A study of the development of the permanent dentition in very low birthweight children

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Abstract

There have been no previous studies on dental maturation of prematurely born, very low birthweight (< 1500 g, VLBW) Caucasian children. This study investigated dental development and prevalence of enamel defects in a group of 55 VLBW children (mean age at dental examination 7.7 ± 2.2 years, mean birthweight 1203 ± 240 g, and mean gestational age 29.8 ± 2.4 weeks) compared to 55 normal birthweight (NBW) children matched for race, sex, and age. Dental maturity was determined from panoramic radiographs. Overall, VLBW children experienced a delay in dental maturation of approximately 0.29 ± 0.54 years compared with NBW children (P < 0.02). The VLBW children younger than 6 years of age showed the greatest delay of 0.31 ± 0.68 years (P < 0.001). In contrast, children aged 9 years and older had no difference in their dental ages compared to controls (P > 0.1), showing that "catch-up" growth had occurred by age 9 years. Children of birthweight < 1000 g with gestational ages < 30 weeks showed the greatest lag period in dental maturation. Clinical examination also showed that VLBW children had a higher percentage of enamel defects in the permanent first molars (21% versus 11%, P < 0.02) and lateral incisors (12% versus 0%, P < 0.01). As the permanent teeth commence their mineralization a few months after premature birth, it is hypothesized that there is persistent systemic derangement sufficient to affect enamel formation postnatally for some time in VLBW children. (Pediatr Dent 18:379–84, 1996)

In recent years, prematurely born, very low birthweight children (VLBW, < 1500 g) have increased survival rates and greatly improved outcomes because of increasing sophistication of neonatal care. Previous dental research on these children had focused mainly on two aspects — enamel hypoplasia in the primary dentition[1–12] and palatal deformities.[13–15] Enamel hypoplasia in VLBW children has been associated with systemic complications of birth prematurity and low birthweight,[1–12] as well as traumatic laryngoscopy, whereas the palatal deformities are most likely related to prolonged endotracheal intubation.[13–15]

These oral complications of prematurely born children are well researched, but other aspects of dental development are not. Only one study[16] examined dental maturity of the permanent dentition on low birthweight (LBW, < 2000 g) children, and this was confined to a sample of African-Americans. Another study[12] of prematurely born children, which included a small group of older children, mentioned that 58% of permanent central incisors were affected by enamel hypocalcification. Our study investigated the rate of dental development and prevalence of enamel hypoplasia in the permanent molars and incisors in a group of Caucasian, VLBW, prematurely born children compared with a group of race-, age-, and sex-matched, healthy normal birthweight (NBW) controls.

Subjects and methods

VLBW children

The prematurely born VLBW children (< 1500 g birthweight) were part of a cohort of children followed for a longitudinal dental study on the oral complications of low birthweight and prematurity. The subjects, aged approximately 4–12 years, were selected randomly from the register of all preterm children kept at the Mater Mothers' Hospital, Brisbane, and invited to participate. Apart from minor illnesses and asthma, the subjects did not suffer serious medical outcomes of their birth prematurity or have systemic syndromes. Of the children whose addresses were known, consent rate for the study was more than 90%.

Normal birthweight (NBW) controls

For every VLBW child recruited into the study, a control child of normal birthweight was selected randomly from the records kept at the University Dental School. These children all had panoramic radiographs exposed as part of their dental examinations. Their chronological ages at time of examination were matched to within 6 months with those of the prema-

turely born group. The NBW control children were healthy and did not have any serious illnesses or systemic syndromes.

