

Enamel defects in permanent incisors after trauma to primary predecessors: inter-observer agreement based on photographs

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Abstract – Trauma to primary teeth may cause mineralization disturbances in the permanent successors. *Objective:* To study the distribution and type of enamel defects in permanent incisors after trauma to primary teeth and to examine inter-observer agreement when registrations were based on photographs. *Material and methods:* Of 266 children who suffered an oral injury to primary teeth in one county of Norway in 2003, 193 were included in a follow-up study to record enamel defects in the permanent successors 7 years later (age, 8–15 years). Clinical examination and intraoral photographs were undertaken by the principal investigator. The photographs were evaluated twice for enamel defects by three paediatric dentists. Evaluation 1: age at the time of injury, traumatized teeth and diagnoses were kept unknown to the examiners. Evaluation 2: age and trauma diagnoses were known to the examiners. Inter-observer agreement was calculated using Cohen's kappa and chi-square test. *Results:* Of 338 successor teeth, 42% exhibited enamel defects. In neighbouring teeth (339) with non-injured predecessors, 30% were registered with defects. The most common enamel disturbance in successors was demarcated opacities, recorded in 18% of the teeth. Enamel defects owing to a previous trauma were registered in 37% of the children in Evaluation 1, kappa 0.88–0.93 and in 21% in Evaluation 2, kappa 0.63–0.84. The examiners disagreed on a higher proportion of the children when all information on the injury was available ($P < 0.001$). *Conclusion:* Demarcated enamel opacities were the most common defects in permanent successors. Although the inter-examiner agreement was good, the results indicated that recordings of enamel disturbances following trauma is associated with uncertainty.

It is well known that injuries to primary teeth may cause mineralization disturbances in the permanent dentition. The more serious the trauma, the higher is the probability (1–7). Intrusion and avulsion injuries are most often reported to cause developmental disturbances, in 40–70% and 30–50% of the cases, respectively (1,3–5,8,9). According to the literature, less severe trauma, like subluxations, may also be the cause of sequelae in permanent teeth. Da Silva Assunção et al. (5) reported a frequency of 10%, von Arx (3) 19%, whereas Andreassen and Ravn (1) recorded that 34% (12 of 35 teeth) had a defect in the permanent successor after a subluxation injury.

The most common developmental disturbances are white and yellow-brown enamel discolouration, regardless of the severity of the injury or the age of the child (1,5,6).

Enamel defects may also be consequences of other conditions. Molar incisor hypomineralization (MIH), a

mineralization disturbance that often involves incisors, has a reported prevalence from 2.4% to 40.2% (10). In the 2003 Children's Dental Health Survey in UK, the prevalence of enamel opacities was 34% in 12-year-old children (11). In that survey, the most prevalent defects were demarcated and diffuse opacities, whereas hypoplasia affected only a few cases. Caries in primary teeth may also increase the risk of defects in permanent successors (12). In an early study by Andreassen & Ravn (13) comparing two groups of children, one with a trauma history and one without, the authors concluded that only 10% of the enamel disturbances in anterior teeth were owing to trauma.

The large range in reported frequencies of enamel defects, either as a result of tooth injury or due to other insults, may depend on diagnostic criteria or on the study design, the number of examiners, and/or the calibration procedures. Although there are many studies regarding the effect of dental injuries in primary

teeth on the permanent successors, several studies lack information on the number of examiners or the inter-examiner agreement (2,4,7,8). There may be either one (3,5) or several examiners (6,14), but in only one study (13) is the possible error of registrations or disagreement between examiners described.

The aim of this study was (i) to examine the distribution and type of enamel defects in permanent incisors after trauma to primary teeth and (ii) to examine the inter-observer agreement on whether the defects evaluated on photographs most likely were due to a trauma.

Material and methods

During 2003, all trauma episodes affecting primary teeth were recorded in the Public Dental Service (PDS) in one county of Norway. A total of 266 children (age 1–8 years) suffered an oral injury during the registration year, and the mean age was 3.8 years (15).

In 2010, parents of all the children with injured primary teeth in 2003 were invited to participate in a follow-up study. Written, informed consent was obtained from all participants, and approval and ethical permission were obtained from the Regional Committee for Medical Research Ethics and the Norwegian Social Science Data Services. A total of 63 children had either moved out of the county, did not want to participate or did not attend for the scheduled appointment. Ten children with unreadable surfaces owing to orthodontic treatment were excluded from analyses. Of the remaining 193 children (age, 8–15 years) with 373 injured primary incisors, 35 immediate successors and 71 neighbouring teeth were unerupted, leaving 338 successors and 339 neighbouring teeth to be evaluated.

