

American Academy of Pediatric Dentistry 63rd Annual Session Hilton Hotel and Towers Chicago, Illinois

Reference Committee Hearing Materials

Saturday, May 29, 2010 10:00 – 11:00 a.m. Marquette



Council on Clinical Affairs



NOTICE TO ACTIVE AND LIFE MEMBERS

(1) REFERENCE COMMITTEE HEARINGS AND REPORTS & (2) GENERAL ASSEMBLY MEETING

Oral health policies and clinical guidelines of the American Academy of Pediatric Dentistry will be the subject matter for the Reference Committee hearings at the Annual Session. Recommendations from the Council on Clinical Affairs concerning oral health policies and clinical guidelines were available under "Latest News" in the Members-Only section of the AAPD web site (<u>www.aapd.org</u>) for the past month. They are reprinted here for your convenience.

The Reference Committee hearings will take place on Saturday, May 29, 2010 from 10:00 to 11:00 a.m. in the Marquette Room of the Hilton Hotel and Towers. Members are strongly encouraged to attend. Non-members may attend, but will be polled and asked to identify themselves by the chair, and are not allowed to comment. The Reference Committees are intended to be the venue for member discussion on any formal resolutions that will be proposed before the General Assembly. This is an opportunity for members to present testimony on proposed oral health policies and clinical guidelines, and other business to come before the General Assembly.

The General Assembly and Awards Recognition will take place on Sunday, May 30, 2010 from 9:00 a.m. to 11:00 a.m. in the Grand Ballroom of the Hilton Hotel and Towers. The General Assembly is a meeting of Active and Life members for the purposes of conducting the business of the AAPD. Final action on recommendations from Reference Committees takes place at the General Assembly.

Reference Committee Reports will be available in the back of **the Grand Ballroom** of the Hilton Hotel and Towers beginning at 8:00 a.m. on Sunday morning May 30,2010 prior to the beginning of the General Assembly at 9:00 a.m. If available in time, copies will also be provided at District Caucuses on Saturday, May 24, 2010 from 1:00 to 2:00 p.m.

2009-2010 Report of the Council on Clinical Affairs

Joseph B. Castellano, Chair Robert L. Delarosa, Board Liaison John Rutkauskas and Mary Essling, Staff Liaisons

District Representative members: Arnold I. Weiss (I) Anupama R. Tate (II) Larry D. Dormois (III) Edward L. Rick (IV) Randall K. Lout (VI)

Consultants and Ex-Officio members: Maria Aslani-Breit, Consultant Elizabeth S. Barr, Consultant Richard S. Chaet, Consultant Noel K. Childers, Consultant Judith R. Chin, Consultant Maria Regina P. Estrella, Consultant Gerald A. Ferretti, Consultant Sara L. Filstrup, Consultant Carolyn Kerins, Consultant Brian J. Sanders, Consultant Issa Sasa, Consultant Jenny Ison Stigers, Consultant Norman Tinanoff, Consultant Marcio A. da Fonseca, Expert Consultant Brian S. Martin, Expert Consultant Deborah Studen-Pavlovich, Expert Consultant and Ex-Officio (Chair, Committee on the Adolescent)

Charge 1.

Status of Charge 1: In Progress

Review all definitions, oral health policies and clinical guidelines at no greater interval than every fifth year. Engage the Council on Scientific Affairs to perform a literature review for scientific validity.

Background and Intent: This is a standing charge to the Council. To be effective advocates for infants, children, adolescents, and persons with special health care needs, AAPD oral health policies and clinical guidelines must be supported by the best available evidence. Documents will be reviewed and revised/reaffirmed/retired in a cycle of not more than 5 year intervals. When there is sufficient reason (e.g., publications from a consensus conference), documents will be evaluated in advance of their scheduled review cycle.

Review the following in 2009-2010:

- a) Definition of the Dental Home
- b) Policy on the Dental Home
- c) Guideline on Adolescent Oral Health Care
- d) Guideline on Pediatric Oral Surgery
- e) Guideline on Oral/Dental Aspects of Abuse
- f) Policy on Hospitalization and OR Access for Dental Care
- g) Policy on Hospital Staff Membership
- h) Guideline on Management of Acute Dental Trauma
- i) Definition of Dental Neglect
- j) Guideline on Acquired TMD in Infants, Children, Adolescents
- k) Policy on Xylitol
- 1) Policy on Prevention of Sports-related Injuries

- m) Policy on Tobacco Use
- n) Policy on Use of a Caries-risk Assessment Tool (CAT) for Infants, Children, and Adolescents

Progress Report for Charge 1

All documents were reviewed by the Council on Clinical Affairs and the Council on Scientific Affairs. If necessary, modifications and/or revisions were made. The final documents are attached for approval.

CCA recommends the "Policy on Use of a Caries–Risk Assessment Tool for Infants, Children, and Adolescents" be changed to a more comprehensive "Guideline on Caries Risk Assessment and Management". The document is attached for review and approval.

Charge 2.

Status of Charge 2: Completed

Annually review all AAPD-endorsed policies and guidelines developed by other healthcare organizations.

Background and Intent: This is a standing charge to the Council to promote optimal standards of care. CCA annually will monitor the policies and guidelines of other dental and medical healthcare organizations to determine when revisions have been made by the authoring group and the appropriateness of AAPD's continued endorsement.

Progress Report for Charge 2

CCA has reviewed these documents and recommends continued endorsement.

Charge 3.

Status of Charge 3: Ongoing

Annually review the tables, charts, graphs and other items found in the resource section of the Reference Manual.

Background and Intent: This is a standing charge to the Council to provide contemporary guidance in clinical practice. CCA will maintain a resource section within the Reference Manual that supplements AAPD oral health policies and clinical guidelines. An annual review will determine the accuracy of information and appropriateness for continued inclusion.

Progress Report for Charge 3

All tables, charts and graphs, and other items were reviewed by CCA.

CCA recommends that the "Decision Tree for Avulsed Teeth" be deleted and replaced with the new "Open Apex Decision Tree" and the "Closed Apex Decision Tree". These documents are attached for review and approval.

CCA recommends the placement of the "Sample Letter for School Absences for Dental Appointments" to be placed in the resource section of the reference manual. The document is attached for review and approval.

Charge 4.

Status of Charge 4: Completed

Identify potential topics for new definitions, oral health policies, clinical guidelines, and items for the resource section. Present a list of potential topics and recommendations to the Board of Trustees annually.

Background and Intent: This is a standing charge to the Council to anticipate and respond effectively to changes in the clinical and scientific environment.

Progress Report for Charge 4

CCA has recommended to the BOT that the following topics be considered for new policies and guidelines.

- a) Policy on Oral Health in Child Care Centers
- b) Policy on Transitioning of Adult Special Health Care Needs Patients
- c) Guideline on Xylitol

Charge 5.

Status of Charge 5: In Progress

Develop definitions, policies, guidelines or other materials as requested by the Board of Trustees. *Background and Intent:* This is a standing charge to the Council. To be effective advocates for infants, children, adolescents, and persons with special health care needs, AAPD must delineate the organization's position on new and emerging health issues and translate science into clinical practice.

Develop the following in 2009-2010:

- a) Policy on Second Opinion
- b) Policy on School Absences for Dental Appointments
- c) Sample Letter for School Absences for Dental Appointments

Background and Intent: Some school principals and some school attendants refused to allow students to be excused from school to attend dental appointments. The intent is to develop a communication device an AAPD member could send to school to stress the importance of dental visits during school hours and allow the students to be excused from school.

Progress Report for Charge 5

All listed documents were developed by the Council on Clinical Affairs and reviewed by the Council on Clinical Affairs and the Council on Scientific Affairs. They are attached for review and approval.

Charge 6.

Status of Charge 6: Completed

At the request of the Committee on Communications, review proposed pamphlets, brochures and other AAPD publications for scientific accuracy and consistency with AAPD Policies and Guidelines.

Background and Intent: This is a standing charge to the Council to ensure that the publications and promotional and educational materials offered to our members, other professionals, and the public are scientifically accurate and consistent with our Policies and Guidelines.

Progress Report for Charge 6

There have been no pamphlets, brochures, or other AAPD publications forwarded to CCA this year.

Charge 7.

Status of Charge 7: Completed

At the request of the Executive Committee of the AAPD, provide timely review of policies, guidelines, and definitions submitted by the AAP Section on Pediatric Dentistry and Oral Health, with particular attention to conformity with AAPD oral health policies and clinical guidelines. *Background and Intent:* This is a standing charge to the Council. This mechanism implements the intent of the Memorandum of Understanding with the AAP Section on Pediatric Dentistry and Oral Health, to review proposed documents for consistency with AAPD policies and guidelines. The Council will review these documents with sensitivity to the embargoed status of the drafts. A summary report will be submitted to the Executive Committee.

Progress Report for Charge 7

There were no policies, guidelines, definitions or other material forwarded to CCA this year.

Additional Comments

I would like to commend this council for their hard work and timeliness in the reviewing, modifying, and revising of all the documents. They have done an exceptional job this year, met each and every deadline with no delays, and are ready for next year's challenges.

I would like to give a special thanks to Margaret Bjerklie for all her administrative support to CCA over the year. She is truly a diamond in the AAPD's crown.

Lastly, thanks to the Board of Trustees. Their direction and support has allowed CCA to continually expand and improve our reference manual.

1 2	Definition of Dental Home
3	Originating Council
4	Council on Clinical Affairs
5	
6	Review Council
7	Council on Clinical Affairs
8	
9	Adopted
10	2006
11	
12	Reaffirmed
13	2010
14 15	
16	The dental home is the ongoing relationship between the dentist and the patient, inclusive
17	of all aspects of oral health care delivered in a comprehensive, continuously accessible,
18	coordinated, and family-centered way. Establishment of a dental home begins no later than
19	12 months of age and includes referral to dental specialists when appropriate.

1 Policy on the Dental Home 2 3 **Originating Council** 4 Council on Clinical Affairs 5 6 **Review Council** 7 Council on Clinical Affairs 8 9 Adopted 10 2001 11 12 Revised 13 2004 14 15 Reaffirmed 16 2010 17 18 19 Purpose

20 The American Academy of Pediatric Dentistry (AAPD) supports the concept of a dental 21 home for all infants, children, adolescents, and persons with special health care needs. The 22 dental home is inclusive of all aspects of oral health that result from the interaction of the 23 patient, parents, nondental professionals, and dental professionals. Establishment of the 24 dental home is initiated by the identification and interaction of these individuals, resulting 25 in a heightened awareness of all issues impacting the patient's oral health. This concept is 26 derived from the American Academy of Pediatrics' (AAP) definition of a medical home 27 which states pediatric primary health care is best delivered where comprehensive, continuously accessible, family-centered, coordinated, compassionate, and culturally-28 29 effective care is available and delivered or supervised by qualified child health specialists.¹⁻⁴ 30 31 Methods 32 This policy is based on a review of the current dental and medical literature related to the

- **33** establishment of a dental home. A MEDLINE search was conducted using the terms
- 34 "dental home", "medical home in pediatrics", and "infant oral health care". Expert
- 35 opinions and best current practices were relied upon when clinical evidence was not
- 36 available.

37

38 Background

The AAP issued a policy statement defining the medical home in 1992.⁵ Since that time, it
has been shown that health care provided to patients in a medical home environment is

- 41 more effective and less costly in comparison to emergency care facilities or hospitals.⁴⁻⁶
- 42 Strong clinical evidence exists for the efficacy of early professional dental care
- 43 complemented with caries-risk assessment, anticipatory guidance, and periodic supervision.
- 44 The establishment of a dental home may follow the medical home model as a cost-effective
- 45 and higher quality health care alternative to emergency care situations.

46 Children who have a dental home are more likely to receive appropriate preventive and

47 routine oral health care. Referral by the primary care physician or health provider has been

48 recommended, based on risk assessment, as early as 6 months of age, 6 months after the first

49 tooth erupts, and no later than 12 months of age.⁷⁻⁹ Furthermore, subsequent periodicity of

50 reappointment is based upon risk assessment. This provides time-critical opportunities to

51 implement preventive health practices and reduce the child's risk of preventable

52 dental/oral disease.¹⁰

53

54 Policy statement

- 55 1. The AAPD encourages parents and other care provi-ders to help every child establish a56 dental home by 12 months of age.
- **57** 2. The AAPD recognizes a dental home should provide:¹¹
- a. comprehensive oral health care including acute care and preventive services in
 accordance with AAPD periodicity schedules¹²;
- 60 b. comprehensive assessment for oral diseases and conditions;
- c. individualized preventive dental health program based upon a caries-risk
 assessment¹³ and a periodontal disease risk assessment¹⁴;
- d. anticipatory guidance about growth and development issues (ie, teething, digit orpacifier habits);
- e. plan for acute dental trauma;

66	f. information about proper care of the child's teeth and gingivae. This would include
67	the prevention, diagnosis, and treatment of disease of the supporting and
68	surrounding tissues and the maintenance of health, function, and esthetics of those
69	structures and tissues;
70	g. dietary counseling;
71	h. referrals to dental specialists when care cannot directly be provided within the
72	dental home;
73	i. education regarding future referral to a dentist knowledgeable and comfortable with
74	adult oral health issues for continuing oral health care; referral at an age determined
75	by patient, parent, and pediatric dentist.
76	3. The AAPD advocates interaction with early intervention programs, schools, early
77	childhood education and child care programs, members of the medical and dental
78	communities, and other public and private community agencies to ensure awareness of
79	age-specific oral health issues.
81 82 83 84 85 86 87 88 90 91 92 93 94 95 96 97 98 90 100 101	 References American Academy of Pediatrics Committee on Children with Disabilities. Care coordination: Integrating health and related systems of care for children with special health care needs. Pediatrics 1999;104(4Pt1):978-81. American Academy of Pediatrics. Committee on Pediatric Workforce. Culturally effective pediatric care: Education and training issues. Pediatrics 1999;103(1):167-70. American Academy of Pediatrics Committee on Pediatric Workforce. Pediatric primary health care. AAP News November 1993;11:7. Reaffirmed June 2001. American Academy of Pediatrics. The medical home. Pediatrics 2002;110(1Pt1):184-6. American Academy of Pediatrics Ad Hoc Task Force on the Definition of the Medical Home. The medical home. Pediatrics 1992;90(5):774. Kempe A, Beaty B, Englund BP, Roark RJ, Hester N, Steiner JF. Quality of care and use of the medical home in a state-funded capitated primary care plan for low-income children. Pediatrics 2000;105(5):1020-8. Nowak AJ, Casamassimo PS. The dental home: A primary oral health concept. J Am Dent Assoc 2002;133 (1):93-8. Nowak AJ. Rationale for the timing of the first oral evaluation. Pediatr Dent 1997;19(1):8-11. American Academy of Pediatrics Section on Pediatric Dentistry. Oral health risk assessment timing and establishment of the dental home. Pediatrics 2003;111(5):1113-6. US Dept of Health and Human Services. Healthy People 2010: Understanding and

- 104 11. Poland C. Pediatric oral health. In: Burns CE, Brady MA, Dann AM, Starr N, eds.
 105 Pediatric Primary Care: A Handbook for Nurse Practitioners. 2nd ed. Philadelphia, Pa:
 106 WB Saunders Co; 2000.
- 107 12. American Academy of Pediatric Dentistry. Guideline on periodicity of examination,
 108 preventive dental services, antic-ipatory guidance, and oral treatment for children.
 109 Pediatr Dent 2004;26(suppl):81-3.
- 110 13. American Academy of Pediatric Dentistry. Policy on use of a caries-risk assessment
 111 tool (CAT) for infants, children, and adolescents. Pediatr Dent 2004;26(suppl):25-7.
- 112 14. American Academy of Periodontology. Periodontal diseases of children and
- adolescents. J Periodontol 2003;74(11):1696-704.

1 2	Guideline on Adolescent Oral Health Care
3	Originating Committee
4	Clinical Affairs Committee
5	
0 7	Review Council Council on Clinical Affairs, Committee on the Adolescent
8	council on clinical Antalis, commutee on the Adolescent
9	Adopted
10	1986
11 12	Payisad
13	1999, 2003, 2005, 2010
14	
15	Purpose
16	The American Academy of Pediatric Dentistry (AAPD) recognizes that the adolescent
17	patient has unique needs. This guideline addresses these unique needs and proposes
18	general recommendations for their management.
19	
20	Methods
21	This guideline is an update of the previous document, revised in 2005. The update includes
22	an electronic search using the search terms, based on a review of the current dental and
23	medical literature related to adolescent oral health. A MEDLINE search was conducted
24	using the term "adolescent" combined with "dental", "gingivitis", "oral piercing",
25	"sealants", "oral health", "caries", "tobacco use", "dental trauma", "orofacial trauma
26	"periodontal", "dental esthetics", "smokeless tobacco", "nutrition", and "diet". Fields: All
27	Fields; Limits: within the last ten years: humans; English; clinical trails. The reviewers
28	agreed upon the inclusion of five thousand eight hundred seventy-four (5874) and four
29	hand searched articles that met the defined criteria. When data did not appear sufficient or
30	were inconclusive, recommendations were based upon expert and/or consensus opinion by
31	experienced researchers and clinicians.
32	

33 Background

- 34 There is no standard definition of "adolescent". ¹ Adolescents are defined very broadly as
- 35 youths between the ages of 10 to 18. Using this definition, there were approximately 39.9

36 41.5 million adolescents in the United States in 2003 2008, according to the US Census 37 Bureau.² The adolescent patient is recognized as having distinctive needs^{3,4} due to: (1) a 38 potentially high caries rate; (2) increased risk for traumatic injury and periodontal disease; 39 (3) a tendency for poor nutritional habits; (4) an increased esthetic desire and awareness; (5) complexity of combined orthodontic and restorative care (e.g., congenitally missing teeth); 40 41 (6) dental phobia; (7) potential use initiation of tobacco, alcohol and other drugs use; (8) 42 pregnancy; (9) eating disorders; and (10) unique social and psychological needs.⁵⁻⁸ 43 Treatment of the adolescent patient can be multifaceted and complex. An accurate, 44 comprehensive, and up-to-date medical history is necessary for correct diagnosis and 45 effective treatment planning. Familiarity with the patient's medical history is essential to 46 decreasing the risk of aggravating a medical condition while rendering dental care. If the 47 parent is unable to provide adequate details regarding a patient's medical history, 48 consultation with the medical health care provider may be indicated. The practitioner also 49 may need to obtain additional information confidentially from an adolescent patient. 50 51 Recommendations 52 This guideline addresses some of the special needs within the adolescent population and 53 proposes general recommendations for their management. 54 55 Caries 56 Adolescence marks a period of significant caries activity for many individuals. Current 57 research suggests that the overall caries rate is declining, yet remains highest during 58 adolescence. 9 These carious lesions often are confined to developmental pits and fissures.¹⁰ 59 Immature permanent tooth enamel,¹¹⁰ a total increase in susceptible tooth surfaces, and 60 environmental factors such as diet, independence to seek care or avoid it, a low priority for 61 oral hygiene, and additional social factors also may contribute to the upward slope of caries 62 in adolescence. ^{1,12-14} It is important for the dental provider to emphasize the positive effects 63 that fluoridation, routine professional care, patient education, and personal hygiene can 64 have in counteracting the changing pattern of caries in the adolescent population. 5.6.11.15 65

66 Management of Caries

67 *Primary prevention*

68 <u>Fluoride:</u> Fluoridation has proven to be the most economical and effective caries prevention

69 measure. The adolescent can benefit from fluoride throughout the teenage years and into

- 70 early adulthood. Although the systemic benefit of fluoride incorporation into developing
- 71 enamel is not considered necessary past 16 years of age, the topical benefits of

72 remineralization and antimicrobial activity still can be obtained through water fluoridation,

- 73 optimally fluoridated water, professionally-applied and prescribed compounds, and
- 74 fluoridated dentifrices.¹⁶
- 75 <u>*Recommendations:*</u> The adolescent should receive maximum fluoride benefit <u>dependent</u>
- 76 <u>on risk assessment</u>: <u>17</u>
- 77 <u>-1. Systemic fluoride intake via optimal fluoridation of drinking water or professionally</u>
- 78 prescribed supplements is recommended to 16 years of age. or the eruption of the
 79 second permanent molars, whichever comes first.
- 80 2<u>1</u>. <u>Brushing teeth twice a day with a F-f</u>luoridated dentifrice is recommended to provide
- 81 continuing topical benefits through adolescence.^{11, 18}
- 82 <u>32</u>. Professionally applied fluoride treatments should be based on the individual patient's
 83 caries-risk assessment, as determined by the patient's dental provider.¹⁸
- 84 4<u>3</u>. <u>Home-applied prescription strength </u><u>T</u>topical fluoride <u>products</u> supplementation via
- 85 home-applied compounds (e.g., 0.4% stannous fluoride gel, 0.5% F gel or paste, or 0.2%
- 86 <u>NaF rinse</u>) should may be a professional recommendation used when indicated by an
- 87 individual's caries pattern or caries risk status.¹⁸
- 88 1<u>4</u>. Systemic fluoride intake via optimal fluoridation of drinking water or professionally
- 89 prescribed supplements is recommended to 16 years of age. Supplements should only
 90 be given after all other sources of fluoride have been evaluated.¹⁸
- 5. The criteria for determination of need and the methods of delivery should be those currently
 recommended by the American Dental Association and the AAPD.⁴⁷⁻¹⁸
- 93
- 94
- 95

96 Oral hygiene: Adolescence can be a time of heightened caries activity and periodontal 97 disease due to an increased intake of cariogenic substances and inattention to oral hygiene 98 procedures. 1,18 19 Tooth brushing with a fluoridated dentifrice and flossing can provide an 99 anticaries benefit through plaque removal from tooth surfaces and the topical effect of the 100 fluoride and plaque removal from tooth surfaces.20 101 Recommendations: 102 1. Adolescents should be educated and motivated to maintain personal oral hygiene 103 through daily plaque removal, including flossing, with the frequency and pattern based 104 on the individual's disease pattern and oral hygiene needs.19,20 105 2. Professional removal of plaque and calculus is recommended highly for the adolescent, 106 with the frequency of such intervention based on the individual's assessed risk for 107 caries/periodontal disease, as determined by the patient's dental provider.¹⁹ 20.21 108 109 Diet management: The role of carbohydrates in caries initiation is unequivocal. Adolescents 110 are exposed to and consume high quantities of refined carbohydrates and acid-containing 111 beverages.^{12, 13, 20} <u>22</u>The adolescent can benefit from diet analysis and modification. 112 **Recommendations:** Diet analysis, along with professionally determined recommendations 113 for maximal general and dental health, should be part of an adolescent's dental health 114 management. 23 A diet analysis and management should consider: 115 1. dental disease patterns; 116 2. overall nutrient and energy needs; 117 3. psychosocial aspects of adolescent nutrition; 118 4. dietary carbohydrate intake and frequency; 119 5. intake and frequency of acid-containing beverages; 120 6. wellness considerations; 121 122 Sealants: Sealant placement is an effective caries-preventive technique that should be 123 considered on an individual basis. Sealants have been recommended for any adolescent 124 tooth, primary or permanent, that is judged to be at risk for pit and fissure caries.^{6, 13,21-24} 24-27

Caries risk may increase due to changes in patient habits, oral microflora, or physical
condition, and unsealed teeth subsequently might benefit from sealant applications.²⁴²⁷
<u>Recommendations:</u> Adolescents at risk for caries should have sealants placed. An
individual's caries risk may change over time; periodic reassessment for sealant need is
indicated throughout adolescence.²⁴²⁷ *Secondary prevention*<u>Professional preventive care:</u> Professional preventive dental care, on a routine basis, may

prevent oral disease or disclose existing disease in its early stages. The adolescent patient
whose oral health has not been monitored routinely by a dentist may have advanced caries,
periodontal disease, or other oral involvement urgently in need of professional evaluation
and extensive treatment.

137 <u>Recommendations:</u>

- Timing of periodic oral examinations should take into consideration the individual's
 needs and risk indicators to determine the most cost-effective, disease-preventive
 benefit to the adolescent.¹⁷
- 141 2. Initial and periodic radiographic evaluation should be a part of a clinical evaluation.
- 142The type, number, and frequency of radiographs should be determined only after an
- 143 oral examination and history taking. Previously exposed radiographs should be
- 144available, whenever possible, for comparison. Currently accepted guidelines for
- 145 radiographic exposures (i.e., appropriate films based upon medical history, caries risk,
- 146 history of periodontal disease, and growth and development assessments) should be

147 followed.²⁵⁻²⁸

148

<u>Restorative dentistry:</u> In cases where remineralization of noncavitated, demineralized tooth
 surfaces is not successful, as demonstrated by progression of carious lesions, dental
 restorations are necessary. Preservation of tooth structure, esthetics, and each individual
 patient's needs must be considered when selecting a restorative material.²⁶²⁹ Molars with
 extensive caries or malformed, hypoplastic enamel – for which traditional amalgam or
 composite resin restorations are not feasible – may require full coverage restorations.^{24, 27, 27, 30}

Council on Clinical Affairs 2009-10 Charge 1(c) G-Adolescent Oral Health Care

155	Recommendations: Each adolescent patient and restoration must be evaluated on an
156	individual basis. Preservation of noncarious tooth structure is desirable. Referral to an
157	appropriately trained and/or experienced dentist should be considered <u>made</u> when
158	treatment needs are beyond the treating dentist's ability or interest scope of practice. 27
159	
160	Periodontal diseases
161	Adolescence can be a critical period in the human being's periodontal status. Epidemiologic
162	and immunologic data suggest that irreversible tissue damage from periodontal disease
163	begins in late adolescence and early adulthood. ⁸ Pubertal changes characteristically affect
164	the periodontium of the young adolescent, with an increase in inflammation, which is, in
165	most cases, manageable through oral hygiene and regular professional care.28 Adolescents
166	have a higher prevalence of gingivitis than prepubertal children or adults. The rise of sex
167	hormones during adolescence is suspected to be the a cause of the increased prevalence.
168	Studies suggest that the increase in sex hormones during puberty affects the composition of
169	the subgingival microflora. ³¹ Other studies suggest circulating sex hormones may alter
170	capillary permeability and increase fluid accumulation in the gingival tissues. This
171	inflammatory gingivitis is believed to be transient as the body accommodates to the ongoing
172	presence of the sex hormones. ³²
173	
174	Acute conditions: The adolescent may be subjected to acute conditions such as acute
175	necrotizing ulcerative gingivitis , and periodontitis, and traumatic injuries, which can
176	require immediate and occasional long-term management. In most cases, early diagnosis,
177	treatment, and appropriate management can prevent irreversible damage. ^{29.3133-35}
178	Recommendations: Acute intraoral infection involving the periodontium and oral mucosa
179	requires immediate treatment. Therapeutic management should be based on currently
180	accepted techniques of periodontal therapy. ^{36, 37} Traumatic injuries to the teeth and
181	periodontium always require dental evaluation and treatment.36 Referral to an
182	appropriately trained and/or experienced dentist should be considered <u>made</u> when the
183	treatment needs are beyond the treating dentist's ability or interest scope of practice.
184	

- 185 Chronic conditions: Chronic conditions affecting the adolescent include, but are not limited 186 to, gingivitis, puberty gingivitis, hyperplastic gingivitis related to orthodontic therapy, 187 gingival recession that may or may not be related to orthodontic therapy, drug-related 188 gingivitis, pregnancy gingivitis, localized juvenile periodontitis, and periodontitis.^{21, 28,29,32} 189 24,31,32,33 Personal oral hygiene and regular professional intervention can minimize occurrence 190 of these conditions and prevent irreversible damage. 191 *Recommendations:* The adolescent will benefit from an individualized preventive dental 192 health program, which includes the following items aimed specifically at periodontal health: 193 1. Patient education emphasizing the etiology, characteristics, and prevention of 194 periodontal diseases, as well as self-hygiene skills. 32-34 195 2. A personal, age-appropriate oral hygiene program including plaque removal, oral 196 health self-assessment, and diet. Sulcular brushing and flossing should be included in 197 plaque removal, and frequent follow-up to determine adequacy of plaque removal and 198 improvement of gingival health should be considered. 32-36 199 3. Regular professional intervention, the frequency of which should be based on 200 individual needs and should include evaluation of personal oral hygiene success, 201 periodontal status, and potential complicating factors such as medical conditions, 202 malocclusion, or handicapping conditions. Periodontal probing, periodontal charting, 203 and radiographic periodontal diagnosis should be a consideration when caring for the 204 adolescent. The extent and nature of the periodontal evaluation should be determined 205 professionally on an individual basis. Those patients with progressive periodontal 206 disease should be referred to an appropriately trained and/or experienced dentist for 207 evaluation and treatment. when the treatment needs are beyond the treating dentist's 208 scope of the practice. <u>32-34,36</u> 209 4. Appropriate evaluation for procedures to facilitate orthodontic treatment including, but 210 not limited to, tooth exposure, frenectomy, fiberotomy, gingival augmentation, and 211 implant placement.3337 212
- 213 Occlusal considerations

214	Malocclusion can be a significant treatment need in the adolescent population as both
215	environmental and genetic factors come into play. Although the genetic basis of much
216	malocclusion makes it unpreventable, numerous methods exist to treat the occlusal
217	disharmonies, temporomandibular joint dysfunction, periodontal disease, and disfiguration,
218	which may be associated with malocclusion. Within the area of occlusal problems are
219	several tooth/jaw-related discrepancies that can affect the adolescent. Third molar
220	malposition and temporomandibular disorders require special attention to avoid long-term
221	problems. Congenitally missing teeth present complex problems for the adolescent and
222	often require combined orthodontic and restorative care for satisfactory resolution.
223	
224	Malocclusion: Any tooth/jaw positional problems that present significant esthetic,
225	functional, physiologic, or emotional dysfunction are potential difficulties for the
226	adolescent. These can include single or multiple tooth malpositions, tooth/jaw size
227	discrepancies, and craniofacial disfigurements.
228	<u>Recommendations:</u> Any <u>mMalposition</u> of teeth, malrelationship of teeth to jaws,
229	tooth/jaw size discrepancy, bimaxillary <u>skeletal</u> malrelationship, or craniofacial
230	malformations or disfigurement that presents functional, esthetic, physiologic, or emotional
231	problems to the adolescent should be <u>referred for evaluated</u> <u>evaluation when the treatment</u>
232	needs are beyond the treating dentist's scope of practice. by an appropriately trained
233	dentist or professional team. Treatment of malocclusion by an appropriately trained and/or
234	experienced dentist should be based on professional diagnosis, available treatment options,
235	patient motivation and readiness, and other factors to maximize progress. 48
236	
237	Third molars: Third molars can present acute and chronic problems for the adolescent.
238	Impaction or malposition leading to such problems as pericoronitis, caries, cysts, or
239	periodontal problems merits evaluation for removal. ^{32, 34} 36,38The role of The third molar as a
240	functional tooth also should be considered. Although prophylactic removal of all impacted
241	or unerupted disease-free third molars is not indicated, consideration should be given to
242	removal by the third decade when there is a high probability of disease or pathology and/or
243	the risks associated with early removal are less than the risks of later removal.

