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Changes in Dental Tissue in Hypoparathyroidism.

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FLEISCHMANN, 1908 (12) was the first to observe changes in dental tissue in patients suffering from tetany. In 1911 ERDHEIM (12) investigated changes in dental tissue found in parathyroidectomized rats, and discovered the connection between the parathyroid glands and calcium metabolism. Also in 1911 TOYOFUKU (12) examined parathyroidectomized rats and found imperfectly calcified dentine and enamel after parathyroidectomy. In 1929 Erdheim's studies were repeated by ERDHEIM & ALBRIGHT (1), who showed that acalcification of dentine and enamel is followed by calcification, as long as parathyroid hormone is given to the parathyroidectomized rats. ALBRIGHT & STROCK, 1933 (2) compared these investigations with experiences from human pathology. They state that the patho-physiological mechanism is *a*calcification of the dentine which is formed when the parathyroidal function has become impared, and that no *de*calcification of the previously formed dentine takes place. Subsequent studies (SCHOUR et al., 1937 (10)) have demonstrated that parathyroidectomized rats, if given an adequate diet, show hypercalcification of the dentine formed during the first twenty days after the operation, and only dentine formed after that time shows hypocalcification and at the same time very irregular enamel formation. In patients with hypoparathyroidism the typical mineralization changes in teeth are found only if the hypoparathyroidism occurs before dental development is complete, and only in the teeth which are not fully developed at the start of the disease. Enamel defects are recognized macroscopically as enamel aplasia, pronounced enamel hypoplasia or hypoplasia in the form of irregular bands lying parallel round the teeth.

Clinical cases are dealt with by different authors (3, 4, 7, 11). MORTELL (9) has reported a case where 12 molars and premolars

were embedded and KEATING (6) 2 cases where the embedded teeth completely lacked enamel.

X-ray examination reveals resorption of radices (MILLER, 1946 (8)). The resorption is seen — in contrast to the mineralization changes — in the teeth developed both before and after the occurrence of the disease. It will only be mentioned in passing that the jaw can show dense bone structure similar to the other bones of the body.

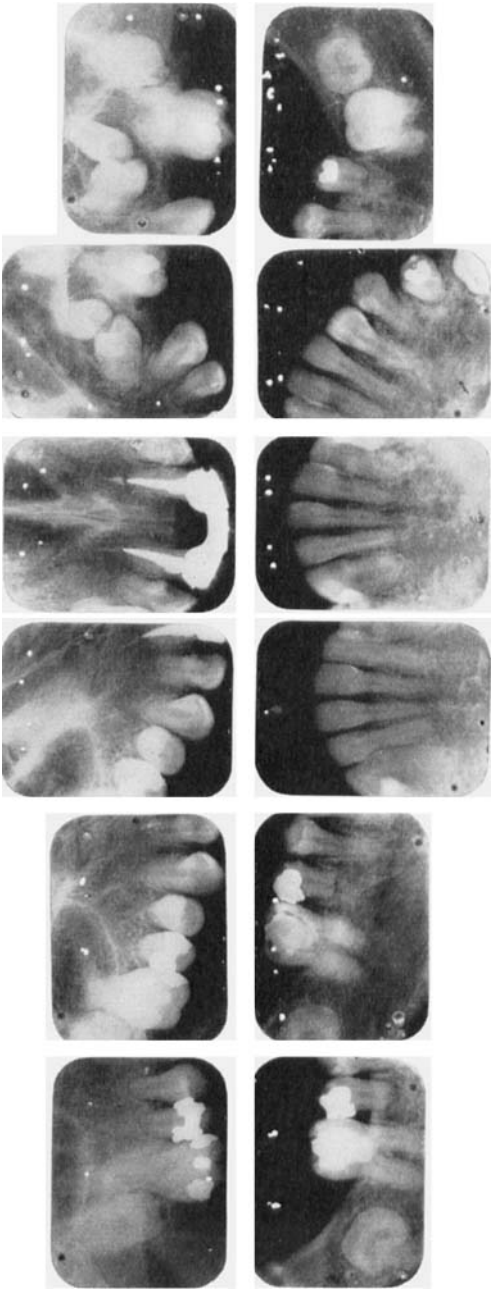
The case reported by the writers concerns dental tissue changes in a 19-year-old girl with idoapatic hypoparathyroidism. The tetanic symptoms commenced when she was 5—6 years of age, but actual causal therapy was only begun after the diagnosis was established when she was 19 years of age. For the more detailed case history, reference should be made to an article published elsewhere (5).

This patient had a pronounced maxillary prognathism, which, in conjunction with retention of several teeth, resulted in severe malocclusion. The erupted teeth found were 6, 5, 4, 3, 2, 1, | 1, 2, 3, 6 and 6, 5, 4, 3, 2, 1 | 1, 2, 3, 4, 5, and the completely embedded teeth 7 |, | 4, 5, | 7 and 7 | and | 6, 7, *i. e.* seven permanent embedded.

The enamel of the erupted teeth was normal on the first molars and incisors. Pronounced enamel hypoplasia was evident on the pre-molars, while the cuspids showed bands with slightly hypoplastic enamel lying in cervical region. X-ray examination (fig.) revealed that the enamel of the embedded premolars and first molars corresponded fairly well to that of the erupted teeth, while the embedded second molars completely lacked enamel. This seems to indicate that the calcification disturbance has started between 4 and 5 years of age, since the crowns of the first molars and incisors calcify before that time, while those of the premolars and second molars calcify between 4—5 and 8 years. The hypoplastic parts of the cuspids corresponded to the same period.

The X-ray pictures showed further the beginning of resorption of the apices of all erupted teeth. The tooth least affected was 6 |, which, however, had no vital pulp.

Finally, a calcified part could be seen in the pulp cavity in the anterior teeth of the lower jaw, lying just as far down in the radices as these were developed when the calcification disturbances started. This condition could be explained by the findings in the



report of SCHOUR et al. (10), which states that hypoparathyroidism first manifests itself as hypercalcification of the hard tissue of the developing permanent teeth. This hypercalcification does not change during the subsequent hypocalcification. In the case reported here, the hypercalcified part, in which even the pulp was involved, must be presumed to be the result of such a transient hypercalcification.

Summary.

The literature concerning changes in dental tissue caused by hypoparathyroidism is reviewed, and a case of idiopatic hypoparathyroidism, with all the characteristic changes present, is reported.

Special attention is drawn to the fact that the signs of transient hypercalcification demonstrated by SCHOUR et al. in parathyroidectomized rats, could be recognized in this patient.

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