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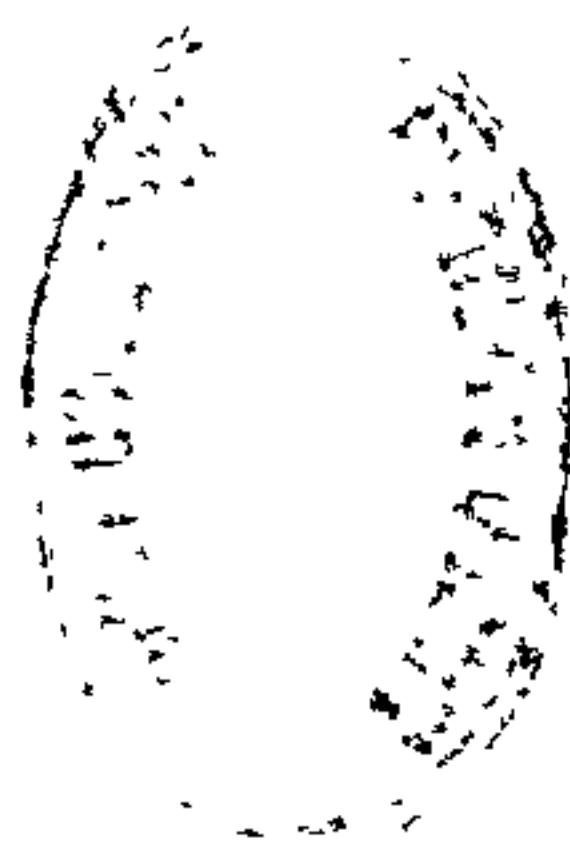
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DENTAL ANOMALIES OF FORM AND STRUCTURE

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A thesis embodying an original research programme
submitted by the undersigned as partial fulfilment
of the requirements for admission to the degree of
Master of Dental Science
within the University of Sydney

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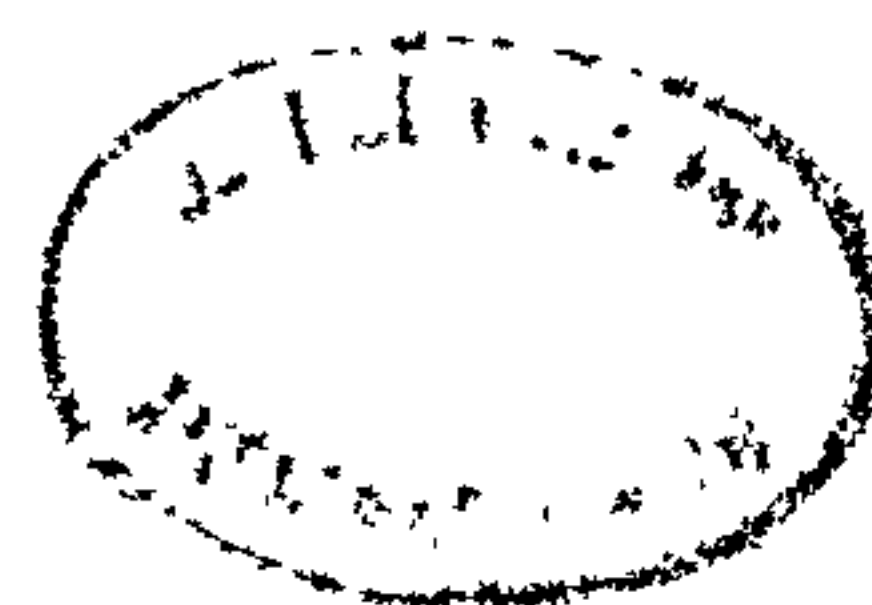
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INTRODUCTION



The correct assessment of anomalies of form and structure, as well as those of eruption and position, is a necessary prerequisite to the establishment of an orthodontic diagnosis and treatment plan. In many instances the early observation of these anomalies may prevent the development of or, at the very least, the further deterioration of, a malocclusion.

Many anomalies are clinically observable, but there are those that may only be discovered by a complete radiographic examination. Accordingly, the purpose of this investigation was to determine the incidence of the anomalies of form and eruption occurring in the permanent dentition by means of a radiographic examination, of a group of children presenting for treatment at the United Dental Hospital, Sydney.

Due to the restricted nature of this sample and the method by which it was obtained, the results of this survey may not correspond to those of similar age groups found in general practice. However, the project was thought to be valuable in directing attention to such phenomena, and as a pilot study for a larger scale survey. Also it was designed to facilitate comparison with other anomalous conditions and syndromes, such as cleft lip and cleft palate conditions, which include dental irregularities.

PART I

Growth and Development of the Deciduous and Permanent Teeth

Before any discussion of the abnormally formed or ectopically positioned tooth can be developed it is essential that normal development be thoroughly identified.

Schour and Massler (1940) have described the tooth as "a living functional organ passing through the following developmental stages in harmonious and sequential manner ..."

1. Growth
 - a. Initiation of development
 - b. Proliferation
 - c. Histodifferentiation
 - d. Apposition
2. Calcification
3. Eruption
4. Attrition

The division into these stages is a matter of convenience as the development is a continuous process with no lines of demarcation, each stage merging imperceptibly with the subsequent one.

Growth

In the early stages of embryological development the ectodermal lining of the primitive oral cavity consists of a basal layer of columnar cells with a superior layer of

squamoid cells. This basal layer is separated from the underlying mesoderm by a basement membrane (Sicher, 1962 p.31). Both these tissues, the ectoderm and the mesoderm, are involved in the development of the dental structures. The ectoderm forms the enamel organ, the changing shape of which is used to identify various stages of development (bud stage, etc.); the mesoderm forms the dentine, cementum, pulp and periodontal membrane.

a. Initiation - Bud stage

The first sign of the activity leading to the development of the teeth is observed in the six weeks old embryo, 11 cm. (Sicher, 1962 p.31). This occurs as increased mitotic activity, in the proliferation of the basal ectodermal layer.

The increased rate of cell division causes the ectodermal epithelium to invaginate into the underlying mesoderm. This germinal epithelial band, which extends along the entire free borders of both the developing jaws, is termed the Dental Lamina. At first it is located superficially, however with further development it becomes more deeply embedded in the mesoderm. During this period of increased activity, mitotic figures are seen, not only in the epithelium but also in the subjacent mesoderm (Sicher, 1962 p.35).

A second germinal epithelial band is formed in close proximity to the outer aspect of the dental lamina in about the seventh

week. Called the Vestibular Lamina, this is the primordium of the oral vestibule. Part of the inner epithelial band and the underlying mesoderm of this lamina will form the gingival mucosa and the labial surface of the alveolar ridge (Schour, 1960).

Soon after the formation of the dental lamina further increased mitotic activity, evidenced by knob-like invaginations from the lamina, is observed in ten areas of both the upper and lower arches. In each arch these sites of proliferation are the primordial structures which form the deciduous teeth. The first buds to form are those for the mandibular anterior teeth - end of the seventh week. By seven-and-a-half weeks the maxillary anterior teeth are observed, and during the eighth week the buds for the deciduous molars have appeared (Provenza, 1964 p.114). These tooth buds are attached laterally to the dental lamina. However, as growth of the bud continues it tends to pull away bringing some of the parent lamina with it.

Continued proliferation and migratory activity occur at the posterior ends of the horse-shoe shaped lamina. This elongation is such that it keeps pace with the lengthening of the maxilla and mandible allowing for the formation of the permanent molar teeth. These arise directly from the lamina in a similar manner to those of the deciduous teeth.

All other permanent teeth - incisors, canines and premolars -

develop from successional lamina lingual to the dental organ of their deciduous predecessors (Logan and Kronfeld, 1933).

By the fourth month the posterior ends of the lamina have given rise to the germs of the first permanent molars. The primordia of the second permanent molars do not occur until nine months after birth and those for the third permanent molar not until about four years of age (Provenza, 1964 p.116). From this it is seen that the dental lamina is active in various parts of the jaws from the young embryo (six weeks) through until the age of four to five years.

The cells which constitute the tooth bud are cytologically similar, but the rate of cell division is not uniform. There is greater activity in the peripheral area of what is to become the base of the tooth buds. As a result of this, with concomitant activity in the subjacent mesoderm, the inferior surface of the bud becomes concave. The embryo is now in about the eighth week of development.

Disturbances of initiation are not common, owing to the young gestation age of the individual and the very basic nature of the initiation process. A lack of initiation results in the absence of teeth. This may occur in isolated areas and involve single teeth, partial anodontia (clinical aspects p. 56), or there may be a complete lack of teeth, total anodontia (clinical aspects p. 49). In total anodontia

of both dentitions there is complete aplasia of the dental lamina. This is usually associated with signs of generalised ectodermal dysplasia (Eisenson, 1956).

Hyperproductivity is responsible for single or multiple supernumary teeth (clinical aspects p. 66). One theory advanced to explain the presence of additional teeth attributes them to excessive growth of the dental lamina which results in an additional tooth germ being given off (Saaremaa 1951).

Excessive numbers of initiation - with cells unco-ordinated and crowded into one area - resemble the complex composite odontome formation (Rushton, 1937) (clinical aspects p. 90).

Teeth and tooth-like structures are capable of being initiated outside the normal dental areas. These occur in dermoid cysts and are found in such areas of the trunk, brain, face, tongue and scalp (Broomall, 1905).

b. Proliferation - Cap stage

This constitutes the most rapid growth of the tooth bud and is characterised by marked alteration of form (Schour and Massler, 1940).

The result of the increased proliferation and the above-mentioned disproportionate growth is a further deepening of the indentation on the inferior surface. The cells of the

tooth germ now show a morphological variation (Hamilton, Boyd and Mossman, 1964). The cell pattern forming the peripheral layer of the crown of the cap, the outer enamel epithelium, is a single layer of cuboidal cells, while those forming the inferior concave surface, inner enamel epithelium, are columnar.

The cells between these two layers, which consist of the bulk of the tooth germ, possess numerous branching and intercommunicating cytoplasmic processes. These begin to separate by an increase in intercellular fluid and they arrange themselves in a network called the stellate reticulum.

The spaces in this reticular network are filled with a mucoid fluid giving it a cushion-like consistency that later supports and protects the delicate enamel forming cells.

During this stage accessory features are formed that are of a temporary nature only. In the centre of the dental organ the cells are densely packed and form an enamel knot. This knot projects partly into the underlying mesoderm, dental papilla, so the epithelial invagination shows a small knob-like area bordered by the labial and lingual grooves (Sicher, 1962 p.39). Provenza (1964 p.116) states that this is due to the epithelial cells in the centre of the concave base being more mitotically active than those adjacent to them.

At the same time there arises a vertical extension from this knot into the lengthening dental organ. This is the enamel cord. Both these structures have disappeared before enamel formation begins.

Under the organising influence of the proliferating epithelium of the dental organ, the mesoderm, partly enclosed by the invagination of the dental organ, proliferates and condenses to form the dental papilla. This occurs side by side with the development of the epithelial portion of the dental organ and should not be considered as a passive crowding by this proliferating epithelium, as it shows active budding of capillaries and mitotic figures (Sicher, 1962 p.39). Concomitantly there is a marginal condensation surrounding the enamel organ and dental papilla. Here the primitive dental sac develops as a denser and more fibrous layer.

The tooth germ now consists of three formative organs.

1. Enamel Organ - derived from epithelium, it forms the enamel
2. Dental Papilla - derived from mesoderm, it forms the dentine and the pulp
3. Dental Sac - derived from mesoderm, it forms the cementum and supporting structures.

Glasstone (1936) has shown that this structure contains the entire growth potential for the future tooth by reason that explants continue to develop in tissue cultures through the

subsequent stages of histodifferentiation and appositional growth.

Disturbances occurring during this stage may lead to the differentiation of additional structures (Schour and Massler 1940). With increased or excessive proliferation these structures may vary from the development of additional cusps to the formation of supernumary teeth. The reverse can also occur. The suppression of proliferation results in the loss of cusps and tooth structures and in extreme cases the absence of the tooth.

McCall (1944) states that

"According to the accepted theories of the embryology of the succedaneous teeth, the permanent tooth germ is given off from the deciduous tooth germ early in the development of the latter, and in accordance with this theory it would be impossible for permanent teeth other than permanent molars, to develop if the corresponding deciduous teeth were congenitally absent."

Epithelial rests may be formed during the proliferative state by the separation of cells from the developing dental organ. These may remain as such or become activated under the stimulus of an irritation. The final result of this separation depends on the degree of differentiation which occurs (Schour and Massler, 1940). If these epithelial

rests remain undifferentiated, then once stimulated the cells will merely proliferate. But should they become partially differentiated or have been separated in the partly differentiated state, then they would assume a secretory function and a cyst would develop. If, however, the cells become fully differentiated or are separated in that state, then the enamel and dentinal matrix would be deposited resulting in the formation of an odontome (Tratman, 1949) (Clinical aspects p. 90).

c. Histodifferentiation - Bell stage

Characterised by its bell shape, formed as the invagination of the epithelium deepens and the marginal growth continues, this stage has two distinct phases (Schour and Massler, 1940).

1. Histodifferentiation
2. Morphodifferentiation

1. Histodifferentiation

The formative cells of the tooth germ undergo histological and chemical changes and acquire their appositional growth potential. With the organising influence of the inner enamel epithelium the peripheral cells of the subjacent mesoderm undergo histodifferentiation, they assume a tall columnar form, odontoblasts, and acquire their specific ability to take part in the formation of dentine (Manly, 1954).

The differentiation of the inner enamel epithelium to ameloblasts does not occur until the odontoblasts have matured and deposited the initial quantity of predentine (Hahn, 1941). The cells of the inner enamel epithelium now lose their capacity to divide and gain their specific ability to secrete the enamel matrix (Marsland, 1951).

It has been demonstrated that the presence of the inner enamel epithelium is essential for the differentiation of the odontoblasts and for the initiation of dentine formation. However, once their functional potential is acquired they can proceed without the presence of this epithelium.

When this dentine formation begins a reversal occurs for, in the absence of dentine, the enamel will not form (Hahn, 1941). Dentine formation therefore precedes and is essential to enamel formation, although the differentiation and chemotactic influence of the epithelial cells precede and are essential to the differentiation of the dentine forming cells.

2. Morphodifferentiation

Concomitantly with histodifferentiation the cells arrange themselves along the site which outlines the basic form and relative size of the future tooth. This outline becomes the Dentino-enamel and Cemento-enamel junctions. Once these commence, the future size and form of the tooth is established (Beust 1928; Hahn 1941).

During this period in which the morphologic specialisation of the developing germ has occurred, the remaining layers of the enamel organ have also undergone differentiation.

The formerly smooth surface of the outer enamel epithelium (columnar cells) is now laid in folds with the cells becoming irregular and flattened. The adjacent mesoderm of the dental sac forms papillae which contain capillary loops, in between these folds (Jump, 1938 p.505). This provides for a rich nutritional supply for the intense metabolic activity of the avascular enamel organ.

Several layers of squamous cells, stratum intermedium, appear between the stellate reticulum and the inner enamel epithelium. These are absent in the areas that outline the roots (Johnson and Bevelander, 1957), and they appear to be essential for enamel formation (Marsland, 1951).

The stellate reticulum still continues to expand by an increase in the intercellular fluid. This fluid is lost and the reticulum shrinks just before enamel formation begins (Sicher, 1962 p.40).

The basal-most area of the enamel organ, which forms a narrowed rim, is termed the cervical loop. This double-layered structure, consisting exclusively of the inner and outer enamel epithelial layers, continues on to become Hertwig's Epithelial Sheath, once the future cemento-enamel junction has been attained (Diamond and Applebaum, 1943). Hertwig's

sheath is responsible for determining the number, size and shape of the roots of the tooth.

Prior to this, the dental lamina maintains a broad connection with the dental organ but in this stage of development it begins to break up by mesodermal invasion (Sicher, 1962 p.44). This invasion which forms the enamel niche first occurs in the central portion and divides the dental lamina into

- (a) Dental lamina proper
- (b) Lateral dental lamina

With continual growth of the enamel organ, further separation from the dental lamina is effected. The epithelial bud for the permanent successor is being formed at the free terminal end of the dental lamina proper, now referred to as the Successional Lamina. As this proliferates, the permanent bud retains its position lingual to that of the deciduous predecessor (Logan and Kronfeld, 1933).

In the advanced bell stage, following the formation of the successional lamina and the permanent tooth bud, the dental lamina loses its continuity and undergoes retrogressive changes. Vestigial remnants of the lamina may be found as epithelial aggregates in the surrounding mesoderm (Manly 1954).

Already stated is the fact that the developmental stages merge and overlap considerably. Therefore, in any tooth

germ, many of the discussed stages may occur at any one time, but at different levels.

The cells of the inner enamel epithelium show this as they demonstrate different degrees of differentiation. The cells located at what will be the crest of the cusp or the incisal edge are the first to differentiate. With departure from the crest the cells become less and less specialised so that those of the cervical loop are the least differentiated. For example, in the mandibular deciduous central incisor, at about five months in utero, apposition and calcification occur at the incisal edge - growth centre, histodifferentiation and morphodifferentiation are progressing in the lower portion of the crown, and proliferation with mitotic activity is found at the future gingival level. After the crown is completed proliferation still proceeds actively in Hertwig's Sheath preparatory to its morphodifferentiative function (Schour, 1948).

Failure of these cells to undergo histodifferentiative changes results in their unhampered proliferation with the formation of a tumor. This mass is characterised by the unorganised proliferation of the cells and by their undifferentiated nature. The degree of non-differentiation of the cells being an index to the malignancy of the tumor.

The detachment of differentiated and partly-differentiated cells and the formation of cysts and odontomes, which can

occur at this time has been discussed in the previous phase.

The ameloblastoma, which is an epithelial tumor of doubtful origin, could begin at this stage from residue cells of the dental lamina (Manly, 1954).

During histodifferentiation disturbances to the development may cause irregularities in size and shape either with or without variation in the function of the specialised cells. In Microdontia and Macrodontia (clinical aspects p. 77), the size of the teeth has been altered without impairment of the formative function. The form and size of the teeth can only be affected if the disturbance occurs at the bell stage of development when the morphogenic pattern is established. If disturbances occur after this time then only those cells functioning at that time will be affected.

The Dens-in-dente or Gestant Odontome (clinical aspects p. 99) is a developmental variation which commences about the time dentine formation begins (Rushton, 1936). This occurs when some cells near the centre of the deep surface of the enamel take on abnormal growth, proliferate in a deep direction at a great rate and by their bulk invaginate the papilla. Another suggestion put forward states that rather than a proliferation of the internal epithelium, a local arrest of development occurs, the surrounding, more actively growing cells then engulf the laggard tissue with a similar resulting invagination (Kronfeld, 1934).

A second variation closely aligned to and often classified with dens-in-dente is the invagination from the lingual pit of the maxillary central and lateral incisors (Hallett, 1953). The invagination is an extension of this pit - foramen caecum - into an abnormal cleft or cavity. This is thought to be formed in a similar manner to dens-in-dente. Differentiation should be made between these two forms as with invagination the cleft starts at the base of the crown but in dens-in-dente the channel, which is enamel-lined, traverses the length of the tooth and extends from the incisal margin (Hallett, 1953; Worth, 1963 p.83).

Fusion and gemination (clinical aspects p. 83) are two developmental disturbances which may be difficult to differentiate. In gemination there is an epithelial invagination into the enamel organ which may partially or completely divide it. When this is partial it may cause varying degrees of separation from a partially bifid crown with a single root canal to a completely bifid crown with the division extending into the root canal.

Fusion is the process in which union occurs between two tooth germs during tooth formation. One suggestion is that this is due to a persistence of the inter-dental lamina (Hitchin and Morris, 1961). Greth (1936) states that it is produced by some physical action, perhaps special pressure, forcing the tooth germs into contact. Fusion may occur between two

normal tooth germs in which case there is a missing tooth, or it may be due to fusion of a tooth germ of a normal series with a supernumary tooth germ. The site of union depends on the stage of tooth formation which has been reached when the two tooth germs come into contact. If this occurs in the very early stages, there will be complete fusion, or fusion of the crowns only and the roots separated. If the crowns are already formed then only the roots will be fused.

Specific disturbances whose end-results constitute disturbance in apposition and calcification have their origin as disturbances during histodifferentiation. These are amelogenesis imperfecta and dentinogenesis imperfecta (Roberts and Schour, 1939). In amelogenesis imperfecta (clinical aspects p. 106) the ameloblasts suffer from a genetic disturbance which leads to a foreshortening of their appositional functional life span (hereditary enamel hypoplasia), or to a defective capacity to assume their normal role in calcification (hereditary enamel hypocalcification) (Weinmann, Svoboda and Woods, 1945). In the former, fairly normal enamel structure is deposited in a subnormal amount, whilst in the latter, enamel of normal thickness is formed but it remains immature and soft. These two disturbances of enamel can be readily correlated with the two phases in normal enamel development, enamel matrix formation and enamel maturation (Weinmann et al. 1945).

Dentinogenesis imperfecta (clinical aspects p.130) is a hereditary disturbance in the histodifferentiation of the odontoblasts. These cells are unable to effect the regular arrangement of the dentinal tubules. Also their functional life span is disturbed so that they continue to form an imperfect dentinal matrix, thereby obliterating the pulp. Roberts and Schour (1939) have shown that this obliteration of the pulp is a posteruptive phenomenon.

Rushton (1954) described a different form of dentinal dysplasia in which there were enormous pulp chambers. This anomaly, 'shell teeth', was due to the restriction of the dentine formation to a thin layer next to the enamel and cementum.

Hertwig's Epithelial Sheath and Root Formation

Root Formation commences once the enamel and dentine formation have reached the future cemento-enamel junction.

It has been shown that Hertwig's Epithelial Sheath is a continuation of the inner and outer epithelial layers of the enamel organ, but also that this does not become manifested until the formation of the enamel matrix of the crown is appreciably advanced (Diamond and Applebaum, 1943).

This epithelial sheath determines the number, size and shape of the root(s) to be formed.

At the level of the cemento-enamel junction, prior to the commencement of the root formation, the inner and outer enamel epithelial layers turn inwardly to a horizontal plane forming the Epithelial Diaphragm (Sicher, 1932).

The diaphragm narrows the cervical opening of the dental organ.

During the development of the tooth the epithelial diaphragm marks a relatively fixed point in the growing alveolus. The growth of the tooth taking place occlusally from this point (Orban and Mueller, 1927).

The inner dental epithelial layer induces the organisation of the adjacent undifferentiated cells of the dental papilla to odontoblasts. These newly maturing cells are continuous with the older functioning odontoblasts which have been depositing the coronal dentinal matrix (Manly, 1954).

As the dentine is deposited, the tooth becomes elongated and moves towards the oral mucosa. With this gross movement the region of the epithelial sheath adjacent to the developing radicular dentine loses its close apposition to the root surface and begins to disintegrate. This allows the adjacent connective tissue cells (mesenchyme) to come into contact with the newly formed dentine, where they undergo rapid differentiation into cementoblasts. Remnants

of Hertwig's Epithelial Sheath persist in the periodontium as the Epithelial Rests of Malassez (Manly, 1954).

In multicrootod teeth expansion of the cervical opening occurs in such a way that long tongue-like extensions of the horizontal diaphragm develop (Orban and Mueller, 1927). Two such extensions are found in double-rooted teeth. In three rooted teeth, tri-lateral proliferation occurs. These projections fuse and so divide the cervical opening. Dentine then forms on the pulpal surface of these dividing bridges.

In the final stages of root development the proliferation of the epithelium in the diaphragm lags behind that of the pulpal connective tissue. This reduces the width of the wide apical foramen - first to the width of the diaphragmatic opening itself and later further narrowing by apposition of dentine and cementum.

Disturbances to the correction function of the epithelial sheath of Hertwig, during root formation, can result in the formation of accessory root canals and enamel pearls (Sicher, 1962 p.47). Occasionally, complete separation of the epithelial sheath from the dentine is not effected. These epithelial cells may differentiate into fully functioning ameloblasts and elaborate enamel deposits, called enamel pearls. These are sometimes in the region of the furcation

of the permanent molars but may occur at any level on the tooth.

On the other hand, if the continuity of Hertwig's Sheath is broken, prior to enamel formation, or if in the case of multi-rooted teeth, the fusion of the horizontal extensions of the diaphragm is not completed, a defect in the dentinal wall of the pulp ensues. Such defects are due to the non-differentiation of the mesodermal cells into odontoblasts in the absence of the epithelial layer - consequently cementum and dentine formation is absent and accessory canals result.

Dilaceration (clinical aspects p.87) is a deformity of the root in which it is unusually curved or bent (Worth, 1963 p.433). It is thought to arise from an injury during development - usually a blow or a fall that severely jars the deciduous teeth and is transmitted to the underlying tooth germ. It may also occur after early surgical intervention in cases of cleft lip. Dilaceration can occur in the absence of external injury, due to an obstacle in the normal direction of growth (Atkinson, 1966). It is most often seen in the region of the maxillary incisors, but may occur anywhere.

Apposition

This stage does not begin until the cells have attained full histodifferentiation and have reached their particular position along the future dentino-enamel junction.

