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Increased prevalence of developmental dental defects in low birth-weight, prematurely born children: a controlled study

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Abstract

This study of prematurely born children shows that the prevalence of enamel defects increases with decreasing birth weight. In very low birth-weight (VLBW, < 1500 g) children, the prevalence was 62.3% compared to 27.3% in the low birth-weight (LBW, 1500-2500 g) group. In children with normal birth weight of > 2500 g, the prevalence was 12.8%. Both systemic and local factors contribute to the etiology of dental defects. In children who were intubated at birth, left-sided defects on maxillary anterior teeth occurred twice as frequently as right-sided defects, probably the result of trauma from left-sided laryngoscopy.

Defects of the enamel can result from various disturbances during amelogenesis. These defects may manifest as surface breaks and decreased enamel thickness known as enamel hypoplasia. Alternatively, the defect may be evident as an abnormality of the translucence of enamel known as enamel opacity. Clinically these enamel defects may present with problems of esthetics. In addition, enamel hypoplasia may predispose to plaque accumulation and caries, and in severe cases even space loss and malocclusion. There is evidence that enamel defects occur with a very high frequency in prematurely born children. As early as 1936 Stein reported that 5 of 12 prematurely born children had enamel hypoplasia (Stein 1947). Later studies revealed markedly discrepant prevalences varying from 20 to 100%.[1]

While the pathogenesis of the dental defects remains unclear, it is probable that both systemic disturbances and local factors contribute to the etiology. In the authors' previous studies of prematurely born children with neonatal rickets, they identified derangement of calcium metabolism as a possible systemic factor in the pathogenesis of dental defects (Seow et al. 1984b). In addition, they found that local traumatic factors such as laryngoscopy and endotracheal intubation applied during the neonatal period may contribute to the etiology of these defects (Seow et al. 1984a). With decreasing birth weight there is greater propensity for systemic illness and the likelihood of endotracheal intubation increases. Hence, it is likely that the lower the birth weight, the greater the prevalence of enamel defects. In this study, the authors attempt to further delineate the causes of enamel defects in prematurely born children by studying the prevalence of enamel defects in 3 groups of children with different birth weights: very low birth-weight (VLBW, < 1500 g); low birth-weight (LBW, 1500-2500 g); and normal birth weight (> 2500 g). This classification of newborn birth weights is an accepted standard recommended by the American Academy of Pediatrics (Silverman 1967).

Patients and Methods

The children in the VLBW group (< 1500 g) were those born in the period 1983-1985, and 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 follow-up of all infants of low birth weights managed at the Mater Mothers' Hospital. Children in the LBW group (1500-2500 g) and the normal birth weight group (> 2500 g) were selected at random from the birth register at the same hospital. These children were born at the same time period as those in the VLBW group. One hundred and fifty-seven (97.0%) of the 162 children who the authors were able to contact consented to the study. The mean (SD) ages of all the children in the study at the time of examination was 25.6 ± 9.1 months (range 9-42 months).

The VLBW group was comprised of 77 children (31 males, 46 females). Their mean birth weight was 1177 ± 193 g (range 783-1499 g) and their mean gestational age was 29.4 ± 2.5 weeks (range 22-33 weeks). Thirty (37.7%) of these children were intubated during the neonatal period while 47 were not.

The LBW (1500-2500 g) was comprised of 33 subjects (14 males, 19 females). Their mean birth weight was 2175

± 273 g (range 1577-2480 g) and their mean gestational age was 36.8 ± 2.2 weeks (range 32-41 weeks). None of the children in the LBW group were intubated.

In the normal birth weight group (> 2500 g) 47 children (25 females, 22 males) were available for study. They were all products of full-term pregnancies and their mean birth weight was 3360 ± 450 g (range 2510-4045 g). None of these children were intubated during the neonatal period.

The dental examinations were performed under ideal conditions at the University Dental School. The teeth were dried and a mirror and probe used to detect dental 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 all dental defects were recorded in a comprehensive chart. Intraoral photographs were taken in some children. Postnatal medical and dental histories were obtained from the patients. Maternal and neonatal medical histories were obtained from hospital records.

