RUSHTON AND COOKE

ORAL HISTOPATHOLOGY

A MANUAL FOR STUDENTS AND PRACTITIONERS OF DENTISTRY

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ORAL HISTOPATHOLOGY

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PREFACE

It is possible in a small book to show and to relate what is happening at a cellular level in the principal disorders of the teeth and neighbouring parts, and that is our intention. To explain in detail why these things are happening, so far as we know this, would require much further discussion of environmental factors and alternative hypotheses than it is the aim of this book to offer. It is hoped, however, that a limited factual presentation will not only assist students and practitioners to comprehend the nature of the changes in the tissues they observe and treat, but will in many arouse the natural desire for a more complete and satisfactory explanation of them than can be provided here or may yet be available. The brief bibliographies offer suggestions for wider reading or indicate the source of photomicrographs previously published. They do not include all the best articles on any aspect, but offer starting points for further enquiry. That they are almost confined to papers in the English language must not be taken as a reflexion on the value of original work published in other languages, but be understood as intended for the convenience of readers using the English tongue.

No authors of a text-book of oral histopathology can be without obligation to the late Dr. R. A. Kronfeld and to Dr. K. H. Thoma. It is a pleasure to express our indebtedness to them and to the leaders of dental research in every land.

We wish to express our particular thanks to Mr. J. E. Hutchinson who has made nearly all the histological preparations and photomicrographs used here; to the Editors of the *British Dental Journal; Oral Surgery, Oral Medicine and Oral Pathology;* the *Journal of Pathology and Bacteriology;* the *Annals of the Royal College of Surgeons of England;* and the *British Journal of Dermatology* who have allowed us to reproduce illustrations used by us in previous papers, of which they hold the copyright; to colleagues who have lent us sections of pathological material or allowed us to reproduce figures from their work, particularly Professors R. V. Bradlaw and A. I. Darling, Drs. G. Gustafson and J. L. Hardwick, Professors R. B. Lucas and E. B. Manley; and to Miss N. Carlton for help in preparation of the manuscript. We are also greatly obliged to Messrs. E. & S. Livingstone for their courtesy and assistance.

M. A. RUSHTON
B. E. D. COOKE

LONDON, 1958

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I

TEETH AND THEIR FORMATIVE ELEMENTS

1. DEVELOPMENTAL ANOMALIES

Developmental anomalies are common and range from minor features scarcely outside normal variation to gross defects as regards number, morphology and structure. The anomalies to be described are those in which defects of structure are important.

STRUCTURAL DEFECTS OF ENAMEL

Genetically Determined: Amelogenesis Imperfecta. Several forms of structural defect of enamel have a familial incidence and are thought

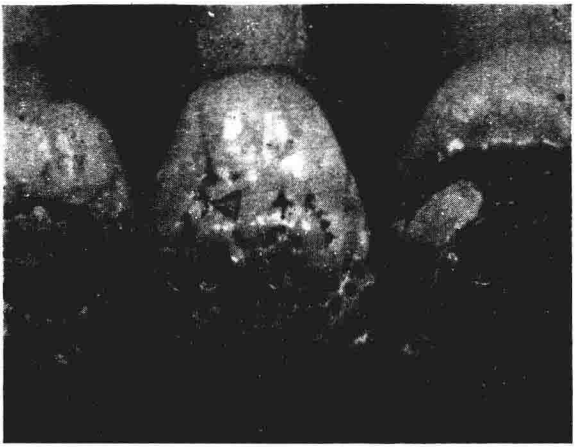

FIG. 1
Hereditary enamel hypoplasia: a moderately severe (female) case showing vertical wrinkles.

to result from genes which are inherited. These disorders are usually divided into two groups (Weinmann *et al.*, 1945): hereditary enamel hypoplasia and hereditary enamel hypocalcification. This is an over-simplification but a convenient one. Various forms can be distinguished by appearance and mode of inheritance (Witkop, 1962). Usually all teeth are affected, though not always equally.

