



Clinical Solutions for Developmental Defects of Enamel and Dentin in Children

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Abstract: Developmental defects of enamel (DDE) are frequently observed in pediatric dental patients. Proper diagnosis may improve the clinician's dental care. The purpose of this article is to present the clinical management of some common dental defects: (1) hypoplasia; (2) diffuse and demarcated opacities; (3) fluorosis; (4) amelogenesis imperfecta (AI); and (5) dentinogenesis imperfecta (DI). The comprehensive management of DDE in children and adolescents should include: (1) active follow-up and observation involving oral hygiene instructions; and (2) dietary consultation. Preventive care should be individually tailored according to the patient's risk-assessment analysis. The treatment of DDE involves an approach that includes several disciplines, including: (1) pediatric dentistry; (2) orthodontics; (3) perioprosthodontics; and (4) psychology. A close follow-up is essential to achieve long-term success. (*Pediatr Dent* 2007;29:330-6)

KEYWORDS: MIH, DDE, ENAMEL OPACITIES, ENAMEL HYPOMINERALIZATION

Developmental defects of enamel (DDE) consist mainly of hypoplasia and of diffuse and demarcated opacities.¹ Fluorosis, amelogenesis imperfecta (AI), and even dentinogenesis imperfecta (DI) may be considered forms of DDE. Weerheijm et al defined the term "molar incisor hypomineralization" (MIH) to describe a more specific pattern of DDE: hypomineralization of systemic origin of 1 to 4 permanent first molars frequently associated with affected incisors.²

Clinically, enamel opacities of MIH can be seen as an abnormality in the translucency of the enamel. Some lesions have significant subsurface porosity, leading to posteruptive breakdown of the surface. This is observed more frequently in permanent first molars than in incisors mainly because molars are subjected to masticatory forces and are generally more severely hypomineralized than the incisors.³

When a severe defect is found in a tooth, it is likely that the contralateral tooth is also affected. Frequently, a combination of enamel defects may be recognized in the same child (hypoplasia, diffuse, and demarcated opacities). The lesions in the permanent first molars are often seen together with those in the maxillary and, more

rarely, in the mandibular incisors.³ When more molars are affected, the relative risk of opacities in the incisors to show opacities is increased.³⁻⁶

Opacities are usually limited to the crown's incisal or cuspal third, more commonly on the facial surfaces. The enamel surface is often smooth and hypermineralized following posteruptive maturation; the subsurface enamel is soft and porous.⁷

Early diagnosis of the type of DDE (eg, MIH/fluorosis/AI/DI) is important for appropriate treatment planning and to prevent future complications. A correct diagnosis may improve the clinician's care in several aspects, such as:

1. assessing the patient's caries risk; and
2. evaluating the quality of the adhesion that affects the retention and durability of restorations and orthodontic brackets.

Furthermore, individual and community perspectives of prevention (dental trauma, enamel fluorosis, genetic counseling, financial considerations, behavioral management, and medicolegal issues) can be affected by identification of the DDE etiology.⁸⁻¹⁸ Medical and dental history and clinical and radiographic findings may contribute to differential diagnosis.¹⁹⁻²⁴ The DDE/MIH patient's chief complaint is often one or more of the following: (1) poor esthetics; (2) thermal sensitivity; (3) attrition; (4) secondary caries; (5) tooth discoloration; (6) malocclusion; and (7) periodontal problems.^{2,25} The patient's complaint, in conjunction with the defects' biochemical and morphohistological characteristics, may affect the prognosis and management.^{7,26,27}

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This article's purpose was to provide recommendations for the clinical management of developmental defects of enamel with emphasis on the relevant modifications necessary in cases of fluorosis, amelogenesis imperfecta, and dentinogenesis imperfecta, as compared to other types of enamel opacities (ie, molar incisor hypomineralization). A few cases restored with direct and indirect intermediate acrylic/polycarbonate crowns are also presented. This article comprises 3 parts:

1. a concise review of the preventive treatment which may be appropriate in most cases of DDE;
2. adhesive system choice and technique used for bonding resin restorations to DDE; and
3. the management of anterior and posterior teeth diagnosed with DDE.