244	<u>Recommendations</u> : Evaluation of third molars, including radiographic diagnostic aids,
245	should be an integral part of the dental examination of the adolescent. ²⁵²⁸ For diagnostic and
246	extraction criteria, refer to AAPD's Guideline on Pediatric Oral Surgery. ³⁵³⁹ Treatment of
247	third molars that are potential or active problems should be performed by an appropriately
248	trained and/or experienced <u>a licensed</u> dentist. <u>Referral should be made if treatment is</u>
249	beyond the treating dentist's scope of practice.
250	
251	Temporomandibular joint (TMI) problems: Disorders of the TMJ can occur at any age, but
252	appear to be more prevalent in adolescence may provide the stimulus to trigger problems. ³⁶⁻
253	39 <u>3940-41</u>
254	<u>Recommendations</u> : Evaluation of the TMJ and related structures should be a part of the
255	examination of the adolescent. Abnormalities should be managed by an appropriately
256	trained and/or experienced dentist following accepted clinical procedures. <u>Referral</u>
257	should be made when the treatment needs are beyond treating dentist's scope of practice.40.
258	4144,45
259	
260	Congenitally missing teeth: The impact of a congenitally missing permanent tooth on the
261	developing dentition can be significant. ³ When treating adolescent patients with
262	congenitally, missing teeth, many factors must be taken into consideration including, but
263	not limited to, esthetics, patient age, and growth potential, as well as periodontal and oral
264	surgical needs. ⁴²⁻⁴⁴⁴⁶⁻⁴⁸
265	Recommendations: Evaluation of congenitally missing permanent teeth should include
266	both immediate and long-term management. Management Referral should be made when
267	the treatment needs are beyond the treating dentist's scope of practice by an appropriately
268	trained and/or experienced dentist, and $-a$. A team approach may be indicated. ⁴⁵⁴⁹
269	
270	Ectopic eruption and impacted teeth: Abnormal eruption patterns of the adolescent's
271	permanent teeth can contribute to root resorption, bone loss, gingival defects, space loss,
272	and esthetic concerns. Early diagnosis and treatment of ectopically erupting teeth can result
273	in a healthier and more esthetic dentition. Prevention and treatment may include extraction

- of deciduous teeth, surgical intervention, and/or endodontic, orthodontic, periodontal,
- and/or restorative care.46-5050-52

276 <u>*Recommendations:*</u> The dentist should be proactive in diagnosing and treating ectopic

- 277 eruption and impacted teeth in the young adolescent. Early diagnosis, including
- 278 appropriate radiographic examination²⁵²⁸ of ectopic eruption, is important. <u>Referral should</u>
- 279 <u>be made when the treatment needs are beyond the treating dentist's scope of practice.</u> An

280 appropriately trained and/or experienced dentist should manage treatment, and A team

- approach may be necessary.⁴⁵⁴⁹
- 282

283 Traumatic injuries

The most common injuries to permanent teeth occur secondary to falls, followed by traffic accidents, violence, and sports.⁵¹⁻⁵⁴⁵⁴⁻⁵⁷ All sporting activities have an associated risk of orofacial injuries due to falls, collisions, and contact with hard surfaces.⁵⁵⁵⁹ The administrators of youth, high school, and college organized sports have demonstrated that dental and facial injuries can be reduced significantly by introducing mandatory protective equipment such as face guards and mouthguards. Additionally, youths participating in leisure activities such as skateboarding, rollerskating, and bicycling also benefit from

291 appropriate protective equipment.^{56, 5760,61}

292 **Recommendations:** Dentists should introduce a comprehensive trauma prevention 293 program to help reduce the incidence of traumatic injury to the adolescent dentition. This 294 prevention plan should consider assessment of the patient's sport or activity, including level 295 and frequency of activity.⁵⁸⁶² Once this information is acquired, recommendation and 296 fabrication of an age-appropriate, sport-specific, and properly-fitted mouthguard/faceguard 297 can be initiated.⁵⁸⁶² Players must be warned about altering the protective equipment that 298 will disrupt the fit of the appliance. In addition, players and parents must be informed that 299 injury may occur, even with properly fitted protective equipment.5862

300

301 Additional considerations in oral/dental management of the adolescent

302 The adolescent can present particular psychosocial characteristics that impact the health303 status of the oral cavity, care seeking, and compliance. The self-concept development

304 process, emergence of independence, and the influence of peers are just a few of the 305 psychodynamic factors impacting dental health during this period.^{1, 5, 7,16} 306 Discolored or stained teeth: Desire to improve esthetics of the dentition by tooth whitening 307 and removal of stained areas or defects can be a concern of the adolescent. Indications for 308 the appropriate use of tooth-whitening methods and products are dependent upon correct 309 diagnosis.5963 310 The dentist must determine the appropriate mode of treatment. Use of bleaching agents, 311 microabrasion, placement of an esthetic restoration, or a combination of treatments all can 312 be considered.⁶⁰⁶⁴ 313 *<u>Recommendations</u>*: For the adolescent patient, judicious use of bleaching can be 314 considered part of a comprehensive, sequenced treatment plan that takes into consideration 315 the patient's dental developmental stage, oral hygiene, and caries status. A dentist should 316 monitor the bleaching process, ensuring the least invasive, most effective treatment method. 317 Dental professionals also should consider possible side effects when contemplating dental 318 bleaching for adolescent patients.^{61, 6265,66} 319 320 <u>Tobacco use:</u> Significant oral, dental, and systemic health consequences and death are 321 associated with all forms of tobacco use. Smoking and other tobacco use almost always are 322 initiated and established in adolescence.63-6867-72 323 Recommendations: Education of the adolescent patient on the oral and systemic 324 consequences of tobacco use should be part of each patient's oral health education. For those 325 adolescent patients who use tobacco products, the practitioner should provide or refer the 326 patient to appropriate educational and counseling services.⁶⁹⁻⁷¹⁷³⁻⁷⁵When associated 327 pathology is present, treatment should be managed by an appropriately trained and/or 328 experienced health care provider referral should be made when the treatment needs are 329 beyond the treating dentist's scope of practice. 330 331 Positive youth development: Treatment and management of adolescent oral health that 332 takes into account the adolescent's psychological and social needs can be approached 333 through the framework of positive youth development (PYD). 7276 The approach goes

334	beyond traditional prevention, intervention, and treatment of risky behaviors and problems
335	and suggests that a strong interpersonal relationship between the adolescent patient and the
336	pediatric dentist can be influential in improving adolescent oral health and transitioning
337	patients to adult care. In the office, dental professionals have a unique opportunity to serve
338	as positive role models.
339	<u>Recommendations:</u> PYD should be recognized as containing a number of key elements
340	relevant to care patients of this age:
341	-1. providing youth with safe and supportive environments; 26
342	-2. fostering relationships between young people and caring adults who can mentor and
343	guide them. 26
344	-3. promoting healthy lifestyles and teaching positive patterns of social interaction.76
345	-4. providing a safety net in times of need. ⁷²⁷⁶
346	Integrating PYD into clinical practice can be attained through continuing education on
347	adolescent development issues, as well as partnerships with community-based
348	organizations and schools. The dentist can be a part of the myriad of adolescent support and
349	services. ⁷⁶
350	
351	Psychosocial and other considerations: Behavioral considerations when treating an
352	adolescent may include anxiety, phobia, or intellectual dysfunction. ¹ These_special needs
353	should be managed by an appropriately trained dentists. Referral should be made when the
354	treatment needs are beyond the treating dentist's scope of practice, to and nondental
355	professionals or a team approach may be indicated.
356	Additional examples of oral problems associated with adolescent behaviors include, but
357	are not limited to:
358	1. oral manifestations of venereal diseases;
359	2. effects of oral contraceptives or antibiotics on periodontal structures;
360	3. perimyolysis <u>(severe enamel erosion)</u> in bulimia ⁷³⁷⁷
361	4. traumatic injury to teeth and oral structures in athletic or other activities (short- and
362	
	long-term management) 57,7476 <u>60,78-80</u>

- 364 The impact of psychosocial factors relating to oral health must include consideration of365 the following:
- 366 1. changes in dietary habits (e.g., fads, freedom to snack, increased energy needs, access to367 carbohydrates);
- **368** 2. use and abuse of tobacco, alcohol and drugs;
- 369 3. motivation for maintenance of good oral hygiene;
- 370 4. potential for traumatic injury;
- **371** 5. adolescent as responsible for care;
- **372** 6. lack of knowledge about periodontal disease.
- 373 Physiologic changes also can account for significant contribute to oral problems
- 374 <u>concerns</u> in the adolescent. These <u>physiologic changes</u> include:
- **375** 1. loss of remaining primary teeth;
- **376** 2. eruption of remaining permanent teeth;
- **377** 3. gingival maturity;
- **378** 4. facial growth;
- **379** 5. hormonal changes.
- 380 <u>Recommendations:</u>
- **381** 1. Oral health care of the adolescent <u>Adolescent's oral health care should be provided by</u>
- a dentist who has appropriate training in managing the patient's specific needs. <u>Referral</u>
- 383 should made when the treatment needs are beyond the treating dentist's scope of
- 384 <u>practice.</u> The primary care dentist should consider referral to a specialist for treatment
- 385 of particular problems outside his or her expertise. This may include both dental and
 386 nondental problems. ⁷⁶
- 387 2. Attention should be given to the particular psychosocial aspects of adolescent dental
 388 care. Other issues such as consent, confidentiality, and compliance should be addressed
 389 in the care of these patients.^{79, 8083,85}
- 390 3. A complete oral health care program for the adolescent requires an educational
- 391 component that addresses the particular concerns and needs of the adolescent patient
- and focuses on:

- a. specific behaviorally-and physiologically-induced oral manifestations in this age
 group; 20
- b. shared responsibility for care and health by the adolescent and provider;²⁰
- 396 c. consequences of adolescent behavior on oral health. <u>86,87</u>
- 397

398 <u>Transitioning to adult care</u>: As adolescent patients approach the age of majority, it is 399 important to educate the patient and parent on the value of transitioning to a dentist who is 400 knowledgeable in adult oral health care. The adult's oral health needs may go beyond the 401 scope of the pediatric dentist's training. The transitioning adolescent should continue 402 professional oral health care in an environment sensitive to his/her individual needs. Many 403 adolescent patients independently will choose the time to seek care from a general dentist 404 and may elect to seek treatment from a parent's primary care provider. In some instances, 405 however, the treating pediatric dentist will be required to suggest transfer to adult care.

Pediatric dentists are concerned about decreased access to oral health care for persons
with special health care needs (SHCN) ⁸⁴⁸⁸ as they transition beyond the age of majority.
Pediatric hospitals, by imposing age restrictions, can create a barrier to care for these
patients. Transitioning to a dentist who is knowledgeable and comfortable with adult oral
health care needs often is difficult due to a lack of trained providers willing to accept the
responsibility of caring for SHCN patients.

412 <u>Recommendations:</u> At a time agreed upon by the patient, parent, and pediatric dentist, the 413 patient should be transitioned to a dentist knowledgeable and comfortable with managing 414 that patient's specific oral care needs. For the SHCN patient, in cases where it is not 415 possible or desired to transition to another practitioner, the dental home can remain with 416 the pediatric dentist and appropriate referrals for specialized dental care should be 417 recommended when needed.⁸²⁸⁸

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- 419 References
- American Psychological Association. Developing Adolescents: A Reference for Professionals. Washington, DC: American Psychological Association; 2002.
 US Census Bureau. (2001) Census 2000 PHC-T-9. Population by age, sex, race for the United States. Census 2003 Summary File I. Available at: "http://factfinder. census.gov". Accessed February 7, 2005.

- 425 US Census Bureau. United States 2008 Population Estimates by age and sex. Available
 426 at: "http://www.factfinder.census.gov/datasets/annual population estimates/detailed
 427 tables/nation/United States/T6-2008 sex by age". Assessed July 19,2009.
- 428 3. Pinkham JR, Casamassimo PS, Fields HW Jr, McTigue DJ, Nowak AJ. Adolescence. In:
 429 Pediatric Dentistry: Infancy Through Adolescence. 4th ed. Philadelphia, Pa: WB
 430 Saunders Co; 2005:649-718.
- 431
 4. National Institutes of Health. Consensus development conference statement: Diagnosis and management of dental caries throughout life, March 26-28, 2001. J Am Dent Assoc 2001;132(8):1153-61.
- 434 5. Macgregor ID, Regis D, Balding J. Self-concept and dentalhealth behaviors in adolescents. J Clin Periodontol 1997;24(5):335-9.
- 436 6. Yu SM, Bellamy HA, Schwalberg RH, Drum MA. Factors associated with use of
 437 preventive dental and health services among US adolescents. J Adolesc Health
 438 2001;29(6):395-405.
- 439 7. American Academy of Pediatric Dentistry. Policy on prevention of sports-related orofacial injuries. Pediatr Dent 20042008;2630(suppl):44-58-60.
- 441 8. US Dept of Health and Human Services. Oral Health In America: A Report of the
 442 Surgeon General Executive Summary. Rockville, Md: US Dept of Health and Human
 443 Services, National Institute of Dental and Craniofacial Research, National Institutes of
 444 Health; 2000.
- 445 9. Kaste LM, Selwitz RH, Oldakowski JA, Brunelle JA, Winn DM, Brown LJ. Coronal caries
 446 in the primary and permanent dentition of children and adolescents 1-17 years of age:
 447 United States, 1988-1991. J Dent Res 1996;75(special issue):631-41.
- 448 11.10.Kirkham J, Robinson C, Strong M, Shore RC. Effects of frequency of acid exposure on
 449 demineralization/remineralization behavior of human enamel in vitro. Caries Res 1994;
 450 28(1):9-13.
- 451 10.11.Burt BA. Prevention policies in light of the changed distribution of dental caries.
 452 ActaOdontol Scand 1998;56(3):179-86.
- 453 12. Howze KA. Health for Teens in Care: A Judge's Guide 2002. Washington, DC: American
 454 Bar Association; 2002.
- 455 13. Majewski RF. Dental caries in adolescents associated with caffeinated carbonated
 456 beverages. Pediatr Dent 2001;23(3):198-203.
- 457 14. Marshall TA, Levy SM, Broffitt B, et al. Dental caries and beverage consumption in young children. Pediatrics 2003;112(3Pt1):e184-e191.
- 459 15. Irwin CE, Millstein SG. Biophysical correlates of risk-taking behaviors during
 460 adolescence. J Adolesc Health Care 1986;(7):825-965.
- 461 16. CDC. Recommendations for using fluoride to prevent and control dental caries in the462 United States. MMWR Recomm Rep 2001;50(RR14):1-42.
- 463 <u>17. American Academy of Pediatric Dentistry. Policy on use of a caries-risk assessment tool</u>
 464 (CAT) for infants, children, and adolescents. Pediatr Dent 2008;30(suppl):29-33.
- 465 1718. American Academy of Pediatric Dentistry. Guideline on fluoride therapy. Pediatr
 466 Dent 20042008;26-30(suppl): 87-8121-4.
- 467 <u>1819</u>.Macgregor ID, Balding J, Regis D. Tooth-brushing schedule, motivation, and 'lifestyle'
 468 behaviours in 7,770 young adolescents. Community Dent Health 1996;13(4):232-7.

469	1920.Dean JA, Hughes CV. Mechanical and chemotherapeutic home oral hygiene. In:
470	McDonald RE, Avery DR, Dean JA, eds. Dentistry for Child and Adolescent. 8th ed. St
471	Louis, Mo: Mosby; 2004: 236-56.
472	19.21. American Academy of Pediatric Dentistry. Guideline on periodicity of examination,
473	preventive dental services, anticipatory guidance, and oral treatment for children.
474	Pediatr Dent 2004 2008; 26 -30(suppl): 81-3 112-18.
475	20 22.Freeman R, Sheiham A. Understanding decision-making process for sugar
476	consumption in adolescents. Community Dent Oral Epidemiol 1997;25(3):228-32.
477	23. American Academy of Pediatric Dentistry. Policy on dietary recommendations for
478	infants, children, and adolescents. Pediatr Dent 2008;(suppl):47-8.
479	2124.Feigal RJ. The use of pit and fissure sealants. Pediatr Dent 2002;24(5):415-22.
480	2225. Ahovuo-Saloranta A, Hiiri A, Nordblad A, Worthington H, Makela M. Pit and fissure
481	sealants for preventing dental decay in the permanent teeth of children and adolescents
482	[review]. Cochrane Database Syst Rev 2004;(3):CD001830.
483	2326.Macek MD, Beltrán-Aguilar ED, Lockwood SA, Malvitz DM. Updated comparison of
484	the caries susceptibility of various morphological types of permanent teeth. J Public
485	Health Dent 2003;63(3):174-82.
486	2427. American Academy of Pediatric Dentistry. Guideline on pediatric restorative dentistry.
487	Pediatr Dent 2004 2008; 26 30 (suppl): 106-14 163-9.
488	2528. American Academy of Pediatric Dentistry. Guideline on pre-scribing dental
489	radiographs for infants, children, adolescents, and persons with special health care
490	needs. Pediatr Dent 2005 2008; 27 30(suppl): 185-6 236-7.
491	2629.Donly K. Pediatric Restorative Dentistry Consensus Conference April 15-16, 2002, San
492	Antonio, TX. Pediatr Dent 2002;24(5):374-6.
493	2730.Croll TP, Castaldi CR. The preformed stainless steel crown for restoration of
494	permanent posterior teeth in special cases. J Am Dent Assoc 1978;97(4):644-9.
495	28. American Academy of Periodontology. Oral health info for the public: Adolescents and
496	oral health care. Available at: http://www.perio.org/consumerchildren.htm. Accessed
497	February 7, 2005.
498	31. Beck JD. Arbes SJ, Jr. Epidemiology of gingival and periodontal disease. In: Newman
499	MG, Taki HH, Klokkevold PR, Carranza FA, eds. Carranza's Clinical Periodontology.
500	10th ed. St. Louis: Saunders Elsevier; 2006: 117-19.
501	29 32.Wilson, TG, Jr. Kornman KS. Fundamentals of Periodontics. 2nd ed. Quitessence
502	Publishing (IL); 2003: 196-7.
503	29 33.Modeer T, Wondimu B. Periodontal diseases in children and adolescents. Dent Clin
504	North Am 2000;44(3):633-58.
505	30 34.Grossi SG, Zambon JJ, Ho AW, et al. Assessment of risk for periodontal disease. I. Risk
506	indicators for attachment loss. J Periodontol 1994;65(3):260-7.
507	31 35.Grossi SG, Genco RJ, Machtei EE, et al. Assessment of risk for periodontal disease. II.
508	Risk indicators for alveolar bone loss. J Periodontol 1995;66(1):23-9.
509	2024 Litering IC Device and the description of infections and the imported third meday I
	3236.Litonjua LS. Pericoronitis, deep fascial space infections, and the impacted third molar. I
510	Philipp Dent Assoc 1996;47(4):43-7.
510 511	 32<u>56</u>.Litonjua LS. Pericoronitis, deep fascial space infections, and the impacted third molar. J Philipp Dent Assoc 1996;47(4):43-7. 33<u>37</u>.American Academy of Periodontology. Periodontal therapy. J Periodontol

- 513 34<u>38</u>.Waller JH, Malden N. Rapid cystic involvement of a lower third molar. Dent Update
 514 1999;26(4):166-7.
- 515 3539. American Academy of Pediatric Dentistry. Guideline on pediatric oral surgery. Pediatr
 516 Dent 20052008;2730(suppl):158-64205-11.
- 517 36.40. American Academy of Pediatric Dentistry. Treatment of temporomandibular
 518 disorders in children: Summary statements and recommendations. J Am Dent Assoc
 519 1990;120(3):265-9.
- 520 37<u>41</u>.Riolo ML, tenHave TR, Brandt D. Clinical validity of the relationship between TMJ
 521 signs and symptoms in children and youth. J Dent Child 1988;55(2):110-3.
- 522 3842. Alamoudi N, Farsi N, Salako N, Feteih R. Temporomandibular disorders among school
 523 children. J Clin Pediatr Dent 1998;22(4):323-9.
- 524 3943.Nydell A, Helkimo M, Koch G. Craniomandibular disorders in children: A critical
 525 review of the literature. Swed Dent J 1994;18(5):191-205.
- 526 4044.National Institutes of Health. Management of Temporomandibular Disorders.
 527 Bethesda, Md; NIH Techno Assess Statement; 1996:1-31. Available at:
- 528 <u>"http://consensus.nih.gov/ta/018/018_statement.htm". Accessed February 7, 2005.</u>
 529 <u>National Institutes of Health Technology Assessment Conference Statement. J Am Dent</u>
 530 Assoc 1996; 127(11): 1595-1606.
- 4145.Skeppar J, Nilner M. Treatment of craniomandibular disorders in children and young
 adults. J Orofac Pain 1993;7(4):362-9.
- 4246.Garg AK. Treatment of congenitally missing maxillary incisors: Orthodontics, bone grafts, and osseointegrated implants. Dent Implantol Update 2002;13(2):9-14.
- 535 4347.Wexler G. Missing upper lateral incisors: Orthodontic considerations in young
 536 patients. Ann R Australas Coll Dent Surg 2000;15:136-40.
- 537 44<u>48</u>.Richardson G, Russell KA. Congenitally missing maxillary incisors and orthodontic
 538 treatment considerations for the single tooth implant. J Can Dent Assoc 2001;67(1):25-8.
- 539 4549. American Academy of Pediatric Dentistry. Guideline on management of the
 540 developing dentition and occlusion in pediatric dentistry. Pediatr Dent
 541 20052009;27(suppl):143-55PENDING.
- 542 46<u>50</u>.Chaushu S, Sharabi S, Becker A. Dental morphologic characteristics of normal versus
 543 delayed developing dentitions with palatally displaced canines. Am J Orthod
 544 Dentofacial Orthop 2002;121(4):339-46.
- 545 47<u>51</u>.Kojima R, Taguchi Y, Kabayashi H, Noda T. External root resorption of the maxillary
 546 permanent incisors caused by ectopically erupting canines. J Clin Pediatr Dent 2002;
 547 26(2):193-7.
- 548 48<u>52</u>.Ericson S, Kurol PJ. Resorption of incisors after ectopic eruption of maxillary canines.
 549 Angle Orthod 2000; 70(6):415-23.
- 4953.Shapira Y, Borell G, Kuftinec MM. Bringing impacted mandibular second premolars
 into occlusion. J Am Dent Assoc 1996;127(7):1075-8.
- 552 5054.Kurol J. Early treatment of tooth eruption disturbances. Am J Orthod Dentofacial
 553 Orthop 2002;121(6):588-91.
- 554 5155.Rocha MJdC, Cardoso M. Traumatized permanent teeth in Brazilian children assisted
 555 at the Federal University of Santa Catarina, Brazil. Dent Traumatol 2001;17(6):245-9.
- 556 5256.deFranca Caldas A Jr, Burgos MEA. A retrospective study of traumatic dental injuries
 557 in a Brazilian dental trauma clinic. Dent Traumatol 2001;17(6):250-3.

- 558 5357.Skaare AB, Jacobsen I. Dental injuries in Norwegians aged 7-18 years. Dent Traumatol
 2003;19(2):67-71.
- 560 54<u>58</u>. Tapias MA, Jiménez-García R, Lamas F, Gil AA. Prevalence of traumatic crown fractures to permanent incisors in a childhood population: Mostoles, Spain. Dent Traumatol 2003;19(3):119-22.
- 553 5559.Gassner R, Bösch R, Tuli T, Emshoff R. Prevalence of dental trauma in 6,000 patients
 with facial injuries: Implications for prevention. Oral Surg Oral Med Oral Pathol Oral
 Radiol Endod 1999;87(1):27-33.
- 566 5660. Tesini DA, Soporowski NJ. Epidemiology of orofacial sports-related injuries. Dent Clin
 567 North Am 2000;44(1):1-18.
- 568 <u>5761</u>.Ranalli DN. Prevention of sport-related dental traumatic injuries. Dent Clin North Am
 2000;44(1):19-33.
- 570 5862.Ranalli DN. A sports dentistry trauma control plan for children and adolescents. J
 571 Southeast Soc Pediatr Dent 2002;(8):8-9.
- 572 <u>5963</u>.Sarrett DC. Tooth whitening today. J Am Dent Assoc 2002;133(11):1535-8.
- 573 6064. Donly KJ. The adolescent patient: Special whitening challenges. Compend Contin Educ
 574 Dent 2003;24(4A):390-6.
- 575 6165.Li Y. Tooth bleaching using peroxide containing agents: Current status of safety issues.
 576 Compend Continu Educ Dent 1998;19(8):783-96.
- 577 6266. American Academy of Pediatric Dentistry. Policy on dental bleaching for child and
 578 adolescent patients. Pediatr Dent 2004200?;26(suppl):45-7PENDING.
- 579 6367.US Dept of Health and Human Services. Preventing Tobacco Use Among Young
 580 People: Report of the Surgeon General. Atlanta, Ga: US Dept of Health and Human
 581 Services, Public Health Service, CDC, National Center for Chronic Disease Prevention
 582 and Health Promotion, Office on Smoking and Health; 1994.
- 583 6468.CDC. Tobacco use among high school students-United States, 1997. MMWR Morb
 584 Mortal Weekly Rep 1998;47(12):229-33.
- 585 6569.Tomar SL, Winn DM, Swango PA, Giovino GA, Kleinman DV. Oral mucosal smokeless
 586 tobacco lesions among adolescents in the United States. J Dent Res 1997;76(6):1277-86.
- 587 6670. Audrain-McGovern J, Rodriguez D, Tercyak KP, Cuevas J, Rodgers K, Patterson F.
 588 Identifying and characterizing adolescent smoking trajectories. Cancer Epidemiol
 589 Biomarkers Prev 2004;13(12):2023-34.
- 6771.Zullig KJ, Valois RF, Huebner ES, Drane JW. Evaluating the performance of the Centers
 for Disease Control and Prevention core health-related quality of life scale with
 adolescents. Public Health Rep 2004;119(6):577-84.
- 593 6872.Johnson CC, Myers L, Webber LS, Boris NW. Profiles of the adolescent smoker: Models
 594 of tobacco use among 9th grade high school students; Acadiana Coalition of Teens
 595 against Tobacco (ACTT). Prev Med 2004;39(3):551-8.
- 596 6973.American Dental Association. Summary of policy and recommendations regarding
 597 tobacco: 1964-present. ADA Resolution 1H-1992. In: ADA Transactions 1992. Chicago,
 598 Ill: ADA; 1993:598.
- 599 7074. American Cancer Society, National Cancer Institute, National Institutes of Health. How
 600 to Help Your Patients Stop Using Tobacco: A National Cancer Institute Manual for the
 601 Oral Health Team. Bethesda, Md: National Institutes of Health, US Dept of Health and
 602 Human Services, Public Health Service; 1998. NIH publication No. 98-3191.

603 7475. American Academy of Pediatric Dentistry. Policy on tobacco use. Pediatr Dent 604 20042008;2730(suppl):42-353-5. 605 7276. US Dept of Health and Human Services Administration for Children and Families. 606 Toward a Blueprint for Youth: Making Positive Youth Development a National Priority. 607 Rockville, Md: US Department of Health and Human Services; 2002. Available at: 608 "http://www.acf.dhhs.gov/programs/fysb/vouthinfo/blueprint2.htm". Accessed 609 February 7, 2005. 610 Larson RW. Toward a Psychology of Positive Youth Development. American 611 Psychologist. 2000;55(1): 170-83. 612 7377. Christensen GJ. Oral care for patients with bulimia. J Am Dent Assoc 613 2002;133(12):1689-91. 614 7478.Cortes MI, Marcenes W, Sheiham A. Impact of traumatic injuries to the permanent 615 teeth on the oral health-related quality of life in 12- to 14-year-old children. Community 616 Dent Oral Epidemiol 2002;30(3):193-8. 617 7579.Gassner R, Tuli T, Hächl O, Moreira R, Ulmer H. Craniomaxillofacial trauma in 618 children: A review of 3,385 cases with 6,060 injuries in 10 years. J Oral Maxillofac Surg 619 2004;62(4):399-407. 620 7680.Barnett F. Prevention of sports-related dental trauma: The role of mouthguards. Pract 621 Proced Aesthet Dent 2003;15(5):391-4. 622 7781. American Dental Association. Statement on intraoral/perioral piercing. Available at: 623 "http://www.ada.org/prof/recoveries/positions/statements/piercings.asp.html". 624 Accessed February 7, 2005. 625 American Academy of Pediatric Dentistry. Policy on intraoral and perioral piercing. 626 Pediatr Dent 2008;30(suppl):56-7. 627 7882.Boardman R, Smith RA. Dental implications of oral piercing. J Calif Dent Assoc 628 1997;25(3):200-7. 629 7983. American Academy of Pediatric Dentistry. Guideline on record-keeping. Pediatr Dent 630 20042007;2630(suppl):134-9226-33. 631 8084. American Academy of Pediatric Dentistry. Guideline on informed consent. Pediatr 632 Dent 20052008;2730(suppl):182-3234-35. 633 8185. American Academy of Pediatric Dentistry. Definition of persons with special 634 healthcare needs. Pediatr Dent 20042009;26(suppl):15PENDING. 635 86. McDonald JL, Jr., Nutritional considerations for the dental patient. In: Dentistry 636 for the Child and Adolescent. 8th ed. St Louis, Mo: Mosby Inc;2004:257-69. 637 87. Macgegor ID, Balding JW: Self-esteem as a predictor of tooth brushing behavior 638 in young adolescents, J Clin Periodontal 18:312-16,1991 639 8288. American Academy of Pediatric Dentistry. Guideline on management of persons with 640 special health care needs. Pediatr Dent 20042008;2630(suppl):77-80107-11.

1 Guideline on Pediatric Oral Surgery

2 3 Originating Council 4 Council on Clinical Affairs 5 6 **Review Council** 7 Council on Clinical Affairs 8 9 Adopted 10 2005 11 12 Revised 13 2010 14

15 Purpose

16 The American Academy of Pediatric Dentistry (AAPD) intends this guideline to define,

- 17 describe clinical presentation, and set forth general criteria and therapeutic goals for
- 18 common pediatric oral surgery procedures that have been presented in considerably more
- 19 detail in textbooks and the dental/medical literature.
- 20

21 Methods

- 22 This guideline is <u>an update of the previous document adopted in 2005. It is based on a</u>
- 23 review of the current dental and medical literature related to pediatric oral surgery-,
- 24 including a systemic literature search of the A-MEDLINE/Pubmed electronic database with
- 25 <u>the following TERMS:</u> search was conducted using the terms "pediatric", "oral surgery",
- 26 <u>"extraction",</u> "odontogenic infections", "impacted canines", "third molars",
- 27 "supernumerary teeth", "mesiodens", "mucocele", "eruption cyst", "eruption hematoma",
- 28 "attached frenum", "ankyloglossia", "gingival keratin cysts", "Epstein pearls", "Bohn's
- 29 nodules", "congenital epulis of newborn", "dental lamina cysts", "natal teeth", and
- 30 "neonatal teeth". Fields: All Fields; Limits: within the last ten years; humans; English;
- 31 clinical trials. 7761 articles matched these criteria. Papers for review were chosen from this
- 32 <u>list and from references within selected articles</u>. When data did not appear sufficient or
- 33 were inconclusive, recommendations were based upon expert and/or consensus opinion by
- 34 experienced researchers and clinicians. <u>Also In addition</u>, the manual Parameters and

- **35** Pathways: Clinical Practice Guidelines for Oral and Maxillofacial Surgery,¹ developed by
- 36 the American Association of Oral and Maxillofacial Surgeons (AAOMS), was consulted.
- 37

38 Background

- **39** Surgery performed on pediatric patients involves a number of special considerations unique
- 40 to this population. Several critical issues deserve to be addressed. These include:
- 41 1. preoperative evaluation;
- 42 a. medical;
- 43 b. dental;
- 44 2. behavioral considerations;
- 45 3. growth and development;
- 46 4. developing dentition;
- 47 5. pathology;
- 48 6. perioperative care.
- 49

50 **Preoperative evaluation**

- 51 *Medical*
- 52 Important considerations in treating a pediatric patient include obtaining a thorough
- 53 medical history, obtaining appropriate medical and dental consultations, anticipating and
- 54 preventing emergency situations, and being prepared to treat emergency situations.^{2,2}
- 55

56 Dental

- 57 It is important to perform a thorough clinical and radiographic preoperative evaluation of
- 58 <u>the dentition as well as extraoral and intraoral soft tissues</u>.^{2,43} Radiographs <u>can include</u>
- 59 intraoral films and extraoral imaging often include 1 or more intraoral films and may
- 60 include extraoral imaging if the area of interest extends beyond the dentoalveolar complex.

