

# Apgar Index as a Correlate of Enamel Defects of Primary Dentition

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**Purpose:** Enamel defects are serious challenges because of their unaesthetic appearance, dentinal sensitivity and the subsequent susceptibility to dental caries. Apgar index (AI) is used for postnatal, general, neural and behavioural assessment of newborns. The aim of the present study was to investigate the correlation of AI with the enamel defects of primary dentition.

**Materials and Methods:** A total of 181 subjects (95 females and 86 males) aged 3 to 5 years were randomly selected. Subjects with a history of systemic or debilitating diseases or local confounding factors, for example traumatic habits leading to enamel abrasion, were excluded. According to the modified index for developmental enamel defects, visual and tactile examinations of the entire primary dentition (buccal, lingual/palatal and incisal/occlusal) were performed. Teeth with caries lesions and restorations were excluded from the examination. After collection of the data regarding AIs of the subjects, statistical analysis was performed based on the Spearman and Mann-Whitney *U* tests.

**Results:** Enamel hypoplasia and hypocalcification were negatively correlated with the AI ( $P < 0.05$ ). Moreover, the number of teeth exhibiting enamel defects was significantly lower in subjects with higher AI ( $P < 0.05$ ). The effect of gender on the distribution of enamel defects as a covariate was not statistically significant ( $P > 0.05$ ).

**Conclusions:** AI is inversely correlated with enamel defects of primary dentition. Both quality and quantity of enamel defects of primary dentition are higher in children with lower AI, delineating a high-risk group demanding more stringent preventive measures.

**Key words:** Apgar index, hypocalcification, hypoplasia, primary dentition

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Enamel is formed during the second month of foetal development. The process of remodelling, which is ubiquitous to various hard tissues such as bone, does not occur in the enamel because of the loss of ameloblasts, and thus enamel defects cannot be repaired. Enamel defects may have different systemic and environmental aetiologies of which hypoxia,

hypocalcaemia, renal disorders, nutritional deficiencies and viral infections are the most commonly encountered causes (Pindborg, 1982). Weinmann et al (1945) for the first time categorised the different classes of enamel defects as hypoplasia and hypocalcification. Enamel hypoplasia, being a developmental disorder, is manifested as a lack of enamel in pits, fissures or other surfaces of the tooth. Hypocalcification is the result of defective mineralisation of enamel, and it is mainly a qualitative disorder.

Enamel defects are serious therapeutic challenges owing to aesthetic problems, dentinal hypersensitivity and vulnerability to dental caries. Considering these sequelae, the delivery of early preventive and therapeutic measures to the patient affected by this malformation is necessary to avoid further morbidity, and the resultant burden is imposed on the patient and the health systems. Moreover, the enamel

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**Table 1 The Apgar scoring system**

Sign	Score		
	1	2	3
Colour	Pale blue	Pink body, blue extremities	Completely pink
Reflex irritability	None	Grimace	Vigorous cry
Heart rate	Absent	Slow (< 100)	> 100
Respiratory effort	Absent	Slow (irregular)	Crying
Muscle tone	Flaccid	Some flexion of extremities	Active motion
<b>Score</b>	<b>Status</b>		
7–10	Normal		
4–6	Moderately depressed		
0–3	Severely depressed		

defects give some clues regarding more serious concomitant concealed health hazards. McDonald et al (1987) reported that neurological disorders accompany enamel defects in 70% of the subjects. Cohen and Diner (1970) found a similar association between enamel and neurological defects, and proposed a diagnostic value for enamel defects. Murray et al (1987) also demonstrated that children with neurological disorders exhibit enamel defects in primary dentition. Moreover, the association of enamel hypoplasia with rickets and respiratory distress in low-birthweight infants has been suggested (Johnson et al, 1984).

Apgar index (AI) is used for the evaluation of general, neural and behavioural status of newborns (American Academy of Pediatrics, 2006). The technique involves first to fifth minute assessment of skin colour, heartbeats, muscular tonicity, primary motor reflex and rate of respiration. While an AI value of 7 to 10 is considered normal, for values < 6 special medical measures are indicated. AI is one of the first indices used for early diagnosis of infant anomalies. The aim of the present retrospective study was to investigate whether AI is of any value in the prediction of enamel formation defects.