Preventive treatment

The preventive treatment following the diagnosis of DDE should be tailored for the individual patient, considering factors such as: (1) risk for dental caries; (2) posteruption breakdown; (3) the presence of symptoms; (4) DDE etiology and severity; and (5) the extent of the defects. The extent of the problem depends on the number of teeth involved and the severity of the lesions (depth, size, color, and enamel breakdown).

William et al have presented a table suggesting different approach for clinical management of MIH that may be used as a guideline in most cases.²⁵ Obviously, mild cases of DDE (eg, diffuse opacities, mild fluorosis), will not demand all measures for prevention.

The cariogenicity and erosivity of the child's diet should be assessed and appropriate recommendations for dietary modification should be provided. Oral hygiene instructions may include proper toothbrushes and desensitizing toothpaste if necessary.²⁸ Weekly topical fluoride gel or varnish applications, and daily sodium fluoride rinses may: (1) improve the resistance to demineralization; (2) decrease tooth sensitivity; and (3) enhance enamel remineralization and post eruptive maturation.²⁹ Daily application of casein phosphopeptide-amorphous calcium phosphate (CPP-ACP) in oral care products is reported to promote remineralization.³⁰⁻³¹ Surface hardening and reduced enamel demineralization and tooth sensitivity as well as esthetic improvement of opacities may occur.²⁵⁻³²

When an erupting tooth is diagnosed with hypomineralized yellow-brown enamel, monthly follow-up visits should be scheduled for inspecting enamel surface integrity and for application of a 5% sodium fluoride varnish (Duraphat, 2.26% fluoride, Colgate-Palmolive, NY, USA or on of the new white varnishes, such as Vanish (OMNII, 2.26% fluoride, Oral Pharmaceuticals, 3M ESPE, USA). The use of glass ionomer cement sealants until the hypoplastic tooth reaches occlusion, to be followed by the application of a conventional resin-based sealant, may also be considered.²⁵ Mechanical prepara-

tion of the enamel prior to sealant application is not recommended, unless enamel integrity is already compromised.

Adhesive system choice and technique used for bonding resin restorations to DDE

The type of adhesive system chosen may determine the clinical outcome of the resin restoration adhesion to hypomineralized enamel. The shear bond strength of resin composite bonded to hypomineralized enamel is significantly lower than bonding to normal enamel.³³ Phosphoric acid, most commonly used for enamel etching, may cause more enamel loss than self-etching primers,³⁴ reducing the adhesion to hypomineralized enamel.³³ Self-etching adhesives may offer an alternative that better meets the challenge of adhesion to hypomineralized enamel for several reasons:

1. They are simpler to use, hydrophilic, and require less time and fewer steps.³⁵⁻³⁶
2. Rinsing is omitted, so wet conditions that inhibit resin infiltration and dilute the water-soluble primer, are prevented.
3. Some self-etching primers (Clearfil SE Bond/Protect bond, Kuraray Medical Inc, Tokyo, Japan) bond both micromechanically and chemically to hydroxyapatite.
4. Some self-etching primers (Clearfil SE Bond/Protect bond, Kuraray Medical Inc) have fluoride releasing properties as well as an antibacterial component.
5. They cause less postoperative sensitivity, which may be important in severely hypomineralized teeth.³⁸

The management of fluorosis and AI, however, may involve other modifications to the bonding technique and will be discussed later.

The management of anterior and posterior teeth diagnosed with DDE

The treatment of anterior and posterior teeth with DDE and MIH may not be alike differ, due to different esthetic and mechanical demands. Moreover, the treatment of fluorosed teeth, AI, and DI may necessitate modifications from the treatment recommended for MIH.

