PEDIATRIC DENTISTRY/Copyright © 1984 by
The American Academy of Pedodontics/Vol. 6 No. 1

Developmental defects in the primary dentition of low birth-weight infants: adverse effects of laryngoscopy and prolonged endotracheal intubation

W. Kim Seow, BDS, FRACDS John P. Brown, BDSc, MSc, PhD, FRACDS
David I. Tudehope, MBBS, FRACP Michael O'Callaghan, MBBS, FRACP

Abstract

Trauma caused by laryngoscopy and orotracheal intubation affects mainly the maxillary anterior teeth. Examination of the primary dentition of 63 low birth-weight, prematurely born children showed that developmental defects of these teeth occurred in 85.0% of 40 intubated children compared to only 21.7% of nonintubated children, a fourfold difference. Trauma caused by laryngoscopy affects mainly the left maxillary anterior teeth; in the intubated group of children with defects of maxillary anterior teeth, 66.1% of the affected teeth were on the left compared with 33.9% on the right, a twofold difference. Traumatic injury caused by laryngoscopy and endotracheal intubation at the critical period of amelogenesis may contribute to defects in the dentition of low birth-weight infants whose dental development already is compromised by derangements of calcium metabolism and other systemic factors.

Previous studies of the primary dentition in premature infants have demonstrated a high prevalence of enamel hypoplasia.[1-5] Although general systemic factors such as hypocalcemia have been implicated in the etiology, local traumatic factors also may have a significant role. Two previous reports[6,7] have suggested that endotracheal intubation and mechanical ventilation may have traumatic effects on the developing unerupted primary dentition of newborn infants. It also has been well documented that the use of the laryngoscope during endotracheal intubation may injure erupted teeth.[8-12] This study examined the prevalence and distribution of developmental dental defects in a group of low birth-weight infants in order to determine the possible adverse effects of laryngoscopy and prolonged endotracheal intubation.

Patients and Methods

The patients in this study were children aged two years and older who were attending the Growth and Development Clinic of the Mater Children's Hospital, South Brisbane. This clinic was established in 1978 to provide a multidisciplinary longitudinal followup of all surviving infants of low birth weights managed at the Mater Mothers' Hospital.[13-16]

A total of 63 children with low birth weights were available for study. They were all prematurely born, with birth weights of between 605 g and 1,500 g, and a mean birth weight of 1,154 g.

There were 37 males and 26 females. Forty received endotracheal intubation and mechanical ventilation in the neonatal period, while 23 did not. At the time of dental examination, the ages of the children ranged from two years, two months to five years, five months, with a mean of three years, eight months. All these children were single births except for a set of twins and three sets of triplets (two sets of which had two surviving members each). Only two children from the third set of triplets were included in the study because the third child had a cleft lip and palate — teeth in the region of orofacial clefts often are defective.

The dental examinations were performed under ideal conditions at the University of Queensland Dental School. The teeth were dried and a mirror and probe used to detect caries, opacities, and enamel hypoplasia. The diagnosis of opacity was restricted to teeth with white or yellow brown areas that did not have hypoplastic enamel, i.e., pitting, ridging, or other disturbances of surface contour. If a tooth showed both opacity and hypoplasia, a diagnosis of hypoplasia was made. All tooth surfaces were examined and the severity and extent of each dental defect recorded in a comprehensive chart. Intraoral photographs were taken in some children. Postnatal medical and dental histories were obtained from the parents. Maternal and neonatal medical histories were

Table 1. Distribution of Defective Teeth in 40 Intubated and 23 Nonintubated Low Birth-Weight Children

Intubated Group

	Number of Defective Maxillary Teeth											*Number of Defective Mandibular Teeth*										
	Right					Left					Total	Right					Left					Total
Tooth	E	D	C	B	A	A	B	C	D	E		E	D	C	B	A	A	B	C	D	E	
Hypoplasia	0	0	3	3	4	15	10	3	2	1	41	1	0	4	0	1	1	0	6	1	0	14
Opacity	5	3	3	3	3	2		4	3	3	1	30	4	8	3	2	1	1	2	3	9	7	40
Total	5	3	6	6	7	17	14	6	5	2	71	5	8	7	2	2	2	2	9	10	7	54

Nonintubated Group

	Number Of Defective Maxillary Teeth											*Number of Defective Mandibular Teeth*										
	Right					Left					Total	Right					Left					Total
Tooth	E	D	C	B	A	A	B	C	D	E		E	D	C	B	A	A	B	C	D	E	
Hypoplasia	0	2	0	1	3	4	2	0	1	0	13	0	2	0	1	0	0	1	0	1	0	5
Opacity	2	2	1	2	3	1	1	1	1	1	15	2	1	1	1	0	0	1	1	0	3	10
Total	2	4	1	3	6	5	3	1	2	1	28	2	3	1	2	0	0	2	1	1	3	15

obtained from hospital records.