## MATERIALS AND METHODS

### Study population

The present descriptive retrospective study was performed at the Department of Pediatric Dentistry,

Tabriz University of Medical Sciences, Tabriz, Iran. A total of 181 subjects (95 females and 86 males) aged 3 to 5 years completed the study.

The subjects were selected through simple random sampling. Subjects with a history of systemic or debilitating diseases or local confounding factors, for example traumatic habits leading to enamel abrasion, were excluded. Teeth with prominent cracks, caries lesions and restorations were excluded from the study. Moreover, children with specific caries patterns, for example baby bottle caries lesion, were not included in the study. The study procedure was explained to the parents of the children and they signed a written consent form. This study was approved by the Ethical and Research Committees of Tabriz University of Medical Sciences.

### Study procedure

The study procedure was performed by a single paediatric clinician. A questionnaire was prepared that covered patient information, prenatal, perinatal and postnatal problems, AI, and enamel hypoplasia and hypocalcification. The data regarding the AIs of the subjects were registered by referring to the past medical documents of the patients.

Virginia Apgar, a physician and anaesthetist, developed the Apgar scoring system in 1952 (Apgar, 1953). The Apgar score is performed at 1 and 5 min of life. The Apgar scoring system is a comprehensive screening tool to evaluate a newborn's condition at birth (Table 1). Newborn infants are evaluated based on five variables: (1) heart rate, (2) respiratory effort, (3) muscle tone, (4) reflex irritability and (5) colour. A numerical score of 0 to 2 is assigned in each category for a maximum score of 10. Apgar scoring is best used in conjunction with additional evaluative techniques such as physical assessment and vital signs.

Intraoral examination of the subjects was performed under direct illumination. According to the modified index for developmental enamel defects, visual and tactile examinations of the entire primary dentition (buccal, lingual/palatal and incisal/occlusal) were performed. After drying the teeth, visual inspection and tactile examination using a sharp sterile dental explorer were performed. Enamel defects were categorised as hypoplasia or hypocalcification. Some subjects (45 females and 40 males) were examined twice on a random basis and also after completion of examination of all subjects to measure the intraexaminer agreement of data.

**Table 2 Apgar-based distribution of children affected by dental hypoplasia and hypocalcification**

Apgar value	Hypoplasia		Hypocalcification		Hypoplasia and hypocalcification		Mean
	No.	Per cent	No.	Per cent	No.	Per cent	
1	0	0	0	0	0	0	0
2	17	17.9	17	15.9	17	18.9	17
3	19	20	19	17.8	19	21.1	21
4	15	15.8	15	14	15	16.7	19
5	18	18.9	20	18.7	18	20	23
6	10	10.5	12	11.2	10	11.1	18
7	4	4.2	5	4.7	3	3.3	20
8	7	7.4	10	9.3	4	4.4	24
9	2	2.1	5	4.7	1	1.1	22
10	3	3.2	4	3.7	3	3.3	17
<b>Total</b>	95	100	107	100	90	100	181

### Statistical analysis

All quantitative data are presented as mean values. The intraexaminer agreement for the diagnosis of enamel hypoplasia and hypocalcification was evaluated by Spearman's correlation coefficient. The analysis of data was performed based on the Mann–Whitney *U* test. In the present study,  $P < 0.05$  was considered to indicate statistical significance.

### RESULTS

A total of 181 subjects (95 females and 86 males) aged 3 to 5 years were included in this study.

#### Intraexaminer agreement of data

The intraexaminer agreement for the diagnosis of enamel hypoplasia ( $r = 0.91$ ) and hypocalcification ( $r = 0.84$ ) was excellent.

In parallel with decreased AI, the number of children exhibiting enamel hypoplasia or hypocalcification increased ( $r(\text{hypoplasia}) = -0.78$ ,  $r(\text{hypocalcification}) = -0.82$ ,  $P < 0.05$ ) (Table 2). The same trend was observed for subjects with combined hypoplasia and hypocalcification ( $r = -0.65$ ,  $P < 0.05$ ). Moreover, in children with enamel defects, the number of affected teeth increased in parallel with the reduction of AI ( $r(\text{hypoplasia}) = -0.86$ ,  $r(\text{hypocalcification}) = -0.81$ ,  $P < 0.05$ ) (Table 3). Below the critical Apgar value of 6, an abrupt surge in both

the number of affected patients and the number of affected teeth was observed. The number of hypoplastic teeth was less than the number of teeth with hypocalcification. However, the difference was not statistically significant ( $P > 0.05$ ). Fewer children demonstrate combined hypoplasia and hypocalcification than those exhibiting one type of enamel defect; but the difference did not reach statistical significance ( $P > 0.05$ ). The distribution of enamel defects in primary dentition was as follows: A (47.8%), B (47.6%), C (51.3%), D (46.6%) and E (36.3%).