The treatment of MIH may depend on the lesion's severity. A definite correlation between color and histological porosity, mineral content, and depth has been established in MIH. Yellow-brown defects tend to be deeper, extending from the dentoenamel junction to the enamel surface, whereas white-creamy defects are usually less porous and variable in depth, typically limited to the inner enamel. In most DDE types and in MIH, the cervical and the most superficial enamel layers are usually more mineralized.⁷

The treatment of fluorosed enamel is also dependent on the severity. Thylstrup and Fejerskov's index (TF index)³⁸ may be used to divide treatment modalities for fluorosed teeth. This index is divided into 9 scores among several levels.

A TF index of:

- a. 1 to 2 is the result of increased porosity along the striae of Retzius, which are clinically seen as emphasized perikymata.
- b. 3 to 4 indicates subsurface porosity, while the enamel rods' shapes are still within the normal range; clinically it is visible as a chalky hypomineralization of enamel.
- c. 5 to 6 is clinically visible as areas of "punched-out" enamel opacities.
- d. >7, the porous enamel surface is exposed due to different degrees of enamel breakdown.
- e. 9, the most severe, exhibits enamel loss with a change in the anatomical tooth shape.

Treatment of mild diffuse opacities and fluorosis (TF index of 1-2)

Usually follow-up is sufficient, since physiologic attrition may improve the appearance.

Treatment of minor lesions (diffuse opacities, white-creamy demarcated opacities) and mild fluorosis (TF index of 3-4)

The clinician may consider enamel microabrasion.³⁹⁻⁴⁰ This technique, however, may cause aggressive reduction of enamel as a function of duration, number, and intensity of applications.⁴¹⁻⁴² Combining treatment with external bleaching may improve the esthetic result and conserve tooth substance.³⁹ The application of CPP-ACP may enhance enamel remineralization, improve esthetic results, and diminish tooth sensitivity after tooth whitening and enamel microabrasion.⁴³⁻⁴⁴

Treatment of moderate cases (creamy-yellow opacities, moderate fluorosis; TF index of 5-6) and hypoplastic AI
Enamel microabrasion may be considered.⁴⁵ Often, in case of a deep, resistant stain, a supplemental composite restoration must follow.⁴⁶ In some of these cases, composite opaquers may be necessary to mask the stained enamel.

Mild and moderate fluorosis does not adversely affect the adhesion of resin composite to fluorosed enamel. Doubling the time for etching may improve adhesion. The adhesion to young fluorosed enamel is better than in adults older than 40. Self-etching primers are not recommended in moderate and severe fluorosis, as they provide a lower shear bond strength.⁴⁸

Compomers are more retentive than glass ionomer cements (GICs)/resin-modified GICs (RMGICs) when restoring fluorosed enamel and may be used for restoration of small occlusal or cervical cavities.⁴⁹

Treatment of severe cases of yellow-brown opacities, fluorosis (TF index of 7-9), severe hypoplastic AI, and hypomaturation and hypocalcification types of AI

Crown restoration with resin composites, polycarbon-

ate crowns, laminate veneers, or porcelain crowns may be necessary. If severe DDE is suspected in an erupting tooth, a temporary glass ionomer or composite restoration of the defect is advised as early as possible, given that progressive deterioration of tooth substance is not predictable.⁵⁰

In cases of MIH with large deep yellow-brown lesions, it is advisable to remove the defective enamel with rotary instruments and roughen the cervical enamel prior to adhesion with a self-etching primer system.²⁵⁻³³

When the defects involve proximal surfaces, celluloid strip crowns offer reasonable esthetics and retention. In MIH, a self-etching primer bonding system is preferred.²⁵⁻³³ In hypoplastic AI, pretreatment with 5% sodium hypochlorite (NaOCl) to remove protein encasing the hydroxylapatite is suggested,⁵¹⁻⁵³ followed by a self-etching, primer-bonding system.