Data were analyzed using 2 x 2 contingency tables and χ^2 tests were used to detect statistical differences between groups.

Results

Prevalence of Enamel Hypoplasia

Table 1 shows the prevalence of enamel hypoplasia in the 3 groups of subjects. In the VLBW groups, 48 of 77 children showed at least one tooth with enamel defect, giving a prevalence of 62.3%. Of these, 8 children (10.4%) had enamel opacities alone and 40 (51.9%) had enamel hypoplasia with or without opacities. However, in the LBW group a much lower prevalence of 27.3% was obtained (9 of 33 children affected). In the normal birth weight group of children there was a much lower prevalence of 12.7% where only 6 of 47 children were affected. Of these, only 3 children (6.1%) had opacities alone. The difference in prevalence among the 3 study groups is statistically significant ($P < 0.001$), indicating that prevalence of enamel hypoplasia varies in direct relation to birth weight.

TABLE 1. The Prevalence of Enamel Hypoplasia in Children With Very Low (VLBW), Low (LBW), and Normal Birth Weights

Group	Birth Weight (g) Mean ± SD	Prevalence of Enamel Defects Opacities	Hypoplasia	Total
VLBW (N = 77)	1177 ± 193	8 (10.4%)	40 (51.9%)	48 (62.3%)
LBW (N = 33)	2175 ± 273	2 (6.1%)	7 (21.2%)	9 (27.3%)
Normal (N = 47)	3360 ± 450	3 (6.4%)	3 (6.4%)	6 (12.7%)

The difference in prevalence of enamel defects in the 3 groups is statistically significant ($\chi^2 = 57.7$, df = 2, $P < 0.001$).

Distribution of Enamel Hypoplasia

The authors also analyzed the distribution of enamel hypoplasia in the 3 groups of children for possible insight into the etiology of the dental defects. Table 2 shows the distribution of enamel hypoplasia in the study groups. In the VLBW group 63.1% of all affected teeth occurred on the left side compared with 36.9% on the right side. This difference was statistically significant ($P < 0.005$). In contrast, in both the low and normal birthweight groups, the dental defects appeared fairly evenly distributed on both left and right sides ($P > 0.1$).

TABLE 2. The Distribution of Enamel Hypoplasia in Children With Very Low (VLBW), Low (LBW), and Normal Birth Weights

Birth Weight	Affected Teeth (per cent of total) Left Sided	Right Sided	P value
VLBW (<1500 g)	63.1	36.9	<0.005
LBW (1500-2500 g)	58.3	41.7	>0.1
Normal (>2500 g)	52.2	47.8	>0.1

The increased numbers of affected teeth on the left side in the VLBW group is most likely related to trauma from laryngoscopy and endotracheal intubation which affect mainly maxillary anterior teeth (Seow et al. 1984a). In order to determine this, the distribution of affected maxillary anterior teeth in intubated and nonintubated children in the VLBW group were analyzed. As shown in Table 3, the intubated children showed a twofold increase in numbers of affected teeth on the left side compared to the right (67.2% of all affected maxillary teeth vs. 32.8%, $P < 0.005$). In contrast, in the nonintubated group, there was no difference in distribution of affected teeth between the left and right sides (53.8% vs. 46.2%, $P > 0.1$).

Discussion

Previous studies of prematurely born, low birthweight children have indicated a high overall prevalence of dental defects ranging from 20 to 100%.[2] However, most of these studies were done on isolated groups without normal birth-weight control children or poorly selected controls. Table 4 shows the summary of clinical studies on prematurely born children which included

TABLE 3. Distribution of Affected Maxillary Anterior Teeth (Incisors and Canines) in 47 Intubated and 30 Nonintubated VLBW Children

	Affected Maxillary Anterior Teeth (% of total) Left	Right	P Value
Intubated	67.2	32.8	<0.005
Nonintubated	53.8	46.2	>0.1

children with normal birth weights. As can be seen from the table, the prevalence of dental defects in LBW children in these studies is around 30%. The present study gives a comparable figure of 27.3% for this birth-weight group. In the VLBW group, however, prevalence figures

