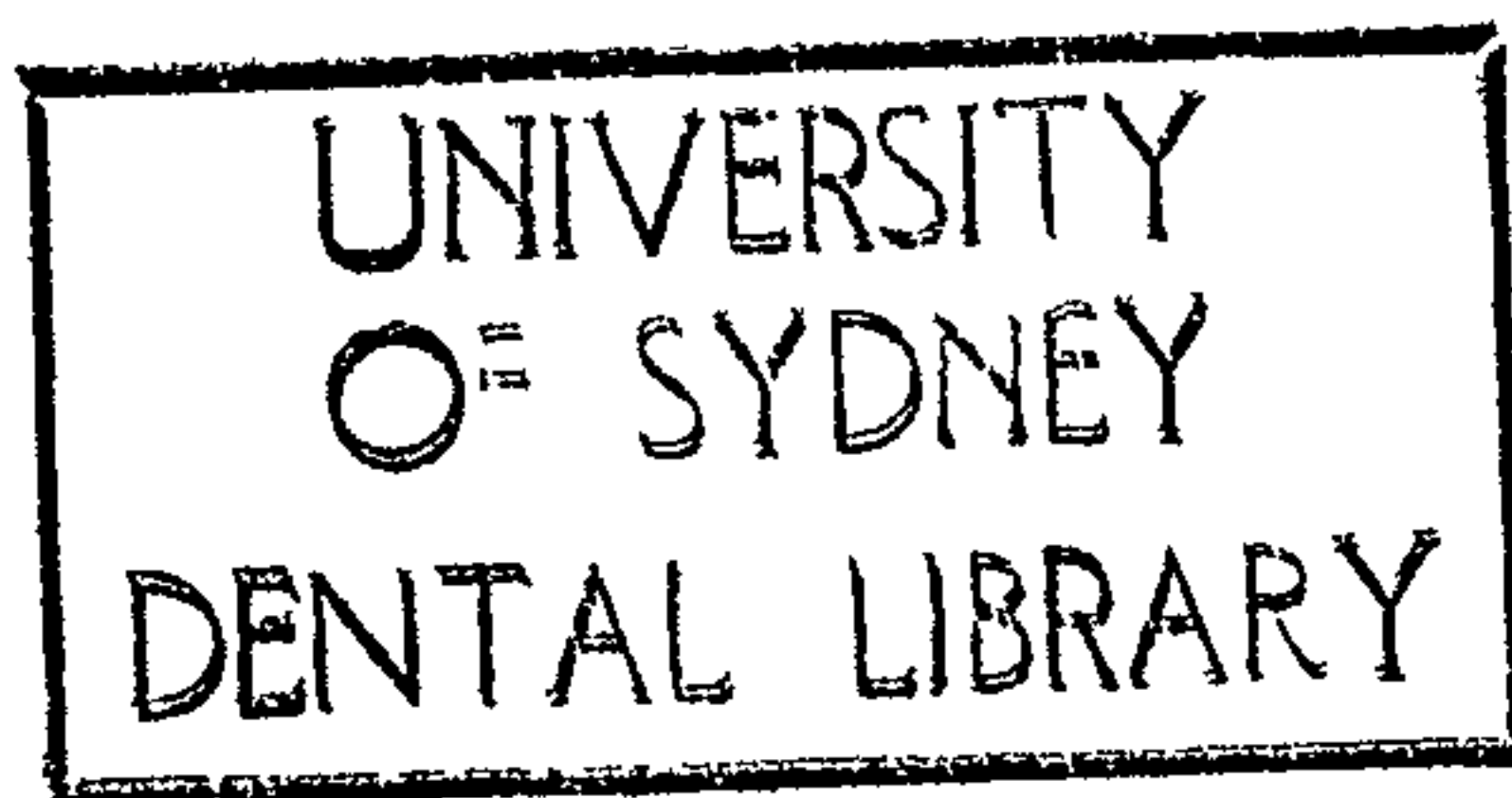


THE OCCURRENCE OF PALATALLY IMPACTED CANINES
IN ASSOCIATION WITH THE ABERRANT (MAXILLARY)
LATERAL INCISOR.

ROBERT WESLEY JOHN CHAPMAN B.D.S. (Syd.Uni.)

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Department of Preventive Dentistry,
Faculty of Dentistry,
University of Sydney,
Australia.

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DEDICATION

To my wife Karen, parents and friends whose patience,
understanding and encouragement have made my career in
orthodontics possible.

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ABSTRACT

This work investigated the apparent association between anomalies of the permanent maxillary lateral incisor and the palatally impacted permanent canine.

After reviewing the etiology and incidence of both impacted canines and anomalous lateral incisors, several theories were postulated to explain this apparent association.

An investigation was undertaken to establish the incidence of anomalies of the lateral incisor in 100 cases presenting with one or more palatally impacted canines, requiring surgical intervention. The incidence was statistically compared to the incidences of similar anomalies occurring in a control group of 100 randomly selected patients, presenting for orthodontic treatment, other than for impaction of the canine.

For the subjects examined, it was determined that no significant difference existed in the incidences of congenitally missing lateral incisors, between the impacted canine and the non-impacted control groups. However when investigating the incidence of lateral incisors with abnormal crown morphology (frankly peg-shaped, peg-shaped or small), a marked significant difference was found between the canine impacted and non-impacted control groups.

It is recommended that:

i) This information may be useful for an earlier diagnosis of deviant canine eruption and the instigation of interceptive measures to prevent palatal impaction.

ii) Further investigation of a general population sample is required to add support to the first recommendation.

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INTRODUCTION

When a patient presents with a palatally impacted permanent canine the clinician faces a treatment-decision dilemma.

Should the clinician remove the impacted canine, compromising the esthetic and functional occlusal result? Should the clinician expose the canine with the hope of stimulating further eruption, but realising the possibility exists of further oral surgery if this proves unsuccessful? Should the clinician commit the patient to full appliance therapy by exposing, placing an attachment and forcefully bring the canine into the arch, accepting this may extend overall treatment time considerably? Or should the clinician just ignore the impacted canine's presence, do nothing and hope that no pathological involvement occurs?

Needless to say, different clinicians select different treatment alternatives depending upon the circumstances related to the particular patient's problem and their previous experiences. However, all clinicians would ponder the wisdom of having been able to instigate interceptive treatment at an early age to prevent the canine becoming palatally impacted.

Dewel (1959), Heath (1961), Mayne (1969) and Moyers (1973) have advocated the use of serial extractions in selected cases to achieve early alignment of the anteriors and correct eruption of the canines. Indeed the use of this technique had prompted Johnston in 1969 to state "I have never seen an impacted canine where serial extraction has been employed."

The phrase 'presurgical recovery' was given by Easthope (1982) to describe his technique of interceptive treatment for palatally impacted

canines in which he applied pressure to the overlying palatal tissue in the hope of altering the canine's path of eruption.

The basis for true success of interceptive procedures lies in the correct and early diagnosis of an impending palatal impaction.

Many authors (Goldsmith, 1931; Mershon and Gunter, 1940; Newcomb, 1959; Williams, 1981; to name a few) advocated the use of serial radiographs, in patients identified as lacking a normal cuspid bulge clinically by the age of 8-9 years, to monitor the eruption of the canines.

It is the belief of the author, that an easier and earlier diagnosis of possible palatal canine impaction, at the age of the permanent lateral incisor eruption, can be made. This belief has been prompted by the author's own experience of observing a number of palatally impacted canines in association with aberrant lateral incisors and by the similar observations of several other investigators (Kettle, 1958; Miller, 1963/4; Bass, 1967; Kay, 1977; Becker, Smith and Behar, 1981).

This thesis was undertaken to determine, firstly, if there exists any possible connection between the suggested etiologies and incidences of palatally impacted canines and aberrant lateral incisors; and secondly, whether the observed association is significant, so that the lateral incisor may be used as an early indication to palatal canine impaction.

LITERATURE REVIEW

CHAPTER 1

THE PERMANENT MAXILLARY CANINE

1.1 The importance of the canine to man's dentition

"Canines are unique in that they have certain responsibilities not fully shared by other teeth". (Dewel, 1971).

The maxillary canine represents an integral unit of the human dentition, being of outstanding importance in both function and esthetics (Bass, 197; Dewel, 1971; Goldsmith, 1931; Hutchin, 1956; Von der Heydt, 1975).

This tooth is found at the angle of the mouth, in a critical strategic position, being the junction between the anterior and posterior teeth, offering one face aligned with the posterior teeth, the other continuous with the incisors; the 'cornerstone' of that arch.

Anatomically the maxillary canines are the "largest and most robust of the anterior teeth and the longest of all teeth" (Gabriel, 1965, p.56). Therefore, they lend themselves admirably to their role in the functional dynamics of occlusion, as advocated by exponents of the theory of cuspid-protected occlusion.

The theory of cuspid-protected occlusion, made popular after the extensive work by D'Amico in 1961 and now being taught by a number of the dental profession (Dawson, 1974; Guichet, 1977; Klineberg, 1983; Ramfjord and Ash, 1971; Roth, 1981; Williamson, 1976); emphasises the dramatic importance of the canine in the functional occlusal scheme.

There can be little doubt that nature intended the maxillary

canine to be one of its foremost mainstays in the human dentition, as shown by its low incidence of agenesis (Hallet and Weyman, 1954).

However, as it will be shown in section 1.4.2 the canine still manages to become impacted within the palate, in a relatively large number of cases.

Most practitioners accept the importance of the maxillary canine to esthetics and function, and would agree "the impacted canine is an object of vital interest to those of us whose duty it is to help preserve the stability and integrity of the human denture" (Goldsmith, 1931).

1.2 Normal development of the permanent maxillary canine

To detect abnormality in the morphology and position of the erupting canine it is helpful to have a clear understanding of the normal development of this tooth.

As with all succedaneous teeth, the permanent canine germ branches off from the dental lamina that connects the deciduous canine bud to the oral epithelium (Noyes, 1960; Provenza, 1972, p.99; Ten Cate, 1980, p.62; Orban, 1976, p.36).

The histologic cross-sectional study by Logan and Kronfeld (1933) indicated that, during the period: birth-to-one year, the different permanent teeth develop to their own unique diversity. The first permanent molars and the permanent central incisors are shown to begin to calcify at, or shortly after birth (Figure 1). At the age of around nine months, the upper and lower central incisors, the lower lateral incisors and the four first permanent molars show considerable progress in calcification (Figure 2). As Logan and Kronfeld (1933) state "the next tooth in line of development is not, as might be expected from the order of eruption, the permanent lateral incisor, but the permanent cuspid which is almost as far advanced in its development as the central incisor." (Figures 1 and 2).

As observed by both Logan and Kronfeld (1933) and Dewel (1949) an interesting and notable event in tooth development is that the canine and the central permanent incisors develop almost simultaneously, although the former erupt five or six years later than the incisors. The upper permanent lateral incisors which erupts practically at the same time as the central incisor is far behind in development but manages to 'catch up', so that at the age of $2\frac{1}{2}$ to 3 years the central

FIGURE 1

DEVELOPMENT OF THE HUMAN DENTITION FROM THE SEVENTH YEAR TO MATURITY

REF: NOYES (1960)

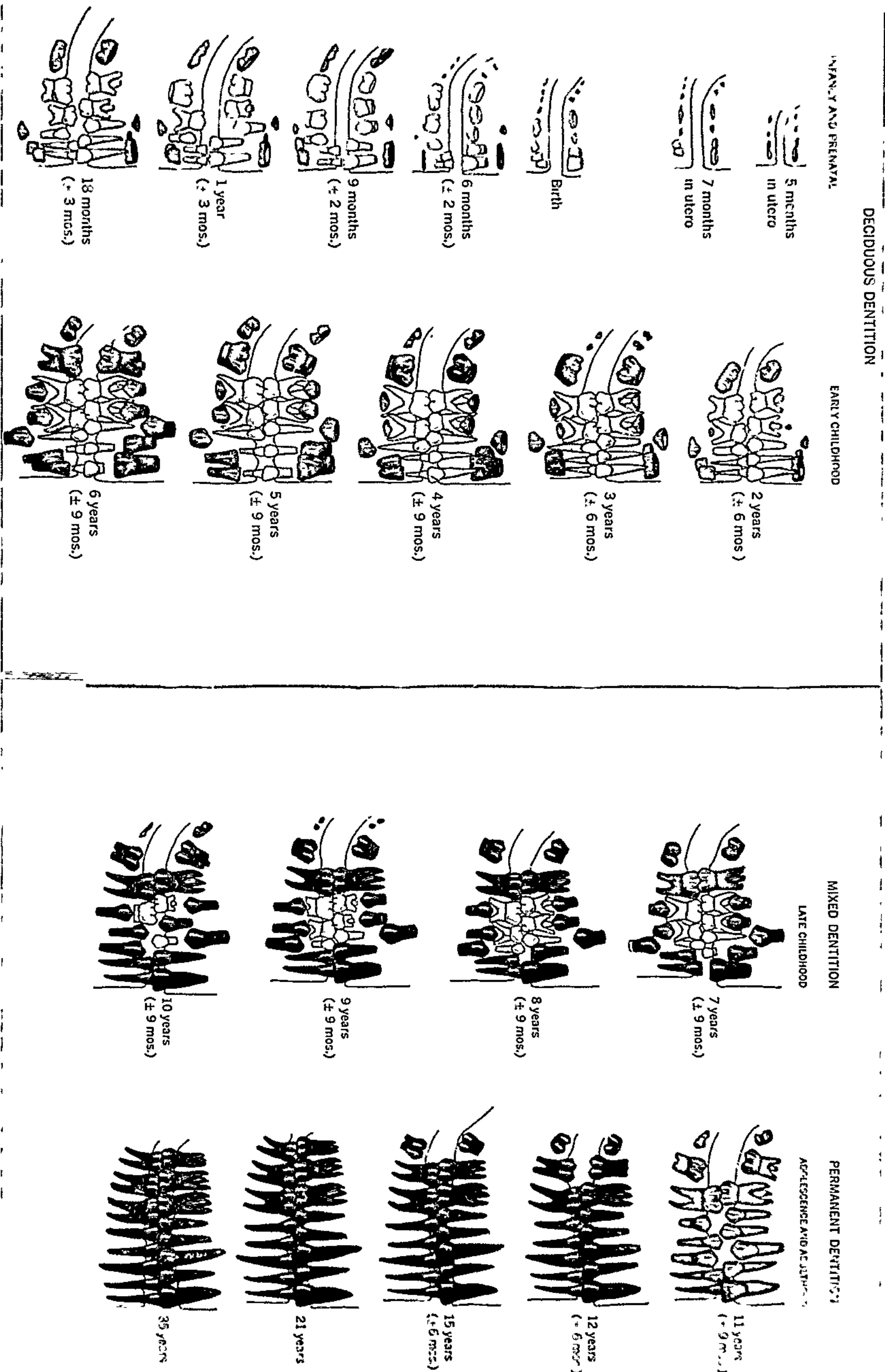
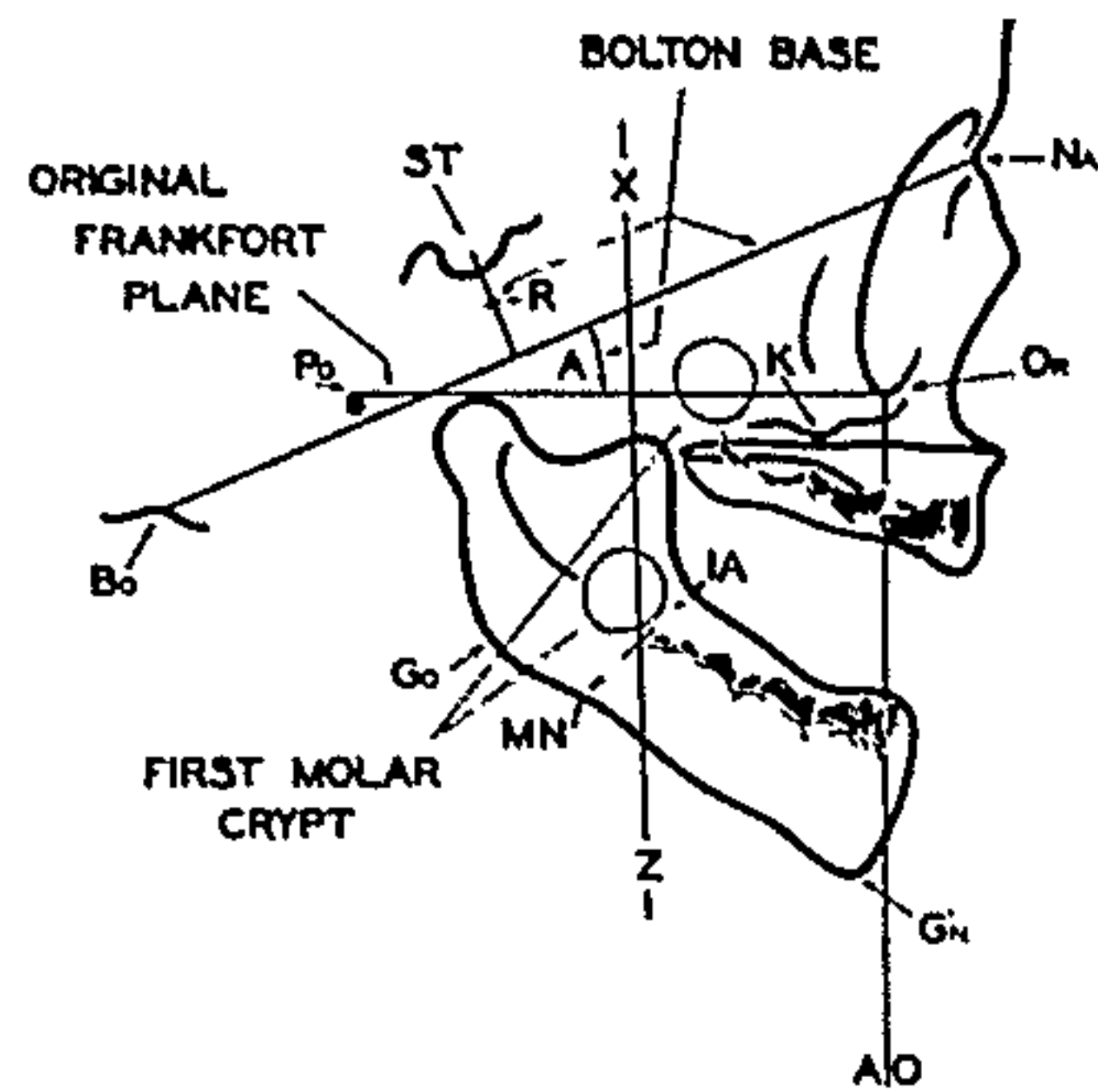