In severely stained fluorosed teeth, the application of 12% HCl, followed by 5% NaOCl, may dissolve the calcified layer that covers the fluorosed enamel and exposes residual organic material, dissolving it. This procedure can enhance esthetics as well as the adhesion of composite resins to the teeth.⁵⁴

A decreased adhesion is expected and restoration with polycarbonate or cast crowns may prove to be more durable^{55,56}: (1) when the enamel defects of MIH are extensive and celluloid strip crowns fail to provide a long-lasting solution; and (2) in severe hypoplastic AI (eg, subtypes: Id-smooth hypoplastic, autosomal dominant, males with subtype Ie-smooth hypoplastic, X-linked dominant, most cases with hypomaturation, AI, hypocalcified AI).

This is particularly true when proximal surfaces are involved. AI patients tend to have an early recession of pulp horns; this and the minimal preparation necessary enable restorations with temporary/permanent crowns at an early age (Figures 1-3).



Fig. 1. Intraoral view of an 11-year-old girl with hypoplastic amelogenesis imperfecta at the initial examination.

The direct technique involves: (1) minimal tooth preparation; (2) adjustment of a preformed polycarbonate crown relining with acrylic resin as necessary; and (3) cementing with GIC.

Alternatively, the crown can be prepared at chairside with

quick-curing acrylic resin. The indirect technique involves: (1) minimal tooth preparation; (2) taking an impression; and (3) cementing a provisional polycarbonate or acrylic crown. An acrylic crown is: (1) prepared in the laboratory; (2) re-lined; (3) adjusted in the patient's mouth as necessary; and (4) cemented with GIC.



Fig. 2. Intraoral view of the incisors and canines restored with celluloid strip crowns (3M/Unitek, St. Paul, Minn) and composite resin Z-100 (3M, St. Paul, Minn).

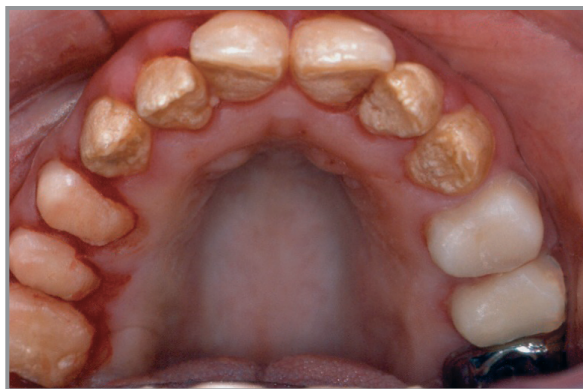


Fig. 3. Intraoral view of the teeth prepared for intermediate crowns (direct technique) and restoration of premolars with intermediate polycarbonate crowns (3M/ESPE St. Paul, Minn), and Unifast, Trad, GC, Tokyo, Japan). The molars were restored with 3M/Unitek stainless steel crowns (3M/ESPE).

Intermediate polycarbonate crowns: (1) are not as technique sensitive or costly as cast restorations; (2) require less cooperation and time to insert; (3) preserve arch length and vertical dimensions; and (4) help in improving oral hygiene, function and esthetics.

The authors have excellent long-term results in severe cases of AI and DI both in primary and permanent dentition.

GIC is recommended for cementing due to its: (1) fluoride-releasing and fluoride-recharging properties at a later date; and (2) improved adhesion characteristics.

These properties may be an asset in cases of severe hypomineralization, enhancing the resistance to secondary caries and decreasing thermal sensitivity.

Whenever possible, porcelain crowns are recommended because they accumulate less calculus.⁵⁷

When a permanent first molar is severely hypomineralized:

1. Early orthodontic and prosthetic assessment is essential. The following factors may influence the long-term prognosis and dictate extraction:
 - a. evaluation of the patient and/or guardians preferences;
 - b. behavior management;
 - c. tooth vitality;
 - d. restorability;
 - e. dental age;
 - f. skeletal relationship and growth;
 - g. buccal segment crowding;
 - h. occlusal relationships;
 - i. presence of wisdom teeth; and
 - j. the condition of other teeth and other developmental anomalies.^{25,58,59}

Moreover, orthodontic advice can help close spaces and improve occlusion. Separation of teeth before crown preparation may conserve tooth material in young permanent teeth.⁵³

2. The permanent molars should be covered with stainless steel crowns (SSC) as early as possible to:
 - a. conserve tooth integrity and vitality;
 - b. diminish tooth sensitivity; and
 - c. establish correct interproximal and occlusal relationships.