The first appearance of the dentine seems to be a critical phase in the life cycle of the inner enamel epithelium.

As long as the epithelium is in contact with the connective tissue of the dental papilla, it receives nutrients from the blood vessels of the papilla, but when dentine forms it cuts off these cells from this source (Marsland, 1951). From thence they are supplied by the capillaries that surround, and may even penetrate, the outer enamel epithelium. This is characterised by the proliferation of the capillaries of the dental sac and the reduction and gradual disappearance of the stellate reticulum (Jump, 1938 p.515).

Amelogenesis

The first function of the ameloblasts is to contribute to the formation of the dentino-enamel junction (Saunders, Nuckolls and Frisbie, 1942), this occurs about when the first layer of dentine matrix begins to calcify and is carried out by the deposition of a layer of matrix deposited extra-cellularly in a thin line on the dentine.

Termed the dentino-enamel membrane, it is continuous with the subsequently formed inter-prismatic substance and accounts for the fact that the distal ends of the enamel rods are not in direct contact with the dentine (Orban, Sicher and Weinmann, 1943).

Microscopic examination of the dentino-enamel junction will show that the surface contiguous to the base of the enamel rods is thrown into numerous folds, thereby presenting an undulated appearance. While this surface is scalloped and calcified in the fully developed crown, such was not the case during earlier stages of development. This condition of the mature dentino-enamel junction probably serves to establish a firmer connection between the enamel and dentine (Provenza, 1964 p.223).

The cyclic organisation of the matrix for the enamel prisms and the interprismatic substance involves two steps.

1. Development on the Tomes' processes and Terminal Bar Apparatus.
2. Transformation of Tomes' Processes.

1. Development of Tomes' Processes and Terminal Bar Apparatus

Once the dentino-enamel membrane is formed, the development of the Tomes' Processes takes place at the distal end of the differentiating ameloblasts. The Tomes' Process is a granulated protoplasmic projection of the cytoplasm of the ameloblast (Orban, Sicher and Weinmann, 1943). The boundaries between the ameloblasts are marked by condensations of the intercellular substance (Marsland, 1951) - this completely surrounds the ends of the cells delineating the Tomes' Processes. In section this matrix appears as a row of projections about 4 microns long extending intracellularly from the dentino-enamel membrane.

The distal ends of the ameloblasts are separated from the Tomes' Processes by the terminal bar apparatus. These are localised condensations of the cytoplasmic substance closely associated with the cell membranes (Reith, 1960).

Both the Tomes' Process and the terminal bar develop with the formation of the enamel matrix and disappear once the enamel matrix is fully formed (Orban et al., 1943).

2. Transformation of the Tomes' Process

This consists of the gradual completion of the distal ends of the Tomes' Process with matrix material to form segments of the enamel rods (Orban et al., 1943). The transformation into matrix material, which is secreted by the ameloblasts, takes place from the periphery inwards. Recent investigations show that the Tomes' Process may be cut off from the parent cell by an infolding of the lateral cell membrane (Nylen and Scott, 1960). As one row of processes is transformed, new processes are outlined basal to the preceding ones. These steps are repeated again and again until the entire thickness of enamel is formed (Orban et al., 1943).

The primary segmentation of the rods, that result from this rhythmic deposition is the basis for the cross striations - incremental lines of Retzius, seen in the mature rods. The lengths of these developing segments, and the distance between these striations is about 4 microns.

The ameloblasts, which are at right-angles to the dentinal surface, are generally at an angle to the developing rod segments (Orban et al., 1943). However, these rods are rarely, if ever, straight. They follow a wavy course from the dentine to the enamel surface. This more or less regular change in direction may be regarded as a functional adaptation minimising the risk of cleavage in the axial direction under the influence of occlusal masticatory stresses. The change in direction of the rods is responsible for the appearance of the Hunter-Schrieger bands - alternate dark and light bands best seen in longitudinal sections under oblique reflected light. They are usually wide enough to be seen with the naked eye (Hollander, Bodecker, Applebaum and Saper, 1935). Each ameloblast forms one enamel rod (Orban et al., 1943).

The end-product of the ameloblasts is a stratified epithelial covering, the enamel cuticle (Ussing, 1955), and is formed when the enamel has fully developed. This protects the mature enamel by separating it from the connective tissue cells until the tooth erupts.

Dentinogenesis

The dentinal matrix, unlike the enamel matrix, is not deposited in globular units but in rather a viscid fluid state which soon becomes calcified. The successive incremental layers, lines of von Ebner, are therefore not so sharply demarcated.

The formation and calcification of the dentine begins nearest the tips of the cusps or incisal edges and proceeds inward by rhythmic apposition of conic layers. One within the other. When the coronal portion is completed the apical layers assume the shape of elongated truncated cones (Schour, 1936).

The dentinal matrix or predentine formed is of two types (Provenza, 1964 p.127);

1. Mantle
2. Circumpulpal

The mantle dentine is that which is formed initially and accordingly will be subjacent to the dentino-enamel junction. At the beginning of the histodifferentiation, the peripheral mesodermal cells, subjacent to the inner enamel epithelium, assume a short columnar shape and become aligned in a single layer along the basement membrane. As differentiation proceeds the cells grow to several times their original length.

Provenza (1964 p.127) states that during this period of differentiation the mesodermal cells migrate into and beyond, and then retreat from, the basement membrane.

The first sign of the predentine is the presence of bundles of fibrils between the differentiating odontoblasts. These, Korff's Fibres, believed to originate from the intercellular

spaces of the dental papilla, are in a fan-like arrangement at right-angles to the separating membrane (Orban, 1929). In the dentinal mantle, of which they are the major constituent, the fibres are between the now-retreating funnel-shaped odontoblasts. It was first thought that these fibres were precollagenous, but more recent electron-micrograph studies reveal all the structural characteristics of collagen itself (Nylen and Scott, 1958).

The remainder of the mantle consists of smaller collagen fibres. These form a network which predominates throughout the circumpulpal predentinal layers, whereas Korff's Fibres, now compact bundles of parallel fibrils, become a minor component (Sicher, 1962 p.99).

By this stage of development the odontoblasts have receded further from the separating medium leaving behind single extensions, the odontoblastic processes, which have become embedded in the mantle.

From this point the dentinal matrix produced is described as circumpulpal and differs from the mantle layer on the basis of the predominating variety of formed intercellular components, (fibre - fibril) (Maximow and Bloom, 1957).

The fully differentiated odontoblasts decrease in size during further matrix formation but otherwise retain their structural

characteristics until the matrix formation is completed.

At this point they enter a quiescent state and, until stimulated to produce reparative dentine, their activity is restricted to an ordinary very slow formation of secondary dentine.

Throughout the matrix formation, the odontoblasts are arranged in a layer on the pulpal surface. Each cell gives rise to one process which traverses the dentine in a dentinal tubule (Orban, 1929).

Daily Rhythmical Activity of Apposition

The dentino-enamel junction of the tooth is characterised by high points which correspond to the position of cusps and lobes in the completed posterior and anterior teeth. These appositional growth centres are characteristic in number and position for each tooth.

Cellular activity commences at these high points (growth centres) and spreads at a rhythmically daily rate in two directions (Schour and Massler, 1940).

1. Successive cells along the dentino-enamel junction at successive daily intervals.
2. Each cell recedes rhythmically from the dentino-enamel junction.

The integration of these two directions results in the basic incremental growth pattern of the tooth. Therefore, the

given incremental layers - 4 microns thick - are in the shape of a cone with the apex directed occlusally. In enamel formation the cones deposited one on top of the other, whereas with dentine formation they are deposited one within the other. Once the full thickness of enamel or dentine has been achieved at the apex, subsequent layers are deposited at the sides in the form of truncated cones. This thickness of both the dentine and the enamel is governed by the life-span of the appropriate cells - ameloblasts or odontoblasts.

While the basic formative plan is the same, the final incremental pattern differs for different classes of teeth (Schour and Massler, 1940). The crown of a five-cusp tooth, e.g. lower deciduous second molar, represents the approximation and peripheral coalescence of the incremental cones of five growth centres. Likewise for the other posterior teeth, the only difference being in the number of cusps.

The anterior teeth are formed from four growth centres (Schour and Massler, 1940). Three are arranged mesodistally and the fourth to the lingual - this forms the cingulum. In the central and lateral incisors the three growth centres are at the same level, while in the cuspid the middle one is above the other two.

Cementogenesis

The commencement of calcification of the radicular dentinal matrix sees evidence of change in the cells of Hertwig's Epithelial Sheath (Gottlieb, 1942). This sheath, which separates the matrix from the surrounding connective tissue, begins to lose its continuity, either by

(a) degeneration of the epithelium

or by

(b) proliferation of the connective tissue.

Thus contact between the connective tissue and the matrix is established. With this the connective tissue cells differentiate into cuboidal cementoblasts (Sicher, 1962 p.132). Prior to the breakdown of the epithelial sheath there had been an increase in the number of collagen fibres found in the intercellular spaces of the future periodontal membrane. These fibres migrated, with the connective tissue, towards the dentine and contributed to the initial layer of developing cementoid. Concomitantly, the mucopolysaccharides of the connective tissue are converted into the ground substance.

Since, under normal conditions, the growth of the cementum is a continuous process, a thin layer of cementoid is always present on the root surface. This tissue responds similarly to osteoid in that it is highly resistant to osteoclastic

activity - whereas mature cementum, dentine and bone are readily resorbable. On this basis a protective function has been attributed to the surface layer of the cementum (Gottlieb, 1942).

Calcification

Calcification of Enamel Matrix

Mineralisation occurs in the matrix segments and interprismatic substances immediately following their elaboration.

Weinmann, Wessinger and Reed (1942) found that in this initial stage 25% of the definitive mineral content is laid down in the matrix in the form of a crystalline apatite.

The second stage of maturation consists of the gradual completion of mineralisation beginning at the dentinal end of the rods in the crown of the tooth and progressing cervically (Weinmann, Wessinger and Reed 1942). This begins before the enamel matrix has reached its required thickness. Crabb and Darling (1962) state that the central core of the enamel prism appears to mineralise before its periphery.

At first, the maturation occurs parallel to the dentino-enamel junction, but later it tends to follow the outer enamel surface (Crabb, 1959). Following this basic pattern, incisal and occlusal areas reach maturity ahead of cervical areas.

Electron microscopic studies have provided the means by which the mineralisation process can be visualised (Provenza, 1964 p.139). In the primary phase initial nucleation and length-wise growth results in the formation of long ribbon-shaped crystals. Maturation is characterised by growth and eventual fusion of these crystals seen in the primary phase (Sicher, 1962 p.169).

During development the incremental striae showed degrees of mineralisation both greater and smaller than that of the surrounding enamel (Crabb and Darling, 1962).

Calcification of Dentinal Matrix

Calcification of dentinal matrix begins when several microns of the matrix have been formed in small areas in the layer closest to the dentino-enamel junction - mantle dentine, at the highest point of the dental papilla (Sicher, 1962 p.99). As calcification progresses, these enlarge and fuse until a uniform band is formed (Takuma, 1960); thereafter the pattern is usually lamellar, paralleling the retreating odontoblasts. Occasionally sporadic calcification occurs in the circum-pulpal dentinal matrix. This is in the form of globules which subsequently fuse.

The earliest mineralisation is in the form of hydroxyapatite on the surface of the fibrils and in the ground substance.

Subsequent to this intrafibrillar deposition occurs (Takuma, 1960). Within the globular areas deposition takes place radially from common centres.

Calcification of Cementum

Morphologically cementum can be divided into two types (Kronfeld, 1938), cellular and acellular but their location is not definite and layers may alternate in almost any arrangement.

1. Acellular cementum, which may cover the root dentine from cemento-enamel junction to apex, but is frequently absent in the apical third. It contains calcified intercellular substance.
2. Cellular cementum, usually formed on the surface of acellular cementum, but may comprise the entire thickness of the apical cementum where it is always thickest. Its growth contributes to the length of the tooth. Both types of cementum are penetrated in their superficial layers, by fibres of the periodontal membrane - Sharpey's Fibres.

In acellular cementum, calcification occurs in the form of lamellae involving the matrix somewhat removed from the base of the cementoblasts. Because this mineralisation is distant to the cementogenesis cells do not become involved. In cellular cementum mineral deposits are found in the form of spherules dispersed through the matrix and are even to be found intercellularly. As these increase in size, the matrix

surrounding the cementoblasts becomes involved. The production of the acellular type is slow in comparison to cellular type. Both cellular and acellular cementum are separated in incremental lines.

The occurrence of a sufficiently severe local or systemic disturbance at any time during the appositional development of the crown may result in the arrested activity of a group of functional ameloblasts (Sarnat and Schour, 1941), their life is terminated prematurely and the resulting rods will be shorter than normal. This evidences as a coronal area lacking in enamel and may be observed as pitting, furrowing or even total absence (clinical aspects p.113).

A disturbance to the calcification of a tooth results in hypocalcification. This is a deficiency in the mineral content of the enamel which is distinct from hypoplasia, the latter being a defect in enamel formation. In the former, the enamel presents as enamel matrix (Weinmann et al., 1945).

Disturbances to calcification are much more common than disturbances to formation (Kronfeld and Schour, 1939), either hypocalcification or hypoplasia may be caused by the same systemic interference depending on the degree of the disturbance. A mild disturbance may cause deficient calcification without affecting the formation of the organic

matrix, but a severe disturbance may cause a hypoplastic defect in addition to deficient calcification.

Thus hypocalcified and hypoplastic defects, although arising from two different development processes, are yet related in as much as they indicate different degrees or intensities of systemic or local disturbances.

In hypoplasia, a difference is seen in the involvement between the ectodermal and the mesodermal dental tissues. The ectodermal formative cells are more vulnerable and less amenable to repair and regeneration than the mesodermal tissue (Kronfeld and Schour, 1939). This is emphasised by hypoplasia formed during the neo-natal period, Schour (1936) found that the neo-natal ring, a disturbance of enamel and dentine formation arising during the change from intra- to extra-uterine environment, is present in 98% of deciduous and first permanent molar teeth. If the metabolic disturbance during this process is severe enough, there will be cessation of the amelogenesis of those cells involved, whereas the dentine formation, after a transitory period of retardation, continues normally (Kronfeld and Schour, 1939).

Similar results are noticed in mild fluorosis and other mild interferences causing mottling, but with no visible change in the dentine. With severe fluorosis and other grave interferences, the enamel is frequently hypoplastic whereas the

dentine shows only interglobular zones. It is only in extreme circumstances - rickets, hypo-avitaminosis A - that vascular inclusions can be found in the dentine (dentine hypoplasia) (Kronfeld and Schour, 1939).

Eruption

Active eruption begins at the time root formation commences and continues throughout the life span of the tooth.

The period of tooth development involving the coronal portion, prior to the root development, is classified as the pre-eruptive stage. Once root formation has commenced the eruptive stage is initiated.

Accordingly, the eruption of both the deciduous and permanent teeth can be divided into the following phases (Weinmann, 1941)

1. Pre-eruptive phase
2. Pre-functional eruptive phase
3. Functional eruptive phase

During these phases the teeth move in different directions.

These movements may be

1. Vertical - occlusal in the direction of the long axis.
2. Horizontal - bodily in mesial, distal, buccal or lingual direction.
3. Tilting - about a transverse axis.
4. Torsion - rotating around longitudinal axis.

Pre-eruptive Phase

Eruption is preceded by a period in which the developing and growing teeth appear to adjust their position in the growing jaw (Brash, 1928).

During this phase the dental organ develops to its full size and the formation of the hard substances of the crown takes place. Corresponding changes occur in the tissues and the tooth germ is surrounded by the loose connective tissue of the dental sac and by the bone of the dental crypt.

The development of the tooth and the growth of the jaws are simultaneous and inter-dependent processes, with the tooth germs maintaining their relation to the growing alveolar border. This occurs by occlusal and buccal movement of the tooth germ (Brash, 1928).

The processes responsible for this movement are (Sicher and Weinmann, 1944) :

1. Bodily movement.

This is a shift of the entire tooth germ involving resorption in front of the moving bud and apposition behind it.

2. Excentric Growth.

Here one part of the tooth germ remains stationary. Excentric growth leads to a shift of the centre of the tooth germ and is characterised by resorption from in front of the moving tooth, but with no apposition behind.

As the alveolar arches expand, the deciduous teeth move in a vestibular direction. Concurrently with this, both bodily shift and excentric growth cause the anterior teeth to move mesially and the posterior teeth distally. Brodie (1934) states that

"Growth studies reveal that at birth the germs of all teeth, except the permanent second and third molars, lie packed in the jaws which are extremely small. In the maxilla they are in close proximity to the orbit. During growth the face descends and goes forward and outward so that by the time the adult stage is reached the teeth have travelled an approximate $1\frac{1}{4}$ - $1\frac{1}{2}$ inches."

The permanent successors to the deciduous teeth undergo intricate movements before they reach their erupted position. The germs of the permanent incisors branch from the lingual side of the dental lamina and are originally located in the connective tissue lingual to, and at the occlusal level of, the deciduous tooth germ (Logan and Kronfeld, 1933). At the end of the pre-eruptive phase they are found lingually to the apical region. Similarly, the developing premolars move from their position lingual and occlusal to the deciduous germ, to between the divergent roots.

Pre-functional Eruptive Phase

This is the period from the commencement of root formation to when the tooth reaches the level of the occlusal plane.

During this phase there is rapid growth of the alveolar ridges of the jaws. To emerge from these growing jaws the developing tooth must move more rapidly than the ridge increases in height (Kronfeld, 1932).

This movement is the result of

1. Primary effects - elongation by radicular apposition
2. Secondary effects
 - (A) re-organisational changes of bony crypts and periodontal connective tissue.
 - (B) increased activity of pulpal tissues.

1. Primary Effect

The growth of the root is initiated by proliferation of Hertwig's Epithelial Sheath and connective tissue of the dental papilla. The proliferation of the epithelium takes place by mitotic division of cells of the epithelial diaphragm.

2. Secondary Effect

(A) Since growth of the root alone is not enough to move the crown to the level of the occlusal plane, the erupting

movement is aided by growth of the bone at the base of the crypt. This lifts the tooth with its hammock ligament towards the occlusal surface.

The hammock or cushioned hammock ligament, is a network of thick fibres adjacent to the growing end of the tooth.

These fibres, which are attached to bone form a strong ligament around the base of the tooth, and have two functions (Sicher, 1942 (b))

1. Correlation of root and bone growth to one directed effort - the axial eruption of the tooth.
2. Supporting structures for the growing end of the tooth.

Weinmann (1941) was able to show apposition of bone in alveolar fundus in two ways

- (a) layer upon layer - slow growth
- (b) new trabeculae formed at some distance from old - rapid growth

The latter is mainly responsible during this pre-functional eruptive stage.

(B) The increased activity of the pulpal tissues causes a slight increase in the pressure in the dental crypt. The hammock ligament is instrumental in preventing resorption at the base of the crypt (Sicher, 1942 (b)). This rise in

pressure is the stimulus for the initiation of tissue changes that will release the pressure - forward axial movement with resorption of bone from in front of the tooth.

Scott (1952) states that active eruption is the result of this growth of the tooth pulp between the calcified part of the tooth and the base of the follicle, and that root formation is a consequence of tooth movement and not a cause.

During this stage three layers of the future periodontal ligament can be distinguished (Orban, 1928; Sicher, 1942 (a))

1. Dental fibres adjacent to tooth surface
2. Alveolar fibres attached to primitive alveolus
3. Intermediate plexus

The alveolar and dental fibres are mainly collagenous and can be traced into, but not through, the intermediate plexus. It is this plexus which permits continuous adjustment to the rapidly erupting tooth (Sicher, 1942 (a)).

As the crown moves towards its clinical eruption - emergence into the mouth - the connective tissue separating the dental from the oral epithelium narrows and then disappears, allowing fusion between the oral and dental epithelium.

Functional Eruptive Phase

This stage consists of occlusal and mesial compensatory movements.

Continued eruption, once the tooth has reached the occlusal plane, compensates for loss of the tooth structure by occlusal and incisal attrition. This is the only way that the occlusal plane level and distance between the jaws can be maintained.

Functional movements also lead to loss of mesio-distal width due to wear at individual contact points. Firm contact is maintained despite this loss of substance by a continuous mesial drift of the teeth.

In the functioning teeth, differential growth appears to be the cause of the occluso-mesial movements. Apposition of cementum occurs on the entire surface, but is greater in furcation and apical areas. Also, bone growth occurs at the fundus and on the distal walls of the socket as well as on the alveolar crest. Resorption on the mesial wall appears to be secondary to the mesial movements. A continual rearrangement of principal fibres of the periodontal ligament occurs, during these movements, at the intermediate plexus (Sicher, 1942 (a)).

Exfoliation of Deciduous Teeth

This is the physiological elimination of the deciduous teeth prior to their replacement by the permanent successors.

The progressive resorption is performed by multi-nucleated giant cells which have differentiated from the cells of the loose connective tissue in response to the pressure exerted by the erupting tooth germ.

Resorption, however, can occur in the absence of a permanent successor. In these cases, resorption appears to be instituted by the continued occlusal stress (Aisenberg, 1941).

The resorption of the roots of the deciduous incisors and canines first occurs on the lingual surface at the apical third. At this time the movement of the permanent incisors is in an occlusal and labial direction, (see section on eruption). In later stages, the permanent tooth is found directly apical, and so resorption of the deciduous predecessor occurs accordingly. Frequently, vestibular movement of the permanent tooth is not completed when clinical eruption occurs and so it appears lingual to its deciduous counterpart.

With the germ of the permanent teeth between the roots of the deciduous molars their resorption is first observed on the inner surfaces of the roots. However, continued

eruption of the deciduous molar often moves it further away from the permanent tooth, allowing for repair by apposition of cementum.

In later stages the erupting permanent tooth closes to the deciduous molar and resorption again takes place.

Resorption of the deciduous teeth is not a continuous process, but one in which periods of activity follow periods of rest. During these periods of rest, resorption not only ceases, but repair may occur. This explains the variation in firmness of the deciduous teeth (Oppenheim, 1922).

A broad band of variation exists in the normal eruption times of the deciduous and permanent teeth for different individuals and because of this it is difficult to determine when eruptive times are outside the limits of the normal range. However, certain cases occur in which these times are grossly beyond the limits (clinical aspects p. 136).

By birth, or soon after, one or two teeth may have erupted. These have been termed natal and neo-natal teeth, respectively (Massler and Savara, 1950). Heredity appears to play an important role in the early eruption of these teeth which are mainly found in the mandibular incisal region. In a few cases they become progressively looser and are lost within a few weeks, but the majority develop normally and are shed at

the normal age (Farmer and Lawton, 1966 p.126).

Rushton (1953) points out that these teeth are erupting normally and it is their superficial position that is abnormal. This allows the crowns to clinically erupt before the roots have had sufficient time to develop.

In the deciduous teeth, localised retardation of their eruption may be due to an eruption cyst which has developed from epithelial remnants. Precocious eruption of the first permanent molar may impact and so prevent eruption of the second deciduous molar (Worth, 1963 p.917).

Generalised retardation of this dentition may be associated with nutritional deficiencies - rickets (Friel, 1922) - or endocrine disturbances - hypopituitarism (Schour and Massler, 1943 p.599) and hypothyroidism (Schour and Massler, 1943 p.603).

Premature loss of the deciduous teeth is predominantly due to their extraction as the result of dental caries, or its sequelae. Sleichter (1963) reports that this will generally expedite the eruption of the permanent successor unless the extraction is carried out at an early stage with the permanent tooth still deeply embedded in the alveolar bone. This then may result in the retardation of eruption. Generalised early loss of the deciduous teeth has been reportedly associated with Pink disease (Nussey, 1954), Papillon-Lafevre Syndrome (Hall, 1963)

and Hand-Schuller-Christian disease (Talley, 1948).

Instead of being shed at the normal time, the deciduous teeth may be retained. This may be a general retardation associated with the delayed eruption of the permanent dentition as occurs in cretinism (Schour and Massler, 1943 p.60), rickets (Friel, 1922), and cleido-cranial dysostosis (Millhon and Austin, 1954); or there may be partial or total anodontia.

Persistence of an individual deciduous tooth may be due to the congenital absence or, more rarely, the late development of the succeeding permanent tooth. It may be due to the malposition of this underlying permanent tooth, so that it is not in its correct position beneath the deciduous predecessor (clinical aspects p.142), or to breakdown in the co-ordinated movements during the pre-functional eruptive stage which may lead to an impaction and embedding of the tooth.

Excessive trauma may result in an arrest of active tooth eruption during the functional eruptive phase if the periodontal ligament has been damaged (Willman, 1930). Resorption of the root may ensue, in which event the deposition of bone in these resorbed spaces may lead to ankylosis by fusion of alveolar bone and tooth (Dixon, 1963) (clinical aspects p.146).

Eruption of the permanent dentition is very much governed by the behaviour of the deciduous teeth for, if there is to be

early eruption of the permanent teeth, it is necessary that there should be premature loss of the deciduous teeth. The premature eruption of the permanent teeth is a rare occurrence and may be found in hyperthyroidism and hyperpituitarism (Schour and Massler, 1943 p.764,766).