An intra-oral examination was undertaken in the public clinics. The teeth were dried by compressed air and, if necessary, cotton rolls were used to remove debris. Standard lighting, mouth mirrors and probes were used. The child sat upright in the dental chair, and a cheek retractor was inserted. A Cannon EOS 30D digital camera with ring flash was used to take the frontal view and close-up photographs by the principal investigator. First permanent molars were always examined but only photographed when MIH was diagnosed or suspected owing to appearance of front teeth. The children's age at the time of injury and the trauma diagnosis were collected from the dental records.

Three paediatric dentists (ABS, ALMA, NJW) were calibrated by examining pictures of enamel defects. Based on a subsample of photographs, the examiners discussed the criteria based on a modified Developmental Defects of Enamel (DDE) index (16). The scores used were from 0 to 9 on labial surfaces as follows: 0 = sound, 1 = demarcated opacities ≤ 3 mm, 2 = demarcated opacities >3 mm, 3 = diffuse opacities ≤ 3 mm, 4 = diffuse opacities >3 mm, 5 = hypoplasia ≤ 3 mm, 6 = hypoplasia >3 mm, 7 = combination of opacity and hypoplasia, 8 = crown dilaceration and 9 = unreadable (unerupted, restoration). Mineralization disturbances <0.5 mm were regarded as sound. All evaluations were

performed in the same room under identical lighting conditions.

Evaluations were performed at 2-week intervals. In Evaluation 1, the photographs were presented to the examiners without information on the age of child at the time of injury, nor trauma diagnosis. Each examiner independently scored the labial tooth surface of the upper and lower incisors using the modified DDE index. The final score for each surface was achieved by majority and formed the basis for the judgement of whether the defect could be due to trauma in the primary dentition.

In Evaluation 2, age and diagnoses at the time of injury, one or more trauma episodes, and whether the presence of MIH or not were known. The examiners were asked, independently of each other, to judge whether the child had an enamel defect most likely due to trauma (yes/no). Neighbouring permanent teeth (successors to uninjured primary teeth) were also taken into account. The tooth surfaces were not given a DDE score as performed in Evaluation 1. Enamel defects owing to trauma were reported when all three examiners agreed.

Data were analysed by cross tabulations and tested by chi-square using the Statistical Package for the Social Sciences (version 16; SPSS, Inc., Chicago, IL, USA). Cohen's unweighted kappa and proportional agreement were used to calculate inter-observer agreement. Cohen's kappa was rated as suggested by Landis and Koch (17). The level of agreement was set at $P < 0.05$.

Results

Table 1 shows the distribution and type of enamel defect in successors and in neighbouring teeth with non-injured predecessors. Enamel defects were registered in 41.7% (141/338) of the successors and in 29.5% (100/339) of neighbouring teeth with non-injured primary predecessors. In total, 35.6% of the examined incisors had a defect.

The most common enamel disturbances in successors were demarcated opacities, recorded in 17.8% of the teeth, followed by diffuse opacities in 13.9%, hypoplasia in 7.1% and combinations of opacity and hypoplasia in 3% of the teeth.

In neighbouring teeth of non-injured predecessors, diffuse opacities were most common, recorded in 15.3% of the teeth. Demarcated opacities were registered in 8.3%, hypoplasia in 5.3% and a combination of opacity and hypoplasia in $<1\%$ of neighbouring tooth defects.

As the majority of the injuries affected the maxilla, inter-examiner agreement was calculated for the upper incisors ($n = 606$). The examiners judged whether the enamel defects most likely were due to a previous trauma or not (age and primary trauma diagnoses known), and the values of Cohen's kappa were in the range 0.60–0.83 and proportional agreement 92–96%.

Table 2 presents the number and proportions of children with enamel defects. One-fourth had enamel defects owing to trauma when injured between 2.5 and

Table 1. Distribution and types of enamel defect in successors ($n = 338$) and in neighbouring teeth with non-injured predecessors ($n = 339$)

	Enamel defects in successors		Enamel defects in neighbouring teeth	
	<i>n</i>	%	<i>n</i>	%
Total demarcated opacities	60	17.8	28	8.3
≤ 3 mm	47		25	
>3 mm	13		3	
Total diffuse opacities	47	13.9	52	15.3
≤ 3 mm	11		12	
>3 mm	36		40	
Total hypoplasia	24	7.1	18	5.3
≤ 3 mm	23		16	
>3 mm	1		2	
Combination	10	3.0	2	0.6
All enamel defects	141	41.7	100	29.5
No enamel defect	197	58.3	239	70.5
Total	338	100	339	100

Table 2. The numbers and proportions of children ($n = 193$) categorized according to enamel appearance in permanent incisors after trauma to the primary dentition

	Unknown age and trauma diagnosis Evaluation 1		Known age and trauma diagnosis Evaluation 2	
	<i>n</i>	%	<i>n</i>	%
No enamel defect	72	37	88	46
Enamel defect owing to trauma	72	37	41	21
Other mineralization disturbance	30	16	32	16.5
Disagreement	19	10	32	16.5
Total	193	100	193	100

3.5 years and none after 6 years of age. When the only information given was a previous trauma to the primary dentition, 37% of the children were estimated by all examiners to have at least one enamel defect most likely due to trauma. There was disagreement between the examiners in 10% of the children, and other causes were given in 16% of the children. Kappa was in the range 0.88–0.93.