SSCs improve oral hygiene and function because they are:

- a. not as technique sensitive or costly as cast restorations;
- b. require little time to insert; and
- c. preserve arch length and vertical dimensions.

Treatment of DI

There is variable expression of the disease with variable levels of attrition and discoloration. Frequently, the primary dentition is more damaged than the permanent, particularly when the expression is part of osteogenesis imperfecta (Shields type I).⁶¹ In a third of the cases, there is also a defect in the enamel's calcification, which may be expressed as hypomineralization or hypoplasia.⁶²

The management is matched to the severity of the disease's clinical expression. In patients without cracks and rapid attrition of the enamel, intracoronal restorations and veneers may be considered for anterior teeth.⁵⁷ External bleaching with carbamide peroxide has been reported with excellent results.⁶³ Most cases are severe, however, and demand early restoration of posterior teeth with stainless steel crowns. In the anterior dentition, celluloid strip crowns may suffer from retention problems and laboratory acrylic crowns in the indirect technique may prove more retentive with better long-term esthetic results (Figures 4-6).⁶⁴ Stain-

less steel anterior crowns with composite facings may also be considered.⁵⁷ Permanent anterior teeth should be covered with composite as soon as they start their eruption. This may reduce the attrition and, soon after further eruption, restoration of those teeth with celluloid strip crowns or polycarbonate crowns is performed. At a later stage, porcelain crowns are suggested.



Fig. 4. Intraoral view of a 4-year-old girl with dentinogenesis imperfecta. Some of the anterior teeth were restored with celluloid strip crowns (Pedo Strip Crowns, 3M/Unitek, St. Paul, Minn) and composite Z-100 (3M/ESPE, St. Paul, Minn); others were restored with laboratory prepared acrylic crowns. The primary maxillary right central incisor (tooth F) is prepared for restoration in the indirect technique following failure of this tooth's composite restoration.



Fig. 5. A plaster-working model of the acrylic crown for tooth F prepared in the laboratory.



Fig. 6. Intraoral view of the same girl 2 years later. Note the durability and esthetics of the laboratory-prepared acrylic crowns compared to the stained and chipped celluloid.

Discussion

The studies published in the field of adhesion of resin materials to hypomineralized enamel:

1. lack standardizations of bonding and adhesive systems used; and
2. differ in:
 - a. storage media;
 - b. testing apparatus;
 - c. specimen preparation;
 - d. bonded surface area (fissures, ground cut or uncut enamel surface); and
 - e. the severity of enamel defects.^{33,51,64}

The extrapolation of conclusions from those articles should, therefore, be taken carefully.

Self-etching primers may not have the same capacity as phosphoric acid to effectively etch fluorosed and uncut or unprepared enamel. Until more investigations are performed, several of the recommendations cited in this article are, at best, educated assumptions.

Many questions are still open, and answers are still unavailable. For example, how do sodium hypochlorite and self-etching system conditioning of hypomineralized enamel affect the adhesion of RMGIC and resin adhesives in various types of DDE?

Conclusions

The treatment of developmental defects of enamel involves an approach that includes several disciplines, including: (1) pediatric dentistry; (2) orthodontics; (3) prosthodontics; and (4) psychology. Proper diagnosis and awareness of the different treatment modalities in each case of DDE may influence the treatments outcome. A close follow-up and maintenance is essential to achieve a long-term success.