Data were analyzed using 2x2 contingency tables and χ^2 tests with Yates correction or the Student's t-test were used to detect statistical differences between groups.

Results

Effect of Endotracheal Intubation

Table 1 shows the distribution of defective teeth in the 40 intubated and 23 nonintubated children. To investigate the effects of endotracheal intubation, it is necessary to consider only the dental defects present in the maxillary incisors and canines as neither the laryngoscope nor orotracheal tube would extend beyond the region of the maxillary canines.

Thirty-four (85%) of the 40 intubated infants had defects of the maxillary anterior teeth (Table 2). These 34 affected children had a total of 56 defective maxillary anterior teeth. Only 5 (21.7%) of the 23 nonintubated children had defective maxillary anterior teeth (Table 2). These 5 children had a total of 19 defective maxillary anterior teeth. The difference in the children affected in the two groups (85% versus 21.7%) was statistically significant ($\chi^2 = 24.46$, $p<0.001$). In contrast, no statistical difference was found in the mandibular anterior teeth between the intubated and nonintubated groups ($\chi^2 = 0.18$, $p<0.1$), suggesting that the maxillary anterior teeth were being affected selectively during the intubation process.

Although the intubated group was comparable in gestational age to the nonintubated group ($t = 1.17$, $p<0.1$), their mean birth weight was 181 g less ($t = 2.53$, $p<0.05$), which indicates that both general and local factors may be operative in causing defective amelogenesis in the intubated group (Table 2). However, low birth weight does not seem to predispose to an increase in prevalence of dental defects in the whole group of 63 children. As can be observed in Table 3, there is no statistical difference in the prevalence of defective dentition between children with birth weight less than 1,000 g, and those of 1,000 g and more ($\chi^2 = 0.91$, $p<0.1$).

Effect of Laryngoscopy

In order to determine whether the endotracheal tube per se is responsible for the selective distribution of dental defects in our study population, the data were analyzed by two separate procedures.

Table 4 shows the relationship between the length of intubation and the prevalence of dental defects. Dental defects occurred in 66.7% of the group receiving intubation for less than 1 day (usually for less than two hours), compared to 74.2% in the group intubated for 2 to 64 days ($\chi^2 = 0.28$, $p<0.1$). These results suggest that laryngoscopy, rather than the endotracheal tube itself, may be the more important cause of the selective distribution of dental defects in our sample.

Further support of this conclusion is provided by

Table 2. Group Characteristics of Intubated and Nonintubated Children

	Intubated Group (40 Children)	**Nonintubated Group** (23 Children)	p value
Number of children with defective maxillary anterior teeth (%)	34(85%)	5(21.7%)	<0.001 ($\chi^2=24.46$)
Number of children with defective mandibular anterior teeth (%)	10(25.0%)	5(21.7%)	<0.1 ($\chi^2=0.18$)
Gestational age in weeks (mean ± S.D.)	29.0±2.7	30.3±1.9	>0.1 (t=1.17)
Birth-weight in grams (mean ± S.D.)	1,088.3±257.9	1,269.1±194.3	<0.05 (t=2.53)

Table 3. Relationship of Birth Weight to Prevalence of Dental Defects

Birth Weights	*Number of Children*	*Number of Children With Defective Teeth**
605-999	16	14 (87.5%)
1,000-1,500 g	47	37 (78.7%)

*$\chi^2 = 0.91$, $p<0.1$

analysis of left-sided and right-sided distribution of the defects in the maxillary anterior teeth, because in low birth-weight infants, the laryngoscope usually is applied on the left side of the midline of the maxillary anterior region. Table 5 shows the distribution of 56 defective maxillary anterior teeth on the right and left sides of the 34 intubated children with dental defects. The number of defective maxillary anterior teeth on the left side is twice that on the right (66.1% versus 33.9%). This difference is statistically significant ($\chi^2 = 7.76$, $p<0.01$). By contrast, in the nonintubated group no statistical difference was found between the numbers of defective anterior teeth on the left and right sides (47.4% versus 52.6%).

Table 1 indicates that in the intubated group the maxillary left central and lateral incisors are by far the most commonly affected teeth (hypoplasia is the defect commonly seen in these teeth). Figure 1 shows the appearance of such typically affected left maxillary incisor teeth in intubated children.