HEREDITARY ENAMEL HYPOPLASIA. Here the enamel is hard but too thin, so that in extreme cases the teeth appear almost denuded of enamel. In less severe cases the surface is pitted or wrinkled (Fig. 1). The teeth suffer quickly from attrition. In ground sections it may be

seen that the quality of the enamel is abnormal: while some areas show the usual rod structure, others have a glassy appearance with fine laminations parallel with the surface (Fig. 2). On decalcification these areas leave a matrix of hyaline, structureless appearance.

HEREDITARY ENAMEL HYPOCALCIFICATION. In these cases the enamel is normal in quantity but incompletely calcified so that it is unduly soft and has a matt surface. After eruption the enamel becomes pigmented buff, orange or brown, and may be quickly chipped and worn

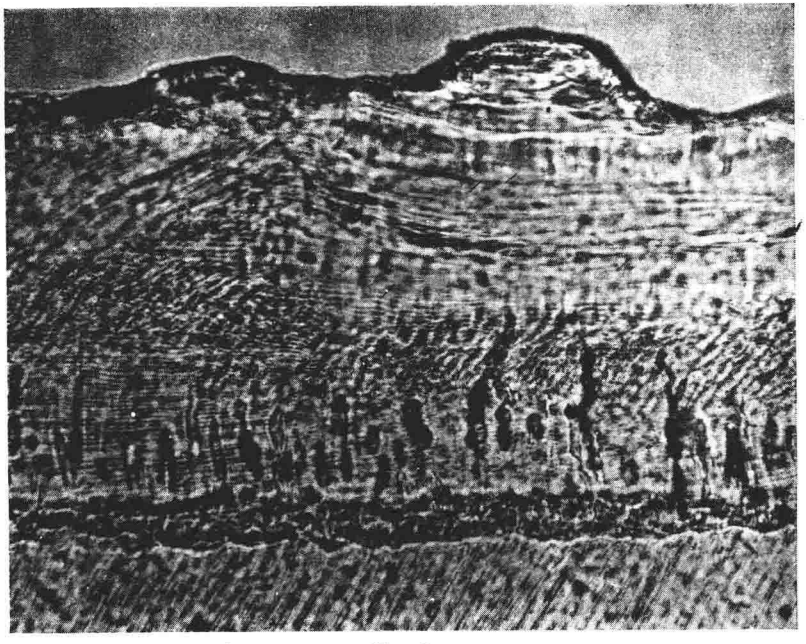

FIG. 2
Hereditary enamel hypoplasia: ground section from a severe (female) case showing thin enamel, of which the superficial layers are of glassy appearance without visible rod structure. × 500.

away. Much of the enamel matrix remains acid-insoluble, as in incomplete maturation, so that it is often preserved in sections of decalcified specimens. The rod pattern of the enamel is approximately normal but a surface layer like a thick calcified cuticle may be present before eruption. Sections of young unerupted teeth show premature degeneration of the enamel organ (Fig. 3).

Metabolic Disorders and General Infections. Disorders of calcium metabolism such as rickets and hypoparathyroidism, if occurring during the period of tooth formation, are likely to result in hypoplastic defects

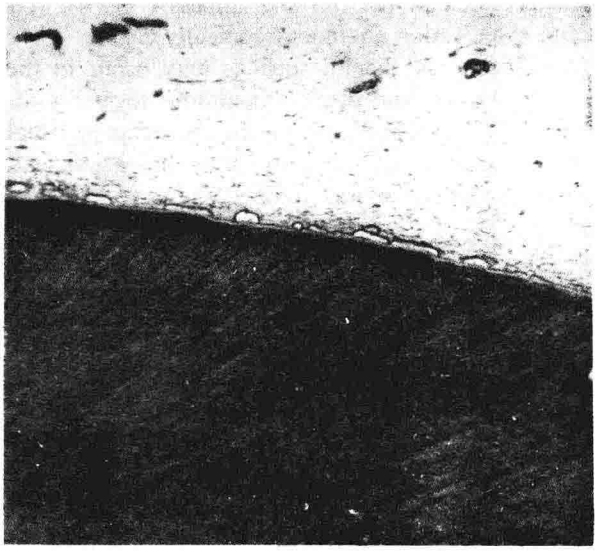

FIG. 3
Hereditary enamel hypocalcification: on a child's unerupted premolar the reduced enamel epithelium has already degenerated, leaving only some hyaline nodules on the surface of the enamel. From a section kindly lent by Professor E. B. Manley. Van Gieson. ×60.

