Essentials of Dental Surgery and Pathology

R. A. Cawson

牙体外科和病理学基础

THIRD EDITION

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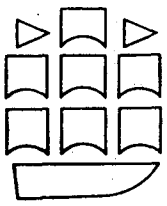

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Contents

1. Disorders of development of the teeth and related tissues

The ideal dentition, that is of perfect appearance and function, depends both on the formation of teeth normal in structure and number, and also their eruption at the appropriate time into their correct positions and relationship with each other.

The function or appearance of the dentition may therefore be impaired by such obvious causes as defective structure of the teeth or irregularities in their position, due to disordered eruption or other factors.

In view of the complexity of the organisation of the development of the teeth, serious defects of structure are remarkably uncommon. By contrast, disorders of occlusion due to irregularities of the teeth in the arch or abnormal relationship of the arches to each other are so common that their treatment has become a specialty in its own right.

The main groups of disorders affecting development of the dentition may be summarized as follows:

1. **Abnormalities in the number of teeth**
   - Anodontia or hypodontia
   - Anodontia or hypodontia associated with systemic disorders
   - Additional teeth. (Hyperdontia)

2. **Disorders of eruption of the teeth**
   - Local obstructions to eruption
   - General disorders of eruption.

3. **Defects of structure of the teeth**
   - Disorders of development of the enamel
   - Disorders of development of the dentine.

4. **Developmental anomalies of several dental tissues**
   - Odontomes.

ABNORMALITIES IN THE NUMBER OF TEETH

These disorders are usually very minor, affecting the dentition alone and without any apparent systemic abnormality.

Isolated hypodontia or anodontia.
Failure of development of one or two teeth is relatively common. The defect is often hereditary. The teeth most frequently missing are third molars, second premolars or maxillary second incisors.

Absence of third molars can be a disadvantage if first or second molars, or both, have been lost as a result of caries. The absence of lower premolars creates a problem if there is already disparity in size between an underdeveloped mandible and a normal upper arch. In other circumstances the absence of these teeth may have little or no noticeable effect.

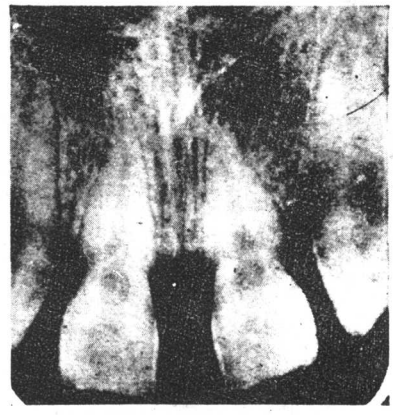

Fig. 1.1 Congenital absence of lateral incisors with spacing of the anterior teeth.

Absence of lateral incisors can sometimes be a conspicuous defect, especially in a girl, because the large and pointed canines come into position in the front of the mouth beside the central incisors. It is usually impossible to prevent the canine from erupting into this empty space, even if the patient is seen early. It is also very difficult to make room between the centrals and canines by orthodontic means to replace the laterals. An attempt has often therefore to be made to disguise the shape of the canines by stoning them down.

Total failure of a complete dentition is exceedingly rare but cases are occasionally seen of absence of the permanent dentition and this may happen in a family who show minor degrees of hypodontia. In such cases of anodontia the deciduous dentition is retained for many years beyond its normal period of function, but when these deciduous teeth become excessively worn or too much damaged by caries then they must be replaced by dentures.

Hypodontia or anodontia associated with systemic defects

Anhidrotic (hereditary) ectodermal dysplasia. This is a rare genetically determined disorder of ectodermal development. Males are mainly affected and there are defects or failure of development of many structures of ectodermal origin. In extreme cases there may be complete failure of development of the dental lamina so that no teeth form. More often the dental lamina develops incompletely; many or all the deciduous teeth form but thereafter development seems to stop and there are few or no permanent teeth. Such teeth as form are usually of somewhat simplified shape, being peg-shaped or of simple conical form.

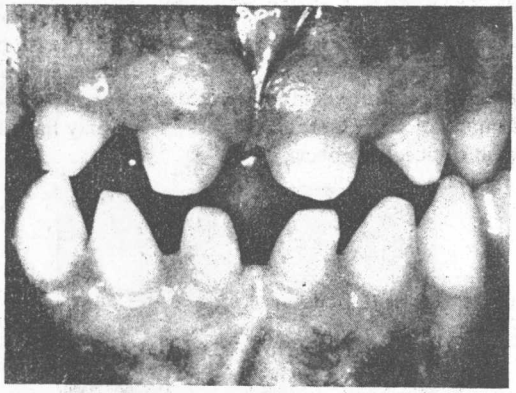

Fig. 1.2 Ectodermal dysplasia. The teeth are deficient in number and of simple conical form. Defects of the hair and nails were associated.

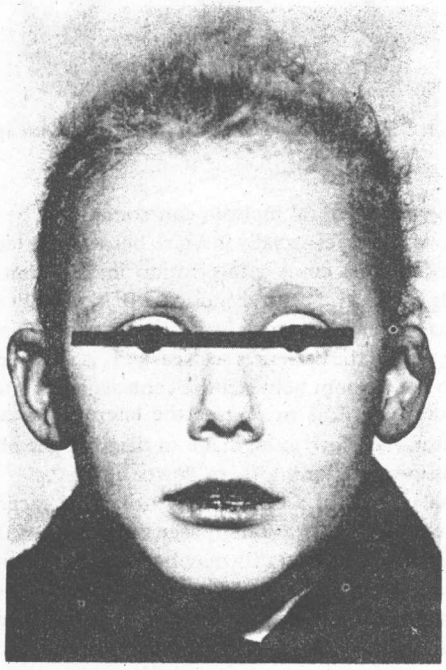

Fig. 1.3 Anhidrotic ectodermal dysplasia. This shows the typical fine and scanty hair. There is also scaling of the lower lip.

In severe cases where one or both dentitions are completely absent the alveolar process, without teeth to support, fails to develop. The alveolar ridges and the profile resemble those of an elderly person because of the gross loss of vertical dimension.

In most cases the hair is fine and somewhat sparse, but even in these cases examination usually shows a very thin or almost bald area in the tonsural region. In more severe cases the hair is represented by little more than thin downy fluff, the skin is smooth and shiny, and dry due to the absence of sweat glands. Heat is therefore poorly tolerated. The finger nails are usually also defective.

All that can be done from the dental viewpoint for these children is to fit dentures to improve the patient's appearance and to improve mastication. Dentures are usually well tolerated by children.

Other conditions associated with hypodontia

There are several rare syndromes where hypodontia is a feature, but the only common one is Down's syndrome (Mongolism). In this condition there seems to be no defined pattern of hypodontia. One or more third molars are absent in over 90 per cent of these patients, while absence of individual teeth scattered about the arch is also common. Total anodontia has also been reported, but is rare.