Dental examinations

The dental examinations were performed at the University Dental School pediatric clinic by the author. It was not possible to perform the examinations blind. The teeth were dried with gauze, and examined for enamel hypoplasia, enamel opacities and dental caries. A simplified DDE (development defects of enamel) index[17] was used to chart the enamel defects. Enamel hypoplasia was defined as a break in the continuity of the enamel in the form of pits, grooves, or missing enamel. Enamel opacity was defined as a change in the translucency of enamel without a break in enamel continuity. In previous studies[8–13], the intraexaminer variability[18] for the diagnosis of enamel defects has been shown to be insignificant ($P > 0.1$)

Assessment of dental ages

Dental ages were assessed from panoramic radiographs by the author using the method of Demirjian et al.[19] employed in previous studies.[20, 21] In this method, the individual radiological appearances of the seven permanent teeth on the left side of the mandible were evaluated according to developmental criteria. Each tooth was categorized into one of eight developmental stages, and a numerical score was obtained from standard reference charts provided in Demirjian's study.[19] The summed scores on all seven teeth gave a dental maturity score. The dental age of each patient was obtained by comparing the dental maturity score with normal standards. The difference between the dental age (DA) and the chronological age (CA) demonstrated the delay or acceleration in dental maturity. Consistency of intraexaminer scoring for dental age assessment had been established in previous studies.[19, 20]

Statistical analysis

The student's *t*-test or chi-square test, wherever appropriate, was used for data analysis. The alpha value was set at 0.05.

Results

Demographics (Table 1)

Altogether 55 VLBW children (25 boys and 30 girls) were recruited into the study. Their mean birthweight was 1203 ± 240 g, and their mean gestational age was 29.8 ± 2.4 weeks.

There were 55 normal birthweight (NBW) control children, case-matched with each VLBW subject for race, sex, and age at panoramic exposure. Their mean birthweight was 3397 ± 243 g and their mean gestational age 40 ± 2.4 weeks. The mean ages of the children at dental examination were 7.7 ± 2.7 and 7.7 ± 0.6 for the VLBW and control groups respectively.

Table 1. Demographic data of subjects in study

	VLBW	*Control*
Total	55	55
Boys	25	25
Girls	30	30
Mean birthweight (g) (Range)	1203 ± 240 (652–1499)	3397 ± 243 (2784–3778)
Mean gestational age (wks) (Range)	29.8 ± 2.4 (24–35)	40 ± 2.4 (38–42)
Mean age at exam (yrs) (Range)	7.7 ± 2.2 (4.0–12.2)	7.7 ± 0.6 (3.5–12.5)

Enamel defects in permanent dentition

As the permanent first molars and central and lateral incisors were the only permanent teeth present in the mean age groups, these teeth were examined for enamel opacities and hypoplasia.

As shown in Table 2, 13 (33%) VLBW children showed enamel opacity of at least one permanent tooth, and another four (10%) had enamel hypoplasia of at least one permanent tooth, giving a total prevalence of 17 (43%) children affected with enamel defects. In contrast, only six (15%) of the NBW children had enamel opacity, and another four (10%) had enamel hypoplasia of at least one permanent tooth, giving a total prevalence of 10 (25%) children with enamel defects. These differences were statistically significant (chi-square = 7.2, df = 1, $P < 0.01$).

The percentages of teeth affected are shown in Fig 1. Overall, the prevalence of enamel defects in the VLBW group was significantly higher than the NBW group (chi-square = 10.86, df = 1, $P < 0.001$). In the VLBW group, four (3%) of the 141 permanent first molars examined showed enamel hypoplasia, and 24 (17%) showed enamel opacity, yielding a total of 30 (20%) teeth affected with enamel defects. Of the NBW children, identical numbers of first permanent molars with enamel hypoplasia compared to the VLBW group were found. However, only 10 (8%)

Table 2. Prevalence of enamel defects in the permanent dentition in VLBW children compared with controls

	Number (%) With at Least One Permanent Tooth Affected		
	VLBW (N = 40)	*NBW (N = 40)*	*P value*
Enamel opacity	13 (33%)	6 (15%)	$P < 0.01$
Enamel hypoplasia	4 (10%)	4 (10%)	NS
Total	17 (43%)	10 (25%)	$P < 0.01$

NS: not significant.