TABLE 4. Analysis of Studies on the Prevalence of Enamel Defects in Prematurely-Born Children Which Included Children With Normal Birth Weights

Study	*Birth Weight*	*Per Cent† Acceptance*	*Prevalence of Enamel Defects**
Grahnen and Larsson 1958	LBW	87.1	32.1
	Normal	77.2	13.1
Rosenzweig and Sahar 1962	LBW	N.R.	23.8
	Normal	N.R.	1.2
Mellander et al. 1982	LBW	72.2	30.7
	Normal	30.9	39.6
Johnsen et al. 1984	VLBW	N.R.	52.0
	Normal	N.R.	26.0
Seow et al. (this study)	VLBW	100	62.3
	LBW	100	27.3
	Normal	97	12.8

N.R.—not reported; *—Enamel defects include both enamel hypoplasia and opacities; †—denotes number studied/number approached × 100.

are comparatively higher. In the study of Johnsen et al. (1984), there was a prevalence of enamel defects of 52%. In the present study, the prevalence for the VLBW group is slightly higher, at 62.3%.

A major difference between this study and previous studies lies in the prevalence of dental defects in normal birth-weight children. In the present study, this prevalence is 12.8%, a figure close to that of the study by Grahnen and Larsson (1958). However, Mellander et al. (1982) and Johnsen et al. (1984) reported exceptionally high prevalence rates of 39.6% and 26%, respectively. The reasons for this gross discrepancy may be due to: (1) the use of subjects from a clinic population as in the study of Johnsen et al. (1984) or (2) sampling error from low acceptance rates as in the study of Mellander et al. (1982, Table 4).

The reasons for the difference in prevalence of enamel hypoplasia in the various birth-weight groups are most likely related to both systemic and local factors. Children with the most premature births and lowest birth weights have the highest tendency to suffer from systemic derangements which can affect dental development adversely. Enamel hypoplasia resulting from systemic disturbances usually results in several teeth being affected as shown in Figure 1. These systemic factors include neonatal asphyxia (Grahnen et al. 1969), respiratory distress syndrome,[3] maternal pre-eclampsia (Via and Churchill 1959), maternal diabetes (Grahnen and Edlund 1967; Tsang et al. 1973), hyperbilirubinemia (Grahnen and Granath 1962; Funakoshi et al. 1981), and neonatal infection (Funakoshi et al. 1981).

A previous study of a group of prematurely born, VLBW children with neonatal rickets showed that every one was affected by enamel malformation (Seow et al. 1984b). As rickets is a major disturbance in calcium metabolism, it is not surprising that such a high prevalence of enamel defects is noted. In fact, all prematurely born children suffer from low calcium stores and disturbed calcium metabolism, with the lowest birth-weight children most severely affected (Tsang et al. 1973; Tsang 1983). Other perinatal factors associated with prematurity such as hypoxia, sepsis, cerebral injuries, and hyperbilirubinemia may also indirectly cause hypocalcemia (Tsang et al. 1983). It is possible that it is through this

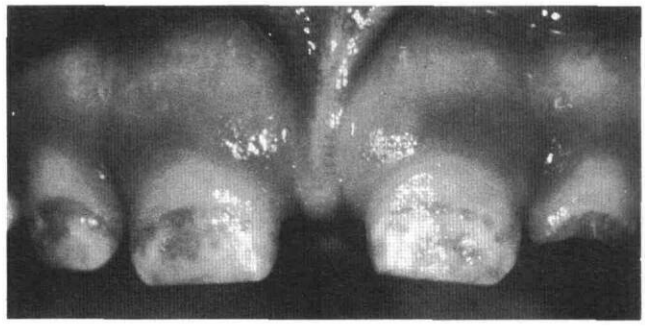

FIG 1. Enamel hypoplasia resulting from systemic disturbances usually result in several teeth being affected.

mechanism that many systemic factors cause enamel hypoplasia in prematurely born children.