References

1. A review of the developmental defects of enamel index (DDE index). Commission on Oral Health Research and Epidemiology. Report of an FDI Working Group. *Int Dent J* 1992;42:411-26.
2. Weerheijm KL, Jalevik B, Alaluusua S. Molar-incisor hypomineralization. *Caries Res* 2001;35:390-1.
3. Weerheijm KL. Molar-incisor hypomineralization (MIH). *Eur J Pediatr Dent* 2003;115-21.
4. Koch G, Hallonsten AL, Ludvigsson N, Hansson BO, Holst A, Ullbro C. Epidemiologic study of idiopathic enamel hypomineralization in permanent teeth of Swedish children. *Community Dent Oral Epidemiol* 1987;15:279-85.
5. Jalevik B, Klingberg G, Barregard L, Noren JG. The prevalence of demarcated opacities in permanent first molars in a group of Swedish children. *Acta Odontol Scand* 2001;59:255-60.

6. Weerheijm KL, Groen HJ, Beentjes VE, Poorterman JH. Prevalence of cheese molars in 11-year-old Dutch children. *J Dent Child* 2001;68:259-62.
7. Jalevik B, Odelius H, Wolfram D, et al. Secondary ion mass spectrometry and X-ray microanalysis of hypomineralized enamel in human permanent first molars. *Arch Oral Biol* 2000;46:239-47.
8. Peretz B, Kafka I. Baby bottle tooth decay and complications during pregnancy and delivery. *Pediatr Dent* 1997;19:34-6.
9. Ellwood RP, O'Mullane DM. Association between dental enamel opacities and dental caries in a north Wales population. *Caries Res* 1994;28:383-7.
10. Leppaniemi A, Lukinmaa P-L, Alaluusua S. Nonfluoride hypomineralizations in the first molars and their impact on the treatment need. *Caries Res* 2001;35:36-40.
11. Burt BA. The changing patterns of systemic fluoride intake. *J Dent Res* 1992;71:1228-37.
12. Curzon MEJ, Spector PC. Enamel mottling in high strontium area of the USA. *Community Dent Oral Epidemiol* 1977;5:243-7.
13. Sae-Lim V, Chulaluk K, Lim LP. Patient and parental awareness of the importance of immediate management of traumatized teeth. *Endod Dent Traumatol* 1999;15:37-41.
14. Welbury RR, Murphy JM. The dental practitioner's role in protecting children from abuse. 2. The orofacial signs of abuse. *Br Dent J* 1998;24:61-5.
15. Borum MK, Andreasen JO. Sequelae of trauma to primary maxillary incisors. I. Complications in the primary dentition. *Endod Dent Traumatol* 1998;14:31-44.
16. Jarvinen S. Incisal overjet and traumatic injuries to upper permanent incisors. A retrospective study. *Acta Odontol Scand* 1978;36:359-62.
17. Buck D, Baker GA, Jacoby A, et al. Patient's experiences of injury as a result of epilepsy. *Epilepsia* 1997;38:439-44.
18. Odoi R, Croucher R, Wong F, Marcenes W. The relationship between problem behavior and traumatic dental injury amongst children aged 7 to 15 years old. *Community Dent Oral Epidemiol* 2002;30:392-6.
19. Seow WK. Clinical diagnosis of enamel defects: Pitfalls and practical guidelines. *Int Dent J* 1997;47:173-82.
20. Cutress TW, Suckling GW. The assessment of noncarious defects of enamel. *Int Dent J* 1982;32:117-22.