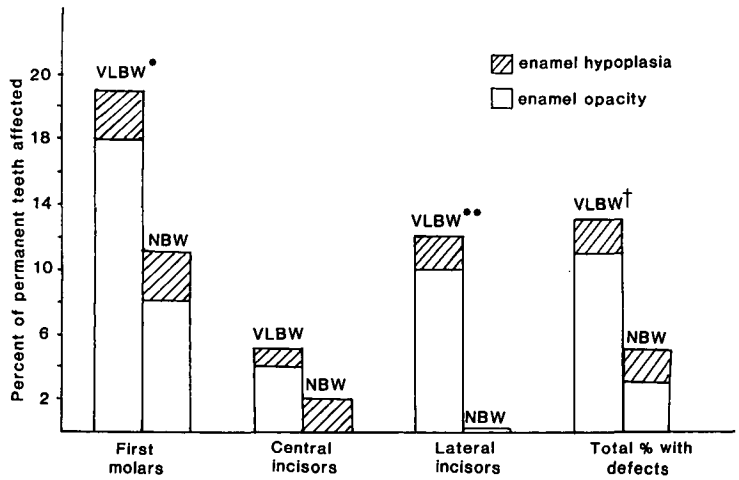

Fig 1. The percentage of permanent first molars, central and lateral incisors affected by developmental enamel defects. * $P < 0.02$; ** $P < 0.01$; † $P < 0.001$.

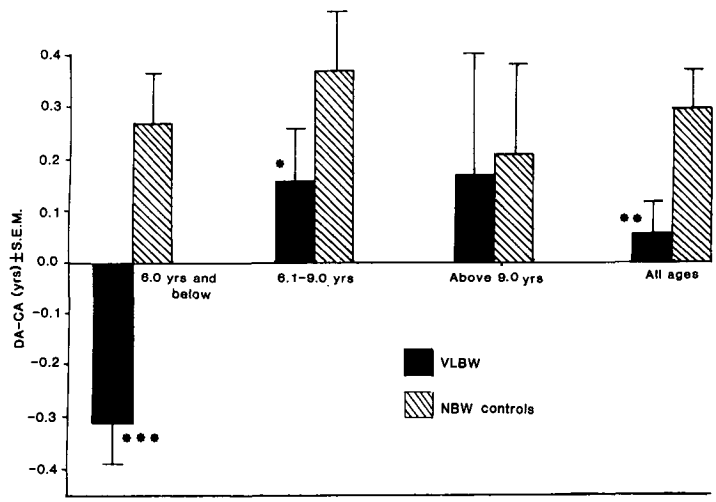

Fig 2. Mean differences between the dental and chronological ages (DA-CA). * $P < 0.05$; ** $P < 0.02$; *** $P < 0.001$.

teeth were affected with enamel opacity, giving a total of 14 (11%) teeth affected with enamel defects. The difference in total prevalence of enamel defects in the permanent first molars between the two groups was statistically significant (chi-square = 5.46, df = 1, $P < 0.02$).

In contrast, in the case of the permanent central incisors, although the VLBW group showed a slightly higher percentage of enamel defects (5%) than did the NBW group (2%), the difference was not significant. However, in the case of the permanent lateral incisors, there was a prevalence of enamel defects of 12% in the VLBW compared with 0% in the NBW group (chi-square = 9.22, df = 1, $P < 0.01$).

Differences between dental ages (DA) and chronological ages (CA)

Table 3 shows the differences between the dental and chronological ages of VLBW children compared with controls. Overall, the VLBW group had a mean CA of 7.74 ± 2.17 years compared with a mean DA of 7.76 ± 2.46 years, giving a mean difference of 0.05 ± 0.59 years. In contrast, the NBW group showed an overall mean DA of 7.78 ± 2.36 years, and a mean CA of 7.48 ± 2.30 years, giving a mean difference of 0.29 ± 0.54 years. The overall difference in dental maturity between the VLBW and NBW groups was statistically significant ($P < 0.02$, Fig 2).