Palatal clefts are another developmental disorder associated with hypodontia.

Additional teeth. Hyperdontia

Additional teeth are relatively common. They are usually of simple conical shape (*supernumerary teeth*) but may resemble teeth of the normal series (*supplemental teeth*). These are the result of excessive but organised growth of the dental lamina of unknown cause.

Additional (supplemental) teeth. Occasionally a supplemental maxillary incisor or premolar and rarely a fourth molar develops.

Supernumerary teeth. Conical or more severely malformed additional teeth are commoner than supplemental teeth and are usually seen in the maxillary incisor region. Occasionally they form in the midline (mesiodens), particularly of the maxilla, or even more rarely in the mandible.

Effects and treatment. Additional teeth usually erupt in an abnormal position labial or buccal to the arch, creating a stagnation area with caries or periodontal disease as a consequence. In other cases a supernumerary tooth

may prevent a normal tooth from erupting. In the vast majority of cases these additional teeth should be extracted.

Fig. 1.4 Additional teeth; there are two additional premolars. One has erupted partially; the other remains embedded within the jaw.

Syndromes associated with hyperdontia

These are all rare but probably the best known is cleido-cranial dysplasia. This condition is also discussed in Chapter 15, but the feature relevant to the present context is the development of many supernumerary teeth. These may be so crowded as to become distorted, as shown in Fig. 15.12. In addition there is a widespread failure of eruption, with the consequence that most of these teeth remain buried in the jaw, producing pseudo-anodontia.

DISORDERS OF ERUPTION

The eruption of deciduous teeth starts at about six months, usually with the appearance of the lower incisors, and is complete by about two and a half years. The eruption of many teeth or of the whole dentition may be delayed or fail altogether but this is very rare. More often a single tooth is prevented from erupting by some local obstruction. In the case of the permanent dentition delay in eruption of a tooth or, more commonly, too early loss of a deciduous predecessor tends to cause irregularities because movement of adjacent teeth closes the available space.

The times at which the deciduous teeth should appear is shown in Table 1.1 but there is considerable individual variation; a delay of six months or even longer is not unusual.

Delayed eruption associated with skeletal disorders

Delayed eruption of the teeth is associated with metabolic diseases affecting the skeleton, particularly *cretinism* and *rickets*. Though these diseases are now uncommon, they are the main causes of delayed eruption of many teeth. Treatment of these diseases usually restores the eruption rate to normal.

In the rare disease *cleido-cranial dysplasia*, as mentioned earlier, most of the permanent teeth may fail to erupt, or their eruption may be long delayed.

In severe *hereditary gingival fibromatosis* eruption may apparently fail merely because the teeth, although normally erupted, are buried in the excessive fibrous gingival tissue and only their tips show in the mouth.

In *familial fibrous dysplasia*, described in Chapter 15, several teeth may be displaced by the proliferating masses of connective tissue containing giant cells and are prevented from erupting. The number of teeth affected in this way depends on the severity of the disease and in some cases both mandibles and maxillae of both sides are affected.

LOCAL FACTORS AFFECTING ERUPTION OF DECIDUOUS TEETH

Having no predecessors, the deciduous teeth are not

Table 1.1 Chronology of tooth development

Tooth	*Calcification begins*	*Appearance in mouth*
Deciduous		
Incisors	4 months (fetal life)	6–9 months
Canines	5 months (fetal life)	16–18 months
1st molars	6 months (fetal life)	12–14 months
2nd molars	6 months (fetal life)	20–30 months
Permanent		
Incisors	3–4 months (upper lateral incisor 10–12 months)	Lower 6–8 years Upper 9–7 years
Canines	4–5 months	Lower 9–10 years Upper 11–12 years
Premolars	1½–2½ years	10–12 years
1st molars	Birth	6–7 years
2nd molars	2½–3 years	11–13 years
3rd molars	7–10 years	17–21 years

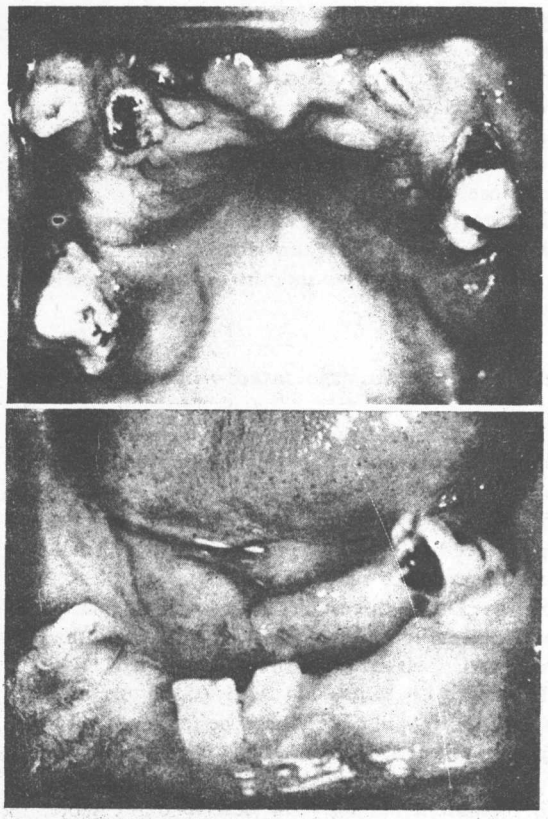

Fig. 1.5 Cleido-cranial dysplasia. The upper and lower jaws of a patient of forty-one. Most of the missing teeth are still embedded in the jaws as shown in the radiographs in Chapter 15.

often prevented from erupting. Occasionally an eruption cyst may overlie a tooth and may need to be incised.

LOCAL FACTORS AFFECTING ERUPTION OF PERMANENT TEETH

A permanent tooth may be prevented from coming into position by such causes as the following:

Loss of space. Early loss of deciduous teeth may allow adjacent teeth to drift together. The lower second premolars are the teeth most frequently displaced by this cause.

Abnormal position of the crypt. The lower third molar and the upper canine are most often misplaced and fail to erupt.

Overcrowding. Inadequate space in the alveolar ridge for the teeth is an important predisposing cause of delayed eruption, especially of lower third molars.

Supernumerary and supplemental teeth. One of these may erupt before, and occupy the space of a tooth of the permanent series.

Dentigerous cysts. The tooth involved in the cyst is displaced away from the alveolus and is prevented from erupting.

Retention of a deciduous tooth. A deciduous tooth occasionally becomes ankylosed to the bone. Resorption may very rarely be delayed by periapical infection, but these teeth are usually shed normally.

Fig. 1.6 Impaction of a premolar. Eruption has been prevented by closure of the space between first premolar and molar, following early loss of the deciduous molar.

The treatment depends on the circumstances but room has usually to be made for the unerupted tooth by orthodontic means or by extractions.