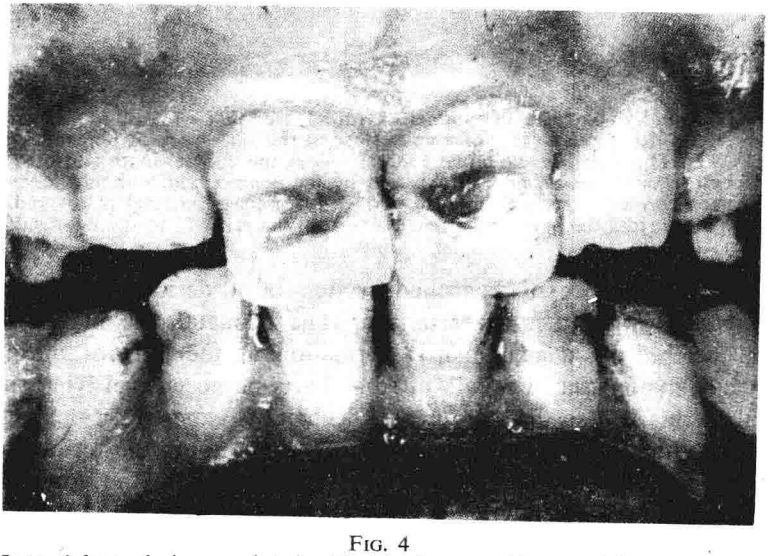

FIG. 4
Enamel hypoplasia associated with tetanic convulsions which occurred at two years of age.

of the enamel formed at the times of illness (Fig. 4). They will also cause defective calcification not macroscopically visible.

Severe disturbances of health such as may occur in the first weeks of life of a premature infant, or exanthematous fevers in childhood may result in hypoplastic pits and grooves in the enamel, usually arranged horizontally. In some cases the cause may be insufficient availability of mineral substances, in others toxic damage to the enamel-forming cells.

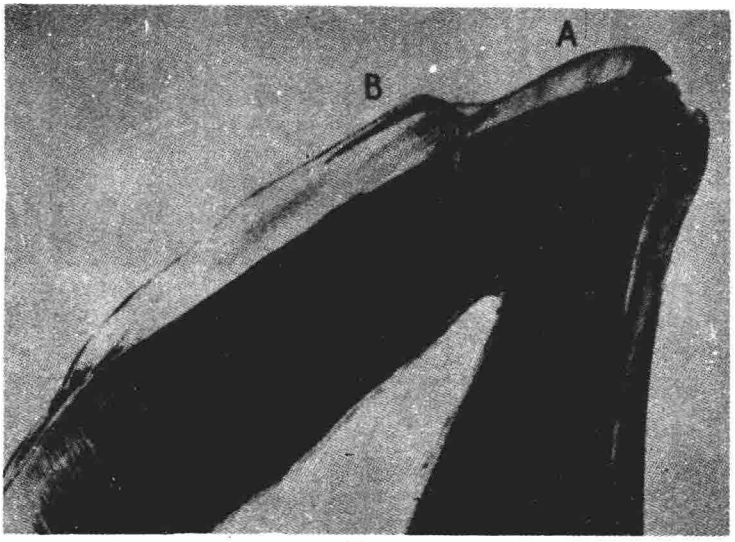

FIG. 5
Neonatal hypoplasia of enamel. Ground section (× 18) of an upper lateral deciduous incisor of a child born at the 28th week of pregnancy. There is a notch in the labial enamel. At A the enamel matrix formed before birth has never been covered by the enamel which should have been formed over it after birth. At B the prenatal enamel is covered by a normal thickness deposited post-natally superficial to the neonatal line.