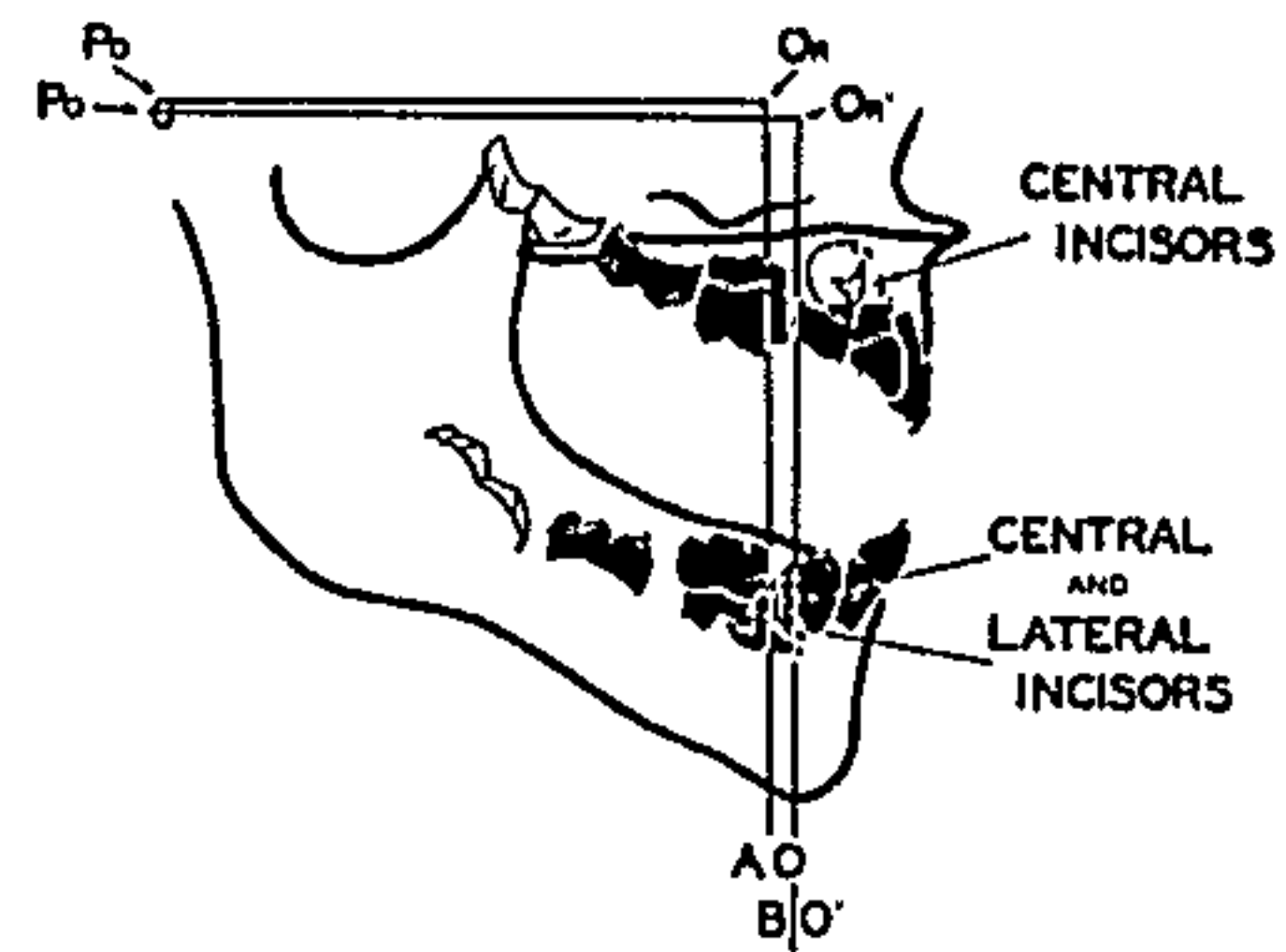
FIGURE 2

DEVELOPMENT OF THE HUMAN DENTITION, 1 MONTH TO 3 YEARS

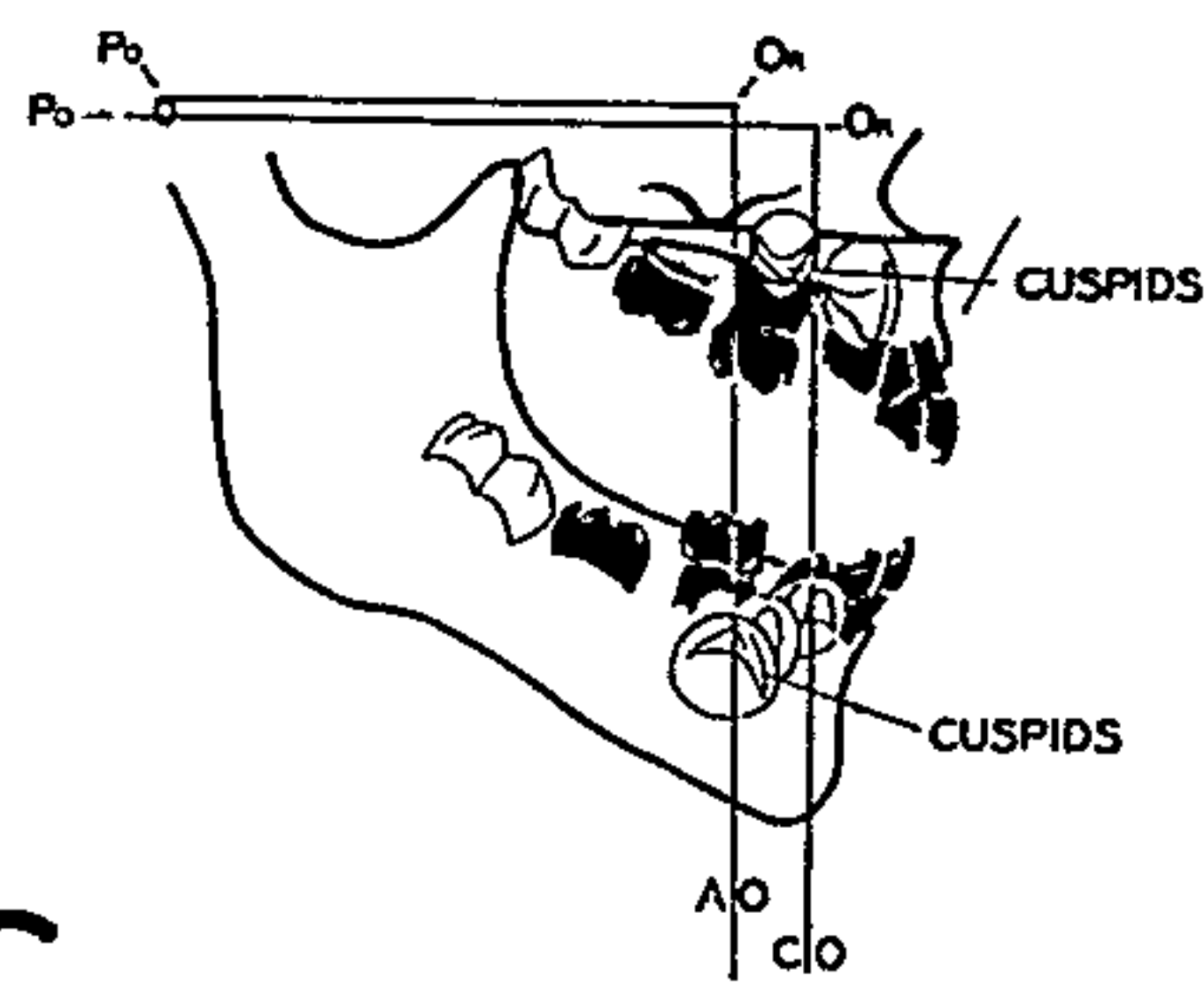
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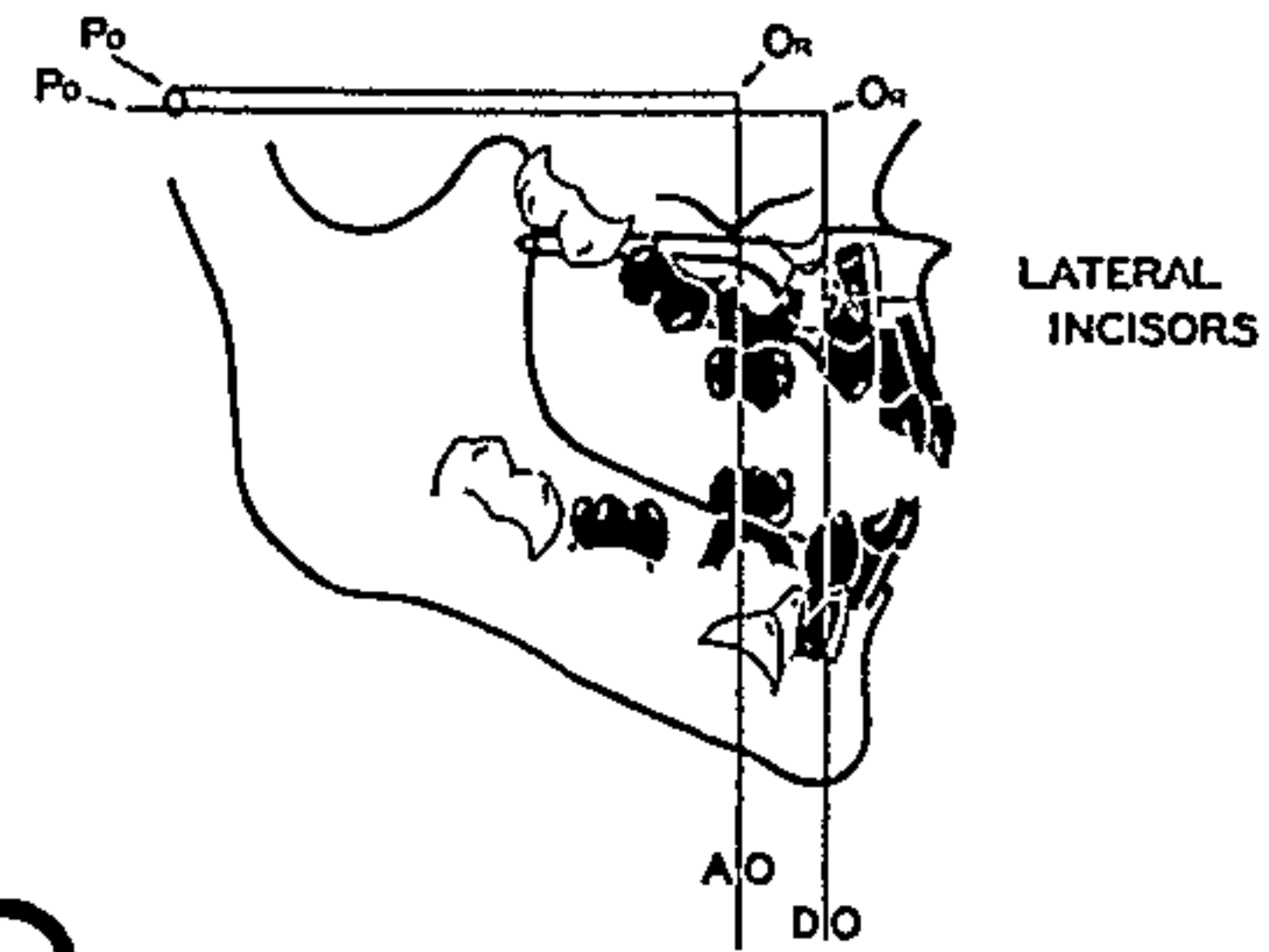
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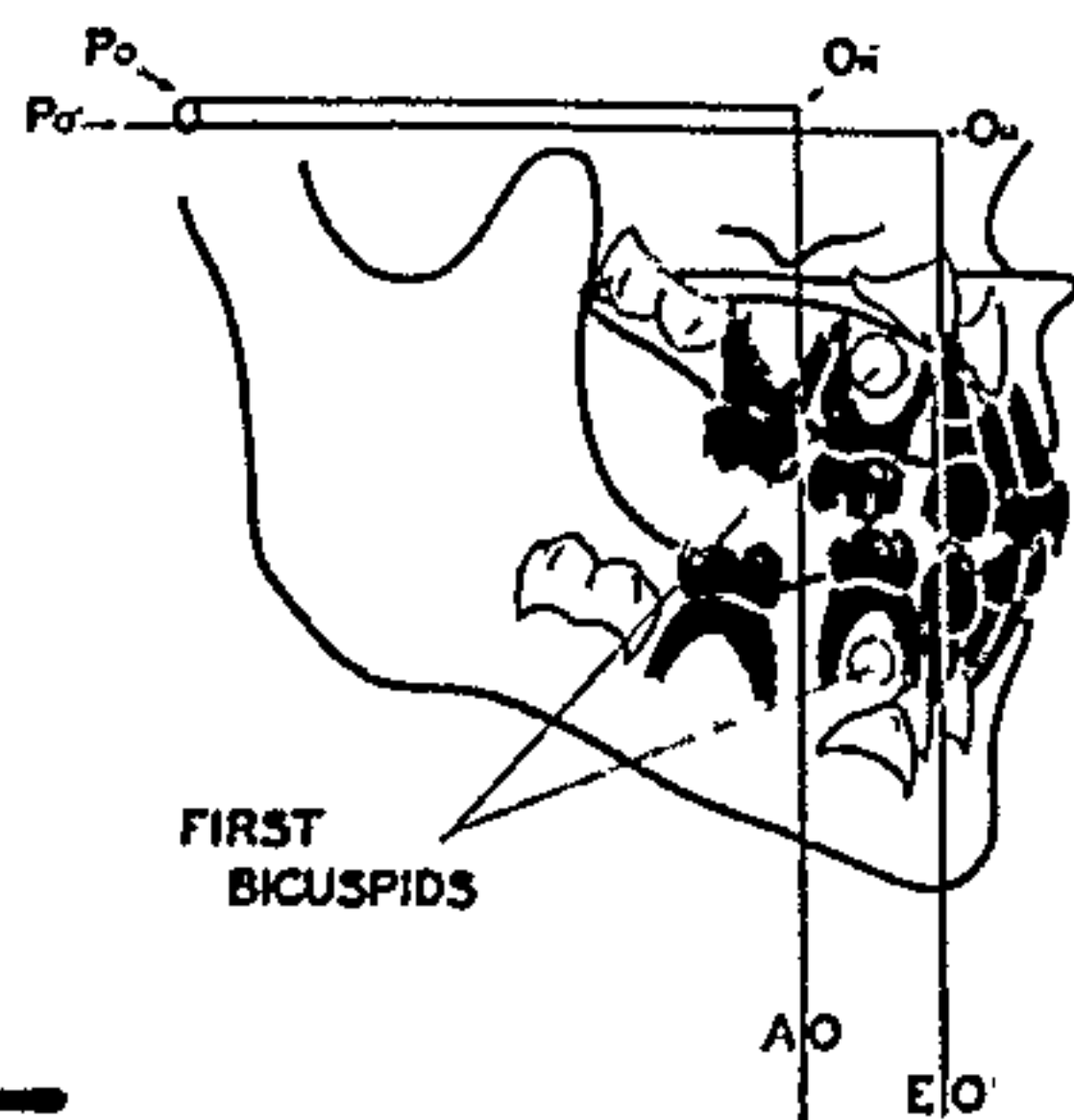
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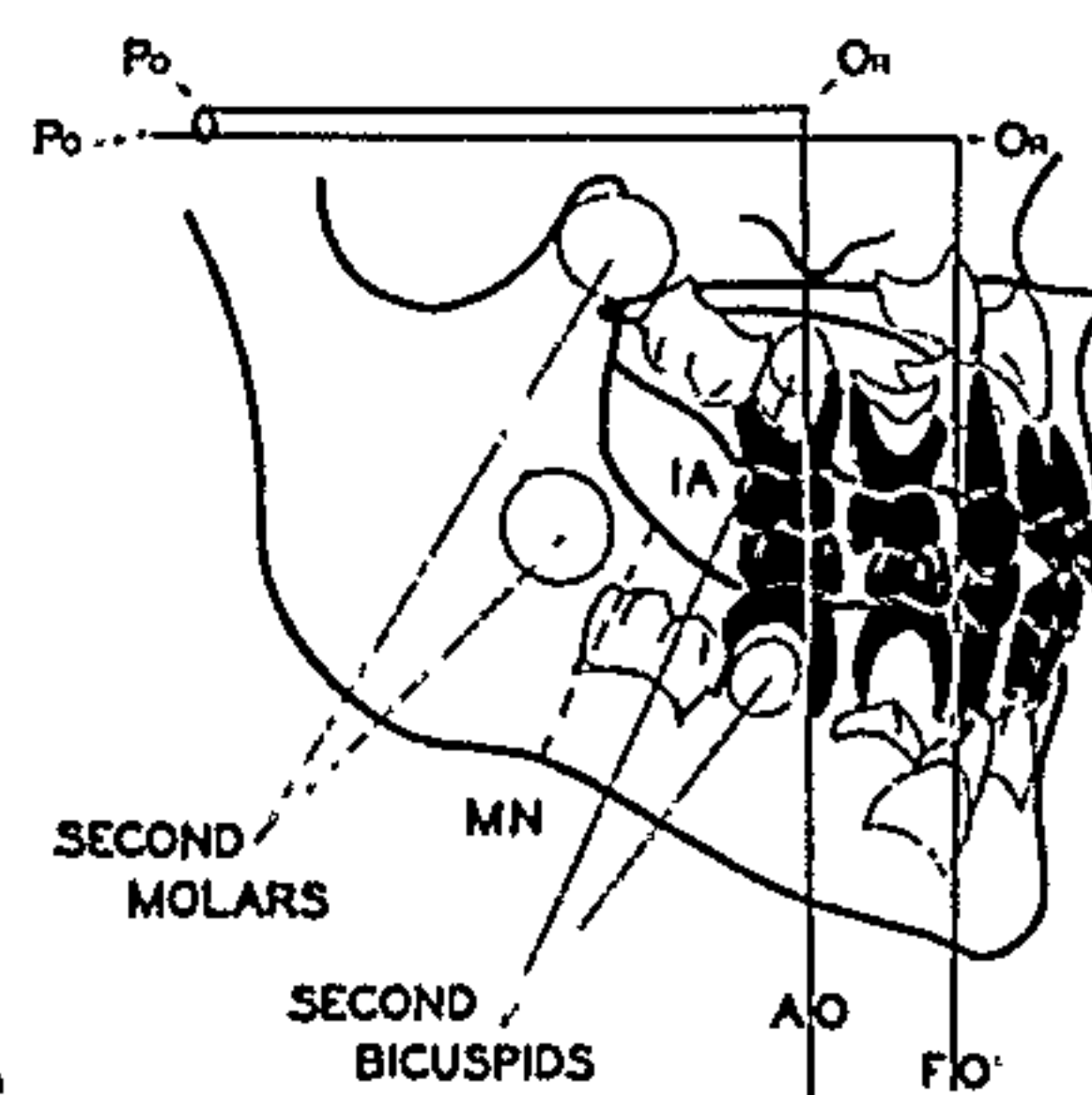
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D 1 1/2 YR.



E 2 1/2 YR.



F 3 YR.

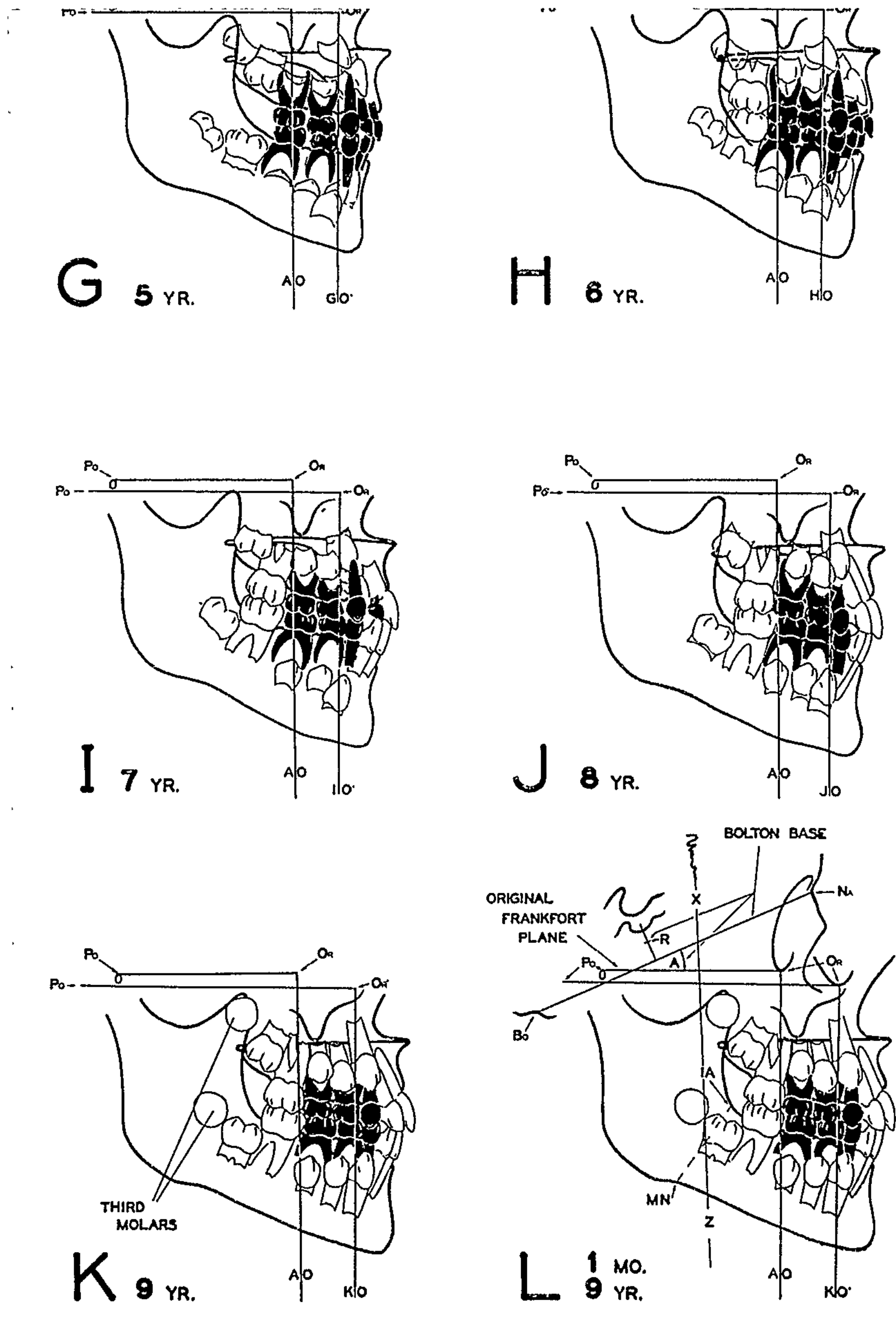
and lateral incisors have reached a nearly identical stage of development. The development of the canine progresses much more slowly. "The calcification of the upper cuspid begins more than half a year before that of the upper lateral incisor, but at the time when the crown of the lateral incisor is fully formed the enamel of the cuspid is still in the process of development" (Logan and Kronfeld, 1933). The crown of the canine is finally complete at approximately 6-7 years (Broadbent, 1941: figure 3).

In studying the calcification of the permanent teeth, Nolla (1960) found that the rate of growth was approximately the same on the right and left sides. Her findings, showing females ahead of males in their dental development, agreed with those of Garn and Lewis (1957).

FIGURE 3

DEVELOPMENT OF THE HUMAN DENTITION, 5 YEARS TO 9 YEARS

REF: BROADBENT (1941)



1.3 Normal eruption of the permanent maxillary canine

Broadbent (1941), F. Noyes, Schour and H. Noyes (1944) and Williams (1981) all emphasized that the term "eruption" had been used throughout the dental literature with two meanings:

Early clinical studies usually referred to eruption as the actual clinical emergence of the tooth into the oral cavity. Later studies with the aid of x-ray diagnosis began to incorporate within the description of eruption, the normal well defined path the tooth travels within the bone, from its crypt to the oral cavity.

Several authors, utilizing this later concept, have accounted in detail the normal eruptive movements of the maxillary canine (Broadbent, 1941; Moyers, 1973; Salzman, 1974).

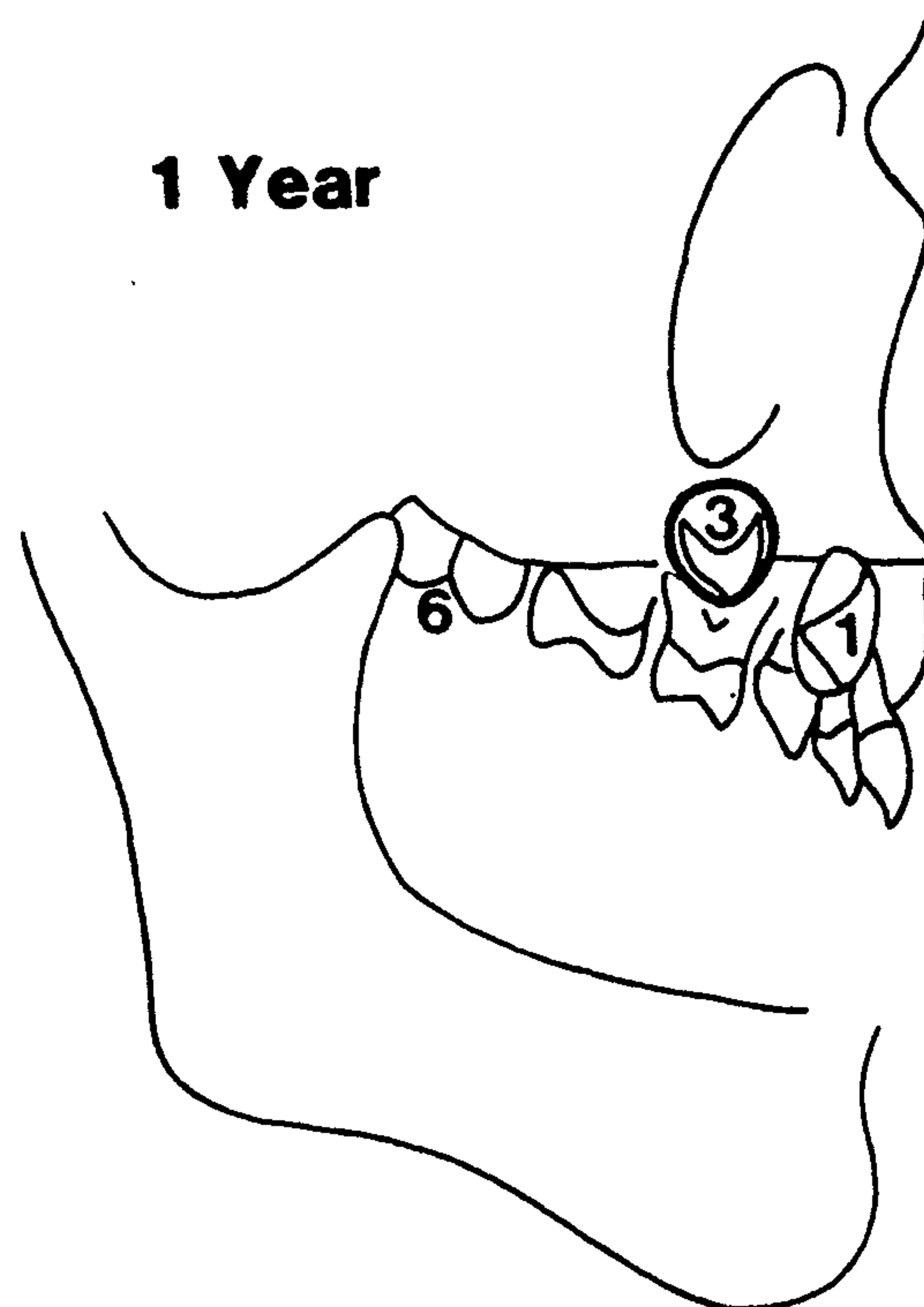
As stated previously the crown of the permanent maxillary canine begins to calcify at 4 or 5 months. At this early developmental stage it lies occlusally and somewhat palatal to the developing deciduous canine, at a higher level than the floor of the nasal cavity and not far below the orbit (Figure 4).

With the maturing of the deciduous canine and increasing alveolar bone the bud of the permanent canine moves farther palatally, so that the deciduous tooth can erupt into the dental arch. At this stage (around 1 year) the permanent canine usually migrates distally as well, so that it is somewhat above the first deciduous molar (Figures 1 and 2). From these relatively deep developmental areas the canines proceed slowly to their final positions in the dental arches.

As the deciduous teeth erupt, the permanent incisor and canine migrate rapidly forward in the maxilla. At age 3 years the canine

FIGURE 4

THE DEVELOPING PERMANENT CANINE (3). THE CANINE IS POSITIONED ABOVE THE BIFURCATION OF THE FIRST DECIDUOUS MOLAR, AT A HIGHER LEVEL THAN THE FLOOR OF THE NASAL CAVITY, AND NOT FAR BELOW THE ORBIT. REF: HUNTER (1981).



now lies just above the apex of its predecessor with its crown directed palatomesially (Figure 2).

According to Atkinson (1942) at the age of $4\frac{1}{2}$ years, the developing canine is changing position above the first premolar and this is in turn above the first deciduous molar, thereby creating the situation where the three teeth are on top of one another (Figure 3).

As facial growth continues to carry the deciduous dentition downwards and forwards, the canine remains high in the maxilla its root development only beginning at the age of 7 years (Nolla, 1960). As the root begins to form the canine moves towards the occlusal plane, the crown lying in close proximity to the root of the lateral incisor (Figure 5).

The cuspid's path brings it down the distal aspect of the lateral incisor root causing the lateral incisor's crown to be distally inclined, a major factor in the 'ugly duckling' stage of normal development (Broadbent, 1941; figure 5).

By approximately 12.5 years the canine has usually reached occlusion, the lateral incisors have uprighed and any remaining spaces between the incisors have closed (Broadbent, 1941; Hunter, 1981).

As can be seen from the foregoing description, and as aptly stated by Dewel in 1949: "Of all the teeth it (maxillary canine) has the longest period of development, the deepest area of development and the most devious course to travel from its point of origin to full occlusion."

FIGURE 5

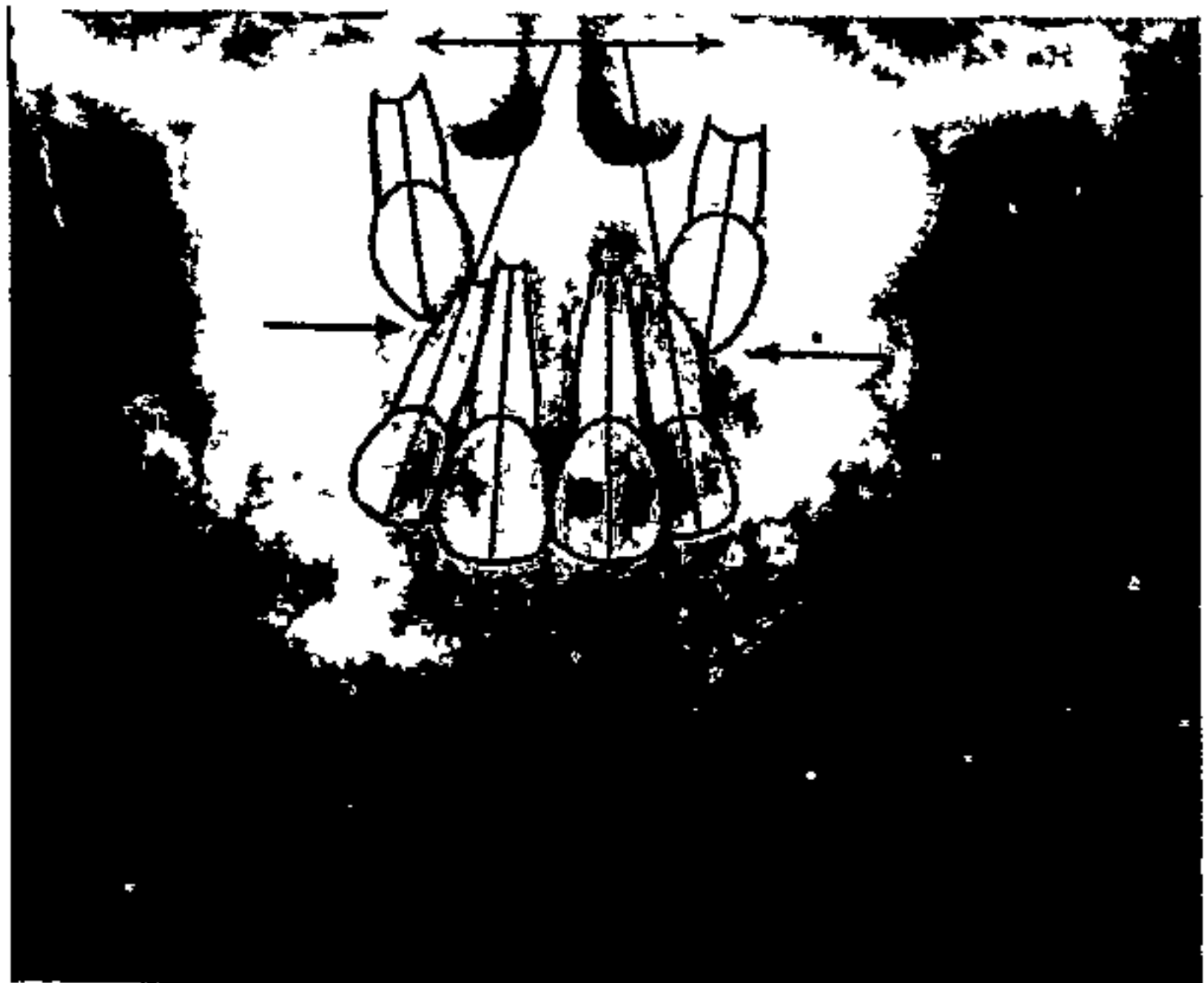
THE 'UGLY DUCKLING' DEVELOPING DENTAL PATTERN
REF: BROADBENT (1941)



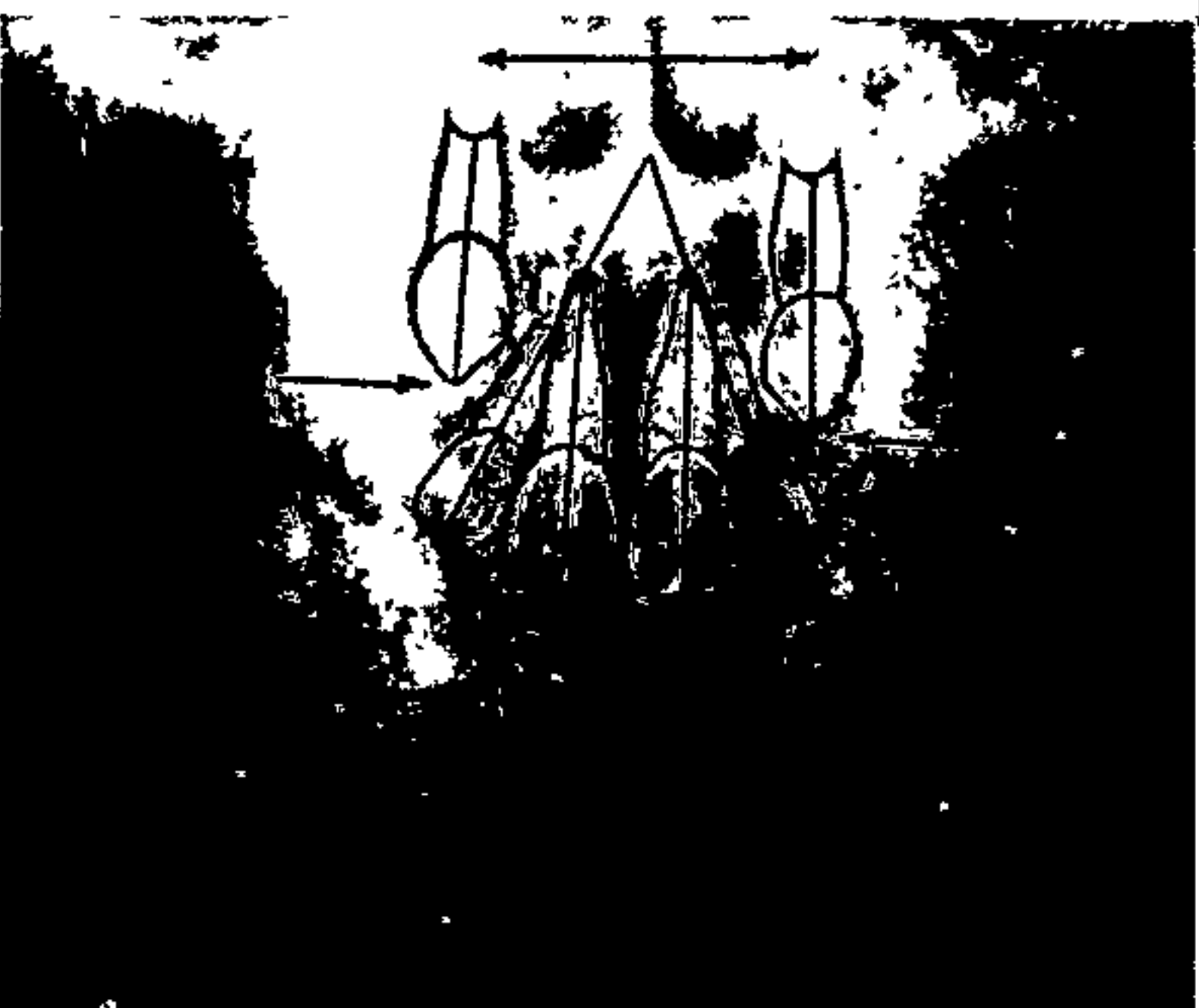
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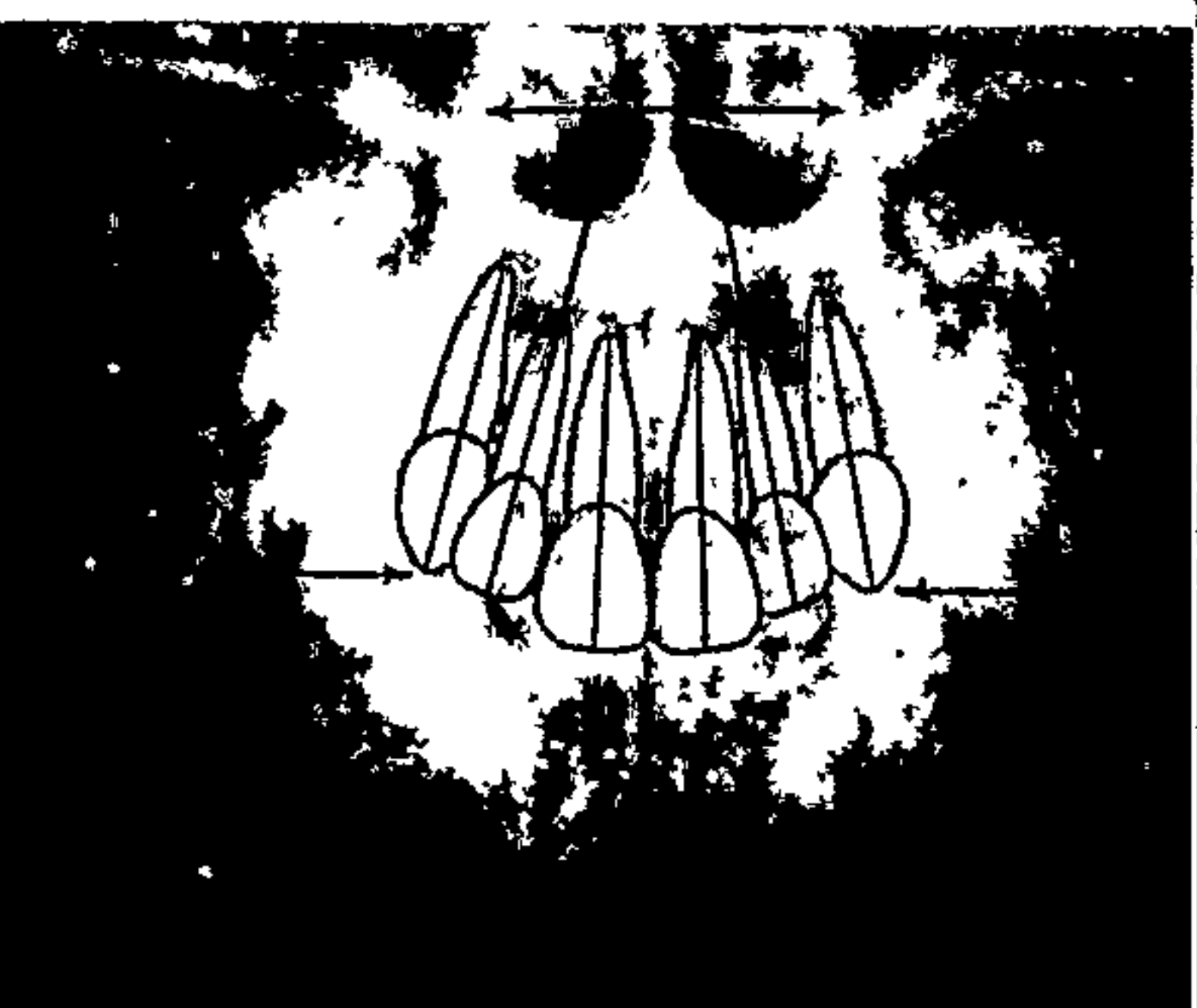
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**"UGLY DUCKLING"
DEVELOPING DENTAL PATTERNS**

1.4 Impaction of the permanent maxillary canine

1.4.1 Definition of impaction

When reviewing the available literature on canine impaction it becomes apparent that several interpretations have been previously utilized for the term 'impaction'.