The delayed eruption of the permanent teeth has been mentioned in conjunction with the prolonged retention of the deciduous teeth. Conditions other than those which may be conducive to retarded eruption are - the lack of space for the erupting tooth and the presence of supernumary teeth, cysts and other developmental abnormalities.

PART II

Review of Literature of the Clinical Aspects of the Anomalies

The review of literature has been divided into the following sections, and each will be dealt with separately

1. Abnormality of Number
 - a. Anodontia - total
partial
 - b. Supernumary teeth
2. Abnormality of Size
 - Megadontia and Microdontia
3. Abnormality of Shape
 - a. Gemination and Fusion
 - b. Dilaceration
 - c. Odontomes -
Complex and Compound
Composite
Gestant and Invagination
4. Abnormality of Structure
 - Hypoplasia - Enamel
 - a. Aplasia
 - b. Amelogenesis Imperfecta
 - c. General developmental defects
 - d. Endemic Fluorosis
 - e. Result of Tetracycline therapy
 - Dentine
 - Dentinogenesis Imperfecta
5. Abnormality of Eruption
 - a. Aberrant eruption times
 - b. Ectopic eruption
 - c. Ankylosis of deciduous molars

1. Abnormality of Number

Total Anodontia

Total anodontia describes the complete developmental absence of teeth (Part I, p. 5). This is a rare condition usually associated with the more generalised disturbance of ectodermal dysplasia (Famrer and Lawton, 1966 p.155). Ectodermal dysplasia is an entity resulting from the suppression of development of the ectodermal tissues in the developing human embryo (Eisenson, 1956). The ectodermal epithelium commences development soon after the second month of foetal life (Thoma and Goldman, 1960 p.25). Other structures affected by this suppression of development are the hair, glands, iris and fingernails.

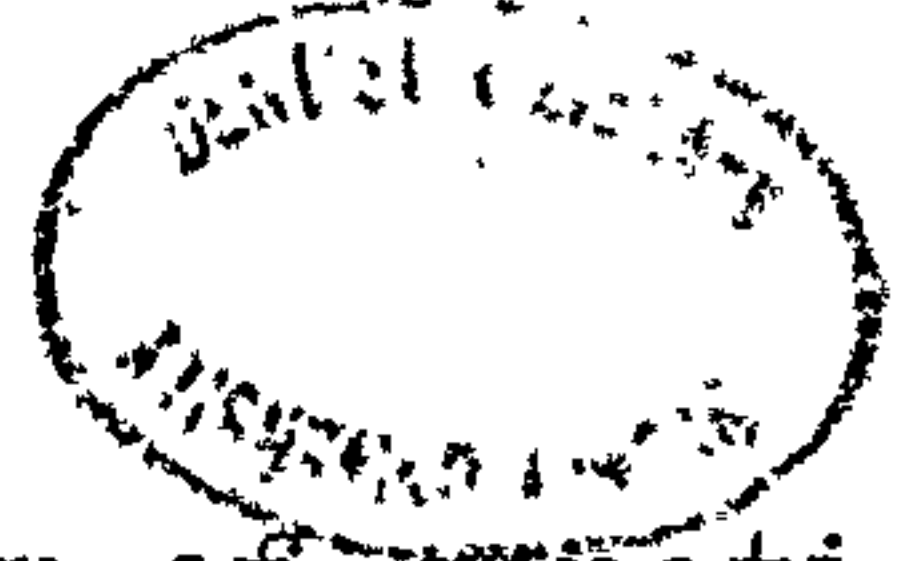
The origin of this anomaly has been investigated by many workers. Owen (1871) cited, as proof of the evils of inter-marriage of blood relatives, a case in which a young man was "prevented from having a tooth" because the father had married his cousin. The young man's sister had two or three small stubs of teeth present.

Thadani (1921) discussed the bald and toothless condition of men in India. These were known as Buddhas. He was able to study families in which the condition occurred in various members and generations and found it to be transmitted by a sex-linked inheritance of recessive nature.

Thoma and Allen (1940) in agreeing with this, state, "The inheritance is of a recessive type, the disease appearing in the grandsons. In the second generation the males are not affected, but the character is carried by all the unaffected daughters. This is due to the fact that the anomaly is inherited with the 'X' chromosome of the father. In the third generation the children may either inherit the affected or the normal 'X' chromosome from the mother, and thus the sons who inherit the defective 'X' chromosome become affected, but those who inherit the normal 'X' chromosome remain normal. Of the daughters, some may also inherit the defective 'X' chromosome while others may inherit the normal. In the third generation, therefore, the females with the defective 'X' chromosome become carriers and pass the disease on to future generations."

Guilford (1883) presented one of the first documented reports, a male who "Although forty years of age, and in perfect physical health, having never been confined to his bed by sickness a day in his life, he has been edentulous from birth, is totally lacking the sense of smell and almost devoid of the sense of taste; the surface of his body is destitute of the fine hairs that should cover it and he has never perspired."

The hereditary facts of this case are interesting. Guilford makes mention of the maternal grandmother, bald and edentulous.



This is an exception to the Mendelain system of genetic transmission and should happen only if a female inherits two affected 'X' chromosomes, one from each parent. In such a case it must be supposed that one parent was a carrier of the defect which had been recessive for several generations (Thoma and Allen, 1940).

In 1936 Battersby reported the complete absence of the deciduous and permanent dentitions in a nine-year-old boy. His head was well covered with fine light-coloured hair, there was no evidence of rickets or any other organic disease and x-ray films showed a complete absence of teeth or rudiments of teeth. The boy could not stand exposure and only his hands and feet perspired. The anomaly was supposedly inherited from the mother, who lacked the maxillary and mandibular permanent lateral incisors and both mandibular central incisors. Her father and great-grandfather had only eleven permanent teeth, spaced and distributed between the maxillary and mandibular jaws.

Dahlberg (1937) stated that endocrine glands, syphilis, diet and heredity have all been discussed in this connection and no doubt are all correct to a certain extent. It appears, however, that the hereditary factor is responsible for a large percentage of these conditions.

Sperber in 1963, suggested that there may be two non-related forms,

1. The inherited form, which might be termed the 'phylogenetic' type, in that the trait could become an evolutionary characteristic.
2. The 'ontogenetic' type, which appears in persons with no previous hereditary history of the condition.

These two types would be clinically undistinguishable but they could be differentiated on the basis of the patient's history.

Everett et al (1952) state that differences of opinion do arise as to the mode of genetic transmission of this abnormality, which may be of a dominant, recessive or of a sex-linked character. However, most patients are described as showing recessive, sex-linked characteristics, males being affected about eight times more frequently than females.

Downs (1928) studies patients affected by various endocrine disturbances and concluded that the dental anomalies found cannot be considered pathognomonic for any definite type of endocrine dyscrasia.

The time of onset of the ectodermal disturbance is instrumental in determining the degree of dental aplasia (Sperber, 1963); the earlier in development the interference takes place, the more severe the result. Thus, in successive

categories of severity, total anodontia, absence of permanent teeth only, or absence of only a few teeth from either dentition would be a reflection of instances of progressively later interference in the normal developmental pattern of the dentition. However, in such cases where the aplasia of the dental lamina is incomplete and allows the formation of a few teeth in either the maxilla or mandible, there is generally insufficient vigor to this tissue, and hypoplastic forms result. The teeth having a rudimentary cone-shaped appearance (Thoma and Allen, 1940).

Thoma and Goldman (1960, p.30) cite Willner (1936) who collected 125 cases of anodontia from the literature in an effort to classify them. Fifty of these were sufficiently documented for his purpose. The following classification is an elaboration of that offered by Willner.

- A. Total anodontia in both jaws -
 - 1. Affecting both dentitions
 - 2. Affecting only the permanent dentition
 - 3. With two or three peg-shaped teeth present
- B. Total anodontia in one jaw only -
 - 1. Affecting lower jaw; in upper, partial anodontia
 - 2. Affecting upper jaw, lower mixed
- C. Total anodontia on one side of face only
- D. Subtotal anodontia in both jaws

In 1953 Sarnat, Brodie and Kubacki reported a clinical and roentgenographic study of a patient, with complete anodontia, up to the age of sixteen years. The patient, who lacked sweat and sebaceous glands, had sparse languo-like hair, which increased over the period of examination, but still lacked pigment. The serial roentgenographic appraisal of the cranio-facial development during childhood and adolescence revealed the patient's overall measurements to be at least equal to small normal.

In 1931 Sainsbury presented a case from the United Dental Hospital, Sydney. This showed the clinical features, as reported by many other authors, as poor growth of hair, no eyebrows, fingernails soft, body and limbs normal and no mental deficiency. The abnormality was not hereditary.

Everett et al. (1952) reported the case of almost total anodontia in an eight-year-old boy. The maxillary right and left second deciduous molars being the only teeth present. They attributed the high caries rate found in these teeth to the fact that very little saliva was produced.

A case of partial anodontia was reported by Etheridge (1913) in which the patient was totally edentulous on the left side of the mandible. The dentition of the maxillary and mandibular right sides was complete except for both the central incisor teeth. In the left maxillary arch the first permanent molar, the second deciduous molar and the deciduous

cuspid teeth were present. A neurotrophic aetiology was postulated. This was supported by the presence of a hemiatrophy on the left side of the body, associated with left side alopecia.

Steadman (1953) presented the case of a five-year-old boy in whom the only tooth present was the maxillary right second deciduous molar; this was of atypical form. Hutchinson (1953), Cramer (1947), Warr (1938) and Cautley (1928) have reported cases of complete anodontia of the permanent dentition, the patients appearing normal in all other respects. In the case of Cautley, the patient was not x-rayed, but at twenty years of age only deciduous teeth were present.

Anodontia must not be confused with multiple retention of the teeth in the jaws. Ivy (1933) describes a patient who, upon superficial examination, showed the lack of the entire permanent dentition. X-ray examination revealed the permanent dentition unerupted. Many teeth were malformed and supernumary teeth were present. The father, sister and brother of this patient also had histories of delayed eruption.

Partial Anodontia

Partial anodontia (oligodontia) is the developmental absence of one or more teeth in either the deciduous or permanent dentition or both (Part I, p. 5). This is not nearly as rare as total anodontia.

Hereditary ectodermal dysplasia accounts for many cases of partial anodontia. Rushton (1934) presented a report of the condition occurring in six members of a family in three generations. Guilford (1883), Battersby (1936) and Eisenson (1956) reported cases of total anodontia with associated ectodermal defects where other members of the family, first and second generations, exhibit partial anodontia.

Thoma and Goldman (1960, p.31) state that the inherited defect may become modified, producing in the offspring after a few generations, only partial anodontia. Hinrichsen (1963) reported the case of dizygous male twins showing severe partial anodontia together with other manifestations of anhydrotic ectodermal dysplasia.

Mutations may play an important part in the aetiology of partial anodontia. If the ectodermal defect is affected later in foetal life, the aplasia of the dental lamina may be incomplete and so fail to develop the anlage of an individual tooth, or group of teeth, or there may be insufficient vigor of the tissue and hypoplastic forms result, the teeth

having a rudimentary cone-shaped appearance (Thoma and Allen, 1940).

Dahlberg (1937) reported the identical anomaly, the absence of six deciduous and permanent incisors, appearing in eighteen of forty-six members of four generations. He states that "as to the etiology little can be said except that mutations appear from time to time for no apparent cause and the new characteristics are transmitted, modified or lost depending on various factors, including types of mating."

Porter and Edwards (1937) reported a case of a thirteen-year-old girl who had twenty-nine permanent teeth missing. Her fifteen-year-old brother had sixteen teeth absent. No abnormal hereditary condition was found in either parent.

Keeler (1935) in a paper entitled 'Heredity in Dentistry' collected ten pedigrees showing twenty-three marriages between normal individuals and persons with congenital absence of one maxillary central incisor tooth, two maxillary central incisor teeth or having one or both under-developed. These matings produced thirty-eight normal offspring and forty-five having some form of defect. The defect inherited as a unit dominant character. Keeler suggests that the expected equality, according to Mendelian Law, was not quite reached, but this may have been due to inaccuracies in recording.

Grahnen (1956) states that tooth agenesis is more frequent in parents and siblings of individuals with missing teeth than in the population as a whole. A finding that strongly supports the hypothesis that this condition is genetically determined.

Identical twins have been recorded, each with the absence of the mandibular second premolar teeth. In both cases, the second deciduous molar was present (Farmer and Lawton, 1966 p.157). However, Greenberg (1961) reports a case of identical twins in one of whom the maxillary second incisors were absent. In the second child these teeth were fully developed. The implication from this being that congenitally missing teeth may not be absent in all circumstances because of a hereditary factor, and even further, that environment may play some part.

Thoma and Goldman (1960, p.33) on the other hand, cite the studies of twins by Zerger and Winkler (1931) in which the deficiency corresponds. In the first case of the identical twins, each individual had both maxillary second premolar teeth missing. In the second case, the maxillary and mandibular second premolar teeth and the mandibular second permanent molar teeth were missing bilaterally. In the case of fraternal twins, however, it was found that one twin was missing all first and second premolar teeth except the mandibular right first and second premolars, while in the other

twin the maxillary premolars only were absent. Zerger and Winkler believed this to be good proof for the hereditary diathesis rather than an aetiology of somatic disease.

Geneticists (Roberts, 1959; Stern, 1960 p.289), from surveying physical and biochemical anomalies occurring in many family pedigrees, have suggested that variations in the expressions of genes may be expected. On the one hand, there is the factor of penetrance, which refers to the presence or absence of a gene factor: it has been observed that an anomaly can skip a generation (non-penetrance) yet appear in subsequent generations (indicating a 'carrier' individual) giving fullest expression of the anomalous form (complete penetrance).

However, in very many genetically distinctive traits or characters we see variable expression, i.e. variable degree of involvement or variable expressivity (incomplete penetrance). This concept could be exemplified in those instances of identical twins which, while both show evidence of the dental anomaly, have the anomaly expressed in different degrees of 'severity'. The instances of variable expressivity of a particular gene allele are considered to be the result of varying internal environments (particular total genetic constitution of the individual being) interacting with the particular external environment. This concept satisfies the idea of hereditary diathesis or predisposition. It does not necessarily rule out somatic disease as the initiating factor, but

also serves to complicate evaluation of causes of congenital anomalies.

The theory of phylogenesis has also been used to explain a reduction in the number of teeth. Dahlberg (1945) stated that the dentition of man is changing in form, size and number. The general trend is towards a simplification of the patterns and a reduction in measurements of the thirty-two teeth found in man.

Werther and Rothenberg (1939) state : "The deficiency of teeth is found more often in the permanent denture. It occurs with such regularity that it leads to the belief that it results from a well-determined reduction. In most cases the maxillary lateral incisors, the mandibular central incisors, the maxillary second premolars and the maxillary third molars are missing, either separately or in any combination at the same time. This cycle (reduction) has not been completed yet. Formerly the tooth formula was $3413/3143$: it means 3 molars, 4 premolars, 1 canine, 3 incisors, 3 incisors, 1 canine, 4 premolars, 3 molars. Today it is $3212/2123$, and the future formula will probably be $2111/1112$, if the process continues."

It is noticed that the observed lack of tooth germs coincides with those teeth, shown by Dahlberg in 1953, to be most variable.

Bolk (1914) stated that the three teeth in our dentition which are on the way to reduction and elimination are : the maxillary lateral incisors, the second premolars and the third molars.

Varying observations have been made about this theory of reduction. Schultz (1932), in support of the theory, cited the high frequency with which third molars, in the posterior part of the arches, and the maxillary lateral incisors and the mandibular central incisors, in the anterior position, are missing. However, he did not consider the second premolar teeth which are also frequently missing and yet do not have a similar anatomical relationship to the ends of the arches.

Hellman (1936) found, amongst 433 dental students, 25.4% had one to four third molars missing, 19.16% had one or two missing. In a series of skulls of nineteen different races and nations, 29.6% had one to four missing third molars, and 18.9% one or two.

Euler (1936), in a similar study, found 26.83% of third molars missing in prehistoric man but only 1.33% amongst 150 students. He does not believe that the human dentition has been developing towards a reduction in the number of molars.

Werther and Rothenberg (1939), reporting on these studies, state that they only cover a short range, 5-10,000 years, and because of this they cannot be considered proof. More than ten times as many years have to pass before any change in development may become dominant enough to occur in a great number of human beings. Also the processes of natural selection which very likely influenced dentition development over the past millennia may no longer exist, and so it would indeed be impossible to forecast future evolutionary trends.

The rare phenomenon of missing permanent or deciduous canines does not support this theory. This defect has been reported by Lubner (1937), Thompson (1936), Fustman (1952) and Goldman (1962).

Many authors suggest that somatic diseases are responsible for partial anodontia. Evan (1947) cites three cases of partial anodontia in children whose mothers contracted rubella in the first three months of pregnancy. Stocker (1951) describes the complete aplasia of the permanent dentition in similar circumstances.

Thoma and Goldman (1960, p.115) believe that children, born to mothers who had severe exanthematous fevers during early pregnancy, may show a deficient dentition.

Stein (1913) stated that there are numerous reports of missing maxillary lateral incisors as the result of congenital syphilis, but that the absence of these teeth is not necessarily a sign of congenital syphilis. The original defect was thought to become a dominant character handed down to numerous generations (Campbell, 1934). This inheritance of acquired characters is now known not to be tenable (Stern, 1960 p.483).

Miller (1937) reported a case of dental dystrophies associated with achondroplasia. The chief findings consisted of maldevelopment and retention of the deciduous dentition and absence, or greatly retarded eruption, of the permanent teeth.

Quinby (1937) cited a case he examined of a seven-year-old boy who had developed rickets within the first year of his life. An examination revealed no teeth in the mandibular arch and only the two second premolars in the maxillary arch.

Aitchison (1953) claimed that hypertelorism with associated partial anodontia is sufficiently common to place the dentist on his guard when about to examine a patient, who shows undue breadth between the eyes. He reports having seen two such cases with missing maxillary lateral incisors and three cases with non-specific peg-shaped maxillary permanent lateral teeth.

Downs (1928) found partial anodontia more frequently in glandular dysfunction, particularly in hypothyroidism or mongolism.

Sperber (1963) states that "These various disease factors which could produce disturbed dental development are not normally inheritable features. Accordingly, the resulting anodontia would be considered to be of the ontogenetic type." He also states "Their (incisor teeth) frequent absence may be attributed to their midline position. The midline, being the most remote point of the blood supply to the jaws, is the region more likely to suffer relative oligemia. This precarious blood supply during the formative period of the incisors is likely to hinder their development and even result in their total absence."

Paul (1967) observed greater incidence of missing teeth in patients with clefts involving the maxillary alveolar ridge.

Some of the studies which have reported on the incidence of congenitally absent teeth in children, in both the permanent and deciduous dentitions, are summarised below.

Percentage Incidence in Permanent Teeth

<u>Investigator(s)</u>	<u>Percentage</u>
Werther and Rothenberg (U.S.A. 1936)	2.3
Dolder (Switzerland 1937)	3.4
Clayton (U.S.A. 1956)	7.1
Brown (U.S.A. 1957)	7.2
Lind (Sweden 1959)	7.4
Castaldi et al. (Canada 1966)	4.1

Percentage Incidence in Deciduous Teeth

<u>Investigator(s)</u>	<u>Percentage</u>
Menczer (U.S.A. 1955)	0.09
Plaetschke (Germany 1938)	0.7
Granen and Granath (Sweden 1961)	0.4

Sabes and Bartholdi (1962) studied 157 cases of varying degrees of partial anodontia selected from 4,000 patients admitted to the dental school at the University of Minnesota. These patients evinced a total of 365 missing teeth. Seventy-nine percent had one or two teeth missing and only seven percent manifested lack of development of five or more. The following table shows the range.

<u>Number of teeth</u>	<u>Cases</u>	<u>Percentage</u>
1	64	40.76
2	61	38.86
3	10	6.37
4	10	6.37
5	2	1.27
6	1	0.64
8	2	1.27
9	4	2.55
10	2	1.27
21	1	0.64

The results of a survey by Grahnen and Granath (1961) indicate that a correlation occurs between the two dentitions. Of eight cases of partial anodontia in the deciduous dentition,

six were found with hypodontia of the permanent dentition in the corresponding regions. Two also had missing teeth in other regions.

b. Supernumary Teeth

A supernumary tooth may be defined as the occurrence of a tooth in excess of those normally found in the human dentition (Part I, p. 6). These may be divided into two groups.

- (a) those resembling teeth in the normal permanent or deciduous dentitions
- (b) those of conical or tuberculate form

These are termed supplemental teeth and supernumary teeth respectively (Farmer and Lawton, 1966 p.162).

In reviewing the literature, one finds various theories advanced concerning the origin of these extra teeth. The theory of atavism (Fastlicht, 1943), which is the recurrence of the ancestral forms of the teeth since eliminated in the process of evolution, is now not so strongly held (Gardiner, 1961).

As stated earlier (anodontia) the formula for the present dentition in man is $3212/2123$, totalling thirty-two teeth. According to phylogenetic studies primitive placental mammals, from which man seems to descent, had the formula $4313/3134$,

forty-four teeth. The distribution of these teeth in man's ancestors was three incisors, one cuspid, three premolars and four molars in each quadrant. The presence of supernumary teeth is attributed to an effort by nature to return these teeth to their primitive place (Fastlicht, 1943).

In 1939 Werther and Rothenberg stated "Formerly it was believed that a lack of individual tooth germs was a sign of general physical degeneration. Today it is considered, by many writers, to be a retrograde development in which the number of teeth is decreasing to conform to the needs of mastication of this era of human development. On the contrary, the often observed supernumary teeth are believed to represent an atavistic reversion."

In 1912 Osburn defined atavism as the reversion to ancestral type and stated "it is thus a sort of long-distance heredity." He feels that this theory is overworked in an attempt to explain the presence of supernumary teeth, and sums up evidence against their origin by reversion in the following manner :

1. A third incisor, a third premolar or a fourth molar in man could only be accounted for, as atavisms, by a reversion covering so long a period of time (ten millions years), that this alone is sufficient to render the theory untenable.

2. There is no ancestral condition which would allow the presence of a second canine.
3. Supernumary teeth seldom present ancestral characters.
4. Duplicate teeth, occurring in or out of the series, are not atavistic in character.
5. The theory offers no explanation as to why
 - (a) teeth may be omitted from any part of a series
 - (b) discontinuous variations of a similar character often occur in other structures of a similar serial nature.
6. The paleontological record of the evolution of the teeth, which is fairly complete in all mammalian orders, lends no support to the theory.

Bolk (1914) stated that the occurrence of supernumary teeth in the molar region of man is not a haphazard phenomenon, but subject to certain regularity. He felt certain that we are not dealing with accidental products of dentition, but with elements that are of the greatest importance for our conception of the problems involved in the genesis and final evolution of the molars.

He introduced and discussed the distinctive terms :

1. Para-molar - the rudimentary tooth which is situated laterally to the molar row, and

2. Disto-molar - the tooth situated behind the third molar, which other writers call the fourth molar.

Previous to this, Bateson (1891) and Black (1909) had rejected the reversion theory (Werther and Rothenberg, 1939).

Werther and Rothenberg state that Black was apparently unaware of Bateson's work, though his theory fitted in generally with the study and conclusions of Bateson.

Black (1909) proposed the theory that supernumary teeth were due to excessive growth in the epithelial tooth band or dental lamina. Abnormal proliferations from this band could arise from three different positions.

- (a) Before the deciduous tooth buds - Pre deciduous
- (b) Between deciduous and permanent tooth buds
- (c) After permanent tooth buds - Post permanent

The predeciduous teeth, seen in the mandibular incisal region at birth or soon after, are called natal and neonatal teeth respectively (Massler and Savara, 1950). These are horny structures with little or no root formation, and are usually shed during the first few months of life. Bodenhoff and Gorlin (1964) state that only ten percent of these teeth are supernumary.

The post-permanent dentition, however, has no apparent clinical distinctions that are additional to teeth of the normal series. Adelstein (1943), Oehlers (1952) and Cowan (1952)

report partially developed supernumary premolars after the normal premolars have completed their root formation.

Blumenthal (1934) describes the radiographs of a case which showed the permanent premolar teeth below the deciduous molar teeth and immediately below these were developing two more premolar teeth.

Morgan, Poyton and Crouch (1959) reported the unusual case of recurring mandibular bicuspid. Three supplemental mandibular bicuspid were removed at the age of eleven years. These teeth re-occurred and were removed at the age of sixteen years. In addition, two maxillary left impacted supplemental bicuspid, not present at eleven years of age, had appeared by sixteen years.

Flint (1939) states that if the proliferations of the epithelial cord are normal in number we may expect the dentition to develop in a normal manner, but we have embryological evidence that abnormal proliferations do occur.

Fastlicht (1943) cites the embryological studies of Bolk (1909) which relate that the epithelial cord, after having generated the enamel organs of the permanent teeth, normally disappears, but it may happen that its resorption is not complete and the epithelial remnants that persist may cause the development of the dental papilla of a future supernumary tooth.

Hemley (1944) in his text 'Fundamentals of Occlusion', illustrates a case of a supernumary lateral incisor in a dental occlusion which he considered demonstrated an 'individual normal condition'.