When a deciduous tooth is retained longer than usual,

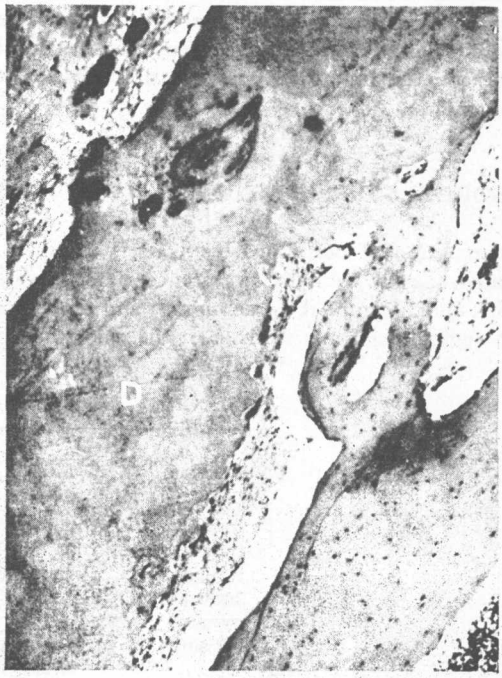

Fig. 1.7 Ankylosis of a deciduous tooth. Resorption is an intermittent process: periods of osteoclastic activity are followed by partial repair. Occasionally excessive repair tissue (A) may form, uniting the dentine (D) to the bone (B) of the jaw. Below the point of ankylosis osteoclasts are actively resorbing the dentine.

radiographs should be taken to make sure that there is a permanent successor before extracting the deciduous tooth.

If the buried tooth is causing symptoms or partially erupts and becomes infected, it may have to be removed; mandibular third molars are the commonest source of trouble of this sort. The treatment of these teeth and of misplaced maxillary canines is discussed among the problems associated with extractions.

Changes affecting buried teeth

Teeth may occasionally remain buried in the jaws, for one of the reasons mentioned earlier, for many years without becoming infected or causing symptoms. The roots of these teeth may undergo hypercementosis or they may become resorbed. Enamel usually resists resorption and if the crown of the buried tooth comes into contact with the roots of an adjacent tooth, the roots are resorbed and the enamel remains undamaged. Similar changes may affect a tooth in a dentigerous cyst. Occasionally the crown becomes resorbed during the course of many years (see Chapter 5).

DEFECTS OF STRUCTURE: HYPOPLASIA AND HYPOCALCIFICATION

Minor structural defects of the teeth, such as pitting or discolouration, are fairly common, and more serious defects may sometimes be seen. These changes may be of interest as indications of past disease, but only on rare occasions is the disease still active.

Hypoplasia of the teeth is not an important contributory cause of dental caries; hypoplasia due to fluorosis is in fact associated with increased resistance, while hypoplastic teeth due to other causes are not especially prone to caries. The main clinical problem associated with hypoplastic teeth is usually how best to improve their appearance.

The deciduous teeth

Calcification of the deciduous teeth begins about the fourth month of intra-uterine life. Disturbances of metabolism, or infections that affect the fetus at this early stage without causing abortion, are rare. Defective structure of the deciduous teeth is therefore uncommon, but in a few places, such as parts of India, where the fluoride content of the water is excessively high, the deciduous teeth may be mottled.

The deciduous teeth may be discoloured by abnormal pigments circulating in the blood. Severe neonatal jaundice may cause the teeth to become yellow or there may be bands of greenish discolouration. In congenital porphyria, a rare disorder of haemoglobin metabolism, the teeth are red or purple. Tetracycline given during dental development can also cause permanent discolouration.

The permanent teeth

The permanent teeth may be affected by local influences and by systemic diseases. Though the permanent teeth are more frequently hypoplastic than the deciduous teeth, the incidence of serious structural defects is small.

Local causes

Infection. Local periodontitis of a deciduous tooth, especially a molar, may damage the underlying permanent successor. This tooth may be malformed to a greater or lesser degree. The enamel is commonly pitted and sometimes the affected part of the crown may be much smaller than the rest.

Systemic disease

a. Hereditary disorders
   (i) Amelogenesis imperfecta
      Hereditary enamel hypoplasia
      Hereditary enamel hypocalcification
   (ii) Dentinogenesis imperfecta
b. Infection
   Congenital syphilis
c. Severe disturbances of metabolism
   Fevers and other childhood infections, rickets and hypoparathyroidism
d. Tetracycline pigmentation
e. Fluorosis

Amelogenesis imperfecta

There are several genetically determined disorders of enamel formation. Genetic factors act more or less severely throughout the whole duration of amelogenesis. Characteristically therefore all the teeth may be affected and defects involve a large part of, or are randomly distributed throughout the enamel. In contrast, exogenous factors affecting enamel formation (with the important exception of fluorosis) tend to act only for a short time and leave a pattern of defects only in that part of the enamel formed during the course of the disease.

Amelogenesis imperfecta can be divided into two main groups. *Hereditary enamel hypoplasia* is characterised by defective matrix formation but normal calcification of the enamel, while in *Hereditary enamel hypocalcification* a normal matrix forms but is poorly calcified. Inheritance of these enamel defects follows several different patterns, but it is rarely possible to obtain a clear family history because dental defects in other members of the family have not been noticed or have been confused with caries, i.e. 'the teeth came through decayed'.

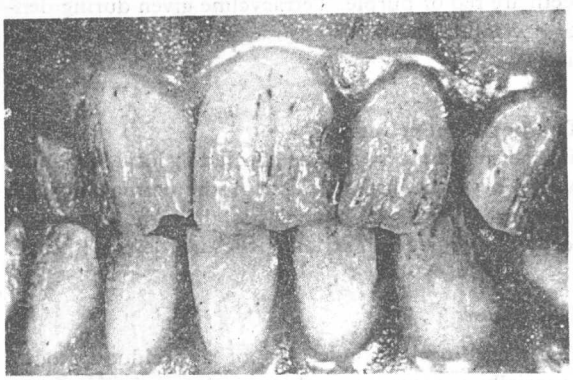

Fig. 1.8 Amelogenesis imperfecta—hypoplastic, sex-linked dominant type. This shows the vertically ridged enamel in a female.

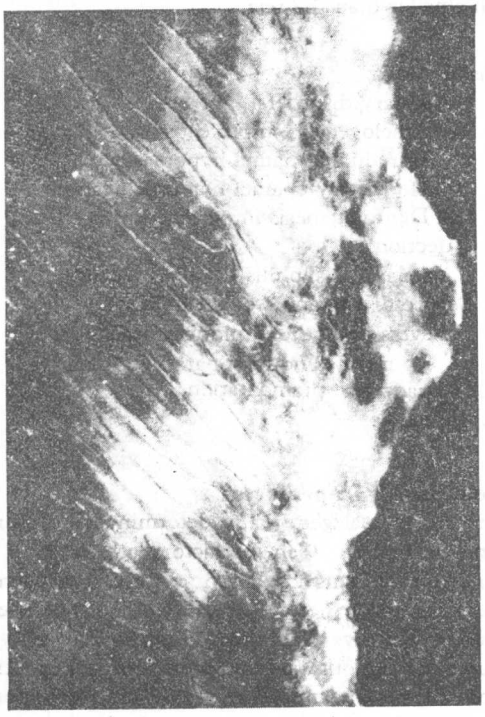

Fig. 1.9 Sex-linked dominant type enamel hypoplasia. This and the three subsequent pictures show the defect as it affects males. The exceedingly thin enamel lacks any prismatic structure.