21. Needleman HL, Leviton A, Allred E. Macroscopic enamel defects of primary anterior teeth: Types, prevalence, and distribution. *Pediatr Dent* 1991;13:208-16.
22. Cutress TW, Suckling GW. Differential diagnosis of dental fluorosis. *J Dent Res* 1990;69:714-20.
23. Jalevik B. Enamel hypomineralization in permanent first molars. *Swed Dent J* 2001;149(suppl):1-82.
24. William V, Messer LB, Burrow MF. Molar incisor hypomineralization: Review and recommendation for clinical management. *Pediatr Dent* 2006;28:224-32.
25. Jalevik B, Noren JG, Klingberg G. Etiologic factors influencing the prevalence of demarcated opacities in permanent first molars in a group of Swedish children. *Eur J Oral Sci* 2001;109:230-4.
26. Jalevik B, Noren JG. Enamel hypomineralization of permanent first molars: A morphological study and survey of possible etiological factors. *Int J Paediatr Dent* 2000;10:278-89.
27. American Academy of Pediatric Dentistry Council on Clinical Affairs. Policy on use of a caries-risk assessment tool (CAT) for infants, children, and adolescents. Reference Manual 2005-06. *Pediatr Dent* 2005;27:25-7.
28. Adair SM. Evidence-based use of fluoride in contemporary pediatric dental practice. *Pediatr Dent* 2006;28:133-42.
29. Giniger M, MacDonald J, Ziemba S, et al. The clinical performance of professionally dispensed bleaching gel with added amorphous calcium phosphate. *J Am Dent Assoc* 2005;136:383-92.
30. Iijima Y, Cai F, Shen P, et al. Acid resistance of enamel subsurface lesions remineralized by a sugar-free chewing gum containing casein phosphopeptide-amorphous calcium phosphate. *Caries Res* 2004;38:551-6.
31. Kilpatrick N, Mahoney EK. Dental erosion: Part 2. The management of dental erosion. *NZ Dent J* 2004;100:42-7.
32. William V, Burrow MF, Palamara JEA, Messer LB. Microshear bond strength of resin composite to teeth affected by molar hypomineralization using 2 adhesive systems. *Pediatr Dent* 2006;28:233-41.
33. Hosein I, Sherriff M, Ireland AJ. Enamel loss during bonding, debonding, and clean-up with use of a self-etching primer. *Am J Orthod Dentofacial Orthop* 2004;126:717-24.
34. Valente RM, De Rijk WG, Drummond JL, et al. Etching conditions for resin-modified glass ionomer cement for orthodontic brackets. *Am J Orthod Dentofacial Orthop* 2002;121:516-20.
35. Cacciafesta V, Sfondrini MF, Scrbante A, et al. Effect of blood contamination on shear bond strength of brackets bonded with a self-etching primer combined with a resin-modified glass ionomer. *Am J Orthod Dentofacial Orthop* 2004;126:703-8.
36. Suda R, Andoh Y, Shionome M, Hasegawa K, Itoh K, Wakumoto S. Clinical evaluation of the sedative effect of HEMA solution on the hypersensitivity of dentin. *Dent Mater J* 1990;9:163-6.
37. Thylstrup A, Fejerskov O. Clinical appearance of dental fluorosis in permanent teeth in relation to histologic changes. *Community Dent Oral Epidemiol* 1978;6:315-28.
38. Croll TP. Enamel microabrasion: Observations after 10 years. *J Am Dent Assoc* 1997;128(suppl):45S-50S.
39. Akpata ES. Occurrence and management of dental fluorosis. *Int Dent J* 2001;51:325-33.