Boucher (1974, p.203) defines an impacted tooth as:

"Tooth, impacted: A condition in which the unerupted or partially erupted tooth is positioned against another tooth, bone, or soft tissue so that complete eruption is unlikely."

The concept of the impacted canine being diagnosed by its forced eruption against a hard tissue impediment such as tooth or bone, had been adopted by several authors (Blum, 1923; Adamson, 1952; Hitchin, 1956; Beeching, 1979). In the case of an impacted third molar or the partly erupted lower second premolar, this concept in most cases would be readily applicable. It, however, falls short in defining canine impaction where no apparent obstruction other than soft tissue is observed; such as in the case of a high buccal canine, where a 'failure to erupt' rather than 'impaction' would possibly be more appropriate. The shortcomings in the term impaction were recognised by several authors (Von der Heydt, 1975; Helmore and Norton, 1953; Fastlicht, 1954; Mead, 1930; Richardson and McKay, 1982) who attempted to modify their interpretation of impaction to incorporate these situations.

Von der Heydt (1975) suggested that the term impacted was convenient but technically inappropriate and suggested "unerupted would be a better term." Shafer, Hine and Levy (1974, p.63) differentiate between impacted and imbedded teeth; applying imbedded to those "individual teeth which are unerupted usually because of a lack of

eruptive force."

Young (1930) when referring to unerupted canines described them as being "malposed to such an extent as to prevent their eruption and development." The view of the tooth being malposed was adopted by both Granerus (1961) and Gensior and Strauss (1974); the latter qualified this by stating the canines were "not likely to erupt even if space is provided for them in the dental arch."

Young (1930) and later Dewel (1949) also described the impacted canine as having an 'ectopic' eruption or position, being interpreted from Boucher's (1974, p.137) definition of ectopic as meaning a deviation from normal in direction and/or position of the tooth concerned.

Although there appears to be some diversity in the interpretation of the term 'impaction', Moyers (1973) and Pindborg (1970) suggest common usage has applied the term impaction, to any tooth that remains unerupted beyond the time it should normally erupt. The impaction generally being determined by clinical observation and radiographic evidence (Thilander and Jakobsson, 1968).

In the situation of a palatally impacted canine, a radiographic and clinical observation would indicate an unerupted maxillary canine in a patient beyond the chronological age of 12.5 years, believed to be late for the canines appearance (Hunter, 1981; Broadbent, 1941; Hurme, 1948), and that this unerupted tooth, was located palatally to its normal position within the arch.

TABLE 1 STUDIES SHOWING THE INCIDENCE OF IMPACTED MAXILLARY PERMANENT CANINES NR = Not Recorded

Investigator	Year	Sample	Sample Size	Canine Impaction Incidence %	Sexual Difference	Side Preference	Method
Rohrer	1929	German	3,000	2	Female 3:1	Left Side	Cross-sectional radiographic study using modified tube shift (age not given)
Mead	1930	White Nth American	1462	1.6	None	None	Cross-sectional study Full mouth radiographs of office cases presenting for treatment (age not given)
		Eskimo Perovian Indian Mongolian Negro Egyptian Hawaiian Bohemian	4927	0	NR	NR	Cross-sectional study. Careful examination and x-rays where acquired of skulls in the Smithsonian Institution
Dachi and Howell	1961	White Nth American	3874	0.92*	Female 2:1	NR	Cross-sectional full mouth x-rays from Dental Schools of Indiana and Oregon (20 yrs of age) * incidence was presented as a percentage of the normally erupted teeth found in the sample and therefore are low compared with other studies
Bass	1967	Anglo-Saxon	9102	1.65	Female 2:1	Left Side	Cross-sectional study of those patients presenting at Royal Victoria Hospital Bournemouth for surgical treatment, as a percentage of total patients examined (no age given)
Thilander and Jakobsson	1968	Scandinavian children	384	1.3	None	NR	Longitudinal study on children over 7 years (mean age 11.5 yrs to mean age 17.8 yrs)
		Scandinavian adults	39	-	Females 2:1	Right Side	Adults for comparison who had been referred because of impaction
Rayne	1969	Anglo-Saxon	12000	1.5	Adults none Teenagers Females 2:1	None	Cross-sectional study of records of patients with impactions at United Oxford Hospital (> 12 years of age)
Aitasalo Lehtinen and Oksala	1972	Finnish	4063	2.95 ^ø	Female 3:2	Left Side	Cross-sectional OPG study of patients presenting at Dental School of Turku (20-69 years of age) ø May include a small number of mandibular canines
Takahama and Aiyama	1982	Japanese	3367	0.27 + 0.09 ^Δ	Female 3:2	NR	Cephalometric cross-sectional study (22-68 years of age) Δ includes cases with cleft lip and palate

1.4.2 Incidence of impaction

The impaction of the maxillary canine is second only to that of the 3rd molars in incidence (Blum, 1923; Rohrer, 1929; Mead, 1930; Montelius, 1932; Field and Ackerman, 1935; Gunter, 1942; Dewel, 1949; Lappin, 1951; Adamson, 1952; Thoma, 1958; Granerus, 1961; Dachi and Howell, 1961; Adreasen, 1971).

Testifying to the importance of canine impactions to the dental fraternity is the substantial number of investigations reported within the literature, undertaken to establish its incidence (see Table 1).

Data on the prevalence of impacted canines vary widely, with the variation also noted between races. Mulick (1979) gave several reasons why he thought the incidence varied between apparently similar racial groups:

a. The studies have not established criteria for the definition of impaction and

b. Most epidemiologic studies are done from full mouth x-rays alone, without clinical confirmation and hence probably only measure severe impactions.

However, from the available literature written concerning impacted teeth and the results of the numerous investigations conducted, certain characteristics of the incidence of canine impactions are apparent -

1. It appears canine impaction has a greater incidence in the civilised races (1-2% - Rohrer, 1929; Dachi and Howell, 1961; Bass, 1967; Thilander and Jakobsson, 1968; Rayne, 1969), than the more primitive coloured races (0-0.4% - Mead, 1930; Takahama and Aiyama, 1982).

2. Displaced canines are much more frequent in the maxilla than the mandible. (Rohrer, 1929; Goldsmith, 1931; Blum, 1923; Mead, 1930; Thilander and Jakobsson, 1968; Rayne, 1969).

3. The tendency of the malposition in the maxilla to be on the palatal is greater than the buccal (Rohrer, 1929; Goldsmith, 1931; Rayne, 1969).

4. The female has a greater preponderance of displaced impacted canines than the male (Rohrer, 1929; Dachi and Howell, 1961; Dewel, 1949; Bass, 1967; Rayne, 1969; JCO co-operative, 1970) and

5. Finally Rohrer (1929), Bass (1967) and Aitasalo et al (1972) suggested a greater number of canine impactions occurred on the left side; other researchers have found the difference between left and right statistically insignificant (Mead, 1930; Rayne, 1969); while Thilander and Jakobsson (1968) have found a greater incidence of canine impactions on the right side.

1.4.3 Etiology of impaction

The views found within the literature on the etiology of canine impaction can be discussed under the following headings:

- A. EVOLUTION
- B. HEREDITY
- C. GLANDULAR DYSFUNCTION
- D. ENVIRONMENT

A. EVOLUTION

The evolutionary trend towards smaller cranial and facial structures was emphasized by Bjork (1950) when he discussed Lundhom's work on domestication in animals. Lundhom suggested there had been a reduction in the size of the jaws and brain case in animals through domestication, without a proportional diminution in the size of the teeth, resulting in the appearance of crowding. Bjork (1950) suggested this may be similar to the effects occurring in man. Hellman (1936) believed this was indeed the case when he stated "... despite the progressive swelling of the human brain case, the face is continuing to withdraw beneath it. Accompanying this is a gradual diminution in the size of the jaw bones with a consequent reduction in space for the accommodation of the full complement of teeth." Fastlicht (1954) wrote "Deficient mastication in our times undoubtedly has influence on the lack of development of the maxillae."

Mead (1930) believed the evolutionary trend towards smaller arches directly related to an increased number of impactions in the permanent dentition in modern man. Citing Dr Wahl's study on Eskimos, he believed the lower incidence of impactions in the Eskimos occurred since this race had not yet been subjected to modern man's softer diet, requiring less mastication and hence less jaw development. He suggested

in modern man the dentition was undergoing 'atrophic changes', with those teeth (molars and canines) that were evolutionary weakened erupting late and hence, more liable to impaction.

Both Young in 1930 and previously Rohrer in 1929, had concurred with the ideas presented by Mead (1930), believing the canine's position at the limit of the maxilla and premaxilla would undoubtedly involve it if there had been an evolutionary size disturbance. Thomas as late as 1960, virtually repeated the ideas of Mead (1930) when he wrote, "The most common etiologic factor producing greatly delayed eruption or non-eruption of the canines and third molars is underdevelopment of the jaws Due to soft foods introduced by the past generations, the amount of masticatory force required and the time consumed in its application have been constantly diminished and today the functional development of the jaws has decreased considerably, with the result the jaws are too short to contain all the teeth."

While agreeing with the principle of evolutionary modification, Goldsmith (1931) draws our attention to the conclusion made by Todd (1929) that the human jaws "are not undergoing evolution ... the crowding of the teeth and consequent malocclusions and other anomalies are due entirely to generations of urbanisation. Just as soon as the proper care of the teeth and jaws is instituted better teeth and occlusion will reappear."

Whether man's facial structures have undergone evolutionary change; whether this be due entirely to the use of a softer diet requiring reduced mastication; and whether this evolutionary modification has produced a decrease in jaw size without a concomitant decrease in tooth size, is still under conjecture. If an evolutionary change has however produced smaller dental arches and crowding of the permanent

teeth, it could be feasible that in some Caucasian and Mongoloid groups an increase in the number of impacted teeth may occur.

B. HEREDITY

The controversy as to the hereditary nature of malocclusions appears to have existed since the beginning of dental research. Whether the dentition of one parent can be inherited with the facial structures of the other parent has still not been resolved (Kinsella 1967).

It is plausible the factors responsible for canine impaction could be inherited. Lappin (1951) reported relatives who were similarly affected in many cases with impacted canines, while Leach (1968) reported twins each with canine impaction. Bass (1967) although not defining the term impaction showed that a high proportion of patients with 'impacted' canines had partial anodontia and suggested that the inherited defect responsible for the agenesis might also be the inherited defect responsible for canine impaction. Similarly, Richardson and McKay (1982) proposed the hypothesis that canine impaction (not defined) may be part of a hereditary syndrome, arriving at this based on their experience and observation of canine impactions within family groups, in monozygotic twins and in association with congenital anomalies of the lateral incisor.

Although the genetics concerning canine impactions can only at this period in time be speculative, circumstantial evidence does exist that canine impaction or factors in association with an impacted canine, may be inherited.

C. GLANDULAR DYSFUNCTIONS

A disturbance in the function of the Endocrine glands have both a direct and indirect bearing on the etiology of impacted teeth, depending upon the stage of development at which the disturbance takes place and the duration of the dysfunction. The thyroid gland controls general metabolism by secretion of thyro-globulin and controls bodily growth and development by complementing the function of the pituitary gland (Shafer, Hine and Levy, 1974). Disturbances in the secretions of the thyroid gland manifest themselves as disturbances in growth and development, reflected dentally as crowding and impactions. Similarly, dysfunction in the pituitary gland in early childhood can result in a delay or lack of growth and development of the cranium and face, contributing to the associated constricted arches, crowding, delayed eruption and impaction of teeth (Salzman, 1974). Retarded eruption was shown to occur in hypothyroidism and hypopituitarism by Bryer (1957).

Although several authors (Kay, 1977; Bishara, et al 1976; Moss, 1972; Rayne, 1969) have suggested glandular dysfunction as a possible cause of canine impaction, the actual role of the glandular secretions in development and eruption of teeth is unknown. It is possible that subclinical disorders of the endocrine glands, although not recognised, may be a factor in impaction of teeth, since gross clinically recognisable disturbances result in skeletal and dental aberrations.

D. ENVIRONMENTAL FACTORS

A large number of environmental factors, local and general, have been suggested as possible causal factors in canine impaction.

Local Environmental Factors

a) Factors associated with the permanent canine

I. Developmental period and path of eruption

Payne in 1930, suggested that the canines being late in the eruptive sequence where the teeth more probable to meet with obstruction on their way into the arch. Goldsmith (1931) reiterated Payne (1930), when he conjectured the canine in making its descent into the line of occlusion ran a "far more hazardous gauntlet than the other teeth" and hence was more often deflected.

Not only was the distance and course of eruption emphasized by Dewel (1949), he also proposed that the overall time for the development of the canine was an important factor in its impaction. As stated previously (sections 1.2 and 1.3), the canine begins to calcify by the age of 6 months yet calcification of the crown is not completed until approximately 6-7 years, with final position within the arch not attained until the 12-13th year. Dewel (1949) concluded that with "so many opportunities in time and position for deflection from a normal course, it is surprising that the cuspid so often finds its way into a reasonably normal occlusion." This conclusion was similarly drawn and restated by many authors as the likely cause of the canine impaction (Lappin, 1951; Helmore and Norton, 1953; Kettle, 1957; Miller, 1963/4; Archer, 1975; Moss, 1972; Gensior and Strauss, 1974; Mulick, 1979; Olow-Nordenram and Anneroth, 1982).

II. Malposed canine tooth germ

Young in 1930 believed the malposition of the canine tooth germ to be the principal cause of the unerupted canine, the reason being that palatal malposition of the tooth germ had been observed in 75% of his impacted canine cases. Thilander and Jakobsson (1968), Craig (1968), Pindborg (1970), Kay (1977) and Mulick (1979), all agreed abnormal location of the developing tooth bud would allow eruption to take place in an atypical direction. It was of interest to note that Young (1930) did not elaborate on the reason behind the malposition of the tooth germ. However Richardson and McKay (1982) proposed their hypothesis that it was genetically determined, this being based on experience rather than scientific investigation. If this hypothesis was correct, it would indicate that not only could the malposition of the tooth germ be through local disturbance but may have been genetically determined as part of an overall hereditary design.

III. Changing Axial inclination of the erupting canine

A palatal deflection of the erupting canine may be the result of a slight change in axial inclination of the tooth during development. Hovell (1966) believed the mesial inclination that occurred during development to be an important factor in canine impaction etiology.

Miller (1963/4), Thilander and Jakobsson (1968), Moyers (1973) and Marsh (1965) all mentioned abnormal axial inclination or rotation of the tooth buds as a possible cause for palatal deflection of the erupting canine.

IV. Presence of an enlarged follicle or dentigerous cyst in association with the canine

It has been suggested, that an enlarged follicle or cyst associated with an erupting canine would act as a cushion and negate the guidance afforded by the adjacent lateral incisor root; allowing the canine to be diverted from its course (Mershon and Gunter, 1940; Fastlicht, 1954; Kettle, 1957; Mulick, 1979; Lappin, 1951; Rayne, 1969; Olow-Nordenram and Anneroth, 1982).

Tradner (1948), cited by Thilander and Jakobsson (1968), stated that enlarged follicles were associated in 30% of impacted canine cases. However, Thilander and Jakobsson (1968) found 96% of all canines have enlarged follicular spaces, there being no difference between their control group (non impacted) and those that were impacted. As an explanation, Richardson and McKay in 1982 suggested that a prominent crypt around an unerupted canine is a frequent radiological finding, but at what point it can be diagnosed as being a cyst or enlarged, is arbitrary.

V. Abnormal morphology of the canine

Hitchin (1956) suggested that the nature of the buccal surface of the canine may contribute to the palatal deflection. An extremely bulbous character would place the incisal tip deep to the apex of the lateral incisor root, increasing the chance of guidance to the palatal side. Abnormal crown morphology was listed by several other authors (Di Biase, 1971; Mulick, 1979; Pindborg, 1970).

b) Factors associated with the deciduous canine

I. Lack of root resorption

The lack of proper root resorption and hence overretention of the deciduous canine, has often been cited as a major obstruction to correct canine eruption (Angle, cited by Goldsmith, 1931; Mershon and Gunter, 1940; Helmore and Norton, 1953; Fastlicht, 1954; Kettle, 1957; Hitchin, 1956; Rayne, 1969; Archer, 1975; Moss, 1972; Moyers, 1973; Kay, 1977; Mulick, 1979; Richardson and McKay, 1982).

However, conclusions drawn by Thilander and Jakobsson (1968) from their longitudinal study into the etiologic factors of canine impaction, suggests the idea of a persistent deciduous predecessor may be the consequence rather than the cause of canine impaction.

II.. Periapical pathology

Over-retention of the deciduous predecessor has also been felt to be associated with periapical pathology of this tooth. Similar to the cushioning effect proposed occurring with an enlarged canine follicle (section a-IV), the collagen-like mass of a granuloma could be capable of deflecting an erupting canine into the palate (Mershon and Gunter, 1940; Fastlicht, 1954; Bloch-Jorgenson, 1931; Kettle, 1957).

Richardson and McKay (1982) although admitting that the granuloma could alter the erupting canine's course, question the susceptibility of this tooth to decay and also suggest that it is not usually vulnerable to accidental trauma.

III. Early loss of the deciduous canine

The early loss of the deciduous canine could produce a loss of arch length in that area, resulting in a subsequent lack of adequate space for the permanent canine, and hence canine impaction.

(Mershon and Gunter, 1940; Cryer, 1911; Adamson, 1952; Fastlicht, 1954; Kettle, 1957; Hitchin, 1956; Rayne, 1969; Dewel, 1949; Lappin, 1951; Kay, 1977).

c. Factors associated with the lateral permanent incisors

Agensis, pegging and abnormal position of the permanent lateral incisor have often been suggested as being in association with an impacted maxillary canine (Hitchin, 1956; Kettle, 1957; Miller, 1963/4; Bass, 1967; Rayne, 1969; Moss, 1972; Kay, 1977; Becker, Smith and Behar, 1981).

Several theories have been advanced to explain this association; these will be discussed in section 3.

d. Other factors associated with obstruction

The presence of supernumeraries in the path of eruption has been suggested as a cause for preventing eruption of the canine (Mershon and Gunter, 1940; Fastlicht, 1954; Thilander and Jakobsson, 1968; Payne, 1930; Kay, 1977; Pindborg, 1970).

However, while contemplating the supernumerary as a factor, Lappin (1951) and Helmore and Norton (1953) suggested that its presence was so rare in the cuspid region it could not be of great importance (0.4% as suggested by Shafe, Hine and Levy 1974, p.44). Hitchin (1956) found only 7 supernumeraries out of the 109 impactions he described (6.4%); these included cleft lip and palate cases. Rayne (1969) found

only 1% in the series investigated by himself.

Several other structures have been implicated to cause obstruction, these have been: roots of adjoining teeth (Case, 1921); Odontomes (Mershon and Gunter, 1940; Pindborg, 1970; Kay, 1977; Olow-Nordenram and Anneroth, 1982); dense bone and fibrous scar tissue (Helmores and Norton, 1953; Moyers, 1973; Kay, 1977) and epithelial remnants (Rayne, 1969).

e) Factors associated with an abnormal eruption sequence

The most frequently found sequences of eruption in the maxilla are (Lo and Moyers 1953): 6 1 2 4 5 3 7 8 (48.72%)

or 6 1 2 4 3 5 7 8 (16.01%)

These sequences, have been verified to also be the most common in New South Wales school children by Gates (1963).

From these sequences, the canine can be seen to normally erupt between the already present lateral incisor and first premolar, and since its predecessor's diameter is much less, it must wedge its way into the arch utilising some of the available leeway space (Dewel, 1949; Gensior and Strauss, 1974). Early loss of a deciduous molar, canine or an unusual eruption sequence whence the second permanent molar erupts prior to the canine, may lead to a loss of available leeway space and the occasional simian space, adversely affecting the eruption of the canine (Mitchell, 1960; Archer, 1975; Moyers, 1972; Salzman, 1966; Dewel, 1949; Richardson and McKay, 1982).

f) Factors associated with crowding

A number of authors (Rohrer, 1929; Mead, 1930; Bass, 1967; Rayne, 1969; Moyers, 1973) have implied crowding within the arch has often been associated with, and may be a major contributing factor in, canine

impaction. Fastlicht (1954) went as far as to state that "all authors are in agreement that one of the principal causes of canine impactions is the lack of space". Thilander and Jakobsson (1968) observed during their longitudinal study into local causes of canine impaction, that crowding tended in general to be associated with impaction, and stated "crowding seems to delay eruption".

Crowding in the maxilla has been suggested to be an indication of an overall reduction in the size of the arch. Cryer in 1911 believed anything that interfered with the normal growth of the maxilla would result in a reduced jaw size and associated crowding, with increased incidence of impaction. Several authors agreed that a reduced arch dimension in both width and length could contribute to canine impactions (Hitchin, 1956; Kettle, 1957; Rayne, 1969; Kay, 1977). Mulick (1979) gave as an example the case of facial stenosis, an extreme form of maxillary hypoplasia. Rohrer (1929) believed reduced arch size could explain the high preponderance of canine impactions in the female, since they were the sex with the smaller diminutive face.

Fastlicht in 1954 proposed that a case of false mandibular prognathism would create a lack of anterior arch development in the maxilla, with subsequent crowding and canine impaction.

Kettle (1957) and Rayne (1961) believed that incisor retroclination could decrease the available space for the canine, while Bass (1967), following similar ideas, found in Class II malocclusions with proclined incisors a lower incidence of canine impactions.

However Dewel (1949), Young (1930) and Helmore and Norton (1953) considered contracted arches played no role in canine impaction with Dewel (1949) stating "curiously unerupted cuspids frequently occur

in quite normal arch form cases. Therefore, the cause is usually not due to crowded or contracted arches." The suggestion being that it was due to a multitude of different causes.

General Environmental Factors

Dewel (1949) believed any environmental factor which retarded growth in the canine region or interfered specifically with tooth development immediately results in a tendency to impaction. Previously, Gunter (1942) had suggested that the causal factors concerning malocclusion and impaction must primarily influence the mechanism of bone growth, regardless of the etiology concerned. Adamson in 1952 reiterated both Gunter (1942) and Dewel (1949) when he stated "many canine impactions occur as the result of abnormal growth and developmental processes."

Possible factors having an influence on canine development and maxillary growth are:

a) Trauma to the maxillary region

Trauma to the developing tooth germ by either an injurious blow to the deciduous dentition, or surrounding alveolar bone may cause abnormal growth of the maxilla or disorientation of the developing canine (Cryer, 1911; Mershon and Gunter, 1940; Di Biase, 1971; Moyers, 1973; Kay, 1977). Pindborg (1970) and Kay (1977) suggested that the resultant dilaceration and ankylosis of the canine would eventuate in its impaction.

Persistent deleterious oral habits have also been implicated in distortion to the developing canine and surrounding region, resulting in possible palatal deflections (Fastlicht, 1954; Moyers, 1973).

b) Developmental abnormalities

Any developmental abnormality having an effect on the growth and development of the maxilla or developing tooth germs can result in canine impaction. Achondroplasia and cleidocranial dysostosis have been seen to influence the development of the maxilla and have been associated with dental impactions and other anomalies (Rayne, 1969; Pindborg, 1970; Moss, 1972).

Cleft lip and palate is an extreme example of a developmental deformity influencing the maxillary structures. Disturbances in the eruption of the maxillary permanent teeth are often seen in cleft patients. This may be due purely to the disturbance of the dental lamina by the surgical procedure (Godfrey, 1983), or by the subsequent distorted underdevelopment of the maxilla.

Takahama and Aiyama (1982) believe, however, canine impaction may be a microform of the cleft lip and palate. They postulated this hypothesis after their finding of a higher incidence of maxillary canine impaction in close relatives of cleft patients, than that found in the general population of Japan.

c) Diseases

M.H. Cryer in 1911 suggested that, deformed impacted and misplaced teeth should be studied not only as diseased conditions in themselves, but be regarded as symptoms of some precedent pathological disturbance. He suggested syphilis, scarlet fever, measles, rickets and scurvy could all manifest themselves as impactions of the dentitions.

d) Nutritional deficiencies

Nutritional deficiency has often been linked to irregularities of the dentition and faulty bony development (Salzman, 1957; Moyers, 1973; Cryer, 1911; Gunter, 1942).

The elements considered essential for normal growth and development of mammals are calcium, phosphorous, magnesium, potassium, sodium, chlorine, iodine, copper, iron, zinc, manganese, cobalt and probably fluorine. Inorganic and organic combinations of these elements are active in many physiologic processes. They constitute the basic structure of bone and teeth (Shafer et al 1974).

Vitamins and minerals also play a major role in correct bone and teeth development.

Dalldorf and Zall (1930) found a lack of vitamin C in guinea pigs retarded eruption.

e) Intra uterine environment

As previously suggested, anything which retarded growth in the canine region or which interfered with the position of cuspid development immediately results in a tendency to impaction. Malnutrition, alcohol, diseases of serious character (rubella in early months of pregnancy) etc. all are thought capable of altering foetal growth. It is possible that the alteration may occur in the midface region influencing the developing maxilla with the consequence of possible canine impaction.

1.5 Summary

As can be seen from the previous review many reasons have been postulated for the canine becoming impacted. These have been discussed under the headings of:

Evolution
Heredity
Glandular dysfunctions
Environmental

If we consider the relatively high incidence of canine impactions, it becomes apparent why there is a seemingly unending variety of conjectured causes for the canine impaction. With the relatively high frequency in the general population, it is statistically possible that canine impaction will be associated with a variety of other events. Whether these events are related can be at times, highly debatable. It is only too easy for a clinician to diagnose two events and assume that those two events are related by suggesting that one event may be the cause or affect of the other event or vica versa. However it must be remembered that the situation of those events being completely isolated entities, is also a distinct possibility.

This possibility becomes extremely probable, when considering many of the conjectured environmental factors said to be directly attributable to canine impaction.

The assertion by Dewel (1949) that the canine has a more tortuous path of eruption than any other tooth and its impaction can be directly attributed to the length of development, and the time and distance covered while erupting, generalizes the reason for the influence of many of the cited factors upon the canine. This is one of the more plausible reasons for the canine impaction, the difficult path of eruption having been

described in detail histologically by Logan and Kronfeld (1933) and radiographically by Broadbent (1941).

The hypothetical ideas that the characteristics of canine development and impaction are in some way a part of a yet undefined hereditary design to reduce the size of man's facial structure, with the evolutionary changes being inherited by succeeding generations, is still within the realms of possibility. This, however, at present remains controversial, since irrefutable proof is yet to be documented.

One thing is certain, the etiology of canine impaction (and that, for any malocclusion) is difficult to define, due to the complex nature of the environment's influence on growth and development and the yet undefined specific role of heredity and evolution.

The conclusion drawn by Goldsmith in 1931 that, "the forces involved in the etiology of impactions are of a conflicting character and many of the statements made are difficult to prove", after many intervening years of research, is still relevant today.