61

62 Behavioral considerations

- **63** Behavioral guidance of children in the operative and perioperative periods presents a
- 64 special challenge. Many children benefit from modalities beyond local anesthesia, nitrous

- 65 <u>oxide-oxygen inhalation</u> and oral premedication sedation to control their anxiety.^{2,5}
- Anesthetic <u>mM</u>anagement of children <u>under deep sedation or general anesthesia</u> requires
 extensive training and expertise.^{2,6}
- 68 Special attention should be given to the assessment of the social, emotional, and
- 69 psychological status of the pediatric patient prior to surgery.⁴⁷ Children have many
- 70 unvoiced fears concerning the surgical experience, and their psychological management
- 71 requires that the dentist be cognizant of their emotional status. Answering questions
- 72 concerning the surgery is important and should be done in the presence of the parent. The
- 73 dentist also should obtain informed consent⁵⁸ prior to the procedure.
- 74

75 Growth and development

- 76 The potential for adverse effects on growth from injuries and/or surgery in the oral and
- 77 maxillofacial region markedly increases the potential for risks and complications in the
- 78 pediatric population. Traumatic injuries involving the maxillofacial region can affect
- 79 growth<u>, development</u>, and function adversely. For example, injuries to the mandibular
- 80 condyle may not only result in restricted growth, but also limit mandibular function as a
- 81 result of ankylosis. Surgery for acquired, congenital, or developmental malformations may,
- 82 in itself, affect growth adversely. This commonly is <u>commonly</u> seen in the cleft patient, for
- 83 example, where palatal scarring following primary palatal repair may result in maxillary
- 84 growth abnormalities constriction.²
- 85

86 Developing dentition

- 87 Surgery involving the maxilla and mandible of young patients is complicated by the
- 88 presence of developing tooth follicles. Alteration or deviation from standard treatment
- 89 modalities may be necessary to avoid injuring the follicles. ⁹ For example, distraction
- 90 osteogenesis can be a successful treatment option for craniofacial abnormalities in pediatric
- 91 patients. Nevertheless, this technique can be associated with long-term dental sequelae,
- 92 including damage to developing teeth, genesis of dentigerous cysts secondary to placement
- 93 of pins in unerupted tooth follicle spaces, and subsequent malocclusion issues.⁶²-To
- 94 minimize the <u>negative</u> effects of surgery on the developing dentition, careful planning using

- 95 radiographs, tomography¹⁰, cone beam computed tomography¹¹, tomograms, and/or 3-D
- 96 imaging techniques is necessary to provide valuable information to assess the presence,
- 97 absence, location, and/or quality of individual crown and root development.⁹
- 98

99 Pathology

- 100 Primary and reconstructive management of tumors in children is affected by anatomical and
- 101 physiological differences from those of adult patients. Tumors generally grow faster in
- 102 pediatric patients and are less predictable in behavior. The same physiological factors that
- 103 affect tumor growth, however, can play a favorable role in healing following primary
- 104 reconstructive surgery. Pediatric patients are more resilient and heal more rapidly than their
- 105 adult counterparts.²
- 106

107 Perioperative care

- 108 Metabolic management of children following surgery frequently is more complex than that
- 109 of adults. Special consideration should be given to caloric intake₂ as well as fluid and
- 110 electrolyte management and blood replacement. Comprehensive management of the
- 111 pediatric patient following extensive oral and maxillofacial surgery usually is best
- accomplished in a facility that has the expertise and experience in the management of young
- 113 patients (ie, a children's hospital).2.3
- 114

115 Recommendations

116 Odontogenic infections

117 In children, odontogenic infections may involve more than 1 tooth and usually are due to

- 118 carious lesions, periodontal problems, or <u>a history of trauma.^{7,812,13}</u> <u>Untreated odontogenic</u>
- 119 infections can lead to pain, abscess and cellulitis. As a consequence of this, Prompt
- 120 treatment of infections is important, as children are prone to dehydration especially if they
- 121 are not eating well due to pain and malaise. <u>Prompt treatment of the source of infection is</u>
- 122 important in order to control pain and prevent the spread of infection.
- 123 With upper face infections to the upper portion of the face, patients usually complain of
- 124 facial pain, fever, and inability to eat or drink. Care must be taken to rule out sinusitis, as

- 125 symptoms may mimic an odontogenic infection. Occasionally in upper face infections, it
- 126 may be difficult to find the true cause. Infections of the lower face usually involve pain,
- swelling, and trismus.⁷¹² They frequently are frequently associated with teeth, skin, local
- 128 lymph nodes, and salivary glands.⁷¹² In lower face swelling, dental infection is diagnosed
- 129 most frequently Swelling of the lower face has been more commonly associated with dental
- 130 <u>infections</u>.⁹¹⁴
- 131 Most odontogenic infections are not serious and can be managed easily. Treatment
- 132 involves with pulp therapy, extraction, or incision and drainage.² Infections of odontogenic
- 133 origin with systemic manifestations (eg, elevated temperature of 102° to 104°F, facial
- 134 cellulitis, difficulty in breathing or swallowing, fatigue, nausea) require antibiotic therapy.
- 135 Severe but rare complications of odontogenic infections include cavernous sinus thrombosis
- 136 and Ludwig's angina.^{2,712} These conditions can be life threatening and may require
- 137 immediate hospitalization with intravenous antibiotics, incision and drainage, and
- 138 referral/consultation with an oral and maxillofacial surgeon. 2,712
- 139
- 140 Extraction of erupted primary teeth
- 141 *Maxillary and mandibular anterior teeth*
- 142 Most primary and permanent maxillary and mandibular central incisors, lateral incisors,
- 143 and canines all have conical single roots. In most cases, extraction of anterior teeth is
- 144 <u>accomplished with a rotational movement, due to their single root anatomies.² However,</u>
- 145 there have been reported cases of accessory roots observed in primary canines.¹⁵⁻¹⁷
- 146 <u>Radiographic examination is helpful to identify differences in root anatomy prior to</u>
- 147 <u>extraction.¹⁵⁻¹⁷ Care should be taken to avoid placing any force on adjacent teeth that could</u>
- 148 <u>become luxated or dislodged easily due to their root anatomy.</u>
- 149
- 150 Maxillary and mandibular molars
- 151 Primary molars have roots that are smaller in diameter and more divergent than permanent
- 152 molars. Root fracture in primary molars is not uncommon due to these characteristics and as
- 153 <u>well as the potential weakening of their roots caused by the eruption of their permanent</u>
- 154 successors.² To avoid inadvertent extraction, or trauma to of the permanent

- successor, consideration should be given to careful evaluation of the relationship of the
 primary roots to the permanent successor crown succedaneous developing tooth should be
- 157 <u>completed</u>. Primary molars with roots encircling the successor's crown may need to be
- 158 sectioned to protect the permanent tooth's location.²
- 159 Molar extractions are accomplished by using slow continuous palatal/lingual and
- 160 buccal force allowing for the expansion of the alveolar bone to accommodate the divergent
- 161 roots and reduce the risk of root fracture.² When extracting mandibular molars, care should
- 162 be taken to support the mandible to protect the temporomandibular joints from injury.²
- 163
- 164 *Maxillary and mandibular anterior teeth*
- 165 Primary and permanent maxillary and mandibular central incisors, lateral incisors, and
- 166 canines all have conical single roots. Extraction of anterior teeth is accomplished with a
- 167 rotational movement, due to their single root anatomies.² Care should be taken to avoid
- 168 placing any force on adjacent teeth that could become luxated or dislodged easily due to
- 169 their root anatomy.
- 170

171 *Fractured primary tooth roots*

- 172 The dilemma to consider when treating a fractured primary tooth root is that removing the
- 173 root tip may cause damage to the succedaneous tooth, while leaving the root tip may
- 174 increase the chance for postoperative infection and delay eruption of the permanent
- 175 successor.² Use of appropriate radiographs can aide assist in the decision process. The
- 176 literature suggests that, if the <u>fractured tooth</u>-root <u>tip</u> can be removed easily, it should be
- 177 removed.² If the root <u>tip</u> is very small, located deep in the socket, situated in close proximity
- 178 to the permanent successor, or unable to be retrieved after several attempts, it is best left to
- 179 be resorbed.²
- 180

181 Unerupted and impacted teeth

- **182** *Impacted canines*
- 183 <u>MPermanent maxillary</u> canines are second to third molars in frequency of impaction.¹⁰¹⁸
- 184 Early detection of an ectopically erupting canine <u>through visual inspection</u>, palpation and
185 radiographic examination is important to minimize such an occurrence.¹⁹ Panoramic and 186 periapical films are useful in locating potentially ectopic canines.²⁰ Routine evaluation of 187 patients in mid-mixed dentition should involve identifying signs such as lack of canine 188 bulges, asymmetry in pattern of exfoliation and eruption of canines and abnormal 189 angulation or ectopic eruption of developing permanent cuspids in a radiograph.¹⁹ When 190 the cusp tip of the permanent canine is just mesial to or overlaying the distal half of the long 191 axis of the root of the permanent lateral incisor, canine palatal impaction usually occurs.¹¹²⁰ 192 Extraction of the primary canines is the treatment of choice when malformation or ankylosis 193 is present, when the risk of resorption of the adjacent tooth is evident or when trying to 194 correct palatally impacted canines, provided there are normal space conditions and no 195 incisor resorption.^{10,12,18,21-23} One study showed that 78% of ectopically-erupting permanent 196 canines normalized within 12 months after removal of the primary canines; 64% 197 normalized when the starting canine position overlapped the lateral incisor by more than 198 half of the root and 91% normalized when the starting canine position overlapped the lateral 199 incisor by less than half of the root.¹⁰¹⁸ If no improvement in canine position occurs in a year, 200 surgical and/or orthodontic treatment were suggested.^{10,18,23} <u>Although a Cochrane review</u> 201 yielded a lack of randomized controlled clinical studies to support extraction of primary 202 canines to facilitate eruption of ectopically erupting permanent maxillary canines,²¹ the 203 literature suggests that this can be considered to minimize complications resulting from 204 impacted canines. Consultation between the practitioner and an orthodontist may be useful 205 in the final treatment decision. 206

00**7** ----

207 *Third molars*

208 Panoramic or periapical radiographic examination is indicated in late adolescence to assess

- the presence, position, and development of third molars.³⁴ AAOMS recommends that a
- 210 decision to remove or retain third molars should be made before the middle of the third
- 211 decade.¹ Little controversy surrounds their removal when pathology (eg, cysts, or tumors,
- 212 nonrestorable or recurrent caries, periodontal disease, infection, or pericoronitis, detrimental
- 213 changes of adjacent teeth or bone) is associated and/or the tooth is malpositioned or
- 214 nonfunctional (ie, an unopposed tooth).^{1,13,24-26} A systematic review of research literature

215 from 1984 to 1999 concluded there is no reliable evidence to support the prophylactic 216 removal of disease-free impacted third molars.¹³ Although prophylactic removal of all 217 impacted or unerupted disease-free third molars is not indicated, consideration should be 218 given to removal by the third decade when there is a high probability of disease or 219 pathology and/or the risks associated with early removal are less than the risks of later 220 removal.^{1,24-2613-15} Removing the third molars prior to complete root formation may be 221 surgically prudent.¹ The American Association of Oral and Maxillofacial Surgeons 222 performed an age-related third molar study among board-certified oral maxillofacial 223 surgeons in 2001.²⁵ They concluded that third molar removal in adults is safe with 224 minimal complications and negative effects on the patient's quality of life. The report 225 showed that mandibular third molars showed more pathology or abnormalities. All 226 intraoperative complications (eg, nerve injury, unexected hemorrhage, unplanned 227 transfusion or parenteral drugs, compromised airway, fracture and other injuries to adjacent 228 tooth/structures) occurred at a frequency less than 1%.²⁵ Excluding alveolar osteitis, 229 postoperative complications (eg, paresthesia, infection, trismus, hemorrage, etc) were 230 similarly low.^{25,26} Factors that increase the risk for complications (eg, coexisting systemic 231 conditions, location of peripheral nerves, history of temporomandibular joint disease, 232 presence of cysts or tumors)^{25,26} and position and inclination of the molar in question²⁷ 233 should be assessed. The age of the patient is only a secondary consideration.²⁷ Referral to 234 an oral and maxillofacial surgeon for consultation and subsequent treatment may be 235 indicated. When a decision is made to retain impacted third molars, they should be 236 monitored for change in position and/or development of pathology, which may necessitate 237 later removal.

238

239 Supernumerary teeth

- 240 Supernumerary teeth and hyperdontia are terms to describe an excess in tooth number.
- 241 Supernumerary teeth are thought to be related to disturbances in the initiation and
- 242 proliferation stages of dental development.^{16, 17<u>15,28</u>} Although some supernumerary teeth
- 243 may be syndrome associated (eg, cleidocranial dysplasia) or of familial inheritance pattern,
- 244 most supernumerary teeth occur as isolated events.¹⁷¹⁵

Supernumerary teeth can occur in either the primary or permanent dentition.^{17-2015,29-31} In
33% of the cases, a supernumerary tooth in the primary dentition is followed by the
supernumerary tooth complement in the permanent dentition.^{21,2232,33} Reports in incidence of
supernumerary teeth can be as high as 3%, with the permanent dentition being affected 5
times more frequently than the primary dentition and males being affected twice as
frequently as females.^{17-1915,29,30}

251 Supernumerary teeth will occur ten times more in the maxillary arch versus the 252 mandibular arch.¹⁵ Approximately 90% of all single tooth supernumerary teeth are found in 253 the maxillary arch, with a strong predilection to the anterior region.^{17,20,15,31} The anterior 254 midline of the maxilla is the most common site, in which case the supernumerary tooth is 255 known as a mesiodens, while the second most common site is in the maxillary molar area 256 (paramolar).^{17-2015,29-31} A mesiodens can be suspected if there is an asymmetric eruption 257 pattern of the maxillary incisors, delayed eruption of the maxillary incisors with or without 258 any over-retained primary incisors, or ectopic eruption of a maxillary incisor.^{18,2229,33} The 259 diagnosis of a mesiodens can be confirmed with radiographs, including occlusal, periapical, 260 or panoramic films²³³⁴ or computed tomography^{10,11}. Three-dimensional information needed 261 to determine the location of the mesiodens or impacted tooth can be obtained by taking 2 262 periapical radiographs and by using either 2 projections taken at right angles to one another 263 or the tube shift technique (buccal object rule or Clark's rule)²³³⁴ or by using cone beam 264 computed tomography¹¹.

Complications of supernumerary teeth can include delayed and/or lack of eruption of the
 permanent tooth, crowding, resorption of adjacent teeth, dentigerous cyst formation,
 pericoronal space ossification, and crown resorption.^{24,2535,36} Early diagnosis and
 appropriately timed treatment are important in the prevention and avoidance of these
 complications.

Because only 25% of all mesiodens erupt spontaneously, surgical management usually is
<u>often</u> necessary.^{22,2633,37} A mesiodens that is conical in shape and is not inverted has a better
chance for eruption than a mesiodens that is tubercular in shape and is inverted.²⁵³⁶ The
treatment objective for a nonerupting permanent tooth-mesiodens is to minimize eruption
problems for the permanent incisors.²⁵³⁶ Surgical management will vary depending on the

size, shape, and number of supernumeraries and the patient's dental development.²⁵³⁶ The
treatment objective for a nonerupting primary tooth-mesiodens differs in that the removal of
these teeth usually is not recommended, as the surgical intervention may disrupt or damage
the underlying developing permanent teeth.²⁴³⁵ Erupted primary tooth mesiodens typically
are left to shed normally upon the eruption of the permanent dentition.²⁴³⁵

280 Extraction of an unerupted primary or permanent tooth-mesiodens is recommended 281 during the mixed dentition to allow the normal eruptive force of the permanent incisor to 282 bring itself into the oral cavity.²⁵³⁶ Waiting until the adjacent incisors have at least two 283 thirds root development will present less risk to the developing teeth but still allow 284 spontaneous eruption of the incisors.¹ In 75% of the cases, extraction of the mesiodens 285 during the mixed dentition results in spontaneous eruption and alignment of the adjacent 286 teeth.^{24,2735.38} If the adjacent teeth do not erupt within 6 to 12 months, surgical exposure and 287 orthodontic treatment may be necessary to aid their eruption.^{26,2837,39} The diagnosing dentist 288 may consider a multidisciplinary approach when treating difficult or complex cases.

289

290 Pediatric oral pathology

291 *Lesions of the newborn*

292 Oral pathologies occurring in newborn children include Epstein's pearls, dental lamina 293 cysts, Bohn's nodules, and congenital epulis. Epstein's pearls are common, found in about 294 75% to 80% of newborns.^{29 324043} They occur in the median palatal raphe area^{29 334044} as a 295 result of trapped epithelial remnants along the line of fusion of the palatal halves. 31,3342,44 296 Dental lamina cysts, found on the crests of the dental ridges, most commonly are seen 297 bilaterally in the region of the first primary molars.³¹⁴² They result from remnants of the 298 dental lamina. Bohn's nodules are remnants of salivary gland epithelium and usually are 299 found on the buccal and lingual aspects of the ridge, away from the midline.^{29,30,3240,41,43} 300 Epstein's pearls, Bohn's nodules, and dental lamina cysts typically present as asymptomatic 301 1 mm to 3 mm nodules or papules. They are smooth, whitish in appearance, and filled with 302 keratin.^{30,3141,42} No treatment is required, as these cysts usually disappear during the first 3 303 months of life.30,3341,44

Congenital epulis of the newborn, also known as granular cell tumor or Neumann's
tumor, is a rare benign tumor seen only in newborns. This lesion is typically a protuberant
mass arising from the gingival mucosa. It is most often found on the anterior maxillary
ridge.^{34,3545,46} Patients typically present with feeding and/or respiratory problems.³⁵⁴⁶
Congenital epulis has a marked predilection for females at 8:1 to 10:1.^{34-3645,47} Treatment
normally consists of surgical excision.^{34,3645,47} The newborn usually heals well, and no future
complications or treatment should be expected.

- **312** *Eruption cyst (eruption hematoma)*
- 313 The eruption cyst is a soft tissue cyst that results from a separation of the dental follicle from
- the crown of an erupting tooth.^{30,3741,48} Fluid accumulation occurs within this created folli-
- 315 cular space.^{29,32,37,3840,43,48,49} Eruption cysts most commonly are found in the mandibular molar
- 316 region.³⁷⁴⁸ Color of these lesions can range from normal to blue-black or brown, depending
- on the amount of blood in the cystic fluid. 29,32,37,3840,43,48,49 The blood is secondary to trauma.
- 318 If trauma is intense, these blood-filled lesions sometimes are referred to as eruption
- 319 hematomas. 29,32,37,3840,43,48,49
- Because the tooth erupts through the lesion, no treatment is necessary.^{29,32,37,3840,43,48,49} If
 the cyst does not rupture spontaneously or the lesion becomes infected, the roof of the cyst
 may be opened surgically.^{29,32,3740,43,48}
- 323

324 Mucocele

325 The mucocele is a common lesion in children and adolescents resulting from the rupture of

- 326 a minor salivary gland excretory duct, with subsequent spillage <u>leakage</u> of mucin into the
- 327 surrounding connective tissues that later may be surrounded in a fibrous capsule.^{30,32,39}
- 328 41,43,50-52 Most mucoceles are well-circumscribed bluish translucent fluctuant swellings
- 329 (although deeper and long-standing lesions may range from normal in color to having a
- whitish keratinized surface) that are firm to palpation.^{32,39,4143,50,52} Local mechanical trauma to
- the minor salivary gland is often the cause of rupture. $\frac{32,39 \cdot 4143,50 \cdot 52}{\text{At least 75\% of cases are}}$
- 332 found on the Mucoceles are most frequently observed on the lower lip, usually lateral to the

- 333 midline.³⁹⁵⁰ Mucoceles also can be found on the buccal mucosa, ventral surface of the
- tongue, retromolar region, and floor of the mouth (ranula).^{39,4050-52}
- 335 Superficial mucoceles and some mucoceles are short-lived lesions that burst
- spontaneously, leaving shallow ulcers that heal within a few days. 32,39 4143,50-52 Many lesions,
- 337 however, require treatment local surgical excision with the removal of adjacent minor
- 338 salivary glands to minimize the risk of recurrence. 32,39-4143,50-52
- 339

340 Structural anomalies

341 Maxillary frenum

342 A high or prominent maxillary frenum in children, although a common finding, is often a

- 343 concern, especially when associated with a diastema. A comparison of attached frena with
- 344 and without diastemas found no correlation between the height of the frenum attachment
- 345 and diastema presence and width.⁴²⁵³ Recent trends justify significantly fewer
- 346 frenectomies.⁴³ Treatment is necessary only suggested when the attachment exerts a
- 347 traumatic force on the gingiva <u>causing the papilla to blanch when the upper lip is pulled</u> or
- 348 <u>if</u> it causes a diastema to remain after eruption of the permanent canines.^{4354,55} Interference
- 349 with oral hygiene measures, esthetics and psychological reasons are contributing factors
- 350 <u>relating to treatment of the maxillary frenum.^{54,56} Treatment options can include</u>
- 351 <u>orthodontics, restorative dentistry, surgery or a combination of these.⁵⁴ When a diastema is</u>
- 352 present, the objectives for treatment involve managing both the diastema of permanent
- 353 <u>teeth and its cause while maintaining stable results in the future.⁵⁴ It is recommended that</u>
- 354 <u>Ttreatment should be is delayed until the permanent incisors and cuspids have erupted and</u>
- 355 the diastema has had an opportunity to close naturally.⁴³⁵⁵ In an older child, if a frenum is
- 356 present and the papilla blanches when the upper lip is pulled, removal can be indicated.⁴⁴
- 357 Again, <u>tT</u>he frenectomy should be performed only after orthodontic treatment is completed
- 358 and the diastema is closed as much as possible to achieve stable results.⁵⁴ When indicated, a
- 359 maxillary frenectomy is a fairly simple procedure and can be performed in the office setting.
- 360

361 Mandibular labial frenum

362	A high frenum sometimes can present on the labial aspect of the mandibular ridge. This is
363	most often seen in the central incisor area and frequently occurs in individuals where the
364	vestibule is shallow.4557 The mandibular anterior frenum, as it is known, occasionally inserts
365	into the free or marginal gingival tissue. ⁴⁵⁵⁷ Movements of the lower lip cause the frenum to
366	pull on the fibers inserting into the free marginal tissue, which, in turn, can lead to food and
367	plaque accumulation. ⁴⁵⁵⁷ Early treatment is- <u>can be considered indicated</u> to prevent
368	subsequent inflammation, recession, pocket formation, and possible loss of the alveolar
369	bone and/or tooth. ⁴⁵⁵⁷ However, if factors causing gingival/periodontal inflammation are
370	controlled, the degree of recession and need for treatment decreases.58
371	
372	Mandibular lingual frenum/ankyloglossia
373	Ankyloglossia is a developmental anomaly of the tongue characterized by a short, thick
374	lingual frenum resulting in limitation of tongue movement (partial ankyloglossia) or if the
375	tongue appears to be fused to the floor of the mouth (total ankyloglossia). ^{3344,58} The reported
376	prevalence is 0.1-10.7% of the population. ^{58,58} It can be categorized into 2 types. Total
377	ankyloglossia is rare and occurs when the tongue is completely fused to the floor of the
378	mouth. Partial ankyloglossia is variable and encompasses the remainder of the cases.
379	The exact cause of ankyloglosia remains unknown ⁵⁸ , however it has been associated with
380	problems with breast-feeding among neonates, 58-61 tongue mobility and speech, 54,58,62
381	malocclusion 58,63,64 and gingival recession.58 During breast-feeding, a short frenum can
382	cause ineffective latch, inadequate milk transfer and intake, and persistent maternal nipple
383	pain, all of which can affect feeding adversely.58-61 When indicated, frenuloplasty or
384	frenotomy may be a successful approach to facilitate breast-feeding, however, there is a lack
385	of reliable indications for or against treatment based on evidence-based literature. This
386	indicates that there is a need to standardize a classification system and justify parameters for
387	surgical correction of ankyloglossia among neonates.58-63 Limitations in tongue mobility and
388	speech pathology have been associated with ankyloglossia. ^{2,54,58,62} There has been varied
389	opinion among health care professionals regarding the correlation between ankyloglossia
390	and speech disorders.58,62 Frenuloplasty or frenectomy in conjunction with speech therapy
391	can be a treatment option to improves tongue mobility and speech. ⁶² Further evidence is

392	needed to determine the benefit of surgical correction of ankyloglossia in resolving speech
393	pathology. ⁵⁸ There is limited evidence to show an association between ankyloglossia and
394	Class III malocclusion. ^{58,64} Speculations have been made that the abnormal tongue position
395	may affect skeletal development.58,63,64 Although there are no clear recommendations in the
396	literature, a complete orthodontic workup, diagnosis, and treatment plan is necessary prior
397	to any surgical intervention. ⁵⁸ Reports have also been made regarding the association
398	between frenal attachment and gingival recession, however, further clinical evidence is
399	warranted to show a clear relationship between these two factors.58 Elimination of plaque
400	induced gingival inflammation can minimize gingival recession without any surgical
401	intervention. ⁵⁸
402	The significance and management of ankyloglossia are very controversial due to the lack
403	of evidence based studies to support frenotomy, frenectomy and frenuloplasty among
404	children and adults affected by ankyloglossia. 58,62 Studies have shown a difference in
405	treatment recommendations among speech pathologists, pediatricians, otolaryngologists,
406	and-lactation specialists, surgeons and dental specialists.46,4758-63,65 Most professionals,
407	however, will agree that there are certain indications for <u>these procedures</u> frenectomy,.63 A
408	short lingual frenum can inhibit tongue movement and create deglutition problems. 4765 If
409	there is no improvement in breast-feeding of a child with ankyloglossia after non-surgical
410	intervention, frenotomy may be indicated. ⁵⁸ Although there is a limited amount of evidence
411	in the literature to promote the timing, indication and type of surgical intervention,
412	<u>f</u> Frenectomy <u>or frenuloplasty</u> for functional <u>problems</u> <u>limitations</u> due to severe ankyloglossia
413	should be considered on an individual basis. ⁵⁸ If evaluation shows that function will may be
414	improved by surgery, treatment should be considered.4765,48
415	Ankyloglossia also can lead to problems with breast-feeding, speech pathology,
416	malocclusion, and periodontal health. During breast-feeding, a short frenum can cause
417	ineffective latch, inadequate milk transfer, and maternal nipple pain, all of which can affect
418	feeding adversely. ^{49,50} When indicated, frenuloplasty seems to be a successful approach to
419	facilitate breast-feeding.49 Although not as common as once thought, speech pathology has
420	been associated with ankyloglossia. ^{2,43,51,52} In such cases, frenectomy can be a treatment
421	option that improves tongue mobility and speech. ⁵² It should not be performed, however,

- 422 until an evaluation and therapy by a qualified speech therapist have been completed.²
- 423 Ankyloglossia also has been associated with Class III malocclusion.^{53,54} The abnormal
- 424 tongue position may affect skeletal development. Although there are no clear
- 425 recommendations, frenectomy in these cases should be deferred until a complete
- 426 orthodontic workup, diagnosis, and treatment plan have been completed and any necessary
- 427 referrals obtained.
- 428

429 <u>Frenectomy Techniques</u>

- 430 <u>Conventional frenectomy involves surgical incision, control of hemostasis and suturing of</u>
- 431 the wound.⁶⁶ Dressing placement or the use of antibiotics are not necessary.⁶⁶
- 432 <u>Recommendations include maintaining a soft diet, regular oral hygiene and analgesics as</u>
- 433 <u>needed. 66</u> Although there is minimal evidence based research available, the use of laser
- 434 <u>technology and electrosurgery for frenectomies have demonstrated a shorter operative</u>
- 435 working time, the ability to quickly control hemostasis, reduced pain and discomfort, less
- 436 post-operative complications (pain, swelling, infection, no need for suture removal), as well
- 437 <u>as increasing patient acceptance. ⁶⁶⁻⁶⁹ These procedures require skilled technique and patient</u>
- 438 <u>management^{66,69}.</u>
- 439

440 Natal and neonatal teeth

- 441 Natal and neonatal teeth can present a challenge when deciding on appropriate treatment.
- 442 Natal teeth have been defined as those teeth present at birth, and neonatal teeth are those
- that erupt during the first 30 days of life.⁵⁵⁷¹ The occurrence of natal and neonatal teeth is
- 444 rare; the incidence varies from 1:1,000 to 1:30,000.^{5670,71} The teeth most often affected are the
- 445 mandibular primary incisors.⁵⁷² In most cases, anterior natal and neonatal teeth are part of
- 446 <u>the normal compliment of the dentition. 70,71</u> Natal or neonatal molar teeth have also been
- 447 <u>identified in the posterior region and may be associated with systemic conditions or</u>
- 448 <u>syndromes (eg, Pfieffer syndrome or Histiocytosis X).⁷²⁻⁷⁴</u> Although many theories exist as
- to why the teeth <u>occurerupt prematurely</u>, currently no studies confirm a causal relationship
- 450 with any of the proposed theories. The superficial position of the tooth germ associated with
- 451 a hereditary factor seems to be the most accepted possibility.^{56,571}

- 452 If the tooth is not excessively mobile or causing feeding problems, it should be preserved
- and maintained in a healthy condition if at all possible.^{56,5871,75} Close monitoring is indicated
 to ensure that the tooth remains stable.
- 455 Riga-Fede disease is a condition caused by the natal or neonatal tooth rubbing the ventral
- 456 surface of the tongue during feeding and causing leading to ulceration.^{58,59,69,70} Failure to
- 457 diagnose and properly treat this lesion can result in dehydration and inadequate nutrient
- 458 intake for the infant.⁵⁸⁷⁵ Treatment should be conservative <u>and focus on creating round</u>,
- 459 <u>smooth incisal edges.</u>, if at all possible, consisting of smoothing rough incisal edges or
- 460 placing resin over the edge of the tooth to round it.⁵⁶⁻⁵⁹⁷¹⁻⁷⁶ If conservative treatment does not
- 461 correct the condition, extraction is the treatment of choice. $\frac{56,5971,76}{100}$
- 462 An important consideration when deciding to extract a natal or neonatal tooth is the
- 463 potential for hemorrhage. Extraction is contraindicated in newborns due to risk of
- 464 hemorrhage.⁶⁰⁷⁷ Unless the child is at least 10 days old consultation with the pediatrician
- 465 regarding adequate hemostasis may be indicated prior to extraction of the tooth.