In the case of the VLBW group aged 6 years and younger, the mean DA was only 4.16 ± 0.64 years compared with their mean CA of 4.74 ± 0.45 years. The difference between these two ages (DA-CA) indicated that there was a delay of dental maturation of 0.31 ± 0.68 years. In contrast, in the similar age group of NBW children, there was an acceleration of dental maturation of 0.26 ± 0.40 years. This difference in the dental maturation rate between the VLBW and NBW groups (0.46 years) was statistically significant ($P < 0.001$).

In the children aged 6.1–9.0 years, the VLBW group also showed a lower mean (DA-CA) of +0.15 ± 0.55 years than the NBW group (+0.3 ± 0.55 years, $P < 0.05$), although the difference is less than that observed in the younger age group (Fig 2).

In contrast, in the children aged 9.0 years and older, the VLBW children showed a mean (DA-CA) of 0.17 ± 0.57 years compared with 0.20 ± 0.67 years in the NBW group, indicating that there were no significant differences in dental maturity rates between the VLBW and the control groups ($P > 0.1$, Fig 2).

The influence of birthweight, gestational age, and gender on dental development

To examine the influence of birthweight on dental development, the VLBW group was divided into those born weighing < 1000 g, and those with birthweights 1000–1500 g. As shown in Table 4, the group with birthweights < 1000 g had a mean difference of -0.30 ± 0.9 years between their mean dental and chronological ages. This figure was significantly different from the value of 0.09 ± 0.53 years of those born with birthweights 1000–1500 g ($P < 0.01$), as well as that of controls (0.20 ± 0.67 years, $P < 0.01$).

The effect of gestational age was examined by grouping the VLBW children into two groups — one of gestational age 24–30 weeks, and another of gestational age > 30 weeks. The results (Table 4) showed that the younger gestational age group showed a mean (DA-CA) of -0.21 ± 0.65 years, whereas higher gestational age group had a mean of 0.23 ± 0.53 years. The difference between the two gestational age groups was

TABLE 3. CHRONOLOGICAL AND DENTAL AGES OF VLBW AND CONTROL NBW CHILDREN

	N	Mean Chronological Age (CA) (yrs ± SD)	Mean Dental Age (DA) (yrs ± SD)
VLBW			
6.0 and younger	16	4.74 ± 0.45	4.16 ± 0.64
6.1–9.0	26	7.71 ± 0.60	7.89 ± 0.75
Older than 9.0	13	10.62 ± 0.93	10.80 ± 1.13
All ages	55	7.74 ± 2.17	7.76 ± 2.46
NBW			
6.0 and younger	16	4.61 ± 0.64	4.88 ± 0.86
6.1–9.0	26	7.69 ± 0.59	8.05 ± 0.76
Older than 9.0	13	10.59 ± 1.06	10.71 ± 1.29
All ages	55	7.48 ± 2.30	7.78 ± 2.36

TABLE 4. INFLUENCE OF BIRTHWEIGHT, GESTATIONAL AGE AND GENDER ON DIFFERENCE BETWEEN DENTAL AGE (DA)

		N	Mean DA–CA (yrs ± SD)	P value*
Birth				
Weight (g)	<1000	10	-0.30 ± 0.90	$P < 0.01$
	1000–1500	45	0.09 ± 0.53	$P < 0.01$
	Control	55	0.20 ± 0.67	
Gestation				
Age (wks)	24–30	26	-0.21 ± 0.65	$P < 0.01$
	30–35	29	0.23 ± 0.53	
	Control	55	0.20 ± 0.67	
Sex				
VLBW	Girls	30	0.09 ± 0.68	$P < 0.001$
	Boys	25	-0.06 ± 0.55	$P < 0.001$
Control	Girls	30	0.32 ± 0.51	
	Boys	25	0.27 ± 0.58	

* Compared to controls.

statistically significant ($P < 0.01$). In contrast, there was no significant difference between (DA-CA) of the group with gestational ages > 30 weeks and the control group ($P > 0.1$).

