Intubation and mineralization disturbances in the enamel of primary teeth

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This study was undertaken to examine the effects of intubation on the enamel development of primary teeth in children intubated during the first 3 months of life. The teeth of 35 children were examined clinically for signs of defects. Dental enamel defects were seen in 26 (74%) patients; enamel hypoplasia was seen in 15 and enamel hypomineralization in 19 cases. In eight patients both enamel hypoplasia and hypomineralization were found. There was a preponderance of enamel defects in the right maxilla, which supports the hypothesis that an early trauma to mineralizing primary teeth caused by laryngoscope may lead to dental enamel hypoplasia. □ *Dental enamel; dental enamel hypoplasia; laryngoscopy; odontogenesis; tooth, deciduous*

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The primary teeth start their mineralization during the 4th gestational month, and the crowns of the incisors are completed at around 6 months of age. The mineralization of the primary molars starts at the end of the 5th gestational month and is completed at the end of the 1st year of life (1). Since the primary teeth form during both intra- and extra-uterine life to 12 months postnatally, they can serve as chymographs of events during their mineralization, as morphologic changes of the enamel will remain.

Enamel hypoplasia in primary dentition is subject to several different systemic etiologic background factors as well as local factors (2–9). Experimental studies on animals have shown that trauma induced to rat maxillary incisors can cause aberrations similar to enamel hypoplasias (10). Clinical studies of enamel defects in primary teeth have shown that laryngoscopy or orotracheal intubation may cause palatal deformation and developmental defects of primary teeth (7, 11–26). There are several reasons for intubation at an early age, such as premature birth, management of respiratory distress, and surgery and postoperative care.

This study was undertaken to elucidate the effects of intubation on the enamel development of primary teeth in children intubated during the first 3 months of life.

Patients and methods

Children were selected from those residing in Göteborg and operated on during their first 3 months of life at Children's Hospital during the year 1 December 1980 to 30 November 1981. Of 55 infants meeting these criteria 35 children appeared for a clinical examination of their primary teeth. Of the 20 non-attenders, 1 child was dead and the others did not respond to the call to come in for the dental examination or did not want to take part in the investigation.

The dental examination, which involved a clinical check-up of the primary teeth for mineralization disturbances, was performed by two of the authors (J. G. Norén and L. Ranggård) in accordance with the agreed-on criteria. The following criteria were used:

Enamel hypoplasia: A macroscopic defect of the enamel with reduced enamel thick-

ness. The borders of the defect should be rounded and smooth.

Enamel hypomineralization: A defect of the enamel seen as a change in the translucency of the enamel, the surface being smooth.

For both types of defects the location on the tooth and tooth surface, number of defects, surface structure, and the color of the defects were noted. Further, the parents were asked about fluorine exposure and trauma during the first 3 years of life.

Results

The non-participants did not differ from the examined children with regard to their medical diagnoses or other medical findings.

The children were divided into three groups in accordance with the time of intubation: <1 month, 1 < 2 months, and 2 < 3 months, respectively, after birth.

Medical findings

Children intubated 0–1 month after birth (11 children). The mean birth weight was 2950 g (range 650–4660 g). The medical diagnoses were inguinal hernia (3), myelomeningocele, omphalocele, and pyloric stenosis (8).

Children intubated 1–2 months after birth (13 children). The mean birth weight was 3256 g (range, 1480–4360 g). The medical diagnoses were pyloric stenosis (5), inguinal hernia (5), cardiac defects (2), and Epstein anomaly (1).

Children intubated 2–3 months after birth (11 children). The mean birth weight was 2844 g (range, 1890–3610 g). The medical diagnoses were inguinal hernia (10) and hydrocele (1).

Dental findings

In none of the cases was there any indication of other early trauma or excessive fluorine exposure.

Dental enamel disturbances of the primary teeth were found in 26 (74%) of the 35 patients. In the other nine (26%) cases, however, no visible enamel disturbances were detected. Hypoplasia was found in 15 and enamel hypomineralization in 19 cases. In 8 of the 35 patients both enamel hypoplasia and hypomineralization were found.