CHAPTER 2

THE PERMANENT MAXILLARY LATERAL INCISOR

2.1 Anomalies of the maxillary lateral incisors

The maxillary lateral incisor has a wide range of variability in its appearance. It is quite often congenitally missing or reduced markedly in size (Scott and Symons, 1964; Gabriel, 1965; Sicher, 1980; Barker, 1973). The most common size deformity being that the teeth are conical or peg shaped (Scott and Symons, 1964), but a range of variations occur between the normal stem type and the peg-lateral (Figure 6).

The normal 'stem' type is shown to be similar to the anatomy of the central maxillary incisor but with its own specific characteristics (Gabriel 1965). It appears to be absolutely smaller in both length inciso-apically and mesio-distal width when compared with its mesial neighbour. When the crown is viewed from both the palatal and labial sides a distinct convergence can be noted towards the cervical margin (Figure 7). The labial surface is rarely grooved with the lingual surface being more deeply concave than the first incisor. Often the lateral incisor has a deep pit incisal to the tubercle on the palatal known as the foramen cecum; this is rarely seen on the central.

It can be seen from the figures presented by Black (1902) and Moorrees (1959) (tables 2 and 3) that the average mesio-distal crown diameters of the upper lateral are less than the upper central incisor but are greater than that of its mandibular counterpart.

FIGURE 6

VARIATION IN MAXILLARY LATERAL INCISOR FORM SHOWING LABIAL,
LINGUAL, DISTAL AND INCISAL VIEWS OF THREE RIGHT AND THREE
LEFT UPPER LATERAL INCISORS

REF: GABRIEL (1965)



FIGURE 7

THE RIGHT MAXILLARY LATERAL INCISOR 'STEM TYPE'
REF: BRESCIA (1961)

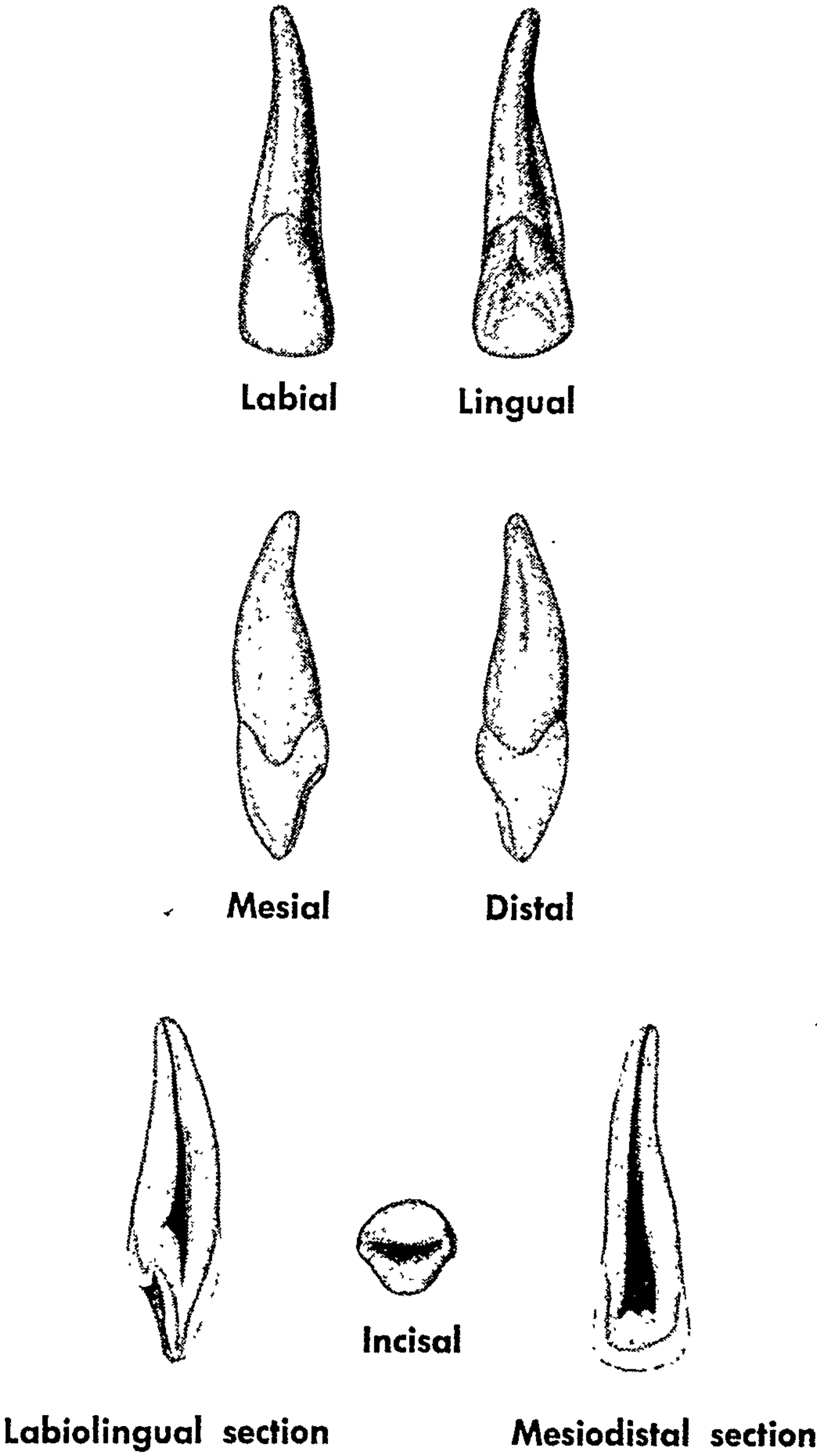


TABLE 2
MESIODISTAL CROWN DIAMETERS OF PERMANENT TEETH
REF: MOOREES (1959)

Tooth	Sex	Mean (mm)	S.E. _M (mm)	S.D. (mm)	C.V. (percent)	Range (mm)	Number
<i>Maxilla</i>							
<i>I</i> ₁	♂	8.78	0.05	0.46	5.29	7.9-10.0	87
	♀	8.40	0.06	0.53	6.30	7.1-9.8	87
<i>I</i> ₂	♂	6.64	0.07	0.63	9.42	4.5-8.2	84
	♀	6.47	0.07	0.62	9.60	4.5-8.5	86
<i>C</i>	♂	7.95	0.05	0.42	5.24	6.6-9.0	87
	♀	7.53	0.04	0.37	4.94	6.9-8.5	85
<i>Pm</i> ₁	♂	7.01	0.04	0.38	5.24	6.1-8.2	87
	♀	6.85	0.05	0.42	6.12	5.8-7.8	84
<i>Pm</i> ₂	♂	6.82	0.04	0.37	5.43	5.9-7.6	86
	♀	6.62	0.05	0.43	6.49	5.2-7.8	81
<i>M</i> ₁	♂	10.81	0.06	0.56	5.18	9.9-12.4	83
	♀	10.52	0.06	0.51	4.86	9.4-11.9	85
<i>M</i> ₂	♂	10.35	0.08	0.63	6.11	8.5-11.7	65
	♀	9.81	0.07	0.49	4.96	8.3-11.8	50
<i>Mandible</i>							
<i>I</i> ₁	♂	5.42	0.03	0.31	5.75	4.5-6.1	85
	♀	5.25	0.04	0.36	6.86	4.3-6.1	87
<i>I</i> ₂	♂	5.95	0.04	0.38	6.32	5.2-6.9	85
	♀	5.78	0.04	0.38	6.60	4.7-6.8	87
<i>C</i>	♂	6.96	0.04	0.36	5.22	6.2-8.1	84
	♀	6.47	0.04	0.32	5.00	5.8-7.4	87
<i>Pm</i> ₁	♂	7.07	0.04	0.35	4.94	6.4-8.1	85
	♀	6.87	0.04	0.38	5.59	5.9-7.5	87
<i>Pm</i> ₂	♂	7.29	0.06	0.52	7.11	6.3-9.6	82
	♀	7.02	0.04	0.40	5.67	6.2-7.9	83
<i>M</i> ₁	♂	11.18	0.05	0.47	4.22	10.0-12.7	76
	♀	10.74	0.06	0.56	5.24	9.4-12.2	84
<i>M</i> ₂	♂	10.76	0.10	0.71	6.62	9.3-12.5	53
	♀	10.34	0.08	0.62	5.96	9.2-11.7	53

TABLE 3

MEASUREMENTS OF THE TEETH
REF: BLACK (1902)

MEASUREMENTS OF THE TEETH.

UPPER TEETH.		Length over all.	Length of crown.	Length of root.	Mesio-distal diameter of crown.	Mesio-distal diameter of neck.	Labio- or bucco-lingual diameter.	Curvature of the gingival line.
UPPER TEETH.								
Central Incisor.	Average.	22.5	10.0	12.0	9.0	6.3	7.0	3.0
	Greatest.	27.0	12.0	16.0	10.0	7.0	8.0	4.0
	Least.	18.0	8.0	8.0	8.0	5.5	7.0	2.0
Lateral Incisor.	Average.	22.0	8.8	13.0	6.4	4.4	6.0	2.8
	Greatest.	26.0	10.5	16.0	7.0	5.0	7.0	4.0
	Least.	17.0	8.0	8.0	5.0	4.0	5.0	2.0
Cuspid.	Average.	26.5	9.5	17.3	7.6	5.2	8.0	2.5
	Greatest.	32.0	12.0	20.5	9.0	6.0	9.0	3.5
	Least.	20.0	8.0	11.0	7.0	4.0	7.0	1.0
First Bicuspid.	Average.	20.6	8.2	12.4	7.2	4.9	9.1	1.1
	Greatest.	22.5	9.0	14.0	8.0	6.0	10.0	2.0
	Least.	17.0	7.0	10.0	7.0	4.0	8.0	0.0
Second Bicuspid.	Average.	21.5	7.5	14.0	6.8	5.3	8.8	0.8
	Greatest.	27.0	9.0	19.0	8.0	6.5	10.0	1.5
	Least.	16.0	7.0	10.0	6.0	4.5	7.5	0.0
First Molar.	Average.	20.8	7.7	13.2	10.7	7.5	11.8	2.2
	Greatest.	24.0	9.0	16.0	12.0	8.0	12.0	3.0
	Least.	17.0	7.0	10.0	9.0	7.0	11.0	1.0
Second Molar.	Average.	20.0	7.2	13.0	9.2	6.7	11.5	1.6
	Greatest.	24.0	8.0	17.0	10.0	8.0	12.5	4.0
	Least.	16.0	6.0	9.0	7.0	6.0	10.0	0.0
Third Molar.	Average.	17.1	6.3	11.4	8.6	6.1	10.6	0.7
	Greatest.	22.0	8.0	15.0	11.0	8.0	14.5	2.5
	Least.	14.0	5.0	8.0	7.0	5.0	8.0	0.0

*There are 25.4 millimeters to the inch.

2.2 Incidence of maxillary lateral incisor anomalies

2.2.1 Congenitally missing laterals

Controversy still surrounds the maxillary lateral incisor's position in the order of frequency of congenitally missing permanent teeth. The majority of authors list the third molars as having the highest incidence (Bot and Salmon, 1977). The upper lateral incisor is listed second by many (Cohen and Anderson, 1931; Werther and Rothenberg, 1939; Brekhus, et al. 1944; Goldman, 1962; Rosenweig and Garbarski, 1965; Muller, et al. 1970); while many list the mandibular second premolar before the maxillary lateral incisor (Dolder, 1937; Byrd, 1943; Grahnen, 1956; Clayton, 1956; Glenn, 1961; Sabres and Bartholdi, 1962; J. Rose, 1966).

Whatever the maxillary lateral incisor's position in the order of frequency of agenesis, its overall importance to research is apparent when we examine the sizeable amount of literature dealing with its occurrence (table 4).

Several observations can be made about agenesis of the lateral:

The frequency of the congenitally missing maxillary lateral in man varies between races. In Caucasians it ranges from 0.5% (Dolder, 1973) to 2.6% (Clayton, 1956; Montagu, 1940) with the figure most quoted being around 2.0%. Davies (1968) found the incidence to be 1.8% in Australian schoolchildren. In coloured races the frequency appears to be lower, ranging from 0.2% in the Chinese to 1.7% in the American Negro (Hrdlicka, 1921).

Support is lent to the theory that evolution has brought about a greater incidence of agenesis by the figures of both Kajava (1912) who found no cases of congenitally missing laterals in the primitive

TABLE 4

STUDIES SHOWING INCIDENCE OF CONGENITALLY MISSING OR PEGGED MAXILLARY LATERAL INCISORS

NR = Not recorded

Author	Year	Sample Size	Material	% Absent	% Pegged	Sexual Difference	Side Dominance
C. Rose cited by Hrdlicka (1921)	1906	18856	North+Central Europeans	1.5	1.9	female	NR
		850	Swedish	2.1	4.0	female	NR
		12250	Swedish+Central European soldiers	1.7	1.8	NR	NR
Kajava cited by Rantanen, (1956)	1912	180	Finnish lap skulls	0	0	NR	NR
Hrdlicka	1921	1000	white Nth American	2.2	3.0	female	none
		807	American negro	1.7	3.7	female	none
		651	Chinese	0.2	7.7	male	left pegged
Shultz	1934	2000	Monkey+ape skulls	0.1	NR	NR	NR
Campbell	1934	2000	New York clinic	1.8	1.0	NR	NR
Dolder	1937	10000	Swiss children	0.5	NR	NR	NR
Werther and Rothenberg	1939	1000	Pennsylvania Dental School USA	2.3	NR	NR	NR
Montagu	1940	528	American Jewish males	2.6	2.5	NR	NR
Brekhus et al.	1944	11487	Minnesota Cental Clinic USA	1.0	1.1	NR	Right agenesis left pegged
Grahnen	1956	1006	Swedish School children 11-14 yr	1.6	1.7	female pegged	NR
		1064	Students+ Dental Nurses 17-43 yr	1.7	1.3	none	NR
Rantanen cited by Brooks (1974)	1956	3200	Finnish undergraduates	NR	1.0	NR	NR
Clayton	1956	3557	Nth Americans	3.6	NR	NR	NR
Meskin and Gorlin	1963	8289	Nth American undergraduates	0.95	0.88	female	left

TABLE 4 continued

NR = Not recorded

Author	Year	Sample Size	Material	% Absent	% Pegged	Sexual Difference	Side Dominance
Brown cited by Glenn (1961)	1957	5276	Nth American	0.9	NR	NR	NR
Glenn	1963	925	Florida whites+ blacks	4.0	NR	female	NR
Niswander and Sujaku	1963	4150	Nagasaki Japan	1.7	NR	NR	NR
J Rose	1966	6000	English ortho. practice	1.3	NR	female	none
Davies	1968	2170	Australian schoolchildren	1.8	1.1	female	none
Muller et al.	1970	14940	Nth American Caucasians	1.3	NR	female	none
Malik	1972	2080	Pakistan	0.52	0.91	female	NR
Chung et al. cited by Brook 1974	1972	833 589 828 6022	Filipinos Caucasians Chinese Japanese	NR NR NR NR	3.1 1.7 1.9 1.6	NR NR NR NR	NR NR NR NR
Thilander and Myrberg	1973	5459	Swedish schoolchildren	NR	0.6	NR	NR
M Elomaa and E Elomma	1973	6264	Finland	2.39	0.4	female	left pegged
Eidelman et al.	1973	21384	Israeli schoolchildren	2.11	NR	female	NR
Brook	1974	1115	British schoolchildren	NR	2.5	female	NR
Bachmann cited by Schwaninger and Shaye 1977	1974	8694	Swiss schoolchildren	1.67	NR	female	NR
Bot and Salmon	1977	5738	French males	1.90	1.59	-	none
Becker et al.	1981	633	Israelian	2.1	1.1	female	none

Finnish skulls and Shultz (1934) who found only 2 cases in 2,000 monkeys and apes.

The frequency of the congenitally missing maxillary lateral incisor varies between the sexes. Generally, females appear to have a higher frequency than males (Hrdlicka, 1921; Moskin and Gorlin, 1963; Glenn, 1963; J. Rose, 1966; Davies, 1968; Muller, et al. 1970; Malik, 1972; M Elomaa and E Elomaa, 1973; Eidemann et al. 1973; Bachmann, 1974).

The pattern of the congenitally missing maxillary lateral incisor can favour the right side (Campbell, 1934; Brekhus, Oliver and Montelius, 1944), the left side (Meskin and Gorlin, 1963) or show no significant preference (Hrdlicka, 1921; J Rose, 1966; Davies, 1968; Muller, et al. 1970; Bot and Salmon, 1977).

2.2.2 Reduced laterals

In the case of reduced lateral dimensions most work has revolved around the occurrence of pegging in this region (Table 4) although the term 'peg-lateral' is seldom defined.

Several interesting differences are noted between the frequency of congenitally missing laterals and peg-laterals:

The frequency of peg-laterals as with congenitally missing laterals varies between the races. The frequency in Caucasians of peg-laterals ranges from 0.4% in the Finnish population (M Elomaa and E Elomaa, 1973) to 3.0% in the American population (Hrdlicka, 1921). This is similar to the range for congenitally missing laterals. However, in coloured races the frequency for peg-laterals appears to be higher than for congenitally missing laterals, ranging from 3.7% in American Negroes to 7.7% in the Chinese (Hrdlicka, 1921). Davies (1968) found

the incidence of peg-laterals to be 1.1% in Australian schoolchildren.

Similar to that found with congenitally missing laterals, peg-laterals have a higher frequency in the female than the male (Becker, et al. 1981; Brook, 1974; Davies, 1968; Grahnen, 1956).

In contrast to congenitally missing laterals which appears to have no distinct pattern to the side involved, peg-laterals have been shown to occur more frequently on the left side than the right (Hrdlicka, 1921; Brekhus, et al. 1944; Meskin and Gorlin, 1963; M Elomaa and E Elomaa, 1973).

2.3 The etiology of maxillary lateral incisor anomalies

As with those found for the etiology of canine impaction, the views as expressed within the available literature dealing with the etiology of maxillary lateral incisor anomalies, can be discussed under the following divisions:

- A EVOLUTION
- B HEREDITY
- C GLANDULAR DYSFUNCTION
- D ENVIRONMENT

2.3.A. EVOLUTION

The evolutionary reduction of man's facial structures has brought about an accompanying reduction in the dimensions of the jaws. This reduction in the size of the jaws as shown previously (section 1.5A), has often been cited as the reason for an increase in canine impactions. It has also been named as the reason for the presence of a high number of anomalies in the maxillary lateral incisor region (Hrdlicka, 1921; Campbell, 1925; Shultz, 1932; Bradlaw, 1934; Werther and Rothenberg, 1939; Montagu, 1940; Dahlberg, 1945; Bot, Eugene and Salmon, 1980; Grundy, 1980; Jorgenson, 1980).

Bradlaw (1934) exemplified this view when he wrote "it is generally agreed that there is a tendency among the highly evolved races toward the loss of the maxillary lateral incisor, concurrently with the reduction in the size of the jaws."

The field concept as proposed by Butler in 1939 formed a theoretical basis for the earlier attempts to explain the mechanics of change occurring through evolution, to man's dentition. In this concept, each tooth occupies a position within one of three fields - incisivation, caninisation and molarisation. The teeth found most distal within their

respective field were said to be those most affected by the evolutionary reduction and hence the most variable. The maxillary second incisors were those teeth in the most distal position within the incisor field.

Bot et al. (1980) believed in the process of evolutionary simplification of man's dentition and proposed a model, based on their research, for this morphological change. The first stage they believed was a change in developmental timing and tooth eruption followed by a modification in the groove patterns of the lower molars. This was then accompanied by first, a reduction in the mesiodistal width of one maxillary lateral incisor, then by a reduction in both. The final phase resulted in the selective disappearance of the upper third molars, the lower second premolars then the upper lateral incisors.

However, Bolk in 1950 (cited by Davies, 1966) an opponent of the theories of evolutionary reduction, believed most cases of agenesis in man were originally due to mutations that have been inherited.

Brekhus et al. (1944) also questioned the thoughts that an evolutionary reduction in the size of the supporting structures had caused pegging of the laterals, when they found adequate basal bone situations accompanied by peg-shaped and missing laterals. Furthermore, the phylogenetic theory of civilization discarding the weakest, most distal tooth within the field appears to be contradicted in the cases of canine and lower first premolar agenesis (Brekhus et al. 1944; Osburn, cited by Werther and Rothenberg, 1939; Thoma and Goldman, 1960; Lavelle and Moore, 1973).

Undoubtedly, a reduction has occurred in the overall size of the human cranial and facial structures, this being irrefutably shown by

ample fossil evidence (Gregory, 1922). However conflicting views (Brekhus et al., 1944; Thoma and Goldman, 1960) still exists as to the influence this has had on the overall size and number of teeth within man's dentition, and specifically the etiology of the congenitally missing or peg-shaped maxillary lateral incisor.

B. HEREDITY

The congenitally missing and peg-shaped lateral incisor has on numerous occasions been documented as an example of the hereditary nature of dental anomalies.

One of the earliest references is that reported by Leroy-d'Etiolles (cited by Campbell, 1934) to the Societe de Bologne in 1851. He described the absence of the maxillary lateral incisor in both the son and daughter of a mother similarly affected. Tomes in 1873 (cited by Campbell, 1934) presented three sisters all with similar 'anomalies' of the lateral incisors. Sergi (1914) observed three generations of a family, who "in all of them the absence of the tooth (lateral maxillary incisor) is accompanied by a noticeable reduction in the size of the corresponding incisor on the left side."

Data was published in 1888 by Lucas (cited by Wheelon 1925) showing parents in whom the lateral incisor was either absent or deformed and in whose children the deformities had been also expressed.

Duval in 1895 showed the models of an entire family with absence of the maxillary lateral incisors, while Wheelon in 1925 presented two families in whom the missing lateral incisor had been expressed over a number of generations.

Six generations of the one family, a total of twenty two individuals were traced by Gardner in 1927, showing in all the congenitally missing

maxillary lateral incisor trait.

More recently, M Elomaa and E Elomaa (1973) examined the parents and siblings of thirty dental students presenting with congenital absence of the maxillary lateral. Of the eighty two relatives investigated 43.9% presented with the identical incisor anomaly.

These are but a few of the large number of reports published on the familial occurrence of the anomalous lateral, strongly suggesting the role genetics has in its etiology. There exists, however, controversy as to the mode of inheritance, with several different genetic models postulated to explain the lateral incisor's inheritance pattern.

Grahnén (1956) theorized, "hypodontia (excluding third molars) is primarily determined by a dominant autosomal gene with incomplete penetrance and variable expressivity The possibility of several genetic entities being involved cannot be excluded. Peg-shaped maxillary lateral incisors are probably a modified manifestation of the same genotype that causes hypodontia."

Several other authors agreed with Grahnén (1956) believing the trait was inherited through a single autosomal dominant gene (Shultz, 1932; Montagu, 1940; Mandeville, 1950; Winter, 1969; Alvesalo and Portin, 1969). To explain the variability of the lateral anomalies within family groups, it had been suggested the trait had "incomplete penetrance and expressivity" (Grahnén, 1956; Woolf, 1971; Huskins, 1930). However, Woolf (1971) speculated that in some instances, the variability may arise from the trait being autosomal recessive. This had been previously reported by Iltis (1948) and Thomsen (1952).

Iltis (1948) favoured the view of a single autosomal recessive gene,

based on the fact that the absence of maxillary lateral incisors in his material occurred mostly in the offspring of consanguineous marriages. Thomsen (1952) after working with the population of Tristan de Cunha concurred with Iltis (1948). However, Davies (1966) when reviewing the work of both Iltis (1948) and Thomson (1952) concluded that none of the pedigrees presented by these workers indicated a certain diagnosis of a recessive mode.

The alternative interpretation for the associated genetic variations lies in the solution that hypodontia may be controlled by more than one gene. The polygenic hypothesis* has been suggested by a number of authors (Rose, 1966; Baum and Cohen, 1971; Roberts, 1973; Davies, 1983; Graber, 1978). Suarez and Spence (1974) applied the observed data of Grahnen (1956) to both single locus and polygenic statistical models and concluded "at least for Grahnen's data, the single-locus hypothesis can be rejected in favor of the polygenic hypothesis."

Baillet (1975) suggested it may be difficult to understand how the presence or absence of a tooth can be considered polygenic since such characteristics usually have a continuous distribution. However, he presents as a possible explanation the ideas proposed by Brook in 1973. Brook (1973) believed that agenesis in Man was indeed polygenically determined but also an example of a "quasi-continuous trait". He suggested the congenital absence of teeth existed as a discontinuous anomaly, tending to arise near the extremes of the continuous variable, tooth size. That is, tooth size is turned into an all or none trait by

* Polygenic inheritance = a mode of inheritance caused by a number of genes each with a relatively small effect and is best known in continuous traits such as stature and intelligence.
(Chosack, Eidemann and Cohen, 1975)

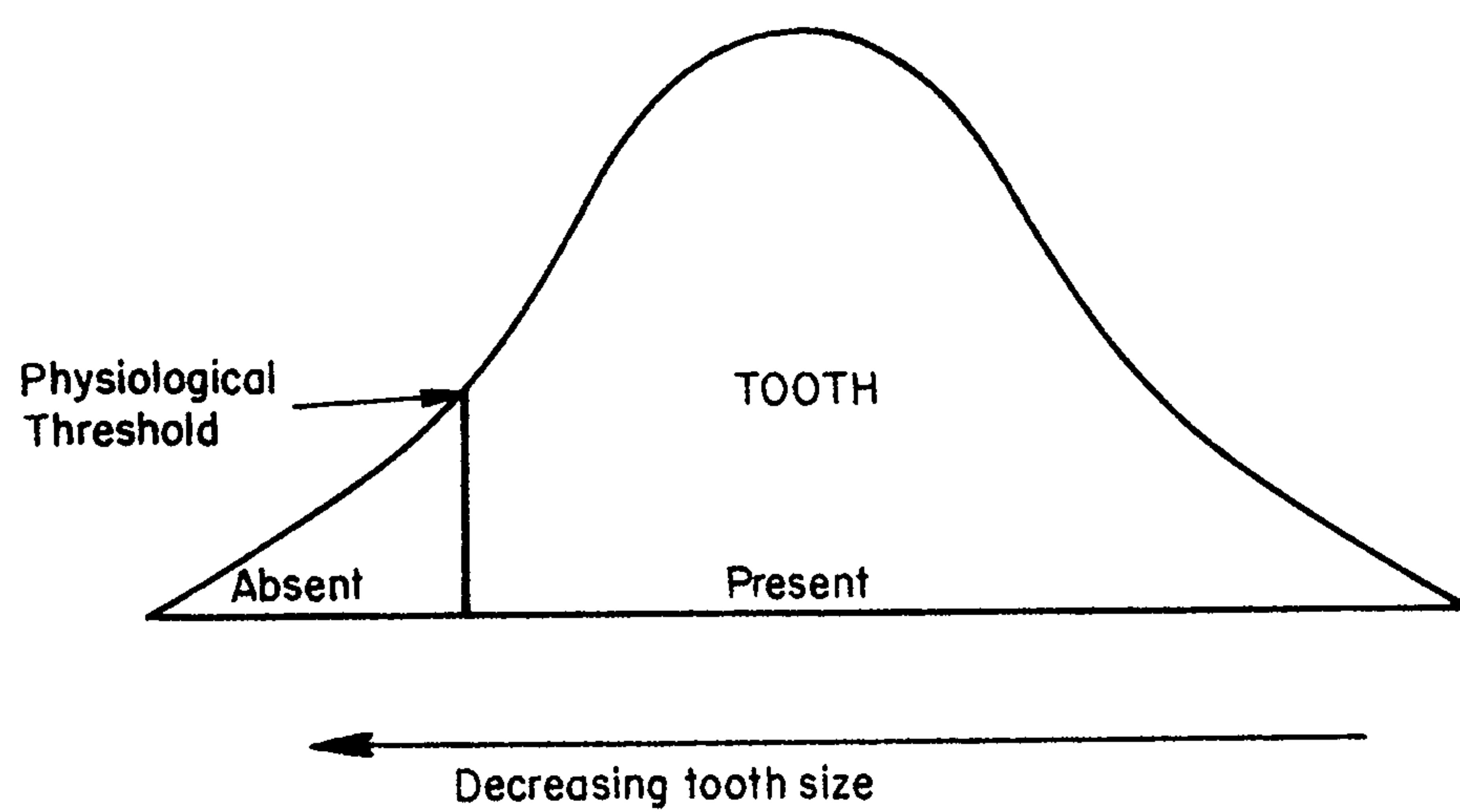
means of a threshold mechanism. The threshold being influenced by many environmental or physiologic factors (Figure 8).

Therefore, Brook (1973) proposed a polygenic, multifactorial model and wrote: "the anomalies behave as quasi-continuous traits determined by the positions of individuals in relation to thresholds superimposed on a continuous distribution."