Local factors also contributed to the difference in prevalence of enamel hypoplasia in the 3 study groups of children. Children with the lowest gestational ages and birth weights tend to suffer most from respiratory problems in the neonatal period and have the greatest need for laryngoscopy and endotracheal intubation. These traumatic procedures have been associated with enamel hypoplasia of the maxillary teeth (Moylan et al. 1980; Wetzel 1980). A previous study has shown that children who underwent laryngoscopy and endotracheal intubation in the neonatal period had a fourfold increase in prevalence of defects of the maxillary anterior teeth compared with nonintubated children (Seow et al. 1984b). In addition, these defects occurred mainly on the left maxillary anterior teeth, suggesting left-sided pressure from the laryngoscope blade (Fig 2, next page). The present study confirms the authors' previous observations. Only the VLBW group, which included intubated children, demonstrated an increase in dental defects on the left side, confirmed by analysis of the distribution of defects on the anterior maxillary teeth.

Conclusions

This study shows that the lower the birth weight of a child the greater the propensity to develop enamel hypoplasia. The pathogenesis of these defects is related

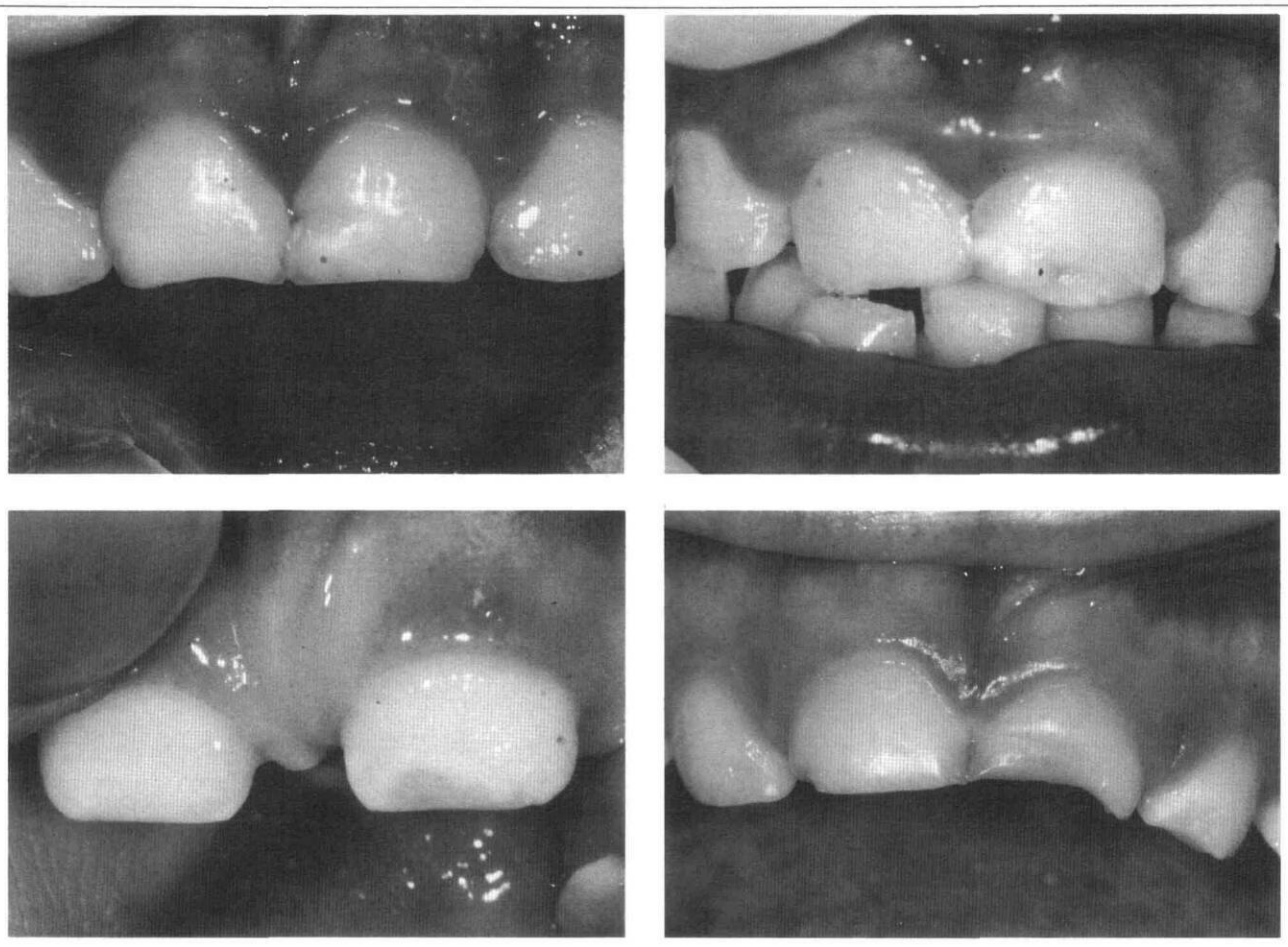

FIG 2. Defects occurred mainly on the left maxillary anterior teeth suggesting left-sided pressure from the laryngoscope blade.

to both systemic and local factors.

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Medications can affect oral health

Medications prescribed for medical conditions can have an effect on the mouth and gums and dentists should obtain a complete medical history before treating patients.

Oral contraceptives, for instance, can worsen existing periodontal disease. Some commonly prescribed drugs as Dilantin (used to treat epilepsy) and Procardia (for heart ailments) can create fibrous tissue that, if untreated, may cause an overgrowth of gum tissue that can lead to periodontal disease.

Some antibiotics or steroid medications may make the patient susceptible to yeast infections because these can cause an imbalance in the bacterial population found in the mouth. Tetracycline, when given to a child whose primary teeth are still forming, may cause permanent staining of the teeth.