Gender-specific distribution of enamel defects was evaluated. Strong negative correlation of Apgar value and enamel defects were observed. Furthermore, there was no gender-dependent statistical difference regarding the prevalence of enamel hypoplasia and hypocalcification between subjects.

### DISCUSSION

The aim of the present retrospective study was to investigate whether or not AI is of any significance in the prediction of enamel-formation defects. Our findings indicated a strong negative correlation between the Apgar value and the existence of enamel hypoplasia and hypocalcification.

Various aetiological factors have been implicated in the development of enamel defects. The aetiological factors involved in the enamel defects can be divided into two: systemic and local (Pindborg, 1982). The systemic factors may be categorised

**Table 3 Apgar-based distribution of hypoplastic and hypocalcified teeth**

Apgar value	Hypoplasia		Hypocalcification		Mean
	No.	Per cent	No.	Per cent	
1	0	0	0	0	0
2	204	31.3	229	31.6	253
3	181	27.8	194	26.8	271
4	121	18.6	133	18.3	291
5	79	12.1	87	12	304
6	27	4.1	31	4.3	289
7	13	2	15	2.1	321
8	11	1.7	16	2.2	329
9	7	1.1	9	1.2	336
10	9	1.4	11	1.5	292
<b>Total</b>	652	100	725	100	2686

as genetically determined, chromosomal anomalies, congenital defects, inborn errors of metabolism, neonatal disturbances, infectious diseases, neurological disturbances, endocrinopathies, nutritional deficiencies, nephropathies, enteropathies, liver diseases and intoxications. The genetically determined enamel defects include amelogenesis imperfecta that may occur as an isolated phenomenon or as a part of other disorders such as epidermolysis bullosa, pseudohypoparathyroidism and taurodontism. The congenital defects include heart disorders and unilateral facial hypoplasia and hypertrophy. Among the inborn errors of metabolism are galactosaemia, phenylketonuria, alkaptonuria, erythropoietic porphyria and primary hyperoxaluria. Neonatal disturbances are important in the development of enamel hypoplasia, and the foremost among these are premature birth and hypocalcaemia.

The goal of Apgar scoring system was to make certain that infants were systematically observed for their need for immediate care at birth. Although prediction of long-term outcomes was never a goal of the Apgar scoring system, research conducted over 40 years ago (Apgar and James, 1962; Apgar, 1966) provided initial evidence to disclaim the reliability of Apgar scores for predicting long-term outcomes of any type (e.g. developmental and neurological). Blackmann (1988) suggested that low Apgar scores warrant developmental surveillance during the early years of life but, if unaccompanied by neonatal seizures, they do not appear to predict more subtle developmental dysfunction evident at the age of school entry. However, Behnke et al (1989) proposed that after controlling for birth weight and gestational

age, the Apgar scores did not predict morbidity in low-birthweight infants and hence should not be used to provide a developmental prognosis. The findings of Hegyi et al (1998) may partially explain the underlying cause of this relative disagreement. They found that among the components of the Apgar score, respiratory effort, muscle tone and reflex activity correlated well with one another; heart rate correlated less well; and colour the least, leading to different responses of AI components.

In recent years, many researchers have attempted to correlate Apgar scores with various outcomes including development (Blackmann, 1988; Behnke et al, 1989; Riehn et al, 1998), later delinquency (Gibson and Tibbetts, 1998), intelligence (Nelson and Ellenberg, 1981) and neurological development (Sommerfelt et al, 1996; Wolf et al, 1997a, b, 1998) for the purposes of research.