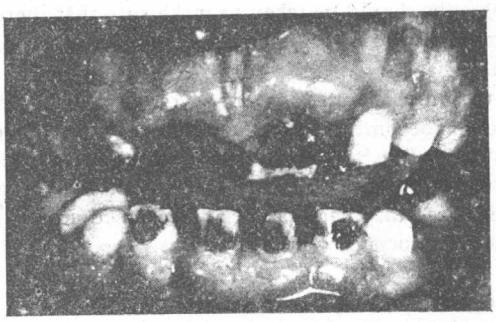

Fig. 1.10 Enamel hypoplasia. A patient with a similar defect to that in the previous picture. The darkening of the incisors is partly due to staining and partly to silver nitrate applied to prevent decay.

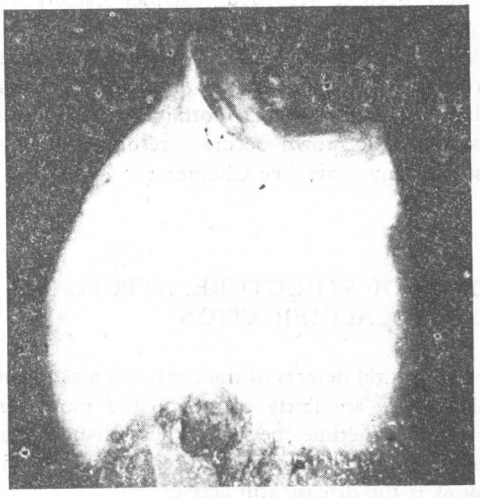

Fig. 1.11 Enamel hypoplasia. The premolar has a cap of enamel so thin that the shape of the tooth is virtually that of the dentine core.

Fig. 1.12 Enamel hypoplasia. Radiographs of the same patient showing the deficiency of enamel.

HEREDITARY ENAMEL HYPOPLASIA

The main defect is in the formation of the matrix. The enamel is randomly pitted, grooved or very thin, but such as there is, is hard and translucent. The defects tend to become stained, but the teeth are not especially prone to caries unless the enamel is very thin and easily damaged. In these cases the enamel may lack the normal prismatic structure and appear lamellated or glassy.

There are three main patterns of inheritance. There are autosomal dominant and recessive forms and in addition—a genetic rarity—a sex linked dominant type. The latter is characterised by almost complete failure of

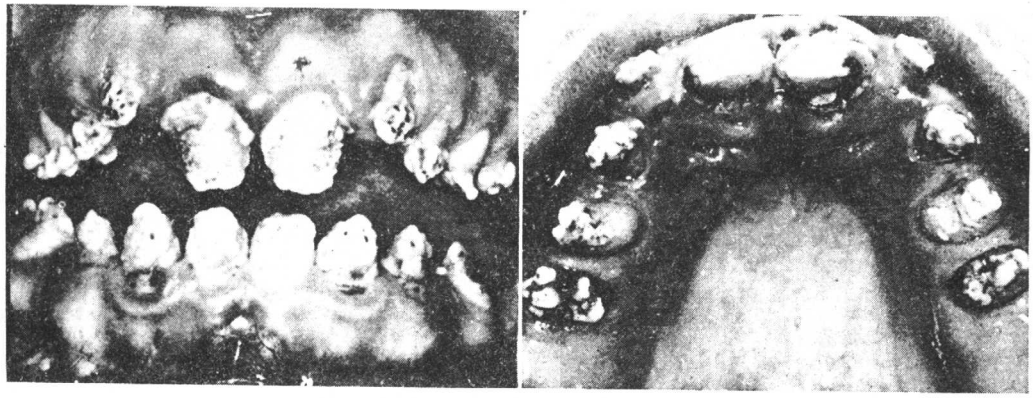

Fig. 1.13 Severe enamel hypoplasia. The enamel consists only of small irregular nodules but is hard and glossy. The pits are stained but the teeth are not carious.

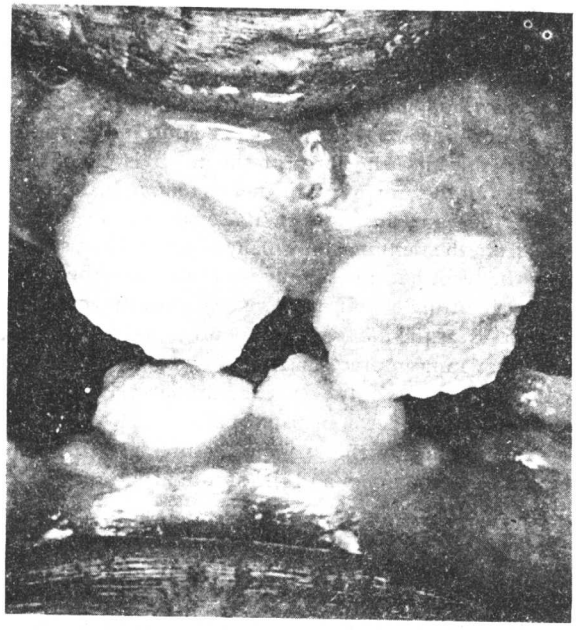

Fig. 1.14 Enamel hypocalcification. The soft, chalky enamel was virtually of normal thickness and form but has chipped away during mastication.

enamel formation in the males, while in females the tissue is vertically ridged (see Figs. 1.8–1.12).

In the United States it has been estimated that the frequency of inheritable enamel hypoplasias is about 1 in 16,000.

HEREDITARY ENAMEL HYPOCALCIFICATION

Enamel matrix is formed in normal quantity and when newly erupted the enamel is normal in thickness and form, but weak and opaque or chalky in appearance.

The enamel is soon chipped away and becomes stained, usually a yellowish colour. The teeth tend to be

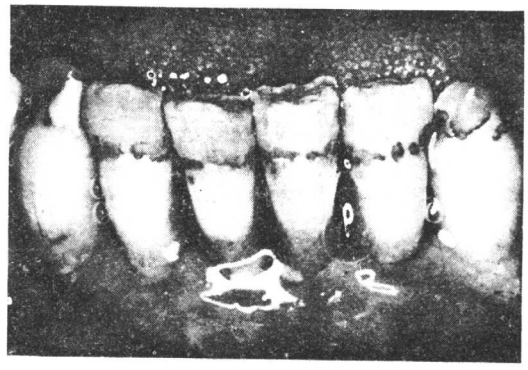

Fig. 1.15 Hypoplasia due to metabolic upset. Unlike the hereditary types of amelogenesis imperfecta the defects are linear and thought to correspond to a short period of amelogenesis disturbed by a concurrent severe illness.

relatively rapidly worn away and the upper incisors develop a characteristic shouldered form due to the chipping away of the thin, soft enamel of the incisal edge.