466

467 References

- 468 1. American Association of Oral and Maxillofacial Surgeons. Parameters and Pathways:
 469 Clinical Practice Guidelines for Oral and Maxillofacial Surgery (AAOMS ParPath01). J
 470 Oral Maxillofac Surg 2001.
- 471 2. Wilson S, Montgomery RD. Local anesthesia and oral surgery in children. In: Pinkham
 472 JR, Casamassimo PS, Fields HW Jr, McTigue DJ, Nowak AJ, eds. Pediatric Dentistry:
 473 Infancy through Adolescence. 4th ed. St. Louis, Mo: Elsevier Saunders; 2005:454, 461.
- 474 <u>3. Kaban L, Troulis M. Preoperative Assessment of the Pediatric Patient. In: Pediatric Oral</u>
 475 <u>and Maxillofacial Surgery. Philadelphia, Pa: Saunders;2004:3-19.</u>
- 476 <u>43</u>. American Academy of Pediatric Dentistry. <u>Guideline Endorsements on prescribing</u>
 477 dental radiographs for infants, children, adolescents, and persons with special health
 478 care needs. Pediatr Dent 2009;31(6):250-2522005;27(suppl):185-6.
- 479 <u>5. Kaban L, Troulis M. Behavior management and conscious sedation of pediatric patients</u>
 480 <u>in the oral surgery office. In: Pediatric Oral and Maxillofacial Surgery. Philadelphia, Pa:</u>
 481 <u>Saunders;2004:75-85.</u>
- 482 <u>6. Kaban L, Troulis M. Deep sedation for pediatric patients. In: Pediatric Oral and</u>
 483 <u>Maxillofacial Surgery. Philadelphia, Pa: Saunders;2004:86-99.</u>
- 484 4<u>7</u>. McDonald RE, Avery DR, Dean JA. Examination of the mouth and other relevant
 485 structures. In: Dentistry for the Child and Adolescent. 8th ed. St. Louis, Mo: Mosby Co;
 486 2004:4.
- 487 58. American Academy of Pediatric Dentistry. Guideline on informed consent. Pediatr Dent
 488 2009;31(6):247-2492005;27(suppl):182-3.

489	69. Murray DJ, Chong DK, Sandor GK, Forrest CR. Dentigerous Cyst After Distraction
490	Osteogenesis of the Mandible. J Craniofac Surg. 2007;18(16):1349-52. Davies J, Turner S,
491	Sandy J. Distraction osteogenesis: A review. Br Dent J 1998;14(9):462-7.
492	10. White S, Pharoah M. Advanced Imaging. In: Oral Radiology: Principles and
493	Interpretation. 6th ed. St. Louis, Mo: Mosby Elseier; 2009:207-224.
494	11. White S, Pharoah M. Cone Beam Computed Tomography. In: Oral Radiology:
495	Principles and Interpretation. 6th ed. St. Louis, Mo: Mosby Elseier; 2009:225-243.
496	712. Kaban L, Troulis M. Infections of the maxillofacial region. In: Pediatric Oral and
497	Maxillofacial Surgery. Philadelphia, Pa: Saunders; 2004:171-186 1990:164-88 .
498	813. Seow W. Diagnosis and management of unusual dental abscesses in children. Aust
499	Dent J 2003;43(3):156-68.
500	914. Dodson T, Perrott D, Kaban L. Pediatric maxillofacial infections: A retrospective
501	study of 113 patients. J Oral Maxillofac Surg 1989;47(4):327-30.
502	15. Regezi J, Sciubba J, Jordan R. Abnormalities of teeth. In: Oral Pathology: Clinical-
503	Pathologic Correlations, 5th edition. St. Louis, MO: Saunders Elsevier; 2008:361-376.
504	16. Mochizuki K, Ohtawa Y, Kubo S, Machida Y, Yakushiji M. Bifurcation, birooted primary
505	canines: a case report. Int J Pediatr Dent. 2001;11(5):380-5.
506	17. Ott N, Ball R. Birooted primary canines: a report of three cases. Pedatr Dent
507	<u>1996;18(4):328-330.</u>
508	1018. Ericson S, Kurol J. Early treatment of palatally erupting maxillary canines by
509	extraction of the primary canines. Eur J Orthod 1988;10(4):283-95.
510	19. Richardson G, Russel K. A Review of Impacted Permanent Maxillary Cuspids -
511	Diagnosis and Prevention. J Can Dent Assoc 2000;66:497-501.
512	1120. Lindauer SJ, Rubenstein LK, Hang WM, Andersen WC, Isaason RJ. Canine impaction
513	identified early with panora-mic radiographs. J Am Dent Assoc 1992;123(3):91-2, 95-7.
514	Erratum in J Am Dent Assoc 1992;123(5):16.
515	21. Parkin N, Benson P, Shah A, Thind B, Marshman Z, Glenroy G, Dyer F. Extraction of
516	<u>primary (baby) teeth for unerupted palatally displaced permanent canine teeth in</u>
517	children. Cochrane Database Syst Rev. 2009;15(2):CD004621.
518	<u>122</u> .Fernandez E, Bravo LA, Canteras M. Eruption of the permanent upper canines: A
519	radiologic study. Am J Orthod Dentofacial Orthop 1998;113(4):414-20.
520	23. Baccetti T, Leonardi M, Armi P. A randomized clinical study of two interceptive
521	approaches to palatally displaced canines. Eur J Orthod 2008;30(4):381-5.
522	<u>1324</u> . Song F, O'Meara S, Wilson P, Goldner S, Kleijnen J. The effectiveness and cost-
523	effectiveness of prophylactic removal of wisdom teeth. Health Technol Assess
524	2000;4(1):1-55.
525	25. Haug R, Perrott D, Gonzalez M, Talwar R. The American Association of Oral and
526	Maxillotacial Surgeons Age-Related Third Molar Study. Oral Maxillotac Surg
527	<u>2005;63:1106-1114.</u>
528	<u>26. Pogrel M, Dodson T, Swift J, Bonine F, Rafetto L, Kennedy J, Malmquist J. White Paper</u>
529	on Third Molar Data. AAOMS. March 2007.
530	14. van der Schoot EA, Kuitert KB, van Ginkel FC, Prahl-Andersen B. Clinical relevance of
531	third permanent molars in relation to crowding after orthodontic treatment. J Dent
JJZ	1997;23(2):167-9.

522	1507 Almonduce Mensues N. Alexico Alexano E. Ovinteres Bergerello M. Berini Artes I
555	<u>1327</u> . <u>Almendros-Marques N, Alaejos-Algarra E, Quinteros-Dorgarello M, Berini-Aytes L,</u>
534	Gay-Escoda C. Factors influencing the prophylactic removal of asymptomatic impacted
535	lower third molars. Int J Oral Maxillotac Surg. 2008 Jan;37(1):29-35. Hicks EP. Third
536	molar management: A case against routine removal in adolescent and young
537	orthodontic patients. J Oral Maxillofac Surg 1999;57(7):831-6.
538	1628. Profitt W, Fields HW Jr, Ackerman J, Sinclair P, Thomas P, Tullock J. The etiology of
539	orthodontic problems. In: Contemporary Orthodontics. 2 nd ed. St. Louis, Mo: Mosby
540	Year Book, Inc; 2000:105-38.
541	17. Regezi J, Sciubba J. Abnormalities of teeth. In: Oral Pathology: Clinical-Pathologic
542	Correlations, Philadelphia, PaSt. Louis, MO: WB Saunders Elsevier: 1993:494-520.
543	$\frac{18}{29}$ Primosch R Anterior supernumerary teeth – Assessment and surgical intervention
544	in children Pediatr Dent 1981:3(2):204-15
545	1030 Dummett CO Ir Anomalies of the developing dentition In: Pinkham IR
546	<u>1750.</u> Duminieu CO JI. Anomanes of the developing dentition. In. I inknam JK,
540	through A delegence. 4th ed. Ct. Lewis, Mey Eleminiar Cours down, 2005-(1, 2
047 E40	10021 Navilla P. Danam D. Allan C. Banamad I. Almannalitica of the testh. In Ourland
548	$\frac{2031}{10}$. Neville B, Damm D, Allen C, Bouquot J. Abnormalities of the teeth. In: Oral and
549	Maxillofacial Pathology <u>, 3rd ed</u> . Philadelphia, Pa<u>St. Louis, MO</u>: WB Saunders<u>Elsevier</u>;
550	$\frac{19952009:77-11044-95}{1000000000000000000000000000000000000$
551	21 32. Taylor G. Characteristics of supernumerary teeth in the primary and permanent
552	dentition. Trans Br Soc Study Orthod 1970-71;57:123-8.
553	2233. American Academy of Pediatric Dentistry. Guideline on the management of the
554	developing dentition and occlusion in pediatric dentistry. Pediatr Dent 2009;31(6):196-
555	<u>2082005;27(suppl):143-55</u> .
556	234. Goaz P, White S, Pharoah M. Projection geometry. In: Oral Radiology: Principles and
557	Interpretation. 3 rd 6 th ed. St. Louis, Mo: Mosby Elsevier; 2009:46-521994:97-105.
558	2435. Neville B, Damm D, White D. Pathology of the teeth. In: Color Atlas of Clinical Oral
559	Pathology, 2 nd ed. Baltimore, Md: Williams & Wilkins; 2003:58-60.
560	2536. Christensen IR, Fields HW Ir, Treatment planning and management of orthodontic
561	problems. In: Pinkham IR, Casamassimo PS, Fields HW Ir, McTigue DI, Nowak AL eds
562	Pediatric Dentistry: Infancy through Adolescence 4thed St Louis Mo: Flsevier
563	Saunders: 2005:624-6
564	2637 Russell K Felwarczna M Mesiodens: Diagnosis and management of a common
565	<u>2057</u> . Russen R, Forwardzha M. Mesiodens. Diagnosis and management of a common
505	2729 Howard P. The uncommuted inciser. A study of the necton archive ementions history of
500	<u>2456</u> . Howard K. The unerupted incisor. A study of the postoperative eruptive history of
507	incisors delayed in their eruption by supernumerary teeth. Dent Pract Dent Rec
508	1967;17(9): 332-41.
569	2839. Giancotti A, Grazzini F, De Dominicis F, Komanini G, Arcuri C. Multidisciplinary
570	evaluation and clinical management of mesiodens. J Clin Pediatr Dent 2002;26(3):233-7.
5/1	<u>2940</u> . American Academy of Pediatric Dentistry. Dental development, morphology,
572	eruption and related pathologies. In: Nowak A J , <u>Casamassimo P,</u> ed. The Handbook : <u>of</u>
573	Pediatric Dentistry. 2nd3rd ed . Chicago, Il <u>: American Academy of Pediatric</u>
574	<u>Dentistry;19992007:7-279-28</u> .
575	<u>3041</u> . Flaitz CM. Differential diagnosis of oral lesions and developmental anomalies. In:
576	Pinkham JR, Casamassimo PS, Fields HW Jr, McTigue DJ, Nowak AJ, eds. Pediatric

- 577 Dentistry: Infancy through Adolescence. 4th ed. St. Louis, Mo: Elsevier Saunders;
 578 2005:18.
- 579 3142. Hays P. Hamartomas, eruption cysts, natal tooth, and Epstein pearls in a newborn. J
 580 Dent Child 2000;67(5):365-8.
- <u>4</u>32.Cameron AC, Widmer RP. <u>Pediatric Oo</u>ral <u>medicine and</u> pathology. In: Handbook of
 Pediatric Dentistry, <u>3rd ed</u>. <u>LondonPhiladelphia, PA</u>: Mosby <u>Elsevier</u>; <u>19972008</u>:<u>143-</u>
 78192-216</u>.
- 584 33<u>44</u>. Neville B, Damm D, Allen C<u>, Bouquot J</u>. Developmental defects of the oral and
 585 maxillofacial region. In: Oral and Maxillofacial Pathology. Philadelphia, PaSt. Louis,
 586 MO: WB Saunders Elsevier; 19952009:1-4326-27.
- 587 345. Lapid O, Shaco-Levey R, Krieger Y, Kachko L, Sagi A. Congenital epulis. Pediatrics
 588 2001;107(2):E22.
- 589 <u>3546</u>. Marakoglu I, Gursoy U, Marakoglu K. Congenital epulis: Report of a case. J Dent
 590 Child 2002;69(2):191-2.
- 591 3647. Neville B, Damm D, Allen C, Bouquot J. Soft tissue tumors. In: Oral and
 592 Maxillofacial Pathology. Philadelphia, PaSt. Louis, MO: WB Saunders Elsevier;
 593 19952009:537-538362-415.
- 594 37<u>48</u>. Neville B, Damm D, Allen C<u>, Bouquot J</u>. Odontogenic cysts and tumors. In: Oral and
 595 Maxillofacial Pathology. Philadelphia, PaSt. Louis, MO: WB Saunders Elsevier;
 596 19952009:678-691493-540.
- 597 3849. Regezi J, Sciubba J, Jordan R. Cysts of the oral region. In: Oral Pathology: Clinical 598 Pathologic Correlations. Philadelphia, PaSt. Louis, Mo: WB Saunders Elsevier;
 599 19932008:241-244322-61.
- 600 3950. Baurmash HD. Mucoceles and ranulas. J Oral Maxillofac Surg 2003;61:369–378.
 601 Neville B, Damm D, Allen C. Salivary gland pathology. In: Oral and Maxillofacial
 602 Pathology. Philadelphia, Pa. WB Saunders; 1995:322-61.
- 603 40<u>51</u>. Regezi J, Sciubba J, Jordan R</u>. Salivary gland diseases. In: Oral Pathology: Clinical604 Pathologic Correlations. Philadelphia, PaSt. Louis, Mo: WB Saunders Elsevier;
 605 19932008:179-182239-302.
- 606 41<u>52</u>. American Academy of Pediatric Dentistry. Oral pathology/oral
 607 medicine/syndromes. In: Nowak AJ, <u>Casamassimo P</u>, ed. The Handbook: <u>of</u> Pediatric
 608 Dentistry. 2nd3rd ed. Chicago, Il: <u>American Academy of Pediatric Dentistry</u>;
 609 19992007:2829-5553.
- 610 42<u>53</u>. Ceremello P. The superior labial frenum and midline diastema and their relation to
 611 growth and development of the oral structures. Am J Orthod Dentofacial Orthop
 612 1993;39(2):120-39.
- 613 <u>543</u>. <u>Gkantidis N, Kolokitha OE, Topouzelis N. Management of maxillary midline</u>
 614 <u>diastema with emphasis on etiology</u>. J Clin Ped Dent 2008;32(4):265-72. Griffen AL.
 615 Periodontal problems in children and adolescents. In: Pinkham JR, Casamassimo PS,
 616 Fields HW Jr, McTigue DJ, Nowak AJ, eds. Pediatric Dentistry: Infancy through
 617 Adolescence. 4th ed. St. Louis, Mo: Elsevier Saunders; 2005:417.
- 618 44<u>55.</u> Griffen AL. Periodontal problems in children and adolescents. In: Pinkham JR,
 619 <u>Casamassimo PS, Fields HW Jr, McTigue DJ, Nowak AJ, eds. Pediatric Dentistry:</u>
 620 <u>Infancy through Adolescence. 4th ed. St. Louis, Mo: Elsevier Saunders; 2005:417.</u>
 621 <u>Leonard M. The maxillary frenum and surgical treatment. Gen Dent 1998;46(6):614-7.</u>

622	4556. McDonald RE, Avery DR, Weddell JA. Acquired and developmental disturbances of
623	the teeth. In: McDonald RE, Avery DR, Dean JA, eds. Dentistry for the Child and
624	Adolescent. 8th ed. St. Louis, Mo: Mosby Co; 2004:142.
625	57. McDonald RE, Avery DR, Weddell JA. Gingivitis and periodontal disease. In:
626	McDonald RE, Avery DR, Dean JA, eds. Dentistry for the Child and Adolescent. 8th ed.
627	St. Louis, Mo: Mosby Co; 2004:440-41.
628	58. Suter VG, Bornstein MM. Ankyloglossia: Facts and Myths in Diagnosis and Treatment.
629	J Periodontol 2009;80:1204-1219.
630	59. Segal L, Stephenson R, Dawes M, Feldman P. Prevalence, diagnosis, and treatment of
631	ankyloglossia. Can Fam Physician 2007;53(6):1027-1033.
632	60. Ballard J, Auer C, Khoury J. Ankyloglossia: Assessment, incidence, and effect of
633	frenuloplasty on the breast-feeding dyad. Pediatrics 2002;110(5):e63.
634	61. Geddes D, Langton D, Gollow I, Jacobs L, Hartmann P, Simmer K. Frenulotomy for
635	Breastfeeding Infants With Ankyloglossia: Effect on Milk Removal and Sucking
636	Mechanism as Imaged by Ultrasound. Pediatrics 2008;122(1):e188-e194.
637	62. Kupietzky A, Botzer E. Ankyloglossia in the Infant and Young Child: Clinical
638	Suggestions for Diagnosis and Management. Pediatr Dent 2005;27(1):40-46.
639	63. Lalakea L, Messner A. Frenotomy and frenuloplasty: If, when and how. Operative
640	Techniques in Otolaryngology-Head and Neck Surgery 2002;13(1):93-97.
641	464. Neville B, Damm D, White D. Developmental disturbances of the oral and maxillofacial
642	<u>region. Color Atlas of Clinical Oral Pathology. 2nd ed. Baltimore, Md: Williams &</u>
643	Wilkins; 2003:10-1. Messner A, Lalakea M. Ankyloglossia: Controversies in management.
644	Int J Pediatr Otorhinolaryngol 2000;54(2-3):123-31.
645	4765. Lalakea M, Messner A. Ankyloglossia: Does it matter? Pediatr Clin North Am
646	2003;50(2):381-97.
647	66. Kaban L, Troulis M. Intraoral Soft Tissue Abnormalities. In: Pediatric Oral and
648	Maxillofacial Surgery. Philadelphia, Pa: Saunders;2004:147-153.
649	67. Shetty K. Trajtenberg C. Patel C. Streckfus C. Maxillary frenectomy using a carbon
650	dioxide laser in a pediatric patient: a case report. General Dentistry, 2008;56(1):60-3.
651	68. Kara C. Evaluation of Patient Perceptions of Frenectomy: A Comparison of Nd:YAG
652	Laser and Conventional Techniques. Photomedicine and Laser Surgery, 2008;26(2):147-
653	<u>152.</u>
654	69. Gontijo I, Navarro R, Haypek P, Ciamponi A, Haddad A. The Applications of Diode and
655	Er:YAG Lasers in Labial Frenectomy in Infant Patients. J Dent Child 2005;72:10-15.
656	48. Whight J. Tongue-tie. J Paediatr Child Health 1995;31(4):276-8.
657	49. Ballard J, Auer C, Khoury J. Ankyloglossia: Assessment, incidence, and effect of
658	frenuloplasty on the breast-feeding dyad. Pediatrics 2002;110(5):e63.
659	50. Messner A, Lalakea M, Aby J, Macmahon J, Bair E. Ankyloglossia: Incidence and
660	associated feeding difficulties. Arch Otolaryngol Head Neck Surg 2000;126(1):36-9.
661	51. García Pola M, Gonzalez García M, García Martin J, Gallas M, Leston J. A study of
662	pathology associated with short lingual frenum. J Dent Child 2002;69(1):59-62.
663	52. Messner A, Lalakea M. The effect of ankyloglossia on speech in children. Otolaryngol
664	Head Neck Surg 2002; 127(6):539-45.

- 53. Mukai S, Mukai C, Asaoka K. Congenital ankyloglossia with deviation of the epiglottis
 and larynx: Symptoms and respiratory function in adults. Ann Otol Rhinol Laryngol
 1993;102(8Pt1):620-4.
- 668 54. Neville B, Damm D, White D. Developmental disturbances of the oral and maxillofacial
 669 region. Color Atlas of Clinical Oral Pathology. 2nd ed. Baltimore, Md: Williams &
 670 Wilkins; 2003:10-1.
- 671 55<u>70</u>. Massler M, Savara BS. Natal and neonatal teeth: A review of the 24 cases reported in
 672 the literature. J Pediatr 1950;36(3):349-59. Cunha RF, Boer FA, Torriani DD, Frossard
 673 WT. Natal and neonatal teeth: Review of the literature. Pediatr Dent 2001;23(2):158-62.
- 674 5671. Leung A, Robson W. Natal Teeth: A Review. J Natl Med Assoc. 2006;98(2):226-8.
- 675 572. Zhu J, King D. Natal and neonatal teeth. J Dent Child 1995;62(2):123-8. Galassi MS,
- 676 Santos-Pinto L, Ramalho T. Natal maxillary primary molas: case report. J Clin Pediatr
 677 Dent 2004;29(1):41-44.
- 678 <u>73.</u> Alvarez MP, Crespi PV, Shanske AL. Natal molars in Pfeiffer syndrome type 3: a
 679 case report. J Clin Pediatr Dent. 1993;18(1):21-4.
- 680 74. Stein S, Paller A, Haut P, Mancini A. Langerhans Cell Histiocytosis Presenting int eh
- 681 Neonatal Period: A Retrospective Case Series. Arch Pediatr Adolesc Med. 2001;155:778-783.
- 5875. Slayton RL. Treatment alternatives for sublingual trauma-tic ulceration (Riga-Fede disease). Pediatr Dent 2000;22(5):413-4.
- 684 <u>5976</u>. Goho C. Neonatal sublingual traumatic ulceration (Riga-Fede disease): Report of
 685 cases. J Dent Child 1996;63(5):362-4.
- 686 6077. Rushmah M. Natal and neonatal teeth: A clinical and histological study. J Clin
 687 Pediatr Dent 1991;15(4):251-3.

1 2	Guideline on Oral and Dental Aspects of Child Abuse and Neglect
3	Originating Group
4	American Academy of Pediatrics Committee on Child Abuse and Neglect and the American
5	Academy of Pediatric Dentistry
6	
/ Q	Review Group
9	Academy of Pediatric Dentistry Council on Clinical Affairs
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14 15	2005
16	2000
17	Reaffirmed
18	<u>2010</u>
19 20	Abstract
21	In all 50 states, physicians and dentists are required to report suspected cases of abuse and
22	neglect to social service or law enforcement agencies. The purpose of this report is to review
23	the oral and dental aspects of physical and sexual abuse and dental neglect and the role of
24	physicians and dentists in evaluating such conditions. This report addresses the evaluation
25	of bite marks as well as perioral and intraoral injuries, infections, and diseases that may be
26	suspicious for child abuse or neglect. Physicians receive minimal training in oral health and
27	dental injury and disease and, thus, may not detect dental aspects of abuse or neglect as
28	readily as they do child abuse and neglect involving other areas of the body. Therefore,
29	physicians and dentists are encouraged to collaborate to increase the prevention, detection,
30	and treatment of these conditions.
31	
32	Physical abuse

33 Craniofacial, head, face, and neck injuries occur in more than half of the cases of child

34 abuse.¹⁻¹⁰ A careful and thorough intraoral and perioral examination is necessary in all cases

- 35 of suspected abuse and neglect. In addition, all suspected victims of abuse or neglect,
- 36 including children in state custody or foster care, should be examined carefully not only for

- 37 signs of oral trauma but also for caries, gingivitis, and other oral health problems. Some
- **38** authorities believe that the oral cavity may be a central focus for physical abuse because of
- 39 its significance in communication and nutrition.¹¹
- 40 Oral injuries may be inflicted with: instruments such as eating utensils or a bottle during
 41 forced feedings; hands; fingers; or scalding liquids or caustic substances.
- The abuse may result in: contusions, burns, or lacerations of the tongue, lips, buccal
 mucosa, palate (soft and hard), gingivae, alveolar mucosa, or frenum; fractured, displaced,
 or avulsed teeth; or facial bone and jaw fractures.
- In one study,¹² the lips were the most common site for inflicted oral injuries (54%),
 followed by the oral mucosa, teeth, gingivae, and tongue. Discolored teeth, indicating
 pulpal necrosis, may result from previous trauma.^{13,14} Gags applied to the mouth may result
 in bruises, lichenification, or scarring at the corners of the mouth.¹⁵
- 49 Some serious injuries of the oral cavity, including posterior pharyngeal injuries and 50 retropharyngeal abscesses, may be inflicted by caregivers with factitious disorder by proxy¹⁶ 51 to simulate hemoptysis or other symptoms requiring medical care; regardless of caregiver 52 motive, all inflicted injuries should be reported for investigation. Unintentional or accidental 53 injuries to the mouth are common and must be distinguished from abuse by judging 54 whether the history, including the timing and mechanism of injury, is consistent with the 55 characteristics of the injury and the child's developmental capabilities. Multiple injuries, 56 injuries in different stages of healing, or a discrepant history should arouse a suspicion of 57 abuse. Consultation with or referral to a knowledgeable dentist may be helpful.
- 58

59 Sexual abuse

- 60 Although the oral cavity is a frequent site of sexual abuse in children,¹⁷ visible oral injuries
- 61 or infections are rare. When oral-genital contact is suspected, referral to specialized clinical
- 62 settings equipped to conduct comprehensive examinations is recommended. The American
- 63 Academy of Pediatrics statement "Guidelines in the Evaluation of Sexual Abuse of
- 64 Children^{"18} provides information regarding these examinations.
- 65 Oral and perioral gonorrhea in prepubertal children, diagnosed with appropriate
 66 culture techniques and confirmatory testing, is pathognomonic of sexual abuse¹⁹ but rare

Council on Clinical Affairs 2009-10

Charge 1(e) G-Oral and Dental Aspects of Child Abuse and Neglect

67 among prepubertal girls evaluated for sexual abuse.²⁰ Pharyngeal gonorrhea is frequently 68 asymptomatic.²¹ When oral-genital contact is confirmed by history or examination findings, 69 universal testing for sexually transmitted diseases within the oral cavity is controversial; the 70 clinician should consider risk factors (eg, chronic abuse, perpetrator with a known sexually 71 transmitted disease) and the child's clinical presentation in deciding whether to conduct 72 such testing. Although human papillomavirus infection may result in oral or perioral warts, 73 the mode of transmission remains uncertain and debatable. Human papillomavirus 74 infections may be sexually transmitted through oral-genital contact, vertically transmitted 75 from mother to infant during birth, or horizontally transmitted through nonsexual contact 76 from a child or caregiver's hand to the genitals or mouth.²² 77 Unexplained injury or petechiae of the palate, particularly at the junction of the hard and 78 soft palate, may be evidence of forced oral sex.²³ As with all suspected child abuse or 79 neglect, when sexual abuse is suspected or diagnosed in a child, the case must be reported 80 to child protective services and/or law enforcement agencies for investigation.²⁴⁻²⁷ A 81 multidisciplinary child abuse evaluation for the child and family should be initiated. 82 Children who present acutely with a recent history of sexual abuse may require 83 specialized forensic testing for semen and other foreign materials resulting from assault. If a 84 victim provides a history for oral-penile contact, the buccal mucosa and tongue can be 85 swabbed with a sterile cotton-tipped applicator, then the swab can be air-dried and 86 packaged appropriately for laboratory analysis. However, specialized hospitals and clinics 87 equipped with protocols and experienced personnel are best suited for collecting such 88 material and maintaining a chain of evidence necessary for investigations. 89 90 **Bite marks**

Acute or healed bite marks may indicate abuse. Dentists trained as forensic odontologists
can assist physicians in the detection and evaluation of bite marks related to physical and
sexual abuse.²⁸ Bite marks should be suspected when ecchymoses, abrasions, or lacerations
are found in an elliptical or ovoid pattern. Bite marks may have a central area of ecchymoses
(contusions) caused by 2 possible phenomena: positive pressure from the closing of the teeth
with disruption of small vessels or negative pressure caused by suction and tongue

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97 thrusting. Bites produced by dogs and other carnivorous animals tend to tear flesh, whereas 98 human bites compress flesh and can cause abrasions, contusions, and lacerations but rarely 99 avulsions of tissue. An intercanine distance (ie, the linear distance between the central point 100 of the cuspid tips) measuring more than 3.0 cm is suspicious of an adult human bite.²⁹ 101 The pattern, size, contour, and color of the bite mark should be evaluated by a forensic 102 odontologist or a forensic pathologist if an odontologist is not available. If neither specialist 103 is available, a physician or dentist experienced in the patterns of child abuse injuries should 104 observe and document the bite mark characteristics photographically with an identification 105 tag and scale marker (eg, ruler) in the photograph. The photograph should be taken such 106 that the angle of the camera lens is directly over the bite and perpendicular to the plane of 107 the bite to avoid distortion. A special photographic scale was developed by the American 108 Board of Forensic Odontology (ABFO) for this purpose, as well as for documenting other 109 patterned injuries, and can be obtained from the vendor (ABFO No. 2 reference scale, 110 available from Lightening Powder Co Inc, Salem, Ore). Names and contact information for 111 ABFO certified odontologists can be obtained from the ABFO Web site (www.abfo.org). 112 In addition to photographic evidence, every bite mark that shows indentations should 113 have a polyvinyl siloxane impression made immediately after swabbing the bite mark for 114 secretions containing DNA. This impression will help provide a 3-dimensional model of the 115 bite mark. Written observations and photographs should be repeated daily for at least 3 116 days to document the evolution of the bite. Because each person has a characteristic bite 117 pattern, a forensic odontologist may be able to match dental models (casts) of a suspected 118 abuser's teeth with impressions or photographs of the bite. 119 Blood group substances can be secreted in saliva. DNA is present in epithelial cells from

the mouth and may be deposited in bites. Even if saliva and cells have dried, they should be collected using the double-swab technique. First, a sterile cotton swab moistened with distilled water is used to wipe the area in question, dried, and placed in a specimen tube. A second sterile dry cotton swab cleans the same area, then is dried and placed in a specimen tube. A third control sample should be obtained from an uninvolved area of the child's skin. All samples should be sent to a certified forensic laboratory for prompt analysis.³⁰ The chain

- 126 of custody must be maintained on all samples submitted for forensic analysis. Questions
- 127 regarding evidentiary procedure should be directed to a law enforcement agency.
- 128

129 Dental neglect

130 Dental neglect, as defined by the American Academy of Pediatric Dentistry,³¹ is the "willful 131 failure of parent or guardian to seek and follow through with treatment necessary to ensure 132 a level of oral health essential for adequate function and freedom from pain and infection." 133 Dental caries, periodontal diseases, and other oral conditions, if left untreated, can lead to 134 pain, infection, and loss of function. These undesirable outcomes can adversely affect 135 learning, communication, nutrition, and other activities necessary for normal growth and 136 development.³² Some children who first present for dental care have severe early childhood 137 caries (formerly termed "baby bottle" or "nursing" caries); caregivers with adequate 138 knowledge and willful failure to seek care must be differentiated from caregivers without 139 knowledge or awareness of their child's need for dental care in determining the need to 140 report such cases to child protective services.

141 Failure to seek or obtain proper dental care may result from factors such as family 142 isolation, lack of finances, parental ignorance, or lack of perceived value of oral health.³³ The 143 point at which to consider a parent negligent and to begin intervention occurs after the 144 parent has been properly alerted by a health care professional about the nature and extent of 145 the child's condition, the specific treatment needed, and the mechanism of accessing that 146 treatment.³³ Because many families face challenges in their attempts to access dental care or 147 insurance for their children, the clinician should determine whether dental services are 148 readily available and accessible to the child when considering whether negligence has 149 occurred.