Actually, the concept of a threshold mechanism is not recent as it had been previously used by Gruneberg in 1951 to explain the apparent association between pegged and missing lateral incisors. He believed the dental lamina had a minimal size (threshold) below which the probability the tooth would fail to develop increased. Witkop (1961) however disagreed, as he believed the rudimentary lateral indicated the influence of environmental and genetic factors after the initiation of tooth development, whereas agenesis indicated their influence before or at initiation. Whatever the mechanism, many authors believe the etiology of the peg lateral incisor is related to the congenitally missing lateral incisor (Davies, 1968; Niswander and Sujaku, 1963; Garn, Lewis and Vicinus, 1962; Demoge, 1973; Sofaer, Chung, Niswander and Runck, 1971; Eastman and Raibley, 1979; Woolf, 1971; Bailet and Sung, 1968; Becker et al. 1981; Jorgenson, 1980). This was emphasized in the findings of Thilander and Myrberg (1973) where it was noted, in the 28 cases presenting with a pegged lateral the lateral on the opposite side in each case was found to be congenitally missing. Several workers have actually shown in cases presenting with agenesis, the remaining teeth in the dentition show an overall size reduction, which can be expressed in the extreme form of the peg lateral incisor (Garn and Lewis, 1969; Baum and Cohen, 1971; Rune and Sarnas, 1974; Bot and Salmon, 1980).

FIGURE 8

THE THRESHOLD MECHANISM OF TOOTH SIZE AS ADVOCATED BY
BROOK (1973)



Although the controversy over the mechanism of genetic control for pegging and agenesis has still not been resolved, recent views tend to favour the ideas proposed by Brooks (1973), that being a polygenic determined, quasi continuous variable, influenced by several possible environmental factors (Chosack, Eidelman and Cohen, 1975; Graber, 1978; Davies, 1983; Charlton, 1983).

C. GLANDULAR DYSFUNCTION

Certain glandular dysfunctions have been cited throughout the literature as playing a major role in the etiology of anomalies of the lateral incisor. Kaplan (1917) associated deformities of the lateral incisor with abnormalities in secretions from the gonads. He wrote "In the male small and stumpy laterals bespeak sexual impotence on the physical basis."

A 'pluriglandular syndrome' involving the pituitary gland was given by Timme (1918) as the reason for certain characteristics in the dentition. The example he gave was ".... the lateral incisors especially in girls are frequently greatly underdeveloped."

Werther and Rothenberg in 1939 offered the explanation that a disturbance in the endocrine glandular system would bring about retarded formation of the ectoderm which they suggested explained the association of tooth anomalies with hair, nails and sweat gland deficiencies. Brekhus, Oliver and Montelius (1944) believed only some of the more extreme cases of missing teeth could be shown as definitely due to endocrine disturbances, while Lavelle and Moore (1973) were of the conviction that no conclusive evidence has been forthcoming to support the view agenesis results from endocrinologic factors.

However, Downs in 1927 on examining the incidence of congenitally

absent teeth in 647 children with various forms of endocrine disturbances, showed a higher number of missing teeth than could be found in the general population, but at the same time he did not try to relate his findings to any particular endocrine dyscrasia. Cohen and Anderson in 1931, likewise showed a higher incidence of upper lateral incisor agenesis in children affected by endocrine disturbances and mongolism. These findings are similar to those found by several other authors (Barkla, 1963; Cohen and Winer, 1965).

It could be concluded that increased incidence of aberrations in both the size and number of teeth have been associated with several glandular disturbances and have been listed as characteristics of many syndromes (Table 5). However, the role of glandular disturbances in the etiology of tooth anomalies is still undetermined.

D. ENVIRONMENTAL FACTORS

Several environmental factors have been reported to be associated with abnormalities of the upper lateral region. These can be best described as being either local or general.

Local Environmental Factors

i. Local disruption of tooth germ

a. Acute and Chronic inflammations

Inflammation occurring within the area of the tooth germ may influence the developmental stages of tooth formation resulting in the possible pegging, or in the extreme case, agenesis of the lateral incisor (Werther and Rothenberg, 1939).

b. Pyogenic infections and tumor formation

Grahnén (1956) suggested the presence of sequestrating

TABLE 5

SYNDROMES WITH HYPODONTIA AS A COMPONENT

REF: JORGENSEN (1980)

Syndrome	Pattern of hypodontia
Autosomal dominant	
Crouzon's syndrome	No pattern (infrequent)
Robinson's syndrome	Generalized (moderately severe)
Ectodermal dysplasia:	
cleft lip, and cleft palate	Generalized (moderately severe)
Freire-Maia syndrome	No pattern
Hypertrichosis lanuginosa	Generalized (severe to anodontia)
Marshall-White syndrome	No pattern
Monilethrix-anodontia syndrome	Generalized (anodontia)
Otodental dysplasia	Premolars (occasional)
PHC (Böck's) syndrome	Premolars
Rieger's syndrome	Maxillary incisors (constant), mandibular incisors, and premolars (occasional)
Tooth and nail syndrome	No pattern
Autosomal recessive (AR)	
Cockayne's syndrome	No pattern
Ectodermal dysplasia:	
hypohidrotic	Generalized (severe)
Ellis-van Creveld syndrome	Mandibular incisors and canines
Gorlin's syndrome	Generalized (severe)
Gorlin-Chaudhry-Moss syndrome	Deciduous molars, secondary premolars, and molars
Lipoid proteinosis	Maxillary lateral incisors, canines, and premolars
Melanoleukoderma	No pattern
Palmoplantar keratosis,	
hypotrichosis, cysts of eyelid	No pattern
Rothmund-Thomson syndrome	No pattern (infrequent)
Toumaala-Haapanen syndrome	Generalized (anodontia)
X-linked	
Coffin-Lowry syndrome	Maxillary lateral incisors, mandibular incisors
Ectodermal dysplasia:	
hypohidrotic	Generalized (severe)
Focal dermal hypoplasia	No pattern
Incontinentia pigmenti	No pattern
Orofaciodigital I syndrome	Mandibular incisors and canines
Genetics uncertain or others	
Cleft lip, cleft palate†	Maxillary lateral incisors and canines
Down's syndrome (chromosomal)	Third molars, mandibular second premolars, and maxillary lateral incisors
Frontometaphyseal dysplasia	No pattern
Glassopalatine ankylosis syndrome	Incisors
Hallermann-Streiff syndrome (?AR)	No pattern
Hanhart's syndrome (?AR)	No pattern
Holoprosencephaly	Maxillary incisors
Hypoglossia-hypodactylia syndrome	Mandibular incisors and canines
Johanson-Blizzard syndrome	Generalized (severe)
Progeria (?AR)	No pattern (infrequent)

*The syndromes listed are those for which hypodontia has been reported as an associated finding. When hypodontia is reported only occasionally in a syndrome, it is difficult to know whether the association is causal or fortuitous. When there is doubt, the syndrome has been listed.

†Includes approximately 100 genetic syndromes with clefting as a feature and isolated clefting.

osteomyelitis, osteitis and tumors can affect the developing tooth germ; agenesis being the result of complete expulsion or suppression of the tooth germ.

c. Local trauma

Werther and Rothenberg (1939) named birth trauma as a local cause of malformation in the permanent dentition

ii. Space Inadequacies

Montagu (1940) believed an overall lack of space the reason for the high incidence of incorrect lateral formation. He suggested the evolutionary reduction in the size of the premaxilla had resulted in a lack of space, preventing development of that part of the dental lamina normally devoted to the lateral incisors. Sofaer et al. (1971) similarly believed the lateral incisor would be the first tooth to manifest effects of premaxillary reduction but believed this to be due to the fact that it begins its development between two previously established neighbours (central and canine), with which it competes for available space; the lack of space could lead to a consequent lack of development.

General environmental factors

i. Nutrition

Robertson (1924) wrote "man is the sickest beast alive ... mainly due to our demineralized devitalized menu." He believed when cells were starved they had trouble in developing and surviving; this being especially apparent in the teeth. Although Robertson was possibly being dramatic, diet deficiencies can cause anomalies in the development of both the teeth and surrounding structures (Shafer, Hine and Levy, 1974) especially during pregnancy (Werther and Rothenberg, 1939).

ii. Systemic Disease

One of the more avid supporters of systemic disease being the sole causal factor of upper lateral incisor anomalies was Campbell (1934). He believed that the syphilologists had "... been able to present a strong case in favour of congenital syphilis being a factor (whatever its mechanism may be) in the production of agenesis and hypoplasia of the upper lateral incisor." However, Campbell (1934) did not believe all people presenting with abnormalities of the lateral incisor were second or third generation syphilitics. Instead, he believed that an original syphilitic injury had caused the defect, which had become a dominant heritable character being handed down through the generations. However Brekhus et al. (1944) believed syphilitic stigmata and dental anomalies should be considered as separate entities and that severe dental deficiencies are recognized symptoms of some diseases. They gave as their example ectodermal dysplasia which has often been cited as an extreme form of disease-related agenesis (Miles, 1954; Thoma, 1960; Craig, 1968; Winter, 1969).

In addition to syphilis and ectodermal dysplasia, several other diseases have been associated with anomalies of the dentition:

Rickets (Grahnen, 1956; Thoma, 1960)

Parotitis (Grahnen, 1956)

exanthematous fevers (Grahnen, 1956; Werther and Rothenberg, 1939;
Thoma, 1960)

and tuberculosis (Werther and Rothenberg, 1939).

iii. Developmental Deformities

The cleft lip and palate is an example of an extreme deformity in association with anomalies of the dentition. Its own etiology is still obscure, having been linked with both genetic and environmental factors

(Godfrey, 1983).

The relationships between cleft lip and cleft palate and lateral incisor defects was noted as early as 1888 by Lucas; he wrote "...the absence of a lateral upper incisor tooth resulting from an arrest of development is to be regarded as a malformation closely related to hare-lip and cleft palate and capable of transmitting each or both of these deformities to succeeding generations."

Although the cause for hypodontia in cleft individuals remains obscure there exists a significantly higher frequency of this dental abnormality accompanying them (Watson and Hardwick, 1971; Craig, 1968; Millhon and Stafne, 1941). The study by Millhon and Stafne (1941) found the incidence of lateral maxillary incisor agenesis to be 11.7% in sixty cases of cleft palate, significantly higher than that found in many caucasian populations (see section 2.2).

Montagu in 1940 appeared to regard the maxillary lateral incisor agenesis as a mild manifestation of a cleft. However, Miles (1954) considered this may not be the case as cleft lip is significantly more common on the left side while agenesis could occur on either side.

iv. Intra uterine environment

Various investigators consider the influence of prenatal conditions of prime importance to the etiology of microdontia and agenesis (Berendt, 1954; Brekhus et al. 1944; Thoma, 1960; Keene, 1966; Charlton, 1983). Diseases, nutritional and metabolic disturbances during pregnancy have been associated with anomalies of the dentition.

Charlton (1983) having previously examined the permanent teeth of a large number of Sydney University students for anomalies, and recorded family, medical and dental histories, suggested irregularities

in prenatal and post natal environment could be reflected in the anomalies of the dentition.

Keene (1966) reported that naval recruits with hypodontia tended to have low birth weights, be the first born in their family and have mothers who were less than 20 years of age at the time of their birth.

Prenatal environment may be related to some instances of hypodontia but at present evidence does not exist to indicate its role in all occurrences of the anomaly.

2.4 Summary

The literature concerning the etiology of lateral incisor anomalies as found with that dealing in canine impaction is extensive and has been discussed under the headings:

EVOLUTION

HEREDITY

GLANDULAR DYSFUNCTION

ENVIRONMENT

In similar fashion to that suggested with the etiology of canine impaction, many of the conjectured causes for lateral incisor anomalies are controversial since in many instances the cause/affect relationship suggested, is based only on circumstantial evidence. Possibly the prominence of its position in the anterior region, the ease of examination and its relatively high frequency of absence, has made this tooth the focus of many investigations and, hence, has been named as an alleged result of many dental and medical anomalies.

Most of the research into the inheritance of dental anomalies appears to have been directed towards the maxillary lateral incisor. Our present understanding in this important area of science is still limited. However Brook's proposed model (1973), of a "quasi-continuous variable influenced by several environmental factors" appears to be in favour at present.

This would then suggest our present knowledge on the etiology of lateral incisor anomalies would still limit us to conclude that it is the result of a complex interrelationship of hereditary genetic, glandular and environmental factors. A similar conclusion has been previously drawn for the impacted canine (chapter 1.5).

Although the possible link between the impacted canine and the

anomalous lateral incisor can at this stage, only be postulated, several instances of similarities between their respective conjectured etiologies and incidences can be found and hypothetical reasons for this association raised. This will be further discussed in the next chapter (3).

CHAPTER 3

THE ASSOCIATION OF THE MAXILLARY IMPACTED CANINE WITH ANOMALIES OF THE MAXILLARY LATERAL INCISOR

The impacted canine has often been stated to be in association with anomalies of its adjacent tooth the lateral incisor (see section 1.4.3). As early as 1891 Mr R.H. Woodhouse reported the case in which both lateral incisors were congenitally absent and both canines had become impacted.

In 1897 C. Robbins presented a case of a lady aged 17 "in whose family there was a history of suppression of laterals and retarded eruption of permanent canines."

Bass (1967) after surveying a large sample of orthodontic patients at the Royal Victorian Hospital, Bournemouth, concluded "the frequency of the congenital absence of permanent teeth (most common were upper laterals) was significantly higher in the patients with misplaced canines compared with the patients selected at random."

Becker et al. (1981) in their study on the relationship of anomalous laterals to impacted canines found a high incidence of peg shaped (17.25%) and otherwise small (25%) lateral incisors adjacent to palatally placed cuspids. They suggested "... in all cases where anomalous lateral incisors are noted, the possibility of resultant palatal displacement of the cuspid should be investigated."

3.1 Hypothetical reasons for this association

There are several hypothetical reasons why anomalies of the lateral incisor could be associated with an impacted canine. The author believes these reasons can be divided into two schools of thought:

3.1.1 Purely mechanical factors

3.2.2 Common etiologic factors

3.1.1 Purely mechanical factors

During the eruption of the canine the lateral incisor provides a guide to the crown of the canine as it moves up the distal aspect of the incisor root; this produces the characteristic 'Ugly duckling' splayed appearance (Broadbent, 1941).

In the case of agenesis, the complete lack of the lateral root guidance allows the cuspid's crown to pass mesially until it encounters the root of the permanent central, this then takes over the role of the lateral (Miller, 1963/4 ; Kettle, 1957). With the added available space (as the result of the agenesis) and the lack of interference, the canine has at this stage an equal opportunity to arrive correctly within the arch or be located either buccally or palatally (Becker et al. 1981).

However, consideration must be given to the following:

i. The canine has had to undergo an increase in distance travelled to meet the guiding influence of the central incisor and hence a longer time to be deflected (Dewel, 1949).

ii. The central incisor that is now its guide, is a tooth that tends to suffer a larger number of childhood traumas and is often found

out of position.

iii. The cuspid's crown would meet the central incisor tooth at a different approach angle than it would the lateral incisor.

In the situation of the mesio-distally reduced lateral, Becker et al. (1981) believes the rudimentary lateral can interfere with the late rectifying movements of the canine producing a palatal deflection, even though there may be sufficient space within the arch. The author is in agreement with Becker et al. (1981) but also believes that in the case of the rudimentary lateral, in the majority of instances, the resultant dento-alveolar discrepancy caused by the anomaly in the anterior region would allow some mesial movement of the buccal segments. This slight mesial movement of the buccal segments, the author contends, would be enough to create a reciprocal change in the direction of the erupting canine, possibly towards the palatal aspect of the rudimentary lateral.

3.1.2 Common Etiologic factors

Bass (1967) after finding a significantly high level of congenitally missing teeth in the buried canine group compared with the random group, theorized "that the malposition of the canine resulted from the same disturbance of the dental lamina that led to the agenesis of the permanent teeth."

This theory is quite feasible when we consider the number of suggested etiological factors mutual to both the impacted canine and the anomalous lateral incisor, and the similarities that exist in their incidence.

For example, the role of evolution in reducing the cranial and facial structures has been suggested as a major etiological factor in

both canine impaction (Chapter 1.4.3A) and lateral incisor anomalies (Chapter 2.3.A). An evolutionary reduction in overall arch dimension may affect the correct dimensional formation of the permanent dentition to the extreme point of selected agenesis. A secondary effect may be the disturbance in the axial inclination of the canine resulting in impaction. Sofaer et al. (1971) suggested in the situation where space within the arch was at a premium, the canine and central incisor since they developed earlier, would be at a size to suppress lateral incisor initiation. Bot et al. (1980) proposed an evolutionary modification model for the dentition that incorporated progressive stages of change in the developmental timing, eruption and morphology of the permanent dentition (Section 1.2.3A).

Payne (1930), Goldsmith (1931) and Dewel (1949) stressed the length of time required for canine development and final eruption, and that this may contribute to the cuspid's own impaction. They gave as the reason, that the teeth arriving late in the eruption sequence were the ones more likely to be affected by environmental factors. Garn, Lewis and Vicinus (1962) noted that dental development time and eruption may be delayed further in the presence of agenesis and pegging. This could then suggest that agenesis and pegging of the lateral incisor may produce a further delay in the already long developmental time of the canine, resulting in an extended period for environmental influence.

The hereditary nature of the anomalous lateral incisor and the hereditary nature of the impacted canine has been reported in the literature (Chapter 1.4.3B and 2.3.B). Whether the same genetic model can be used to explain the inheritance of both of these occurrences is open to conjecture. Although the evidence may be only circumstantial, it is interesting to note that both can occur simultaneously and appear

from the report by C. Robbins (1897) to be able to be transmitted through the generations. Also of interest, is the occurrence of both cuspid impaction and lateral incisor anomalies with another genetic stigma, the cleft lip and cleft palate. As noted earlier, Montagu (1940) regarded the lateral incisor agenesis as a mild manifestation of a cleft, while Takahama and Aiyama (1982) believed canine impaction to be a microform of the cleft. There may be a link between all three, with its basis within in some yet undefined genetic combination.

The association of glandular dysfunctions with cases of agenesis and impactions (Chapters 1.4.3C and 2.3.C) suggests that glandular secretions may have an influence on both the formation and eruption of teeth. Minor imbalances in the gland secretions may influence only specific areas within the dentition such as canine impaction and lateral incisor anomalies.

Any environmental factors influencing the development of the lateral incisor could have a subsequent effect on the developing canine, as it has been shown (Chapter 1.2), the canine development begins before the lateral incisor, with its eventual eruption occurring a considerable time after that of the lateral incisor. Early childhood diseases or nutritional imbalances have been suggested as factors in both cuspid impaction and incisor development (Chapters 1.4.3D and 2.3.D). Obstructions to canine eruption, occurring through the influence of local factors such as enlarged follicles, acute and chronic local infections, dentigerous cysts, supernumeraries, tumour formations and local trauma; may have also influenced the formation of the lateral incisor.

Finally if we compare the incidences of cuspid impaction (Chapter 1.4.2) with the incidences of lateral incisor anomalies (Chapter 2.2)

in the Caucasian races we find the differences not to be so great. In addition, there appears to be a higher incidence of both impactions (Table 1) and lateral anomalies in the female (Table 4).

Whether the existence of a reduced or missing lateral incisor mechanically influences the eruption of the canine, or similar etiologic factors are common to both these events, is still uncertain. However, in support of the idea that there is some underlying mechanism mutual to both anomalies, it has been shown that reduced mesio-distal dimensions and agenesis of teeth are not isolated phenomena, having been associated with delayed calcification and eruption of the remaining teeth (Garn, Lewis and Vicinus, 1962; Garn and Lewis, 1969; Rune and Sarnas, 1974; Baum and Cohen, 1971a, 1971b; Wisth, Thunold and Boe, 1974; Lysell, 1953, cited Wisth et al. 1974; Grahnen, 1956).

Whatever the etiologic connection, if a possible association can be found between the palatally impacted canine and the anomalous lateral incisor, the lateral would afford the clinician of an early diagnostic "warning sign" to a future palatally impacting canine, in a substantial proportion of cases.

ORIGINAL STUDYCHAPTER 4AIM OF THE STUDY

The aim of this study was to test the hypothesis: In patients presenting with unilateral or bilateral palatally impacted permanent canines, there exists an associated significant incidence of maxillary lateral incisor anomalies.

CHAPTER 5

INVESTIGATIVE METHOD

Two groups were examined for the presence of lateral incisor anomalies: A palatally impacted canine group and a non-impacted control group.

The palatally impacted canine group

A large number of patients, after careful examination and the collating of records by the Orthodontic Department of the United Dental Hospital, Sydney, were referred to the Oral Surgery Department of the same Hospital for surgical intervention of palatal impacted canines. Palatal impaction had been previously diagnosed by the examining orthodontist and consulting oral surgeon from both clinical observation and radiographic investigation. From this group, a sample of 100 cases were randomly selected for this investigation. Their available dental histories, radiographs and plaster models were examined to determine the incidence of anomalous lateral incisors.

The non-impacted control group

In addition to those patients diagnosed as having palatal impactions of the canine, an even greater number of patients were diagnosed by the same Orthodontic Department as having malocclusions other than impaction. From this group a random sample of 100 patients were selected as a control. Their available dental histories, radiographs and plaster models were similarly examined to determine the incidence of anomalous lateral incisors.

5.1 Method of Assessment

An anomalous lateral incisor was defined as a permanent maxillary lateral incisor being either congenitally missing or having abnormal crown morphology.

5.1.(i) Congenitally missing

The absent maxillary lateral incisor was first observed on the plaster model and then confirmed as being congenitally missing by both the patient's dental history and radiographs. (Figures 9,10).

5.1.(ii) Abnormal crown morphology

The abnormal maxillary lateral incisor crown morphology was defined as, any deviation away from the stem type as described in the literature (Chapter 2.1).

The author for ease of examination and measurement and hence increased reproducibility, divided the abnormal crown morphologies into three groups:

i. Frankly peg-shaped: This represented those lateral incisors that appeared dramatically reduced when compared to the antimere and surrounding teeth; to the point of being distinctly conical in shape. (Figures 11,12).

ii. Peg-shaped: This represented those lateral incisors with the mesio-distal width greatest at the cervical margin. (Figure 13)

iii. Small: This represented those lateral incisors with the mesio-distal width equal to or smaller than that of its mandibular counterpart. (Figure 14)

Groups (ii) and (iii) were similar to those used by Becker et al. (1981) in their investigation and were determined by careful measurement of the tooth on the available plaster models.