As with the hypoplastic type of amelogenesis imperfecta, autosomal dominant and recessive patterns of inheritance have been described.

In the United States the hypocalcified type of amelogenesis imperfecta is apparently commoner than the hypoplastic type.

Dentinogenesis (odontogenesis) imperfecta

This is an uncommon hereditary disease, also known as hereditary opalescent dentine, transmitted as an autosomal (simple) dominant. The gene is closely related to that of osteogenesis imperfecta. The dental disorder may be regarded as one of defective odontoblastic activity, and the skeletal disorder as one of defective osteoblastic activity. In osteogenesis imperfecta, bone formation is inadequate and the fragile bones fracture repeatedly when stress is imposed on them.

A patient may be affected by either of these diseases

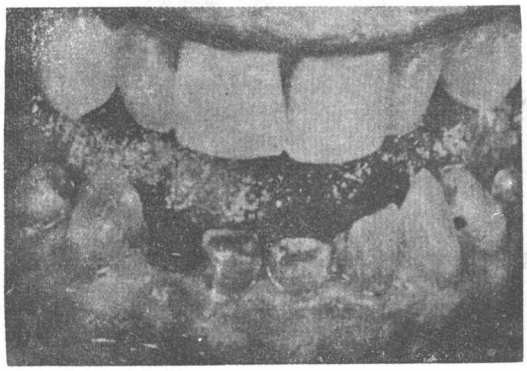

Fig. 1.16 Dentinogenesis imperfecta. The affected teeth are brownish and translucent, but of normal form. The enamel is hard but poorly attached and has chipped off, especially from the lower central incisors. The upper centrals appear unaffected.

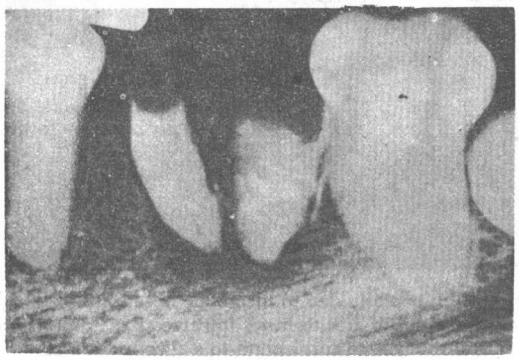

Fig. 1.17 Dentinogenesis imperfecta. Radiographs show the characteristic bulbous crowns, short roots and obliterated pulp chambers.

alone, but in certain families both dentinogenesis imperfecta and osteogenesis imperfecta are present together in affected members. In addition, in some of the patients, the sclera of the eyes may be blue.

Histological features

The dentine is soft and has an abnormally high water content. The earliest formed dentine under the amelo-dentinal junction usually appears normal. The deeper tissue is more defective, tubules become few, calcification incomplete and the matrix imperfectly formed. The pulp chamber becomes obliterated at an early stage and odontoblasts degenerate, but cellular inclusions in the dentine are common. Scalloping of the amelo-dentinal junction is sometimes absent and the enamel tends to split from the dentine. The enamel is normal in typical cases.

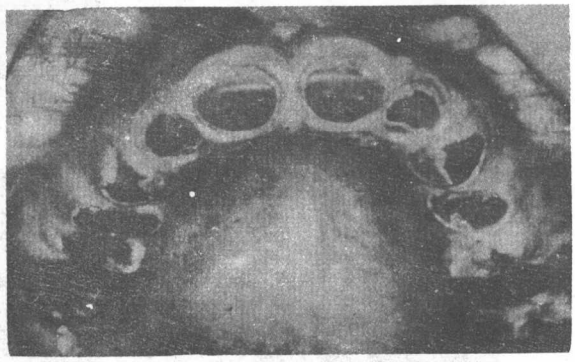

Fig. 1.18 Dentinogenesis imperfecta. A boy of fourteen with complete destruction of the crowns of the teeth by attrition due to progressive chipping away of the enamel and wearing away of the relatively soft dentine. Obliteration of the pulps has prevented their exposure.

Clinical features

The enamel appears normal in texture but the tooth is of a uniform brownish or purplish colour and abnormally translucent. The form of the teeth is essentially normal, but the crowns of the molars tend to be bulbous and the roots are usually short. The enamel tends to chip away from the dentine abnormally easily until in severe cases the teeth become rapidly worn down to the gum. The early fitting of full dentures becomes inevitable in these cases, as the relatively soft dentine and short roots make crowning impractical.

In some patients only a few teeth are severely affected, while the remainder appear normal.

Radiographically the striking features are the obliterated pulp chambers and the stunted roots.

Infection. Congenital syphilis

Prenatal syphilis, the result of maternal infection, may give rise to a characteristic deformity of the teeth, described by Hutchinson in 1858. The effects are due to

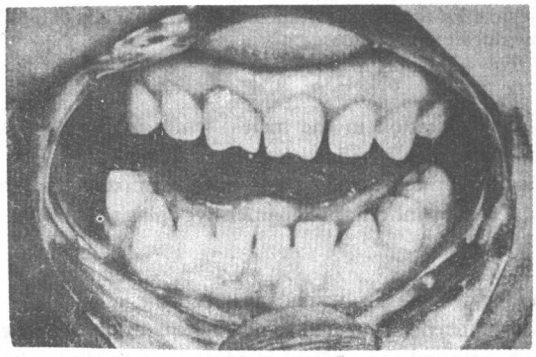

Fig. 1.19 Congenital syphilis; Hutchinson's teeth. The characteristics are the notched incisal edge and the peg shape, tapering fron neck to tip.

direct action of the *Treponema pallidum* within the dental follicle and it has been suggested that the organisms provoke a typical chronic inflammatory reaction and fibrosis of the tooth sac. This was thought to cause hypoplasia by compressing the developing tooth and distorting the ameloblast layer. More recently it has been shown that *Treponema pallidum* causes proliferation of the odontogenic epithelium which bulges into the dentine papilla causing the characteristic central notch.

Clinical features

If the fetus becomes infected at a very early stage, abortion follows. Infants born with stigmata of congenital syphilis are the result of later infection of the fetus and it is the permanent teeth that are affected. The characteristic signs are seen most often in the upper central incisors, less frequently in the first molars and sometimes in the canines and lateral incisors.

The incisors (Hutchinson's incisors) are small, barrel-shaped, and taper towards the tip. The incisal edge sometimes shows a crescentic notch or deep fissure which forms before eruption and can be seen radiographically. An anterior open bite is also a characteristic feature.

The first molars may be dome-shaped (Moon's molars) or may have a rough pitted occlusal surface with compressed nodules instead of cusps (mulberry molars). This defect is exceedingly rare nowadays.