Table 5. The Left-sided and Right-sided Distribution of Defective Maxillary Anterior Teeth in Intubated and Nonintubated Children

	Total Number of Maxillary Anterior Teeth Per Side (A B C)	*Number of Defective Maxillary Anterior Teeth*		*p value*
		Left side	Right Side	
Intubated	102 (34 children)	37 (66.1%)	19 (33.9%)	<0.01 ($\chi^2=7.96$)
Nonintubated	15 (5 children)*	9 (47.4%)	10 (52.6%)	<0.1 ($\chi^2=0.29$)

* See Table 2.

Discussion

The present study indicates that the adverse influences of systemic factors on amelogenesis may be compounded by traumatic factors, the most obvious being laryngoscopy and orotracheal intubation. This reasoning is supported by the observation that in the intubated group of children, the distribution of the defective dentition is localized selectively to the region of the maxillary anterior teeth, notably the left central and lateral incisors.

This distribution can be explained better by the traumatic effects of laryngoscopy, rather than by orotracheal

Table 4. Relationship of Length of Intubation to Prevalence of Dental Defects

Length of Intubation	*Number of Children*	
	Total	Defective maxillary anterior teeth (%)†
<1 day*	9	6 (66.7%)
2 days and greater**	31	23 (74.2%)

* Length of intubation usually less than two hours.
** Length of intubation from 2 days to as long as 64 days.
† $\chi^2 = 0.28$, $p<0.1$.

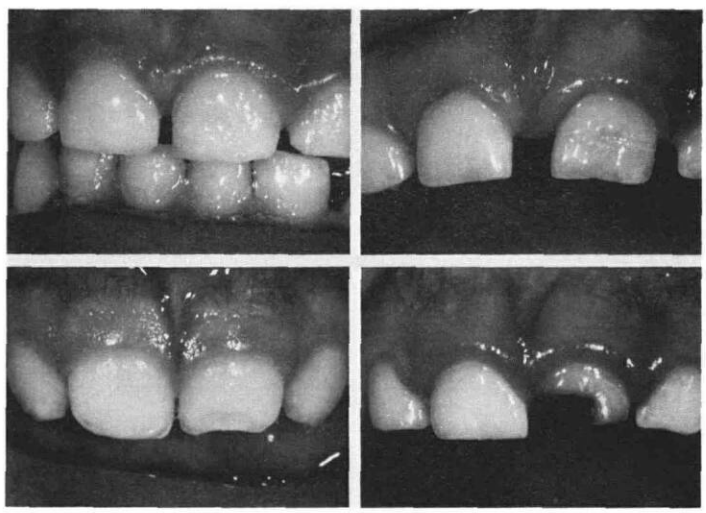

Figure 1. Defective maxillary left central incisor teeth in intubated children.

intubation for the following reasons. First, the orotracheal tube is taped routinely to the midline, and with regular turning of the infant, it should be displaced evenly on both sides of the midline, rather than predominantly on the left. Second, the prevalence of dental defects did not correlate with the duration of orotracheal intubation. Third, it previously has been reported that traumatic damage to erupted maxillary anterior teeth can be inflicted by laryngoscope leverage.[8-12]

In the neonate, the process of laryngoscopy involves insertion of the laryngoscope blade into the right side of the mouth, but the blade has to be brought across just to the left of the midline in order to create enough room for the insertion of the orotracheal tube. The instrument is so constructed that it is always held with the left hand, while the right hand is occupied with insertion of the orotracheal tube along the groove on the right side of the laryngoscope blade. Ideally, no force should be applied to the maxillary alveolar ridge during the process of laryngoscopy. However, in very small infants, especially those of low birth weight, the mandible is so hypoplastic that it does not provide a sufficient fulcrum for lifting the anterior oropharynx and tongue in order to expose the laryngeal opening. Thus, an inadvertent leverage

force sometimes is exerted on the maxillary anterior alveolar ridge, on the left side adjacent to the midline.

The importance of the orotracheal tube cannot be discounted completely as another possible local cause of dental defects. Boice et al.[7] reported a nonsurviving low birth-weight infant who showed a notable concavity of the anterior left maxillary ridge clearly outlining the placement of the orotracheal tube. Sections taken through the notched alveolar ridge showed severe disruption of the developing enamel organ. Other workers,[17,18] have observed development of an indentation on the anterior ridge of mechanically ventilated infants due to continual trauma from orotracheal tubes.

Conclusions

The selective distribution of dental defects in intubated infants to the left maxillary anterior teeth indicates that traumatic injuries produced by laryngoscopy and orotracheal intubation may compound further the already high predisposition of low birth-weight infants to developmental defects in their primary dentitions.