FIGURE 9

BILATERAL CONGENITALLY MISSING LATERAL INCISORS

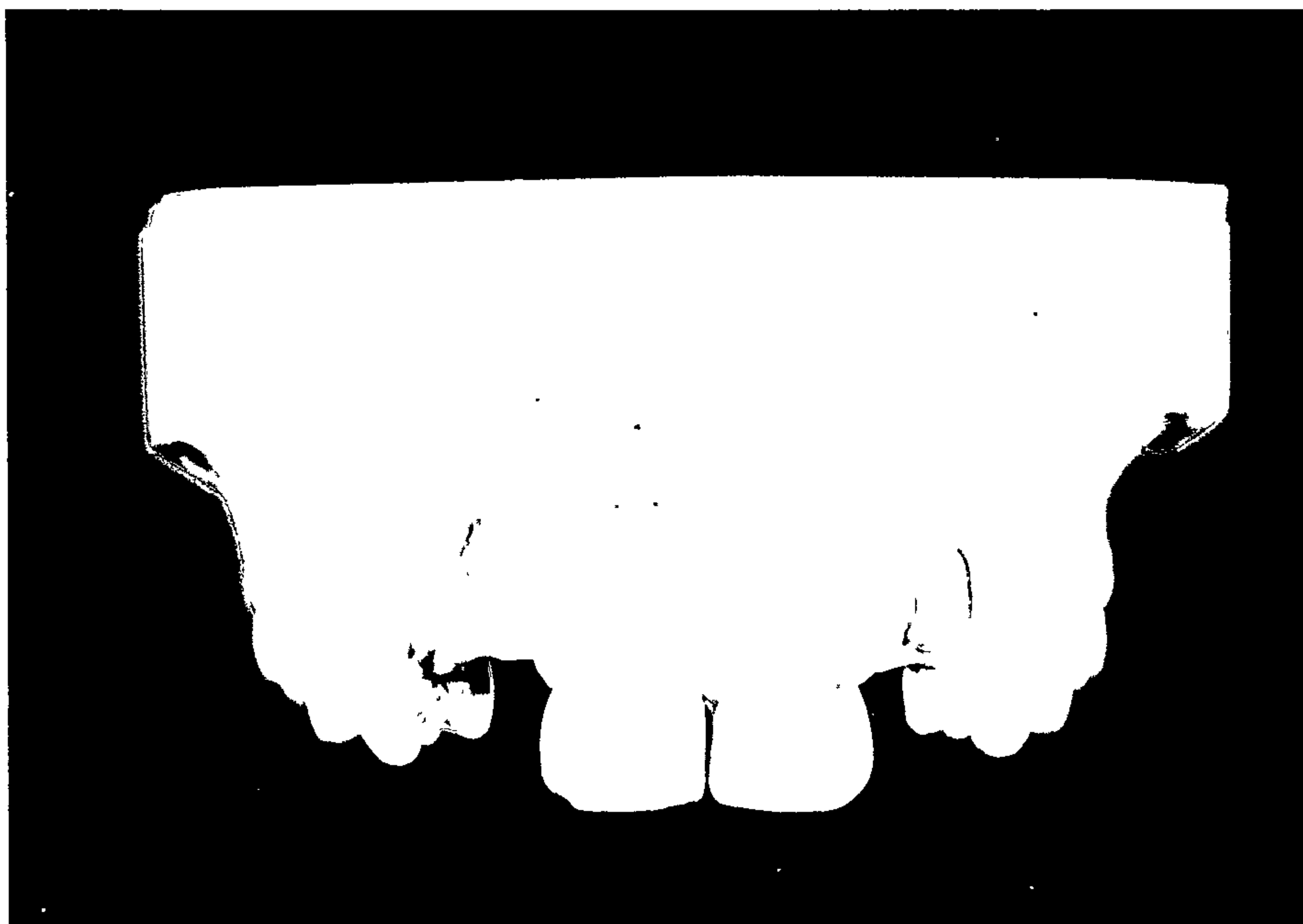


FIGURE 10

A UNILATERAL CONGENITALLY MISSING LATERAL INCISOR

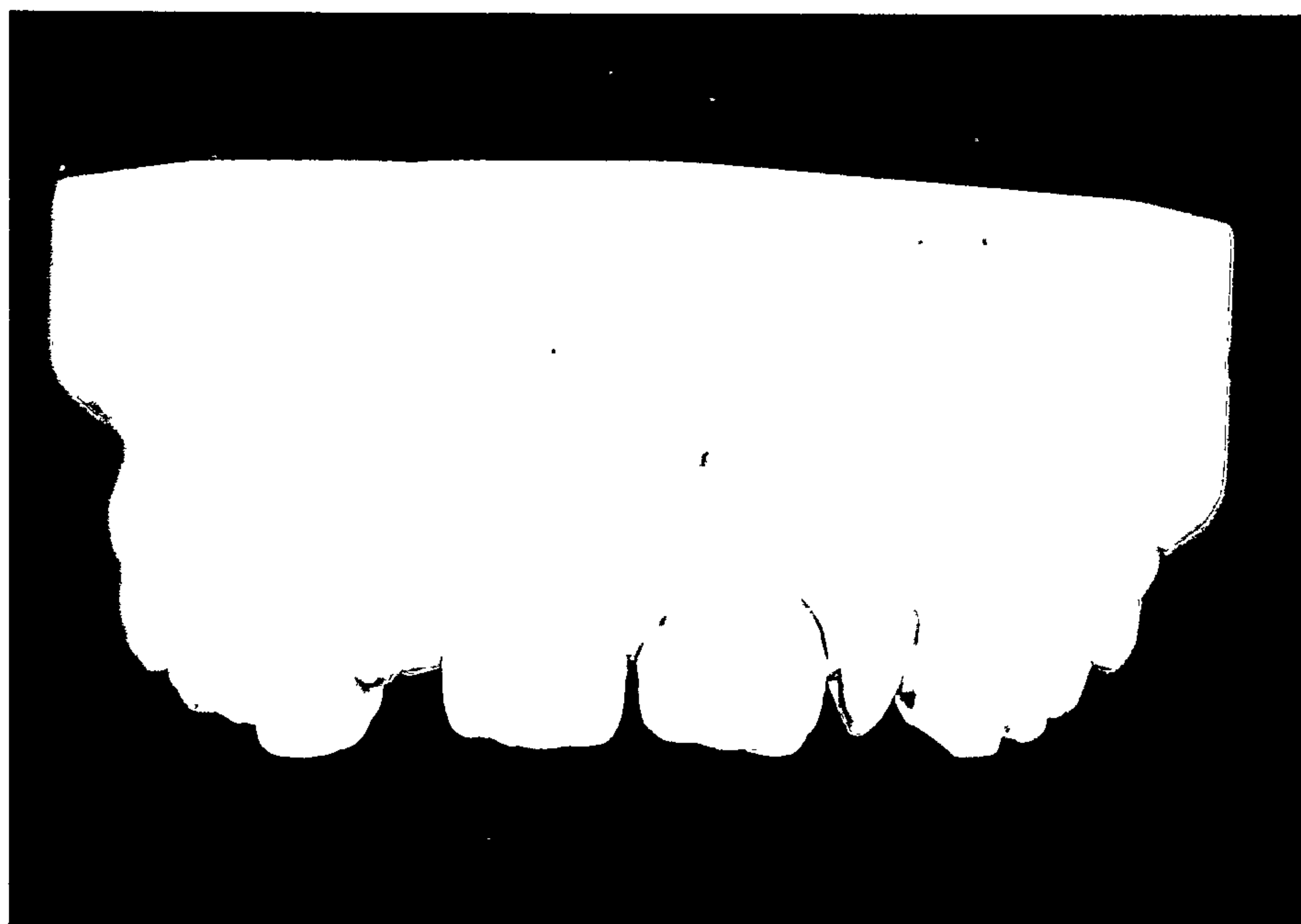


FIGURE 11

A UNILATERAL FRANKLY PEG-SHAPED LATERAL INCISOR

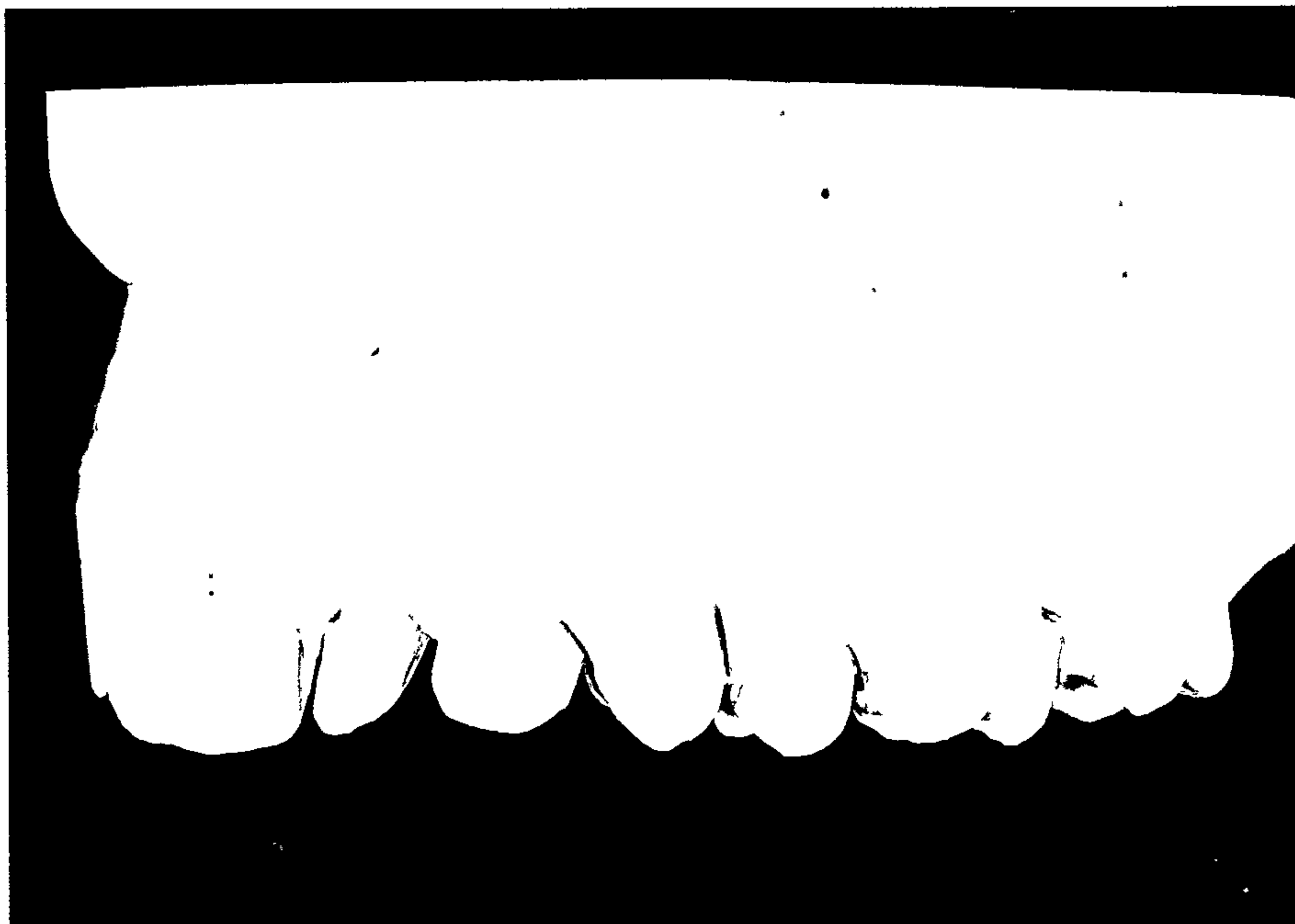


FIGURE 12

BILATERAL FRANKLY PEG-SHAPED LATERAL INCISORS

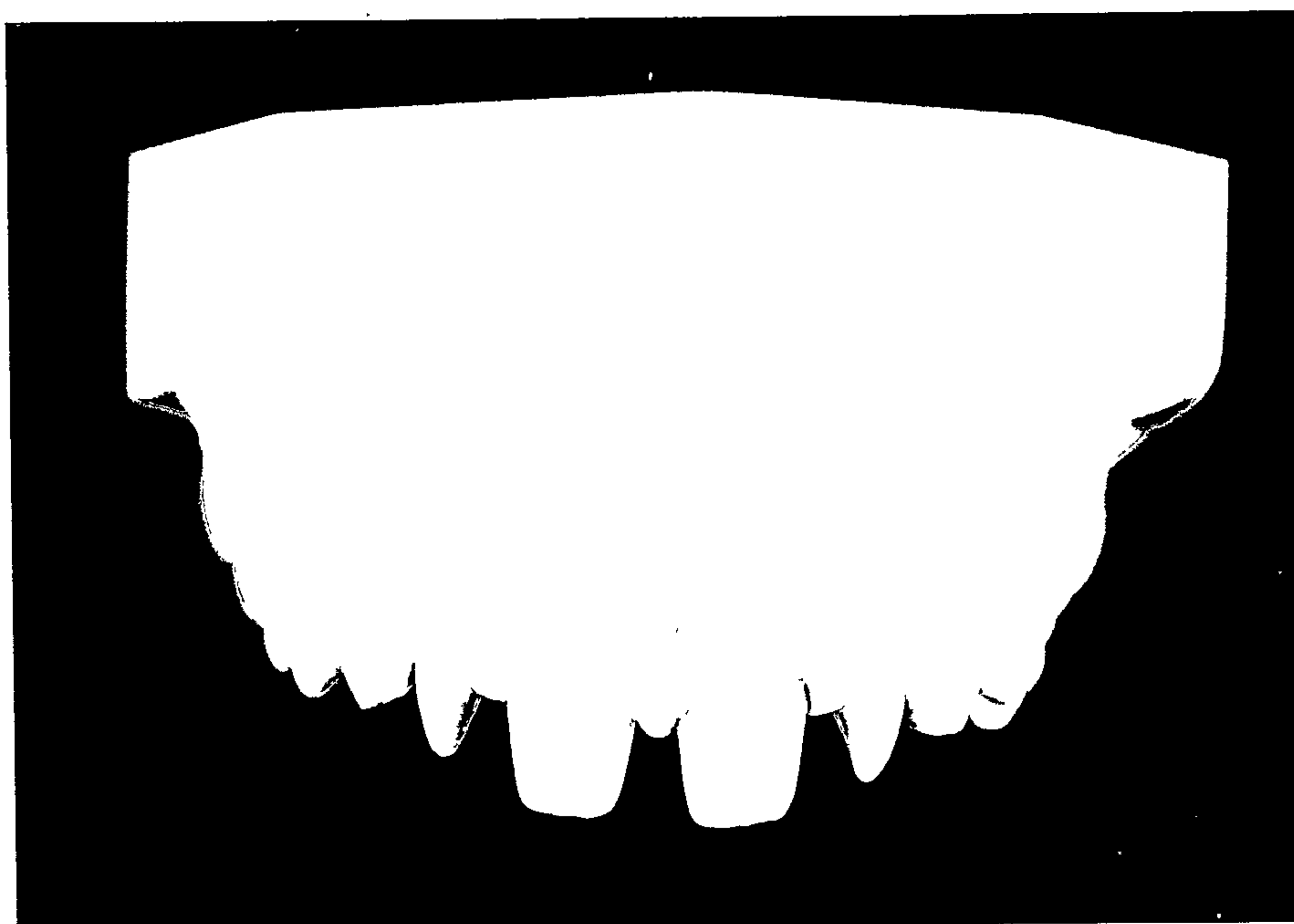


FIGURE 13

A PEG-SHAPED LATERAL INCISOR



FIGURE 14

A SMALL LATERAL INCISOR



Hence for this study, the maxillary permanent lateral incisors were classified according to either of the following categories:

- A. Congenitally missing
- B. Frankly peg-shaped
- C. Peg-shaped
- D. Small
- E. Normal (none of the above)

5.2 Method of Measurement

The maximum mesio-distal diameter of the upper permanent lateral incisors were measured on the plaster study models in order to identify those lateral incisors with abnormal crown morphology according to the foregoing definitions. The lower permanent lateral incisors were similarly measured so as comparison of the upper lateral incisor measurement could be made, allowing the determination of the group 'small' lateral incisors.

5.2.(I) Instrumentation

The Caliper used for measurement

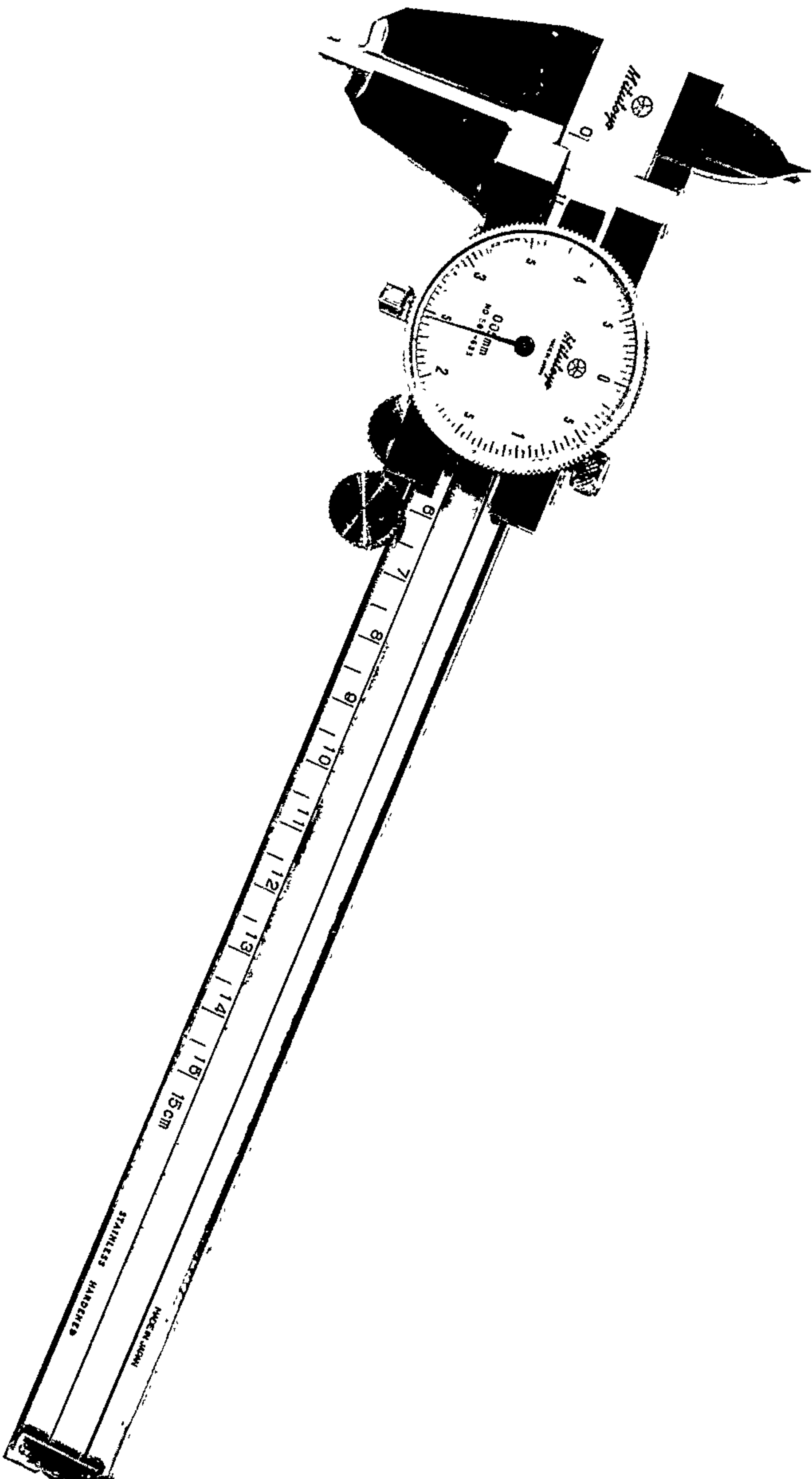
All measurements were made with the same Mitutoyo Dial Calipers (No.505-633) size 15 cm (Figure 9).

To facilitate measuring of the lateral incisor in cases with a distinct lack of space the beaks were reduced to fine points as recommended by Sandilands (1965), Watson (1971), Peck and Peck (1972), and Bau (1973).

The reduction was undertaken similarly to that done by Bau (1973) using heatless stones mounted in a laboratory handpiece, with final polishing being done with rubber polishing wheels. Alcohol was used as a coolant

FIGURE 15

THE MITUTOYO DIAL CALIPERS USED IN THIS STUDY. NOTE THE REDUCED MEASURING TIPS



throughout the reduction to prevent overheating of the instrument which would have caused loss of temper and subsequent distortion. Additional care was taken not to place excessive force on the reduced beaks during the measuring procedure to overcome any subsequent "play" in the tips. This involved light pressure when closing the beaks at the contact points and the avoidance of resting the entire caliper's weight on the fine points. Furthermore, the beaks were fully closed and the caliper dial checked to be correctly zeroed at the completion of each measurement.

5.2.(II) The measuring technique

To familiarize the author with both the instrument and the measuring technique, a training period of 1 week as suggested by Hunter and Priest (1960) was undertaken.

As far as possible, the maximum mesio-distal measurements of the lateral incisors were recorded by the technique advocated by Sandilands (1965) and Watson (1971), wherein the beaks of the caliper were placed:

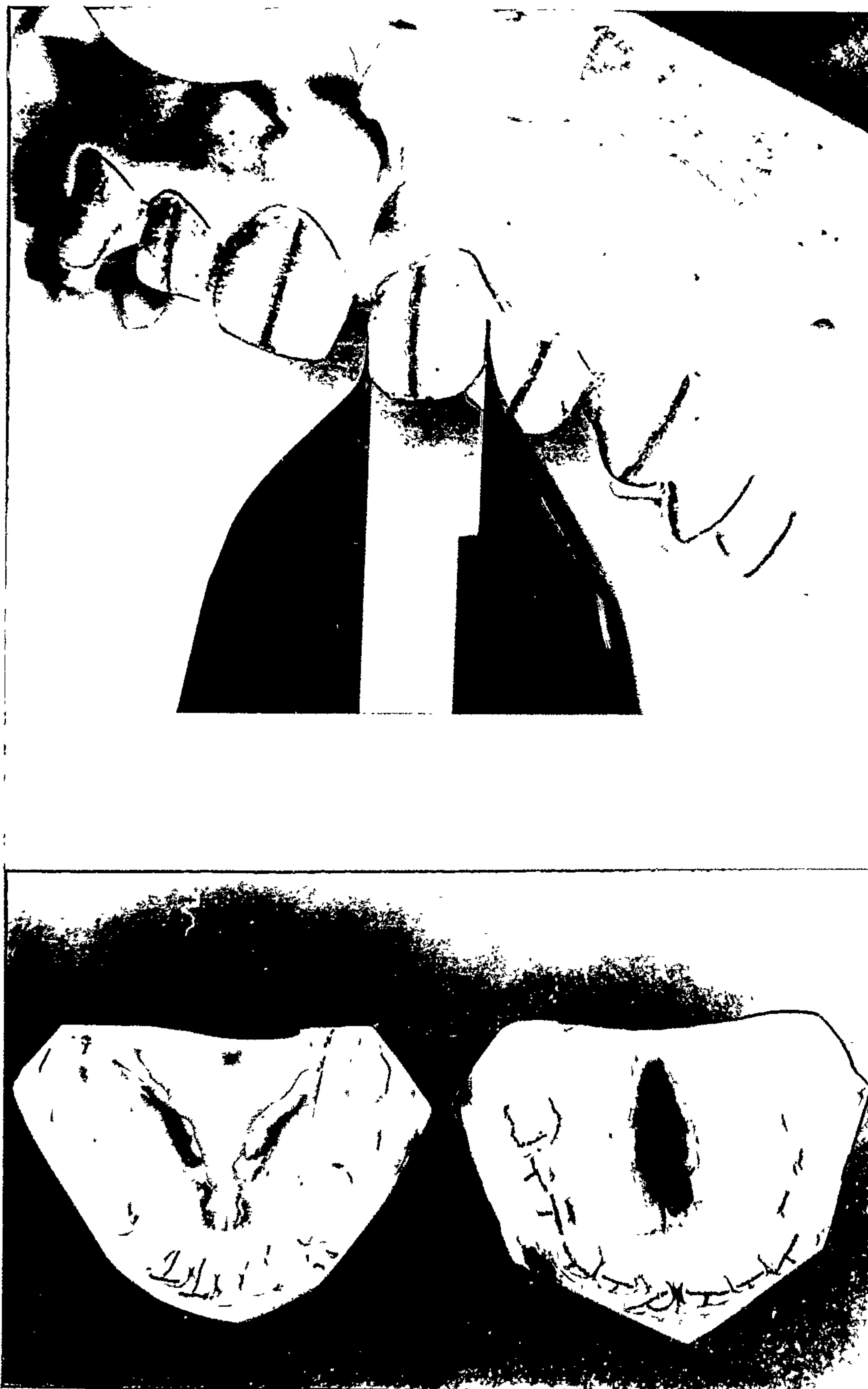
- i) Parallel to the longitudinal axis of the crown
- ii) Perpendicular to the labio-lingual plane
- iii) Perpendicular to the mesio-distal plane (Figure 16).

However, two situations arose where it was found that this method could not be used:

Situation 1: where the greatest mesio-distal diameter of the lateral incisors occurred somewhere between the incisal edge and the cervical margin, the cervical margin measurement could not be obtained by this method (Figure 17).

FIGURE 16

MEASUREMENT METHOD ADVOCATED BY SANDILANDS (1965)
AND WATSON (1971)



Situation 2: where extremely tight contacts made it impossible to insert the calipers into the position advocated, this method again could not be used (Figure 18).

In either of the above two situations a point contact measurement was made by inserting the reduced tips from the labial or lingual; whichever allowed best access to the required measurement (Figure 19).

However, it was apparent that the presence of a reduced lateral incisor or anterior crowding (with lateral incisors rotated), actually facilitated measurements for maximum mesio-distal width made by the advocated method.

The measurements were recorded by the author immediately in millimeters to the nearest 0.05 millimeter, being the accuracy limit placed upon the operator by the recording instrument. As previously stated, the instrument was re-examined with the beaks closed for accuracy at zero before each new measurement. This was carried out to reduce any errors that may have been induced by beak distortion on the previous measurement.

5.2.(III) Reliability test for the method of measurement*

In addition to the training period undertaken to standardize the measuring technique, the same measurement for several cases were recorded on four separate occasions. The time interval between the successive measurements was a minimum of three weeks (Appendix 1).

The maximum difference found between any one of the measurements for the same case was 0.05 mm which is within the limitation imposed by the calibration of the Mitutoyo Dial Calipers.

FIGURE 17

SITUATION 1 IN WHICH THE MEASUREMENT METHOD ADVOCATED BY
SANDILANDS (1965) AND WATSON (1971) COULD NOT BE USED



FIGURE 18

SITUATION 2 IN WHICH THE MEASUREMENT METHOD ADVOCATED BY
SANDILANDS (1965) AND WATSON (1971) COULD NOT BE USED

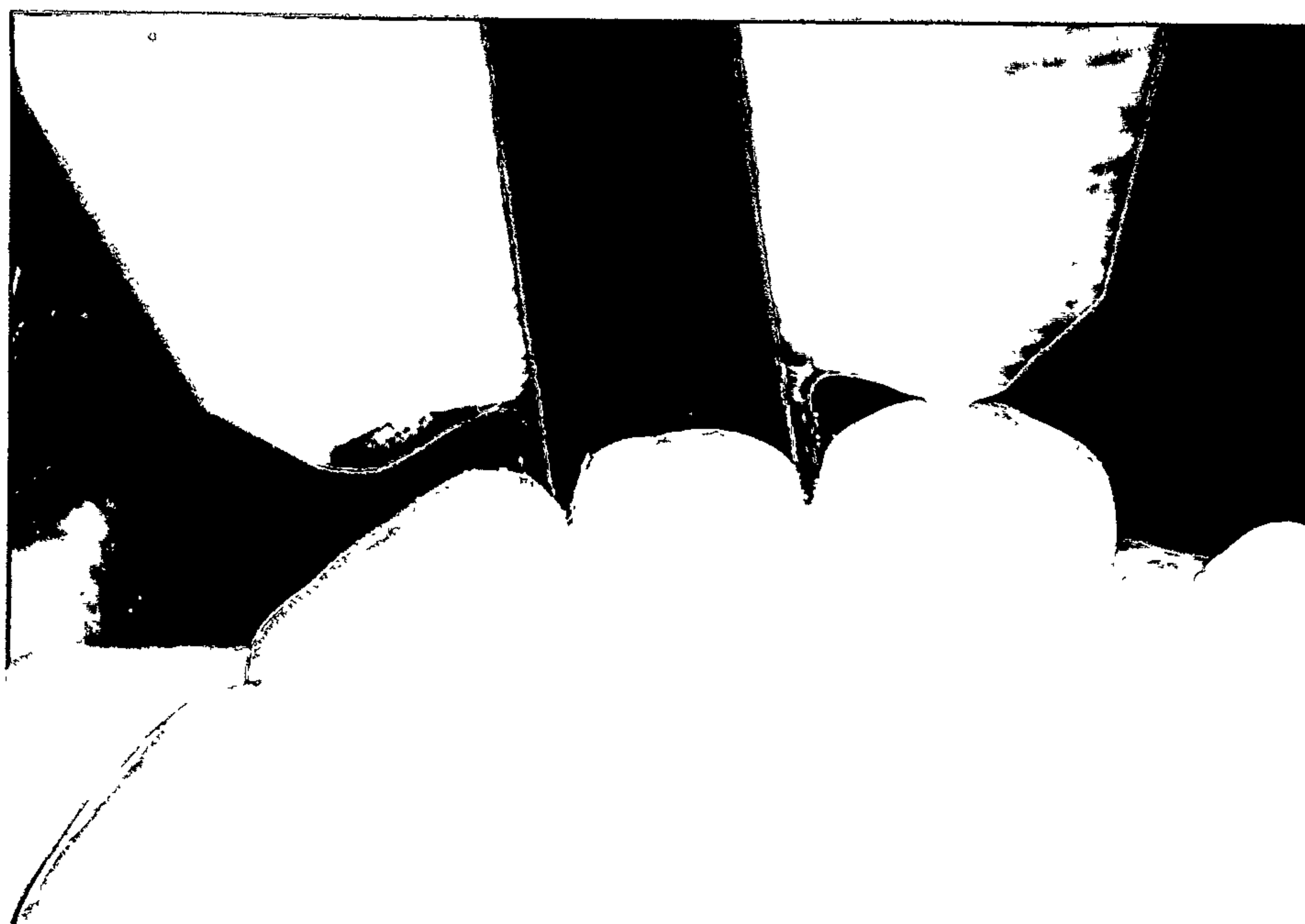


FIGURE 19

THE POINT CONTACT MEASUREMENT METHOD USED TO OVERCOME SITUATIONS 1 AND 2 IN WHICH THE METHOD ADVOCATED BY SANDILANDS (1965) AND WATSON (1971) COULD NOT BE USED. NOTE MEASURING POINTS ARE NOW ACCURATELY PLACED TO OBTAIN THE CORRECT MEASUREMENT.



CHAPTER 6

RESULTS

The maximum mesio-distal width of both the maxillary and mandibular permanent lateral incisors were recorded for the palatally impacted canine group and the non-impacted control group (Appendix 2.)

The incidence of a right or left maxillary lateral incisor congenitally missing or exhibiting abnormal crown morphology (frankly peg-shaped, peg-shaped or small), occurring in a patient with a unilateral or bilateral palatal canine impaction (independent to the site of impaction), were recorded (Appendix 2.i)

The incidence of a maxillary lateral incisor congenitally missing or exhibiting abnormal crown morphology (frankly peg-shaped, peg-shaped or small) occurring on the same side as the palatally impacted canine, were recorded (Appendix 2.vii, 2.xiii)

The incidence of the same maxillary lateral incisor anomalies occurring in the non-impacted control group, as per patient and per side affected, were recorded (Appendix 2.iv, 2.x, 2.xvi)

The difference in incidence of the maxillary lateral incisor anomalies as seen between the impacted group and the non-impacted control group were subjected to a 't' test for statistical significance (Appendix 3) using the following formulae

't' test of percentages (%A vs %B)

$$'t' = \frac{\text{Difference}}{\text{Standard error of the Difference}}$$

$$\text{Difference} = \%A - \%B$$

$$\text{Standard error of the Difference}$$

$$= \sqrt{(\text{SE } A\%)^2 + (\text{SE } B\%)^2}$$

$$SE A\% = \sqrt{\frac{\% A \times q}{N}}$$

$$SE B\% = \sqrt{\frac{\% B \times q}{N}}$$

where q = probability of event failing to occur

N = size of sample

All of the preceding information was then summarized and presented for ease of discussion in the following tables:

TABLE 6

The incidence of an anomalous right or left maxillary lateral incisor occurring in the selected groups

Lateral Incisor Anomaly	Impacted Group %	Control Group %	't' values for significance
A. Congenitally missing	4	2	1.15 (P > 0.20)
B. Frankly peg-shaped	16	2	3.57 (P < 0.001)
C. Peg-shaped	68	24	9.44 (P < 0.001)
D. Small	34	6	5.30 (P < 0.001)

Where the impacted group sample size = 100(N) consisting of 68 females and 32 males and the control group sample size = 100(N) consisting of 49 females and 51 males.

TABLE 7

The incidence of the anomalous maxillary lateral incisor occurring on the same side as the impacted maxillary canine

Lateral Incisor Anomaly	Impacted Group %	Control Group %	't' values for significance
A. Congenitally missing	1.49	2.00	1.44 (P > 0.20)
B. Frankly peg-shaped	14.92	1.00	4.00 (P < 0.001)
C. Peg-shaped	52.96	14.50	7.72 (P < 0.001)
D. Small	29.85	4.50	5.83 (P < 0.001)

Where the impacted group sample size = 134(N) and the non-impacted control group sample size = 200(N).

Overall, no significant differences were found in the incidences of congenitally missing lateral incisors between the impacted group and the non-impacted control, regardless of whether we examine the incidence of the lateral incisor agenesis occurring on the same side as the impaction or whether examined independent from the impaction site. However, significant differences were observed in the incidence of lateral incisor abnormal crown morphologies (frankly peg-shaped, peg-shaped, small), when the impacted and non-impacted control groups were compared. This was apparent in the incidence of the lateral incisor abnormal crown morphologies both on the same side as the impacted canine and also when examined independently of the impaction site.

6.1 Congenitally missing lateral incisors

The difference in the percentage incidence of congenitally missing lateral incisors occurring independent to the impaction site, between the impacted canine group and the non-impacted control group was found to be 2% (Table 6, row A). The difference in the percentage incidence of congenitally missing lateral incisors occurring on the same side as the impacted canine between the impacted and non-impacted control groups was found to be 0.51% (Table 7, row A). When these percentage differences were subject to a 't' test they were found to be statistically insignificant for $P < 0.001$.

6.2 Abnormal crown morphology of the lateral incisors

The comparison of abnormal crown morphology incidences yielded significant differences between the canine impacted group and the non-impacted control. The impacted group tended to be significantly larger in all instances:

6.2.(I) Frankly peg-shaped

The incidence of right or left frankly peg-shaped lateral incisors was found to be greater in the impacted group (16%) than the non-impacted control group (2%), as shown in Table 6, row B. The difference in incidence (14%) when subjected to a 't' test was found to be statistically significant for $P < 0.001$. This significant difference was also shown to exist in the incidence of the lateral incisor on the same side as the impacted canine between the impacted group (14.92%) and the control (1.00%), as shown in Table 7, row B.

6.2.(II) Peg-shaped

The largest differences in incidence of abnormal crown morphology between impacted and non-impacted groups, were found with 'peg-shaped lateral incisors. The incidence of peg-shaped lateral incisors occurring independent to the site of impaction in the impacted group was 68% (Table 6, row C), a significant difference of 44% from that found in the non-impacted control group (24%). Also, a significant difference of 28.36% was found between the impacted group (52.96%) and the non-impacted control group (14.50%) in the incidence of peg-shaped lateral incisors occurring on the same side as the canine impaction (Table 7, row C).

