MORPHOLOGY OF THE PRIMARY TEETH

An accurate chronology of primary tooth calcification is of clinical significance to the dentist. It is often necessary to explain to parents the time sequence of calcification in utero and during infancy.

The common observations of tetracycline pigmentation, developmental enamel defects, and generalized hereditary anomalies can be explained if the calcification schedule is known. A brief discussion of the morphology of the primary teeth is also appropriate before restorative procedures are considered for children.

LIFE CYCLE OF THE TOOTH

INITIATION (BUD STAGE)

Evidence of development of the human tooth can be observed as early as the sixth week of embryonic life. Cells in the basal layer of the oral epithelium proliferate at a more rapid rate than do the adjacent cells. These proliferating cells contain the entire growth potential of the teeth. The result is an epithelial thickening in the region of the future dental arch that extends along the entire free margin of the jaws.

This thickening is called the primordium of the ectodermal portion of the teeth and what results is called the dental lamina. At the same time, 10 round or ovoid swellings occur in each jaw in the position to be occupied by the primary teeth.

The permanent molars, like the primary teeth, arise from the dental lamina. The permanent incisors, canines, and premolars develop from the buds of their primary predecessors. The congenital absence of a tooth is the result of a lack of initiation or an arrest in the proliferation of cells. The presence of supernumerary teeth is the result of continued budding of the enamel organ.

PROLIFERATION (CAP STAGE)

Proliferation of the cells continues during the cap stage. As a result of unequal growth in the different parts of the bud, a cap is formed. A shallow invagination appears on the deep surface of the bud. The peripheral cells of the cap later form the outer and inner enamel epithelium.

As with a deficiency in initiation, a deficiency in proliferation results in failure of the tooth germ to develop and in fewer numbers of teeth than the normal.

Excessive proliferation of cells may result in epithelial rests. These rests may remain inactive or become activated due to an irritation or stimulus.

If the cells become partially differentiated or detached from the enamel organ in their partially differentiated state, they assume the secretory functions common to all epithelial cells, and a cyst develops. If the cells become more fully differentiated or detached from the enamel organ, they produce enamel and dentin, which results in an odontoma or a supernumerary tooth. The degree of differentiation of the cells determines whether a cyst, an odontoma, or a supernumerary tooth develops.

HISTODIFFERENTIATION AND MORPHODIFFERENTIATION (BELL STAGE)

The epithelium continues to invaginate and deepen until the enamel organ takes on the shape of a bell.

It is during this stage that the cells of the dental papilla differentiate into odontoblasts and those of the inner enamel epithelium differentiate into ameloblasts. Histodifferentiation marks the end of the proliferative stage as the cells lose their capacity to multiply. This stage is the forerunner of appositional activity.

Disturbances in the differentiation of the formative cells of the tooth germ result in abnormal structure of the dentin or enamel. One clinical example of the failure of ameloblasts to differentiate properly is amelogenesis imperfecta. The failure of the odontoblasts to differentiate properly, with the resultant abnormal dentin structure, results in dentinogenesis imperfecta.

In the morphodifferentiation stage, the formative cells are arranged to outline the form and size of the tooth. This process occurs before matrix deposition.

The morphologic pattern of the tooth becomes established when the inner enamel epithelium is arranged so that the boundary between it and the odontoblasts outlines the future dentino-enamel junction.

Disturbances and aberrations in morphodifferentiation lead to abnormal forms and sizes of teeth, resulting in conditions such as peg teeth, other types of microdontia, and macrodontia.

APPOSITION

Appositional growth is the result of a layer-like deposition of a nonvital extracellular secretion in the form of a tissue matrix. This matrix is deposited by the formative cells, ameloblasts, and odontoblasts, which line up along the future dentino-enamel and dentinocemental junction at the stage of morphodifferentiation. These cells deposit the enamel and dentin

matrix in a definite pattern and at a definite rate. The formative cells begin their work at specific sites that are referred to as growth centers.

Any systemic disturbance or local trauma that injures the ameloblasts during enamel formation can cause an interruption or an arrest in matrix apposition, which results in enamel hypoplasia. Hypoplasia of the dentin is less common than enamel hypoplasia and occurs only after severe systemic disturbances.

CALCIFICATION

Calcification (mineralization) takes place following matrix deposition and involves the precipitation of inorganic calcium salts within the deposited matrix. The process begins with the precipitation of a small nidus, and further precipitation occurs around it. The original nidus increases in size by the addition of concentric laminations.

There is an eventual approximation and fusion of these individual calcospherites into a homogeneously mineralized layer of tissue matrix.

If the calcification process is disturbed, there is a lack of fusion of the calcospherites. These deficiencies are not readily identified in the enamel, but in the dentin they are evident microscopically and are referred to as interglobular dentin.

EARLY DEVELOPMENT AND CALCIFICATION OF THE ANTERIOR PRIMARY TEETH

It was found that the first macroscopic indication of morphologic development occurs at approximately 11 weeks in utero. The maxillary and mandibular central incisor crowns appear identical at this early stage as tiny, hemispheric, mound-like structures. The lateral incisors begin to develop morphologic characteristics between 13 and 14 weeks. There is evidence of the developing canines between 14 and 16 weeks.

Calcification of the central incisor begins at approximately 14 weeks in utero, with the maxillary central incisor slightly preceding the mandibular central incisor. The initial calcification of the lateral incisor occurs at 16 weeks and that of the canine at 17 weeks.

EARLY DEVELOPMENT AND CALCIFICATION OF THE POSTERIOR PRIMARY TEETH AND THE FIRST PERMANENT MOLAR

The maxillary first primary molar appears macroscopically at 12½ weeks in utero. It had been observed that, as early as 15½ weeks, the apex of the mesiobuccal cusp may undergo calcification. At approximately 34 weeks the entire occlusal surface is covered by calcified tissue. At birth, calcification includes roughly three fourths of the occluso-gingival height of the crown.

The maxillary second primary molar also appears macroscopically at about 12½ weeks in utero. There is evidence of calcification of the mesiobuccal cusp as early as 19 weeks. At birth, calcification extends occlusogingivally to include approximately one fourth of the height of the crown.

The mandibular first primary molar initially becomes evident macroscopically at about 12 weeks in utero. Calcification may be observed as early as 15½ weeks at the apex of the mesiobuccal cusp. At birth, a completely calcified cap covers the occlusal surface.

The mandibular second primary molar also becomes evident macroscopically at 12½ weeks in utero. Calcification may begin at 18 weeks. At the time of birth, the five centers have coalesced, and only a small area of uncalcified tissue remains in the middle of the occlusal surface. There are sharp conical cusps, angular ridges, and a smooth occlusal surface, all of which indicate that calcification of these areas is incomplete at birth. Thus there is a calcification sequence of central incisor, first molar, lateral incisor, canine, and second molar.

It had been indicated that the adjacent second primary and the first permanent molars undergo identical patterns of morphodifferentiation but at different times, and the initial development of the first permanent molar occurs slightly later. The first permanent molars are uncalcified before 28 weeks of age; calcification may begin at any time thereafter. Some degree of calcification is always present at birth.