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Lack of stability in enamel defects in primary teeth of children with cerebral palsy or mental retardation

Mohandas Bhat, MDS, DrPH Karin B. Nelson, MD
Philip A. Swango, DDS, MPH

Abstract

The Developmental Defects of Enamel (DDE) index was used to study the reliability across time of assessment of enamel defects in primary teeth in neurologically handicapped children. Fourteen of the 48 children originally examined were available for re-examination 6 months later. The original findings were confirmed in only 6 of these children (5 with no enamel defects and 1 with an enamel defect). Discrepancies noticed between the 2 examinations in the other 8 children could be explained on the basis of biological processes that occurred during the 6-month interim, namely, attrition, abrasion, crown fracture, caries, and possibly remineralization of a hypomineralized defect. These findings suggest that the optimum time for studying such defects is as soon as possible after the teeth erupt.

The developing tooth germ is sensitive to a range of systemic disturbances, some of which also may affect neurologic development (Levine et al. 1979). Because the enamel cannot recover once it is damaged, it provides a repository of information on the timing and perhaps the nature of insults potentially affecting other ectodermally derived structures, including the nervous system (Cohen and Diner 1970). However, several authors (Watson 1955; Via and Churchill 1957; Pimlott et al. 1985) have mentioned loss of tooth structure as a potential confounding factor in studies relating the appearance and location of enamel defects in primary teeth to gestational age and the presumed intrauterine insult. Therefore, as a first step in evaluating a relationship between the appearance and location of the enamel defect and the timing of onset and nature of the presumed insult, it would be desirable to have information on the stability of the findings over time.

This paper reports the results of a pilot study undertaken to determine the reliability across time of assessment of enamel defects in the primary teeth of neurologically handicapped children.

Materials and Methods

Subjects of the initial examination were 48 neurologically handicapped children enrolled in a specialized day care facility in Montgomery County, Maryland. Only 14 of these children were available 6 months later when a second series of examinations was conducted. Ages at the first examination ranged from 19 months to 5 years. The handicaps in these children included cerebral palsy, mental retardation, infantile spasms, and spina bifida.

The children were examined under an artificial light while seated in a portable dental chair or in the lap of an adult. A mouth mirror and an explorer were used to examine all the primary teeth present for both qualitative and quantitative enamel defects. The Developmental Defects of Enamel (DDE) index suggested by the Federation Dentaire Internationale (FDI; 1982) was used. No oral prophylaxis was administered before the examination. Tooth surfaces were dried with a cotton pellet and inspected visually. Defective areas were probed by passing the tip of an explorer over them to determine abnormalities of surface contour. When defects were diagnosed, their type was noted, and their exact size, and precise location were drawn to scale on diagrams of the teeth depicting the buccal, lingual, and in the case of molars, the occlusal surfaces. A specially designed chart similar to the one suggested by the FDI (1982) was used for this purpose.

A second and identical examination was conducted by the same examiner on the remaining 14 children after 6 months. The examiner was unaware of the results of the previous session.

Results

In the 5 children in whom no enamel defects were noted initially, absence of enamel defects was confirmed 6 months later (Table). The remaining 9 children were considered at the initial examination to have enamel defects on the labial surfaces of the incisors; in

TABLE. **Comparison of First and Six-Month Repeat Examination of Enamel Defects in Primary Teeth**

ID	*Age**	*Tooth*	*Initial Exam*	*Repeat Exam*	*Comments*
23	36 mo.	Max. Lateral	Missing enamel	Normal enamel	Tooth chipped at defect site
		Max. Central	Missing enamel	Missing enamel	
		Max. Lateral	Missing enamel	Missing enamel	
22	42	Max. Central	Shallow pit	Normal enamel	Attrition at defect site
		Max. Central	Missing enamel	Normal enamel	Attrition at defect site
		Max. Lateral	Shallow horiz. groove	Shallow horiz. groove	Heavy abrasion at defect site made assessment difficult
31	42	Max. Lateral	Shallow pit	White opacity	Abrasion at defect site
		Max. Lateral	Shallow pit	Shallow pit	
17	46	Max. Central	Shallow horiz. groove	Large white opacity	Abrasion at defect site
3	24	Max. Central	Missing enamel	Caries	Caries at defect site
		Max. Central	Missing enamel	Caries	Caries at defect site
18	24	Max. Lateral	Missing enamel	Missing enamel	
		Max. Central	Missing enamel	Missing enamel	
		Max. Central	Missing enamel	Missing enamel	
		Max. Lateral	Missing enamel	Missing enamel	
9	47	Max. Central	White opacity	Normal enamel	Attrition at defect site
5	19	Max. Lateral	White opacity	Normal enamel	Remineralized?
		Max. Central	White opacity	White opacity	
		Max. Central	White opacity	White opacity	
		Max. Lateral	White opacity	Normal enamel	Remineralized?
10	32 mo.	Max. Central	Yellowish-brown opacity	Normal enamel	Attrition at defect site

* Age at initial examination in months.
Note: All cases had cerebral palsy and mental retardation, except ID 10 who had spina bifida.

only 1 of these was the enamel defect fully confirmed at the second examination. In 8 of the children, several discrepancies were noted between the 2 examinations. Although initially these discrepancies appeared puzzling, on further consideration it became apparent that all could be explained by biological processes that were active during the 6-month interim (Table).