Dr. Seow is a lecturer in pediatric dentistry and Dr. Brown is senior lecturer in pediatric dentistry, University of Queensland Dental School, Turbot St., Brisbane, Queensland, Australia 4000; Dr. Tudehope is director of neonatal intensive care, and Dr. O'Callaghan is developmental pediatrician, Mater Public Hospitals, South Brisbane, Australia 4101. Requests for reprints should be sent to Dr. Seow.

1. Stein, G. Enamel damage of systemic origin in premature birth and diseases of early infancy. Am J Orthod 33:831-41, 1947.
2. Grahnen, H., Larsson, P.G. Enamel defects in the deciduous dentition of prematurely born children. Odont Revy 9:193-204, 1958.
3. Rosenzweig, K.A., Sahar, M. Enamel hypoplasia and dental caries in the primary dentition of prematuri. Br Dent J 113:279-80, 1962.
4. Funakoshi, Y., Kushida, Y., Hieda, T. Dental observations of low birth-weight infants. Pediatr Dent 3:21-25, 1981.
5. Mellander, M., Noren, J.G., Fredén, H., Kjellmer, I. Mineralization defects in deciduous teeth of low birth-weight infants. Acta Paediatr Scand 71:727-33, 1982.
6. Moylan, F.M.B., Selden, E.B., Shannon, D.C., Todres, J.D. Defective primary dentition in survivors of neonatal mechanical ventilation. J Paediatr 96:106-8, 1980.
7. Boice, J.B., Krous, H.F., Foley, J.N. Gingival and dental complications of orotracheal intubation. JAMA 236:957-58, 1976.
8. Wasmuth, C.E. Legal pitfalls in the practice of anesthesiology. Part 2. Complications of endotracheal anesthesia. Anesth Analg 39:138-40, 1960.
9. Bamforth, B.J. Complications during endotracheal anaesthesia. Anesth Analg 42:727-32, 1963.
10. Powell, J.B., Keown, K.K. Endotracheal aspiration of a tooth. Anesth Analg 44:355-57, 1965.
11. Henry, P.J. Mouth guards in general anesthesia. Med J Aust 1:911-12, 1969.
12. Stevens, D.O. Prevention of traumatic dental and oral injuries, in Traumatic Injuries of the Teeth, Andreasen, J.O., ed. Philadelphia; W.B. Saunders, Co., 1981, pp 419-37.
13. Tudehope, D.I., Burns, Y.R., O'Callaghan, M., Mohay, H. Minor neurological abnormalities during the first year of life in infants of birth-weight less than 1,500 g. Aust Paediatr J 17:265-68, 1981.
14. Cleghorn, G.J., Tudehope, D.I., Masel, J.P. Rickets in extremely low birth-weight infants. Aust Paediatr J 17:285-89, 1981.
15. Tudehope, D.I., Burns, Y.R., O'Callaghan, M., Mohay, H., Silcock, A. The relationship between intrauterine and postnatal growth on the subsequent psychomotor development of very low birth-weight infants. Aust Paediatr J 13:3-10, 1983.
16. Masel, J.P., Tudehope, D., Cartwright, D., Cleghorn, G. Osteopenia and rickets in the extremely low birth-weight infant — A survey of the incidence and a radiological classification. Aust Radiol 26:83-96, 1982.
17. Wetzel, R.C. Defective dentition following mechanical ventilation (letter to the editor). J Pediatr 97:334, 1980.
18. Krous, H. Defective dentition following mechanical ventilation (letter to the editor). J Pediatr 97:334, 1980.

Quotable quote: esthetics and society

The ability to alter appearance has affected how we respond esthetically to differences. An example of this situation is the case of relative maxillary protrusion, the Class II malocclusion. A child who may have been acceptably "buck-toothed"in the 1950s and who may have been a candidate for orthodontic braces in the 1950s and 1960s, now often has a dentofacial deformity, the treatment for which is maxillofacial surgery. The expansion of medical attention toward this nonlife-threatening condition serves to increase its unacceptability as normal. As a result, a minor variant of normal becomes a deformity.

When something is correctable, our willingness to accept it untouched is reduced. The more esthetic variation can be controlled, the less likely we are to tolerate difference. Those who choose not to avail themselves of surgical correction experience peer and community pressures. New forms of treatment also may serve to reawaken concerns in people who had accepted their appearance and were adapted to their social roles.

Strauss, R.P. Ethical and social concerns in facial surgical decision making. Plastic Reconstr Surg 72:727, 1983.