The examiners disagreed on a higher proportion of the children, 16.5%, when all information on the injury was available. Fewer children, 21% vs 37%, were considered to have enamel defects most likely due to a trauma when more information was known ($P < 0.001$), (Table 2). The inter-examiner agreement was lower than in Evaluation 1 with kappa values in the range 0.63–0.84.

In 29% of the individuals, enamel defects were recorded on a neighbouring tooth where the primary predecessor had not been injured; an example is shown in Fig 1. Figures 1–3 show examples of cases where the examiners disagreed on the cause of enamel defects.



Fig. 1. The figure shows a lateral incisor with a demarcated opacity and two central incisors with normal enamel. Trauma at 3 years of age. Diagnoses: 52 no previous injury, 51 subluxation, 61 intrusive luxation.



Fig. 2. The figure shows 11 and 21 with demarcated opacities. Trauma at 3 years of age. Diagnoses: 51 subluxation, 61 concussion. Tooth 61 had a transient (bleeding) discoloration. The 6-year molars were unaffected. No anamnestic information on illness in early years or caries that could explain the enamel defects.



Fig. 3. The figure shows 11 with a hypoplastic defect and 21 with a demarcated opacity. Trauma at 5 year of age. Diagnoses: 51 and 61 subluxation. The 6-year molars were unaffected. No history of other possible aetiological factors.

Discussion

In this study, enamel defects were registered in nearly half of permanent incisors after trauma to primary

predecessors and in nearly one-third of the neighbouring teeth. The most common enamel disturbances to the immediate predecessor were demarcated opacities, followed by diffuse opacities and hypoplasia. These results are in accordance with previous findings (1,5,6) and consistent with results in a survey from UK reporting that demarcated and diffuse opacities were the most prevalent defects regardless of a previous trauma (12).

There is a wide range in reported prevalence of mineralization defects in permanent successors after luxation injuries to primary teeth. According to the literature, the prevalence of these disturbances varies from 10% to 70% (1,3,4,8,9). This wide range may be due to the severity of trauma and the age of the child as well as material and method (14). The examination may be performed in paediatric specialist clinics (6–8) or in general practice/public dental health service (9) or there may be differences owing to techniques like lighting and magnification. As evaluations are mostly performed by one examiner, the prevalences may vary even more. Evaluations are subjective assessments, and more examiners may possibly prevent an extreme recording. Based on the present results with disagreement on the cause of defect in 16.5% of the children and on results by Andreasen and Ravn (13) who reported an error of registration to be 9%, the frequencies of enamel defects may vary from almost zero to 80–90%. In the aforementioned study (13), the authors concluded that only about 10% of the enamel defects were owing to trauma.

In the present study, more enamel defects were judged to be consequences of previous trauma when there was lack of information on the injury. In a recent publication (18), the authors point out the importance of preinjury factors like age of patient, tooth development stage, trauma type and severity when registering consequences. The age of the patient and diagnoses were known to the examiners only in the second evaluation in our study. When this information was available, the inter-examiner agreement was lower regarding whether or not enamel defects were owing to previous trauma. This may be due to the fact that enamel defects, idiopathic or owing to trauma, may have the same appearance. When age and diagnoses were known, fewer defects were judged as caused by trauma.

This investigation was designed as a prospective study and had the advantage of collecting data on the trauma when the injury occurred. Many clinicians were involved in diagnosing and recording trauma, and the retrospective data collection at the follow-up examination revealed some examples of deficient dental records. This may have influenced the registration of trauma diagnoses and, subsequently, the frequencies of developmental disturbances related to a specific diagnosis. Despite this limitation, there are, as far as we know, no studies reporting reliability testing of examiners who assess enamel disturbances in permanent teeth caused by trauma to the primary dentition.