The appearance of a ground section of a deciduous tooth with *neonatal hypoplasia* is characteristic. The neonatal line indicates the level reached by enamel matrix formation at the time of birth. In neonatal hypoplasia there will at some parts be no enamel lying superficial to this line so that the prenatal enamel is exposed on the surface of the tooth (Fig. 5). Nearer to the neck of the tooth the formation of enamel has been resumed. This means that some of the ameloblasts active at the time of the disturbance—sometimes all of them—perished, but that the younger ameloblasts which had not yet begun to produce enamel matrix escaped.

A ground section of a *rachitic* tooth shows depressions on the enamel surface corresponding to the grooves and pits seen macroscopically (Fig. 6). The contour lines of the enamel have the appearance of having collapsed together under these depressions and it has been suggested that a collapse of poorly calcified enamel matrix has in

FIG. 6
Ground section of human rachitic tooth. Two deep grooves seen on the enamel surface correspond to periods of disturbed enamel formation. Interglobular dentine (best seen opposite the lower groove) was formed at the same periods. × 30.

fact occurred. However, a reduced rate of matrix formation by the ameloblasts in the affected areas would give the same appearance. The disturbances in the enamel can usually be seen to correspond chronologically with areas of interglobular dentine in the same specimen.

The appearance of tooth germs in human rickets has been described by Gottlieb (1920). There are degenerative changes in the enamel organ which may be partly separated from the enamel surface by an exudate containing cells. Ameloblasts may no longer be recognised

in some areas, and the enamel matrix varies in thickness from one part to another or may in some places have failed to form at all. Most information about the effects of vitamin D deficiency has been derived from experiments on animals. In human rickets vitamin D deficiency is

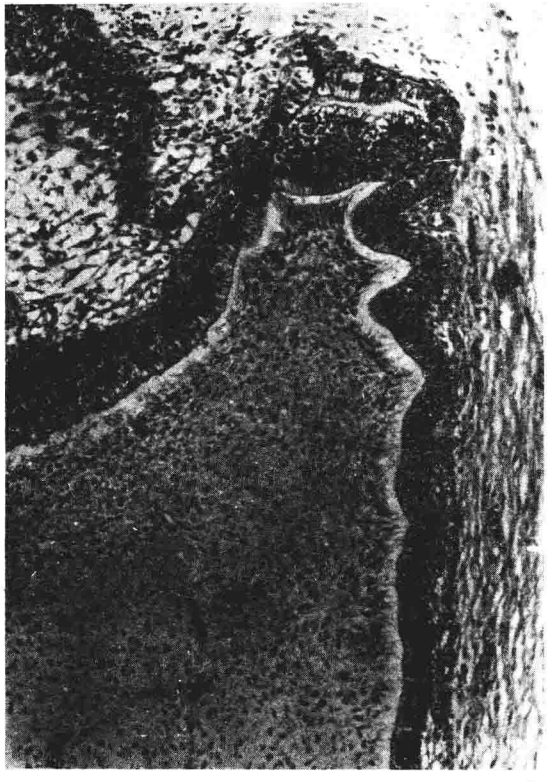

FIG. 7
Tooth germ of Hutchinsonian incisor showing abnormal outline of the dentine papilla with ridges and central depression (left) which will correspond to the incisive notch. Haematoxylin and eosin. (Bradlaw, 1953. *Oral Surg.* **6**, 147.)

often only one of several deficiencies concerned in the illness and the results may differ from those described in animal experiments.

Less severe disturbances than those necessary to produce macroscopic defects of the enamel will cause marked incremental lines visible in ground sections, or areas of defective mineralisation which can be recognised more certainly by other physical methods than simple optical examination, for example by radiographs of sections or by the polarizing microscope.

