

Review of Current Literature

Teeth in Fetal Rickets

JOHN J. WOLFE, M.D., L.D.S. (Eng.)

Peiping, China

American Journal of Diseases of Children, Vol. 49, No. 4, April 1935, pp.905.

A case of hypoplasia in the deciduous dentition is reported which, in the author's opinion, is attributable to fetal rickets. The mother's blood calcium on admission to the hospital, at the time of delivery, was markedly below the normal level as was also the phosphorus. There were clinical evidences of a calcium deficiency. Roentgenological examination of the bones of the infant showed rachitic changes and the blood calcium and phosphorus was slightly below normal. Within the first twenty months the clinical and X-ray evidence of rickets had disappeared. The deciduous teeth showed areas of extreme hypoplasia or absence of enamel as follows: incisal tips of the canines, occlusal one-third of the first deciduous molars, gingival two-thirds of the incisors. Calcification was definitely disturbed from the last part of the fifth month to immediately after birth. (Anti-rachitic therapy was instituted at once after birth.)

A second case is reported with histologic and gross evidence of rickets in an infant delivered by embryotomy following a diagnosis of fetal death. Microscopic evidence of osteomalacia in the calcification of both enamel and dentine are described. Another and similar condition is reported in the deciduous incisors of an infant delivered stillborn by Caesarean section.

With this evidence, Dr. Wolfe feels that "Rickets occurring in the fetus manifests itself in the teeth by gross hypoplasia of the enamel." He calls attention to the assistance which the teeth may afford in determining the period of greatest deficiency of calcifying factors, particularly as the evidence recorded in bony changes gradually disappear on a therapeutic regimen.

H.J.]

Mottled Teeth: An Experimental and Histologic Analysis

ISAAC SCHOUR, D.D.S., Ph.D., *Chicago, Illinois and*

MARGARET CAMMACK, Ph.D., *Tucson, Arizona*

The Journal of the American Dental Association, May, 1935.

The material upon which this report is based consists of thirty-two rats, aged 90 to 270 days old, which were given from one to eight injections of 0.1 to 0.9 of 2.5 percent sodium fluoride at varying intervals.

The histologic findings on the rat incisor reveals that each injection produces a pair of light and dark incremental layers in both enamel and dentin. The width of each light layer is approximately 16 microns and represents the immediate response to each injection of sodium fluoride. The width of the layer gives the approximate growth rate of the rat incisor. The light layers are imperfect in formation and calcification. The dark layer represents the recovery response to the injection and follows the light layer. The dark layers are normal in formation and normal or excessive in calcification. The width of the dark layer varies with the number of days following each injection.

This study reveals that the rate of growth of the rat incisor is approximately 16 microns each day. The earliest response recognized was that found in a single injection of 0.3 cc of 2.5 percent of sodium fluoride and the rat killed one hour after injection. The change was noted in the enamel forming organ at the base of the tooth. From this observation it is thought that fluorine exerts a local action on the enamel and dentin forming organs rather than producing general metabolic changes.

E. 1

First Permanent Molar
Its Condition At Birth and Its Postnatal Development

RUDOLF KRONFELD, B.S., M.D., D.D.S.

Chicago, Illinois

The Journal of the American Dental Association, July, 1935.

This article is based upon an x-ray and histological study of twenty-six human jaws ranging in age from birth to fifteen years. The x-ray findings in some specimens of the new born revealed a complete absence of calcification of the first permanent molar. Wherever calcification had taken place it occurred at the mesial buccal cusp. Study in specimens of later date reveal that calcification occurs in a definite sequence with the mesial cusps before the distal cusps. The mesio-buccal cusp may begin to calcify two months earlier than the disto-lingual cusp. The histologic study bears out the x-ray findings.

At the time of calcification the centers do not change their relative positions and do not move apart with the onset of calcification. Kronfeld divides the development of the first permanent molar into five stages.

1. Prenatal phase (fifth fetal month to birth): Differentiation of the tooth germ; growth of enamel organ and dental papilla; modeling of the pulp and arrangement of the pulp horns corresponding to the later centers of calcification in the cusps.
2. Birth to 6 months: Formation of four or five centers of calcification, beginning about the time of birth with the mesio-buccal cusp and ending about two months later with the disto-lingual cusp; fusion of the individual cusps at about 6 to 8 months.
3. Six months to 1 year: Formation and calcification of occlusal surface; enamel immature.
4. One year to 2 years: Formation and calcification of entire crown; maturation of enamel beginning at the cusps and progressing cervically.
5. Two years to three and half years: Complete maturation of enamel; beginning formation of root.

The first permanent molar in its postnatal growth changes moves occlusally with the vertical growth of the jaw and Hertwig's sheath is the relatively fixed point.

E. M.