150 The physician or dentist should be certain that the care-givers understand the 151 explanation of the disease and its implications and, when barriers to the needed care exist, 152 attempt to assist the families in finding financial aid, transportation, or public facilities for 153 needed services. Parents should be reassured that appropriate analgesic and anesthetic 154 procedures will be used to ensure the child's comfort during dental procedures. If, despite

- 155 these efforts, the parents fail to obtain therapy, the case should be reported to the
- 156 appropriate child protective services agency.^{31,33}
- 157

158 Conclusions

159 Pediatricians should be aware that physical or sexual abuse may result in oral or dental 160 injuries or conditions that sometimes can be confirmed by laboratory findings. Furthermore, 161 injuries inflicted by one's mouth or teeth may leave clues regarding the timing and nature of 162 the injury as well as the identity of the perpetrator. Pediatricians are encouraged to be 163 knowledgeable about such findings and their significance and to meticulously observe and 164 document them. When questions arise or when consultation is needed, a pediatric dentist or 165 a dentist with formal training in forensic odontology can ensure appropriate testing, 166

- diagnosis, and treatment.
- 167 Pediatric dentists and oral and maxillofacial surgeons, whose advanced education
- 168 programs include a mandated child abuse curriculum, can provide valuable information
- 169 and assistance to physicians about oral and dental aspects of child abuse and neglect. The
- 170 Prevent Abuse and Neglect Through Dental Awareness [also known as PANDA; telephone
- 171 (501) 661-2595 or e-mail Lmouden@healthyarkansas.com] coalition, which has trained
- 172 thousands of physicians, nurses, teachers, child care providers, dentists, and dental
- 173 auxiliaries, is another resource for physicians seeking information on this issue. Physician
- 174 members of multidisciplinary child abuse and neglect teams are encouraged to identify such
- 175 dentists in their communities to serve as consultants for these teams. In addition, physicians
- 176 with experience or expertise in child abuse and neglect can make themselves available to
- 177 dentists and to dental organizations as consultants and educators. Such efforts will
- 178 strengthen our ability to prevent and detect child abuse and neglect and enhance our ability
- 179 to care for and protect children.
- 180

181 References

- 182 1. Mouden LD, Bross DC. Legal issues affecting dentistry's role in preventing child abuse 183 and neglect. J Am Dent Assoc 1995;126:1173-80.
- 184 2. Schwartz S, Woolridge E, Stege D. The role of the dentist in child abuse. Quintessence 185 Int 1976;7:79-81.

- 186 3. Sognnaes RF, Blain SM. Child abuse and neglect. I: Diagnostic criteria of special interest to dentists [abstract]. J Dent Res 1979;58(special issue A):367.
- Donly KJ, Nowak AJ. Maxillofacial, neck, and dental lesions of child abuse. In: Reece
 RM, ed. Child Abuse: Medical Diagnosis and Management. Philadelphia, Pa: Lea &
 Febiger; 1994:150-66.
- 191 5. Baetz K, Sledziewski W, Margetts D, Koren L, Levy M, Pepper R. Recognition and management of the battered child syndrome. J Dent Assoc S Afr 1977;32:13-8.
- 193 6. Becker DB, Needleman HL, Kotelchuck M. Child abuse and dentistry: Orofacial trauma and its recognition by dentists. J Am Dent Assoc 1978;97:24-8.
- 195 7. Cameron JM, Johnson HR, Camps FE. The battered child syndrome. Med Sci Law 1966;6:2-21.
- 197 8. Jessee SA. Physical manifestations of child abuse to the head, face and mouth: A198 hospital survey. J Dent Child 1995;62:245-9.
- 199 9. Jessee SA, Rieger M. A study of age-related variables among physically abused children.
 200 J Dent Child 1996; 63:275-80.
- 201 10. Malecz RE. Child abuse, its relationship to pedodontics: A survey. J Dent Child
 202 1979;46:193-4.
- 203 11. Needleman HL. Orofacial trauma in child abuse: Types, prevalence, management, and
 204 the dental profession's involvement. Pediatr Dent 1986;8(Spec Iss 1):71-80.
- 205 12. O'Neill JA Jr, Meacham WF, Griffin JP, Sawyers JL. Patterns of injury in the battered
 206 child syndrome. J Trauma 1973;13:332-9.
- 207 13. Skinner AE, Castle RL. Seventy-eight Battered Children: A Retrospective Study.
 208 London, England: National Society for the Prevention of Cruelty to Children; 1969.
- 209 14. Tate RJ. Facial injuries associated with the battered child syndrome. Br J Oral Surg 1971;9:41-5.
- 211 15. Vadiakas G, Roberts MW, Dilley DC. Child abuse and neglect: Ethical issues for dentistry. J Mass Dent Soc 1991;40:13-5.
- 213 16. Naidoo S. A profile of the oro-facial injuries in child physical abuse at a children's hospital. Child Abuse Negl 2000;24:521-34.
- 215 17. Kittle PE, Richardson DS, Parker JW. Two child abuse/child neglect examinations for
 216 the dentist. J Dent Child 1981;48:175-80.
- 217 18. Blain SM, Winegarden T, Barber TK, Sognnaes FR. Child abuse and neglect. II: Role of dentistry [abstract]. J Dent Res 1979;58(special issue A):367.
- 219 19. McNeese MC, Hebeler JR. The abused child: A clinical approach to identification and management. Clin Symp 1977;29:1-36.
- 20. Levin AV. Otorhinolaryngologic manifestations. In: Levin AV, Sheridan MS, eds.
 Munchausen Syndrome by Proxy: Issues in Diagnosis and Treatment. New York, NY:
 Lexington Books; 1995:219-30.
- 224 21. Folland DS, Burke RE, Hinman AR, Schaffner W. Gonorrhea in preadolescent children:
 225 An inquiry into source of in-fection and mode of transmission. Pediatrics 1977;60:153-6.
- 22. American Academy of Pediatrics Committee on Child Abuse. Guidelines for the
 evaluation of sexual abuse of children: A subject review. Pediatrics 1999;103:186-91.
- 23. DeJong AR. Sexually transmitted diseases in sexually abused children. Sex Transm Dis
 1986;13:123-6.

- 24. Everett VD, Ingram DL, Flick LAR, Russell TA, Tropez-Sims ST, McFadden AY. A
 comparison of sexually transmitted diseases (STDs) found in a total of 696 boys and
 2973 girls evaluated for sexual abuse [abstract]. Pediatr Res 1998;43:91A.
- 233 25. Nelson JD, Mohs E, Dajani AS, Plotkin SA. Gonorrhea in preschool- and school-aged
 234 children: Report of the Prepubertal Gonorrhea Cooperative Study Group. JAMA
 235 1976;236:1359-64.
- 236 26. Stevens-Simon C, Nelligan D, Breese P, Jenny C, Douglas JM Jr. The prevalence of
 237 genital human papillomavirus infections in abused and nonabused preadolescent girls.
 238 Pediatrics 2000;106:645-9.
- 239 27. Schlesinger SL, Borbotsina J, O'Neill L. Petechial hemorrhages of the soft palate
 240 secondary to fellatio. Oral Surg Oral Med Oral Pathol 1975;40:376-8.
- 241 28. Sperber ND. Bite marks, oral and facial injuries: Harbingers of severe child abuse?242 Pediatrician 1989;16:207-11.
- 243 29. Wagner GN. Bitemark identification in child abuse cases. Pediatr Dent 1986;8:96-100.
- 30. National Research Council, Committee on DNA Techno-logy in Forensic Science, Board
 of Biology, Commission on Life Sciences. DNA Technology in Forensic Science.
 Washington, DC: National Academy Press; 1992.
- 247 31. American Academy of Pediatric Dentistry. Definition of dental neglect. Pediatr Dent
 248 2003;25(suppl):7.
- 32. Sanger RG, Bross DC, eds. Clinical Management of Child Abuse and Neglect: A Guide
 for the Dental Professional. Chicago, Ill: Quintessence Publishing Co, Inc; 1984.
- 251 33. California Society of Pediatric Dentists. Dental neglect: When to report. Calif Pediatricia
 252 1989;Fall:31-2.

- 1 Policy on Hospitalization and Operating Room Access for Dental Care of Infants,
- 2 Children, Adolescents, and Persons With Special Health Care Needs
- 3 4 **Originating Committee** 5 Dental Care Committee 6 7 Review Council 8 Council on Clinical Affairs 9 10 Adopted 11 1989 12 13 Revised 14 1997, 2001, 2005, 2010 15 16 Reaffirmed 17 1993 18 19 20 Purpose 21 The American Academy of Pediatric Dentistry (AAPD) advocates, when indicated, 22 hospitalization and equal access to operating room facilities for dental care of infants, 23 children, adolescents, and persons with special health care needs. The AAPD recognizes 24 that barriers to hospital dental care for patients best treated in that setting need to be 25 addressed. 26 27 Method 28 This policy is an update of the previous document revised in 2005 based upon a review of 29 current dental literature and policies of dental and medical organizations. A MEDLINE 30 search was performed using the keywords "hospitalization" and "operating room access." 31 An updated electronic search using the search terms "Access to care for dental care in 32 hospitals", "Operating room access for dental care" was conducted. Fields: All Fields; 33 Limits: within the last ten years. When data did not appear sufficient or were inconclusive, 34 recommendations were made based upon expert and /or consensus opinion by experienced 35 researchers and clinicians.
 - Council on Clinical Affairs 2009-10 Charge 1(f) P-Hospitalization and Operating Room Access for Dental Care

36

37 Background

38 Pediatric dentists often treat patients who present special challenges related to their age, 39 behavior, medical status, developmental disabilities, intellectual limitations, or special 40 needs. Caries, periodontal diseases, and other oral conditions, if left untreated, can lead to 41 pain, infection, and loss of function.¹⁻⁴ These undesirable outcomes adversely can affect 42 learning, communication, nutrition, and other activities necessary for normal growth and 43 development.⁵⁻⁸ ^ZMany medical conditions (eg, hematological, oncological) are compounded 44 by the presence of oral maladies and disease. To address these challenges and meet these 45 treatment needs effectively, pediatric dentists have developed and employ a variety of 46 management techniques, including accessing anesthesia services and/or the provision of 47 dental care in a hospital setting with or without general anesthesia. Hospital dentistry is an 48 integral part of the curriculum of all accredited advanced pediatric dental training 49 programs. Pediatric dentists are, by virtue of training and experience, qualified to recognize 50 the indications for such an approach and to render such care.98 51 Pediatric dentists occasionally have experienced difficulty in gaining an equal 52 opportunity to schedule operating room time, postponement/delay of nonemergency 53 dental care, and economic credentialing. Economic credentialing (ie, the use of economic 54 criteria not related to quality of care or professional competency) to determine qualifications 55 for granting/renewing an individual's clinical staff membership or privileges should be 56 opposed.^{10,11} ²Additionally, economic credentialing by third party payors in which economic 57 factors are placed above quality of care is unwarranted.¹²-The American Academy of 58 Pediatric Dentistry and the American Dental Association urge hospital insurance carriers to 59 include hospitalization benefits for dental treatment in both private and public insurance 60 programs so that the resources of a hospital are available to patients whose condition in the 61 judgment of the dentist warrants treatment in the operating room.¹⁰ 62 The mutual objective of both the governing board and the medical staff is to improve the 63 quality and efficiency of patient care in the hospital. Decisions regarding hospital privileges 64 should be based upon the training, experience, and demonstrated competence of candidates, 65 taking into consideration the availability of facilities and the overall medical needs of the

- 66 community, the hospital, and especially the patients. Privileges should not be based on
- 67 numbers of patients admitted to the facility or the patient's economic or insurance status.¹³<u>11</u>
- 68

69 Policy statement

- 70 The AAPD shall work with all concerned medical and dental colleagues and organizations
- 71 to remove barriers to hospital <u>and operating room access for</u> dental care for patients best
- 72 treated in that those settings. The AAPD affirms that hospitals or outpatient settings
- 73 providing surgical treatment should not discriminate against pediatric dental patients
- 74 requiring care under general anesthesia. Such patients and their care providers need access
- 75 to these facilities. The dental patient, as with any other patient, should have the right to be
- 76 seen in a timely manner.

77

78 References

- Acs G, Pretzer S, Foley M, Ng MW. Perceived outcomes and parental satisfaction
 following dental rehabilitation under general anesthesia. Pediatr Dent 2001;23(5):419-23.
- 81 2. Low W, Tan S, Schwartz S. The effect of severe caries on the quality of life in young
 82 children. Pediatr Dent 1999;21(6):325-6.
- 83 3. Milano M, Seybold SV. Dental care for special needs patients: A survey of Texas
 84 pediatric dentists. J Dent Child 2002;69(2):212-5.
- 4. American Academy of Pediatric Dentistry. Definition of dental disability. Pediatr Dent 2004;26(suppl):12. 2008-09;29:12.
- 87 5. American Academy of Pediatric Dentistry. Definition of dental neglect. Pediatr Dent
 88 2004;26(suppl):11. 2008-09;29:11.
- 6. American Academy of Pediatric Dentistry. Special Patients. In: Nowak AJ, ed. The
 90 Handbook. 3 nd 2nd ed. Chicago, Ill: AAPD; 1999:224 2007:260.
- 91 7. Badger GR. Caries incidence in child abuse and neglect. Pediatr Dent 1986;8(1 Spec
 92 No.):101-2.
- 93 <u>78.</u> Thomas CW, Primosch RE. Changes in incremental weight and well-being of children
 94 with rampant caries following complete dental rehabilitation. Pediatr Dent
 95 2002;24(2):109-13.
- 89. American Dental Association Commission on Dental Accreditation. Accreditation
 97 standards for advanced specialty education programs in pediatric dentistry: Hospital
 98 and adjunctive experiences. Chicago, Ill; 1998:25-9.

99 <u>911. American Medical Association. Policy H-230.975 Economic credentialing. Adopted 1991;</u> 100 reaffirmed 1998. Available at: <u>"http://www.ama-</u>

- 101 <u>assn.org/ama/noindex/category/11760.html". Accessed February 28, 2005. March</u>
- 102 <u>24,2009. http://www.ama-assn.org/ama/pub/physician-resources/legal-</u>
- 103 topics/medical-staff-topics/economic-credentialing.shtml October 21, 2009
- 104

- 105 ¹⁰<u>10</u>. American Dental Association. Current policies, economic credentialing (1993:692).
- 106 Available at: "http://www.ada.org/prof/resources/positions/policies.asp". Accessed
- 107 February 28, 2005. March 25, 2009. October 21, 2009.
- 108 11. American Medical Association. Policy H-230.975 Economic credentialing. Adopted 1991;
 109 reaffirmed 1998. Available at: "http://www.ama-
- 110 assn.org/ama/noindex/category/11760.html". Accessed February 28, 2005.
- 111 1212. American Medical Association. Policy H-180.967 Economic credentialing by
 112 insurance companies. Adopted 1992; reaffirmed 1996. Available at: "http://ama-assn.org/noindex/categor v/11760.html". Accessed February 28, 2005. March 24,2
- assn.org/ noindex/categor y/11760.html". Accessed February 28, 2005. <u>March 24,2009</u>
 114 111133. American Medical Association. Policy E-4.07 Staff privileges. Issued July 1983;
- 114 <u>111-133</u>. American Medical Association. Policy E-4.07 Starr privileges. Issued July 1985;
 115 updated June 1994. Available at: <u>"http://www.ama-assn.org/ama/noindex/category/</u>
- 116 11760.html". Accessed February 28, 2005. March 24,2009. http://www.ama-
- 117 assn.org/ama/pub/physician-resources/medical-ethics/code-medical-
- 118 <u>ethics/opinion407.shtml October 21, 2009</u>
- 119 120

1	Policy on Hospital Staff Membership			
2 3 4 5	Originating Committee Hospital Guidelines for Pediatric Dentistry Ad Hoc Committee			
5 6 7 8	Review Council Council on Clinical Affairs			
9 10 11	Adopted 1977			
12 13 14 15	Revised 1979, 1991, 1999, 2002, 2005, <u>2010</u>			
16	Purpose			
17	The American Academy of Pediatric Dentistry (AAPD) recognizes that dentists have the			
18	opportunity to play a significant role within a hospital. Staff membership is necessary to			
19	provide comprehensive, consultative, and/or emergency dental services for infants,			
20	children, adolescents, and persons with special health care needs within the hospital setting.			
21				
22	Method			
23	This guideline is an update of the previous document, revised in 2005. The update included			
24	an electronic search using the search terms, "Hospital Staff" and "Dentistry". Fields: All			
25	Fields; Limits: within the last ten years; humans; English; clinical trials. The reviewers			
26	agreed 2 articles met the defined criteria. When data did not appear sufficient or were			
27	inconclusive, recommendations were based upon expert and/or consensus opinion by			
28	experienced researchers and clinicians.			
29	This policy is based on a review of the 2004 Comprehensive Accreditation Manual for			
30	Hospitals: The Official Handbook. ¹			
31				
32	Background			
33	Pediatric dentists contribute in multiple ways as members of the hospital staff. Most			
34	commonly, the pediatric dentist can provide essential comprehensive dental services to			
35	patients within an operating room setting. Additionally, the pediatric dentist can provide			

36 consulting and emergency services.² ¹ "Team" (eg, cleft lip/palate, hemophilia) evaluations 37 of patients often require dental input, and certain medical protocols (eg, hematopoietic cell 38 transplantation) require an oral examination. Beyond patient services, a pediatric dentist 39 may participate within the hospital's organizational structure through committee 40 memberships of either clinical or administrative purpose. A pediatric dentist can partner 41 with medical colleagues in self-development through hospital-sponsored continuing 42 medical education. 43 Health care providers Pediatric dentists seeking hospital staff membership must contact 44 the medical staff office at an area hospital. Board certification or candidacy (previously

45 <u>known as board eligibility</u>) is increasingly being required for hospital staff membership.

46 Following a credentialing process and appointment to a medical staff, a pediatric dentist

47 must accept and fulfill certain responsibilities. Among them are patient care within the

48 limits of approved clinical privileges, <u>possible</u> participation in emergency department on-

49 call rotations, timely medical records completion of medical records, and compliance with

the rules and regulations of the medical/dental staff and the policies and procedures of thehospital.

Although hospital and medical/dental staffs have some individual latitude, the
standards for all hospital services are issued by national commissions such as the Joint
Commission on Accreditation of Healthcare Organization (JCAHO).⁴ ² Standards for dental
services are integrated intimately and inseparably within the overall hospital organizational
structure and, therefore, are stringently subject to the standards established by these

57 commissions.

58 Economic credentialing (ie, the use of economic criteria not related to quality of care or 59 professional competency) to determine qualifications for granting/renewing an individual's 60 clinical staff membership or privileges should be opposed.³ The mutual objective of both the 61 governing board and the medical staff is to improve the quality and efficiency of patient 62 care in the hospital. Decisions regarding hospital privileges should be based upon the 63 training, experience, and demonstrated competence of candidates, taking into consideration 64 the availability of facilities and the overall medical needs of the community, the hospital,

65	and especially the patients. Privileges should not be based on numbers of patients admitted		
66	to t	the facility or the patient's economic or insurance status. ⁴	
67			
68	Po	licy statement	
69	Th	e AAPD:	
70	1.	Encourages the participation of pediatric dentists on hospital medical/dental staffs.	
71		Beyond having the capability to provide valuable services to patients, the pediatric	
72		dentist can be an effective, contributing member to the hospital through consultative	
73		services, educational opportunities, leadership initiatives, and committee membership.	
74	2.	Recognizes the American Dental Association as a corporate member of the JCAHO and	
75		further recognizes the standards for hospital governance, as established by the JCAHO.	
76	3.	Encourages hospital member pediatric dentists to maintain strict adherence to the rules	
77		and regulations of the medical/dental staff and the policies and procedures of the	
78		hospital.	
79	4.	Is opposed to the use of economic credentialing to determine qualifications for	
80		granting/renewing an individual's clinical staff membership or privileges should be	
81		opposed	
82			
83			
84	Re	ferences	
85	2 .	<u>1.</u> Weddell JA, Jones JE. Hospital dental services for children and the use of general	
86		anesthesia. In: McDonald RE, Avery DR, Dean JA, eds. Dentistry for the Child and	
87	1	Adolescent. 8th ed. St. Louis, Mo: Mosby; 2004:314.	
00 80	÷.	<u>2.</u> Joint Commission on Accreditation of Health Care Organizations. Medical staff	
90		Official Handbook, Oakbrook Terrace, Ill: Joint Commission on Accreditation of Health	
91		Care Organizations; 2004 2009.	
92	3.	American Medical Association. Policy H 230.975 Economic Credentialing. Adopted 1991;	
93		reaffirmed 1998. Available at http://www.ama-	
94		assn.org/ama/noindex/category/11760.html Accessed June 16, 2009.	
95	4.	American Medical Association. Policy E-4.07 Staff privileges. Issued July 1983; updated	
96		June 1994. Available at: "http://www.ama-assn.org/ama/noindex/category/	
97 98		11/60.ntml ⁻ . Accessed June 16, 2009.	

- 1 Guideline on Management of Acute Dental Trauma 2 3 Originating Council 4 Council on Clinical Affairs 5 6 **Review Council** 7 Council on Clinical Affairs 8 9 Adopted 10 2001 11 12 Revised 13 2004, 2007, 2010 14 15 Purpose 16 The American Academy of Pediatric Dentistry (AAPD) intends these guidelines to define,
- 17 describe appearances, and set forth objectives for general management of acute traumatic
- 18 dental injuries rather than recommend specific treatment procedures that have been
- 19 presented in considerably more detail in textbooks and the dental/medical literature.
- 20

21 Methods

- 22 This guideline is an update of the previous document revised in 2007. It is based on a
- 23 review of the current dental and medical literature related to dental trauma. An electronic
- 24 MEDLINE search was conducted using the <u>following: *t*T</u>erms: "teeth", "trauma",
- 25 "permanent teeth", and "primary teeth"; Field: All Fields; Limits: within the last 10 years;
- 26 <u>humans; English</u>. <u>5,269 articles matched these criteria</u>. Papers for review were chosen from
- 27 <u>this list and references within selected articles</u>. <u>Also In addition</u>, a review of the journal
- 28 *Dental Traumatology* was conducted for the years 2000-2006<u>9</u>. The recommendations are
- 29 congruent with the 20017 guidelines developed by the International Association of Dental
- 30 Traumatology.^{1-5 1-3} <u>When data did not appear sufficient or were inconclusive</u>,
- 31 recommendations were based upon expert and/or consensus opinion including those from
- 32 the 2008 symposium of the AAPD titled "Symposium on Trauma: A Comprehensive Update
- 33 <u>on Permanent Tooth Trauma in Children" (Chicago, Ill).</u>
- 34

35 Background

Facial trauma that results in fractured, displaced, or lost teeth can have significant negative
functional, esthetic, and psychological effects on children.^{6 4, 5} Dentists and physicians
should collaborate to educate the public about prevention and treatment of oral traumatic
injuries to the oral and maxillofacial region.
The greatest incidence of trauma to the primary dentition teeth occurs at 2 to 3 years of

41 age, when motor coordination is developing.^{7 6} The most common injuries to permanent
42 teeth occur secondary to falls, followed by traffic accidents, violence, and sports.^{8-11 7-10} All

43 sporting activities have an associated risk of orofacial injuries due to falls, collisions, and

44 contact with hard surfaces.^{12 11} The AAPD encourages the use of protective gear, including

45 mouth-guards, which help distribute forces of impact, thereby reducing the risk of severe
46 injury.^{13,14} ^{12,13}

47 Dental injuries could have improved outcomes if the public were aware of first-aid
48 measures and the need to seek immediate treatment.¹⁵⁻¹⁸ ¹⁴⁻¹⁷ Because optimal treatment
49 results follow immediate assessment and care,¹⁹ ¹⁸ dentists have an ethical obligation to
50 ensure that reasonable arrangements for emergency dental care are available.²⁰ ¹⁹ The
51 history, circumstances of the injury, pattern of trauma, and behavior of the child and/or
52 caregiver are important in distinguishing non-abusive injuries from abuse.²⁴ ²⁰

53 Practitioners have the responsibility to recognize, differentiate, and either appropriately
54 manage or refer children with acute oral traumatic injuries, as dictated by the complexity of
55 the injury and the individual clinician's training, knowledge, and experience. Compromised

56 airway, neurological manifestations such as altered orientation, hemorrhage, nausea,

57 <u>vomiting</u>, or suspected loss of consciousness requires further evaluation by a physician.

58 To efficiently determine the extent of injury and correctly diagnose injuries to the teeth,

59 periodontium, and associated structures, a systematic approach to the traumatized child is

60 essential.^{22,23} ^{21,22} Assessment includes a thorough <u>medical and dental</u> history, visual <u>clinical</u>

61 and radiographic examination, and additional tests such as palpation, percussion,

62 <u>sensitivity</u>, and mobility evaluation. Intraoral radiography is useful for the evaluation of

- 63 dentoalveolar trauma. If the area of concern extends beyond the dentoalveolar complex,
- 64 extraoral imaging may be indicated. Treatment planning takes into consideration the

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patient's health status and developmental status as well as extent of injuries. Advanced
behavior guidance techniques or an appropriate referral may be necessary to ensure that
proper diagnosis and care are given.

68 All relevant diagnostic information, treatment, and recommended follow-up care are 69 documented in the patient's record. Appendix I is a sample document for recording 70 assessment of acute traumatic injuries. This sample form, developed by the AAPD, is 71 provided as a practice tool for pediatric dentists and other dentists treating children. It was 72 developed by pediatric dentistry experts and offered to facilitate excellence in practice. This 73 form, however, does not establish or evidence a standard of care. In issuing this form, the 74 AAPD is not engaged in rendering legal or other professional advice. If such services are 75 required, competent legal or other professional counsel should be sought. Well-designed 76 follow-up procedures are essential to diagnose complications.

77 After a primary tooth has been injured, the treatment strategy is dictated by the concern 78 for the safety of the permanent dentition.^{7,22,24} 6, ^{21, 23} If determined that the displaced 79 primary tooth has encroached upon the developing permanent tooth germ, removal is 80 indicated.^{2,7,25-29} ^{3, 6, 24-28} In the primary dentition, the maxillary anterior region is at low risk 81 for space loss unless the avulsion occurs prior to canine eruption or the dentition is 82 crowded.²⁴ ²³ Fixed or removable appliances, while not always necessary, can be fabricated 83 to satisfy parental concerns for esthetics or to return a loss of oral or phonetic function.⁷ ⁶ 84 When an injury to a primary tooth occurs, informing parents about possible pulpal 85 complications, appearance of a vestibular sinus tract, or color change of the crown 86 associated with a sinus tract can help assure timely intervention, minimizing complications 87 for the developing succedaneous teeth.^{2,7,30} ^{3, 6, 29, 30} Also, it is important to caution parents 88 that the primary tooth's displacement may result in any of several permanent tooth 89 complications, including enamel hypoplasia, hypocalcification, crown/root dilacerations, or 90 disruptions in eruptions patterns or sequence.^{30 29} The risk of trauma-induced 91 developmental disturbances in the permanent successors is greater in children whose 92 enamel calcification is incomplete.^{24,31} ^{23,31} 93 The treatment strategy after injury to a permanent tooth is dictated by the concern for

94 vitality of the periodontal ligament and pulp. Subsequent to the initial management of the

- 95 dental injury, continued periodic monitoring is indicated to determine clinical and
- 96 radiographic evidence of successful intervention (ie, asymptomatic, positive sensitivity to
- 97 pulp testing, root continues to develop in immature teeth, no mobility, no periapical
- 98 pathology).^{3-5,22,29,32} 1, 2, 21, 24, 32 Initiation of endodontic treatment is indicated in cases of
- 99 spontaneous pain; abnormal response to pulp <u>sensitivity</u> tests; lack of continued root
- 100 formation or apexogenesis; or breakdown of periradicular supportive tissue.^{3-5,22,29,32} 1, 2, 21,
- 101 ^{24, 32} To restore a fractured tooth's normal esthetics and function, reattachment of the crown
- 102 fragment is an good alternative that can should be considered.^{22,29} 21, 24, 33
- 103 To stabilize a tooth following traumatic injury, a splint may be necessary.^{29,33-37} ^{24, 34-38}
- 104 Flexible splinting assists in healing.^{22,38} ^{21,39} Characteristics of the ideal splint include:
- 105 1. easily fabricated in the mouth without additional trauma;
- 106 2. passive unless orthodontic forces are intended;
- 107 3. allows physiologic mobility;
- 108 4. nonirritating to soft tissues;
- 109 5. does not interfere with occlusion;
- 110 6. allows endodontic access and vitality testing;
- 111 7. easily cleansed;
- 112 8. easily removed.
- 113 Instructions to patients having a splint placed include to:
- 114 1. consume a soft diet
- 115 2. avoid biting on splinted teeth;
- 116 3. maintain meticulous oral hygiene;
- 117 4. use chlorhexidine/antibiotics as prescribed;
- **118** 5. call immediately if splint breaks/loosens.
- 119

120 Recommendations

- 121 Infraction
- **122** Definition: incomplete fracture (crack) of the enamel without loss of tooth structure.
- 123 Diagnosis: normal gross anatomic and radiographic appearance; craze lines apparent,
- 124 especially with transillumination.