40. Dalzell DP, Howes RI, Hubler PM. Microabrasion: Effect of time, number of applications, and pressure on enamel loss. *Pediatr Dent* 1995;17:207-11.
41. McDonald RE, Hartsfield JK. Acquired and developmental disturbances of the teeth and associated oral structures. In: McDonald RE, Avery DR, eds. *Dentistry for the Child and Adolescent*. St. Louis, Mo: Mosby; 2000:105-50.
42. Giniger M, MacDonald J, Ziembra S, et al. The clinical performance of professionally dispensed bleaching gel with added amorphous calcium phosphate. *J Am Dent Assoc* 2005;136:383-92.
43. Iijima Y, Cai F, Shen P, et al. Acid resistance of enamel subsurface lesions remineralized by a sugar-free chewing gum containing casein phosphopeptide-amorphous calcium phosphate. *Caries Res* 2004;38:551-6.
44. Ashkenazi M, Sarnat H. Microabrasion of teeth with discoloration resembling hypomaturation enamel defects: Four-year follow-up. *J Clin Pediatr Dent* 2000;25:29-34.
45. de Araujo EB, Zis V, Dutra CA. Enamel color change by microabrasion and resin-based composite. *Am J Dent* 2000;13:6-7.
46. Ateyah N, Akpata E. Factors affecting shear bond strength of composite resin to fluorosed human enamel. *Oper Dent* 2000;25:216-22.
47. Weerasinghe DS, Nikaido T, Wettasinghe KA, Abayakoon JB. Micro-shear bond strength and morphological analysis of self-etching primer adhesive system to fluorosed enamel. *J Dent* 2005;33:419-26.
48. Awliya WY, Akpata ES. Effect of fluorosis on shear bond strength of glass ionomer-based restorative materials to dentin. *J Prosthet Dent* 1999;81:290-4.
49. Alaluusua S, Backman B, Brook AH, et al. Developmental defects of dental hard tissue and their treatment. In: Koch G, Poulsen S, eds. *Pediatric Dentistry: A Clinical Approach*. Munksgaard, Copenhagen: Blackwell Publishing Limited 2001:273-99.
50. Venezie RD, Vadiakas G, Christensen JR, et al. Enamel pretreatment with sodium hypochlorite to enhance bonding in hypocalcified amelogenesis imperfecta: Case report and SEM analysis. *Pediatr Dent* 1994;16: 433-6.
51. Wright JT, Hall KI, Yamauche M. The enamel proteins in human amelogenesis imperfecta. *Arch Oral Biol* 1997;42:149-59.
52. Wright JT. The etch-bleach-seal technique for managing stained enamel defects in young permanent incisors. *Pediatr Dent* 2002;24:249-52.
53. Belkhir MS, Douki N. A new concept for removal of dental fluorosis stains. *J Endod* 1991;17:288-92.
54. Seow WK, Amaratunge A. The effects of acid-etching on enamel from different clinical variants of amelogenesis imperfecta: An SEM study. *Pediatr Dent* 1998;20:37-42.
55. Quionez F, Hoover R, Wright JT. Transitional anterior esthetic restorations for patients with enamel defects. *Pediatr Dent* 2000;22:65-7.
56. Wright JT. The diagnosis and treatment of dentinogenesis imperfecta and amelogenesis imperfecta. *Hellenic Dent J* 1992;2:17-24.
57. Fayle SA. Molar-incisor hypomineralization: Restorative management. *Eur J Pediatr Dent* 2003;121-6.
58. Penchas J, Peretz B, Becker A. The dilemma of treating severely decayed first permanent molars in children: To restore or to extract. *J Dent Child* 1994;61:199-205.
59. Jalevik B, Klingberg G. Dental treatment, dental fear, and behavior management problems in children with severe enamel hypomineralization in permanent first molars. *Int J Paediatr Dent* 2002;12:24-32.
60. Shields ED, Bixter D, El-Kafrawy AM. Proposal classification for heritable human dentin defects with a description of a new entity. *Arch Oral Biol* 1973;18:543-53.
61. Witkop CJ, Rao S. *Inherited Defects in Tooth Structure*. Baltimore, Md: Williams and Wilkins; 1971:p.153.
62. Croll TP, Sasa IS. Carbamide peroxide bleaching of teeth with dentinogenesis imperfecta. Discoloration: Report of a case. *Quintessence Int* 1995;26:683-6.
63. Sapir S, Shapira Y. Dentinogenesis imperfecta: An early treatment strategy. *Pediatr Dent* 2001;23:232-7.
64. Whigham R, Salama F, Barenie J, Fairhurst C, Hanes C. Sealant bond strength on dysplastic molars. *Pediatr Dent* 1989;11:246-7.