The present retrospective study demonstrated that decreased AI is strongly correlated with the development of enamel defects. Subjects with lower Apgar values exhibited more hypoplastic and hypocalcified teeth. However, below the critical Apgar value of 6 there was a sudden surge in the number of affected subjects. Thus, it may be proposed to carry out more intense and stringent oral screening for the subjects with Apgar values < 6. Gender does not influence the final outcome.

Future research may be directed towards the investigation of correlation of Apgar scoring system components with developmental enamel defects. Moreover, the study of association of developmental, craniofacial and musculoskeletal anomalies with AI seems to be interesting.

It may be concluded that the Apgar value can be reliably used for the risk assessment of further development of enamel defects of primary dentition. Moreover, there is a critical Apgar value below which the vulnerability to the development of enamel defects is substantially increased, and the implementation of specific preventive measures may prove to be highly beneficial.

## REFERENCES

1. American Academy of Pediatrics. The Apgar score. *Obstet Pract Pediatr* 2006;117:1444–1447.
2. Apgar V. A proposal for a new method of evaluation of the newborn infant. *Curr Res Anesth Analg* 1953;32:260–267.
3. Apgar V. The newborn scoring system: reflections and advice. *Pediatr Clin North Am* 1966;113:645–650.
4. Apgar V, James L. Further observation of the newborn scoring system. *Am J Dis Child* 1962;104:419–428.
5. Behnke M, Eyler FD, Carter RL, Hardt NS, Cruz AC, Resnick MB. Predictive value of Apgar scores for developmental outcome in premature infants. *Am J Perinatol* 1989;6(1):18–21.
6. Blackmann JA. The value of Apgar scores in predicting developmental outcome at age five. *J Perinatol* 1988; 8(3):206–210.
7. Cohen HJ, Diner H. The significance of developmental dental enamel defects in neurological diagnosis. *Pediatrics* 1970;46(5):737–747.
8. Gibson C, Tibbetts S. Interaction between maternal cigarette smoking and Apgar scores in predicting offending behavior. *Psychol Rep* 1998;83:579–586.
9. Hegyi T, Carbone T, Anwar M, Ostfeld B, Hiatt M, Koons A et al. The apgar score and its components in the preterm infant. *Pediatrics* 1998;101.
10. Johnson D, Krejci C, Hack M. Distribution of enamel defects and the association with respiratory distress in very low birth weight infants. *J Dent Res* 1984;3:59–64.
11. McDonald RE, Avery DR, Lynch TR. Management of trauma to the teeth and supporting tissues. In: *Dentistry for the Child and Adolescent*, ed 5. St. Louis: Mosby, 1987.
12. Murray GS, Johnsen DC, Weissman BW. Hearing and neurologic impairment: insult timing indicated by primary tooth enamel defects. *Ear Hear* 1987;8:68–73.
13. Nelson K, Ellenberg J. Apgar scores as predictors of chronic neurologic disability. *Pediatrics* 1981;68:36–44.
14. Pindborg JJ. Aetiology of developmental enamel defects not related to fluorosis. *Int Dent J* 1982;32(2):123–134.
15. Riehn A, Petzold C, Kuhlisch E, Distler W. Fetal acidemia and neonatal encephalopathy. *Zeitschrift für Geburtshilfe und Neonatologie* 1998;202:187–191.
16. Sommerfelt K, Pedersen S, Ellertsen B, Markestad T. Transient dystonia in non-handicapped low-birthweight infants and later neurodevelopment. *Acta Paediatr* 1996;85:1445–1449.
17. Weinmann JP, Svoboda JF, Woods RW. Hereditary disturbances of enamel formation and calcification. *J Am Dent Assoc* 1945;32:397–418.
18. Wolf M, Beunen G, Casaer P, Wolf B. Neurological findings in neonates with low Apgar in Zimbabwe. *Eur J Obstet Gynecol Reprod Biol* 1997a;73:115–119.
19. Wolf M, Beunen G, Casaer P, Wolf B. Neonatal neurological examination as a predictor of neuromotor outcome at 4 months in term low-Apgar-score babies in Zimbabwe. *Early Hum Dev* 1998;51:179–186.
20. Wolf M, Wolf B, Bijleveld C, Beunen G, Casaer P. Neurodevelopmental outcome in babies with a low Apgar score from Zimbabwe. *Dev Med Child Neurol* 1997b;39:821–826.