- 125 Treatment objectives: to maintain structural integrity and pulp vitality.^{29, 39, 40} ^{24, 40, 41}
- 126 General prognosis: Complications are unusual.⁴¹ ⁴²
- 127

128 Crown fracture-uncomplicated

- 129 Definition: an enamel fracture or an enamel-dentin fracture that does not involve the pulp.
- 130 Diagnosis: clinical and/or radiographic findings reveal a loss of tooth structure confined to
- 131 the enamel or to both the enamel and dentin. 1,3,7,19 22,24 26,32,39,42,43 1, 3, 6, 18-21, 23, 26, 30, 32, 41, 43, 44
- **132** Treatment objectives: to maintain pulp vitality and restore normal esthetics and function.
- 133 Injured lips, tongue, and gingiva should be examined for tooth fragments. <u>Radiographs</u>,
- 134 <u>including lip and cheek lacerations, are recommended.</u> For small fractures, rough margins
- and edges can be smoothed. For larger fractures, the lost tooth structure can be
- 136 restored. 1,3,7,22,24 26,30,32,39,41 43 1, 3, 6, 21, 23, 26, 29, 30, 32, 41-44
- 137 General prognosis: The prognosis of uncomplicated crown fractures depends primarily
- 138 upon the concomitant injury to the periodontal ligament and secondarily upon the extent of
- 139 dentin exposed.²² ²¹ Optimal treatment results follow timely assessment and care.
- 140

141 Crown fracture-complicated

- **142** Definition: an enamel-dentin fracture with pulp exposure.
- 143 Diagnosis: clinical and radiographic findings reveal a loss of tooth structure with pulp
- 144 exposure.^{1,3,7,22} 1, 3, 6, 21</sup>
- 145 Treatment objectives: to maintain pulp vitality and restore normal esthetics and function.^{30 29}
- 146 Injured lips, tongue, and gingiva should be examined for tooth fragments. <u>Radiographs</u>,
- 147 <u>including lip and cheek lacerations, are recommended.</u>1
- Primary teeth: Decisions often are based on life expectancy of the traumatized
 primary tooth and vitality of the pulpal tissue. Pulpal treatment alternatives are
- 150 pulpotomy, pulpectomy, and extraction.^{1,7,24-26} 3, 6, 23, 26, 30
- Permanent teeth: Pulpal treatment alternatives are direct pulp capping, partial
- 152 pulpotomy, and pulpectomy (start of root canal therapy).^{3,22,41,44} 1, 21, 42, 43 There is
- 153 <u>increasing evidence to suggest that utilizing conservative vital pulp therapies for</u>

- 154 <u>mature teeth with closed apices is as appropriate a management technique as when</u>
- 155 <u>used for immature teeth with open apices.</u>⁴⁵
- 156 General prognosis: The prognosis of crown fractures appears to depend primarily upon a
- 157 concomitant injury to the periodontal ligament.²² ²¹ The age of the pulp exposure, extent of
- 158 dentin exposed, and stage of root development at the time of injury secondarily affect the
- tooth's prognosis.²² ²¹ Optimal treatment results follow timely assessment and care.
- 160

161 Crown/root fracture

162 Definition: an enamel, dentin, and cementum fracture with or without pulp exposure.

- 163 Diagnosis: Clinical findings usually reveal a mobile coronal fragment attached to the
- 164 gingiva with or without a pulp exposure. Radiographic findings may reveal a radiolucent
- 165 oblique line that comprises crown and root in a vertical direction in primary teeth and in a
- 166 direction usually perpendicular to the central radiographic beam in permanent teeth. While
- 167 radiographic demonstration often is difficult, root fractures can only be diagnosed
- 168 radiographically.^{1,3,7,22,29} 1, 3, 6, 21, 30

169 Treatment objectives: to maintain pulp vitality and restore normal esthetics and function.¹¹¹⁰

- Primary teeth: When the primary tooth cannot or should not be restored, the entire tooth should be removed unless retrieval of apical fragments may result in damage to the succedaneous tooth.^{1,7} ^{3,6}
- Permanent teeth: The emergency treatment objective is to stabilize the coronal
- 174 fragment. Definitive treatment alternatives are to remove the coronal fragment
- followed by a supragingival restoration or necessary gingivectomy; osteotomy; or
- 176 surgical or orthodontic extrusion to prepare for restoration. If the pulp is exposed,
- pulpal treatment alternatives are pulp capping, pulpotomy, and root canal
- 178 treatment.^{3,22,41} 1, 21, 42
- 179 General prognosis: Although the treatment of crown-root fractures can be complex and
- 180 laborious, most fractured permanent teeth can be saved.²² ²¹ Fractures extending
- 181 significantly below the gingival margin may not be restorable.
- 182

183 Root fracture

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- 184 Definition: a dentin and cementum fracture involving the pulp.
- 185 Diagnosis: Clinical findings reveal a mobile coronal fragment attached to the gingiva that
- 186 may be displaced. Radiographic findings may reveal 1 or more radiolucent lines that
- 187 separate the tooth fragments in horizontal fractures. Multiple radiographic exposures at
- 188 different angulations may be required for diagnosis. A root fracture in a primary tooth may
- 189 be obscured by a succedaneous tooth.^{1,3,7,22} 1, 3, 6, 21
- 190 Treatment objectives: to reposition as soon as possible and then to stabilize the coronal
- 191 fragment in its anatomically correct position to optimize healing of the periodontal ligament
- and neurovascular supply, while maintaining esthetic and functional integrity.²⁹ ²⁴
- Primary teeth: Treatment alternatives include extraction of coronal fragment without
 insisting on removing apical fragment or observation.^{1,7,24} 3, 6, 23 It is not
- 195 recommended to reposition and stabilize the coronal fragment.³
- Permanent teeth: Reposition and stabilize the coronal fragment.^{3,22} 1,21
- 197 General prognosis: Pulp necrosis in root-fractured teeth is attributed to displacement of the
- 198 coronal fragment and mature root development.^{22,46} ^{21,46} In permanent teeth, the location of
- 199 the root fracture has not been shown to affect pulp survival after injury.^{22,47} ^{21,47} Therefore,
- 200 preservation of teeth with root fractures occurring in the tooth's cervical third should be
- attempted.^{22,47} ^{21,47}Young age, immature root formation, positive pulp sensitivity at time of
- injury, and approximating the dislocation within 1 mm have been found to be advantageous
- to both pulpal healing and hard tissue repair of the fracture.^{38,47,48} ^{39,47,48}
- 204

205 Concussion

- 206 Definition: Injury to the tooth-supporting structures without abnormal loosening or
- displacement of the tooth.
- 208 Diagnosis: Because the periodontal ligament absorbs the injury and is inflamed, clinical
- 209 findings reveal a tooth tender to pressure and percussion without mobility, displacement, or
- sulcular bleeding. Radiographic abnormalities are not expected.^{2,4,7,22,24,32} 1, 3, 6, 21, 23, 32
- 211 Treatment objectives: to optimize healing of the periodontal ligament and maintain pulp
- 212 vitality. 2,4,7,22,24,29,32,49 1, 3, 6, 21, 23, 24, 32, 49

- 213 General prognosis: For primary teeth, unless associated infection exists, no pulpal therapy is
- 214 indicated.⁷ ⁶ Although there is a minimal risk for pulp necrosis, mature permanent teeth
- 215 with closed apices may undergo pulpal necrosis due to associated injuries to the blood
- 216 vessels at the apex and, therefore, must be followed carefully.²² ²¹
- 217

218 Subluxation

- 219 Definition: injury to tooth-supporting structures with abnormal loosening but without tooth220 displacement.
- 221 Diagnosis: Because the periodontal ligament attempts to absorb the injury, clinical findings
- reveal a mobile tooth without displacement that may or may not have sulcular bleeding.
- **223** Radiographic abnormalities are not expected.^{2,4,7,22} 1, 3, 6, 21
- 224 Treatment objectives: to optimize healing of the periodontal ligament and neurovascular
- 225 supply.^{2,4,7,22,24-29,32,49} 1, 3, 6, 21, 23, 24, 26-28, 30, 32, 49
- Primary teeth: The tooth should be followed for pathology.
- Permanent teeth: Stabilize the tooth and relieve any occlusal interferences. For
- comfort, a flexible splint can be used. Splint for no more than 2 weeks.
- 229 General prognosis: Prognosis is usually favorable.^{24,32} ^{23,32} The primary tooth should return
- to normal within 2 weeks.⁷ ⁶Mature permanent teeth with closed apices may undergo
- 231 pulpal necrosis due to associated injuries to the blood vessels at the apex and, therefore,
- 232 must be followed carefully.²² ²¹
- 233

234 Lateral luxation

- 235 Definition: displacement of the tooth in a direction other than axially. The periodontal
- ligament is torn and contusion or fracture of the supporting alveolar bone occurs.^{24,32,50} ^{23, 32,}
 ⁵⁰
- 238 Diagnosis: Clinical findings reveal that a tooth is displaced laterally with the crown usually
- in a palatal or lingual direction and may be locked firmly into this new position. The tooth
- 240 usually is not mobile or tender to touch. Radiographic findings reveal an increase in
- 241 periodontal ligament space and displacement of apex toward or though the labial bone
- 242 plate.^{2,4,7,22,50} 1, 3, 6, 21, 50

243 Treatment objectives:

244	•	Primary teeth: to allow passive <u>or spontaneous</u> repositioning <u>if there is no occlusal</u>
245		interference. ³ When there is occlusal interference, the tooth can be gently
246		repositioninged or slightly reduced if the interference is minor.3 - actively reposition
247		and splint for 1 to 2 weeks as indicated to allow for healing, except w<u>W</u>hen the
248		injury is severe or the tooth is nearing exfoliation, extraction is the treatment of
249		<u>choice</u> . ^{2,7,25-29} 3, 6, 24, 26-28, 30
250	•	Permanent teeth: to reposition as soon as possible and then to stabilize the tooth in
251		its anatomically correct position to optimize healing of the periodontal ligament and
252		neurovascular supply, while maintaining esthetic and functional integrity.
253		Repositioning of the tooth is done with digital pressure and little force. The \underline{A}
254		displaced tooth may need to be extruded to free itself from the apical lock in the
255		cortical bone plate. Splinting an additional 2 to 4 weeks may be needed with
256		breakdown of marginal bone. 4,22,29,49,50 1, 21, 24, 49, 50

- **257** General prognosis: Primary teeth requiring repositioning have an increased risk of
- developing pulp necrosis compared to teeth that are left to spontaneously reposition.⁷ ⁶ In
- 259 mature permanent teeth with closed apices, pulp necrosis and pulp canal obliteration are
- $260 \qquad \text{common healing complications while progressive root resorption is less likely to occur.^{50} 50}$
- 261

262 Intrusion

263 Definition: apical displacement of tooth into the alveolar bone. The tooth is driven into the

socket, compressing the periodontal ligament and commonly causes a crushing fracture of

265 the alveolar socket.^{24,32,51} ^{23, 32, 51}

266 Diagnosis: Clinical findings reveal that the tooth appears to be shortened or, in severe cases,

it may appear missing. The tooth's apex usually is displaced labially toward or through the

268 labial bone plate in primary teeth and driven into the alveolar process in permanent teeth.

- 269 The tooth is not mobile or tender to touch. Radiographic findings reveal that the tooth
- appears displaced apically and the periodontal ligament space is not continuous.
- 271 Determination of the relationship of an intruded primary tooth with the follicle of the
- succedaneous tooth is mandatory. If the apex is displaced labially, the apical tip can be seen

- radiographically with the tooth appearing shorter than its contralateral. If the apex is
 displaced palatally towards the permanent tooth germ, the apical tip cannot be seen
 radiographically and the tooth appears elongated. An extraoral lateral radiograph also can
 be used to detect displacement of the apex toward or though the labial bone plate. An
 intruded young permanent tooth may mimic an erupting tooth.^{247,22,51} 1,3,6,21,51</sup>
 Treatment objectives:
- Primary teeth: to allow spontaneous re-eruption except when displaced into the developing successor. Extraction is indicated when the apex is displaced toward the permanent tooth germ.^{2,7,25-29} 3, 6, 24, 26-28, 30
- 282 • Permanent teeth: For immature teeth with more eruptive potential (root 1/2 to 2/3 283 formed), allow to reposition passively (allowing re-eruption to its preinjury 284 position), actively (repositioning with traction), or surgically and then to stabilize the 285 tooth with a splint for up to 4 weeks in its anatomically correct position to optimize 286 healing of the periodontal ligament and neurovascular supply while maintaining 287 esthetic and functional integrity. In teeth with immature root formation, the objective 288 is to allow for spontaneous eruption. In mature teeth, the goal is to reposition the 289 tooth with orthodontic or surgical extrusion and initiate endodontic treatment
- within the first 3 weeks of the traumatic incidence.^{4,22,29,49,51} 1, 21, 24, 49, 51
- 291 General prognosis: In primary teeth, 90% of intruded teeth will reerupt spontaneously 292 (either partially or completely) in 2 to 6 months.^{24,52} ^{23,52} Even in cases of complete intrusion 293 and displacement of primary teeth through the labial bone plate, a retrospective study 294 showed the re-eruption and survival of most teeth for more than 36 months.⁵³ ⁵³ Ankylosis 295 may occur, however, if the periodontal ligament of the affected tooth was severely 296 damaged, thereby delaying or altering the eruption of the permanent successor.⁷ ⁶ In mature 297 permanent teeth with closed apices, there is considerable risk for pulp necrosis, pulp canal 298 obliteration, and progressive root resorption.^{54 51} Immature permanent teeth that are 299 allowed to reposition spontaneously demonstrate the lowest risk for healing 300 complications.^{54,55} ^{54,55} Extent of intrusion (7mm or greater) and adjacent intruded teeth 301 have a negative influence on healing.⁵⁴ ⁵⁴ 302

303	Extrusion		
304	Definition: partial displacement of the tooth axially from the socket; partial avulsion. The		
305	periodontal ligament usually is torn. ^{24,32,56} ^{23, 32, 56}		
306	Diagnosis: Clinical findings reveal that the tooth appears elongated and is mobile.		
307	Radiographic findings reveal an increased periodontal ligament space apically. ^{2,4,7,22,56} 1, 3, 6,		
308	21, 56		
309	Treatment objectives:		
310	• Primary teeth: to allow tooth to reposition spontaneously or reposition and allow for		
311	healing for minor extrusion (<3mm) in an immature developing tooth except		
312	when there are i- Indications for an extraction (ie, the injury is include severe		
313	extrusion or mobility, or the tooth is nearing exfoliation, the child's inability to cope		
314	with the emergency situation, or when the primary tooth is fully formed). If the		
315	treatment decision is to reposition and stabilize, splint for 1 to 2 weeks. ^{2,7,24-29} 6, 23, 24, 26-		
316	28, 30, 57		
317	• Permanent teeth: to reposition as soon as possible and then to stabilize the tooth in		
318	its anatomically correct position to optimize healing of the periodontal ligament and		
319	neurovascular supply while maintaining esthetic and functional integrity.		
320	Repositioning may be accomplished with slow and steady apical pressure to		
321	gradually displace coagulum formed between root apex and floor of the socket.		
322	Splint for up to <u>3</u> <u>2</u> weeks. <u>4,22,29,49,56</u> 1, 21, 24, 49, 56		
323	General prognosis: There is a lack of clinical studies evaluating repositioning of extruded		
324	primary teeth. ⁷ ⁶ In permanent mature teeth with closed apices, there is considerable risk		
325	for pulp necrosis and pulp canal obliteration. ⁵⁶ ⁵⁶ These teeth must be followed carefully. ^{4,22}		
326	1, 21		
327			
328	Avulsion		
329	Definition: complete displacement of tooth out of socket. The periodontal ligament is		
330	severed and fracture of the alveolus may occur. ^{24,32} ^{23,32}		

- 331 Diagnosis: Clinical and radiographic findings reveal that the tooth is not present in the
- 332 socket or the tooth already has been replanted. Radiographic assessment will verify that the
- tooth is not intruded when the tooth was not found. $\frac{2,7,22,24,32}{1,3,6,21,23,32}$
- **334** Treatment objectives:
- Primary teeth: to prevent further injury to the developing successor. Avulsed
 primary teeth should not be replanted because of the potential for subsequent
 damage to developing permanent tooth germs.^{2,7,22,24,28,29} 3, 6, 21, 23, 24, 27
- Permanent teeth: to replant as soon as possible and then to stabilize the replanted
 tooth in its anatomically correct location to optimize healing of the periodontal
- 340 ligament and neurovascular supply while maintaining esthetic and functional
 341 integrity except when replanting is contraindicated by:
- 342 1. the child's stage of dental development (risk for ankylosis where considerable343 alveolar growth has to take place);
- 344 2 compromising medical condition; or
- 345 3. compromised integrity of the avulsed tooth or supporting tissues. Flexible splinting
 346 for 4 <u>2</u> weeks is indicated.⁵ ² Tetanus prophylaxis and antibiotic coverage should be
 347 considered.^{5,22,29,57,63} ², ²¹, ²⁴, ⁵⁸, ⁵⁹ Treatment strategies are directed at avoiding
 348 inflammation that may occur as a result of the tooth's attachment damage and/or
 349 pulpal infection.^{64,65} ^{60, 61}
- 350 General prognosis: Prognosis in the permanent dentition is primarily dependent upon
- formation of root development and extraoral dry time.^{5,22} ², ²¹ The tooth has the best
- 352 prognosis if replanted immediately.^{29,64} ^{24,61} If the tooth cannot be replanted within 5
- minutes, it should be stored in a medium that will help maintain vitality of the periodontal
 ligament fibers.^{30,66} ^{29,62}
- 355 <u>The best</u> ∓transportation media (physiologic) for avulsed teeth include are (in order of preference) Viaspan[™], Hank's Balanced Salt Solution (tissue culture medium), and cold
 357 milk, 57,61-63,67 58, 59, 63-65 <u>Next best would be a non-physiologic media such as saliva (buccal</u>
 358 vestibule), physiologic saline, or water. 57,61-63,67 58, 59, 63-65 <u>Although water is detrimental to</u>
 359 <u>cell viability due to its low osmolality and long term storage in water (ie more than 20</u>
 360 minutes) has an adverse effect on periodontal ligament healing, it is a better choice than dry

361 storage. ²⁴ Limited tooth storage in a cell-compatible medium prior to replantation has 362 produced similar healing results as compared with immediately-replanted teeth.⁶⁸ 363 The risk of ankylosis increases significantly with an extraoral dry time of 15 20 minutes.^{30,69} 364 ^{29, 62, 67, 68} An extraoral dry time of 60 minutes is considered the point where survival of the 365 root periodontal cells is unlikely.⁶⁴ ⁶¹ In permanent avulsed teeth, there is considerable risk 366 for pulp necrosis, root resorption, and ankylosis. , and subsequent infraocclusion during 367 adolescent growth.^{61,70,71} 63, 69, 70 368 Additional considerations: Recent evidence suggests that success of replantation is 369 dependent upon many factors, some of which the clinician can manipulate in a manner that 370 favors more successful outcomes. The Decision Trees in Figures 1 and 2 offer the most up-371 to-date information in an easy-to-use flow-chart format.⁶⁰ 372 Revascularization: An immature or open apex tooth has the potential to establish 373 revascularization when there is a minimum of a 1.0 mm apical opening.⁷¹ Complete pulpal 374 revascularization has been shown to occur at a rate of 18% among immature teeth.²² It 375 appears that antibiotic treatment reduces contamination of the root surface and / or pulp 376 space, thereby creating a biological environment that aids revascularization. ⁷³ On the other 377 hand, a mature tooth or closed apex tooth (apex opening < 1.0 mm) has little or no chance of 378 revascularization. Researchers have demonstrated that immature teeth soaked in 379 doxycycline solution have a greater rate of pulp revascularization.73,74 380 PDL Management - Transitional Therapy: When a tooth has been out of the oral cavity and 381 in a dry environment for greater than 60 minutes, the PDL has no chance of survival. If such 382 a tooth is replanted, it is likely to undergo osseous replacement resorption and over time, 383 the tooth will become ankylosed and will ultimately be lost.²⁵ Because pediatric dentists 384 need to consider the growth and development of the child patient, the goal for a tooth that 385 has been avulsed for greater than 60 minutes with dry storage is to delay the osseous 386 replacement and hence, ankylotic process as long as possible. To slow down this process, 387 the remaining PDL should be removed because otherwise it becomes a stimulus for 388 inflammation that accelerates infection-related resorption. The remaining PDL can be 389 removed by several methods: gentle scaling and root planning, soft pumice prophy, gauze 390 or by soaking the tooth in 3% citric acid for 3 minutes.74,76 This should be followed by a

- 391 <u>sodium fluoride treatment for 20 minutes</u>. The rationale for this fluoride soak is based on
- 392 evidence that this procedure will delay but not prevent ankylosis. Despite these
- 393 recommendations, teeth that have been out of the oral cavity for greater than 60 minutes
- 394 with dry storage have a poor prognosis and will not survive long-term; however, when
- 395 <u>teeth are soaked in fluoride before replantation, it has been shown to significantly reduce</u>
- 396 <u>the risk of resorption after a follow-up of five years.</u>⁷⁷
- **397** <u>Possible contraindications to replanting:</u> There are possible contraindications to tooth
- **398** replantation. Examples are immunocompromise<u>d health</u>, severe congenital cardiac
- anomalies, severe uncontrolled seizure disorder, severe mental disability, severe
- 400 uncontrolled diabetes, and lack of alveolar integrity.

401 Current Research

- 402 Antiresorptive-regenerative therapies may have potential for enhancing the prognosis of
- 403 avulsed teeth.⁶⁸ ⁶⁶ Treatment strategies are directed at avoiding or minimizing
- 404 inflammation, increasing revascularization, and producing hard barriers in teeth with open
- 405 apices.^{5,64,72.81} ^{2, 61, 73, 78-86} New treatment strategies are also directed at specific clinical
- 406 <u>challenges that include decoronation as an approach to treat ankylosis in growing children</u>
- 407 <u>and transplantation of premolars as an approach for replacing avulsed teeth.</u>^{87,88} Dental
- 408 practitioners should follow current literature and consider carefully evidenced-based
- 409 recommendations that may enhance periodontal healing and revascularization of avulsed
- 410 permanent teeth.
- 411 Orthodontic Movement of Traumatized Teeth
- 412 <u>Teeth that have been traumatized must be evaluated carefully prior to beginning or</u>
- 413 <u>continuing orthodontic movement</u>. Even with more simple crown / root fractures without
- 414 pulpal involvement, a 3 month wait is recommended before tooth movement should begin.
- 415 Other minor trauma to the tooth and periodontium such as minor concussions,
- 416 <u>subluxations, and extrusions also require a 3 month rest. When there has been moderate to</u>
- 417 <u>severe trauma / damage to the periodontium, a minimum of 6 months wait is</u>
- 418 <u>recommended.^{89,90} Teeth that have sustained root fractures cannot be moved for at least one</u>
- 419 <u>year.^{89,90} Where there is radiographic evidence of healing, these teeth can be successfully</u>

420	<u>moved.91</u>	In teeth that require endodontics	, movement can begin once	<u>healing is evident.^{89,}</u>
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- 421 <u>90</u>
- 422 Since teeth that have sustained severe periodontal injury have been found to undergo pulp
- 423 <u>necrosis when orthodontic movement was initiated even after a rest period, 92, 93 light</u>
- 424 <u>intermittent forces are recommended along with avoidance of prolonged tipping forces and</u>
- 425 <u>contact with the buccal or lingual cortical plates.</u>90
- 426 <u>The use of a mouthguard during fixed appliance therapy is recommended</u>. Studies have
- 427 <u>found the most effective are a modified custom mouthguard.94,95</u> The newer stock ortho-
- 428 <u>channel mouthguards may be more convenient but there are no studies to date on their</u>
- 429 <u>effectiveness.94</u>
- 430
- 431 References
- 432 1. Flores MT, Andreasen JO, Bakland LK, et al. Guidelines for the evaluation and
 433 management of traumatic dental injuries (part 1 of the series). Dental Traumatol
 434 2001;17(1):1-4.
- 435 -2. Flores MT, Andreasen JO, Bakland LK, et al. Guidelines for the evaluation and
 436 management of traumatic dental injuries (part 2 of the series). Dental Traumatol
 437 2001;17(2):49-52.
- 438 -3. Flores MT, Andreasen JO, Bakland LK, et al. Guidelines for the evaluation and
 439 management of traumatic dental injuries (part 3 of the series). Dental Traumatol
 440 2001;17(3):97-102.
- 441 -4. Flores MT, Andreasen JO, Bakland LK, et al. Guidelines for the evaluation and
 442 management of traumatic dental injuries (part 4 of the series). Dental Traumatol
 443 2001;17(4):145-8.
- 444 -5. Flores MT, Andreasen JO, Bakland LK, et al. Guidelines for the evaluation and
 445 management of traumatic dental in juries (part 5 of the series). Dental Traumatol
 446 2001;17(5):193-6.
- 447 <u>1. Flores M, Andersson L, Andreasen J, Bakland L, Malmgren B, Barnett F, et al. Guidelines</u>
 448 <u>for the management of traumatic dental injuries. I. Fractures and luxations of</u>
 449 permanent teeth. Dental Traumatol 2007;23(2):66-71.
- 450 <u>2. Flores M, Andersson L, Andreasen J, Bakland L, Malmgren B, Barnett F, et al. Guidelines</u>
 451 <u>for the management of traumatic dental injuries. II. Avulsion of permanent teeth.</u>
 452 <u>Dental Traumatol 2007;23(3):130-36.</u>
- 453 <u>3. Flores M, Malmgren B, Andersson L, Andreasen J, Bakland L, Barnett F, et al. Guidelines</u>
 454 <u>for the management of traumatic dental injuries. III. Primary teeth. Dental Traumatol</u>
 455 <u>2007;23(4):196-202.</u>
- 456 64. Cortes MI, Marcenes W, Shelham A. Impact of traumatic injuries to the permanent teeth
 457 on the oral health-related quality of life in 12- to 14-year old children. Community Dent
 458 and Oral Epidemiol 2002;30(3):193-8.

459	5. Lee J, Divaris K. Hidden consequences of dental trauma: the social and psychological
460	effects. Pediatr Dent 2009;31(2):96-101.
461	76. Flores MT. Traumatic injuries in the primary dentition. Dental Traumatol 2002;18(6):287-
462	98.
463	<u>97</u> . Caldas AF Jr, Burgos ME. A retrospective study of traumatic dental injuries in a
464	Brazilian dental trauma clinic. Dental Traumatol 2001;17(6):250-3.
465	8. Rocha MJ, Cardoso M. Traumatized permanent teeth in Brazilian children assisted at
466	the Federal University of Santa Catarina, Brazil. Dental Traumatol 2001;17(6):245-9.
467	109. Skaare AB, Jacobsen I. Dental injuries in Norwegians aged 7-18 years. Dental
468	Traumatol 2003;19(2):67-71.
469	44 <u>10</u> . Tapias MA, Jiménez-Garcia R, Lamas F, Gil AA. Prevalence of traumatic crown
470	fractures to permanent incisors in a childhood population: Móstoles, Spain. Dental
471	Traumatol 2003;19(3):119-22.
472	1211. Gassner R, Bosch R, Tuli T, Emshoff R. Prevalence of dental trauma in 6000 patients
473	with facial injuries: Implications for prevention. Oral Surg Oral Med Oral Pathol Oral
474	Radiol Endod 1999;87(1):27-33.
475	14 <u>12</u> . American Academy of Pediatric Dentistry. Policy on prevention of sports-related
476	orofacial injuries. Pediatr Dent 2006;28(suppl):48-50.
477	13. Ranalli DN. Sports dentistry and dental traumatology. Dental Traumatol 2002;18(5):231-
478	6.
479	1814. Lin S, Levin L, Emodi O, Fuss Z, Peled M. Physician and emergency medical
480	technicians' knowledge and experience regarding dental trauma. Dental Traumatol
481	2006;22(3):124-6.
482	1715. Pacheco L, Filho P, Letra A, Menezes R, Villoria G, Ferreira S. Evaluation of the
483	knowledge of the treatment of avulsions in elementary school teachers in Rio de Janeiro,
484	Brazil. Dental Traumatol 2003;19(2):76-8.
485	16. Sae-Lim V, Chulaluk K, Lim LP. Patient and parental awareness of the importance of
486	immediate management of traumatized teeth. Endod Dent Traumatol 1999;15(1):37-41.
487	1517. Saroğlu I, Sönmez H. The prevalence of traumatic injuries treated in the pedodontic
488	clinic of Ankara University, Turkey, during 18 months. Dental Traumatol 2002;18(6):299-
489	303.
490	1918. Andreasen JO, Andreasen FM, Skeie A, Hjørting-Hansen E, Schwartz O. Effect of
491	treatment delay upon pulp and periodontal healing of traumatic dental injuries: A
492	review article. Dental Traumatol 2002;18(3):116-28.
493	2019. American Academy of Pediatric Dentistry. Policy on emergency oral care for infants,
494	children, and adolescents. Pediatr Dent 2007;29(suppl):21.
495	2120. DiScala C, Sege R, Guohua L, Reece RM. Child abuse and unintentional injuries. Arch
496	Pediatr Adolesc Med. 2000;154(1):16-22.
497	2221. Andreasen JO, Andreasen FM. Essentials of Traumatic Injuries to the Teeth. 2 nd ed.
498	Copenhagen, Denmark: Munksgaard and Mosby; 2000:9-154.
499	2322. Day P, Duggal M. A multicentre investigation into the role of structured histories for
500	patients with tooth avulsion at their initial visit to a dental hospital. Dental Traumatol
501	2003;19(5):243-7.
502	2423. Holan G, McTigue D. Introduction to dental trauma: Managing traumatic injuries in
503	the primary dentition. In: Pinkham JR, Casamassimo PS, Fields HW Jr, McTigue DJ,

- Nowak A, eds. Pediatric Dentistry: Infancy through Adolescence. 4th ed. St. Louis, Mo:
 Elsevier Saunders; 2005: 236-56.
- 506 <u>24.Andreasen J, Andreasen F. Textbook and Color Atlas of Traumatic Injuries to the Teeth.</u>
 507 <u>4th ed. Copenhagen, Denmark: Munksgaard; 2007.</u>
- 508 25. Borum M, Andreasen JO. Sequelae of trauma to primary maxillary incisors. 1.
 509 Complications in the primary dentition. Endod Dent Traumatol 1998;14(1):31-44.
- 510 26. Fried I, Erickson P. Anterior tooth trauma in the primary dentition: Incidence,511 classification, treatment methods, and sequelae: A review of the literature. J Dent Child
 - 1995(4):256-61.

512

- 513 2827. Ravn JJ. Sequelae of acute mechanical trauma in the primary dentition. J Dent Child
 514 1968;35(4):281-9.
- 515 2728. Soporowski NJ, Allred EN, Needleman HL. Luxation injuries of primary anterior
 516 teeth: Prognosis and related correlates. Pediatr Dent 1994;16(2):96-101.
- 517 29. Andreasen JO, Andreasen FM. Textbook and Color Atlas of Traumatic Injuries to the
 518 Teeth. 3rd ed. Copenhagen, Denmark: Munksgaard; 1994:219-425, 750.
- 519 3029. American Academy of Pediatric Dentistry. Pediatric Dental Trauma Card-Primary
 520 Teeth, Permanent Teeth. Chicago, Ill: American Academy of Pediatric Dentistry; 2002:2.
- 521 <u>30. Borum M, Andreasen J. Sequelae of trauma to primary maxillary incisors.</u>
 522 Complications in the primary dentition. Endod Dent Traumatol 1998;14(1):31-44.
- 523 31. Christophersen P, Freund M, Harild L. Avulsion of primary teeth and sequelae on the
 524 permanent successors. Dental Traumatol 2005;21(6):320-3.
- 32. McTigue DJ. Managing traumatic injuries in the young permanent dentition. In:
 Pinkham JR, Casamassimo PS, Fields HW Jr, McTigue DJ, Nowak A, eds. Pediatric
 Dentistry: Infancy through Adolescence. 4th ed. St. Louis, Mo: Elsevier Saunders;
 2005:593-607.
- 529 <u>33. Macedo G, Ritter A. Essentials of rebonding tooth fragments for the best functional and esthetic outcomes. Pediatr Dent 2009;31(2):110-16.</u>
- 531 37<u>34</u>. Cengiz S<u>B</u>, Atac A<u>S</u>, Cehreli Z<u>C</u>. Biomechanical effects of splint types on traumatized
 532 tooth: A photoelastic stress analysis. Dental Traumatol 2006;22(3):133-8.
- 533 35. McDonald N, Strassler HE. Evaluation for tooth stabilization and treatment of
 534 traumatized teeth. Dent Clin North Am. 1999;43(1):135-49.
- 535 3336. Olikarinen K. Tooth splinting: Review of the literature and consideration of the versatility of a wire composite splint. Endod Dent Traumatol 1990;6(6):237-50.
- 537 34<u>37</u>. Olikarinen K, Andreasen JO, Andreasen FM. Rigidity of various fixation methods
 538 used as dental splints. Endod Dent Traumatol 1992;8(3):113-9.
- 539 3638. von Arx T, Filippi A, Lussi A. Comparison of a new dental trauma splint device
 540 (TTS) with three commonly used splinting techniques. Dental Traumatol 2001;17(6):266541 74.
- 542 3839. Cvek M, Andreasen J, Borum M. Healing of 208 intraalveolar root fractures in patients aged 7-17 years. Dental Traumatol 2001;17(2):53-62.
- 40. Ravn JJ. Follow-up study of permanent incisors with enamel cracks as a result of acute
 trauma. Scand J Dent Res 1981;89(2):117-23.
- 546 39<u>41</u>. Robertson A. A retrospective evaluation of patients with uncomplicated crown fractures and luxation injuries. Endod Dent Traumatol 1998;14(6):245-56.