Prenatal Syphilis causes the characteristic deformity of the permanent upper central incisors (Hutchinsonian teeth) and of the first permanent molars. The abnormal shape reflects changes in the odontogenic epithelium (Fig. 7). According to Bradlaw (1953), perivascular infiltration and oedema of the follicle are followed by hyperplasia of the external enamel epithelium, stratum intermedium and ameloblasts

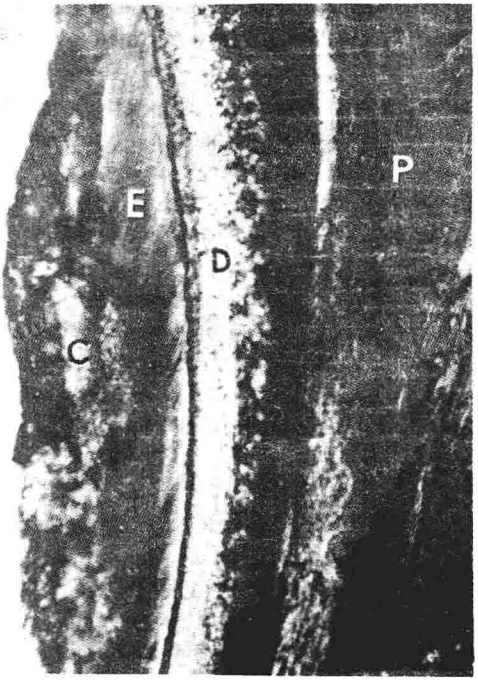

FIG. 8
Stained ground section of a permanent tooth damaged during formation by an infective process (osteomyelitis of the jaw). The irregular surface of the enamel E is covered with cementum C. The dentine D has remained thin and was only calcified at its outer surface, the predentine remaining very wide and here stained dark. The pulp P has pulled away from this slightly in preparation Picrothionin. × 70.

which bulge into the dental papilla, producing the characteristic notch and an irregular amelodentinal junction. Enamel is defectively formed and may in parts be entirely lacking and dentine is often hypoplastic. By appropriate methods *Treponema pallida* may be found throughout the tooth germs.

Fluorosis. Where the fluoride content of water habitually drunk in childhood much exceeds one part per million, the structure of the

enamel formed during that period is altered in such a way that visible effects are produced. These effects vary from opaque white patches to irregular brown areas, producing the characteristic 'mottled enamel'. Fluorosis is, however, not the only cause of opaque white patches. When the fluorosis is moderate, the enamel retains a fine surface gloss, but when severe, the surface may be dull and pitted and the enamel may have lost its usual strength. In ground sections the affected enamel appears defectively calcified both as regards the rods and interprismatic material. The rods may be sharply outlined by a brown substance lying between them.

Local Infection and Trauma. Hypoplastic defects of the enamel of single teeth may result from infection about the roots of a deciduous predecessor or mechanical injury to the developing tooth (see also page 69). In the case of infection around a tooth germ, the enamel may be missing over a limited area due to local destruction of the enamel organ or may be thin and irregular. Defects in its surface are often partly filled up by apposition of cementum before the tooth erupts (Fig. 8). In severe cases such as in osteomyelitis of the jaw, the apposition of dentine may also be permanently arrested so that a stunted tooth is formed. The quality of dentine may also be most abnormal, resembling that formed during rickets.

STRUCTURAL DEFECTS OF DENTINE

Genetically Determined: Hereditary Opalescent Dentine. A disorder due to an inherited dominant gene, and affecting all the teeth of both dentitions in most cases, is known as hereditary opalescent dentine or dentinogenesis imperfecta. A similar anomaly occurs in some patients with the bone disorder osteogenesis imperfecta. The teeth tend to be small with bulbous crowns and small roots (Figs. 9, 10, 11), are somewhat translucent on eruption and later become gradually grey or brown with bluish reflections from the enamel. The teeth wear away quickly. The disorder is principally in the formation of the dentine, but the enamel is often poorly calcified and tends to break away and become lost early in some cases.