6.2.(III) Small

Similar to the findings expressed with frankly peg-shaped and peg-shaped lateral incisors, the difference in incidences of small lateral incisors between impacted and non-impacted control groups was found to be significant, when examined for occurrence independent to the site of impaction (Table 6, row D), and occurrence on the same side as the impaction (Table 7, row D); the difference being 28% in the former and 25.33% in the latter.

CHAPTER 7

DISCUSSION

The primary finding of this study is that for the sample examined, subjects with palatal canine impaction were found to have a significantly higher incidence of lateral incisors with abnormal crown morphology (frankly peg-shaped, peg-shaped and small) than subjects presenting without palatal canine impaction. This finding was also reflected in the incidence of lateral incisors with abnormal crown morphology occurring on the same side as the palatal canine impaction. Furthermore, no significant difference could be found in the subjects examined in the incidence of congenitally missing lateral incisors occurring with or without palatal canine impaction.

When interpreting the findings of this study, the following limitations should be kept in mind:

(i) The delineation of the lateral incisors into the groups of congenitally missing, frankly peg-shaped, peg-shaped, small and normal, as found by Becker et al (1981) allowed specific categorization by the operator of the lateral incisor anomalies and easier result tabulation. In the instances of congenitally missing, peg-shaped, small and normal lateral incisors the categorization process was accomplished objectively with both ease and accuracy. In the case of frankly peg-shaped lateral incisors, the classification became more of a subjective nature being based upon the investigator's past experience and interpretation.

This group may then vary between individual operators.

(ii) Both groups of subjects examined, the impacted and non-impacted control, were derived from patients presenting at the

Orthodontic Department of the United Dental Hospital, having been referred from other Departments within the same Hospital, specifically for the treatment of malocclusions. However, there may exist a large number of Hospital patients with abnormalities of the upper lateral incisors but without any other form of malocclusions, who have not had the need to be referred to the Orthodontic Department. These patients would then have not been included in the sample groups. Therefore, both groups may be seen as being biased in that they represent patients with malocclusions, and hence may not be true cross-sectional samples of the general community.

(iii) Furthermore, the impacted canine group represents those patients that had undergone oral surgery to remove, expose and/or chain their palatally impacted canines. This group then, may incorporate another bias in that it represents only those palatal canine impactions requiring oral surgery as part of their treatment, being completely devoid of palatal canine impactions recovered by other means, or left in situ untreated.

However, given these limitations it is interesting to observe that the randomly selected canine impacted group consisted of 68 females and 32 males; a ratio predisposition of 2:1 to females. This sexual dimorphism is similar to figures quoted by several previous investigators (Dewel, 1949; Rohrer, 1929; Dachi and Howell, 1961; Bass, 1967; Aitasalo et al., 1972; Takahama and Aiyama, 1982). Also the incidence of 2% found for congenitally missing maxillary lateral incisors in the randomly-selected non-impacted control group (Table 6, row A) is similar to that found by other researchers (Rose, 1906; Campbell, 1934; Grahnen, 1956; Eidelman et al., 1973; Bot and Salmon, 1977; Becker et al., 1981), and only slightly above the 1.8% found by Davies (1968) in a group of Sydney

schoolchildren. These observations seem to indicate that even though the sample groups may contain biases, the groups are not that diverse from the general population. Hence, the limitations of sampling may not have had that significant an influence on the overall result found, of an association between the impacted palatal canine and lateral incisor anomalies.

The finding of this study, that the frequency of lateral incisors with abnormal crown morphology is significantly higher in subjects with canine impaction, is in agreement with the findings of the only other similar cited investigation conducted by Becker et al (1981). The additional finding of this study, that the frequency of congenitally missing lateral incisors is not significantly higher in subjects with palatal canine impaction, although agreeing with the findings of Becker et al (1981), disagrees with the findings of Bass (1967). Bass (1967) found a significantly higher level of "congenitally missing teeth in the buried canine group, compared with the random group" he had investigated. However, Bass (1967) made no distinction as to the position of the 'buried canine', only stating "the patients had unerupted upper canine teeth which were surgically treated", and also included premolars in his group of "congenitally missing teeth".

According to the previously discussed conjectured causes for the association of lateral incisor anomalies with palatal canine impactions (Chapter 3), the findings of this present study come as no great surprise to the author. It was suggested by the author that in the case of a congenitally missing lateral incisor, the canine although possibly affected by the same etiologic factors as the lateral, or by the loss of its eruptive guide, would however have an equal opportunity to erupt normally within the arch, due to the additional space created by the

missing lateral incisor. This would then account for the finding of no significant difference in the occurrence of congenitally missing lateral incisors, between the impacted and non-impacted control groups. The situation changes when we consider the lateral with abnormal crown morphology. In these instances the rudimentary lateral would tend to act as a deflection to the late rectifying movements of an erupting canine (Chapter 3.1.1); this being reflected in the finding of a highly significant difference in their occurrence between the canine impacted and non-impacted control groups.

The limitations of sampling imposed upon this study indicate that further investigations need to be conducted to establish the relevance of the results to the clinician when dealing with the general population. A cross-sectional sample of the general population would need to be examined both for the total number of palatal canine impactions in association with lateral incisor anomalies and conversely, the total number of lateral incisor anomalies having an associated palatal canine impaction. The ideal study would endeavour to investigate a random population sample of lateral incisor anomalies longitudinally, from emergence of the lateral incisor into the oral cavity, to the emergence or impaction of the maxillary canine.

Nevertheless, the ease of identification of a lateral incisor with abnormal crown morphology and the early appearance of this tooth in the permanent dentition series, would ideally suit it to the role of an advanced indicator of palatal canine impaction. The results of this study and that of Becker et al (1981), seem to indicate in the samples investigated, for a highly significant number of palatal canine impactions, the lateral incisor's role as an early indicator to the palatal impaction, would have been justified.

CHAPTER 8

SUMMARY AND CONCLUSIONS

8.1 After reviewing the literature, it was found that similarities do exist in the incidences and conjectured etiologies of maxillary impacted canines (Chapter 1) and the maxillary lateral incisor anomalies (Chapter 2). Several reports of, and studies on, the association of the palatally impacted canines with the anomalous maxillary lateral incisor have been cited and several hypothetical reasons for this association suggested (Chapter 3). However, controversy arises since many of the suggested etiologies for either event, could be themselves the result of some yet undefined hereditary design and/or environmental influence. Therefore they may have no specific cause/effect relationship. Until further research uncovers the exact nature for canine impaction or anomalous lateral incisors, the only possible conclusion that can be made, is that these two events are the result of a complex interrelationship of many evolutionary, hereditary, glandular and environmental factors. Somewhere within this as yet undefined complexity may be a specific association between the etiology of palatally impacted canines and the anomalous maxillary lateral incisor.

8.2 An investigation was undertaken to establish if there existed any significant increase in the incidence of maxillary lateral incisor anomalies in patients presenting with palatally impacted canines (Chapter 4).

8.3 Two groups of subjects; a palatally impacted canine group and a non-impacted control group, were selected from patients presenting for

Orthodontic treatment at the Orthodontic Department of the United Dental Hospital, Sydney. For both groups, the congenital absence and the occurrence of "frank pegging" of the maxillary lateral incisor was noted. The mesio-distal dimensions of the maxillary lateral incisors were also recorded, using an accurately calibrated instrument (Chapter 5). The incidence of a lateral maxillary incisor being congenitally absent, frankly peg-shaped, peg-shaped or small (as defined in Chapter 5.1) were calculated for both groups of subjects examined (Tables 6 and 7).

8.4 It was found for the subjects examined, that no significant difference existed in the incidence of congenitally missing lateral incisors between the palatally impacted canine group (4%) and the non-impacted control group (2%). However, a marked significant difference existed in the incidence of lateral incisors with abnormal crown morphology, between the palatally impacted canine group (frankly peg-shaped, 16%; peg-shaped, 68%; small, 34%) and the non-impacted control group (frankly peg-shaped, 2%; peg-shaped, 24%; small, 6%). Similar findings were noted by Becker et al (1981). A similar significant difference was also found to be apparent between the two groups, when examining the incidence of a maxillary lateral incisor with abnormal crown morphology occurring on the same side as the canine impaction (Table 7).

8.5 Several limitations were discussed in the method of obtaining the impacted canine group, the non-impacted control group and the subjective delineation of the "frankly peg-shaped" lateral maxillary incisor. It was felt these limitations did not markedly effect the overall results obtained. However, it was recommended that further investigation of a general population sample would be advantageous in determining if indeed a similar significant association, as that found in this study, exists between lateral incisor anomalies and palatal

impacted canines for the general population (Chapter 7).

8.6 It can be concluded for the subjects studied, the lateral incisor with abnormal crown morphology has a higher incidence in patients presenting with palatal impacted canines. Hence, in the subjects with palatal canine impactions, in the majority of instances, the form of the maxillary lateral incisor could have been utilized as an early diagnostic 'warning sign' to that impaction. This information may be useful to the clinician as an earlier indicator of an impending palatal canine impaction, and the earlier instigation of interceptive procedures to prevent that impaction.

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APPENDIX 1

TABLE 8: RELIABILITY MEASUREMENTS

Measurements made to verify reliability of the measuring technique.

TABLE 8

CASE	PATIENT	MEASURE	DATE	... 42	RIGHT SIDE 12 cerv.	... 22	LEFT SIDE 32 cerv.	...
5	m	1st	6.1.	5.85	5.65	6.20	6.00	5.55
		2nd	20.3.	5.80	5.65	6.20	6.05	5.50
		3rd	18.4.	5.80	5.70	6.20	6.00	5.50
		4th	19.5.	5.80	5.65	6.20	6.00	5.50
16	S	1st	6.1.	6.00	7.05	7.25	6.90	6.75
		2nd	20.3.	6.00	7.10	7.25	6.90	6.75
		3rd	18.4.	6.00	7.10	7.25	6.90	6.80
		4th	19.5.	6.00	7.10	7.20	6.90	6.75
24	V	1st	10.2.	5.95	6.85	6.80	6.75	6.75
		2nd	20.3.	6.00	6.80	6.80	6.80	6.80
		3rd	18.4.	6.00	6.85	6.80	6.80	6.80
		4th	19.5.	6.00	6.80	6.80	6.80	6.80
32	O	1st	10.2.	5.50	6.40	6.05	6.05	6.20
		2nd	20.3.	5.55	6.40	6.00	6.00	6.20
		3rd	18.4.	5.50	6.40	6.00	6.00	6.25
		4th	19.5.	5.50	6.40	6.00	6.00	6.20
47	T	1st	15.2.	5.45	5.15	5.55	5.60	5.10
		2nd	20.3.	5.45	5.15	5.55	5.60	5.10
		3rd	18.4.	5.45	5.10	5.50	5.60	5.15
		4th	19.5.	5.40	5.15	5.55	5.60	5.10
59	M	1st	10.3.	6.35	6.05	6.00	6.35	6.00
		2nd	20.3.	6.35	6.05	6.00	6.30	6.00
		3rd	18.4.	6.35	6.00	6.00	6.35	6.00
		4th	19.5.	6.30	6.00	6.00	6.35	6.00
68	M	1st	10.3.	5.40	5.50	5.60	5.55	5.60
		2nd	20.3.	5.40	5.55	5.60	5.50	5.60
		3rd	18.4.	5.40	5.55	5.60	5.50	5.55
		4th	19.5.	5.40	5.55	5.60	5.50	5.60
78	C	1st	11.3	6.05	7.20	7.15	7.10	7.25
		2nd	20.3.	6.00	7.20	7.10	7.10	7.25
		3rd	18.4.	6.00	7.20	7.10	7.10	7.20
		4th	19.5.	6.00	7.20	7.10	7.10	7.25
86	D	1st	14.3.	6.20	6.90	7.20	6.70	6.65
		2nd	20.3.	6.20	6.90	7.20	6.75	6.60
		3rd	18.4.	6.20	6.95	7.20	6.70	6.60
		4th	19.5.	6.15	6.90	7.20	6.70	6.60
4	C	1st	14.3.	6.65	7.70	8.20	8.20	7.80
		2nd	20.3.	6.60	7.65	8.20	8.20	7.80
		3rd	18.4.	6.60	7.65	8.20	8.20	7.75
		4th	19.5.	6.60	7.70	8.20	8.20	7.80

APPENDIX 2

Compilation of record sheets for the incidence of the maxillary lateral incisor anomalies occurring in the canine impacted and non-impacted groups.

TABLE 9 THE CANINE IMPACTED GROUP
indicating

- i) Sample size = 100
- ii) Male:Female = 32:68
- iii) All mesio-distal measurements of the maxillary and
mandibular lateral incisors
- iv) The incidence of right or left congenitally missing
lateral incisors (θ) = 4
- v) The incidence of right or left frankly peg-shaped
lateral incisors (*) = 16
- vi) The incidence of right or left peg-shaped lateral
incisors (∇) = 68
- vii) The incidence of right or left small lateral incisors
(+) = 34

TABLE 9

C.M. = congenitally missing

	PATIENT	SEX	IMPACTION	RIGHT SIDE....			 LEFT SIDE ...		
				42	12		21	32	
				cerv.			cerv.		
+V1	F	F	UR3	6.40	6.15	6.15	6.30	6.35	6.35
V2	N	F	UL3	5.30	5.75	5.75	5.95	6.05	5.25
+V*3	B	M	bilat	5.20	4.10	4.15	4.25	4.70	5.30
+V*04	M	F	UR3	6.00	4.20	3.05	C.M.*		5.90
5	M	F	UL3	5.85	5.65	6.20	6.00	5.55	5.30
6	B	F	UR3	6.15	6.90	7.55	7.50	7.25	6.25
7	B	F	UL3	6.75	6.65	6.90	6.80	6.45	6.70
8	A	M	UR3	5.95	6.10	6.50	6.25	5.80	6.05
+V*9	T	F	UR3	5.55	4.95	3.65	3.45	4.65	5.80
10	S	F	UL3	6.20	7.35	7.40	6.95	6.90	5.70
11	A	M	UL3	6.45	6.70	6.90	7.25	6.85	6.05
V12	G	M	bilat	5.90	6.15	6.20	6.41	6.50	5.65
+V*13	H	M	bilat	3.85	4.30	3.40	3.60	4.20	4.00
+V*14	S	F	UR3	5.65	6.10	5.75	5.60	5.80	5.55
+V15	D	F	bilat	6.15	6.00	6.15	6.40	6.65	6.30
16	S	M	UL3	6.00	7.05	7.25	6.90	6.75	6.65
+V*17	T	F	UL3	5.35	5.90	5.65	5.60	5.85	5.40
+V*18	W	F	UL3	5.80	5.70	5.55	5.05	5.35	5.45
19	E	F	UL3	6.30	6.55	6.65	7.25	6.80	6.30
20	B	M	bilat	6.00	7.55	7.60	7.40	7.15	6.70
21	L	F	UR3	5.70	6.35	6.40	5.95	5.90	5.50
22	M	M	UR3	5.80	7.45	7.50	6.95	6.55	4.94
23	G	F	bilat	6.00	6.65	7.00	7.15	6.95	6.75
V24	V	M	UL3	5.95	6.85	6.80	6.75	6.75	5.80
V25	V	M	bila-	6.30	6.60	6.45	6.60	7.00	6.20
V26	B	V	UF3	5.95	6.30	6.20	6.15	6.15	5.90
+27	S	F	UR3	6.05	5.15	5.25	5.70	5.20	6.00
V28	F	F	bilat	5.90	6.45	6.30	6.30	6.05	6.05
V29	J	M	UL3	6.50	6.95	6.90	7.25	7.30	6.85
V30	J	F	UR3	5.65	6.40	6.00	5.55	5.85	5.45
V31	K	F	bilat	5.90	6.70	6.85	6.85	7.00	absent
V32	O	M	UL3	5.50	6.40	6.05	6.05	6.20	5.45
V33	J	F	UL3	5.00	5.85	5.65	6.40	6.15	5.00
34	D	F	UL3	5.75	6.00	6.20	5.90	5.80	5.80
V35	S	F	UL3	5.70	6.55	6.70	6.60	6.60	5.50
36	I	F	bilat	6.60	8.55	8.70	8.10	7.90	6.50
37	N	M	UR3	6.40	8.30	8.55	8.00	7.85	6.35
38	H	F	UR3	6.00	6.50	7.20	7.05	7.00	5.95
39	H	M	UR3	5.45	6.30	6.45	6.40	5.90	5.45
V40	G	F	UR3	6.50	6.75	6.95	7.50	7.65	6.30
+41	B	F	bilat	6.45	6.25	6.45	6.30	5.85	6.40
+V42	M	F	UL3	5.65	5.65	5.35	5.45	5.85	5.65
V43	D	F	UL3	6.30	7.15	7.10	7.55	7.30	6.50
+V*044	H	F	UR3	6.50	4.10	3.55	C.M.*		5.90
45	W	M	UR3	6.50	7.60	7.80	8.30	8.20	6.90
+V*46	J	F	UL3	5.45	4.40	4.10	3.80	4.30	5.50
47	T	F	UR3	5.45	5.15	5.55	5.60	5.10	5.25
48	S	F	UR3	6.75	6.95	7.30	7.80	6.70	6.80
V49	K	M	UR3	6.40	7.30	7.30	7.00	6.60	6.40
50	U	F	bilat	5.60	6.95	7.00	6.80	6.60	5.55
V51	W	M	bilat	6.00	6.95	6.90	7.70	7.50	5.60
+V52	D	F	UL3	6.75	6.65	5.95	5.85	6.80	6.80
53	F	F	UL3	6.30	7.15	7.45	7.25	6.65	6.15

TABLE 9 continued

	PATIENT	SEX	IMPACTION	RIGHT SIDE...			 LEFT SIDE ...		
				42	12		21	32	
				cerv.			cerv.		
54	T	M	bilat	5.90	6.65	6.70	7.05	6.90	5.95
55	F	F	UL3	6.60	7.10	7.50	7.40	7.00	6.60
∇56	W	F	UL3	6.80	7.45	7.30	7.55	7.30	6.85
∇57	S	F	bilat	6.10	6.60	6.20	6.70	6.55	5.90
+∇58	M	F	bilat	5.75	5.50	5.30	5.50	5.65	5.80
+∇59	M	F	UR3	6.35	6.05	6.00	6.35	6.00	6.05
∇60	C	F	UL3	5.65	6.75	6.75	6.35	6.25	5.60
∇61	D	M	UR3	6.15	7.00	7.10	7.20	7.35	6.00
+∇62	S	M	UL3	5.90	6.05	5.90	5.60	5.65	5.85
∇63	S	M	UR3	6.75	7.60	7.60	7.50	7.40	6.60
+∇*64	F	F	UR3	5.65	5.95	5.90	5.60	5.90	5.60
∇65	J	F	UL3	5.40	6.10	6.10	6.10	6.00	5.60
∇66	T	F	bilat	6.10	6.80	6.70	6.70	6.80	5.80
+∇*67	M	F	bilat	6.10	5.90	5.70	5.55	5.70	6.15
+∇68	M	F	UL3	5.40	5.50	5.60	5.55	5.60	5.60
+∇69	R	F	UR3	6.40	6.20	6.20	6.70	6.50	6.40
+∇*70	D	F	UR3	6.10	6.20	5.80	6.60	6.40	6.10
+∇71	L	F	UL3	5.90	6.50	6.20	5.70	6.00	6.00
+∇72	S	F	bilat	6.40	6.30	6.20	6.30	5.75	6.60
∇73	S	F	UL3	5.30	5.90	5.70	6.20	6.45	5.55
+∇74	L	F	UL3	5.70	5.60	5.40	5.20	5.40	5.90
+∇75	B	F	UR3	5.90	6.00	5.90	6.10	5.95	5.70
∇76	E	M	bilat	5.65	5.85	5.80	6.00	5.85	5.50
∇077	G	M	UL3	absent	C.M.*		7.50	7.20	6.85
∇78	C	M	UR3	6.05	7.20	7.15	7.10	7.25	6.05
79	B	F	bilat	7.00	7.70	7.90	7.70	7.60	7.00
+∇*80	B	F	bilat	6.20	6.00	5.65	4.50	5.40	6.05
+∇*81	H	F	bilat	6.30	6.80	6.50	6.15	6.60	6.25
∇82	H	F	bilat	6.00	6.80	6.80	6.80	6.70	6.30
∇83	M	M	UL3	6.75	7.40	7.50	7.10	7.30	6.80
∇84	S	M	bilat	6.20	7.40	7.40	6.90	7.10	6.50
85	M	F	UL3	6.05	6.90	7.15	6.70	6.35	6.00
86	D	F	UL3	6.20	6.90	7.20	6.70	6.65	5.85
+∇87	M	F	bilat	5.90	6.50	6.20	5.50	5.50	5.90
∇88	P	M	UR3	6.00	7.80	7.40	7.00	7.10	5.90
+∇*89	W	M	bilat	6.20	6.80	6.80	4.60	5.50	6.55
+∇90	H	F	UL3	5.80	5.50	5.50	5.85	5.70	6.05
∇91	B	F	UR3	6.35	6.60	6.60	7.30	7.25	6.50
92	W	M	bilat	6.60	8.40	8.55	8.50	8.45	6.35
93	E	F	bilat	6.30	6.40	6.45	6.55	6.50	6.40
∇094	B	F	bilat	5.80	C.M.*		C.M.*		5.85
∇95	K	F	UR3	6.15	7.70	7.50	7.40	7.80	6.30
∇96	T	M	bilat	6.30	6.80	6.80	6.90	7.30	6.35
+∇97	W	F	bilat	6.10	6.80	6.80	6.20	6.15	6.20
98	S	F	bilat	6.20	6.65	7.20	6.95	6.05	6.20
+∇*99	S	F	bilat	6.00	5.85	5.00	4.90	5.80	6.10
∇100	N	M	UL3	6.40	6.95	7.30	7.00	7.25	6.35

TABLE 10 THE NON-IMPACTED CONTROL GROUP

indicating

- i) Sample size = 100
- ii) Male:Female = 51:49
- iii) All mesio-distal measurements of the maxillary and mandibular lateral incisors
- iv) The incidence of right or left congenitally missing lateral incisors (θ) = 2
- v) The incidence of right or left frankly peg-shaped lateral incisors (*) = 2
- vi) The incidence of right or left peg-shaped lateral incisors (∇) = 24
- vii) The incidence of right or left small lateral incisors (+) = 6

TABLE 10

C.M. = congenitally missing

PATIENT			SEX	 RIGHT SIDE			 LEFT SIDE		
				42	12	21	32		
				cerv.			cerv.		
∇1	M	M	6.20	7.70	7.50	7.60	7.70	6.50	
∇2	R	M	6.15	7.20	7.00	6.90	6.80	6.30	
3	P	F	5.30	6.80	6.90	7.10	6.70	5.90	
∇4	C	M	6.65	7.70	8.20	8.20	8.20	6.60	
5	M	M	6.15	7.40	7.45	7.50	7.30	6.10	
6	W	M	6.25	7.30	7.45	7.55	7.30	6.20	
7	R	M	5.80	6.90	7.10	7.30	7.20	5.90	
+8	J	M	6.70	6.40	6.45	6.60	6.50	6.20	
9	C	M	6.05	6.70	6.80	6.80	6.70	6.00	
∇10	L	M	6.90	7.30	7.10	6.70	6.70	6.60	
11	Z	M	6.05	6.40	6.80	6.65	6.60	6.00	
∇12	C	F	6.15	7.60	7.70	7.50	7.50	6.00	
∇13	H	M	5.40	6.00	6.10	6.05	6.10	5.30	
14	H	M	5.80	6.30	6.45	6.50	6.40	6.00	
15	H	F	6.10	6.35	6.45	6.55	6.50	6.05	
∇16	W	M	6.35	8.00	7.90	7.90	8.00	6.40	
∇17	D	F	6.20	7.10	6.70	6.60	6.75	6.15	
∇18	B	M	6.55	7.40	7.30	7.95	7.30	6.50	
19	P	M	5.90	6.30	6.70	6.60	6.40	5.90	
∇20	S	M	6.85	7.60	7.65	7.60	7.70	7.00	
∇21	K	M	6.05	6.60	6.50	6.95	7.10	5.70	
22	S	F	5.80	7.50	7.55	7.20	7.10	5.80	
23	K	F	5.95	6.85	6.85	6.90	6.85	5.90	
24	G	F	5.50	6.00	6.25	6.00	6.00	5.60	
25	S	F	5.95	6.50	6.55	6.60	6.80	6.00	
+26	S	F	6.30	6.10	6.20	6.30	6.40	6.30	
27	S	M	5.70	6.30	6.50	6.45	6.30	5.55	
+∇28	D	M	6.25	6.75	6.70	6.40	6.60	6.45	
29	P	M	6.30	7.30	7.50	7.20	7.10	6.35	
30	M	F	6.25	7.80	8.00	7.45	7.30	6.20	
31	C	F	6.10	6.65	6.70	6.70	6.50	6.15	
32	R	F	6.30	7.00	7.10	7.15	7.00	6.10	
33	S	M	6.90	7.60	7.65	7.60	7.55	6.80	
34	H	F	6.00	6.65	6.70	6.80	6.70	5.80	
35	G	M	6.50	7.65	7.70	7.40	7.35	6.15	
36	B	F	6.15	7.05	7.20	7.30	7.15	6.00	
37	B	M	5.55	6.30	6.45	6.30	6.25	5.75	
38	O	M	6.60	8.10	8.30	8.50	8.30	6.70	
39	C	F	5.75	6.65	6.80	6.90	6.70	5.65	
40	E	F	6.70	7.45	7.60	7.45	7.40	6.65	
41	G	M	6.60	7.90	8.20	7.60	7.50	6.50	
42	H	F	6.30	7.10	7.20	7.10	6.95	6.50	
+∇43	G	F	6.65	6.80	6.60	7.15	7.00	6.65	
44	I	M	absent	7.35	7.45	7.60	7.30	absent	
45	F	M	6.80	8.10	8.20	8.45	8.25	6.75	
46	M	F	6.00	6.40	6.65	6.55	6.45	5.95	
47	B	M	6.25	6.85	7.00	7.05	6.90	6.40	
48	S	F	6.60	6.95	7.10	7.50	7.25	6.75	
49	H	F	6.20	6.25	6.40	6.50	6.30	6.05	
50	R	F	8.70	9.05	9.35	9.10	8.95	7.50	

TABLE 10 continued

	PATIENT	SEX	 RIGHT SIDE			 LEFT SIDE		
			42	12		21	32	
			cerv.			cerv.		
51	N	M	7.20	7.40	7.60	7.80	7.65	6.90
52	H	F	6.20	6.75	7.00	6.70	6.45	6.50
53	W	F	6.40	7.10	7.25	6.80	6.50	6.55
54	T	M	6.25	6.25	6.40	6.70	6.50	6.40
V55	W	F	5.70	6.40	6.65	6.60	6.60	5.65
56	S	F	6.45	6.40	6.85	7.25	7.05	6.45
57	T	F	6.00	6.30	6.55	6.35	6.10	5.50
058	S	F	5.30	C.M.		C.M.		5.80
59	T	M	6.60	6.70	6.75	6.95	6.90	6.40
60	W	M	6.70	6.90	7.05	7.35	7.20	6.70
61	A	F	6.15	7.35	7.45	7.25	7.15	6.25
V62	A	F	6.35	6.90	7.10	7.75	7.80	6.40
63	B	M	6.10	6.55	6.70	6.70	6.45	6.40
64	C	F	6.75	8.20	8.35	8.00	7.95	7.10
65	B	M	6.05	6.85	6.95	6.95	6.80	6.70
66	B	F	5.60	6.00	6.25	6.80	6.40	6.20
67	A	F	5.45	5.65	5.80	5.60	4.55	5.35
68	O	M	6.60	6.65	6.90	7.00	6.85	6.30
69	K	F	6.10	6.85	7.00	7.15	7.00	6.00
70	P	F	5.70	6.05	6.20	6.15	6.10	5.55
71	L	M	5.90	6.85	7.10	6.85	6.70	5.90
72	Z	M	6.20	7.00	7.05	7.50	7.35	6.15
73	G	M	6.20	7.00	7.05	7.50	7.35	6.70
74	C	M	6.00	7.55	7.80	7.75	7.60	5.95
75	L	M	6.10	7.00	7.40	7.00	6.40	6.20
V76	M	M	5.60	6.30	6.25	6.25	6.20	5.55
V77	R	F	6.50	7.80	7.90	7.90	7.85	6.20
78	M	F	6.20	6.80	6.85	6.70	6.55	6.05
79	S	F	6.35	7.30	7.35	7.40	7.30	6.25
80	S	F	6.75	7.90	8.30	8.10	8.00	6.70
V81	S	M	6.50	7.10	7.10	7.80	7.70	6.45
082	S	M	6.00	C.M.		C.M.		6.20
83	P	M	6.50	7.00	7.15	7.10	7.00	6.65
84	L	F	5.85	6.10	6.25	6.20	6.05	6.00
85	S	F	6.15	7.75	7.80	7.55	7.45	6.00
86	E	F	6.80	6.60	6.65	6.60	6.55	6.45
+V*87	F	M	6.15	5.70	5.70	5.50	5.70	6.35
+V*88	F	F	6.30	5.60	5.45	7.05	5.60	6.30
89	D	F	6.50	7.45	7.60	7.45	7.30	6.40
90	H	F	6.75	7.35	7.55	7.35	7.10	6.70
91	L	M	6.90	8.05	8.10	8.15	8.00	7.20
92	D	M	5.70	7.15	7.25	7.10	7.00	5.75
93	B	F	5.40	7.10	7.20	6.75	6.60	5.55
94	W	F	6.80	7.10	7.20	6.95	6.80	6.75
95	M	F	6.00	6.30	6.40	6.40	6.25	6.05
96	L	M	6.20	7.30	7.35	7.40	7.30	6.40
97	B	F	6.25	7.55	7.60	6.95	6.80	6.20
98	J	M	6.40	6.90	7.10	7.10	6.95	6.45
99	B	M	5.45	7.25	7.30	6.75	6.70	5.45
100	D	M	7.15	7.45	7.60	7.50	7.45	6.80