- 548 41<u>42</u>. Olsburgh S, Jacoby T, Krejci I. Crown fractures in the permanent dentition: Pulpal and restorative considerations. Dental Traumatol 2002;18(3):103-15.
- 4243. Ravn JJ. Follow-up study of permanent incisors with enamel fractures as a result of acute trauma. Scand J Dent Res 1981;89(3):213-7.
- 4344. Ravn JJ. Follow-up study of permanent incisors with enamel-dentin fractures as a result of acute trauma. Scand J Dent Res 1981;89(5):355-65.
- 554 44. Cvek M. A clinical report on partial pulpotomy and capping with calcium hydroxide in
 555 permanent incisors with complicated crown fractures. J Endod 1978;4(8):232-7.
- 45. Jackson N, Waterhouse P, Maguire A. Factors affecting treatment outcomes following
 complicated crown fractures managed in primary and secondary care. Dental
 Traumatol 2006;22(4):179-85.
- 559 46. Freely L, Mackie IC, Macfarlane T. An investigation of root-fractured permanent incisor teeth in children. Dental Traumatol 2003;19(1):52-4.
- 47. Andreasen J, Andreasen F, Mejàre I, Cvek M. Healing of 400 intra-alveolar root
 fractures. 1. Effect of pre-injury and injury factors such as sex, age, stage of root
 development, fracture type, location on fracture and severity of dislocation. Dental
 Traumatol 2004;20(4):192-202.
- 48. Andreasen JO, Andreasen FM, Mejàre I, Cvek M. Healing of 400 intraalveolar root
 fractures. 2. Effect of treatment factors such as treatment delay, repositioning, splinting
 type and period of antibiotics. Dental Traumatol 2004;20(4):203-11.
- 568 49. Crona-Larsson G, Bjarnason S, Norén J. Affect of luxation injuries on permanent teeth.
 569 Endod Dent Traumatol. 1991;7(5):199-206.
- 570 50. Nikoui M, Kenny D, Barrett E. Clinical outcomes for permanent incisor luxations in a pediatric population. III. Lateral luxations. Dental Traumatol 2003;19(5):280-5.
- 572 51. Humphrey J, Kenny D, Barrett E. Clinical outcomes for permanent incisor luxations in a pediatric population. I. Intrusions. Dental Traumatol 2003;19(5):266-73.
- 574 52. Gondim JO, Moreira Neto JJ. Evaluation of intruded primary incisors. Dental
 575 Traumatol 2005;21(3):131-3.
- 576 53. Holan G, Ram D. Sequelae and prognosis of intruded primary incisors: A retrospective
 577 study. Pediatr Dent 1999;21(4):242-7.
- 578 54. Andreasen JO, Bakland L, Andreasen FM. Traumatic intrusion of permanent teeth. Part
 579 2. A clinical study of the effect of preinjury and injury factors, such as sex, age, stage of
 580 root development, tooth location, and extent of injury including number of intruded
 581 teeth on 140 intruded permanent teeth. Dental Traumatol 2006;22(2):90-8.
- 55. Andreasen JO, Bakland L, Andreasen FM. Traumatic intrusion of permanent teeth. Part
 3. A clinical study of the effect of treatment variables such as treatment delay, method of
 repositioning, type of splint, length of splinting and antibiotics on 140 teeth. Dental
 Traumatol 2006;22(2):99-111.
- 586 56. Lee R, Barrett E, Kenny D. Clinical outcomes for permanent incisor luxations in a pediatric population. Dental Traumatol 2003;19(5):274-9.
- 588 <u>57. Flores M, Andreasen J, Bakland L, Feiglin B, Gutmann J, Oikarinen K, et al. Guidelines</u>
 589 <u>for the evaluation and management of traumatic dental injuries (part 2 of the series).</u>
 590 <u>Dental Traumatol 2001;17(2):49-52.</u>

- 591 57<u>58</u>. American Association of Endodontists. Treatment of the avulsed permanent tooth.
 592 Recommended guidelines of the American Association of Endodontists. Dent Clin
 593 North Am 1995;39(1):221-5.
- 58. Andreasen JO, Borum MK, Jacobsen HL, Andreasen FM. Replantation of 400 avulsed
 permanent incisors: 1. Diagnosis of healing complications. Endod Dent Traumatol
 1995;11(2):51-8.
- 597 59. Andreasen JO, Borum MK, Jacobsen HL, Andreasen FM. Replantation of 400 avulsed
 598 permanent incisors: 2. Factors related to pulpal healing. Endod Dent Traumatol
 599 1995;11(2):59-68.
- 600 60. Andreasen JO, Borum MK, Jacobsen HL, Andreasen FM. Replantation of 400 avulsed
 601 permanent incisors: 3. Factors related to root growth. Endod Dent Traumatol 1995;
 602 11(2):69-75.
- 603 6359. Barrett EJ, Kenny DJ. Avulsed permanent teeth: A review of the literature and treatment guidelines. Endod Dent Traumatol. 1997;13(4):153-63.
- 605 606 60. McIntyre J, Lee J, Trope M, Vann WJ. Permanent tooth re-plantation following avulsion: Using a decision-tree to achieve the best outcome. Pediatr Dent 2009;31(2):137-44.
- 607 64<u>61</u>. Trope M. Clinical management of the avulsed tooth: Present strategies and future
 608 directions. Dental Traumatol 2002;18(1):1-11.
- 609 6662. Sigalas E, Regan J, Kramer P, Witherspoon D, Opperman L. Survival of human
 610 periodontal ligament cells in media proposed for transport of avulsed teeth. Dental
 611 Traumatol 2004;20(1):21-8.
- 612 6163. Andreasen JO, Borum MK, Jacobsen HL, Andreasen FM. Replantation of 400 avulsed
 613 permanent incisors: 4. Factors related to periodontal ligament healing. Endod Dent
 614 Traumatol. 1995;11(2):76-89.
- 615 6264. Barrett EJ, Kenny DJ. Survival of avulsed permanent maxillary incisors in children
 616 following delayed replantation. Endod Dent Traumatol. 1997;13(6):269-75.

617 65. American Academy of Pediatric Dentistry. Decision tree for an avulsed tooth. Pediatr
 618 Dent 2007;29(suppl):264.

- 619 67<u>65</u>. Hiltz J, Trope M. Vitality of human lip fibroblasts in milk, Hank's Balanced Salt
 620 Solution, and Viaspan storage media. Endod Dent Traumatol 1991;7(2):69-72.
- 621 6866. Pohl Y, Filippi A, Kirschner H. Results after replantation of avulsed permanent teeth.
 622 II. Periodontal healing and the role of physiologic storage and antiresorptive-
- regenerative therapy. Dental Traumatol 2005;21(2):93-101.
- 69<u>67</u>. Chappuis V, von Arx T. Replantation of 45 avulsed permanent teeth: A 1-year follow up study. Dental Traumatol 2005;21(5):289-96.
- 626 68.Donaldson M, Kinirons M. Factors affecting the time of onset of resorption in avulsed
 627 and replanted incisor teeth in children. Dental Traumatol 2001;17(5):205-09.
- 628 70<u>69</u>. Barrett E, Kenny D, Tenenbaum H, Sigal M, Johnston D. Replantation of permanent incisors in children using Emdogain[®]. Dental Traumatol 2005;21(5):269-75.
- 630 7470. Malmgren B, Malmgren O. Rate of infraposition of reimplanted ankylosed incisors
 631 related to age and growth in children and adolescents. Dental Traumatol 2002;18(1):28632 36.
- 633 <u>71. Kling M, Cvek M, Mejare I. Rate of predictability of pulp revascularization in</u>
 634 <u>therapeutically reimplanted permanent incisors. Endod Dent Traumatol 1986;2(3):83-</u>
 635 <u>89.</u>

636	72. Cvek M, Cleaton-Jones P, Austin J, Lownie J, Kling M, Fatti P. Pulp revascularization in
637	reimplanted immature monkey incisors - predictability and the effect of antibiotic
638	systemic prophylaxis. Endod Dent Traumatol 1990;6(4):157-69.
639	7673. Cvek M, Cleaton-Jones P, Austin J, Lownie J, Kling M, Fatti P. Effect of topical
640	application of doxycycline on pulp revascularization and periodontal healing in
641	reimplanted monkey incisors. Endod Dent Traumatol 1990;6(4):170-6.
642	74. Yanpiset K, Trope M. Pulp revascularization of replanted immature dog teeth after
643	different treatment methods. Endod Dent Traumatol 2000;16(5):211-17.
644	75. Kinirons M, Boyd D, Gregg T. Inflammatory and replacement resorption in reimplanted
645	permanent incisor teeth: a study of the characteristics of 84 teeth. Endod Dent
646	Traumatol 1999;15(6):269-72.
647	76. Nyman S, Houston F, Sarhed G, Lidhe J, Karring T. Healing following reimplantation of
648	teeth subjected to root planing and citric acid treatment. J Clin Periodontol
649	<u>1985;12(4):294-305.</u>
650	77. Coccia C. A clinical investigation of root resorption rates in reimplanted young
651	permanent incisors: a five-year study. J Endod 1980;6(1):413-20.
652	8178. Bryson E, Levin L, Banchs F, Trope M. Effect of minocycline on healing of replanted
653	dog teeth after extended dry times. Dental Traumatol 2003;19(2):90-5.
654	7379. Filippi A, Pohl Y, von Arx T. Treatment of replacement resorption with Emdogain® –
655	Preliminary results after 10 months. Dental Traumatol 2001;17(3):134-8.
656	7480. Finucane D, Kinirons M. External inflammatory and replacement resorption of
657	luxated and avulsed replanted permanent incisors: A review and case presentation.
658	Dental Traumatol 2003;19(3):170-4.
659	<u>8081</u> . Khin Ma M, Sae-Lim V. The effect of topical minocycline on replacement resorption of
660	replanted monkeys' teeth. Dental Traumatol 2003;19(2):96-102.
661	7782. Maroto M, Barberia E, Planells P, Vera V. Treatment of a non-vital immature incisor
662	with mineral trioxide aggregate (MTA). Dental Traumatol 2003;19(3):165-9.
663	7983. Rafter M. Apexification: A review. Dental Traumatol 2005;21(1):1-8.
664	7584. Schjøtt M, Andreasen JO. Emdogain [®] does not prevent progressive root resorption
665	after replantation of avulsed teeth: A clinical study. Dental Traumatol 2005;21(1):46-50.
666	7285. Trope M. A futuristic look at dental trauma. New York, NY: Presentation at the
667	American Academy of Pediatric Dentistry 56th Annual Session; 2003.
668	7886. Villa P, Fernández R. Apexification of a replanted tooth using mineral trioxide
669	aggregate. Dental Traumatol 2005;21(5):306-8.
670	87. Andreasen J, Schwartz O, Kofoed T, Daugaard-Jensen J. Transplantation of premolars as
671	an approach for replacing avulsed teeth. Pediatr Dent 2009;31(2):129-32.
672	88. Sigurdsson A. Decoronation as an approach to treat ankylosis in growing children.
673	Pediatr Dent 2009;21(2):123-28.
674	89. Kindelan S, Day P, Kindelan J, Spencer J, Duggal M. Dental trauma: an overview of its
675	influence on the management of orthodontic treatment. Part 1. J Orthod
676	<u>2008;35(2):68-78.</u>
6//	<u>90. Malmgren O, Malmgren B. Orthodontic management of the traumatized dentition. In:</u>
678	Andreasen J, Andreasen F, Andersson L, editors. Textbook and Color Atlas of
679	<u>Traumatic Injuries to the Teeth. 4th ed. Ames, Iowa: Blackwell Munksgaard; 2007.</u>

680	91. Duggan D, Quinn F, O'Sullivan M. A long-term follow up of spontaneously healed root
681	fractures later subjected to orthodontic forces - two case reports. Dental Traumatol
682	<u>2008;24(2):231-34.</u>
683	92. Bauss O, Rohling J, Rahman A, Kiliaridis S. The effect of pulp obliteration on pulpal
684	vitality of orthodontically intruded traumatized teeth. J Endod 2008;34(4):417-20.
685	93. Bauss O, Rohling J, Sadat-Khonsari R. Influence of orthodontic intrusion on pulpal
686	vitality of previously traumatized maxillary permanent incisors. Am J Orthod
687	Dentofacial Orthop 2008;134(1):12-17.
688	94. Salam S, Caldwell S. Mouthguards and orthodontic patients. J Orthod 2008;35(4):270-75.
689	95. Sigurdsson A. Prevention of dental and oral injuries. In: Andreasen J, Andreasen F,
690	Andersson L, editors. Textbook and Color Atlas of Traumatic Injuries to the Teeth.
691	<u>4th ed. Ames, Iowa: Blackwell Munksgaard; 2007.</u>
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Fig 1: Clinical Management of Avulsed Permanent Incisors with an Open Apex



Fig 2: Clinical Management of Avulsed Permanent Incisors with a Closed Apex



1	Definition of Dental Neglect
2	
3	Originating Committee
4	Child Abuse Committee
5	
6	Review Council
7	Council on Clinical Affairs
8	
9	Adopted
10	1983
11	
12	Revised
13	1987, 1992
14	
15	Reaffirmed
16	1996, 2001, 2006 <u>, 2010</u>
17	
18	
19	Dental caries, periodontal diseases, and other oral conditions, if left untreated, can lead
20	to pain, infection, and loss of function. These undesirable outcomes can adversely affect
21	learning, communication, nutrition, and other activities necessary for normal growth
22	and development.
23	
24	Dental neglect is willful failure of parent or guardian to seek and follow through with
25	treatment necessary to ensure a level of oral heath essential for adequate function and

26 freedom from pain and infection.

- 1 Guideline on Acquired Temporomandibular Disorders in Infants, Children, and
- 2 Adolescents
- 3
- 4 Originating Committee
- 5 Clinical Affairs Committee Temporomandibular Joint Problems in Children Subcommittee
- 6 Review Council
- 7 Council on Clinical Affairs
- 8 Adopted
- 9 1990
- 10 Revised
- **11** 1999, 2002, 2006, <u>2010</u>
- 12
- 13 Purpose
- 14 The American Academy of Pediatric Dentistry (AAPD) recognizes that disorders of the
- 15 temporomandibular joint (TMJ) occasionally occur in infants, children, and adolescents.
- 16 This guideline is intended to assist the practitioner in the recognition and diagnosis of
- 17 temporomandibular disorders (TMD) and to identify possible treatment options. It is
- 18 beyond the scope of this document to recommend the use of specific treatment modalities.
- 19

20 Methods

- 21 This guideline was developed following the AAPD's 1989 symposium on TMD in children
- 22 and adolescents.¹-is an update of the previous document, revised in 2006. This revision is
- 23 based upon a review of current dental and medical literature related to TMD in children and
- 24 adolescents. A MEDLINE search was conducted using the terms "temporomandibular
- 25 disorder", "adolescent", and "children". The update included an electronic search using the
- 26 search terms "temporomandibular disorders", "TMJ dysfunction", "TMD and adolescents",
- 27 <u>"TMD and gender differences", "TMD and occlusion", and "TMD and treatment". Fields:</u>
- 28 <u>All Fields; Limits: Recent literature was limited to within the last fifteen years; humans;</u>
- 29 English; clinical trials. The reviewers agreed upon the inclusion of sixty-seven articles that
- 30 met the defined criteria. When data did not appear sufficient or were inconclusive,
- 31 recommendations were based upon expert and/or consensus opinion by experienced
- 32 <u>researchers and clinicians.</u>
- 33

34 Background

Council on Clinical Affairs 2009-10 Charge 1(j) G-Acquired Temporomandibular Disorders

35 36 Development of the TMJ 37 Function influences form as development and growth of the TMJ proceeds. The TMJ is 38 comprised of three major components: the mandibular condyle, the mandibular fossa, and 39 the associated connective tissue (including the articular disk).¹ The first evidence of 40 development of the TMJ in humans is seen at 8 weeks after conception.² During the first 41 decade of life, the mandibular condyle becomes less vascularized and most of the major 42 morphological changes are completed. The shape of the mandibular condyle may change 43 significantly during growth with approximately 5% undergoing radiographic changes in 44 shape between 12-16 years of age.³ During the second decade of life, there is continued but 45 progressive slowing of growth. From adolescence to adulthood, the condyle changes in 46 length and width going from a more rounded form to a form that is greater in width than 47 length. Although the TMJ experiences active growth in the first two decades, it undergoes 48 adaptive and remodeling changes throughout life. 49 50 Definition of TMD 51 Temporomandibular disorder is a term adopted by the American Dental Association in 1983 52 to facilitate coordination of research and communication.⁴ Okeson defines TMD as 53 "functional disturbances of the masticatory system."⁵ Sessle et al., included masticatory 54 muscle disorders, degenerative and inflammatory TMJ disorders and TMJ disk 55 displacements under the umbrella of TMD.⁶ Certain medical conditions are reported to 56 occasionally mimic TMD. Among them are trigeminal neuralgia, central nervous system 57 lesions, odontogenic pain, sinus pain, otological pain, developmental abnormalities, 58 neoplasias, parotid diseases, vascular diseases, myofascial pain, cervical muscle 59 dysfunction, and Eagle's syndrome. Other common medical conditions (eg, otitis media, 60 allergies, airway congestion, rheumatoid arthritis) can cause symptoms similar to TMD.⁷ 61 62 Etiology of TMD 63 Temporomandibular disorders have multiple etiological factors.⁸ In fact, the TMJ and 64 masticatory system is complex and thus requires a thorough understanding of the anatomy

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65	and physiology of the structural, vascular and neurological components in order to treat
66	TMD. Many studies show a poor correlation between any single etiological factor and
67	resulting signs and symptoms. ⁸ Alterations in any one or a combination of teeth, periodontal
68	ligament, the TMJ or the muscles of mastication can eventually lead to TMD. ⁹ Research is
69	insufficient to predict reliably which patient will or will not develop TMD. Etiologic factors
70	suggested as contributing to the development of TMD are:
71	1. Head and neck trauma Trauma: Impact injuries such as trauma to the chin. As a
72	common occurrence in childhood because of a fall, chin trauma is reported to be a
73	factor in the development of TMD in pediatric patients. ¹⁰⁻¹² Unilateral and bilateral
74	intracapsular or subcondylar fractures are the most common mandibular fractures in
75	children. ¹⁸ Closed reduction and prolonged immobilization can result in ankylosis. ^{14,15}
76	2. Occlusal factors: There is a relatively low association of occlusal factors and the
77	development of temporomandibular disorders. ^{16,17} However, several occlusal features
78	characterize <u>malocclusions:</u>
79	 skeletal anterior open bite; ¹⁸
80	 overjet greater than 6 to 7 mm; ¹⁸⁻²¹
81	• retrocuspal position (centric relation) to intercuspal position (centric occlusion) with
82	slides greater than 4 mm; ²²
83	 unilateral posterior-lingual cross bite; ^{18,23}
84	• 5 or more missing posterior teeth; ^{24,25}
85	<u>Class III malocclusion.²⁶</u>
86	3. Parafunctional habits: Bruxism, clenching, hyperextension, and any other repetitive
87	habitual behavior. Bruxism is thought to contribute to the development of TMD by joint
88	overloading that leads to cartilage breakdown, synovial fluid alterations and other
89	changes within the joint. These parafunctional habits may occur while the patient is
90	asleep or awake. A study of 854 patients less than 17 years old found the prevalence of
91	bruxism to be 38%. ²⁷ The literature on the association between parafunction and TMD in
92	pediatric patients is contradictory. ²⁸⁻³⁰ However, childhood parafunction was found to
93	be a predictor of the same parafunction 20 years later. ³¹ Others found correlations
94	between reported bruxism and TMD ³² with a 3.4 odds ratio ³³ and that 1 in 5 bruxers

95	report orofacial pain. Children who grind their teeth were found to complain more
96	often of pain and muscle tenderness when eating. ³⁴ . The literature on the association
97	between parafunction and TMD in pediatric patients is contradictory. ²³
98	4. Posture: Craniocervical posture has been associated with occlusion and with
99	dysfunction of the TMJ, including abnormalities of the mandibular fossa, condyle,
100	ramus, and disc. ³⁵⁻³⁷
101	5. Changes in "free-way" dimension of the rest position: Normally 2-4 mm. May be
102	impinged by occlusal changes, disease, muscle spasms, nervous tension, and/or
103	restorative prosthestics. ⁹
104	6. Orthodontic treatment: Current literature does not support that the development of
105	TMD is caused by orthodontic treatment. ^{19,38-41} regardless of whether premolars were
106	extracted prior to treatment.44
107	
108	Prevalence of TMD in children and adolescents
109	The reported prevalence of TMD in infants, children, and adolescents varies widely in the
110	literature. ⁴²⁻⁴⁵ Prevalence of signs and symptoms increases with age. One study of the
111	primary dentition reported 34% with signs and/or symptoms of TMD. ⁴⁶ An epidemiological
112	study of 4724 children aged 5-17 years resported 25% with symptoms. Clicking was seen in
113	2.7% of children in the primary dentition and increased to 10.1% in late mixed dentition and
114	further increased to 16.6% in patients with permanent dentition. ¹⁸ A similar study in
115	preschool children found TMJ sounds and clicking in 16.6% of the patients. ⁴⁷ A study of 217
116	adolescents found that over 20% had either signs and/or symptoms of dysfunction;
117	muscular tenderness exhibited wide variability whereas An epidemiological study of 440
118	children aged 7-14 reported 36% with symptoms (15% of those having recurrent headaches
119	and 13% clicking sounds).¹² More than half of the children (64%) claimed pain upon
120	palpation of the TMJ muscles. ¹² In a study of 285 17 year-olds, 62% had either signs or
121	symptoms of dysfunction, with fatigue in the jaw, TMJ sounds, and headache tenderness in
122	the lateral pterygoid muscle as were the most common findings. ⁴⁸ Clicking is seen more
123	frequently than either locking or luxation and affected girls more than boys. In general, the
124	prevalence of signs and symptoms of TMD are lower in children compared to adults and are

even less the younger the child but increase with increasing age.²³ Recent surveys have

125

126	indicated that significantly higher prevalence of symptoms and greater need for treatment
127	in girls than boys ⁴³ with the development of symptomatic TMD correlated with the onset of
128	puberty in girls. ^{49,50}
129	Controversy surrounds the significance of signs and symptoms in this age group, the
130	value of certain diagnostic procedures, and what constitutes effective therapy. It is not clear
131	whether these signs and symptoms constitute normal variation, preclinical features, or
132	manifestations of a disease state. Whether these signs and symptoms warrant treatment as
133	predictors of TMD in adulthood is questionable. ³¹
134	
135	Recommendations
136	<u>Diagnosis of TMD</u>
137	All comprehensive dental examinations should include a screening evaluation of the
138	temporomandibular joint (TMJ) and surrounding area. ⁵¹⁻⁵³ Diagnosis of TMD is based upon
139	a combination of historical information, clinical examination, and/or craniocervical and TMJ
140	imaging. ^{54,55} The findings are classified as symptoms (those reported by the child or parent)
141	and signs (those identified by the dentist during the examination). ⁵¹
142	For a positive diagnosis of TMD, patients must have a positive history of facial pain
143	combined with positive physical finding, supplemented by radiographic or imaging data
144	when indicated. ⁵⁶
145	A screening history, as part of the health history may include questions such as:57
146	 Do you have difficulty opening your mouth?
147	 Do you hear noises within your jaw joint?
148	 Do you have pain in or around your ears or your cheeks?
149	 Do you have pain when chewing?
150	 Do you have pain when opening your mouth wide or when yawning?
151	• <u>Has your "bite" felt uncomfortable or unusual?</u>
152	 Does your jaw ever "lock" or "go out"?
153	• Have you ever had an injury to your jaw, head or neck? If so, when? How was it
154	treated?

155	0	Have you previously been treated for a temporomandibular disorder? If so,
156		when? How was it treated?
157	Clinical a	nd physical assessment of the TMJ may include: ²¹
158	0	Manual palpation of the muscles and TMJ: to evaluate for tenderness of intraoral
159		and extraoral jaw muscles, neck muscles and TMJ capsule;
160	0	Evaluation of jaw movements including mandibular range of motion assessment
161		using a millimeter ruler: 1) maximum unassisted opening, 2) maximum assisted
162		opening, 3) maximum lateral excursion, and 4) maximum protrusive excursion.
163		5) mandibular opening pattern – is it symmetrical? Restricted manibular
164		opening combined with or without pain on mandibular movement may be
165		interpreted as signs of TMJ internal derangement.58
166	0	Determination of TMJ sounds by palpation and auscultation with a stethoscope
167	0	Radiographs (panoramic and full mouth), TMJ tomography, MRI, lateral
168		cephalometric X rays to examine for TMJ pathology and/or dental pathology.
169		TMJ arthography is not recommended as a routine diagnostic procedure. ^{4,57}
170	<u>Evaluatio</u>	n of pyscholgical etiologies
171	As some 1	mental disorders can greatly influence a patient's pain experience, psychosocial
172	factors rel	ated to temporomandibular symptoms should be considered including: mood
173	<u>disorders</u>	, anxiety disorders, musculoskeletal problems, migraine headaches, tension
174	headache	s, emotional factors, ulcers, colitis, occupational factors, and
175	<u>developm</u>	nental/acquired craniofacial anomalies. ⁹
176	<u>Differentia</u>	l Diagnosis of TMD
177	<u>There is a</u>	need for improved classification of TMDs, however they can largely be grouped
178	into three	<u>classes:</u>
179	1. <u>D</u>	isorders of the muscles of mastication (including protective muscle splinting,
180	<u>m</u>	uscle spasm, and muscle inflammation)
181	2. <u>Di</u>	sorders of the TMJ(including internal disk derangement, disk displacement with
182	re	duction accompanied by clicking, and anterior disk displacement without
183	re	duction seen as mechanical restriction or closed lock)

184 3. Disorders in other related areas that may mimic TMD such as chronic mandibular 185 hypomobility, inflammatory joint disorders such as juvenile rheumatoid arthritis, 186 degenerative joint disease, extrinsic trauma such as fracture.⁴ 187 Treatment of TMD 188 Few studies document success or failure of specific treatment modalities for TMD in infants, 189 children, and adolescents on a long-term basis. They suggest that simple, conservative, and 190 reversible types of therapy are effective in reducing most TMD symptoms in children.^{59, 64} 191 The goal of treatment should be to find a balance between active and passive modalities. 192 Active modalities include participation of the patient whereas passive modalities may 193 include wearing a stabilization splint. The most common form of treatment of TMD in 194 children was information combined with occlusal appliance therapy.⁶⁰ It has been shown 195 that combined treatment approaches are more successful at treating TMD than single 196 treatment modalities.^{60,61} Treatment of TMD can be divided into reversible treatment and 197 irreversible. 198 Reversible therapies include: 199 Patient education: This may include relaxation training, developing behavior coping 200 strategies, modifying inadequate perceptions about TMD; patient awareness of 201 clenching and bruxing habits, if present 57 202 • Physical therapy: Jaw exercises or transcutaneous electrical nerve stimulation 203 (TENS), ultrasound, iontophoresis, massage, thermotherapy and coolant therapy 204 5,62,63 205 Behavioral therapy: (eg, eliminating chewing gum)avoiding excessive chewing of • 206 hard foods or gum; decreasing stress, anxiety and/or depression; voluntary 207 avoidance of stressors and habit reversal 64 208 Prescription medications: Non-steroidal anti-inflammatory drugs, anxiolytic agents, • 209 or muscle relaxers. Anti-depressants have proved to be beneficial, however should 210 be prescribed by a physician.⁶⁵ 211 • Occlusal splints: The goal of an occlusal appliance is to provide orthopedic stability 212 to the TMJ. These alter the patient's occlusion temporarily and may be used to 213 decrease parafunctional activity.^{60,66,67}

- 214 Irreversible therapies can include:
- Occlusal adjustment: <u>This permanently alters the occlusion or mandibular position</u>
 by selective grinding or full mouth restorative dentistry
- Mandibular repositioning: <u>This is designed to alter the growth or permanently</u>
- 218 reposition the mandible. Examples are headgear and functional appliances.
- Orthodontics
- 220 <u>Referral to other specialists: TMD specialists, oral surgeons, pain specialists</u>
- 221

222 Recommendations

223 Every comprehensive dental history and examination should include a TMJ history and 224 assessment. The history should include questions concerning the presence of head and neck 225 pain and mandibular dysfunction, previous orofacial trauma, and history of present illness 226 with an account of current symptoms. In the presence of a positive history and/or signs 227 and symptoms of TMD, additional information is suggested and a referral may be 228 considered. A more comprehensive exam should be done. The This examination should 229 may include palpation of masticatory and associated muscles and the TMJ's, documentation 230 of joint sounds, occlusal analysis, and assessment of range of mandibular movements 231 including maximum opening, protrusion, and lateral excursions. 232 Joint imaging is indicated on a selected basis may be recommended by other specialists 233 to investigate for joint sounds in the absence of other TMD signs and symptoms. For 234 example, the presence of crepitus may indicate degenerative change that is not yet painful.