The dentine near the amelo-dentinal junction is usually normal but that lying more deeply shows disordered structure with a diminished number of tubules, poor calcification, imperfect formation of the collagenous matrix and marked irregular incremental lines (Figs. 12, 13). The pulp cavity becomes obliterated early and there may be numerous pulp stones included in the dentine. The tubules are very irregular in size and in their course. It appears that odontoblasts are normally differentiated, but soon degenerate and are then replaced by

others which also disappear and are again replaced. Patches of more normal dentine often occur beneath the cusps and sometimes in the whole crown. Small blood vessels and cells may be included in the

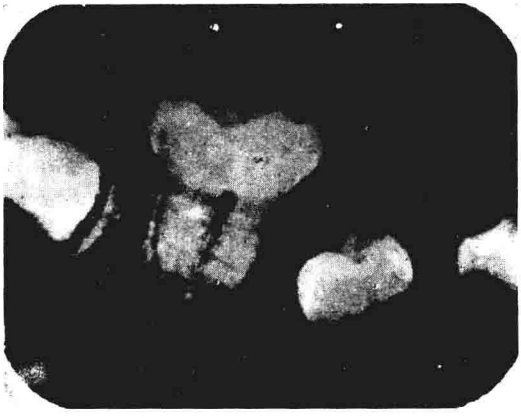

FIG. 9
Hereditary opalescent dentine. Radiograph shows a first permanent molar with bulbous crown and small roots. Although the tooth has just erupted at seven years, the pulp cavity is obliterated. (*Ann. roy. Coll. Surg. Engl.* 1955.)

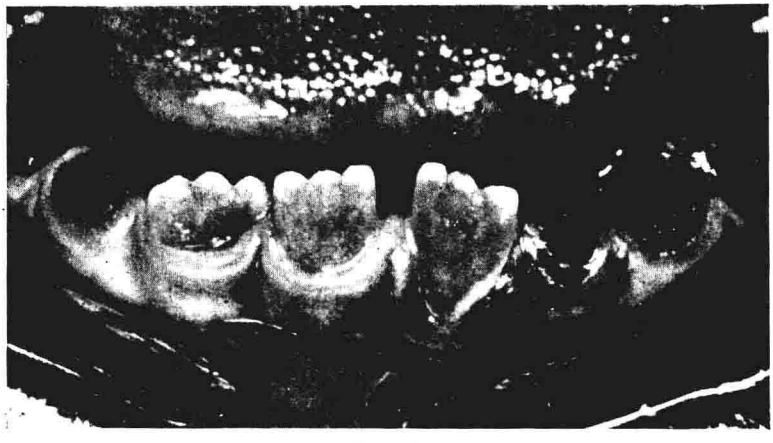

FIG. 10
The deciduous teeth are brown and worn to the level of the gum. The permanent incisors are beginning to change colour. (*Ann. roy. Coll. Surg. Engl.* 1955.)

dentine. In the deciduous teeth sometimes the pulp cavity is not obliterated, but the dentine on the contrary remains thin and the pulps may be exposed by attrition.

A similar condition of the permanent teeth, 'shell teeth', is due to the same gene. In another hereditary condition, 'dentinal dysplasia', the roots are very short and composed of a mass of embedded pulp stones.

FIG. 11

Ground section of upper central incisor from a patient with osteogenesis imperfecta. Note stumpy shape, obliteration of pulp cavity, embedded pulp stones, and radiating vascular inclusions in the dentine. × 7.

In hereditary vitamin D resistant rickets the dental defects are as in acquired rickets (see below).

Metabolic Disorders and Infections. RICKETS. Forming teeth examined during the active stage of rickets show increased width of the predentine layer and incomplete calcification of recent dentine (Fig. 14). The completed teeth of those who have suffered from rickets during the period of tooth formation show bands and areas of interglobular dentine corresponding to the periods of illness (Fig. 15) and often accompanied by enamel defects. While the bands of interglobular dentine lie along the incremental lines, the defect will be more severe at the ends of the lines near the surface of the tooth but not quite reaching the enamel or cementum. An impression that the defects lie in a line parallel with the outer surface of the dentine is thus often produced (Fig. 16). Other disturbances of calcium metabolism will cause analogous effects. Thus in idiopathic *hypoparathyroidism* the dentine will show lines of disturbed calcification corresponding with periodic bouts of tetany (Fig. 17). Interglobular dentine is not, however, a prominent feature, perhaps because the rate of growth of the dentine matrix is retarded at the same time that calcification is disturbed, as shown by the stunted root growth.

SCURVY. In the tooth germ will be found old and recent haemorrhages of the pulp, some of which may be followed by calcification,