Severe disturbances of nutrition and metabolism

General disturbances of metabolism affecting the development of the teeth may be caused by the acute specific fevers and by infantile gastro-enteritis if sufficiently severe. More specific disorders of metabolism such as rickets, tetany and scurvy are exceedingly rare and do not appear to cause clinically characteristic types of hypoplasia. Unlike inherited forms of hypoplasia only a restricted area of enamel is defective, corresponding to the state of development at the time of the metabolic disturbance. Measles with severe secondary bacterial infection was perhaps the commonest cause of this limited type of dental defect. This seems to have become very much more uncommon, presumably due to the more effective treatment available nowadays (see Fig. 1.15).

Clinically, the commonest type of defect is one or more rows of horizontally disposed pits or grooves across the crown of the incisors. These are usually placed in the incisal third, suggesting that the disorder had its effect during the first year or two of life, at a time when the exanthemata and other infections are most dangerous.

Rickets can cause hypocalcification of the teeth, as shown in Fig. 1.20 but has to be unusually severe for the effects to be apparent.

Idiopathic hypoparathyroidism of early onset is rare but causes ectodermal effects; the reasons for this are not known. The teeth may therefore be hypoplastic, the nails may be defective and there may be complete absence of hair. Patients with early onset idiopathic hypoparathyroidism may later develop other endocrine deficiencies (polyendocrinopathy syndrome).

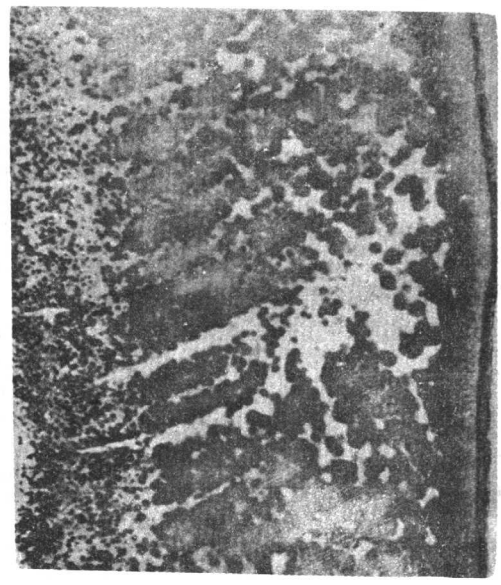

Fig. 1.20 Rickets. In this case of renal rickets defective calcification has left a wide band of predentine and many interglobular spaces (× 45).

Tetracycline pigmentation of the teeth

Tetracycline is taken up by calcifying tissues and the band of tetracycline-stained bone or tooth substance fluoresces bright yellow under ultraviolet light.

The teeth become stained only when tetracycline is given during the period of tooth formation, but when given to the mother during pregnancy, it crosses the placenta and stains the developing teeth of the fetus. More often permanent teeth are stained by tetracycline given during infancy.

The stain is deposited along the incremental lines of the dentine and, to lesser extent, of the enamel. The more prolonged the course of treatment the broader the band of stain, and the deeper the discolouration. It has been suggested (but not proved) that very heavy dosage of tetracycline may cause hypoplasia but usually the teeth are of normal form.

The colour is at first bright yellow, but becomes a dirty brown or grey. The stain is permanent and when the permanent incisors are affected the ugly appearance can only be disguised by fitting jacket crowns later on or by facing the tooth with a composite resin.

When the history is vague the brownish colour of

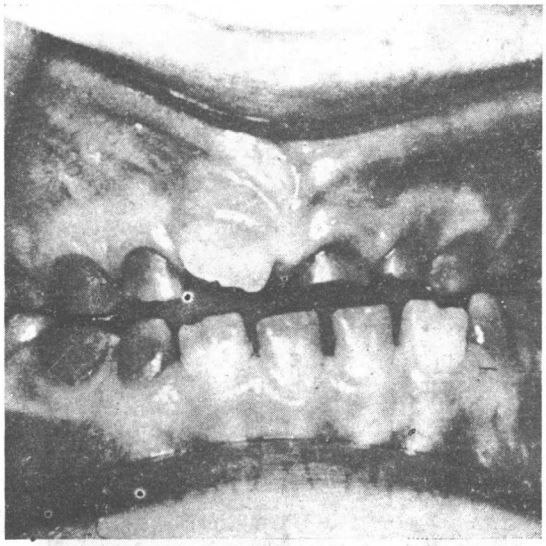

Fig. 1.21a Tetracycline pigmentation. The deciduous teeth, all affected by tetracyclines given to the mother during pregnancy, have acquired the typical greyish brown discolouration. The permanent teeth stained by tetracyclines given during infancy are still yellow but will gradually become brown or greyish.

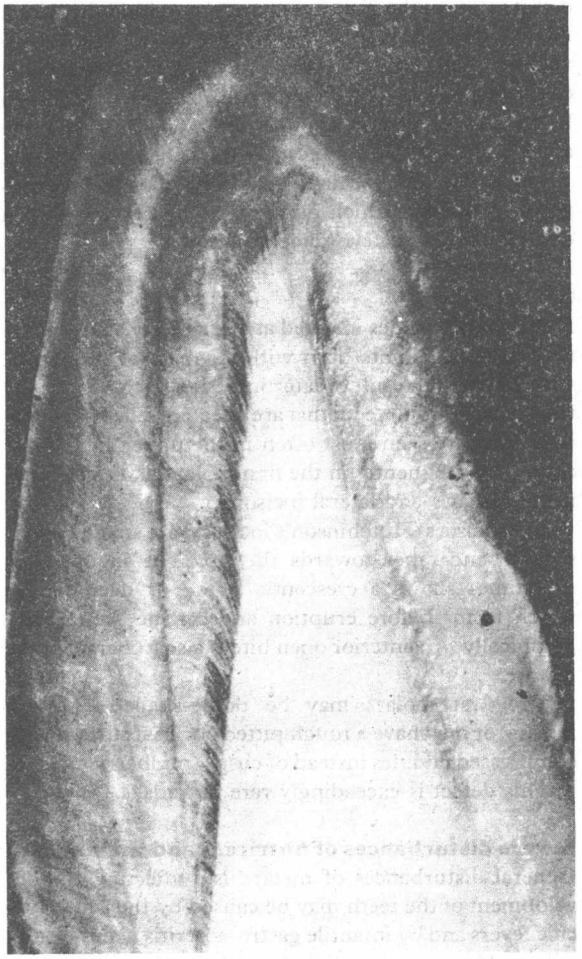

Fig. 1.21b Tetracycline deposition. Under ultraviolet light the whole of the dentine is intensely fluorescent while the incremental lines of the enamel are well demarcated in this undecalcified section of a heavily pigmented incisor.

tetracycline stained teeth must be distinguished from the appearance due to dentinogenesis imperfecta. In dentinogenesis imperfecta the teeth are usually obviously more translucent than normal and in many cases, chipping of the enamel from the dentine can be seen. In the tetracycline-induced defect the teeth appear somewhat *less* translucent than normal and show no structural defect. In very severe cases the intact teeth fluoresce under ultraviolet light. Otherwise the diagnosis can only be confirmed after a tooth has been extracted. An undecalcified section can then be made and the brilliant yellow fluorescence of the tetracycline deposited along the incremental lines can be easily seen. Needless to say, teeth should not be extracted merely for diagnostic purposes.