The most common side effect of medication is dry-mouth syndrome (xerostomia), or lack of saliva. People with this condition need to sip fluids throughout the day. They often have difficulty chewing dry food without liquid. The most common drugs that can cause dry mouth are antihistamines and antichlinergics, which interfere with the involuntary nervous system. Other medications that can dry up saliva are tranquilizers, anti-depressants, anti-psychotics, anti-spasmodics, anti-hypertensives, diuretics, appetite suppressants, and certain anti-inflammatory drugs such as Motrin.

The following medications are trade names of some of the 400 drugs that can cause dry mouth syndrome: Actifed, Benadryl, Chlor-Trimeton, Dimetane, Dimetapp, Donnatal, Diuril, Elavil, Eutonyl, Haldol, Ismelin, Lasix, Librium, Lomotil, Ornade, Preludin, Serpasil, Sinequan, Sudafed, Tenuate, Thorazine, Triavil, and Valium.

Pediatric Dentistry: a step backward to leap forward

This issue of *Pediatric Dentistry* is coming to you very late — for the last time. This is the first issue that has been generated completely in-house. Every word, all of the rules, each page number, even the white space on pages has been put there by the editorial staff. All two of us. While we did plan that the September issue would be the first to be produced completely by our desktop publishing system, the many problems associated with setting up a new computer system for the Central Office *and* moving the publications department fromIowa to Chicago this summer could not have been foreseen. In some ways it was a step backward — a completely new way of doing things was in order.

But now we can leap forward. The new system will allow us to serve our advertisers better. In the past we had a three-month publication deadline; we now will be able to take ads 30 days prior to press time instead of 90. Our authors also will be better served; their work will get published with less turn-around time than ever before. But most importantly, our readers will benefit.

With more control in the publications department we will be able to eliminate many frustrations associated with the traditional printing cycle of editing, proofing, corrections, and galleys that take up so much valuable time. There will be more articles per dollar of production, they will be more readable, and, this issue notwithstanding, your *Journal* will be more timely.

Look at your upcoming issues closely — you will notice many minor changes and a few major ones as well. Let us know what you think; this is your *Journal* and we want to do everything we can to make it a superb publication that represents our specialty in the best light.