The distribution of teeth with mineralization disturbances is given in Fig. 1. In the 35 patients 25 teeth had enamel hypoplasia; 19 were maxillary teeth and 6 mandibular teeth. The highest frequency was found for teeth 51, 52, and 61 (11 teeth; 44%). Teeth with hypomineralized enamel were found in

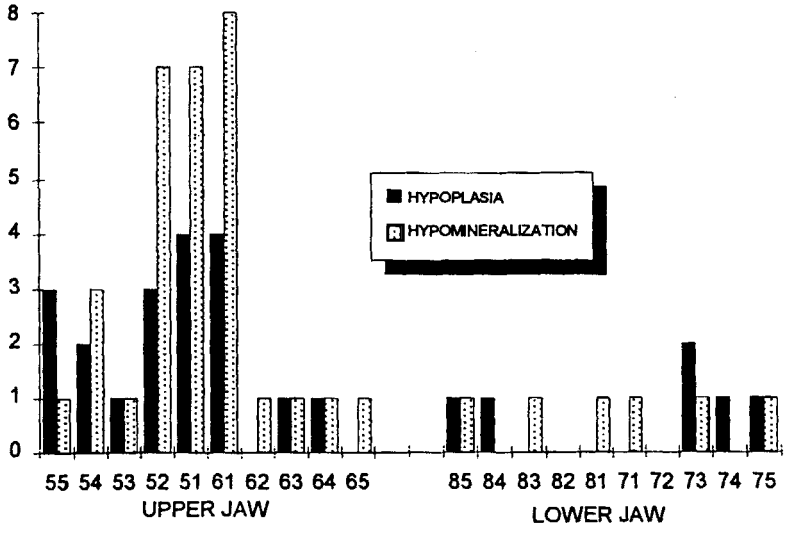

Fig. 1. Distribution of enamel defects, hypoplasias and hypomineralizations, in the primary dentition of children intubated at 0–3 months of age.

Table 1. Distribution of enamel defects in the primary teeth of children intubated during the 1st month of life

Patient no.	Enamel hypoplasia	Enamel hypomineralization
1	61	
2		54, 64
3		73, 83
4		53
5		
6		
7	52	
8		
9		
10		51
11	55, 61, 73	61

a similar distribution; of a total number of 37 primary teeth, 31 were in the maxilla and 6 in the mandible, with the highest frequency for teeth 52, 51 and 61 (22 teeth; 60%).

The enamel defects were located on the buccal side of the teeth in all cases but two, and in these the defects were both buccal and lingual.

Children intubated between 0 and 1 month of age (Table 1). Of the 11 patients 3 (28%) had enamel hypoplasia. Two of these were located in the upper jaw on 51 and 61, whereas in the third patient the enamel hypoplasia was found in both jaws on teeth 55, 61, and 73. Enamel hypomineralization (36%) was found in four patients; in three cases it was located in the upper jaw on teeth 54, 53, 51, and 64. One child had symmetric hypomineralizations on 73 and 83 in the lower jaw. Another had enamel hypoplasia and hypomineralization in both the upper and lower jaws on teeth 55, 61, and 73. Four children (36%) had no clinically visible mineralization defects of their teeth.

Children intubated between 1 and 2 months of age (Table 2). Enamel hypoplasia was found in 6 (46%) of the 13 children. In three children the location was in the upper jaw on teeth 55, 51, and 63, and in one child only it was in the lower jaw on 73. In the last two children the enamel hypoplasia was located in both jaws on teeth 55, 84 and 54, 52, 51, 61, 74, 75, 85. Enamel hypomineralization was found in five patients (38%), mainly located on 52, 51, and 61. In one of these children the enamel disturbances appeared symmetric; two children had both enamel hypoplasia and hypomineralization, and in the remaining two (14%) no enamel defects were observed.

Children intubated between 2 and 3 months of age (Table 3). Of the 11 children 6 (55%) had enamel hypoplasia, all of which was located in the upper jaw on teeth 54, 53, 52, 51, 61, and 64. Two (18%) had enamel hypomineralization only, which was located in the upper jaw on teeth 52, 51, 61, and 63; one of these was symmetric. The last patient had symmetric hypomineralization in the lower jaw, located on 71, 81. Four of the children had both enamel hypoplasia and

Table 2. Distribution of enamel defects in the primary teeth of children intubated between the 1st and 2nd months of life

Patient no.	Enamel hypoplasia	Enamel hypomineralization
12	54, 52, 51, 61, 74, 75, 85	
13	63	
14	73	
15		
16		52
17		52
18	51	52
19	55, 84	54, 75
20		51, 61
21		52, 51
22		55, 61, 65, 85
23		
24	55	54, 52, 51, 61

Table 3. Distribution of enamel defects in the primary teeth of children intubated between the 2nd and 3rd months of life

Patient no.	Enamel hypoplasia	Enamel hypomineralization
25	51	51, 61
26	54, 64	
27		52, 63
28	53	52, 61, 62
29		51, 61
30	61	51, 61
31		
32	51	71, 81
33		
34	52	
35		

hypomineralization. In three patients (27%) no signs of enamel defects could be discerned.