It is rarely essential nowadays to give tetracycline during infancy as many other equally effective antibiotics are available. The period when tetracycline must be avoided is that when the crowns of the permanent anterior teeth are calcifying, i.e. from approximately the 4th month to the 6th year of childhood.

Dental fluorosis. Mottled enamel

Mottling of the enamel is the most frequently seen and most reliable sign of excessive quantities of fluorides in the drinking water. There are important features of mottling that distinguish it from other forms of hypoplasia of the teeth. These are as follows:

1. Mottling is endemic to areas where fluorides in the drinking water exceed about 2 parts per million, mottling therefore has a geographical distribution.

2. Neighbouring communities with a different (fluoride-free) water supply do not suffer from the disorder.

3. Only those people who have lived in a high-fluoride area during the period of development of the teeth show mottling. The defect is not acquired by visitors to the area.

4. The permanent teeth are affected; mottling of deciduous teeth is rare.

5. Those with mottled teeth have a lower incidence of caries than those from low-fluoride areas with normal teeth.

6. The brown stain seen in severe mottling is acquired after eruption of the teeth.

In its mildest form mottling consists of paper-white, opaque patches in the enamel. Only small areas of enamel are affected, the surface is shiny and otherwise unimpaired; the rest of the enamel is normal. In severe mottling the enamel is opaque, stained brown and pitted; it is brittle and chips easily. It is sometimes difficult to distinguish fluorotic defects from amelogenesis imperfecta (hereditary or idiopathic) clinically when the degree of exposure of the patient to fluoride is unknown.

Pathology

Fluorine combines to form calcium fluorapatite in place of part of the normal hydroxy-apatite in the enamel. Damage to ameloblasts leading to defective matrix formation, is seen only when the concentrations of fluorides are exceptionally high. At intermediate levels (2 to 6 ppm) the matrix is normal in structure and quantity; the form of the tooth is unaffected, but there are patches of incomplete calcification beneath the surface layer. The enamel is opaque in these areas due to high organic and water content but is resistant to caries. Where there are high concentrations of fluorides (over 6 ppm) the enamel ... is pitted and brittle, while staining is more severe and widespread.

Clinical features

There is considerable individual variation in the effects of fluorides. A few patients may have mild mottling after exposure to relatively low concentrations of fluorides while others exposed to relatively high concentrations may escape their effect.

Changes due to mottling are graded as follows:

Very mild. Small opaque areas on the tooth involving less than 25 per cent of the surface.

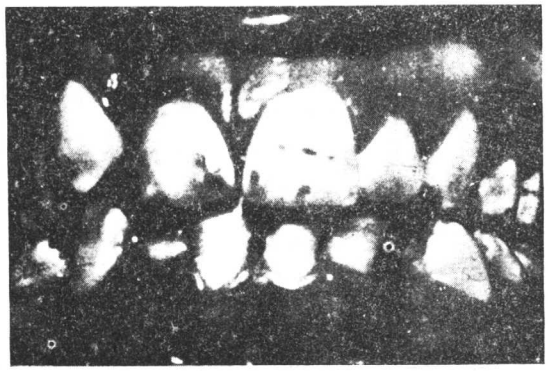

Fig. 1.22 Fluorosis. Moderately severe mottling in a patient from Maldon (Essex). All the teeth show irregular opaque white areas and some brown staining. The enamel surface is intact.

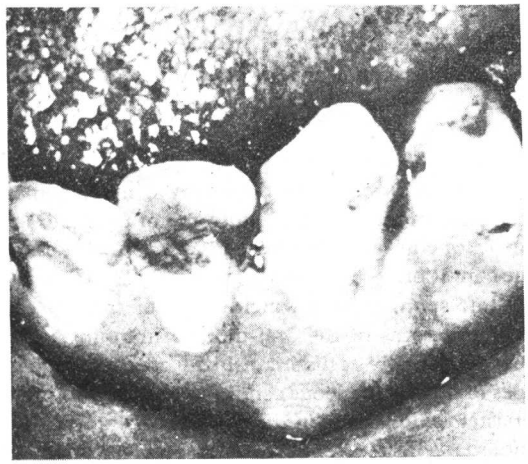

Fig. 1.23 Mottled enamel. Close-up view to show extensive paper white areas due to hypomineralisation beneath the surface and brown staining associated with minute defects in the surface.

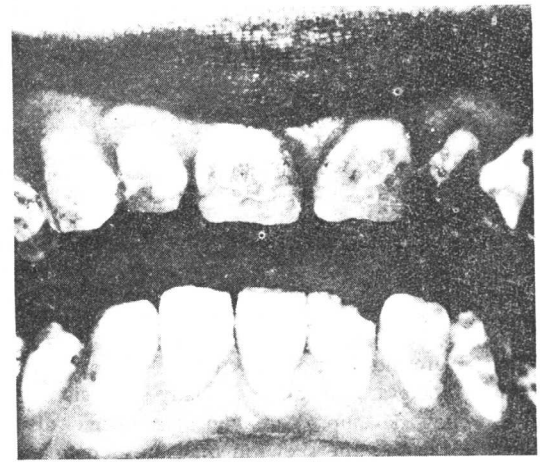

Fig. 1.24 Severe mottling. The entire enamel is opaque or brown and severely pitted; the increased brittleness has caused the incisal edges to be chipped. The patient is from Naples.

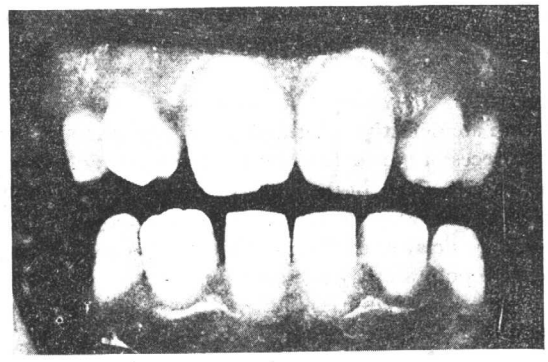

Fig. 1.25 Non-fluoride defects. The opaque flecks are distributed uniformly over the surface of the enamel.

Mild. Opaque areas involving up to 50 per cent of the surface.

Moderate. The whole of the enamel surface may be affected with chalky white areas or yellowish or brown staining. Surfaces subjected to attrition become worn.

Severe. The enamel is grossly defective, opaque, pitted, stained brown and brittle in texture. The tooth may have a corroded appearance.