To examine the effects of gender, the mean values of (DA-CA) of girls and boys in both the VLBW and control groups respectively, were compared. The results (Table 4) indicated that both groups with VLBW differed significantly from their respective NBW control groups. VLBW boys showed the greatest lag in dental maturation, with a mean (DA-CA) value of -0.06 ± 0.55 years, compared with 0.27 ± 0.58 years in the NBW boys ($P < 0.001$). VLBW girls showed less delay in dental maturation than the VLBW boys, with a mean (DA-CA) of 0.09 ± 0.68 years. However, this figure was significantly less than the mean of 0.32 ± 0.51 years for NBW girls ($P < 0.001$).

Discussion

Prematurely born (< 37 weeks gestation) children generally comprise approximately 5–7% of all live births in industrialized Western countries.[22] In recent years, there is increasing interest in not only the survival rates of these children, but also their physical and cognitive growth and development. Long-term studies have reported delay in physical and cognitive development in most VLBW (< 1500 g) children, including those who were apparently "normal" at birth.[23–25] The developmental delay was evident well into school age, with the lowest birthweight children most affected.[23–25]

Our investigation shows that the permanent dentition, namely the first permanent molars and incisors, are affected by mineralization defects. One other report,[12] which included a group of 38 premature children with at least one maxillary incisor, gave a prevalence of enamel hypoplasia in the permanent central incisors of 58%, but other permanent teeth were not examined.

In a previous study, Seow et al.[10] showed that poor mineralization of long bones in the premature infants was correlated with enamel hypoplasia of the primary teeth and hypothesized that the teeth and bones were deprived of normal mineral stores due to low supplies of calcium and phosphate. Inadequate mineral supply occurs commonly in prematurity as a complication of many systemic medical conditions associated with premature birth, such as kidney and liver immaturity and inadequate gastrointestinal absorption.[26] As the permanent teeth commence mineralization a few months after the premature birth, the presence of mineralization defects in these teeth suggests that there is persistent systemic derangement sufficient to affect enamel formation.

The present study also showed that development of the permanent dentition is delayed in VLBW children. The delay in dental maturity was most severe in 6-years-olds in whom there was a mean lag of 0.57 years compared to control children. At ages 6.1 to 9 years, the delay was less, with a mean difference of 0.16 years compared with the controls. However, after age 9 years, there were no differences between the VLBW and control groups, indicating that catch-up growth had occurred. In relation to other physical growth parameters, it is interesting to note that similar catch-up growth has also been reported for skeletal development in some older preterm children.[23–25]

Although the dental maturity standards used in this study were those of French-Canadian children pro-

vided in the report of Demirjian et al.,[19] the employment of a race, age, and sex-matched control group in this study should have canceled sampling error from using the published standards derived from a different population and ethnic group.

Of interest is the finding from this study that the subjects with the lowest birthweights of < 1000 g, showed the greatest lag period in their dental development, although the rest of the subjects with higher birthweights of 1000–1500 g were also significantly delayed compared with control children. Related to this is the finding that only children with gestational ages of 24–30 weeks had a mean delay in dental development, whereas those with gestational ages from 30–35 weeks did not differ significantly in their dental ages from the full-term control children. Of clinical importance is the fact that the mean delay in dental age of those with mean gestational ages 28 weeks was 0.21 years or 10.9 weeks, which corresponded approximately with the difference between their biological and chronological ages. It is interesting to note that in a previous study on emergence of the primary dentition,[27] it was found that premature children also were delayed in their dental eruption by the same extent.