There was a slight tendency for the enamel disturbances to be located towards the maxillary right side.

Discussion

A high prevalence of enamel defects was observed in infants intubated between 0 and 1 month of age with a corresponding increase of defects in infants intubated between 1 and 2 and 2 and 3 months of age. Further, the highest occurrence was found in the right anterior maxillary region. The frequency of dental enamel disturbances (74%) was considerably higher than the normal prevalence figures for Scandinavian populations (27). Since the non-participants did not differ from those taking part in the study, it is reasonable to suggest that their level of defects would have been similar.

The high prevalence of enamel hypoplasia seen in this study is in agreement with earlier investigations of infants intubated at an early age (7, 12–14, 16–18, 21, 25, 30). Also noted in previous studies is the distribution of enamel defects that can be ascribed to the trauma caused by the pressure of the laryngoscope on the alveolar ridge during intubation. This is supported by earlier findings of a positive correlation between enamel hypoplasia in permanent dentition and trauma to the primary teeth (27, 31–34). Trauma, as an important etiologic factor predisposing to enamel hypoplasia, has been presented in animal studies (10, 28, 29, 35).

The enamel hypoplasia was mainly located on the right side of the maxilla, thus suggesting an exogenous cause. In the literature there is a lack of consistency concerning the location of enamel defects after intubation. A higher incidence on the left side of the maxilla is reported in some studies (16, 36), whereas others have found a preponderance on the right (13). In a literature review in 1989 Angelos et al. (26) pointed out that differences in individual laryngoscopy techniques may account for these inconsistent data. The enamel hypoplasia found in the molar regions can be regarded as less relevant since molars mineralize at a later age than the age at which patients were intubated in this study (1) and can hardly be caused by normal handling of a laryngoscope.

Identifying the time of origin of dental hypomineralization is difficult (37); therefore conclusive evidence as to the influence of laryngoscopy cannot be given.

Notwithstanding the great number of clinical and histologic studies, the pathogenetic mechanisms of developmental defects is still poorly understood. The development of enamel hypoplasia and enamel hypomineralization may be regarded as multifactorial from an etiologic point of view. Clinical findings and the relationship to medical factors only present indirect but, nevertheless, sometimes valid evidence.

References

1. Lunt RC, Law DB. A review of the chronology of calcification of the deciduous teeth. J Am Dent Assoc 1974;89:599–606.
2. Seow WK. Enamel hypoplasia in the primary dentition: a review. J Dent Child 1991;58:441–52.
3. Funakoshi Y, Kushida Y, Hieda T. Dental observations of low birth-weight infants. Pediatr Dent 1981;3:21–5.
4. Nikiforouk G, Fraser D. The etiology of enamel hypoplasia: a unifying concept. J Pediatr 1981;98: 888–93.
5. Norén JG. Human deciduous enamel in perinatal disorders. Morphological and chemical aspects [thesis]. Göteborg: University of Göteborg, 1983.