Heredity is not without importance in regard to the frequency of this anomaly and has been put forward as a further explanation for the presence of supernumary teeth. Levine (1962) states that the interaction of transmitted genes is a suggested cause but, although many supernumary teeth appear to be congenital in origin, others do not show a familial pattern. Flint (1939) suggests that enough case histories have been recorded to show that heredity undoubtedly plays some part in their presence, but investigators have found it quite difficult to trace supernumary teeth in ancestors, perhaps because their existence has been unknown or unnoticed.

Stafne (1931) in his rather full study of supernumary teeth states "a sufficient number of persons gave histories of the same abnormality having been seen in other members of their families to corroborate the belief that it has a hereditary tendency to occur."

Finn (1957) reported the presence of a supernumary lateral incisor in fourteen members of a family in two generations.

Fastlicht (1943) however, cites the example of a brother and sister both having supernumary teeth, but neither parent has suffered from the anomaly.

Bellinghausen (1956) reports the occurrence of more than thirty supernumary teeth in one patient. An examination of this patient's mother revealed eight unerupted supernumary teeth in an apparently edentulous mouth. Bellinghausen states "this seems to furnish proof that cumulative or intermediate heredity causes the occurrence of supernumary teeth; the ultimate cause, however, still is an enigma".

Keeler (1935) presented two pedigrees of the occurrence of a palatal supernumary tooth. The first showed the anomaly present in a woman, her daughter and her two brothers. In the second, which was more extensive, four members over three generations were affected. Both pedigrees show that the defect is inherited. Keeler states that it is of a dominant unit character.

A further theory is that the tooth germ may undergo dichotomy (Tannenbaum and Alling, 1963). If this division is equal the result is a supplemental tooth resembling the normal series, but if unequal, one tooth may be conical or rudimentary in form.

Levitas (1965) questions whether these 'twin' teeth, which Tannenbaum and Alling (1963) state result from equal division of the tooth bud, come from this division or whether there was

a twin bud from the beginning. Saarenmaa (1951) discussed this theory in his thesis 'Studies of Erupted Supernumary Teeth' and stated that this seems possible only where there is atrophy of the adjacent teeth and the supernumary tooth is small. He states "The most probable explanation appears to be that supernumary teeth in all parts of the dental arch are the result of proliferation of the dental lamina and arise from independent dental elements, judging from the fact that they are often of normal size as compared with the adjoining teeth and form their frequently normal shape. These seem to be part of the dentition with which their eruption coincides in time."

Shafter, Hine and Levy (1958, p.36) state that it is of interest, and as yet unexplained, that approximately 90% of all supernumary teeth occur in the maxilla.

Levine (1962) has suggested that it would seem possible that in a patient with a history of trauma in this region (maxilla), during the growth period, a tooth bud could be divided. He states that, regardless of the precipitating cause, supernumary teeth result from hyper-activity of the dental lamina.

Supernumary teeth are also found in association with general disturbances. Thoma and Goldman (1960, p.39) state "The occurrence of supernumary teeth may be merely due to hyper-activity of the dental lamina. This may be associated with

with hyperplasia of other epithelial structures, just as in anodontia the remaining products of ectodermal origin are underdeveloped or completely suppressed." They report the case of a twenty-year-old girl with cleido-cranial dysostosis and scoliosis of the spine. She had an exuberant growth of blond hair of a coarse texture, rapidly growing fingernails and a large number of teeth. Although many deciduous teeth were still present, x-rays showed at least forty-five permanent ones.

Hodgin (1963) reported a case where twenty-eight unerupted supernumary teeth were found in a twenty-eight-year-old patient with cleido-cranial dysostosis.

Chips (1951) has also reported a higher percentage of supernumary teeth in cleido-cranial dysostosis.

Paul (1967) reported that patients with alveolar clefts also showed a greater percentage of supernumary teeth.

In 1932 Stafne presented the most complete report on supernumary teeth to that time, the following is a tabulation of his results of the supernumary teeth found in 48,550 persons.

TABULATION AND NUMBER OF SUPERNUMARY TEETH IN EACH REGION

	Central Incisors	Lateral Incisors	Canines	Pre- molars	Para molars	Fourth molars	Total
Max- illa	227	19	2	9	58	131	446
Mand- ible	10	0	1	33	0	10	54

Stafne found 441 patients with 500 supernumary teeth resulting in an incidence of 0.91%. He believes that as the average age of these patients was forty years, then supernumary teeth were probably more numerous.

Dolder (1937) found an incidence of 0.6% in 10,000 Swiss school children. These results are considerably lower than most other studies have shown. Clayton (1956) and Lund (1959) reported 2.25% and 3.6% respectively.

Castaldi et al. (1966) studies a group of 457 children ranging in age from six to nine years. They found in fourteen children a total of sixteen supernumary teeth - 3.06%; six of them between the maxillary central incisors. Of these, called mesiodens by Dr. Bolk, three were erupting normally and three were inverted. In his report Stafne (1932) states that more than half of these teeth, mesiodens, were inverted.

In 1955 Menczer reported an incidence of .23% for supernumary teeth appearing in the deciduous dentition. Grahnen and Granath (1961) found a similar result of .3%. Of twenty supernumary teeth found in the deciduous dentition they observed sixteen cases carried through into the corresponding position in the permanent dentition.

Townend (1953) and Munro (1952) have also reported cases in which the duplication of deciduous laterals have been followed by the duplication of the permanent laterals.

2. Abnormality of Size

Megadontia and Microdontia

Megadontia and Microdontia are the terms used to denote teeth that are, respectively, unusually large or small (Farmer and Lawton, 1966 p.164) (Part I, p.15). These conditions can be general, or only one or two teeth may be affected. Worth (1963, p.80) states that megadontia or microdontia affects the entire dentition very rarely, more commonly only a few teeth are of abnormal size.

Pituitary dysfunction, provided it was present during the early stages of tooth development, would influence tooth size, as well as that of the skeletal structure. Schour and Massler (1943, p.603) observed that this rarely occurs, as the life-span of the cells has been fulfilled before the condition begins, the size of the tooth being determined at the time of outlining the dentino-enamel junction. Cohen and Wagner (1948) and Salzmann and Wein (1952) investigated the dental aspects of pituitary dwarfism and stated that the size of the teeth is not affected by the disease. However, the effect appears to be on those parts that are of mesodermal origin (Farmer and Lawton, 1966 p.164). Thus, in hypopituitarism or hyperpituitarism, the crowns of the teeth would appear normal while the roots were smaller or larger, respectively.

Disproportionate differences in size, between the teeth and jaws, are suggestive of cross inheritance - teeth from one parent and skeletal pattern from the other. Hyde (1938) observed that tooth size is markedly influenced by heredity. He indicated that large teeth may be inherited as a dominant characteristic. He also stated, in agreement with Hrdlicka (1935), that tooth size presents a great variation so that we are probably observing a blend rather than a single dominance. This would be especially true for people of mixed stock.

The twin studies of Horowitz, Osborne and De George (1958), and Korkhaus (1930) show the mesiodistal dimensions of the teeth to be influenced by hereditary factors.

Osborne, Horowitz and De George (1958) point out that, in addition to specific genetic factors which control the size of the individual teeth, there appears to be some generalised genetic regulation of tooth size that is common to adjacent teeth.

Butler (1939) suggested that a field of influence governing size and form existed in each of the tooth groups. It was his observation that morphologic variability became greater in the more distal teeth in each of the morphologic groups of maxillary incisors, cuspids, and molars and mandibular cuspids and molars. The mandibular incisors became less variable towards the distal.

Steinberg, Warren and Warren (1961) reported a family in which generalised microdontia occurred in three generations.

Boyle (1955, p.14) states that "While the fundamental pattern of tooth size and shape is determined by genes, this pattern may be modified by mechanical, hormonal or nutritional influence, or more commonly by infection in the developing tooth germ, e.g. Hutchinson incisor."

Regression or atavism may be the cause of rudimentary development of individual teeth which take on the cone or haplodont form of the reptile dentition. This abnormality is frequently inherited and occurs especially with the maxillary lateral incisors (Thoma and Goldman, 1960 p.94). Horowitz and Hixon (1966) state that these teeth show the highest genetic component of variability.

Microdontia may also occur when the dental epithelium is "too weak" to produce a normal tooth - clearly an indication of developmental suppression. These teeth also have a rudimentary cone appearance (Thoma and Allen, 1940).

Brown (1944) presented a case in which the roots of the tooth, in particular, were underdeveloped. The patient's father showed a similar condition, whilst his uncle had total anodontia. This pointed to a marked hereditary weakness in

the odontogenic epithelium.

Worth (1963, p.80) reported the rare occurrence of one very large tooth - maxillary central incisor - with all other teeth of normal size. Whether there has been complete fusion with a supernumary or whether the tooth developed from one dental germ is not known.

3. Abnormality of Shape

a. Geminatio and Fusion

These anomalies will be reviewed together because of the similarity of their clinical picture, and due to the fact that some authors do not distinguish between them.

Heslop (1954), and Burley and Reynolds (1965) have referred to the subject of the conjoined teeth as a "little confusing".

Levitas (1965) refers to this as a "typical British attitude" but states that "as so frequently happens in the use of the jargon of dentistry, there appears to be a number of definitions for almost any condition described".

Thoma and Goldman (1960, p.80) distinguish between gemination and fusion, and discuss them under these headings.

Farmer and Lawton (1966, p.915) on the other hand, recognise them as dichotomy and fusion - two processes of maldevelopment that have been put forward to explain the formation of the geminated composite odontoma.

Worth (1963, p.430) stated of the terms gemination and geminated composite odontoma : "These two terms appear to be used interchangeably or if there is any difference between them, it is difficult to determine. It is reasonable that

the simple developmental fusion of two teeth, with no other gross deformity, should be regarded as gemination, while the addition of some more or less mishape of one or both of the components may be termed geminated composite odontoma."

Tannenbaum and Alling (1963) stated that twinning, which for them meant complete dichotomy, has been used as a synonym for gemination, and fusion has been called false gemination.

Levitas (1965) doubts the merit of including twinning in a discussion of gemination and states "One may wonder whether these extra teeth came from the division of a tooth bud or whether there was a twin tooth bud from the beginning."

Flint (1939) states that we have embryological evidence that abnormal proliferations of the dental lamina do occur. The extent of their development is probably related to their position in the jaw. If located too closely to the normal germ, these may become fused in the process of their development and give rise to various kinds of fused, twin or partially divided teeth.

In 1957 de Jonge define two new terms - Schizodontia (literally - split tooth), "the term applied to geminate teeth which originate by a division of the tooth anlage into mesial and distal components", and Synodontia (literally - jointed tooth), "geminate teeth which are formed by a complete or partial inability of two adjacent dental germs to retain their identity."

Worth (1963, p.430) goes on to say "The nomenclature is not at all clear, but in view of the fact that it is very easy to determine the important features of the abnormality from clinical and radiographic examination, the name is not very significant."

It is agreed by most authors, that whatever the final terminology, there are two processes involved, viz. dichotomy (gemination), and fusion. The most popular descriptions for these are :

Gemination (Twinning) - This is an incomplete division of the tooth bud which is produced by an epithelial invagination into the enamel organ. This partial division is halted before development is completed and the result is a single tooth with a bifid crown and a single radicular pulp (Part I, p. 16).

Fusion - This is the process in which union occurs between two tooth germs during the process of tooth formation. In this condition there will probably be two separate root canals but only a single root (Part I, p. 16).

When gemination exists, there will be in the area concerned, a normal number of teeth if the bifid crown is counted as one. In fusion, however, it will appear that there is a congenitally missing tooth, unless the fusion occurred between a normal tooth and a supernumary tooth.

The aetiology of these anomalies is obscure. Moody and Montgomery (1934) state that at times heredity can be traced. They reported the occurrence of geminated deciduous lower lateral incisors in four generations of a family. Only females were affected and they transmitted the condition.

Kloeppel (1958) reported a similar condition over four generations.

Bier (1958) cited a case in which the parents had no history of such anomalies, but that the patient, his brother and sister had a "double tooth".

Hitchin and Morris (1961) reported that gemination occurs as an inherited characteristic in Lakeland terriers. In a study of serial sections they concluded that the primary anatomical anomaly was the persistence of the interdental lamina.

According to Menczer (1955) fusion is one of the three most common anomalies occurring in the deciduous dentition. The others being partial anodontia and supernumary teeth. In his examination of 2,209 pre-school children he found an incidence of 0.14%. However, he does not specify whether the heading 'fusion' involves geminated teeth or not.

Clayton (1956), with again no differentiation between fused and geminated teeth, found an incidence of 0.47%.

Plaetschke (1938) examined 1,000 pre-school children and found fusion in four cases (0.4%) and gemination in one (0.1%).

Grahnen and Granath (1961) used the term 'double teeth', as the genesis of fusion and gemination is unknown. They found an incidence of 0.5%. They found also that these anomalies were more common in deciduous dentition and occurred mainly in the premaxilla and anterior regions. They observed eight cases in the primary dentition and found that four followed-through into the permanent dentition.

Farmer and Lawton, (1966, p.917) state that in either dentition, fusion involving a supernumary tooth occurs most often in the maxillary incisor region.

Turkheim (1949) reports two cases of fusion of the deciduous maxillary incisor teeth. One between a central incisor and a supernumary tooth, and the other between central and lateral incisors.

Clem and Natkin (1966) presented the treatment of a permanent maxillary central incisor fused to a mesiodens.

Burley and Reynolds (1965) reported the occurrence of a supernumary tooth fused to the left deciduous maxillary central and lateral incisors in a four-year-old girl. Her two-and-a-half year old brother showed a similar condition without the supernumary tooth.

An earlier report of 'triple dental fusion' was by Marques (1957). This was in a young boy where three lower deciduous incisors had almost completely fused, but each 'tooth' had retained its own well-formed root canal.

In 1963 Stafne reported a rare anomaly which he called "fusion of mandibular incisors with dens-in-dente".

In the permanent dentition fusion is also observed in the molar region. Heslop (1954) presented a short review of gemination and fusion, and reported the fusion of a lower third molar tooth with a supernumary tooth. This is not to be confused with concrescence, which Bier (1958) describes as a "pathological entity found only after dental development has been completed". He described fusion and gemination as physiological processes which occur during the developmental stage.

In 1948 Boucher reported the rare finding of fusion occurring twice in the same dentition. This was observed between the mandibular left central and lateral incisors and the mandibular left cuspid and first premolar. This case was most unusual when one notes the infrequency with which premolars are involved.

b. Dilaceration

Dilaceration is an angulation or sharp bend or curve in the root or crown of the formed tooth that results from an injury during its development (Part I, p. 21).

Shafer, Hine and Levy (1961, p.29) state that the curve or bend may occur anywhere along the length of the tooth, sometimes at the cervical portion, and at other times midway along the root, or even just at the apex of the root depending on the amount of root formed when injury occurred.

Frey (1966) presented a case in which the maxillary central incisor was dilacerated. He stated that this was caused "by trauma at an early age."

Atkinson (1966), in his paper titled "Jaws Out of Balance", stated

"If the developing crown or root of a tooth should encounter an obstacle in the path of its growth, it would not attempt to force its way. It would blindly follow the line of least resistance, departing from its inherited pattern by growing around the obstacle and thereby becoming permanently deformed."

He cited the case of a malformed crown of a tooth caused by the tooth germ developing within a loop in a piece of gold wire.

Thoma and Goldman (1960, p.96) state that the partly or completely erupted tooth may be forced into a different location than originally intended, either by pressure exerted by the crowding of a neighbouring tooth or by orthodontic means. If the root is not completely formed then the uncalcified part will bend. Atkinson (1966) presents examples of dilaceration caused by orthodontic movement and states that the angle of distortion depends on the rapidity of movement.

Townend (1952) presented an interesting case where a three-and-a-half year old child had caused dilaceration of both maxillary central incisors by thumb sucking.

Kronfeld (1956) was more specific in his definition stating that dilaceration, partial fracture, is a deformity that consists of a bend or crease at the junction between the crown and the root. The cause for this is a pre-eruptive injury, usually a blow or fall that severely jars the deciduous tooth and is transmitted to the crown of the underlying permanent tooth.

Castaldi (1959) stated that, although traumatic injury to unerupted incisors following a blow to the primary tooth is not a common occurrence neither is the condition extremely rare. Castaldi reported a case where a child of two years received a blow from a swing. The gross appearance of the tooth suggests that the incisal half of the crown was displaced palatally by approximately ninety degrees. They

stated that this appears to be the most common displacement pattern for the maxillary incisors. For the mandibular incisors this pattern is reversed in that the incisal part of the crown is displaced labially. This agrees with Rushton (1958) who showed "abrupt palatal angulation" on the part of the crown already formed. He showed that partial duplication of the affected tooth may be the result of mechanical injury.

Levine (1962) has suggested that complete division of the tooth under these circumstances could give rise to the formation of supernumary teeth.

The literature revealed no documented cases of dilaceration due to cleft conditions. Thoma and Goldman (1960, p.96) stated "In Brophy's method of cleft palate operation the teeth were frequently injured when wires were passed through the alveolar bone. Operative injury is more common in the region of the alveolar cleft and is often associated with the maldevelopment of the canine and second teeth."

Bohn (1963) in his report "Dental Anomalies in Harelip and Cleft Palate", showed many figures of teeth affected by dilaceration.

c. Odontomes

Odontomes are tumors composed of one or more of the various elements that make up the tooth structure (Olech, 1963).

They are caused by neoplastic proliferation of the odontogenic cells of the tooth germ (Thoma and Goldman, 1960, p.1221) (Part I, p.6,10).

The term 'odontome' appears to have been first applied to these abnormalities by Paul Broca in 1867. Sprawson (1937, p.177) cited him as describing them as tumors formed by the overgrowth of transitory or complete dental tissues. Broca classified odontomes according to the period of tooth formation at which the abnormal growth took place.

Shafer, Hine and Levy (1961, p.199) state that the term 'odontoma' has come to mean a growth in which both epithelial and mesenchymal cells exhibit complete differentiation with the result that functional ameloblasts and odontoblasts form enamel and dentine. The enamel and dentine are usually laid down in an abnormal pattern because the organisation of the odontogenic cells fails to reach a normal state of morpho-differentiation.

Worth (1963, p.420) states it is relatively rare for only one of the calcified tissues to be present and, when this does

occur, the probability is that enamel or cementum make up the lesion. Odontomes consisting of dentine only are extremely rare.

The Complex Composite Odontome is a conglomerate mass of enamel, dentine and cementum in no ordered arrangement, with nutrient canals entering it and containing disordered pulp tissue (Hitchin, 1962). It may be smooth, lobulated or of irregular shape (Northrop, 1963; Vianna, 1966). This is a fairly rare condition (Farmer and Lawton, 1966 p.918).

Thoma and Goldman (1960, p.1221) state that it is produced by a neoplastic development of the tooth germ of a tooth from the normal series or from a supernumary sprout given off the dental lamina.

Farmer and Lawton (1966, p.918) state that the cause of the abnormality is unknown, but it is probably the most closely allied of any of the composite odontomes to a neoplastic condition. This agrees with Sprawson (1937, p.197) who also believes that even though nearly all composite odontomes are developmental in origin, trauma and infection may also produce them by interfering with the co-ordination of normal growth.

Tratman (1949) states that they have potentially unlimited growth but that they do not invade the tissues and so must be regarded as true benign tumors.

Hitchin (1962) states that the calcified composite odontomes are the end results of abnormal developmental processes. As yet little is known about these processes and so diagnosis tends to be purely morphological. He has reviewed the present knowledge of their causation and advanced the simple hypothesis that "the calcified composite odontomes are inherited or due to a disturbance or change in the genetic control of tooth development."

Rushton (1936) suggested that the complex composite odontome is formed in a similar manner to the dilated composite odontomes by a single, but branching, invagination. He termed this invagination 'adenoma'. Rushton stated that in the dilated composite odontome the invagination takes place at the bell stage but in the case of the complex composite odontome the abnormality may arise in the tooth band prior to the bud or germ stage.

Tratman (1949) believes that the development begins later than suggested by Rushton because enamel is formed. This necessitates the stage of differentiation of the internal enamel epithelium being reached (cf. Part I). Tratman agrees with Rushton that the formation is by invagination but he suggests multiple instead of a single branching adenoma. These develop on the internal surface of the bell and completely disrupt its normal configuration. These invaginations push their way into the underlying mesodermal tissue, which in turn becomes

split up into a series of papillae. The usual metaplasia occurs and calcification commences.

Vianna (1966) states that the disturbance may affect a single tooth germ or it may result from an excessive focal overgrowth of the dental lamina. In the latter instance, multiple small tooth buds are united to form an irregular conglomeration of all dental tissues.

A contributing factor to the cessation of enamel formation is the gradual restriction of space occupied by the adenoma as more enamel is formed. This impairs the nutrition and thus tends to hasten the beginning of the atrophic changes (Tratman, 1949). Tratman (1949) states that encapsulation appears to play an important part in bringing about cessation of growth. As the odontome grows a sac appears, the anlage of the normal tooth sac, and grows around it. If the sac is sufficiently thick it will tend to reduce the blood supply to, and hence the nutrition of, the interior of the odontome.

In the Compound Composite Odontome the organisation of the calcified dental tissues is more advanced, so that definite small denticles or tooth-like structures are formed in a stroma of fibrous connective tissue. These denticles mature with the dentition (Blackwood, 1965).

Thoma and Goldman (1960, p.1222) state that the compound composite odontome is formed if the normal enamel epithelium produces

many small enamel organs, all of which develop into tooth germs and give rise to all kinds and shapes of small teeth.

Farmer and Lawton (1966, p.920) present two theories, the first of which is similar to that put forward by Thoma and Goldman. The second, in agreement with Tratman, is that the odontome is formed as the result of the tooth germ undergoing repeated dichotomy. This germ may be either of the normal series or an additional one.

Blackwood (1965) states that, like the complex odontome, the compound form represents an error in the development of the dental tissues and is, therefore, not a true neoplasm.

Farmer and Lawton (1966, p.922) state that cases occur in which there is apparently a mineralised mass together with several denticles, that are separate, so that it is difficult to decide whether it should be classified as a complex or compound composite odontome. Tratman (1949) distinguishes between the two by the arrangement of the calcified tissues. He states "even when a compound composite odontome is formed by a single irregular mass of calcified tissue an examination will show that the mass is essentially composed of a series of small irregular denticles in which the dichotomous process has not proceeded to completion, and hence in which the denticles are still fused together, and in which the tissues are arranged in the normal manner with no invaginations. The external and internal enamel

epithelium are both involved, whereas in the complex composite odontomes it is essentially only the internal enamel epithelium that is involved in a series of buds growing into the mesodermic tissue and there is no dichotomous process".

Northrop (1963) disagrees with Tratman. If the tumor combines both the formation of the rudimentary teeth with a limiting capsule and the formation of a conglomerate mass of dental tissue, then whichever predominates determines the classification.

Henriksson and Kjellman (1964) discuss Husted and Pindborg (1953) who distinguished between ameloblastic, complex and composite odontomes. The ameloblastic odontome presents, in the periphery, a soft tissue consisting of an odontogenic epithelium and an embryonic connective tissue. In the central part all the stages of predentine, dentine, enamel matrix and enamel may be found. Their complex and composite odontomes are the complex composite and compound composite odontomes respectively, which were defined earlier. Husted and Pindborg considered the ameloblastic odontome as the first stage of a development which may lead to either the composite or compound formation. Henriksson and Kjellman, who use this classification, presume it to be supported by the fact that ameloblastic odontomes often occur in children between the ages of one to four years.

Eli Olech (1963) suggests that the odontome represents the inactive or mature form of the odontoblastoma; it is the end result, the calcified remains, after the odontogenic activity of the cells becomes exhausted. This agrees with Thoma and Goldman (1960) who regard the odontome, in its early formative stage as the odontoblastoma made up of neoplastic epithelial and mesenchymal cells which are in the process of differentiating into cells able to produce calcified tooth substances, enamel, dentine and cementum. These tissues are laid down in an abnormal arrangement.

Hitchin (1962) stated that in the early stages of calcification it is impossible by radiography, and even on section, to diagnose differentially between complex and compound composite odontomes. Indeed it is possible for the latter to become extremely similar to, if not actually, the former by deposition of cementum. Hitchin, who defines the compound composite odontome as "consisting of more than one supernumary denticle, mass or particle of hard dental tissues in close relation to each other", divides the structure on morphological grounds into :

1. A denticular type composed of two or more separate denticles.
2. A particulate type composed of two or more separate masses of hard dental tissue having no macroscopic resemblance to a tooth in form.
3. A denticulo-particulate type, in which denticles and conglomerate masses or particles are present side by side.

The clinical picture for the composite odontomes is almost identical. They are usually observed first during childhood or in the young adult. Often the only indication, apart from routine radiographic examination, is the absence of a tooth from the arch. In a small percentage of patients, however, they grow large enough to expand the jaws (Hitchin, 1962). Occasionally they form around the crown of a normally developed unerupted tooth (Henriksson and Kjellman, 1964). Sprawson (1937) states that in some cases an odontome may erupt and become secondarily infected, causing clinical symptoms of pericoronitis, sometimes with complications, similar to those found in ostitis, with formation of sequestra and fistulas. However, such cases are rare.

