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Occurrence of Enamel Hypoplasia in Children with Congenital Allergies

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The causes of enamel hypoplasia have been examined by several investigators. A definition of enamel hypoplasia, by Kronfeld and Schour,\(^1\) recognizes an arrested or deficient state of enamel formation manifested by irregularities and/or indentations on the enamel surface, the result of interference with ameloblast metabolism. There are three categories of events that can interfere with this metabolic process: localized mechanical trauma, systemic disease, and hereditary factors. Sarnat and Schour\(^2\) found a possible correlation between rickets and enamel defects but none between enamel hypoplasia and childhood diseases. Witkop\(^3\) has shown five types of enamel hypoplasia associated with hereditary factors. Forrester and Miller\(^4\) found that sites of hypoplasia in the enamel of the teeth of 13 patients with kernicterus corresponded to the age at which interference with the metabolism occurred. Stein\(^5\) found enamel hypoplasia in 8 of 16 prematurely born children. Kreshover\(^6–8\) has artificially produced enamel hypoplasia in rodents similar to that found in humans with specific disease. He also found it possible to correlate abnormalities of parturition or gestation with abnormalities of enamel.

Via and Churchill\(^9\) found a high correspondence of enamel abnormalities in children with cerebral disorders. In 1959 Via and Churchill\(^10\) determined significant correlations of abnormal enamel formation in children who had suffered a gestatory or birth insult.

By extension, there should be some correlation between congenital systemic involvement of the ectodermal tissues, such as congenital allergy and interference with ameloblast metabolism.

The statement of a null hypothesis for investigation was made that there would be no difference between the enamel formation of the teeth of children with congenital allergies and the enamel formation of the teeth of children not affected by congenital allergies.

EXPERIMENTAL METHODS

This investigation was projected as a pilot study. Earlier, Via and Churchill\(^10\) had found an incidence of 9 per cent enamel hypoplasia in the normal population, and this was accepted as the norm for the study. Patients were limited to those suffering from a definite allergy requiring desensitization procedures by their attending pediatrician. The group was limited to ages three to nine. This allowed inclusion of patients with a majority of the deciduous dentition present. No sex distinctions were made in the choice of patients. They were taken in the order referred by private pediatricians and the Stanford Convalescent Home, a diagnostic and treatment center for chronically ill children associated with the Stanford University School of Medicine, Department of
Pediatrics. Most of the children examined had an asthmatic or upper-respiratory distress symptomatology. Some had generalized skin lesions (eczema), and a few had reactions to specific allergens. No generalizations as to weight, height, or facies could be made.

The number of patients to be examined for significant levels of positive correlation was determined in the following manner: Assume two populations, (1) a normal one with an incidence of hypoplasia of 10 per cent and (2) an allergic population with an incidence of hypoplasia greater than 10 per cent. One and sixty-five hundredths times the standard error on either side of the means gives 90 per cent of the cases in either population. The curves overlap such that the 5 per cent ranges adjacent to each other belong to either population. If the normal value is 10 per cent and the abnormal is 20 per cent, then the difference (10) is equal to $1.65 \times \frac{1.65}{X}$ the standard error. The standard error may be approximated by $\sqrt{(PQ/n)}$, where $P =$ number of cases with lesions, $Q = \text{cases without lesions}$, and $n =$ total number of cases.

Using this relationship, we may conclude that if abnormal incidence $= 20$ per cent, then $n$ must equal 144; if abnormal incidence $= 30$ per cent, then $n$ must equal 44; if abnormal incidence $= 40$ per cent, then $n$ must equal 23. This allows 5 per cent error of Type I (seeing a difference where none exists) and 5 per cent error of Type II (not seeing a difference where one does exist).

Because of limitations of finances, an $n$ value of 45 was chosen for this study. A positive finding in 13 or more children out of 45 (29 per cent) could be analyzed as significant.

The method followed was one of clinical examination and evaluation, accompanied by roentgenographic examination and charting. The roentgenographic examination consisted of 8 films: a periapical film of each quadrant, a bite film of upper and lower anterior, and right and left bite-wing films. Charting was done on a special punch card devised by Rattner. Only the principle investigator examined the children, thus minimizing individual error.

**RESULTS**

Forty-five children were examined: 17 females and 28 males. Twenty-six exhibited one or more enamel defects and 19 had normal enamel formation. This gives a percentage positive value of 45 per cent relative to the 30 per cent incidence that was assumed for purposes of planning the study. The findings are shown in Table 1.

In a population of 45 children, if Via's figure of approximately 10 per cent is correct, we can expect 5 children to exhibit hypoplasia of the enamel and 40 children to have normal enamel (no lesion). These and the values given in Table 1 were applied to the formula for $\chi^2$, which is

$$\chi^2 = \sum \left( \frac{(f_1 - F_1)^2}{F_1} \right),$$

where $F_1 =$ theoretical frequency and $f_1 =$ observed frequency.

On analysis, the data in Table 1 give a value for $\chi^2$ of 99. Using appropriate tables, we may state that if no difference in enamel lesions in the two populations actually existed, we would find $\chi^2$ values as large as 99 much less frequently than once in a thousand such studies.

The seemingly high incidence of positive findings in male children was also analyzed
in the same manner, using the assumption of an equal distribution of lesions in males and females. The \( \chi^2 \) value of 5.0 derived indicates that twice in every 100 such studies we would find \( \chi^2 \) values greater than 5.0 when no actual difference in incidence between males and females existed. We therefore choose to disregard this finding until further data can be assembled.

The main location of the defects was the occlusal one-third of the deciduous cuspids and deciduous first molars. Using Logan and Kronfeld’s chart\(^{12} \) of dental development, the lesions were judged as occurring during the seventh to tenth month of development. Radiographic examination revealed no significant departures from normal development of tooth or supporting structure.

### TABLE 1

**Distribution by Age and Sex of Enamel Lesions**

<table>
<thead>
<tr>
<th>Age</th>
<th>Total</th>
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<tbody>
<tr>
<td>9</td>
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<th>M</th>
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<tbody>
<tr>
<td>20</td>
<td>6</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>11</td>
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</tbody>
</table>

Fig. 1.—A typical example of the group studied. The lesions appear on the incisal one-third of the four upper anterior incisors.
DISCUSSION

A correlation, much greater than could have occurred by chance, has been shown to exist between enamel defects of the deciduous dentition and the presence of severe allergic reactions. This suggests a relation between severe allergies and ameloblast metabolism. Localization of the site of the enamel defect in order to time the onset of the allergic reaction may be of use in diagnosis. Where complete prenatal histories are available, a differential diagnosis may be made, possibly definitive enough to include the inciting allergen. Although a preponderance of male children exhibited a positive lesion, statistical analysis left the significance of this open to some doubt. The positive correlation certainly suggests that this avenue is worthy of continued study.

SUMMARY

Forty-five children with congenital allergies were examined clinically and roentgenographically for enamel defects. A total of 28 males and 17 females ranging in age from three to nine gave 26 positive findings. The enamel lesions were localized in the occlusal one-third of the deciduous cuspids and first molars. This timed the onset to the last trimester of gestation. Chi-square gave a probability of occurring by chance of less than one in a thousand.

REFERENCES