6. Norén JG. The effects of perinatal disorders on the developing dentition. In: Nowak AJ, Erenberg A, editors. Factors influencing orofacial development in the ill, preterm low-birth-weight, and term neonate. Iowa City [IA]: University of Iowa, Proceedings of a conference, 1984.
7. Johnsen D, Krejci C, Hack M, Fanaroff A. Distribution of enamel defects and the association with respiratory distress in very low birth-weight infants. J Dent Res 1984;3:59–64.
8. Bhat M, Nelson KB. Developmental enamel defects in primary teeth in children with cerebral palsy, mental retardation, or hearing defects: a review. Adv Dent Res 1989;3:132–42.
9. Norén JG. Enamel structure in deciduous teeth from low-birth-weight infants. Acta Odontol Scand 1983;41:355–62.
10. Engström C, Norén JG. Effects of orthodontics force on enamel formation in normal and hypocalcemic rats. J Oral Pathol 1985;15:78–81.
11. Boice JB, Krous HF, Foley JN. Gingival and dental complications of orotracheal intubation. JAMA 1976;236:957–8.
12. Krous HF. Defective dentition following mechanical ventilation [letter]. J Pediatr 1980;97:334.
13. Moylan FMB, Selden EB, Shannon DC, Todres JD. Defective primary dentition in survivors of neonatal mechanical ventilation. J Pediatr 1980;96:106–8.
14. Wetzel RC. The present role of the general practitioner in orthodontics. Dent Clin North Am 1968 July;483–508.
15. Erenberg A, Nowak AJ. Palatal groove formation in neonates in infants with orotracheal tubes. Am J Dis Child 1984;138:974–5.
16. Seow WK, Brown JP, Tudehope DI, O'Callaghan M. Developmental defects in the primary dentition of low birth-weight infants: adverse effects of laryngoscopy and prolonged endotracheal intubation Pediatr Dent 1984;6:28–31.
17. Seow WK, Brown JP, Tudehope DI, O'Callaghan M. Dental defects in the deciduous dentition of premature infants with low birth weight and neonatal rickets. Pediatr Dent 1984;6:88–92.
18. Seow WK, Brown JP, Tudehope DI, O'Callaghan M. Effect of neonatal laryngoscopy and endotracheal intubation on palatal symmetry in 2- to 5-year-old children. Pediatr Dent 1985;7:30–5.
19. Erenberg A, Nowak A. Palatal grooves in orally intubated newborns [letter]. Am J Dis Child 1986;140:973–4.
20. Ginther WH. Patal grooves in orally intubated newborns [letter]. Am J Dis Child 1986;140:1097.
21. Seow WK. Oral complications of premature birth. Aust Dent J 1986;31:23–9.
22. Molteni RA, Bumstead DH. Palatal grooves in orally intubated newborns [letter]. Am J Dis Child 1986;140:974.
23. Molteni RA, Bumstead DH. Development and severity of palatal grooves in orally intubated newborns. Am J Dis Child 1986;140:357–9.
24. Molteni RA, Bumstead DH. Palatal grooves in orally intubated newborns. Am J Dis Child 1986; 140:1097.
25. Kopra DE, Creighton PR, Buckwald S, Kopra LB, Carter JM. The oral effects of neonatal intubation. J Dent Res 1988;67:165.
26. Angelos GM, Smith DR, Jorgenson R, Sweeney EA. Oral complications associated with neonatal oral tracheal intubation: a critical review. Pediatr Dent 1989;11:133–40.
27. Norén J, Koch G, Rasmussen P. Disturbances in tooth development and eruption. In: Koch G, Modéer T, Poulsen S, Rasmussen P, editors. Pedodontics—a clinical approach. Copenhagen: Munksgaard, 1991:260–1.
28. Lenstrup K. On injury by fractures of the jaws to teeth in course of formation. Acta Odontol Scand 1955;13:181–202.
29. Suckling GW. Defects of enamel in sheep resulting from trauma during tooth development. J Dent Res 1980;59:1541–8.
30. Seow WK, Humphrys C, Tudehope DI. Increased prevalence of developmental dental defects in low birth-weight, prematurely born children: a controlled study. Pediatr Dent 1987;9:221–5.
31. Ideberg M, Persson B. Development of permanent tooth germs involved in mandibular fractures in children. J Dent Res 1971;50:721–3.
32. Andreasen JO. The influence of traumatic intrusion of primary teeth on their permanent successors. A radiographic and histologic study in monkeys. Int J Oral Surg 1976;5:207–19.
33. Andreasen JO, Ravn JJ. Enamel changes in permanent teeth after trauma to their primary predecessors. Scand J Dent Res 1972;81:203–9.
34. Ravn JJ. Developmental disturbances in permanent teeth after exarticulation of their primary predecessor. Scand J Dent Res 1975;83:131–4.
35. Suckling GW. Developmental defects of enamel—historical and present-day perspectives of their pathogenesis. Adv Dent Res 1989;3:87–94.
36. Mellander M, Norén JG, Fredén H, Kjellmer I. Mineralization defects in deciduous teeth of low birth-weight infants. Acta Paediatr Scand 1982;71: 727–33.
37. Suckling GW, Nelson DGA, Patel MJ. Macroscopic and scanning electron microscopic appearance and hardness values of developmental defects in human permanent tooth enamel. Adv Dent Res 1989;3: 219–33.

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