The complex composite odontome is most frequently found in the molar region (Farmer and Lawton, 1966 p.918; Vianna, 1966), though it also develops in any part of the maxilla and mandible. The compound composite odontome is most frequently found in the anterior part of either jaw (Farmer and Lawton, 1966 p.920). In discussing odontomes in general, Henriksson and Kjellman (1964) state that they are most frequently found in the maxilla.

The compound composite odontomes are not common and are particularly rare in association with the deciduous dentition (Hitchin 1962). Hitchin (1962) presented two such cases. From a child of five years of age six denticles were removed. Their incomplete root formation suggested, in reality, that it was

associated with the permanent dentition. The second was a patient of eight years of age in whom the odontome caused the impaction of both the deciduous and permanent cuspids. For this to occur, it is reasonable to suppose that the odontome was related to the deciduous dentition.

Tratman (1949) states that "no references have been found of these odontomes (complex composite) occurring in connection with the deciduous dentition, though admittedly, published specimen histories are not always explicit as to the dentition from which the specimen has been derived. The majority of specimens have been recorded as coming from areas of the jaws posterior to those occupied by the deciduous dentition and it would appear that the deciduous dentition is not prone to the particular type of aberration which produces these odontomes."

The incidence of the complex and compound composite odontomes has been described as "fairly rare" and "comparatively rare" respectively (Farmer and Lawton, 1966 p.917, 920).

Blackwood (1965) states that in his experience their incidence is about equal. This is in agreement with Gorlin and Chaudhry's (1961) findings, although a review of the literature by Gorlin et al (1961) revealed about twice as many reported cases of the compound variety to the complex form.

Vianna (1966) reports that males and females are about equally affected.

Gestant Odontome

The Gestant Odontome or "dens-in-dente" as it has been commonly called, is a developmental variation which is thought to arise as the result of an invagination in the tooth crown before calcification has occurred (Shafer, Hine and Levy, 1958, p.29)(Part I, p. 15).

Hunter (1951) submitted a report "to question the advisability of perpetuating an erroneous concept by continuing to use the common name for this condition". He supported Rushton (1937), and discussed the formation under the heading 'Dilated Composite Odontome'.

Thoma and Goldman (1960, p.85, 1222) discuss dens-in-dente and dilated composite odontome as two separate entities.

Farmer and Lawton (1966, p.923) call the anomalies 'Invaginated Odontomes'.

Worth (1963, p.433) states that such a wide variation in appearances of the various conditions are embraced by the above terms that it may be thought that they are not synonymous.

The term 'dens-in-dente', meaning 'tooth within a tooth', was first suggested by Busch in 1897 (Hunter, 1951); Busch believed that this phenomenon resulted because one tooth surrounded another by the process of growing around it. This supposes that two

tooth germs are involved, but the concept is no longer accepted by most investigators, even though the terminology is retained.

There are two theories concerning the aetiology of this anomaly. In the first, developed by Kronfeld (1934), it is thought that the anomaly represents a retardation at one or more points of growth of the tooth germ whose growth is regarded as centrifugal in nature. This theory was elaborated on by Kitchen (1935).

Swanson and McCarthy (1947) disagree with this theory. They stated that assuming Kronfeld to be correct, the inner enamel of the invaginations should end at the cemento-enamel junction of the outer tooth. This does not seem to be accurate as the greater majority of sectioned teeth reported present evidence of the inner enamel extending a considerable distance apically below the cemento-enamel junction. Swanson and McCarthy believe dens-in-dente to be a proliferation of cells causing an ingrowth of the enamel organ apically into the dental papilla. They state that the proliferation into the dental papilla occurs during the stage of differentiation of the developing tooth germ at the inner enamel epithelium. This agrees with Rushton (1936) who stated that the development occurred about the time dentine formation began. He believed that this growth process presses the incompletely calcified dentine walls and papilla outwards, so that they are thinned and the deeper part of the

bony crypt becomes enlarged. The part already calcified cannot be expanded, so, in cases where the pressure is great, the tooth becomes apically wider from the cusp rootwards. The central pressure must force the crown in a superficial direction and may help to cause its conical shape. In his paper, Rushton describes nineteen specimens occurring in both molar and incisal crowns and those presumed to have been invaginated from Hertwig's Sheath.

Rabinowitch (1949) suggests that the formation is due to the continued differentiation of some cells of the inner enamel epithelium within the developing tooth remaining attached to the developing tooth, somewhat similar to a tumor mass. This may be compared to remnants of the dental lamina which sometimes continue differentiation outside the tooth forming a supernumary tooth.

The abnormal proliferation, after a time, gradually ceases. Rushton (1937) suggests that this is chiefly due to the proliferation having a limited life activity, but that it may be due to the fact that its maximum blood supply was fixed from the beginning by the calcified ring around the pedicle.

A variation, closely aligned to, and often classified with, dens-in-dente, is the invagination from the lingual pit of the maxillary central and lateral incisors (Hallet, 1953). This will be discussed with dens-in-dente because many surveys carried out do not distinguish between them.

Hallett (1953) differentiated in classification between those invaginations arising from the foramen caecum or palatal pit of the incisors and those arising from the incisal edge. He suggested the term 'invagination of the enamel' for those arising from the lingual pit.

Worth (1963, p.83), as the result of studying a large number of these conditions, agrees with Hallett.

Colyer (1926) suggests that all invaginations are caused by the same basic phenomenon, the different forms being due to varying degrees of affection only.

Atkinson (1943) considers the lack of development of the jaws and the constriction of the arches as a possible cause. In discussing the development of the permanent lateral incisor he shows that the environment of the developing tooth has a direct bearing. Constriction of the forming crown is caused by a restriction on either side, often causing a pit to be formed on the lingual. As the root continues to grow it closes over the pit at about the cingulum, opening up again just below the height of the labio-lingual extreme width. These true invaginations are at times lined with enamel but at other times the walls are just bare dentine.

Hallett (1953) summarised the various theories put forward for the aetiology of invaginations, but states that it is still not understood. He notes that it is not possible to extract any

figures supporting or denying the recurrence of invagination on Mendelian principles of inheritance, as most people are unconscious of their existence.

Present-day theory suggests that this particular condition results from an invagination of a single enamel organ.

The incidence of these anomalies has been reported ranging from 0.25% (Poyton and Morgan, 1966) to 10% (Atkinson, 1943). In the former, Poyton and Morgan studied 5,000 x-rays and restricted their examination to the maxillary incisors, cuspids, premolars and first permanent molars. Their classification of true dens-in-dente included those teeth which displayed an enamel form within the crown (coronal type), or in the root (radicular type or dilated odontome). They reported that the formation was confined to the anterior teeth. Three cases presented with bilateral conditions.

Atkinson, on the other hand, found an incidence of 10% in maxillary lateral incisors, but he did not define his requirements of dens-in-dente.

4. Abnormality of Structure

a. Aplasia of the Enamel

Aplasia of the enamel is a rare inherited disease of tooth formation, brought about by the failure of the enamel rods to form.

Fish (1948) states that the dentine appears to develop normally and that the teeth may be normal in length and size. Bernier (1959, p.80) however, has found that the teeth associated with aplasia of the enamel are deformed. They may be peg-shaped or rounded and usually have a distinct yellowish-brown tinge. Holder (1928) described a case of a four-year-old child in whom the enamel was missing from all the primary teeth. The anterior teeth were conical in shape; the posterior teeth were level with the gingiva, as a result of marked attrition, and had the appearance of glazed glass.

Worth (1963, p.84) states that, from the radiographic point of view, the only abnormality may be in the absence of the normal enamel caps.

Hopewell-Smith (1913) reported a case of a twenty-one year old girl in whom the crowns were absent and no enamel had formed. The teeth had the appearance of being filed off, but there was no appearance of attrition or abrasion. Other members of the family were similarly affected.

Colyer and Sprawson (1942) stated that in many cases the exposed dentine is unable to withstand the force of attrition and is rapidly worn down to the gingival margin.

Histologically there is complete absence of enamel structure, sclerosis of the dentine, and partial and complete occlusion of the pulp chamber and canal (Bernier, 1959 p.80).

Olson (1938) reported a pedigree of three generations in which seven of the fifteen members were affected. He presented the case of a twenty-year-old male in which there was enough dentine present to restore appearance with porcelain jacket crowns.

Bernier (1959 p.80) states that both sexes are affected about equally. He cites Miller (1946) who reported a case where the enamel was absent from all permanent and deciduous teeth in three generations of one family. The involved teeth showed no evidence of enamel formation. They are yellowish in appearance, stained, and in some instances are extremely sensitive to thermal changes.

Schimmelpfennig and McDonald (1955) reported the very rare condition of nearly complete aplasia of the enamel and dysplasia of the dentine. The pulp chambers were extremely large and showed no evidence of secondary dentine formation.

b. Amelogenesis Imperfecta

Generalised defective enamel formation (amelogenesis imperfecta) is a relatively uncommon condition which occurs much less frequently than disturbances in the enamel formation affecting only part of the dentition (Toller, 1959). Shafer, Hine and Levy (1961, p.38) call it a "poorly understood condition that has been described in the literature under a variety of terms including 'hereditary enamel hypoplasia', 'hereditary brown enamel' and 'hereditary brown opalescent teeth' ".

Spokes (1890) gave the first account of, what he termed "faulty enamel" to the Odontological Society of London in 1890. He drew attention to the vertical grooves in the enamel of one member of the family whom he had examined. He stated that

"In ordinary cases of defective enamel we are accustomed to see transverse grooves across the teeth marking the period at which something occurred to interfere with the due formation of this tissue, but in the present case there are grooves in the long axis of the tooth, showing that the process was modified throughout the whole of the time occupied by calcification. In some places there is a total absence of enamel, and the exposed surface of the dentine seems to have been sufficiently hard to survive without the protection of the enamel covering. In other places the enamel is laid on in irregular masses."

Colyer and Sprawson (1942) classified this case as one of hereditary hypoplasia.

Rushton (1964) agreed with Colyer and Sprawson, and in discussing Spoke's case stated "this grooving in the long axis of the tooth is something which we can recognise as highly characteristic of, if not peculiar to, a certain variety of hereditary enamel hypoplasia as seen in affected females."

In 1938 Finn reviewed the literature of forty-one cases of hereditary discolouration of the teeth. He found much of the information lacking, but was still able to differentiate two groups. He classified one group as hereditary opalescent dentine and the other as brown hyperplasia of the enamel. Of the forty-one cases reviewed, twenty-eight could be identified as the former and only three as the latter. Ten cases lacked differential characteristics.

Cockayne (1933) describes hereditary hypoplasia in part, as follows : "The enamel may be entirely absent or it may be laid on quite irregularly, or there may be deep longitudinal grooves where the enamel is very thin, but nowhere is it as thick as the normal tooth. The teeth are often brown when first erupted, but the colour always deepens afterward and the most pigmented parts are those where the enamel is thinnest, and at these points it is almost structureless."

Weinmann, Svoboda and Woods (1945) divide amelogenesis imperfecta into two types - enamel hypoplasia and enamel hypocalcification. The difference is thought to be due to the stage of enamel development at which the defect occurs. In enamel hypoplasia the enamel matrix appears to be imperfectly formed and, although calcification subsequently occurs in the matrix so that the enamel is hard, it is deficient in amount and very irregular in deposition. In enamel hypocalcification matrix formation appears to be normal so that the enamel is normal in thickness, but calcification is deficient and the enamel is soft. They state that these two types of disturbance of enamel development can be readily correlated with the two phases in normal development - enamel matrix formation and enamel calcification (Part I, p. 17). The disturbance being more severe in enamel hypoplasia than in enamel hypocalcification.

Robinson (1946) in his definition of hypoplasia made a similar distinction between these two phases of enamel formation.

Darling (1956), who does not support the process of maturation as described by Weinmann et al, states that in hypomineralisation the line of junction between the mature and immature enamel usually runs roughly parallel to the enamel surface. He also states that hypoplasia and hypomineralisation can occur together. Darling noted five morphological and clinical varieties :

1. General pitting of the enamel

2. Vertical grooving and wrinkling
3. Marked deficiency in the enamel thickness and resorption of unerupted teeth
- 4.) Defective calcification differing in degree
- 5.) and distribution

Rushton (1964) agrees with Darling in that the hereditary hypoplasia and hereditary hypocalcification may be present concurrently. He states that there must be more than two genetic types.

Keeler in 1935 presented pedigrees of 'soft brown teeth lacking enamel' in which he reviews sixty-two matings between normal and affected persons. These matings produced eighty-seven normal children and one hundred and nineteen affected children. He states that this character is an orthodox unit dominant, affected individuals having one affected parent.

Weinmann et al (1945) report a family in which three generations were affected. Camerson and Bradford (1957), on the other hand, report a case in which three of five brothers were affected even though parents and grandparents showed no evidence of the disorder.

Rushton (1964) discusses hereditary enamel hypoplasia in two separate groups,

1. sex-linked dominant type
2. autosomal dominant type with hard enamel or random pitting.

In the former, he states that the abnormal gene is presumed to be dominant (or more properly, intermediate). The effect of this is that an affected heterozygous mother will be expected to hand on the abnormality to half her sons and half her daughters, but the affected father, with a normal wife, will hand it on to all of his daughters and none of his sons or their offspring. A further interesting feature in this sort of hypoplasia is that males and females are differently affected. The males have only a very thin smooth layer of enamel which appears nearly homogeneous and lacks the usual rod structure; but females have enamel which is in parts much thicker and in many parts prismatic. Macroscopically the girls' teeth show a remarkable pattern of vertical grooves separating vertical stripes of better enamel.

Under the second heading, Rushton discusses a family reported by Weinmann et al. (1945), who showed no differences in manifestation between males and females, and where the pedigree was consistent with autosomal dominant. Moreover, in this case the very thin smooth enamel showed normal structural elements and many teeth failed to erupt. Vertical markings were not evident. Rushton, in discussing random pitting in the permanent dentition but with smooth hard enamel in the deciduous dentition, states that in these types one must suppose that the pathological gene either operates in all the ameloblasts or in a randomly scattered proportion of them, unrelated to groups of stem cells from which the ameloblasts are derived. Its effect is to limit the thick-

ness of prismatic enamel at all or some parts of the surface with the production of a thick layer of a glassy substitute.

Witkop (1962) showed that generalised enamel defects may be attributed to both genetic and environmental factors. He describes five distinct hereditary diseases of the enamel, without generalised disease, in which both permanent and deciduous dentitions are involved.

1. hereditary hypoplasia - sex-linked dominant
2. hereditary hypocalcification - autosomal dominant
3. hereditary hypomaturation - sex-linked recessive
4. hereditary pigmented hypomaturation - probably an autosomal recessive
5. hereditary local (pitted) hypoplasia - autosomal dominant with incomplete penetrance.

Witkop lists epidermolysis bullosa dystrophica, various forms of vitamin D-resistant rickets and erythroblastosis fetalis which may have associated generalised defects of the deciduous dentition. Rh factor and A-O blood group incompatibility also may show defects of the permanent anterior teeth and the first molars. Environmental amelogenesis imperfecta affecting deciduous teeth may be the result of severe febrile disease in intra-uterine or early post-natal life. Usually, however, only selected teeth are defective. He suggests the hereditary amelogenesis imperfecta occurs about once in 4,000 children.

Rushton (1964), in discussion of Witkop's classification, states that he has not observed the two special types distinguished as hypomaturation.

Farmer and Lawton describe the anomaly under the headings :

1. Hereditary enamel hypoplasia (Farmer and Lawton, 1966 p.183)
2. Hereditary and idiopathic enamel hypomineralisation (Farmer and Lawton, 1966 p.187)

and describe the following clinical features :

Hereditary enamel hypoplasia : Much of the enamel is missing but any remaining portions are hard. In most cases the teeth are affected with severe attrition until they are worn level with the gums and are usually discoloured dark brown or yellow. Sometimes the teeth have a semi-transparent appearance due to mineralisation of the dentinal tubules.

Hereditary idiopathic enamel hypomineralisation : The teeth usually show confluent pitting or there are areas of complete loss of enamel. The latter may have a consistency of chalk and in a typical case is so soft that it can be easily scraped away. The colour of the enamel may be opaque white or discoloured to a yellowish tinge. Due to the loss of enamel, the teeth have not the normal contour and may appear cylindrical. Large areas of dentine are exposed and their colour varies from orange-brown to brown, and there is marked attrition.

c. General Developmental Defects

Hyperplastic defects are produced by rickets, tetany, exanthematous fevers and other acute childhood diseases, especially those of the digestive and respiratory systems (Thoma and Goldman, 1960 p.113). This results in localised developmental defects of the tooth and relates to a decreased or complete lack of, function of the ameloblasts in a given area. The affected cells do not recover, and, although adjacent cells take over their activity, a defective area remains (Bernier, 1959 p.77).

The clinical picture varies widely. In mild cases the enamel is intact except for several shallow depressions or grooves on an otherwise smooth enamel surface. In more extensive cases the grooves or pits are arranged in horizontal rows around the crown extending into the enamel as far as the dentino-enamel junction. In severe cases the enamel on the incisal edge of the cuspids and the incisors is lacking, and the dentine on the occlusal surface of the first permanent molars is exposed.

The enamel of several teeth is usually involved, the defects being symmetrically distributed in each tooth at a place corresponding to the stage of dental development when the disturbance occurred.

A type of hypoplasia is occasionally seen which is unusual in that only a single tooth is involved. Called 'Turner's hypoplasia' (Shafer, Hine and Levy, 1958 p.44), it affects the permanent teeth and is the result of trauma or periapical infection of the deciduous tooth during the formation of the crown of the permanent successor. The severity of this hypoplasia will depend of the severity of the infection, the degree of tissue involvement and disturbance and the stage of permanent tooth formation during which the disturbance occurred.

In general hypoplasia, the permanent dentition is more often involved than the deciduous one. The enamel formation of the deciduous dentition commences before birth and consequently can be divided into an inner pre-natal enamel and an outer post-natal enamel. The dividing plane between these two layers is visible in most deciduous teeth as an area of hypoplasia. This has been described by Schour (1936) as the neonatal ring. Generally the enamel formation prior to this ring or line is undisturbed and regular. Rarely, a severe disturbance in the mother during pregnancy may be reflected in hypoplastic defects in the deciduous teeth then in the process of formation. De Wilde (1943) describes such a case in which a syphilitic mother reacted severely to treatment during pregnancy. The child was born at term and upon eruption the incisal area of the deciduous teeth was found to be hypoplastic. The characteristic Hutchinsonian incisors were demonstrated by x-ray examination of the unerupted maxillary central incisors.

Schour and Kronfeld (1938) describe the dentition of a full term infant who received a severe brain injury at birth. Most of the ameloblasts active at birth ceased to function, giving rise to a condition which Schour and Kronfeld termed "neonatal hypoplasia of the enamel and the dentine". Portions of the enamel organ, where the ameloblasts were not actively depositing enamel at birth, commenced to deposit enamel when the infant was approximately two months old.

Hypoplastic defects of the teeth in prematurely born infants were described by Stein (1947). These occurred as defective rings nearer the middle third of the incisors, and correspondingly in earlier stages than in normal development, of the other deciduous teeth. Forrester and Miller (1955) reported similar defects in normal premature infants. They also studied children who had survived kernicterus, these all showed hypoplasia in the deciduous dentition.

Kreshover and Clough (1958) observed that it was possible to correlate abnormalities of parturition and gestation with abnormalities of enamel formation. Kreshover (1944) has also artificially produced hypoplasia in rodents similar to that found in humans with a specific disease.

Varying observations have been made concerning the effect of rickets on the developing tooth. Farmer and Lawton (1966, p.83) describe the enamel of the teeth as hypoplastic with a rough and pitted surface, but state that recent investigations have thrown

doubt on whether rickets alone is responsible for these defects. Sarnat and Schour (1942) found a possible correlation between rickets and enamel formation but none between enamel hypoplasia and childhood diseases.

Weinmann and Schour (1945), discussing the effect of a rachitogenic diet on rats, state that enamel formation and mineralisation are apparently unaffected by vitamin D deficiency. The enamel organ shows no change during enamel formation, but on reaching the stage when it is beginning to atrophy, cystic degeneration may occur. Farmer and Lawton (1966 p.40) cite Sjoquist (1937) as suggesting that when enamel hypoplasia occurs with rickets it is due to some accompanying factor, such as hypothyroidism and tetany.

Hypoparathyroidism causes defectively formed enamel through the lowering of the serum calcium (Thoma and Goldman, 1960 p.113). Farmer and Lawton (1966 p.40) cite the experiments on rats of Schour, Chandler and Tweedy (1937) who reported that there is first a hypermineralisation and that this is subsequently followed by a deficiency of mineralisation and irregularly formed dentine, which shows occasional vascular inclusions.

Via and Churchill (1957) found a high correlation of enamel abnormalities in children with cerebral disorders. They have also stated (Via and Churchill, 1959) that the incidence of enamel hypoplasia in the normal population is nine percent.

Any disease causing prolonged fever early in childhood is likely to have a marked deleterious effect on enamel formation.

Measles, smallpox, diphtheria and scarlet fever, especially severe attacks, are generally considered to affect the highly specialised ameloblasts. The injury is probably due to the effect of toxin which acts on the enamel organ just as it affects the growing portion of the hair and nails (Thoma and Goldman, 1960 p.115). These produce well demarkated defects due to their sudden onset. Marshall (1936) reasons that the fact that not all children show these defects is due to the active and latent periods of tooth activity.

Children born to mothers who contracted severe exanthematous fevers during pregnancy may show dental defects. Evans (1937) reports the occurrence of hypoplasia in thirteen of sixty-seven children whose mothers had rubella during pregnancy.

Rattner and Myers (1962) examined forty-five children with congenital allergies, both clinically and roetgenographically, for enamel defects. They found twenty-six positive findings and timed the onset to the last trimester of gestation.

The cause of these defects in the enamel is associated with either matrix formation or mineralisation (Part I, p. 34). Gottlieb (1920) believed the defects to be due to the collapse of normally formed but poorly calcified enamel matrix, the atrophy of the ameloblasts being secondary. If the matrix

is mineralised soon after deposition then normal enamel structure is achieved. If, however, the normal deposition of calcium is delayed the matrix may collapse with the resultant defective formation of the enamel at this site.

Others believe that the defects are produced by the disturbance of the ameloblastic activity of the enamel organ. Klein (1931) and Kreshover (1940), after examination of hypoplastic tooth germs, observed the ameloblasts in all stages of degeneration. The final stage of these cystic changes is the complete destruction of the ameloblasts. Klein (1931) who produced enamel hypoplasia in rats and swine by means of a rachitic diet, states that the outstanding effect is an extreme wave-like hypoplasia. The alignment of the ameloblast layer is disturbed and folding, which is the cause of hypoplastic defects, results.

Croft, Witkop and Glas (1965) in their discussion of Pseudohypoparathyroidism in a twelve-year-old girl, state that the random distribution of pits over the surface of the crown and the indication that these were ascribable to defective enamel formation rather than to the resorption of fully formed enamel, suggests that a defect in vascularisation of the stellate reticulum might be the mechanism responsible for this pattern of enamel hypoplasia. According to Jump (1938) capillaries adjacent to the outer enamel epithelium invade the stellate reticulum immediately before enamel formation. This vascularisation serves for the adsorption of the intercellular fluid of

of the reticulum and for distribution of required nutrition to the inner enamel epithelium. A partial failure in this vascularisation of the stellate reticulum, or a selected failure of some of the capillary loops to function properly after vascularisation, could account for the distribution of the hypoplasia observed.

The hypoplasia due to pre-natal syphilis presents a characteristic, almost pathognomonic, appearance. The upper central incisor is 'screw-driver' shaped, the mesial and distal surfaces of the crown tapering towards the incisal edge, rather than the cervical margin. In addition, the incisal edge is usually notched. The mandibular central and lateral incisors may be similarly involved, although the maxillary laterals may be normal. The crowns of the first molars are very irregular, the enamel appearing to be arranged in an agglomerate mass of globules. There is some difference of opinion as to the mechanism of the changes in the tooth, whether it is due to an indirect effect or the actual action of the organisms on the enamel organ.

Bauer (1944) noted that there were spirochaetes in abundance in the highly vascular tooth follicle and that they migrate through the stellate reticulum to the ameloblastic layer. He considers it to be the chronic productive syphilitic inflammation of the tooth sac which affects the ameloblasts and exerts pressure on the early tooth bud to bring about the alteration in shape.

Bradlaw (1953) stated that this would not explain the uniformity of the abnormality. He reports that the spirochaetes may be seen in and around the follicular blood vessels and in their walls, in the dental papilla and in the dentine. The changes in these structures becomes increasingly marked and perivascular infiltration and oedema of the follicle are observed, followed by hypoplasia of the external enamel epithelium. In the next state there is proliferation of the stratum intermedium and the ameloblasts which bulge into the dentine papilla, thus producing the characteristic notch. Degeneration of the central ameloblasts may then take place when no enamel will be formed at the central notch. A similar degeneration at what should become the angles of the incisors accounts for their subsequent rounding.