1. In 1 case (ID 23), missing enamel located at the distoincisal edge of a tooth was not found on repeat examination. Loss of tooth substance at the site of the defect due to fracture (chipped enamel) apparently caused the defect to disappear.
2. Three cases of enamel defects, pitted or missing enamel (ID 22), a white opacity (ID 9), and a yellowish brown opacity (ID 10), located on the incisal third of incisors, were not confirmed at the second examination. Heavy incisal attrition apparently had caused the sites of the original defects to be lost.
3. A defect diagnosed initially as missing enamel had become carious in 1 case (ID 3), making it difficult to reconfirm the diagnosis at the second examination.
4. In several subjects, shallow grooves (IDs 17 and 22), and a shallow pit (ID 31) diagnosed at the first examination could either be barely detected at the repeat examination or had changed in appearance and appeared to be a white opacity. Heavy abrasion in these subjects had worn the enamel surface at the site of the defect to the level of the bottom of the groove or pit.
5. In 1 case (ID 5), white opacities detected at the first examination were diagnosed as normal at the repeat examination. It is possible that the change in appearance resulted from remineralization.

Discussion

This pilot study suggests that several processes influence the likelihood of stable findings of enamel defects in groups of seriously disabled young children who, because of their youth and handicap, have special problems likely to contribute to early loss of tooth structure. These children generally have poor oral hygiene and it is often impossible for them to brush their teeth. Brushing by an adult helper is difficult (Wessels 1960) and the vigorous brushing often resorted to by an adult could easily lead to severe abrasion of the tooth enamel, especially of incisors. It is not uncommon to find many cases of neglected oral hygiene and an increased incidence of dental caries in children with cerebral palsy (Wessels 1960). Bruxism or grinding of teeth is a common finding in children with cerebral palsy (Watson 1955; Kastein 1957; Wessels 1960) and can produce severe attrition. Also, many children with cerebral palsy experience falls due to convulsive disorders, so traumatic injuries to the incisor teeth are a relatively

frequent occurrence (Wessels 1960).

In addition to the destructive forces active in the dentition of these children, there is the possibility of reparative forces, such as remineralization of white opacities (Aasenden and Peebles 1978) which derive from subsurface hypomineralization (Norén 1983).

The findings of this pilot study clearly indicate that the optimum time for conducting such a study is soon after the eruption of the teeth. Since the majority of enamel defects noted in children with cerebral palsy or mental retardation occur in incisors (McMillan 1961; Gullikson 1969), the optimum age of the subjects would be around 10-18 months and not later than 30 months when eruption of the primary dentition is completed. Older children are less suitable as subjects as some of their enamel defects may change in appearance or disappear altogether.

Dr. Bhat is a visiting scientist and Dr. Swango is a dental epidemiologist, NIDR Epidemiology and Oral Disease Prevention Program, and Dr. Nelson is a neurology medical officer, Neuroepidemiology Branch, National Institute of Neurological and Communicative Disorders, Bethesda, Maryland. Reprint requests should be sent to: Dr. Mohandas Bhat, Epidemiology and Oral Disease Prevention Program, National Institute of Dental Research, 5333 Westbard Ave., Room 549, Bethesda, MD 20892.

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Tooth decay may be inherited

Mothers with poor dental hygiene can pass caries-causing bacteria on to their infants, according to researchers.

The practice of blowing on babies' food or thermal taste testing may be a common vehicle for bacteria transmission, according to researchers at the University of Alabama Institute of Dental Research. Mothers, who typically have the most contact with babies are identified as transmitters of *Streptococcus mutans*, the bacteria known to cause caries.

The Alabama researchers extracted DNA from the mouths of both mothers and children participating in the study. The DNA then was broken down using enzymes, and by comparing the fragments the researchers identified the source of the bacteria. The Alabama studies, funded by a $1.2 million grant from the National Institute of Dental Research (NIDR), traced the bacteria back to the mother.

These findings support previous research done in Sweden indicating that when parents practice good oral hygiene their children often develop fewer caries.