Recording developmental defects by means of photographs has been assessed by other researchers (19–21) who conclude that a photographic method is reliable

for the evaluation of enamel defects and is as sensitive as a clinical examination (21). It may be a challenge to assess the cause of a developmental disturbance, in particular when there are mild periodontal injuries where frequencies of enamel defects are low (1,3,5,6). In the previous study by Andreasen and Ravn (13), where two groups of schoolchildren were compared, the examinations were carried out by two observers and repeated in 100 children. The authors registered a difference in the recordings, and the error of the registration method was found to be 9%. This variation is seldom taken into account when frequency and type of tooth sequelae are reported. As the present study showed, there was obvious uncertainty among examiners about whether trauma was the most probable cause of an enamel defect. Figures 1–3 show examples of such uncertainty.

The conclusion is that demarcated opacities are the most common enamel defect in permanent successors after trauma to the primary dentition. Although the inter-examiner agreement was good, the results show that recordings of enamel disturbances following trauma were associated with uncertainty.

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References

1. Andreasen JO, Ravn JJ. The effect of traumatic injuries to primary teeth on their permanent successors. II. A clinical and radiographic follow-up study of 213 teeth. *Scand J Dent Res* 1971;79:284–94.
2. Brin I, Fuks A, Ben-Bassat Y, Zilberman Y. Trauma to the primary incisors and its effect on the permanent successors. *Pediatr Dent* 1984;6:78–82.
3. von Arx T. Developmental disturbances of permanent teeth following trauma to the primary dentition. *Aust Dent J* 1993;38:1–10.
4. Sennhenn-Kirchner S, Jacobs HG. Traumatic injuries to the primary dentition and effects on the permanent successors – a clinical follow-up study. *Dent Traumatol* 2006;22:237–41.
5. Da Silva Assunção LR, Ferelle A, Iwakura ML, Cunha RF. Effects on permanent teeth after luxation injuries to the primary predecessors: a study in children assisted at an emergency service. *Dent Traumatol* 2009;25:165–70.
6. do Espírito Santo Jácomo DR, Campos V. Prevalence of sequelae in the permanent anterior teeth after trauma in their predecessors: a longitudinal study of 8 years. *Dent Traumatol* 2009;25:300–4.
7. de Amorim Lde F, Estrela C, da Costa LR. Effects of traumatic dental injuries to primary teeth on permanent teeth – a clinical follow-up study. *Dent Traumatol* 2011; 27:117–21.
8. Altun C, Cehreli ZC, Güven G, Acikel C. Traumatic intrusion of primary teeth and its effects on the permanent successors: a clinical follow-up study. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2009;107:493–8.
9. Christophersen P, Freund M, Harild L. Avulsion of primary teeth and sequelae on the permanent successors. *Dent Traumatol* 2005;21:320–3.

10. Jälevik B. Prevalence and Diagnosis of Molar-Incisor- Hypomineralisation (MIH): a systematic review. *Eur Arch Paediatr Dent* 2010;11:59–64. Review.
11. Chadwick BL, White DA, Morris AJ, Evans D, Pitts NB. Non-carious tooth conditions in children in the UK, 2003. *Br Dent J* 2006;200:379–84.
12. Broadbent JM, Thomson WM, Williams SM. Does caries in primary teeth predict enamel defects in permanent teeth? A longitudinal study. *J Dent Res* 2005;84:260–4.
13. Andreasen JO, Ravn JJ. Enamel changes in permanent teeth after trauma to their primary predecessors. *Scand J Dent Res* 1973;81:203–9.
14. Rasmusson CG, Koch G. Assessment of traumatic injuries to primary teeth in general practise and specialized paediatric dentistry. *Dent Traumatol* 2010;26:129–32.
15. Skaare AB, Jacobsen I. Primary tooth injuries in Norwegian children (1-8 years). *Dent Traumatol* 2005;21:315–9.
16. Clarkson J, O'Mullane D. A modified DDE Index for use in epidemiological studies of enamel defects. *J Dent Res* 1989;68:445–50.
17. Landis JR, Koch GG. The measurement of observer agreement for categorical data. *Biometrics* 1977;33:159–74.
18. Andersson L, Andreasen JO. Important considerations for designing and reporting epidemiologic and clinical studies in dental traumatology. *Dent Traumatol* 2011;27:275–80.
19. Cochran JA, Ketley CE, Sanches L, Mamai-Homata E, Oila AM, Arnadóttir IB et al. A standardized photographic method for evaluating enamel opacities including fluorosis. *Community Dent Oral Epidemiol* 2004;32(Suppl 1):19–27.
20. Wong HM, McGrath C, Lo EC, King NM. Photographs as a means of assessing developmental defects of enamel. *Community Dent Oral Epidemiol* 2005;33:438–46.
21. Golkari A, Sabokseir A, Pakshir HR, Dean MC, Sheiham A, Watt RG. A comparison of photographic, replication and direct clinical examination methods for detecting developmental defects of enamel. *BMC Oral Health* 2011;11:16.

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