Bradlaw also states that the affected teeth develop approximately between the fourth and sixth month in utero. This is the time that placental changes permit foetal infection to take place. This would explain why deciduous teeth are unaffected, as their tooth germs are fully differentiated by the tenth week of uterine life.

De Wilde (1943) and Brauer and Blackstone (1941) examined hypoplastic teeth of congenitally syphilitic children. In both cases the hypoplasia was thought to have been the result of anti-syphilitic drugs.

The notched incisors were first described by Hutchinson who, in 1857, described three diagnostic symptoms of congenital syphilis - inflammation of the eye, inflammation of the inner ear and dwarfed notched permanent incisor teeth (Boyle, 1955 p.65).

The clinical appearance of the molars may present one of two shapes

1. Moons molars. In this, the mesio-distal measurement of the occlusal surface is less than at the mid crown and so the molar is dome-shaped.
2. Mulberry molars. This has a similar compression of the occlusal surface. The sides are covered with normal enamel but the crown is rough and pitted. In the place of cusps there are poorly developed nodules compressed together.

Bradlaw (1953) reported the incidence of dental characteristic features in patients with pre-natal syphilis as :

maxillary incisor	6% - 28.5%
mandibular incisor	5%
maxillary and mandibular cuspids	4.5%
first permanent molars	3% - 37%
Hutchinson's triad	less than 1%

d. Endemic Fluorosis (Mottled Enamel)

Mottled enamel, or chronic endemic fluorosis, is characterised by chalky or pitted areas which appear as white, brown or even black defects. The clinical history of the patient invariably indicates an excess of fluorine in the drinking water (Bernier, 1959 p.82).

This was first studied by Black and McKay (1916), who recognised it as a lesion exhibiting a geographic distribution and suggested that it was due to the water supply. The term 'mottled enamel' was used by Black as a descriptive name for the condition. He states "the teeth are of normal form, but not of normal colour. When not stained yellow or brown, they are a ghastly opaque white. In many cases the teeth appear absolutely black. Mottled enamel is distinguished especially by the absence of cementing substance between the enamel rods in the outer fourth, more or less, of the enamel, and presenting a great variety of colour."

It was not until 1931 when Churchill and Smith, Lantz and Smith reported that the mottled teeth were associated with a small amount of fluoride in the drinking water. This has since been confirmed by other workers.

In 1935 Smith, Lantz and Smith reported that the lowest concentration that will produce the defect is 0.9 ppm (parts per million). They stated that the higher the concentration the more

severely are the teeth damaged and the greater the proportion of people affected.

In Black and McKay's (1916) first description of the lesion, it was made to appear that the enamel rods were themselves intact and of normal form and contour but with a disturbance of interprismatic substance.

Subsequent to this Williams (1923), who used higher magnification, reported the defective formation and calcification of both the rods and the interprismatic substances.

Schour and Massler (1935) confirmed the findings of Williams. Thoma and Goldman (1960, p.106) cite Schour as stating that the disturbance in both formation and calcification which results in the amorphous enamel and a matrix which shows, instead of a prismatic arrangement, a globular make-up.

Massler and Schour (1959) state that the developing enamel is the first structure to react to the ingestion or injection of fluorides. The ameloblasts show a selective response, in the form of abnormal globules within their cytoplasm. This is followed by sharp rings of disturbed calcification. This disturbance in calcification is probably brought about by the deposition of calcium fluoride in the tissue instead of the normal calcium salts, and possibly also by the disturbances of the enzymatic phosphatase system concerned in calcification.

Dean and Arnold (1943) observed that the appearance of the teeth varies with the severity of the lesion and can be correlated with the fluoride content of the water in the district.

Continuous use of water containing 1.1 ppm. causes only the mildest grade of mottling in only 10% of the population drinking the water. However, the effects become progressively worse with increased amounts until when the concentration approaches 6.6 ppm. the incidence is about 100%, of which about 90% is the severe grade.

In the very mild grade there are small white opaque areas which involve less than 25% of the tooth surface (Dean, 1934). In the mild grade these are more extreme. Most enamel surfaces are involved in the moderate grade, the surfaces subject to attrition showing marked wear. Minute pitting is often present, and a brown disfiguring complication. In the severe grade all enamel surfaces are involved and the morphology of the tooth may be affected. There is discrete or confluent pitting, the brown stain is widespread and the teeth often have a corroded appearance.

The brown stain, which may vary from light yellow to brown or black, is not present before the tooth erupts but gradually occurs afterwards (Kempf and McKay, 1930). Farmer and Lawton (1966, p.209) state the cause of the change has not yet been definitely elucidated, but it may be associated with dietary conditions or be due to chromogenic bacteria.

Massler and Schour (1952) reported that malnutrition aggravates the degree of mottling for a given level of fluoride intake.

Mottling of the teeth only occurs provided the child lives in the district responsible during the time of enamel mineralisation. As a considerable part of the enamel of the deciduous dentition is mineralised before birth, and nearly all the remainder during the period of lactation, the deciduous teeth, in most cases do not show any mottling. Kempf and McKay (1930) reported some slight indications. Coumoulos (1949) reported "some degree of mottling" in 58.3% of deciduous teeth.

Smith and Smith (1935) state that there is clinical evidence that the maternal organs appear to exert a protective influence, even though a pregnant woman drinks water containing as much as 6 ppm. of fluorine. However, they state that there is severe mottling of the enamel of the deciduous teeth in breast-fed infants in areas of 12-18 ppm. This points to the passage of fluorine into the foetal system or into the milk supply of the nursing mother - but these are severe circumstances.

e. Hypoplasia Associated with Tetracycline Therapy

Discolouration of the teeth by the Tetracycline group of antibiotics was first observed by Shwachman et al (1958). Their report describes dark staining of the teeth occurring in the deciduous or permanent dentitions, or both. The suggestion that the stain was due to localisation of the Tetracyclines in the hard tissues was supported by Bevelander, Rolle and Cohlman (1961).

Recent reports (Weyman and Porteous 1962; Witkop and Wolf 1963) indicate an increasing incidence of this yellow to brown discolouration, Tetracycline being the drug of choice in many infections in which the consequences of the infection outweighs the possible damage to the teeth.

Storey (1963) investigated the effect of Tetracycline on the enamel of rat molars. He found that with low dose levels (20 mg/kg/day) the molar teeth were coloured bright yellow and fluoresced under ultra-violet light. With higher dosages (200-250 mg/kg/day) results varied. In some molar teeth were yellow, in others brown, while some showed hypoplastic areas in the enamel of the incisor teeth. Under ultra-violet light all coloured molars and the area adjoining the hypoplastic enamel of the incisor teeth fluoresced a bright yellow.

Fluorescent microscopy was also carried out. Teeth formed and yet with incomplete calcification showed initial localisation of the Tetracycline to the margins of the calcifying dentine and throughout the areas of calcifying enamel. Following maturation, labelling remained in the dentine but faded in the enamel.

Storey states that the results were consistent with observations in man, that enamel hypoplasia may be induced by high dose levels of Tetracycline given before completion of amelogenesis.

The findings of Storey agree with the clinical study carried out by Witkop and Wolf (1963). Fifteen of seventeen children examined had hypoplasia of the enamel of the deciduous teeth and two of the permanent teeth. The latter two children had been given Tetracycline when they were nine and eleven months, respectively, and had hypoplasia of the incisal edges of the permanent incisor teeth and occlusal surfaces of the first permanent molars. A child who at two weeks of age received the drug had hypoplasia involving the middle third of the deciduous incisors, the tips of the cuspid, the occlusal surface of the first deciduous molar and the cusp tips of the second deciduous molar. It is evident that the hypoplasia of the enamel was localised to those teeth and portions of teeth which underwent calcification at a time which coincided, in each case, with the

time of Tetracycline therapy. In those with high doses severe hypoplasia resulted. In addition, all children had a yellow to brown staining in the areas of hypoplasia and in the enamel immediately adjacent to the hypoplastic defect. Examination of these teeth under an ultra-violet light produced a bright fluorescence, most intense at the junction of the hypoplastic and normal enamel. They also examined patients with normal and carious deciduous and permanent teeth, normal dentine exposed by attrition, hereditary hypocalcified amelogenesis imperfecta, hereditary hypoplastic amelogenesis imperfecta, hypoplasia of the enamel secondary to erythroblastosis fetalis, vitamin D-refractory rickets, and snow capped teeth, who had not had Tetracycline. None of these conditions showed yellow fluorescence.

Wallmann and Hilton (1962) examined a group of twenty-one children who had been given Oxytetracycline during their neo-natal period. Only two of these children had abnormal teeth, but in both cases other factors were probably involved.

Rendle-Short (1962) reports the case of a mother who, after receiving tetracyclines during the last six weeks of pregnancy, bore a child who at the age of one year had teeth of a bright yellow colour. It appears that the drug will pass the placental barrier.

Weyman (1967) states that the degree of fluorescence is no indication of the amount of Tetracycline actually present. She also states that there is little doubt that the clinical discolouration is carried by staining in the enamel and not in the dentine.

Witkop and Wolf (1963) state that "exposure of the yellow pigmented area to sunlight turns it to brown colour and, at the same time, there is a loss of the ability of the pigment to fluoresce a bright yellow. In general, the older the child, the browner the teeth.

Weyman (1965) examined the effect of other members of the Tetracycline group. It was found that children who had been given chlortetracycline had grey-brown teeth and those who had any of the other three types of Tetracycline had yellow teeth. The yellow stain tended to darken to brown only on the surfaces exposed to light. The severest staining occurred in those who had prolonged chlortetracycline therapy, those who received maternal tetracycline, or those who had demethylchlortetracycline. It is suggested that oxytetracycline may well be the type of choice when the drug is required as it appears to produce the least objectionable discolouration.

e. Dentinogenesis Imperfecta

Dentinogenesis imperfecta is a localised form of mesodermal dysplasia affecting the dentine of the tooth (Part I, p. 18). This may occur as an isolated defect or as part of a generalised mesenchymal disturbance, osteogenesis imperfecta.

Goldweber and Low (1957) described the defect as having the following characteristics :

1. the teeth have a bluish or amber opalescence
2. there is a disproportion in size of crown and roots, the roots being considerably diminished
3. the pulp tissue is partially or completely obliterated
4. the enamel is easily worn or fractured.

An examination of the literature reveals several terms which have been used to describe this condition.

Wilson and Steinbrecher (1929) favoured "hereditary hypoplasia of the dentine."

Hodge (1936) suggested "hereditary opalescent dentine".

Finn (1938) reviewed twenty-eight cases using Hodge's terminology.

Becks (1931) suggested "odontogenesis imperfecta".

Chaudhry, Wittich, Stickel and Holland (1961) used "odontogenesis imperfecta" to describe the rare condition in which there was a disturbance in both amelogenesis and dentinogenesis.

Weinmann, Svoboda and Woods (1945) used the term "dentinogenesis imperfecta".

Roberts and Schour (1939), in discussing these terms state that "dentinogenesis imperfecta" is preferable as it has the advantage of bringing hereditary opalescent dentine and the dental changes in osteogenesis imperfecta, with which it is often associated, under a common denominator. They pointed out that hereditary opalescent dentine and hypoplastic dentine, are terms describing the result not the pathology.

In dentinogenesis imperfecta there is a marked hereditary factor. Wilson and Steinbrecher (1929) reported the defect in a family over five generations. A similar case was presented by Noyes (1935). Roberts and Schour (1939) cited the pedigree of a family of five generations, with forty-five persons, twenty-two of whom were affected. They found that the condition was not found in the children unless it was visibly possessed by one of the parents. It is therefore a dominant characteristic. This was similar to the conclusion arrived at by Finn (1938)

in his tabulation of forty-one families. Rushton (1955) reported the unusual case where the anomaly was transmitted through a parent with teeth of normal appearance. He states that cases of this happening are rare. This may be contrasted with osteogenesis imperfecta, in which more than half the cases have no affected relatives.

Helmers and Finn (1966) stated that of all the tooth defects to be genetically determined, dentinogenesis imperfecta has the most evidence for hereditary transmissibility. From a review of the literature it is evident that dentinogenesis imperfecta is inherited as a simple autosomal heterozygous dominant trait, with modifying factors and good penetrance.

Roberts and Schour (1939) state that the defect is in no way the result of faulty nutrition, though conceivably it might be accentuated by it. A dominant character such as this can be eliminated permanently, only if the affected individuals do not bear children.

Dentinogenesis imperfecta may occur as an isolated defect or as part of the generalised mesenchymal disturbance, osteogenesis imperfecta. Osteogenesis imperfecta is a congenital, primary, generalised, arrested malformation of the bone building system and both osteoblasts and odontoblasts are involved. These cells produce defective bone and dentine. Rushton (1955) suggested that the two conditions, as regards the teeth, were very much the same.

Helmers and Finn (1966) do not consider there to be any significant difference between the independent dystrophy and that observed in conjunction with osteogenesis imperfecta.

Roberts and Schour (1939) had suggested there is an "intimate relationship" between osteogenesis imperfecta and hereditary opalescent dentine, and give this as their reason for preferring the term 'dentinogenesis imperfecta'.

Blattner, Heyes and Robinson (1942) agree that there is an associated between these two conditions and wonder whether they are :

1. different degrees of fundamental hereditary dysplasia of the mesoderm,
2. two abnormalities brought about by a single hereditary factor,

or

3. two associated conditions, one being dominant, the other recessive.

Winter and Maicocco (1949) reported the study of osteogenesis imperfecta occurring in a family for six generations, but found no signs of the disease associated with the dental condition.

Wilson and Steinbrecher (1929) and Ivancie (1954) have reported families with dentinogenesis imperfecta not associated with osteogenesis imperfecta; the former for six generations and the latter for two.

Barnett and Barnett (1963) discussed a case of dentinogenesis imperfecta associated with osteogenesis imperfecta and stated that both can occur simultaneously as well as independently.

Thoma and Goldman (1960, p.138) state that "in dentinogenesis imperfecta the part of the tooth which is mesenchymal in origin is affected, the dentine, and sometimes the cementum, principally the root of the tooth. However, in most instances dentinogenesis imperfecta is accompanied by amelogenesis imperfecta. In diseases which affect bone formation, dentinogenesis imperfecta may be found without the enamel formation being involved. In such cases the roots are underdeveloped in spite of crowns of normal dimensions made up of perfectly formed enamel."

Noyes (1935) summarised his observations of the condition, in both the deciduous and permanent dentitions as follows : peculiar bluish-brown colour of the teeth; normal enamel, complete obliteration of the pulp and atypical dentine showing increased matrix and the irregular arrangement of tubules.

Rushton (1955) stated that it was usual for every tooth to be affected, but cites Chaput (1952) who had reported a girl in whom all teeth were affected except the maxillary permanent central incisors, which were considered quite normal. This defies chronological interpretation.

Roberts and Schour (1939) found the teeth normal in size and form but they showed marked abrasion and the enamel was lost except near the gingival level. The enamel chipped and fractured readily. The teeth, especially the exposed dentine, showed an amber-like translucency or opalescence.

Rushton (1955) found that in nearly all children he examined, the pulps of the deciduous dentition remained patent and that sometimes the dentine walls remained unduly thin.

Helmers and Finn (1966) observed that although the enamel layer is of normal thickness and hardness, it tends to flake away from the underlying dentine. This is due to the lack of scalloping of the dentino-enamel junction (Part I, p. 23), the poorly calcified nature of the dentine base or a basic fault in the enamel. Because of the friability of the enamel, the soft underlying dentine is ultimately exposed, thus accelerating the tendency of the teeth to wear. As the occlusal and incisal portions become worn, their bulbous appearance becomes more pronounced. The teeth are frequently worn to the gum line.

Witkop and Wolf (1963) report the occurrence of dentinogenesis imperfecta to be about once in every 8,000 children.

5. Abnormality of Eruption

a. Aberrant Tooth Eruption

It is recognised that a broad range of variation exists in the normal eruption of the deciduous and permanent teeth in different individuals. Because of this inherent biologic variation it is difficult to determine when the eruption dates of teeth of a given individual are outside the limits of the normal range.

Gates (1964) found that the range of eruption times varied with the tooth under consideration from a minimum of 2 years 3 months for the maxillary and mandibular central incisors in females, to a maximum of 5 years 11 months for the mandibular second bicuspids in males. The following table, adapted from Gates, shows the median age of eruption for the permanent teeth in New South Wales children.

Maxilla	Tooth	Males		Females	
	1	7 yrs	1 mth.	6 yrs	10 mths.
	2	8	2	7	9
	3	11	4	10	8
	4	10	3	9	10
	5	11	0	10	8
	6	6	3	6	1
	7	12	1	11	9
Mandible	1	6	2	5	11
	2	7	4	6	11
	3	10	6	9	5
	4	10	10	10	3
	5	11	7	11	1
	6	6	2	5	11
	7	11	8	11	3

Nevertheless cases do occur in which the eruption time is grossly outside and may be considered to be abnormal.

Bodenhoff and Gorlin (1964) reviewed the literature on natal and neonatal teeth, which are the teeth present at birth or that erupt within thirty days of birth (Massler and Savara, 1950), and they suggested that their incidence is at least once in every 3,000 births. They state that, although the aetiology is unknown, about 15% of subjects had parents, siblings or other near relatives with a history of such teeth. About 85% of natal and neonatal teeth reported were mandibular incisors and only about 10% supernumary.

Rushton (1953) states that these are ectopically placed teeth and that it is their superficial position that causes the premature eruption.

Histologic investigation of these teeth has revealed a failure of root formation despite eruption, a large vascular pulp, irregular genesis of the dentine and a failure of cementum formation (Bodenhoff and Gorlin, 1964).

Delayed eruption of the deciduous dentition is rare, the important causes being diseases in which the development of the skeleton as a whole is retarded as in rickets and cretinism. Cawson (1962) states that if the underlying disorder is treated effectively then the eruption of the teeth proceeds normally.

Bone dysplasias are also responsible for retarded eruption of the permanent dentition. In cleido-cranial dysostosis much of the deciduous dentition may be retained until adult life. Thoma and Goldman (1960, p.627) state that the inhibition of the eruptive force affects both the deciduous and permanent dentitions. The deciduous teeth are very slow in becoming exfoliated. The permanent dentition, however, presents the extreme form of delayed eruption and in some instances is almost completely inhibited throughout life.

Millhon and Austen (1944) observed that the deciduous dentition may erupt normally.

Miller (1937) reported retardation of eruption associated with achondroplasia.

In 1933, Ivy recorded the non-eruption of the entire permanent dentition associated with dwarfism. This was thought to be due to an endocrine dysfunction.

The eruption of the dentition is also retarded in Leontiasis Ossea. This is due to the dense nature of the surrounding bone (Thoma and Goldman, 1960 p.629).

Baume, Becks and Evans (1954) examined the hormonal control of tooth eruption. They found that in thyroidectomy and hypophysectomy the eruption rate of the teeth was reduced, the latter being the more dramatic. It was also found that injections of

thyroxin increased the eruption rate. This was consistent with the statements made by Schour and Massler (1943) who observed that hyperthyroidism and hyperpituitarism resulted in premature eruption, while hypothyroidism and hypopituitarism resulted in retarded eruption.

Baume, Becks and Evans (1954) also found that injections of growth hormone had little effect on the eruption of teeth but that it considerably enhanced alveolar growth.

Hereditary patterns have also been reported. Farmer and Lawton (1966, p.134) cite the pedigree of a family in which delayed eruption is observed in various members.

Garn, Lewis and Polacheck (1960) observed that siblings are similar both in the timing of tooth calcification and in tooth movement.

Niswander (1963) showed that familial effects may be partially due to environmental factors.

Premature eruption of the permanent dentition necessitates early loss of the deciduous ones. This is mainly due to the prevalence of dental caries and its sequelae, i.e. by premature extraction. Adler (1963) demonstrated that this seemed to exert an accelerating effect on the eruption of the permanent successors. This agrees with Sleichter (1963) who also stated that a retardation may result if the extraction is carried out at a very early stage.

As mentioned earlier, general early loss of the deciduous teeth has been reported associated with Pink disease (Nussey, 1954), Papillon-Lafevre syndrome (Hall, 1963) and Hand-Schuller-Christian disease (Talley, 1948).

b. Ectopic Eruption

Ectopic eruption denotes deviation from the normal eruptive path which may result in malposition, impaction or transposition of the tooth or teeth involved.

When the developing tooth follows an eruptive path, well outside that of normal deviation for the tooth concerned, it is presumed to have developed from an abnormally located tooth germ (Farmer and Lawton, 1966 p.137). Nodine (1943) attributes this abnormality of the location of the tooth germ to a redundancy of the dental lamina, and the abnormality of its axis to a pressure of unknown origin. He states that :

"In the tooth follicle, the developing incisal edge of the anterior teeth and the cuspidate surfaces of the posterior teeth are parallel to the oral ridge or surface. Too much importance cannot be placed upon this relation, for upon its continuance during the development of the teeth, depends their uniform normal eruption. Any deviation will cause rotated, misplaced or impacted teeth."

He goes on to say that various formative processes are taking place around the developing follicles. At this stage, the follicles are not fixed in their normal relations to the oral surface and any pressure may produce an alteration. This altered relationship is proportionate to the force exerted.

Kloeppel (1959) believes that the emerging tooth starts normally from its crypt when a small deviation from the complicated path of eruption results in a malposition. He describes the jaws of a four-year-old in whom "the whole spongiosa is occupied by the crowns of the permanent teeth, which are in a state of physiological crowding."

Miller (1963) states that of all the teeth in the maxillary arch the cuspid develops the farthest from the position that it will finally occupy when fully erupted. It is, therefore, more prone to be diverted from its normal eruptive course.

Hitchin (1956) states that because of the pyramidal shape of the maxilla and the more bulbous nature of the buccal surface, the incisal tip of the cuspid is deep to the apex of its deciduous predecessor, and thus the permanent cuspid is very liable to be deflected palatally.

Transposition, which is the interchanged position of two teeth, most often occurs in connection with the maxillary canine and first premolar teeth. Miller (1963) states that it has been suggested that defective or missing lateral incisors are in some way instrumental in the aetiology of transposition of these teeth. However, he believes that ectopic developmental positions of the tooth germs of the cuspid and first premolar would still seem the best theoretical cause for their transposition. Townend (1949) recorded the bilateral occurrence of the transposition of these teeth. Less frequent is the interchange between maxillary

canine and lateral incisor. Jackson (1951) presented a case of transposition of the maxillary canine to the position of the central incisor.

The small jaws of man are a most frequent cause of impaction and maleruption. MacGregor (1945) commented that the early deciduous cuspid loss, resulting from ectopic eruption of the permanent lateral incisors was due to insufficient lateral or antero-posterior growth of the jaws. This may also occur where a tooth to base bone discrepancy is recorded.

O'Meara (1962) cites Nikiforuk (1948) who stated that the prime factor was not the abnormal position of the permanent tooth germ but "a deficiency of the intensity and time gradients of growth".

Prolonged retention of the deciduous teeth causes malpositioning of the permanent ones. Morgan (1938) reported that if the deciduous tooth was retained, beyond the normal exfoliation time, the succeeding permanent tooth had a tendency to rotate.

Kim, Shiere and Fogels (1961) observed that there were other contributing factors, such as asymmetrical root resorption, pathological involvement of the deciduous teeth and premature loss of the first permanent molar teeth, but that prolonged retention did not appear to be the cause of rotation. They found that the lower first and second premolars had a characteristic tendency to rotate in a mesial direction. This tendency, as well as the severity, was found to be greater in the second premolars.

Distal eruption of the mandibular second premolar is frequently observed following extraction of the first permanent molar.

Friel (1945) states that in such cases the second premolar only appears to move distally. His explanation is that the loss of distal contact for the crown allows it to lag behind while growth of the bone is carrying the root forward.

Cryer (1965) states this type of change in the premolar position is too rapid to result from growth alone. Rose (1958) suggests that the distal movement may result from the force of eruption modified by an atypical guide plane.

Godfrey (1967) suggests that this mainly occurs where there is a distal inclination of the tooth germ and subsequent asymmetrical root resorption of the second deciduous molar.

Kim, Shiere and Fogels (1961) say that this distal eruption contra-indicates the extraction of the mandibular first permanent molar, prior to the eruption of the second premolar, if at all possible.

Moyers (1963) states that the maxillary first permanent molar erupts ectopically more often than any other tooth.

Morgan (1938) believes that this is caused by the impaction of the molar against the distal of the bell-shaped deciduous molar.

Willett (1933) stated that the alveolar process separating these teeth is relatively soft compared with the tough fibrous tissue

overlying the unerupted permanent molar, and hence the erupting tooth may seek a path of lesser resistance.

MacGregor (1945) and Graber (1962) consider it to be due to a lack of antero-posterior arch length, while Cheyne and Wessells (1947) believe the physiological mesial drift to be an important feature.

Moyers (1963) cites Pulver (1962) who states that the following, in combination, usually accounts for this ectopic eruption.