Table 1.2 Fluoride concentration and its effects

Fluoride Concentration	*Effects*	
Less than 0.5 ppm	Up to 6 per cent of patients show 'very mild' or 'mild' defects.	Inconspicuous
0.5 to 1.5 ppm	At the upper limit of this range 22 per cent show 'very mild' and 3 per cent show 'mild' defects.	
2.5 ppm	Over 50 per cent show 'very mild' or 'mild' defects; nearly 10 per cent show 'moderate' or 'severe' defects.	Apparent
4.5 ppm	Nearly all patients affected in some degree; 46 per cent have 'moderate' and 18 per cent 'severe' defects.	Disfiguring
6.0 ppm and more	All patients affected. 50 per cent severely.	

With severe mottling of the enamel, other effects of excessive intake of fluorides, especially sclerosis of the skeleton may develop. Radiological evidence of increased density of the skeleton may be seen in areas where the fluoride content of the water exceeds 8 ppm but is not harmful. Toxic effects of fluorides are discussed in Chapter 7.

The milder degrees of mottling are not easily distinguished from non-specific defects causing opacities in the enamel. There is also evidence that non-specific defects are commoner in low-fluoride areas where the water contains less than 1 ppm of fluorine.

TREATMENT OF HYPOPLASTIC DEFECTS

If teeth are defectively formed there is no remedy, but when necessary the defects can be disguised. This is important in the case of the anterior teeth where the only practical procedure is to prepare jacket crowns. This should be delayed until adult life. In younger patients the large pulp is easily damaged during the preparation of the tooth, and injuries to the teeth are also more frequent than in older patients.

Odontomes

These are due to aberrant development of the dental lamina. The most minor examples, though they are not usually called odontomes, are slight malformations such as an exaggerated cingulum or extra roots or cusps on otherwise normal teeth. All gradations exist between these and composite odontomes where the dental tissues have developed in a completely irregular and haphazard manner bearing no resemblance to a tooth and occasionally forming a large mass.

Odontomes are discussed more fully in Chapter 14.

GENETICALLY DETERMINED ABNORMALITIES OF THE SOFT TISSUES

These are uncommon; they fall into two main groups as follows:

a. *The gingivae*

The main example is *hereditary gingival fibromatosis* discussed in Chapter 6.

b. *The oral mucosa*

White sponge nevus characterised by white oedematous thickening of the oral epithelium and often also of other mucous membranes is inherited as a simple dominant. The condition is discussed in more detail in Chapter 18.

White sponge nevus which is one of the few causes of white lesions of the oral mucosa in young children must incidentally be distinguished from the rare familial types of muco-cutaneous candidosis which also produce persistent oral lesions in the mouths of children. These lesions are of much more limited extent than white sponge nevus.

Epidermolysis bullosa is characterised by vesicle and bulla formation of ectodermal structures particularly of skin

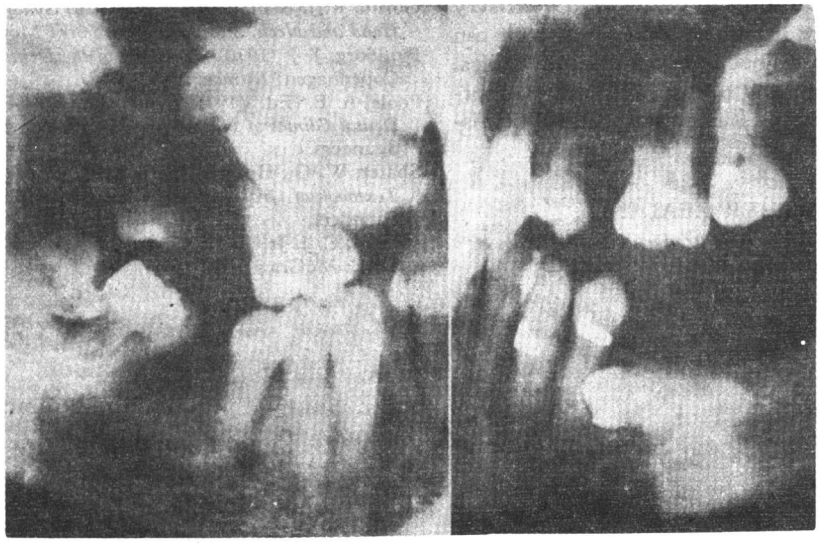

Fig. 1.26 Multiple defects of dental development. On the left in the upper jaw, there is a large composite odontome, above and behind which is the canine. In the lower jaw on the left the third molar is impacted in a mesioangular position behind the second molar. On the right, the second molar lies horizontally, embedded in the jaw and has caused resorption of the root of the tooth in front of it.

and mucous membranes. The bullae are followed by scarring and when this affects the mouth the sulcuses may become obliterated and the tongue tied down to the floor of the mouth. Epidermolysis bullosa shows several patterns of inheritance and because of the vesiculating defect of epithelia, can affect enamel formation causing a characteristic hypoplastic defect of the teeth. This degree of severity and of early onset of the disease is very rarely however compatible with life.

HEREDITARY DEFECTS AFFECTING THE JAWS

For historical reasons hereditary prognathism is the best known of these defects in that the Spanish branch of the Hapsburg royal family showed the defect in striking form as is obvious from contemporary portraits. The disease has been traced through nine generations of this family beginning with Ernst in the 14th century and ending with Karl II in the 17th century.

Cleido-cranial dysplasia. This rare syndrome is inherited as an autosomal dominant but, as with other conditions inherited in this way, isolated cases are also seen due either to mutation or to a gene with poor penetrance.

The syndrome consists of aplasia or hypoplasia of one or both clavicles, exaggerated development of the transverse diameter of the cranium and delayed ossification of the fontanelles. In addition there are usually great numbers of supernumerary teeth and delay or failure of eruption of most of them. Cysts can therefore form round these buried teeth.

Patients are short in stature with long necks and narrow shoulders. The partial or complete absence of the clavicles allows the patient to bring their shoulders together in front of the chest.

The face usually looks small with a depressed nasal bridge, while the cranium shows prominent bossing, particularly on the frontal, parietal and occipital regions. The fontanelles remain open, sometimes for life, and there are usually excessive numbers of wormian bones.

Familial fibrous dysplasia, cherubism. This uncommon disease, inherited as a simple dominant, is characterised usually by symmetrical soft tissue lesions containing many osteoclast-like giant cells, particularly at the angles of the mandibles and in severe cases in the maxillae. The condition is discussed in Chapter 15.

Skeletal anomalies, multiple cysts of the jaws and basal cell naevi. This rare hereditary defect is mainly of interest in consideration of the differential diagnosis of cysts. The disease is important to recognise however as it may be important to warn the patient or parent of the likelihood of development of any of the other anomalies associated with this disorder and also to examine other members of the family for unsuspected cysts which might otherwise cause trouble later on. The condition is discussed in more detail in Chapter 13.