In contrast, the findings of this study differ from those of Harris et al.[16] on LBW African-American children in which developmental delay was found to be limited to only the first molars and central incisors, and evident mainly in the older children. The disparity in results of the two studies is most likely due to racial and ethnic differences (African-American vs white Australians), as well as differences in birthweights (LBW, < 2000 g versus VLBW, < 1500 g) and the employment of different methods of assessment of dental maturity.

The clinical significance of dental developmental delay would be in relation to orthodontic treatment. In the age groups of preterm children who have significant differences between dental and chronologic ages, orthodontic intervention is rarely performed. On the other hand, it is noteworthy that preterm children older than 9 years of age may be considered to have the same dental ages as their full-term peers when planning for orthodontic treatment.

Conclusions

1. VLBW, prematurely born children have delayed dental development of the permanent dentition, with greatest delay being experienced in those younger than 6 years of age and least in those aged 9 years and older.
2. There was a higher prevalence of enamel defects in the first permanent molars and incisors in the VLBW group than in the NBW children.

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Surgery can reduce frequency of epileptic seizures

REDUCED MORTALITY AND INCREASED EMPLOYMENT RESULT

Patients with a certain form of epilepsy can get long-term relief from seizures through an operation that removes a small amount of brain tissue, according to an article in a recent issue of *The Journal of the American Medical Association.*

Michael R. Sperling, MD, from the Comprehensive Epilepsy Center, Graduate Hospital, University of Pennsylvania, and colleagues studied the efficacy of anterior temporal lobectomy on 89 patients with refractory epilepsy.

The five-year follow-up found that 70% of patients were seizure free, 9% had seizures on fewer than three days per year or only suffered from nighttime seizures, 11% had greater than 80% reduction in seizure frequency, 6% had less than 80% reduction in seizure frequency and 4% died of causes unrelated to surgery.

The authors continue: "Recurrences were experienced by year five in 18% of patients who were seizure free in the first year after surgery, by 6% of patients who remained seizure free for two years, and by 4% of patients who were seizure free for three years after surgery. Remission by year five was found in 36% of patients who had seizures in the first postoperative year, in 13% of patients who had seizures in each of the first two years after surgery, and in no patients who had seizures in each of the three years after surgery."

The researchers state: "The majority of seizure recurrences occur in the first two years after surgery, and outcome at two years reasonably predicts outcome at five years ... The overwhelming majority of recurrences occurred early after surgery; 93% (37 patients) of all first recurrent seizures happened within the first two years, and more than 55% occurred within the first 6 months."

The researchers suggest obtaining at least two years of follow-up, both for studies indicating outcome results and for those that determine preoperative predictors of results.

They write: "People with epilepsy have an increased risk of death compared with the general population. Our patients had a relatively high mortality rate, and all deaths in our series occurred in patients who had persistent seizures after surgery."

Of the four patient deaths during the study, three were sudden and unexplained—probably related to seizures, and one death resulted from suicide. The higher sudden unexplained death rates for people with epilepsy may be related to either acute cardiac or pulmonary derangements during seizures or interictal disturbances. Depression and suicide rates are also higher for this group, according to the researchers.

The authors report: "Five years after surgery, full-time employment levels substantially increased, unemployment decreased by more than 50%, and part-time employment also decreased. However, this benefit accrued mainly in those patients whose seizures had stopped, while individuals whose seizures persisted continued to have a high unemployment rate." But, while employment improved, general cognitive abilities did not.

The authors caution: "Whether the added risk and expense of a new therapy is worthwhile should be questioned if the result is simply a reduction in seizure frequency without measurable improvement in other domains."

The researchers conclude: "With careful application of patient selection criteria and an experienced surgeon, anterior temporal lobectomy is a safe and effective method for treating intractable temporal lobe epilepsy and its consequences. Patients should be encouraged to pursue this form of therapy as soon as the intractability of their condition becomes apparent."

Information cited in the study estimates the prevalence of active epilepsy at 0.6% among the general population. Despite medical therapy, between 25% and 60% of people with epilepsy continue to experience seizures.