TABLE 11 THE CANINE IMPACTED GROUP. RIGHT SIDE IMPACTIONS ONLY

indicating

- i) Sample size = 65
- ii) All mesio-distal measurements of the right maxillary and mandibular lateral incisors
- iii) The incidence of right congenitally missing lateral incisors (θ) = 1
- iv) The incidence of right frankly peg-shaped lateral incisors (*) = 10
- v) The incidence of right peg-shaped lateral incisors (∇) = 38
- vi) The incidence of right small lateral incisors (+) = 19

TABLE 11

C.M. = congenitally missing

	PATIENT	SEX	IMPACTION	 RIGHT SIDE		
				42	12 cerv.	
+∇1	F	F	UR3	6.40	6.15	6.15
+∇*2	B	M	bilat	5.20	4.10	4.15
+∇*3	M	F	UR3	6.00	4.20	3.05
4	B	F	UR3	6.15	6.90	7.55
5	A	M	UR3	5.95	6.10	6.50
+∇*6	T	F	UR3	5.55	4.95	3.65
7	G	M	bilat	5.90	6.15	6.20
+∇*8	H	M	bilat	3.85	4.30	3.40
∇*9	S	F	UR3	5.65	6.10	5.75
+10	D	F	bilat	6.15	6.00	6.15
11	B	M	bilat	6.00	7.55	7.60
12	L	F	UR3	5.70	6.35	6.40
13	M	M	UR3	5.80	7.45	7.50
14	G	F	bilat	6.00	6.65	7.00
∇15	V	M	bilat	6.30	6.60	6.45
∇16	B	F	UR3	5.95	6.30	6.20
+17	S	F	UR3	6.05	5.15	5.25
∇18	F	F	bilat	5.90	6.45	6.30
∇19	J	F	UR3	5.65	6.40	6.00
20	K	F	bilat	5.90	6.70	6.85
21	I	F	bilat	6.60	8.55	8.70
22	N	M	UR3	6.40	8.30	8.55
23	H	F	UR3	6.00	6.50	7.20
24	H	M	UR3	5.45	6.30	6.45
25	G	F	UR3	6.50	6.75	6.95
+26	B	F	bilat	6.45	6.25	6.45
+∇*27	H	F	UR3	6.50	4.10	3.55
28	W	M	UR3	6.50	7.60	7.90
+29	T	F	UR3	5.45	5.15	5.55
30	S	F	UR3	6.75	6.95	7.30
∇31	K	M	UR3	6.40	7.30	7.30
32	U	F	bilat	5.60	6.95	7.00
∇33	W	M	bilat	6.00	6.95	6.90
∇34	T	M	bilat	5.90	6.65	6.70
∇35	S	F	bilat	6.10	6.60	6.20
+∇36	M	F	UR3	5.75	5.50	5.30
+∇37	M	F	UR3	6.35	6.05	6.00
38	D	M	UR3	6.15	7.00	7.10
∇39	S	M	UR3	6.75	7.60	7.60
∇40	F	F	UR3	5.65	5.95	5.90
∇41	T	F	bilat	6.10	6.80	6.70
+∇*42	M	F	bilat	6.10	5.90	5.70
+∇43	R	F	UR3	6.40	6.20	6.20
+∇*44	D	F	UR3	6.10	6.20	5.80
+∇45	S	F	bilat	6.40	6.30	6.20
+∇46	B	F	UR3	5.90	6.00	5.90
∇47	E	M	bilat	5.65	5.85	5.80
∇48	C	M	UR3	6.05	7.20	7.15

TABLE 11 continued

PATIENT	SEX	IMPACTION	 RIGHT SIDE		
			42	12	
			cerv.		
49	B	F	bilat	7.00	7.20 7.15
+V50	B	F	bilat	6.20	6.00 5.65
V*51	H	F	bilat	6.30	6.80 6.50
V52	H	F	bilat	6.00	6.80 6.80
V53	S	M	bilat	6.20	7.40 7.40
V54	M	F	bilat	5.90	6.50 6.20
V55	P	M	UR3	6.00	7.90 7.40
V56	W	M	bilat	6.20	6.80 6.80
V57	B	F	UR3	6.35	6.60 6.60
58	W	M	bilat	6.60	8.40 8.55
59	E	F	bilat	6.30	6.40 6.45
060	B	F	bilat	5.80	C.M.
V61	K	F	UR3	6.15	7.70 7.50
V62	T	M	bilat	6.30	6.80 6.80
V63	W	F	bilat	6.10	6.80 6.80
64	S	F	bilat	6.20	6.65 7.20
+V*65	S	F	bilat	6.00	5.85 5.00

TABLE 12 THE NON-IMPACTED CONTROL. RIGHT SIDE MEASUREMENTS ONLY.

Indicating

- i) Sample size = 100
- ii) All mesio-distal measurements of the right maxillary and mandibular lateral incisors
- iii) The incidence of right congenitally missing lateral incisors ($\emptyset$) = 2
- iv) The incidence of right frankly peg-shaped lateral incisors (*) = 1
- v) The incidence of right peg-shaped lateral incisors (∇) = 14
- vi) The incidence of right small lateral incisors (+) = 6

TABLE 12

C.M. = congenitally missing

	PATIENT	SEX	 RIGHT SIDE		
			42	12 cerv.	
∇ 1	M	M	6.20	7.70	7.50
∇ 2	R	M	6.15	7.20	7.00
3	P	F	5.30	6.80	6.90
4	C	M	6.65	7.70	8.20
5	M	M	6.15	7.40	7.45
6	W	M	6.25	7.30	7.45
+8	J	M	6.70	6.40	6.45
9	C	M	6.05	6.70	6.80
∇10	L	M	6.90	7.30	7.10
11	Z	M	6.05	6.40	6.80
12	C	F	6.15	7.60	7.70
13	H	M	5.40	6.00	6.10
14	H	M	5.80	6.30	6.45
15	H	F	6.10	6.35	6.45
∇16	W	M	6.35	8.00	7.90
∇17	D	F	6.20	7.10	6.70
∇18	B	M	6.55	7.40	7.30
19	P	M	5.90	6.30	6.70
20	S	M	6.85	7.60	7.65
∇21	K	M	6.05	6.60	6.50
22	S	F	5.80	7.50	7.55
∇23	K	F	5.95	6.85	6.85
24	G	F	5.50	6.00	6.25
25	E	F	5.95	6.50	6.55
+26	S	F	6.30	6.10	6.20
27	S	M	5.70	6.30	6.50
∇28	D	M	6.25	6.75	6.70
29	P	M	6.30	7.30	7.50
30	M	F	6.25	7.80	8.00
31	C	F	6.10	6.65	6.70
32	R	F	6.30	7.00	7.10
33	S	M	6.90	7.60	7.65
34	H	F	6.00	6.65	6.70
35	G	M	6.50	7.65	7.70
36	B	F	6.15	7.05	7.20
37	B	M	5.55	6.30	6.45
38	O	M	6.60	8.10	8.30
39	C	F	5.75	6.65	6.80
40	E	F	6.70	7.45	7.60
41	G	M	6.60	7.90	8.20
42	H	F	6.30	7.10	7.20
+∇43	G	F	6.65	6.80	6.60
44	I	M	absent	7.35	7.45
45	F	M	6.80	8.10	8.20
46	M	F	6.00	6.40	6.65
47	B	M	6.25	6.85	7.00
48	S	F	6.60	6.95	7.10

TABLE 12 continued

	PATIENT	SEX	 RIGHT SIDE		
			42		12
			cerv.		
49	H	F	6.20	6.25	6.40
50	R	F	8.70	9.05	9.35
51	N	M	7.20	7.40	7.60
52	H	F	6.20	6.75	7.00
53	W	F	6.40	7.10	7.25
54	T	M	6.25	6.25	6.40
55	W	F	5.70	6.40	6.65
56	S	F	6.45	6.40	6.85
57	T	F	6.00	6.30	6.55
058	S	F	5.20	C.M.	
59	T	M	6.60	6.70	6.75
60	W	M	6.70	6.90	7.05
61	A	F	6.15	7.35	7.45
62	A	F	6.35	6.90	7.10
63	B	M	6.10	6.55	6.70
64	C	F	6.75	8.20	8.35
65	B	M	6.05	6.85	6.95
66	B	F	5.60	6.00	6.25
67	A	F	5.45	5.65	5.80
68	O	M	6.60	6.65	6.90
69	K	F	6.10	6.85	7.00
70	P	F	5.70	6.05	6.20
71	L	M	5.90	6.85	7.10
72	Z	M	6.20	7.00	7.05
73	G	M	6.65	7.75	7.90
74	C	M	6.00	7.55	7.80
75	L	M	6.10	7.00	7.40
776	M	M	5.60	6.30	6.25
77	R	F	6.50	7.80	7.90
78	M	F	6.20	6.80	6.85
79	S	F	6.35	7.30	7.35
80	S	F	6.75	7.90	8.30
781	S	M	6.50	7.10	7.10
082	S	M	6.00	C,M.	
83	P	M	6.50	7.00	7.15
85	S	F	6.15	7.75	7.80
+86	E	F	6.80	6.60	6.65
787	F	M	6.15	5.70	5.70
7*88	F	F	6.30	5.60	5.45
89	D	F	6.50	7.45	7.60
90	H	F	6.75	7.35	7.55
91	L	M	6.90	8.05	8.10
92	D	M	5.70	7.15	7.25
93	B	F	5.40	7.10	7.20
94	W	F	6.80	7.10	7.20
95	M	F	6.00	6.30	6.40
96	L	M	6.20	7.30	7.35
97	B	F	6.25	7.55	7.60
98	J	M	6.40	6.90	7.10
99	B	M	5.45	7.25	7.30
100	D	M	7.15	7.45	7.60

TABLE 13 THE CANINE IMPACTED GROUP. LEFT SIDE IMPACTIONS ONLY

indicating

- i) Sample size = 69
- ii) All mesio-distal measurements of the left maxillary and
mandibular lateral incisors
- iii) The incidence of left congenitally missing
lateral incisors ($\emptyset$) = 1
- iv) The incidence of left frankly peg-shaped lateral
incisors (*) = 10
- v) The incidence of left peg-shaped lateral
incisors (∇) = 33
- vi) The incidence of left small lateral
incisors (+) = 21

TABLE 13

C.M. = congenitally missing

	PATIENT	SEX	IMPACTION		LEFT SIDE	
				21		32
					cerv.	
V1	M	F	UL3	5.95	6.05	5.25
+V*2	B	M	bilat	4.25	4.70	5.30
3	M	F	UL3	6.00	5.55	5.30
4	B	F	UL3	6.80	6.45	6.70
5	S	F	UL3	6.95	6.90	5.70
6	A	M	UL3	7.25	6.85	6.05
V7	G	M	bilat	6.41	6.50	5.65
+V*8	H	M	bilat	3.60	4.20	4.00
V9	D	F	bilat	6.40	6.65	6.30
10	S	M	UL3	6.90	6.75	6.65
V*11	T	F	UL3	5.60	5.85	5.40
+V*12	W	F	UL3	5.05	5.35	5.45
13	E	F	UL3	7.25	6.80	6.30
14	B	M	bilat	7.40	7.15	6.70
15	G	F	bilat	7.15	6.95	6.75
V16	V	M	UL3	6.75	6.75	5.80
V17	V	M	bilat	6.60	7.00	6.20
18	F	F	bilat	6.30	6.05	6.05
V19	J	M	UL3	7.25	7.30	6.85
20	K	F	bilat	6.85	7.00	absent
21	O	M	UL3	6.05	6.20	5.45
22	J	F	UL3	6.40	6.15	5.00
23	D	F	UL3	5.90	5.80	5.80
V24	S	F	UL3	6.60	6.60	5.50
25	I	F	bilat	8.10	7.90	6.50
+26	B	F	bilat	6.30	5.85	6.40
+V27	H	F	UL3	5.45	5.85	5.65
28	D	F	UL3	7.55	7.30	6.50
+V*29	J	F	UL3	3.80	4.30	5.50
30	U	F	bilat	6.80	6.60	5.55
31	W	M	bilat	7.70	7.50	5.60
+V32	D	F	UL3	5.85	6.80	6.80
33	F	F	UL3	7.25	6.65	6.15
34	T	M	bilat	7.05	6.90	5.95
35	F	F	UL3	7.40	7.00	6.60
36	W	F	UL3	7.55	7.30	6.85
37	S	F	bilat	6.70	6.55	5.90
+V38	M	F	bilat	5.50	5.65	5.80
39	C	F	UL3	6.35	6.25	5.60
+V40	S	M	UL3	5.60	5.65	5.85
41	J	F	UL3	6.10	6.00	5.60
V42	T	F	bilat	6.70	6.80	5.80
+V*43	M	F	bilat	5.55	5.70	6.15
+V44	M	F	UL3	5.55	5.60	5.60
+V45	L	F	UL3	5.70	6.00	6.00
+46	S	F	bilat	6.30	5.75	6.60
V47	S	F	UL3	6.20	6.45	5.55
+V48	L	F	UL3	5.20	5.40	5.90

TABLE 13 continued

				 LEFT SIDE		
				21		32
				cer.		
49	E	M	bilat	6.00	5.85	5.50
50	G	M	UL3	7.50	7.20	6.85
51	B	F	bilat	7.70	7.60	7.00
+∇*52	B	F	bilat	4.50	5.40	6.05
+∇*53	H	F	bilat	6.15	6.60	6.25
54	H	F	bilat	6.80	6.70	6.30
∇55	M	M	UL3	7.10	7.30	6.80
∇56	S	M	bilat	6.90	7.10	6.50
57	M	F	UL3	6.70	6.35	6.00
58	D	F	UL3	6.70	6.65	5.85
+∇59	M	F	bilat	5.50	5.50	5.90
+∇*60	W	M	bilat	4.60	5.50	6.55
+61	H	F	UL3	5.85	5.70	6.05
62	W	M	bilat	8.50	8.45	6.35
63	E	F	bilat	6.55	6.50	6.40
064	B	F	bilat	C.M.		5.85
∇65	T	M	bilat	6.90	7.30	6.35
+66	W	F	bilat	6.20	6.15	6.20
67	S	F	bilat	6.95	6.05	6.20
+∇*68	S	F	bilat	4.90	5.80	6.10
∇69	N	M	UL3	7.00	7.25	6.35

TABLE 14 THE NON-IMPACTED CONTROL GROUP. LEFT SIDE MEASUREMENT ONLY.

Indicating

- i) Sample size = 100
- ii) All mesio-distal measurements of the left maxillary and mandibular lateral incisors
- iii) The incidence of left congenitally missing lateral incisors (θ) = 2
- iv) The incidence of left frankly peg-shaped lateral incisors (*) = 1
- v) The incidence of left peg-shaped lateral incisors (∇) = 15
- vi) The incidence of left small lateral incisors (+) = 3

TABLE 14

C.M. = congenitally missing

	PATIENT	SEX	 LEFT SIDE		
			21	cerv.	32
1	M	M	7.60	7.70	6.50
2	R	M	6.90	6.80	6.30
3	P	F	7.10	6.70	5.90
∇4	C	M	8.20	8.20	6.60
5	M	M	7.50	7.30	6.10
6	M	M	7.55	7.30	6.20
7	R	M	7.30	7.20	5.90
8	J	M	6.60	6.50	6.20
9	G	M	6.80	6.70	6.00
∇10	L	M	6.70	6.70	6.00
11	Z	M	6.65	6.60	6.00
∇12	C	F	7.50	7.50	6.00
∇13	H	M	6.05	6.10	5.30
14	H	M	6.50	6.40	6.00
15	H	F	6.55	6.50	6.05
∇16	W	M	7.90	8.00	6.40
∇17	D	F	6.60	6.75	6.15
18	B	M	7.95	7.30	6.50
19	P	M	6.60	6.40	5.90
. ∇20	S	M	7.60	7.70	7.00
∇21	K	M	6.95	7.10	5.70
22	S	F	7.20	7.10	5.80
23	K	F	6.90	6.85	5.90
∇24	G	F	6.00	6.00	5.60
∇25	E	F	6.60	6.80	6.00
+∇26	S	F	6.30	6.40	6.30
27	S	M	6.45	6.30	5.55
+ 28	D	M	6.40	6.60	6.45
29	P	M	7.20	7.10	6.35
30	M	F	7.45	7.30	6.20
31	C	F	6.70	6.50	6.15
32	R	F	7.15	7.00	6.10
33	S	M	7.60	7.55	6.80
34	H	F	6.80	6.70	5.80
35	G	M	7.40	7.35	6.15
36	B	F	7.30	7.15	6.00
37	B	M	6.30	6.25	5.75
38	O	M	8.50	8.30	6.70
39	C	F	6.90	6.70	5.65
40	E	F	7.45	7.40	6.65
41	G	M	7.60	7.50	6.50
42	H	F	7.10	6.95	6.50
43	G	F	7.15	7.00	6.65
44	I	M	7.60	7.30	absent
45	F	M	8.45	8.25	6.75
46	M	F	6.55	6.45	5.95
47	B	M	7.05	6.90	6.40
48	S	F	7.50	7.25	6.75
49	H	F	6.50	6.30	6.05
50	R	F	9.10	8.95	7.50

TABLE 14 continued

	PATIENT	SEX	 LEFT SIDE		
			21	32	
			cerv.		
51	N	M	7.80	7.65	6.90
52	H	F	6.70	6.45	6.50
53	W	F	6.80	6.50	6.55
54	T	M	6.70	6.50	6.40
∇55	W	F	6.60	6.60	5.65
56	S	F	7.25	7.05	6.45
57	T	F	6.35	6.10	5.50
⊖58	S	F	C.M		5.80
59	T	M	6.95	6.90	6.40
60	W	M	7.35	7.20	6.70
61	A	F	7.25	7.15	6.25
∇62	A	F	7.75	7.80	6.40
63	B	M	6.70	6.45	6.40
64	C	F	8.00	7.95	7.10
65	B	M	6.95	6.80	6.70
66	B	F	6.80	6.40	6.20
67	A	f	5.60	4.55	5.35
68	O	M	7.00	6.85	6.30
69	K	F	7.15	7.00	6.00
70	P	F	6.15	6.10	5.55
71	L	M	6.85	6.70	5.90
72	Z	M	7.50	7.35	6.15
73	G	M	7.70	7.45	6.70
74	C	M	7.75	7.60	5.95
75	L	M	7.00	6.40	6.20
76	M	M	6.25	6.20	5.55
77	R	F	7.90	7.85	6.20
78	M	F	6.70	6.55	6.05
79	S	F	7.40	7.30	6.25
80	S	F	8.10	8.00	6.70
81	S	M	7.80	7.70	6.45
∇82	S	M	C.M,		6.20
83	P	M	7.10	7.00	6.65
85	S	F	7.55	7.45	6.00
86	E	F	6.60	6.55	6.45
+∇*87	F	M	5.50	5.70	6.35
88	F	F	7.05	5.60	6.30
89	D	F	7.45	7.30	6.40
90	H	F	7.35	7.10	6.70
91	L	M	8.15	8.00	7.20
92	D	M	7.10	7.00	5.75
93	B	F	6.75	6.60	5.55
94	W	F	6.95	6.80	6.75
95	M	F	6.40	6.25	6.05
96	L	M	7.40	7.30	6.40
97	B	F	6.95	6.80	6.20
98	J	M	7.10	6.95	6.45
99	B	M	6.75	6.70	5.45
100	D	M	7.50	7.45	6.80

TABLE 15 INCIDENCE OF LEFT AND RIGHT LATERAL INCISOR ANOMALIES
IN THE CANINE IMPACTED AND NON-IMPACTED CONTROL GROUPS

	Canine Impacted			Non-impacted		
	R	L	Total	R	L	Total
Sample size	65	69	134	100	100	200
Congenitally missing (Θ)	1	1	2 1.49%	2	2	4 2.00%
Frankly peg-shaped (*)	10	10	20 14.92%	1	1	2 1.00%
Peg-shaped (∇)	38	33	71 52.96%	14	14	29 14.50%
Small (+)	19	21	40 29.85%	6	3	9 4.50%

APPENDIX 3

Calculation of 't' values for determination of the significance of the percentage difference found between the impacted group and the non-impacted control group.

1. INCIDENCE OF RIGHT OR LEFT LATERAL INCISOR ANOMALIES1.A. CONGENITALLY MISSING LATERALS

Impacted group = 4%

Control group = 2%

SE % Impacted group

SE % Control group

$$\begin{aligned}
 p &= 4 \\
 q &= 96 \\
 N &= 100
 \end{aligned}
 \quad = \sqrt{\frac{pq}{N}} = 3.84$$

$$\begin{aligned}
 p &= 2 \\
 q &= 98 \\
 N &= 100
 \end{aligned}
 \quad = \sqrt{\frac{pq}{N}} = 1.96$$

Standard error of
the difference

$$\begin{aligned}
 &= \sqrt{(SE \% Imp.)^2 + (SE \% Control)^2} \\
 &= \sqrt{(3.84)^2 + (1.96)^2} \\
 &= \sqrt{5.24} \\
 &= 2.29
 \end{aligned}$$

't' test = $\frac{\text{difference}}{\text{SE of diff}}$

$$= \frac{4 - 2}{2.29}$$

$$'t' = 1.15$$

1.B. FRANKLY PEG-SHAPED LATERALS

Impacted group = 16%

SE % Impacted group

$$\begin{aligned}
 p &= 16 \\
 q &= 84 \\
 N &= 100
 \end{aligned}
 \quad
 \begin{aligned}
 &= \sqrt{\frac{pq}{N}} \\
 &= 13.44
 \end{aligned}$$

Control group = 2%

SE % Control group

$$\begin{aligned}
 &= \sqrt{\frac{pq}{N}} \\
 &= 1.96
 \end{aligned}
 \quad
 \begin{aligned}
 p &= 2 \\
 q &= 98 \\
 N &= 100
 \end{aligned}$$

SE of difference

$$\begin{aligned}
 &= \sqrt{(SE\% \text{ Imp})^2 + (SE\% \text{ Control})^2} \\
 &= \sqrt{(13.44)^2 + (1.96)^2} \\
 &= \sqrt{13.44 + 1.96} \\
 &= 3.92
 \end{aligned}$$

't' test = $\frac{\text{difference}}{\text{SE of diff}}$

$$\begin{aligned}
 &= \frac{16 - 2}{3.92} \\
 't' &= 3.57
 \end{aligned}$$

1.C. PEG-SHAPED LATERALS

Impacted group = 68%

SE % Impacted group

$$\begin{aligned}
 p &= 68 \\
 q &= 32 \\
 N &= 100
 \end{aligned}
 \quad
 \begin{aligned}
 &= \sqrt{\frac{pq}{N}} \\
 &= 21.76
 \end{aligned}$$

Control group = 24%

SE % Control group

$$\begin{aligned}
 &= \sqrt{\frac{pq}{N}} \\
 &= 18.24
 \end{aligned}
 \quad
 \begin{aligned}
 p &= 24 \\
 q &= 76 \\
 N &= 100
 \end{aligned}$$

SE of difference

$$\begin{aligned}
 &= \sqrt{(SE\% \text{ Imp group})^2 + (SE\% \text{ Control group})^2} \\
 &= \sqrt{(21.76)^2 + (18.24)^2} \\
 &= 4.66
 \end{aligned}$$

't' test = $\frac{\text{difference}}{\text{SE of difference}}$

$$\begin{aligned}
 &= \frac{68 - 24}{4.66} \\
 't' &= 9.44
 \end{aligned}$$

1.D. SMALL LATERALS

Impacted group = 34%

SE % Impacted group

$$\begin{aligned}
 p &= 34 \\
 q &= 66 \\
 N &= 100
 \end{aligned}
 = \sqrt{\frac{pq}{N}}
 = 22.44$$

Control group = 6%

SE % Control group

$$\begin{aligned}
 &= \sqrt{\frac{pq}{N}} \\
 &= 5.64
 \end{aligned}
 \begin{aligned}
 p &= 6 \\
 q &= 94 \\
 N &= 100
 \end{aligned}$$

SE of difference

$$\begin{aligned}
 &= \sqrt{(SE\% \text{ Imp. group})^2 + (SE\% \text{ Control group})^2} \\
 &= \sqrt{(22.44)^2 + (5.64)^2} \\
 &= \sqrt{28.08} \\
 &= 5.30
 \end{aligned}$$

$$'t' \text{ test} = \frac{\text{difference}}{\text{SE of difference}}$$

$$= \frac{34 - 6}{5.30}$$

$$'t' = 5.28$$

2. INCIDENCE OF THE LATERAL INCISOR ANOMALY OCCURRING ON THE SAME SIDE AS THE CANINE IMPACTION

2.A. CONGENITALLY MISSING LATERALS

Impacted group = 1.49%

Control group = 4%

SE % Impacted group

SE % Control group

$$\begin{aligned} p &= 1.49 \\ q &= 98.51 \\ N &= 134 \end{aligned} \quad = \quad \sqrt{\frac{pq}{N}} = 1.10$$

$$\begin{aligned} &= \sqrt{\frac{pq}{N}} \quad p = 4 \\ & \quad q = 96 \\ &= 1.92 \quad N = 200 \end{aligned}$$

SE of difference

$$\begin{aligned} &= \sqrt{(1.10)^2 + (1.92)^2} \\ &= 1.74 \end{aligned}$$

't' test = $\frac{\text{difference}}{\text{SE of difference}}$

$$= \frac{4 - 1.49}{1.74}$$

$$'t' = 1.44$$

B. FRANKLY PEG-SHAPED LATERALS

Impacted group = 14.92%

Control group = 2%

SE % Impacted group

SE % Control group

$$\begin{aligned} p &= 14.92 \\ q &= 85.08 \\ N &= 134 \end{aligned} \quad = \quad \sqrt{\frac{pq}{N}} = 9.47$$

$$\begin{aligned} &= \sqrt{\frac{pq}{N}} \quad p = 4 \\ & \quad q = 98 \\ &= 0.98 \quad N = 200 \end{aligned}$$

SE of difference

$$\begin{aligned} &= \sqrt{(9.47)^2 + (0.98)^2} \\ &= 10.45 \\ &= 3.23 \end{aligned}$$

't' test = $\frac{\text{difference}}{\text{SE of difference}}$

$$= \frac{14.92 - 2.00}{3.23}$$

$$'t' = 4.00$$

C. PEG-SHAPED LATERALS

Impacted group = 52.96%

SE % Impacted group

$$\begin{aligned}
 p &= 52.96 \\
 q &= 47.04 \\
 N &= 134
 \end{aligned}
 = \sqrt{\frac{pq}{N}}
 = 18.59$$

Control group = 14.50%

SE % Control group

$$\begin{aligned}
 &= \sqrt{\frac{pq}{N}} & p &= 14.50 \\
 & & q &= 85.50 \\
 &= 6.20 & N &= 200
 \end{aligned}$$

SE of difference

$$\begin{aligned}
 &= \sqrt{(18.59)^2 + (6.20)^2} \\
 &= \sqrt{24.79} \\
 &= 4.98
 \end{aligned}$$

$$'t' \text{ test} = \frac{\text{difference}}{\text{SE of difference}}$$

$$= \frac{52.96 - 14.50}{4.98}$$

$$'t' = 7.72$$

D. SMALL LATERALS

Impacted group = 29.85%

SE % Impacted group

$$\begin{aligned}
 p &= 29.85 \\
 q &= 70.15 \\
 N &= 134
 \end{aligned}
 = \sqrt{\frac{pq}{N}}
 = 15.63$$

Control group = 14.50%

SE % Control group

$$\begin{aligned}
 &= \sqrt{\frac{pq}{N}} & p &= 4.50 \\
 & & q &= 95.50 \\
 &= 2.15 & N &= 200
 \end{aligned}$$

SE of difference

$$\begin{aligned}
 &= \sqrt{(15.62)^2 + (2.15)^2} \\
 &= \sqrt{17.77} \\
 &= 4.22
 \end{aligned}$$

$$'t' \text{ test} = \frac{\text{difference}}{\text{SE of difference}}$$

$$= \frac{29.10 - 4.50}{4.22}$$

$$'t' = 5.83$$

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