1. teeth in ectopica are significantly larger than than normal
2. abnormally placed tooth germ of the first permanent molar
3. maxillary alveolar arch length is normal, but the tuberosity may lag significantly
4. the morphology of the distal surface of the maxillary second deciduous molar and of the mesial surface of the maxillary first permanent molar is ideally suited for the locking of the latter tooth during its eruption.

c. Ankylosis of the Deciduous Molars

Ankylosis of the tooth implies that there is a bony continuity between the alveolar bone and the cementum or dentine, with no soft tissue intervening. The tooth most commonly affected by this is the mandibular second deciduous molar.

The term 'submerged' is generally given to this condition as it describes the clinical appearance.

Dixon (1963) defines a submerging tooth "as one which fails to maintain its position in the developing occlusion, and may become partly or completely re-enclosed in the oral tissues". He found the incidence of submerged teeth, amongst 400 school children, to be 2.5%.

This nomenclature, submerged tooth, is objected to by Vorhies, Gregory and McDonald (1952) on the grounds that the tooth is enveloped in the growing tissues and does not sink into them. They stated that it seems not to take into account the existence of continuous bone growth and tooth eruption and the static retention of the ankylosed tooth in the arch.

The original observation of ankylosis, by histological methods, was made by Noyes (1932), the picture being one of hyperactivity of bone growth. Previous to this he considered the possibility of an early history of rickets with recovery, but found

that the histological appearance did not support this hypothesis.

Vorhies et al (1952) presented further histological evidence to support Noyes. They stated that the osseous ankylosis lies between the dentine and bone and is carried on in close proximity with osteoclastic activity. In one area osteoclastic activity on old dentine is prevalent and a few microns away osteoblasts are laying down osteoid tissue which is hypoplastic. The picture appears to be one in which this double activity of resorption and osseous deposition seemingly bores through solid tooth structure leaving in its wake atypical bone.

Noyes (1943) has observed that even enamel is vulnerable to the absorption process.

Dixon (1963) found a similar histopathological appearance. He states that the process is not affected by the absence of the succeeding premolar tooth, a similar picture being found in a patient with agenesis of the second premolars.

Many theories have been advanced concerning the predisposing factors. Noyes (1932), previous to histological evidence, considered the possibility of early rickets.

Capon (1944) suggested that a localised failure or disturbance of alveolar bone growth has occurred. Evidence supporting this is the tendency of the submerging deciduous molars to occur

in groups and combinations, occasionally with failure of the succeeding premolars to reach full occlusion.

Dixon (1963) considered that ankylosis is not the primary cause but part of the tissue reaction to a submerging tooth.

A number of cases have been reported in which several members of the same family have shown the condition. Capon (1944) described two such examples in brothers. Tulley (1963) reports having seen cases in a brother and sister, and also in identical twins.

Vorhies et al (1952), in discussing the two possible theories, attributed as the cause of this pathological condition, state :

"One is mechanical and the other physiological. In effect the mechanical theory uses for its support the fact that the process is one in which the permanent teeth physically block out and submerge the deciduous teeth. In our opinion the tilting and malposition of the teeth is one of effect, not cause, due to the disengagement of the balance of the arch and its bony support. As for the direct physiologic cause of ankylosis, very little can be said. However, it is agreed that a vital cementum is necessary for a fibrous union of a tooth in its socket. If the cementum is destroyed through resorptive processes and not replaced, an osseous union is feasible with the dentine".

Biederman (1962) discounts the general accepted theory of mechanical trauma and suggests a local disturbance of metabolism as a possible cause.

Dixon (1963) in discussing the 'ankylosis' or 'firming' of deciduous teeth during an inactive period in their physiological resorptative process, states that it is of a different type to that seen with submerging molars. It is not replacing part of the tooth, but is adhered to it. The essential difference was the different form of resorption which preceded the ankylosis.

PART III

Present Study

Radiographic Survey

1. Sample

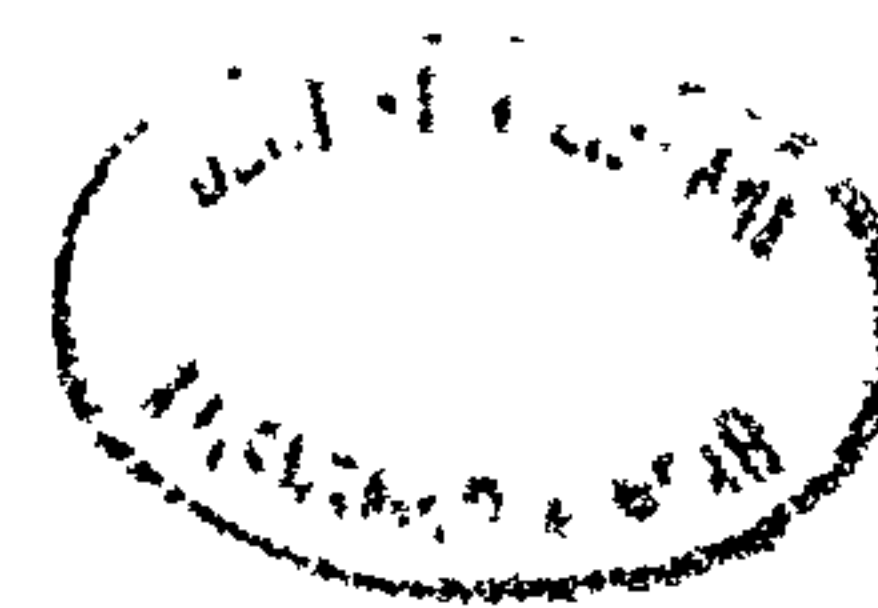
The material for the present study consisted of the full mouth and bitewing radiographs of the permanent and mixed dentitions, of 531 New South Wales children in attendance at the United Dental Hospital, Sydney.

The radiographs examined are part of the records routinely taken for each patient.

Two hundred and twenty-seven of these attended the Operative Clinic for routine dental care. The remaining 304 were obtained from the orthodontic files of the Graduate Clinic and were patients specifically referred for orthodontic treatment. The age range of the children was from six to fourteen years.

Patients not included in this survey were those with cleft lip and palate conditions and those with a history of extractions or orthodontic treatment.

Distribution of the sample is as shown in Table I.



2. Method

An independent sample of 100 radiographs was examined prior to the commencement of the survey. The results of this was compared with study models and other clinical data available, to determine the suitability of the examination of radiographs for this study.

Each radiograph was carefully examined for the following anomalies.

- a. Congenitally missing teeth - (Fig. 1) Radiographically seen as the absence of the developing tooth or tooth germ.
- b. Supernumary teeth - (Fig. 1) The development of an additional tooth.
- c. Abnormality of size - (Fig. 2) Macrodonia and Microdonia. The condition in which one or more teeth is larger or smaller than normal. The antimeres were used as a control where the condition was considered to be unilateral.
- d. Gemination - (Fig. 3) The partial division of a single tooth germ by an invagination. This appears as complete or incompletely separate crowns with a single root and root canal.
- e. Fusion - (Fig. 3) The complete or incomplete union of two normally separate tooth germs during development. This may show a single root with two root canals.
- f. Dilaceration - (Fig. 2) An angulation, sharp bend, or curve in the root of a normal tooth.

g. Odontomes :

Complex composite - (Fig. 4) A single irregular mineralised mass consisting of two or more dental tissues in any proportion and bearing no resemblance to the shape of the tooth.

Compound composite - (Fig. 4) A mineralised mass consisting of a number of separate denticles, made up of two or more dental tissues.

h. Dens-in-dente - (Fig. 5) This appears as an enamel invagination of the crown and root.

i. Invagination - (Fig. 5) An extension of the lingual pit of the maxillary central and lateral incisors into an abnormal cavity.

j. Amelogenesis Imperfecta - (Fig. 6)

Hereditary enamel hypoplasia. Teeth affected by this abnormality have the normal enamel cap replaced by a thin covering of enamel of varying thickness, so that the crowns are devoid of their normal mesial and distal contours. The cusps may be replaced by a series of serrations of varying sharpness. There is a great tendency for the crowns of the teeth to be abraded.

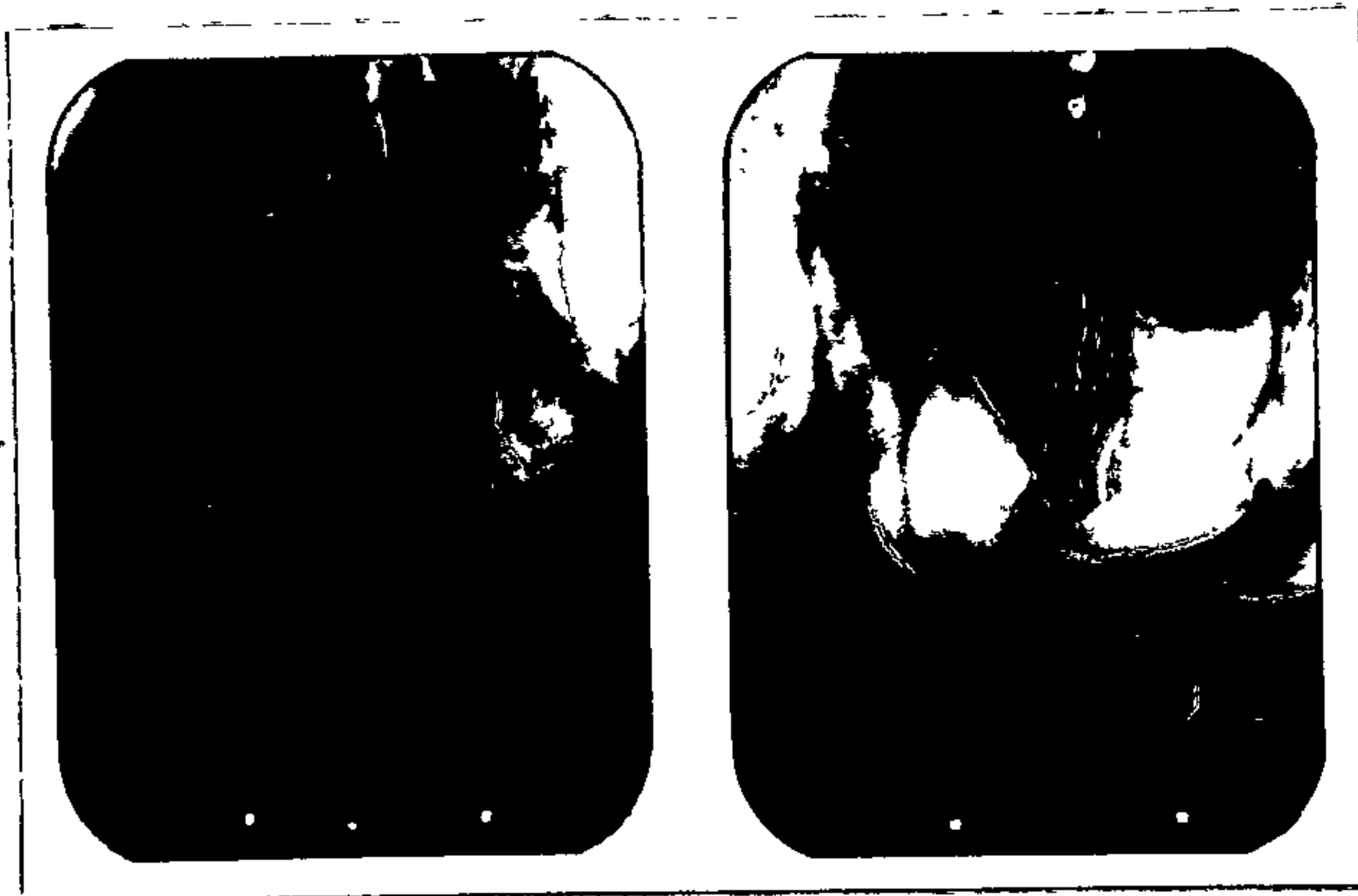
Hereditary enamel hypocalcification. The radiographic appearance is characterised by lack of contrast between the enamel and dentine, the whole crown presenting a uniform density. There may or may not be lack of mesial and distal contours.

- k. Enamel hypoplasia - (Fig. 7) The radiographic appearance varies greatly. To a lesser degree the crown may produce a good shadow, differing from normal only in slight irregularities of the surface. In others there is a mere shell of a partially formed crown, the margins of which are irregular. In place of uniform density the uncalcified tooth may present a spotty appearance.
- l. Dentinogenesis imperfecta - (Fig. 8) In this the teeth are shorter than normal, the roots being abnormally small in proportion to the normally formed crowns. The pulp chambers and root canals may be completely obliterated without any indication of change in density of the dentine in any part of the teeth.
- m. Ectopic position of tooth germ - (Fig. 9) Maxillary and mandibular second bicuspid.

This is registered by mesial or distal inclination of the tooth germ causing asymmetrical resorption of either the mesial or distal root of the deciduous first or second molars. Rotation is recorded when both lingual and buccal cusps were positioned mesio-distally.
- n. Ectopic eruption - Transposition - (Fig. 9) This is applied to a tooth which has erupted into a position in the arch not normally occupied by it, or to the interchange in position of two adjacent teeth.
- o. Impaction of maxillary cuspid teeth - (Fig. 5)
- p. Ankylosis - (Fig. 10) Teeth presenting this condition are usually below the level of the occlusal plane. Radiographically at least partial absence of the periodontal membrane is seen, with areas of apparent blending between

the tooth root and the bone.

q. Multi-root formation - (Fig.11)



(a)

(b)

Fig. 1 (a) Congenitally Missing Maxillary Lateral Incisor
(b) Maxillary Supernumary Central Incisors



(a)

(b)

Fig. 2 (a) Microdontia, Maxillary Lateral Incisor
(b) Dilaceration of Maxillary Second Premolar



(a)

(b)

Fig. 3 (a) Fusion between mandibular deciduous cuspid
and lateral incisor

(b) Geminated mandibular lateral incisor



(a)

(b)

Fig. 4 (a) Compound Composite Odontome

(b) Complex Composite Odontome



(a)

(b)

Fig. 5 (a) Impacted Maxillary Cuspid against a Lateral Incisor showing severe Invagination

(b) Dens-in-Dente

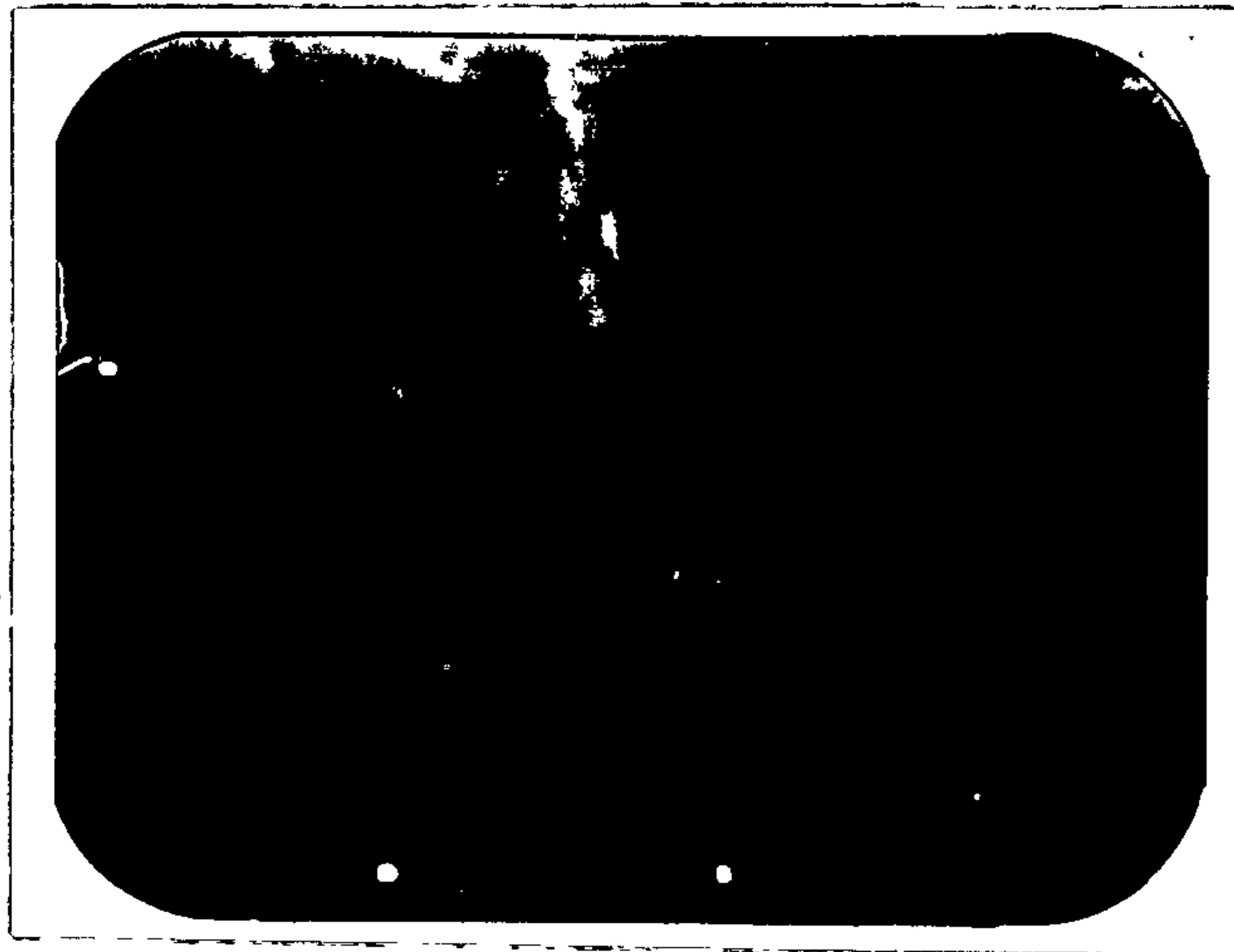


Fig. 6 Amelogenesis Imperfecta

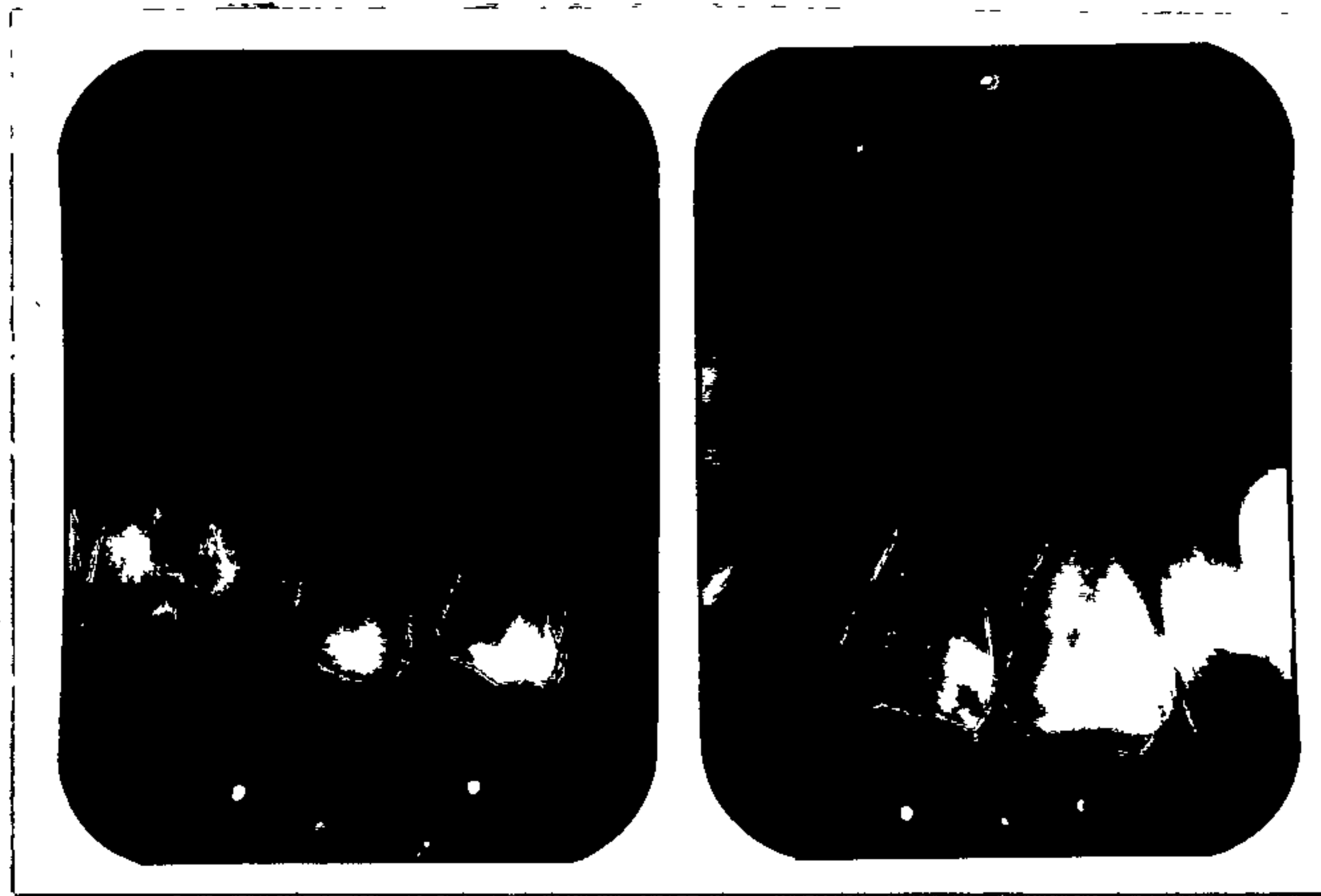


Fig. 7 Enamel Hypoplasia

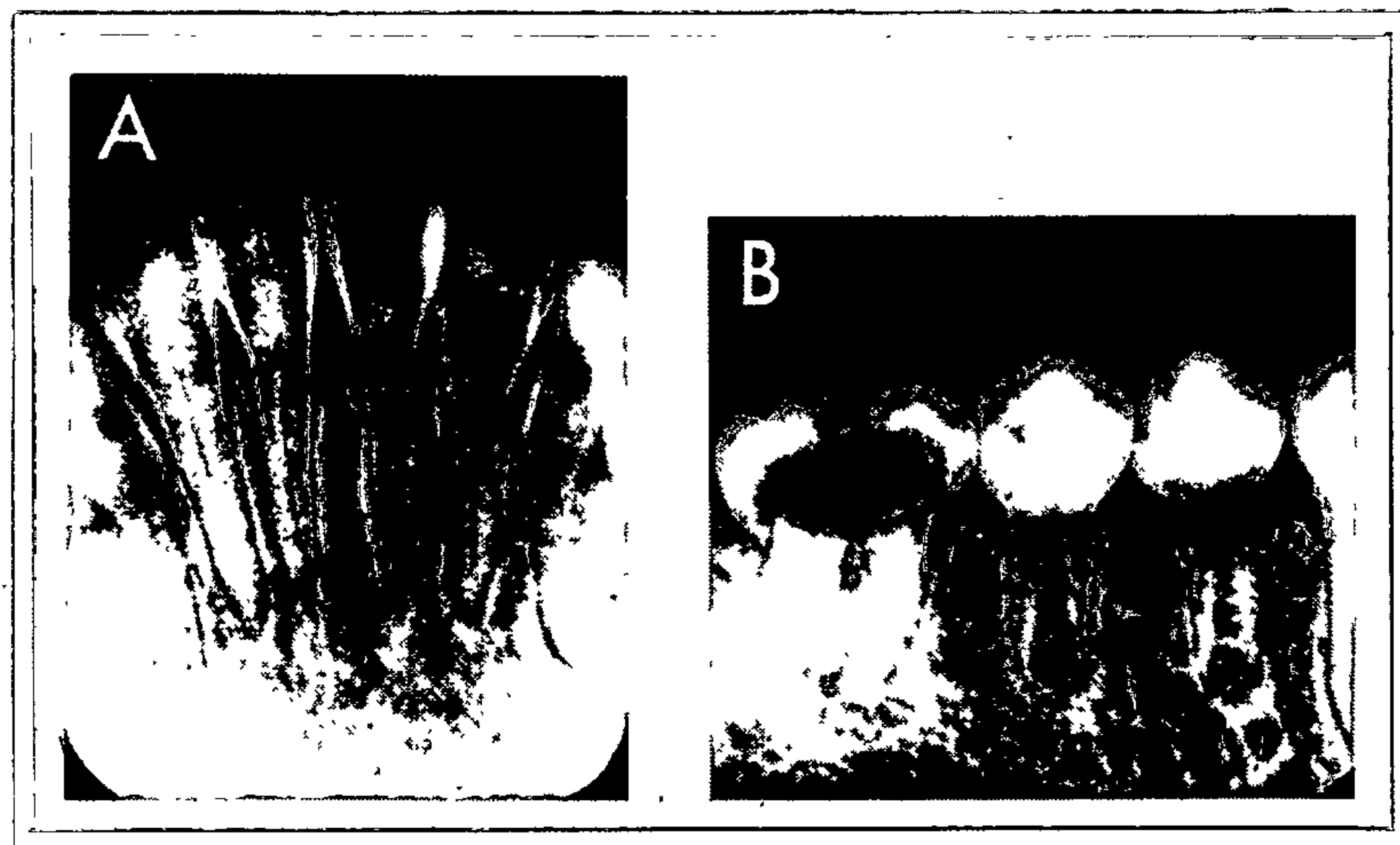
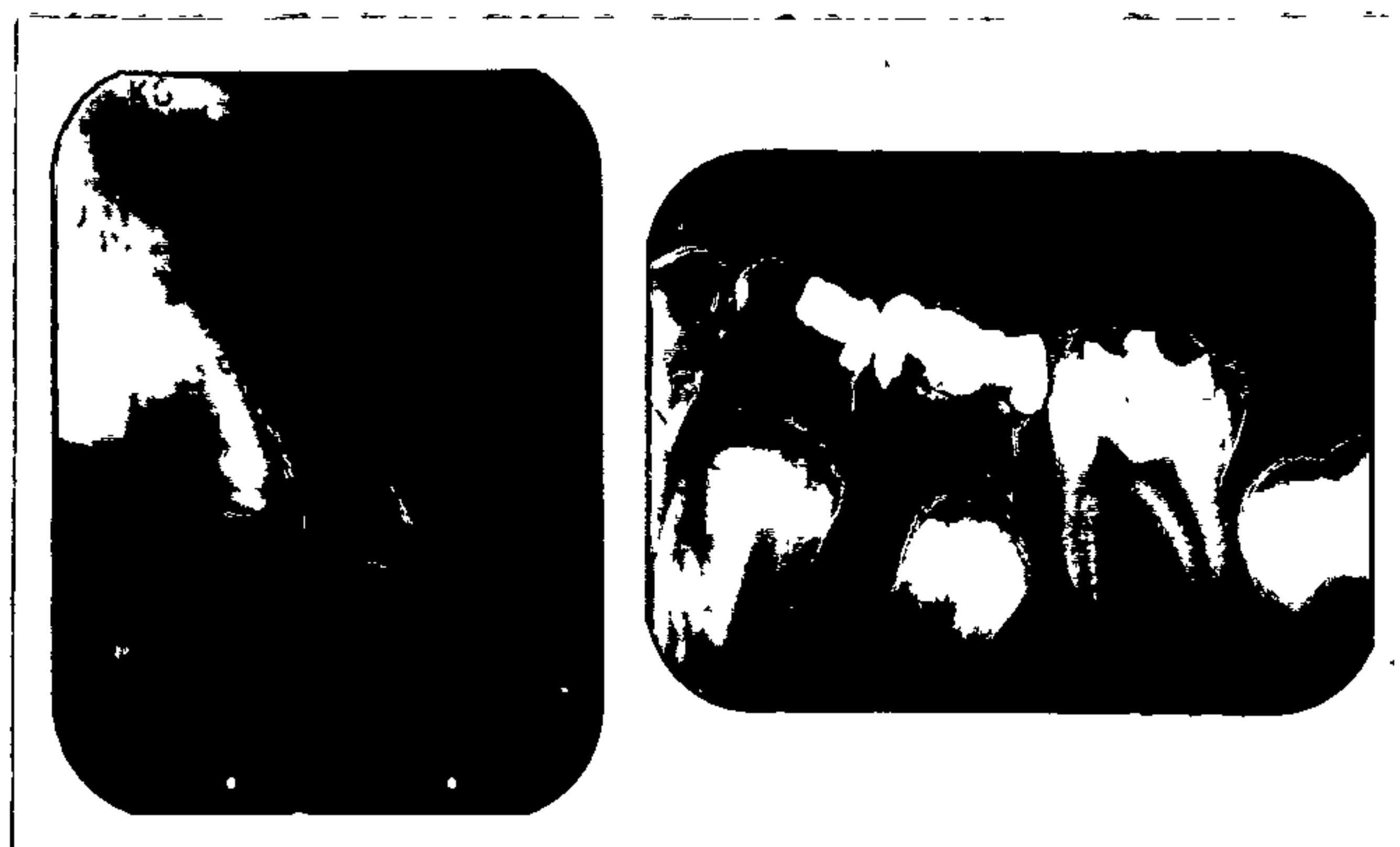


Fig. 8 Dentinogenesis Imperfecta



(a)

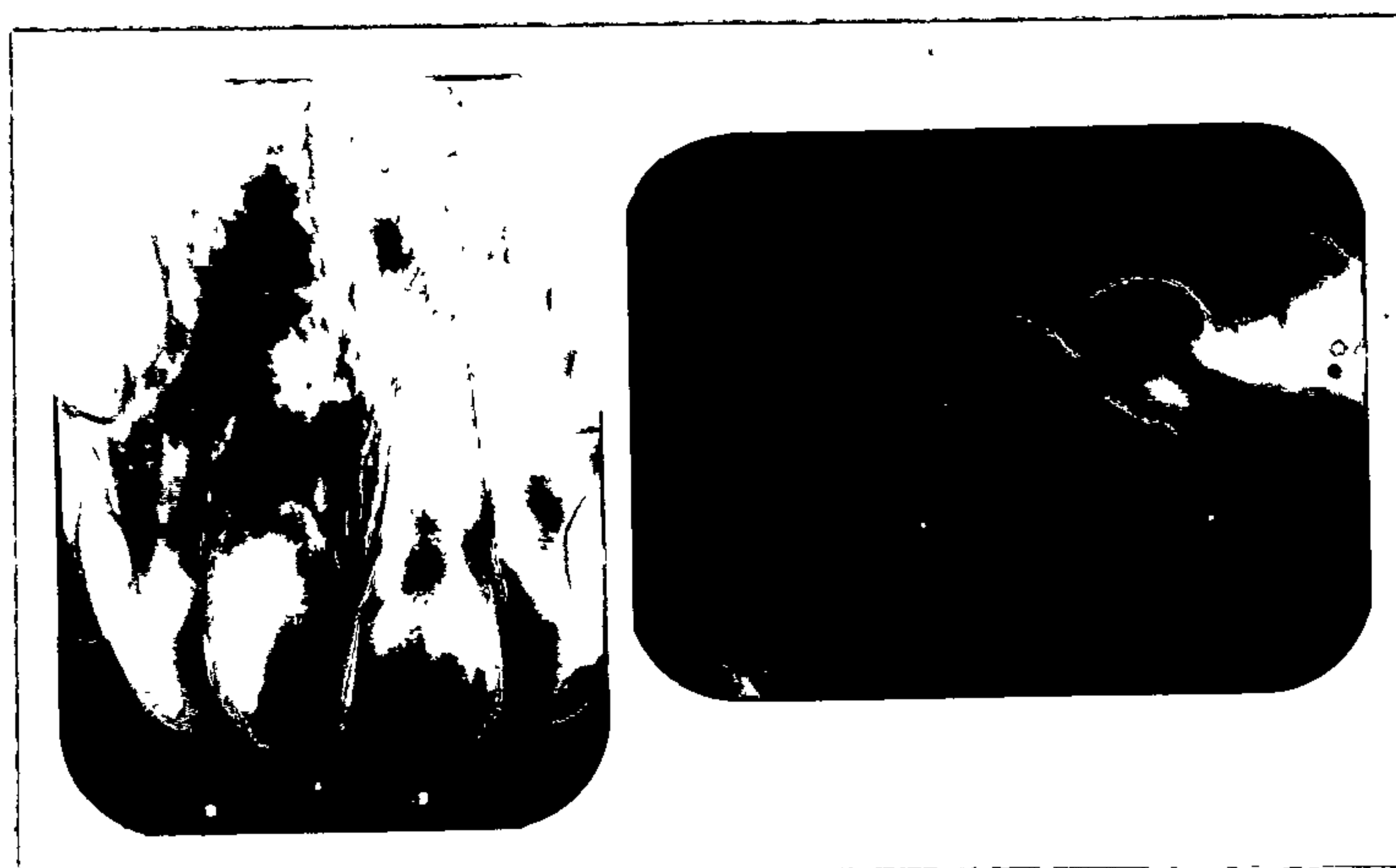
(b)

Fig. 9 (a) Transposition of Maxillary Cuspid and 1st Premolar

(b) Ectopic Eruption of Mandibular 2nd Premolar



Fig.10 Ankylosis of deciduous second molar



(a)

(b)

Fig.11 (a) Absence of Root Formation

(b) Multi-root Formation

RESULTS
TABLES I - XX

TABLE I
DISTRIBUTION OF CHILDREN USED IN SURVEY

Source	Males			Females			Total		
	Permanent Dentition	Mixed Dent.	Total	Permanent Dentition	Mixed Dent.	Total	Permanent Dentition	Mixed Dent.	Total
Orthodontic Clinic	51	69	120	78	106	184	129	175	304
Operative Clinic	53	52	105	74	48	122	127	100	227
Total	104	121	225	152	154	306	256	275	531

TABLE II

COMPARISON OF DIAGNOSIS OF RADIOGRAPHS TO OTHER DATASTUDY OF 100 CASES

	X-rays		Study Models etc.		Total	
	Persons	Teeth	Persons	Teeth	Persons	Total
Congenitally Missing	5	12	5	12	5	12
Supernumary Teeth	4	6	4	6	4	6
Microdontia	2	3	2	4	2	4
Cuspid Impaction	7	11	7	11	7	11
Gemination	1	1	1	1	1	1
Dilaceration	12	21	12	21	12	21
Ectopic Germ Position or Direction	8	15	8	15	8	15
Ectopic Eruption	1	1	1	1	1	1
Multi-rooted Teeth	2	4	2	4	2	4
Ankylosis	1	4	1	4	1	4
Enamel Hypoplasia	1	1	2	2	2	2

TABLE III
INCIDENCE OF TOOTH ANOMALIES

Anomaly	N	Male		Female		Total	
Missing Teeth	531	11	4.88%	18	5.88%	29	5.46%
Supernumary Teeth	531	8	3.55%	4	1.03%	12	2.25%
Microdontia	531	7	3.11%	7	2.28%	14	2.63%
Gemination	531	1	0.44%	-	-	1	0.18%
Enamel Hypoplasia	531	5	2.22%	5	1.63%	10	1.88%
Odontomes	531	1	0.44%	1	0.32%	2	0.37%
Invagination	531	4	1.77%	9	2.94%	13	2.44%
Dilaceration	531	20	8.88%	29	9.47%	49	9.22%
Ectopic Tooth Germ	275	10	8.26%	23	14.93%	33	12%
Ectopic Eruption	531	-	-	4	1.03%	4	0.75%
Impacted Canines	531	4	1.77%	15	4.09%	19	3.59%
Ankylosis	531	1	0.44%	1	0.32%	2	0.37%
Lack Root Formation	531	1	0.44%	1	0.32%	2	0.37%
Multi-rooted Teeth	256	7	6.73%	2	1.31%	9	3.51%

TABLE IV

INCIDENCE OF CONGENITALLY MISSING TEETH

	Operative Clinic		Orthodontic Clinic		Total	
Male	5	4.76%	6	5%	11	4.88%
Female	5	4.09%	13	7.11%	18	5.88%
Total	10	4.4%	19	6.25%	29	5.46%

TABLE V

INCIDENCE OF SUPERNUMARY TEETH

	Operative Clinic		Orthodontic Clinic		Total	
Male	1	0.95%	7	5.83%	8	3.55%
Female	1	0.81%	3	1.63%	4	1.3%
Total	2	0.88%	10	3.28%	12	2.25%

TABLE VI

TOOTH SUMMARY OF CONGENITALLY MISSING TEETH

Tooth	[?] (Operative Clinic		[?] Orthodontic Clinic		Total
	Male	Female	Male	Female	
<u>1</u> 2	1	4	-	1	6
<u>1</u> 5	2	2	-	-	4
<u>2</u> /	1	7	-	4	9
<u>5</u> /	2	-	-	-	2
<u>1</u>	1	2	2	-	5
<u>5</u>	1	6	2	2	11
<u>7</u>	1	-	-	-	1
<u>1</u> 7	1	2	1	-	4
<u>3</u> 7	-	1	-	-	1
<u>5</u> 7	1	4	3	2	10
<u>7</u> 7	1	-	-	-	1
Total	12	28	8	6	54

TABLE VII
CASE SUMMARY OF MISSING TEETH

Orthodontic Clinic				Operative Clinic			
Case No.	Sex	Number of Teeth	Teeth	Case No.	Sex	Number of Teeth	Teeth
1	M	2	$\overline{5 5}$	1	M	1	$\overline{5 }$
2	M	4	$\frac{5 5}{7 7}$	2	M	2	$\overline{5 5}$
3	M	1	$\overline{ 5}$	3	M	1	$\overline{ 5}$
4	M	3	$\frac{ 5}{1 1}$	4	M	3	$\overline{5 1 1}$
5	M	1	$\overline{2 }$	5	M	1	$\overline{ 1}$
6	M	1	$\overline{ 2}$	6	F	1	$\overline{ 5}$
7	F	1	$\overline{2 }$	7	F	1	$\overline{5 }$
8	F	1	$\overline{ 5}$	8	F	1	$\overline{5 }$
9	F	5	$\frac{2 2}{1 15}$	9	F	1	$\overline{ 5}$
10	F	3	$\frac{ 5}{5 5}$	10	F	2	$\overline{2 2}$
11	F	7	$\frac{2 2}{53 1 15}$				
12	F	1	$\overline{2 }$				
13	F	2	$\overline{5 5}$				
14	F	1	$\overline{2 }$				
15	F	2	$\overline{2 2}$				
16	F	2	$\overline{2 2}$				
17	F	1	$\overline{ 5}$				
18	F	1	$\overline{ 5}$				
19	F	1	$\overline{ 5}$				

TABLE VIII
CASE SUMMARY OF SUPERNUMARY TEETH

	No.	Sex	Position	No. Teeth
Orthodontic Clinic	1	M	$\overline{5 \ 4 /}$	1
	2	M	$\begin{array}{c} \uparrow \\ \underline{1 \ / \ 1} \end{array}$	2
	3	M	$\begin{array}{c} \uparrow \\ \underline{1 \ / \ 1} \end{array}$	1
	4	M	$\begin{array}{c} \downarrow \quad \downarrow \\ \underline{1 \ / \ 1} \end{array}$	2
	5	M	$\begin{array}{c} \downarrow \quad \downarrow \\ \underline{1 \ / \ 1} \end{array}$	2
	6	M	$\begin{array}{c} \uparrow \\ \underline{1 \ / \ 1} \end{array}$	1
	7	M	$\begin{array}{c} \downarrow \quad \downarrow \\ \underline{1 \ / \ 1} \end{array}$	2
	8	F	$\begin{array}{c} \downarrow \quad \downarrow \\ \underline{/12 \ 67} \end{array}$	2
	9	F	$\begin{array}{c} \downarrow \\ \underline{1 \ / \ 1} \end{array}$	1
	10	F	$\begin{array}{c} \uparrow \\ \underline{1 \ / \ 1} \end{array}$	1
Operative Clinic	11	M	$\begin{array}{c} \downarrow \\ \underline{1 \ / \ 1} \end{array}$	1
	12	F	$\begin{array}{c} \uparrow \\ \underline{1 \ / \ 1} \end{array}$	1

The arrow indicates the direction of the crown of the supernumary tooth, and its position in relation to adjacent teeth in the arch.

TABLE IX

INCIDENCE OF ODONTOMES

Operative Clinic ¹			Orthodontic Clinic ²			Total
Males	1	0.95%	-	-	1	0.44%
Females	-	-	1	0.54%	1	0.32%
Total	1	0.44%	1	0.32%	2	0.37%

1 = Mandibular right cuspid
region

2 = Maxillary incisal
region

TABLE X

INCIDENCE OF MICRODONTIA*

	Operative Clinic		Orthodontic Clinic		Total	
Males	2	1.9%	5	4.16%	7	3.11%
Females	2	1.63%	5	2.71%	7	2.28%
Total	4	1.76%	10	3.28%	14	2.63%

* Consisted entirely of maxillary lateral incisor teeth

TABLE XI

INCIDENCE OF INVAGINATION

	Operative Clinic		Orthodontic Clinic		Total	
Males	1	0.95%	3	2.5%	4	1.77%
Females	5	4.09%	4	2.17%	9	2.94%
Total	6	2.64%	7	2.3%	13	2.44%

TABLE XII

TOOTH SUMMARY OF INVAGINATION

Tooth	Operative Clinic		Orthodontic Clinic		Total
	Males	Females	Males	Females	
<u>1/</u>	-	4	-	-	4
<u>/1</u>	-	3	1	-	4
<u>2/</u>	1	1	2	4	8
<u>/2</u>	1	2	3	3	9
Total	2	10	6	7	25

TABLE XIII

INCIDENCE OF DILACERATION

	Operative Clinic		Orthodontic Clinic		Total	
Males	11	10.47%	9	7.5%	20	8.88%
Females	15	12.29%	14	7.6%	29	9.47%
Total	26	11.45%	23	7.56%	49	9.22%

TABLE XIV

INCIDENCE OF ECTOPIC POSITION OF PERMANENT TOOTH GERM(MAXILLARY AND MANDIBULAR SECOND BICUSPID)

	Operative Clinic		Orthodontic Clinic		Total	
Males	5	9.61%	5	7.24%	10	8.26%
Females	7	14.58%	16	15.09%	23	14.93%
Total	12	12%	21	12%	33	12%

TABLE XV

TOOTH SUMMARY OF DILACERATION

Tooth	Operative Clinic		Orthodontic Clinic		Total
	Male	Female	Male	Female	
<u>2</u> /	1	2	1	2	6
<u>3</u> /	-	2	-	4	6
<u>4</u> /	-	1	2	3	6
<u>5</u> /	-	1	-	3	4
<u>6</u> /	-	4	-	-	4
<u>7</u> /	-	1	-	-	1
<u>/2</u>	2	2	1	1	6
<u>/4</u>	3	-	3	2	6
<u>/5</u>	1	1	3	3	8
<u>/6</u>	-	3	-	1	4
<u>/2</u>	1	-	-	1	2
<u>/3</u>	-	-	-	1	1
<u>/4</u>	-	1	-	-	1
<u>/5</u>	-	-	-	1	1
<u>/6</u>	1	1	1	-	3
<u>/7</u>	-	1	-	-	1
<u>4</u> /	-	-	1	-	1
<u>5</u> /	1	-	-	-	1
<u>6</u> /	1	1	1	-	3
<u>7</u> /	1	1	1	-	3
Total	11	21	12	22	66

TABLE XVI

TOOTH SUMMARY OF ECTOPIC POSITION OF SECOND PREMOLARS

		5/		5		5/		5		Total
Ectopic Position		M.	F.	M.	F.	M.	F.	M.	F.	
Operative Clinic	Rotation	-	-	-	-	1	1	1	1	4
	Distal Inclination	-	-	-	-	1	6	4	5	16
	Mesial Inclination	-	-	-	1	-	1	-	-	2
Orthodontic Clinic	Rotation	-	1	-	1	-	2	-	-	4
	Distal Inclination	1	4	1	3	2	6	-	5	22
	Mesial Inclination	1	-	1	1	-	-	-	2	5
Total	Rotation	-	1	-	1	1	3	1	1	8
	Distal Inclination	1	4	1	3	3	12	4	10	38
	Mesial Inclination	1	-	1	2	-	1	-	2	7

TABLE XVII

INCIDENCE OF IMPACTED MAXILLARY CUSPIDS

	Operative Clinic		Orthodontic Clinic		Total	
Males	2	1.9%	2	1.66%	4	1.77%
Females	1	.81%	14	7.6%	15	4.9%
Total	3	1.33%	16	5.26%	19	3.57%

TABLE XVIII

INCIDENCE OF HYPOPLASIA OF THE ENAMEL

	Operative Clinic		Orthodontic Clinic		Total	
Males	-	-	5	4.16%	5	2.22%
Females	2	1.63%	3	1.63%	5	1.63%
Total	2	0.88%	8	2.63%	10	1.88%

TABLE XIX

INCIDENCE OF MULTI-ROOTED TEETH

	Operative Clinic		Orthodontic Clinic		Total	
Males	5	9.43%	2	3.92%	7	6.73%
Females	1	1.35%	1	1.28%	2	1.31%
Total	6	4.72%	3	2.32%	9	3.51%

TABLE XX

TOOTH SUMMARY OF MULTI-ROOTED TEETH

Tooth	Operative Clinic		Orthodontic Clinic		Total
	Males	Females	Males	Females	
Maxillary					
1st Premolar	2	1	-	-	3
2nd Molar	-	-	-	2	2
Mandibular					
Central	1	-	-	-	1
Cuspid	1	-	-	-	1
1st Molar	1	-	1	-	2
2nd Molar	-	-	1	1	2
	5	4	2	3	11

4. Discussion

In order to test the accuracy of radiographic diagnosis for this study a sample of 100 radiographs was compared with other available data. Table II shows that variability in the diagnosis of enamel hypoplasia and abnormal tooth size was recorded. The enamel hypoplasia, which consisted of a slight pitting, was such that it was not discernable radiographically. The tooth of abnormal size, however, was a border-line case. It was therefore noted that the radiographic interpretation was sufficient for the purpose of this survey.

In the present study of 531 children, 198 were observed to have dental anomalies of form or eruption. These are summarised in Table III.

Congenital absence of teeth (Tables IV, VII and VI) was found in 5.5% of children examined. In these twenty-nine cases a total number of fifty-four units were found to be missing, the most frequent being the mandibular second bicuspid (39.1%) and the maxillary lateral incisor (27.7%). These findings compare with those of Dolder (1937), Sabes and Bartholdi (1962) and Brown (1957), although the high percentage (15.8%) of mandibular central incisors does not. In the present study, missing teeth occurred more frequently in females than in males (34:20), a finding which supports that of Castaldi et al (1966).

The finding of 2.3% of children with supernumary teeth (Tables V and VIII) agrees with the results of Clayton (1956). These twelve children presented a total of seventeen supernumary teeth, thirteen of which were in the maxillary incisor region. Of these, four were mesiodens, three being inverted and one erupting normally.

As was expected from the pilot sample, the percentage of children showing hypoplastic defects of the enamel (1.9% - Table XVIII) was considerably below that recorded by Barnard (1966). He found, through clinical examination, 6-8% amongst 2,429 New South Wales school children. Similarly, the results for abnormality of tooth size (Table X) would be expected to be greater than those recorded here. Microdontia, which was found to be confined to the maxillary lateral incisors, showed an incidence of 2.63%. No cases of macrodontia were recorded.

Ectopic positions of the developing second premolars (Tables XIV and XVI) were found in 12% of the mixed dentition, of these the greater proportion (71.6%) were distally inclined. The remainder were divided evenly between mesial inclination and rotation. During the course of the survey several series of radiographs were examined in which the distally inclined mandibular second premolar teeth erupted into a distal position subsequent to the loss of the mandibular first permanent molar. Loss of these molars had removed the guide planes which result

in the ectopically inclined tooth erupting into its normal position in the arch.

Anomalies capable of causing malposition of the teeth were found to be of greater incidence in those children examined from the orthodontic clinic, the greatest difference being recorded in the incidence of impacted maxillary cuspids (Table XVII). This anomaly was most frequent in the girls of this group. The possible significance of this difference between males and females of the same group is beyond the scope of this investigation. On the other hand, little difference was found between the two groups of children exhibiting dilaceration (Tables XIII and XV), invagination (Tables XI and XII), and multi-root formation (Tables XIX and XX).

Rarer anomalies (Table III) such as gemination (one case), lack of root formation (two cases) and ankylosis (two cases) were restricted to the children examined from the orthodontic clinic. An interesting feature was the successful eruption of a maxillary central incisor despite the complete lack of root formation (Fig. 11). Two cases of odontomes were observed, one being in each group.

In the four cases of ectopic eruption (Table III), two involved transposition of the maxillary cuspid with the first premolar and one with a maxillary lateral incisor. The fourth case was the eruption of a mandibular lateral incisor distal to the position of the cuspid. All cases (transposition) were found

in the group from the orthodontic clinic.

Although the findings in this study resemble somewhat the findings in other studies, reliable comparisons cannot be made because of different age ranges and because of the differing method in which the sample was collected. For this reason also, these figures may not be pertinent to other groups of New South Wales school children.

Summary

The normal development of the deciduous and permanent teeth has been discussed, together with the identification of anomalies of form and eruption precipitated by disturbances at particular stages during this development.

The literature discussing the clinical aspects of each anomaly was then reviewed.

In the present investigation dental anomalies of form and eruption were identified, by radiographic examination, in the permanent and mixed dentitions of 531 New South Wales children between the ages of six and fourteen years.

The material was obtained from the Graduate Orthodontic Clinic (♂ 120, ♀ 184) and the Operative Clinic (♂ 105, ♀ 122) of the United Dental Hospital, Sydney.

Preliminary comparison (100 cases) of radiographic identification of these anomalies to other diagnostic data showed that mild enamel hypoplasia, pitting of the labial surface, may not be evidenced; also abnormal sized teeth may not be recognised. This led to the conclusion that incidence

underestimated ✓
figures would be below normal in both cases.

A summary of results found in this investigation is contained in Table III.

Accurate comparison cannot be made with the results of other studies because of the variations in age range and the method by which the sample was taken.

The purpose of the investigation, in determining the incidence of these anomalies, was to facilitate comparison with other anomalous oro-facial conditions in which the dental irregularities occur, and to act as a pilot study for a larger survey.

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