- Therapeutic modalities to prevent TMD in the pediatric population are yet to be
 supported by controlled studies. For children and adolescents with signs and symptoms of
 TMD, reversible therapies should be considered. Because of inadequate data regarding their
 usefulness, irreversible therapies should be avoided.^{59,67}
- Referral to a medical specialist may be indicated when otitis media, allergies, abnormal
 posture, airway congestion, rheumatoid arthritis, or other medical conditions <u>are present</u> or
 suspected.⁵⁷
- 242
- 243

244 References

245 1	Carlson DS. Growth of the Temporomandibular Joint. In: Zarb GAC, G.E.; Sessle BJ,
246	Mohl ND, eds. Temporomandibular Joint and Masticatory Muscle Disorders 2nd ed.
247	Copenhagen: Munksgaard; 1994. p. 128-50.
248 2	Dixon AD. Formation of the Cranial Base. In: Dixon AD, Howyte DAN, Rönning O.
249	eds. Fundamentals of Craniofacial Growth. Boca Raton, FL; CRC press, 1997: 100-29.
250 3	Dibbet JMH and Van der Weele LT. Prevalence of structural bony change in the
251	mandibular condyle. J Craniomand Dis Fac Oral Pain 1992;6:254-9.
252 4	Griffiths R. Report of the President's Conference on the Examination, Diagnosis and
253	Management of Temporomandibular Disorders. J Am Dent Assoc 1983;106(1):75-7.
254 5	Okeson J. Management of Temporomandibular Disorders and Occlusion. 6th ed. St.
255	Louis: Mosby Year Book Publication, Inc.;2008:130-163.
256 6	Sessle BB, Bryant PS; Dionne RA, editors. Temporomandibular Disorders and Related
257	Pain Conditions. Seattle: IASP Press; 1995. Progress in Pain Research and
258	Management;Vol. 4: 3-30;79-113;119-132.
259 7	Loos PJ, Aaron GA. Standards for management of the pediatric patient with acute pain
260	in the temporomandibular joint or muscles of mastication. Pediatr Dent 1989;11(4):331.
261 8	Greene CS. The etiology of temporomandibular disorders: Implications for treatment. J
262	<u>Orofac Pain 2001;15:93-105.</u>
263 9	Hiatt JL, Gartner LP. Textbook of Head and Neck Anatomy. 2nd ed. Baltimore, MD;
264	Williams and Wilkins; 1987:223.
265 1). Greco CM, Rudy TE, Turk DC, Herlich A, Zaki HH. Traumatic onset of
266	temporomandibular disorders: Positive effects of a standardized conservative
267	treatment program. Clin J Pain 1997;13(4):337-47.
268 1	1. Fischer DJ, Mueller BA, Critchlow CW, LeResche L. The association of
269	temporomandibular disorder pain with history of head and neck injury in adolescents.
270	<u>J Orofac Pain 2006;20(3):191-8.</u>
271 1	2. Imahara SD, Hopper RA, Wang J, Rivara FP, Klein MB. Patterns and outcomes of
272	pediatric facial fractures in the United States: A survey of the National Trauma Data
273	<u>Bank. J Am Col of Surg 2008;207(5): 710-6.</u>
274 1	3. Posnick JC, Wells M, Pron GE. Pediatric facial fractures: Evolving patterns of treatment.
275	J Oral Maxillofac Surg 1993;51(5):836-44; discussion 844-5.
276 1	4. Kaban L. Acquired abnormalities of the temporomandibular joint. In: Kaban L, Troulis
277	M, eds. Pediatric Oral and Maxillofacial Surgery. Philadelphia, Pa: Saunders; 2004:340-
278	76.
279 1	5. <u>Güven, O. A clinical study on temporomandibular joint ankylosis in children. J</u>
280	<u>Craniofac Surg 2008;19(5):1263-9.</u>
281 1	6. De Boever JA, Carlsson GE, Klineberg IJ. Need for occlusal therapy and prosthodontic
282	treatment in the management of temporomandibular disorders. Part I. Occlusal
283	interference and occlusal adjustment. J Oral Rehabil 2000;27:367-79.
284 1	7. <u>Taskaya-Yilmaz N, Öğütcen-Toller M, Saraç YŞ. Relationship between the TMJ disc and</u>
285	condyle position on MRI and occlusal contacts on lateral excursions in TMD patients. J
286	Oral Rehab 2004; 31:754-8.
287 1	8. <u>Thilander B, Rubio G, Pena L, De Mayorga C. Prevalence of temporomandibular</u>
288	dysfunction and its association with malocclusion in children and adolescents: An

289		epidemiologic study related to specified stages of dental development. Angle Orthod
290		<u>2002;72(2):146-54.</u>
291	19.	Henrikson T, Nilner M. Temporomandibular disorders, occlusion and orthodontic
292		treatment. J Orthod 2003;30(2):129-37; discussion 27.
293	20.	Phillips JT. What skeletal and dental characteristics do TMD patients have in common?
294		Funct Orthod 2007;24(1):24-6,28,30
295	21.	Pahkala R, Qvarnström. Can temporomandibular dysfunction signs be predicted by
296		early morphological or functional variables? Euro J Orthod 2004;26(4):367-73.
297	22.	Pullinger AG, Seligman DA. Quantification and validation of predictive values of
298		occlusal variables in temporomandibular disorders using a multifactorial analysis.[see
299		<u>comment]. J Prosthet Dent 2000;83(1):66-75.</u>
300	23.	Seligman DA, Pullinger AG. Analysis of occlusal variables, dental attrition, and age for
301		distinguishing healthy controls from female patients with intracapsular
302		temporomandibular disorders.[see comment]. J Prosthet Dent 2000;83(1):76-82.
303	24.	Ciancaglini R, Gherlone EF, Radaelli G. Association between loss of occlusal support
304		and symptoms of functional disturbances of the masticatory system. J Oral Rehabil
305		1999;26(3):248-53.
306	25.	Tallents RH, Macher DJ, Kyrkanides S, Katzberg RW, Moss ME. Prevalence of missing
307		posterior teeth and intraarticular temporomandibular disorders. J Prosthet Dent
308		2002;87(1):45-50.
309	26.	Rey D, Oberti G, Baccetti T. Evaluation of temporomandibular disorders in Class III
310		patients treated with mandibular cervical headgear and fixed appliances. Am J Orthod
311		Dentofac Orthop 2008;133(3):379-81.
312	27.	Cheifetz AT, Osganian SK, Allred EN, Needleman HL. Prevalence of bruxism and
313		associated correlates in children as reported by parents. J Dent Child 2005;72(2):67-73.
314	28.	Barbosa Tde S, Miyakoda LS, Pocztaruk RdL, Rocha CP, Gavião MBD.
315		Temporomandibular disorders and bruxism in childhood and adolescence: Review of
316		the literature. Int J Pediatr Otorhinolaryngol 2008;72(3):299-314.
317	29.	Castelo PM, Gaviao MB, Pereira LJ, Bonjardim LR, Gavião MBD. Relationship between
318		oral parafunctional/nutritive sucking habits and temporomandibular joint dysfunction
319		in primary dentition. Int J Paediatr Dent 2005;15(1):29-36.
320	30.	Winocur E, Gavish A, Finkelshtein T, Halachmi M, Gazit E. Oral habits among
321		adolescent girls and their association with symptoms of temporomandibular disorders.
322		<u>J Oral Rehabil 2001;28(7):624-9.</u>
323	31.	Carlsson GE, Egermark I, Magnusson T. Predictors of signs and symptoms of
324		temporomandibular disorders: A 20-year follow-up study from childhood to
325		adulthood. Acta Odontol Scand 2002;60(3):180-5.
326	32.	Magnusson T, Egermarki I, Carlsson GE, Magnusson T, Egermarki I, Carlsson GE. A
327		prospective investigation over two decades on signs and symptoms of
328		temporomandibular disorders and associated variables. A final summary. Acta
329		Odontol Scand 2005;63(2):99-109.
330	33.	Gesch D, Bernhardt O, Mack F, et al. Association of malocclusion and functional
331		occlusion with subjective symptoms of TMD in adults: results of the Study of Health in
332		Pomerania (SHIP). Angle Orthod 2005;75(2):183-90.
333	34.	Alamoudi N. Correlation between oral parafunction and temporomandibular disorders

334		and emotional status among Saudi children. J Clin Pediatr Dent 2001;26(1):71-80.
335	35.	Sonnesen L, Bakke B, Solow B. Temporomandibular disorder in relation to craniofacial
336		dimensions, head posture and bite force in children selected for orthodontic treatment.
337		Eur J Orthod 2001;20(2):179-92.
338	36.	Kondo E, Nakahara R, Ono M, et al. Cervical spine problems in patients with
339		temporomandibular disorder symptoms: An investigation of the orthodontic treatment
340		effects for growing and nongrowing patients. World J Orthod 2002;3:295-312.
341	37.	Motovshi M, Shimazaki T, Namura S. Biomechanical influences of head posture on
342		occlusion: An experimental study using finite element analysis. Eur J Orthod
343		2002:24(4):319-26.
344	38.	Egermark I, Carlsson GE, Magnusson T. A prospective long-term study of signs and
345		symptoms of temporomandibular disorders in patients who received orthodontic
346		treatment in childhood. Angle Orthod 2005;75(4):645-50.
347	39.	Henrikson T, Nilner M, Kurol J. Symptoms and signs of temporomandibular disorders
348		before, during and after orthodontic treatment. Swed Dent J 1999;23(5-6):193-207.
349	40.	Henrikson T, Nilner M, Kurol J. Signs of temporomandibular disorders in girls
350		receiving orthodontic treatment. A prospective and longitudinal comparison with
351		untreated Class II malocclusions and normal occlusion subjects. Eur J Orthod
352		2000:22(3):271-81.
353	41.	Kim MR, Graber TM, Viana MA, Orthodontics and temporomandibular disorder: A
354		meta-analysis. Am I Orthod Dentofac Orthop 2002;121(5):438-46.
355	42.	Alamoudi N, Farsi N, Salako NO, Feteih R, Temporomandibular disorders among
356		school children. I Clin Pediatr Dent 1998:22(4):323-8.
357	43.	List T, Wahlund K, Wenneberg B, Dworkin SF. TMD in children and adolescents:
358		Prevalence of pain, gender differences, and perceived treatment need. I Orofac Pain
359		1999:13(1):9-20.
360	44.	Stockstill IW, Bowley IF, Dunning D, Spalding P, Stafford K, Erickson L, Prevalence of
361		temporomandibular disorders in children based on physical signs. I Dent Child
362		1998:65(6):459-67.
363	45.	Paesani D, Salas E, Martinez A, Isberg A. Prevalence of temporomandibular joint disk
364		displacement in infants and young children. Oral Surg Oral Med Oral Pathol Oral
365		Radiol Endod 1999:87(1):15-9.
366	46.	Bonjardim L.R. Bayjao MB. Carmagnani FG. Pereira L.F. Castelo PM. Signs and
367	10.	symptoms of temporomandibular joint dysfunction in children with primary dentition
368		I Clin Pediatr Dent 2003:28(1):53-8
369	47	Widmalm SE, Christiansen RL, Gunn SM, Crepitation and clicking as signs of TMD in
370		preschool children. Cranio 1999:17(1):58-63.
371	48.	Bonjardim L.R. Gavião MB, Pereira LL, Castelo PM, Garcia RC, Signs and symptoms of
372		temporomandibular disorders in adolescents. Braz Oral Res 2005;19(2):93-8.
373	49.	LeResche L. Mancl LA. Drangsholt MT. Saunders K. Von Korff M. Relationship of pain
374		and symptoms to pubertal development in adolescents. Pain 2005:118(1-2):201-9.
375	50.	LeResche L, Mancl LA, Drangsholt MT, Huang G, von Korff MV. Predictors of onset of
376		facial pain and temporomandibular disorders in early adolescence. Pain
377		2007;129(3):269-78.
		· · · · · · · · · · · · · · · · · · ·

378	51.	McDonald RE, Avery DR, Dean JA. Examination, diagnosis, and treatment planning.
379		In: Dentistry for the Child and Adolescent. 8 th ed. St. Louis, Mo: Mosby; 2004:1-22.
380	52.	Casamassimo PS, Christensen JR, Fields HW Jr, Ganzberg S. Examination, diagnosis,
201		Casamassima DC MaTigua DL Eiglda HW/Jr Naval, AL ada Dadiatria Dantiatru
302 202		Casamassimo PS, McTigue DJ, Fields HW Jr, Nowak AJ, eds. Fediatric Dentistry:
303 204	F 0	Infancy through Adolescence. 4 ^{ar} ed. St. Louis, Mo; Elsvier Saunders 2005:661-89.
304 205	55.	American Academy of Pediatric Dentistry. Guideline on recordicepting. Pediatr Dent
300	⊏ 4	2005;27(suppl):176-81.
380	54.	American Academy of Orofacial Pain. Assessment of orofacial pain disorders. In:
387		Okeson J, ed. Orofacial Pain: Guidelines for Assessment, Diagnosis, and Management.
388		Carol Stream, III: Quintessence Publishing Co Inc. 1996:19-44.
389	55.	Wahlund K, List I, Dworkin SF. Temporomandibular disorders in children and
390		adolescents: Reliability of a questionnaire, clinical examination, and diagnosis. J Orofac
391	- /	Pain 1998;12(1):42-51.
392	56.	De Boever JA, Nilner M, Orthlieb JD, Steenks MH. Educational Committee of the
393		European Academy of Craniomandibular Disorders. Recommendations by the EACD
394		tor examination, diagnosis, and management of patients with temporomandibular
395		disorders and orofacial pain by the general dental practitioner. Orofac Pain
396		<u>2008;22(3):268-78.</u>
397	57.	Brooks, SL, Brand JW, Gibbs SJ, et al. Imaging of the tempromandibular joint: A
398		position paper of the American Academy of Oral and Maxillofacial Radiology. Oral
399		Surg Oral Med Oral Pathol Oral Radiol Endod 1997;83:609-18.
400	58.	Hu YS, Schneiderman ED, Harper RP. The temporomandibular joint in juvenile
401		rheumatoid arthritis: Part II. Relationship between computed tomographic and clinical
402		<u>findings. Pediatr Dent 1996;18(4):312-9.</u>
403	59.	Bodner L, Miller VJ. Temporomandibular joint dysfunction in children: Evaluation of
404		treatment. Int J Pediatr Otorhinolaryngol 1998;44(2):133-7.
405	60.	Wahlund K, List T, Larsson B. Treatment of temporomandibular disorders among
406		adolescents: a comparison between occlusal appliance, relaxation training, and brief
407		information. Acta Odontol Scand 2003;61(4):203-11.
408	61.	Kurita H, Kurashina K, Kotani A. Clinical effect of full coverage occlusal splint therapy
409		for specific temporomandibular disorder conditions and symptoms. J Prosthet Dent
410		<u>1997;78(5):506-10.</u>
411	62.	Michelotti A, Steenks MH, Farella M, Parisini F, Cimino R, Martina R. The additional
412		value of a home physical therapy regimen versus patient education only for the
413		treatment of myofascial pain of the jaw muscles: short-term results of a randomized
414		clinical trial.[erratum appears in J Orofac Pain. 2006 Spring;20(2):106]. J Orofac Pain
415		<u>2004;18(2): 114-25.</u>
416	63.	Medlicott, MS, Harris SR. A systematic review of the effectiveness of exercise, manual
417		therapy, electrotherapy, relaxation training, and biofeedback in the management of
418		temporomandibular disorder.[see comment]. Phys Ther 2006;86(7): 955-73.
419	64.	Crider, AB, Glaros AG. A meta-analysis of EMG biofeedback treatment of
420		temporomandibular disorders. J Orofac Pain 1999;13(1): 29-37.

421	65. List T, Axelsson S, Leijon G. Pharmacologic interventions in the treatment of
422	temporomandibular disorders, atypical facial pain, and burning mouth syndrome. A
423	qualitative systematic review. J Orofac Pain 2003;17(4):301-10.
424	66. Fujii T, Torisu T, Nakamura S. A change of occlusal conditions after splint therapy for
425	bruxers with and without pain in the masticatory muscles. Cranio 2005;23(2):113-8.
426	67. Koh H, Robinson PG. Occlusal adjustment for treating and preventing
427	temporomandibular joint disorders. The Cochrane Database of Systematic Reviews
428	2003;1:Art. No. CD003812. DOP: 10.1002/146751858.CD003812.
429	12. Nilner M, Lassing SA. Prevalence of functional disturbances and diseases of the
430	stomatognathic system in 7-14 year olds. Swed Dent 1981;5(5-6):173-87.
431	13. Wanman A, Agerberg G. Relationship between signs and symptoms of mandibular
432	dysfunction in adolescents. Community Dent Oral Epidemiol 1986;14(4):225-30.
433	14. Green CS. Etiology of temporomandibular disorders. Seminar Orthod 1995;1:222-8.
434	15. Pullinger AG, Seligman DA. Trauma history in diagnostic groups of
435	temporomandibular disorders. Oral Surg Oral Med Oral Pathol 1991;71(5):529-34.
436	17. Kaban LB, Mulliken JB, Murray JE. Facial fractures in children: An analysis of 122
437	fractures in 109 patients. Plast Reconstr Surg 1977;59(1):15-20.
438	20. Pullinger AG, Seligman DA, Gornbein JA. A multiple logistic regression analysis of the
439	risk and relative odds of temporomandibular disorders as a function of common
440	occlusal features. J Dent Res 1993;72(6):968-79.
441	21. McNamara JA Jr, Seligman DA, Okeson JP. Occlusion, orthodontic treatment, and
442	temporomandibular disorders: A review. J Orofac Pain 1995;9:73-90.
443	22. Widmer CG, Evaluation of Temporomandibular Disorders. In: Krause SL, ed. TMJ
444	Disorders: Management of the Craniomandibular Complex. Clinics in Physical Therapy.
445	Vol 18. New York, NY. Churchill Livingstone Inc. 1988:105-9.
446	23. Widmalm SE, Christiansen RL, Gunn SM. Oral parafunctions as temporomandibular
447	disorder risk factors in chil-dren. Cranio 1995;13(4):242-6.
448	27. McNamara JA Jr, Turp JC. Orthodontic treatment and temporomandibular disorders: Is
449	there a relationship? Part 1: Clinical studies. J Orofac Orthop 1997;58(2):74-89.
450	28. Hirata RH, Heft MW, Hernandez B, King GJ. Longitudinal study of signs of
451	temporomandibular disorders (TMD) in orthodontically treated and nontreated groups.
452	Am J Orthod Dentofacial Orthop 1992;101(1):35-40.
453	31. Skeppar J, Nilner M. Treatment of craniomandibular disorders in children and young
454	adults. J Orofac Pain 1993;7(4):362-9.
455	

- 1 Policy on the Use of Xylitol in Caries Prevention 2 3 **Originating Council** 4 Council on Clinical Affairs 5 6 **Review Council** 7 Council on Clinical Affairs 8 9 Adopted 2006 10 Revised 2010 11 12 13 Purpose 14 The American Academy of Pediatric Dentistry (AAPD) recognizes the benefits of caries 15 preventive strategies involving sugar substitutes, particularly xylitol, on the oral health of 16 infants, children, adolescents, and persons with special health care needs. This policy is 17 intended to assist oral health care professionals make informed decisions about the use of 18 xylitol-based products in caries prevention. 19 20 Methods 21 A MEDLINE literature search was conducted using the terms "xylitol AND dental caries", 22 "caries prevention", "plaque reduction", "maternal Streptococcus mutans (MS) 23 transmission", and "Streptococcus mutans long term suppression with xylitol". 24 This policy is an update of the previous policy, adopted in 2006. The update is based upon a 25 review of current dental and medical literature related to the use of xylitol in caries 26 prevention. A MEDLINE search was conducted using PUBMED with the following: 27 TERMS: "xylitol", "caries prevention"; FIELD: All Fields; LIMITS: within the last 10 years; 28 humans; English; birth through 18. 240 of articles matched these criteria. 25 papers were 29 reviewed at this revision. When data did not appear sufficient or were inconclusive, 30 recommendations were based upon expert and/or consensus opinion by experienced 31 researchers and clinicians. 32 33 Background
- 34 Xylitol is a five-carbon sugar alcohol derived primarily from forest and agricultural
- 35 materials. It has been used since the early 1960's in infusion therapy for post-operative,

36 burn, and shock patients; in the diet of diabetic patients; and, most recently, as a sweetener 37 in products aimed at improved oral health.¹ Dental benefits of xylitol first were recognized 38 in Finland in 1970, using animal models.² The first chewing gum developed with the aim of 39 reducing caries and improving oral health was released in Finland in 1975 and in the United 40 States shortly after. The first xylitol studies in humans, known as the Turku Sugar 41 Studies,^{3,4} demonstrated the relationship between dental plaque and xylitol, as well as the 42 safety of xylitol for human consumption. These early studies showed the decayed, missing, 43 and filled (dmf) incidence in teeth in a sucrose chewing-gum group was 2.92 compared to 44 1.04 in the xylitol gum group. The most comprehensive study with xylitol gum, conducted 45 in 1995, compared the effect on caries incidence for xylitol, sorbitol, and sucrose 46 consumption.⁵ The group that received 100% xylitol gum 5 times/day had significantly 47 lower levels of sucrose and free sialic acid in whole saliva than at baseline, and significantly 48 lower plaque index scores.⁵ The xylitol group also exhibited the lowest levels of salivary 49 lactobacilli at endpoint, and this group did not experience the age-related increase in MS as 50 did the other groups.⁵

51 Xylitol studies show varying results in the reduction of the incidence of caries or MS 52 levels.⁵⁻¹¹¹³ Studies suggest xylitol intake that consistently produces positive results ranged 53 from 4-10 grams per day divided into 3 to 7 consumption periods.⁵⁻¹⁴¹³ Higher amounts did 54 not result in greater reduction in incidence of caries and may lead to diminishing anticariogenic results.⁵⁻¹¹¹³ Similarly, consumption frequency of less than 3 times per day at 55 56 optimal xylitol amount showed no effect.¹²⁻¹⁴14-16 Abdominal distress and osmotic diarrhea 57 have been reported following the ingestion of xylitol.¹⁵⁻¹⁶<u>17-18</u> Diarrhea has been reported in 58 patients who have consumed 3-60 grams of xylitol per day.¹⁷⁻²¹ 19-23

59 Xylitol reduces plaque formation and bacterial adherence (ie, is antimicrobial), inhibits 60 enamel demineralization (ie, reduces acid production), and has a direct inhibitory effect on 61 MS. Prolonged use of xylitol appears to select for a "xylitol-resistant" mutant of the MS 62 cells.²² ²⁴ These mutants appear to shed more easily into saliva than the parent strains,²³ ²⁵ 63 resulting in a reduction of MS in plaque²⁴ ²⁶ and possibly hampering their 64 transmission/colonization from mother to child. Long-lasting effects have been 65 demonstrated up to 5 years after 2 years of using xylitol chewing gum.²⁵27-28</sup> Use of xylitol

66 gum by mothers (2-3 times per day) starting 3 months after delivery and until the child was

67	2 years old, reduced the MS levels in children up to 6 years of age and was significantly
68	better than applying fluoride varnish or chlorhexidine varnish at 6, 12, and 18 months after
69	delivery. At 5 years of age, the xylitol group had 70% reduction in caries (dmf) as compared
70	with the varnish and chlorhexidine groups. Fluoride varnish alone had little effect on total
71	salivary levels of MS. ²⁵ ℤ Some studies suggest the chewing process may enhance the caries
72	inhibitory effect of xylitol chewing gum. ²⁶⁻²⁹ 28-31
73	Xylitol currently is available in many forms (eg, gums, mints, chewable tablets, lozenges,
74	toothpastes, mouthwashes, cough mixtures, nutraceutical products). ^{30 <u>32-34</u>Xylitol chewing}
75	gum has been shown to be effective as a preventive agent <u>.</u> ; however<u>The</u> usefulness
76	effectiveness of other xylitol products that have not been is being studied. is uncertain and
77	cannot be recommended at this time <u>.</u> because the delivery system and dose/frequency of
78	use both impact the effectiveness of products.
79	
80	Policy statement
81	The AAPD:
82	1. supports preventive strategies aimed specifically at long term caries pathogen
83	suppression and caries (dmf) reduction using commercially available non-cariogenic
84	sugar substitutes such as xylitol. the use of xylitol as part of a preventive strategy
85	specifically aimed at long term caries pathogen suppression and caries reduction in
86	higher risk populations.
87	2. recommends <u>that as</u> further research- on xylitol to improve the <u>and</u> evidence-based <u>ed</u>
88	knowledge <u>is available</u> , <u>protocols be established to further clarify</u> the impact of delivery
89	vehicle, and the <u>frequency of exposure and</u> -identification of optimal-prevention
90	strategies and <u>dosage</u> to reduce caries (dmf) and improve the oral health of children.
91	3. recommends that when xylitol supplementation is prescribed as part of a caries
92	prevention strategy, it be administered topically 2 or 3 times daily at a total daily dose
93	<u>of 4-8 g. 28,30,32</u>
94	<u>4.</u> encourages xylitol-containing products be to be labeled clearly labeled with regard to
95	their xylitol content to enable dentists and consumers to evaluate fully their therapeutic
96	value insure therapeutic levels of exposure 2931
97	

98

99 References

- Mäkinen KK. Biochemical principles of the use of xylitol in medicine and nutrition with
 special consideration of dental aspects. Experientia Suppl 1978;30:1-16.
- Muhlemann HR, Regolati B, Marthaler TM. The effect on rat fissure caries of xylitol and sorbitol. Helv Odontol Acta 1970;14(1):48-50.
- Scheinin A, Mäkinen KK, Tammisalo E, Rekola M. Turku sugar studies. XVIII. Incidence
 of dental caries in relation to 1-year consumption of xylitol chewing gum. Acta Odontol
 Scand 1975a;33(5):269-78.
- 4. Scheinin A, Mäkinen KK, Ylitalo K. Turku sugar studies. V. Final report on the effect of sucrose, fructose and xylitol diets on caries incidence in man. Acta Odontol Scand 1976; 34(4):179-216.
- 5. Mäkinen KK, Benett CA, Hujoel PP, et al. Xylitol chewing gums and caries rates: A 40month cohort study. J Dent Res 1995;74(12):1904-13.
- 6. Mäkinen KK, Hujoel PP, Bennett CA, et al. A descriptive report of the effects of a 16month xylitol chewing-gum programme subsequent to a 40-month sucrose gum
 programme. Caries Res 1998;32(2):107-12.
- 7. Milgrom P, Ly KA, Roberts M, Rothen M, Mueller G, Yamaguchi DK. Mutans
 Streptococci dose response to Xylitol chewing gum. J Dent Res 2006;85(2):177-81.
- 8. Hujoel PP, Mäkinen KK, Bennett CA, et al. The optimum time to initiate habitual xylitol gum-chewing for obtaining long-term caries prevention. J Dent Res 1999;78(3):797-803.
- 9. Mäkinen KK. The rocky road of xylitol to its clinical application. J Dent Res 2000;79(6):1352-5.
- 121 10. Mäkinen KK, Chiego DJ Jr, Allen P, et al. Physical, chemical, and histologic changes in dentin caries lesions of primary teeth induced by regular use of polyol chewing gums.
 123 Acta Odontol Scand 1998;56(3):148-56.
- 124 11. Mäkinen KK, Mäkinen PL, Pape HR, et al. Conclusion and review of the Michigan
 125 Xylitol Programme (1986-1995) for the prevention of dental caries. Int Dent J
 126 1996;46(1):22-34.
- 127 12. Deshpande, A. and A.R. Jadad, The impact of polyol-containing chewing gums on
 128 dental caries: a systematic review of original randomized controlled trials and
 129 observational studies. J Am Dent Assoc, 2008. 139(12): p. 1602-14.
- 130 13. Stecksen-Blicks, C., P.L. Holgerson, and S. Twetman, Effect of xylitol and xylitol-fluoride
 131 lozenges on approximal caries development in high-caries-risk children. Int J Paediatr
 132 Dent, 2008. 18(3): p. 170-7.
- 133 12. 14. Isokangas P. Xylitol chewing gum in caries prevention. A longitudinal study on
 134 Finnish school children. Proc Finn Dent Soc 1987;83(suppl 1):1-117.
- 135 13. 15. Rekola M. Correlation between caries incidence and frequency of chewing gum sweetened with sucrose or xylitol. Proc Finn Dent Soc 1989;85(1):21-4.
- 137 14. <u>16.</u> Thaweboon S, Thaweboon B, Soo-Ampon S. The effect of xylitol chewing gum on 138 mutans streptococci in saliva and dental plaque. Southeast Asian J Trop Med Public 139 Health 2004;35(4):1024-7.
- 140 15. <u>17. Scheienin</u> AA, Fijerskov O. Xylitol in caries prevention: What is evidence for clinical efficacy? Oral Dis 1998;4(4):268-78.
- 142 16. 18. Mäkinen KK. Dietary prevention of dental caries by xylitol Clinical effectiveness and safety. J Appl Nutr 1992;44:16-28.

- 144 17. <u>19.</u> Akerblom HK, Koivukangas T, Puuka R, Mononen M. The tolerance of increasing amounts of dietary xylitol in children. Int J Vitam Nutr Res Suppl 1982;22:53-66.
- 146 18. 20. Giertsen E, Emberland H, Scheie AA. Effects of mouth rinses with xylitol and fluoride on dental plaque and saliva. Caries Res 1999;33(1):23-31.
- 148 19. 21. Salminen EK, Salminen SJ, Porkka L, Kwasowski P, Marks V, Koivistoinen PE.
 149 Xylitol vs glucose: Effect on the rate of gastric emptying and motilin, insulin, and gastric inhibitory polypeptide release. Am L Clin Nutr 1989;49(6):1228-32.
- 151 20. 22. Uhari M, Kontiokari T, Koskela M, Niemela M. Xylitol chewing gum in prevention of acute otitis media: Double blind randomized trial. Brit Med J 1996;313(7066):1180-4.
- 153 <u>21.</u> <u>23.</u> Waler SM, Rolla G. [Xylitol, mechanisms of action and uses]. Nor Tannelaegeforen
 154 Tid 1990;100(4):140-3.
- 155 <u>22.</u> <u>24.</u> Trahan L, Mouton C. Selection for *Streptococcus mutans* with an altered xylitol transport capacity in chronic xylitol consumers. J Dent Res 1987;66(5):982-8.
- 157 23. 25. Trahan L, Söderling E, Dréan MF, Chevrier MC, Isokangas P. Effect of xylitol
 158 consumption on the plaque-saliva distribution of mutans streptococci and the
 159 occurrence and longterm survival of xylitol-resistant strains. J Dent Res
 160 1992;71(11):1785-91.
- 161 24. <u>26.</u> Söderling E, Trahan L, Tammiala-Salonen T, Hakkinen L. Effects of xylitol, xylitol162 sorbitol, and placebo chewing gums on the plaque of habitual xylitol consumers. Eur J
 163 Oral Sci 1997;105(2):170-7.
- 164 25. 27. Söderling E, Isokangas P, Pienihakkinen K, Tenovuo J, Alanen P. Influence of 165 maternal xylitol consumption on mother-child transmission of mutans streptococci: 6 166 year follow-up. Caries Res 2001;35(3):173-7.
- 167 <u>28. Makinen, K.K., et al., Thirty-nine-month xylitol chewing-gum programme in initially 8-</u>
 168 <u>year-old school children: a feasibility study focusing on mutans streptococci and</u>
 169 <u>lactobacilli. Int Dent J, 2008. 58(1): p. 41-50.</u>
- 170 26. 29. Machiulskiene V, Nyvad B, Baelum V. Caries preventive effect of sugar-substituted chewing gum. Community Dent Oral Epidemiol 2001;29(4):278-88.
- 172 27. <u>30.</u> Scheie<u>nin</u> AA, Fejerskov O, Danielsen B. The effects of xylitol-containing chewing gums on dental plaque and acidogenic potential. J Dent Res 1998;77(7):1547-52.
- 174 28. 31. Van Loveren C. Sugar alcohols: What is the evidence for caries-preventive and caries-therapeutic effects? Caries Res 2004;38(3):286-93.
- 176 29. 32. Ly KA, Milgrom P, Rothen M. Xylitol, sweeteners, and dental caries. Pediatr Dent 2006;28(2):154-63. Discussion 92-8.
- 30. <u>33.</u> Lynch H, Milgrom P. Xylitol and dental caries: An overview for clinicians. J Calif Dent Assoc 2003;31:205-9.
- 180 <u>34. Lund, A.E., Xylitol gummy bears reduce plaque in young children. J Am Dent Assoc,</u>
 181 <u>2008. 139(10): p. 1312.</u>
- 182
| 1
2 | Policy on Prevention of Sports-related Orofacial Injuries |
|----------|--|
| 2 | Originating Committee |
| 4 | Clinical Affairs Committee |
| 5 | |
| 6 | Review Council |
| /
8 | Council on Clinical Affairs |
| 9 | Adopted |
| 10 | 1991 |
| 11 | |
| 12 | Revised |
| 13
14 | 1995, 1999, 2002, 2006, <u>2010</u> |
| 15 | Purpose |
| 16 | The American Academy of Pediatric Dentistry (AAPD) is concerned about recognizes the |
| 17 | prevalence of sports-related orofacial injuries in our nation's youth and the need for |
| 18 | prevention. This policy is intended to educate dental professionals, health care providers, |
| 19 | educational and athletic personnel on the prevention of sports-related orofacial injuries. |
| 20 | |
| 21 | Methods |
| 22 | A MEDLINE literature search was conducted using the terms "sports injuries", "injury |
| 23 | prevention", "dental injuries", "orofacial injuries", and "mouthguard". This guideline is an |
| 24 | update of the previous document, revised in 2006. The update included an electronic search |
| 25 | using the search terms, "sports injuries", "injury prevention", "dental injuries", "orofacial |
| 26 | injuries", and "mouthguards". FIELD: All Fields; LIMITS: within the last ten years; |
| 27 | humans; English; clinical trials; literature reviews. The reviewers agreed upon the inclusion |
| 28 | of 48 articles that met the defined criteria. When data did not appear sufficient or were |
| 29 | inconclusive, recommendations were based upon expert and/or consensus opinion by |
| 30 | experienced researchers and clinicians. |
| 31 | |
| 32 | Background |
| 33 | Increased competitivenessThe tremendous popularity of organized youth sports and the |
| 34 | high level of competiveness has resulted in a significant number of dental and facial injuries |

35 which represent a high percentage of the total injuries experienced in youth sports.^{1,2}Over

36 the past decade, approximately 46 million youths in the United States were involved in 37 "some form of sports".³ It further is It is estimated that 30 million children in the US 38 participate in organized sport programs.⁴ All sporting activities have an associated risk of 39 orofacial injuries due to falls, collisions, and contact with hard surfaces, and contact from 40 sports-related equipment. Sports accidents reportedly account for 10-39% of all dental 41 injuries in children.⁵ Children are most susceptible to sports- related oral injury between the 42 ages of 7 and 11 years of age. 5.6.38.39 The administrators of youth, high school, and college 43 football, lacrosse, and ice hockey have demonstrated that dental and facial injuries can be 44 reduced significantly by introducing mandatory protective equipment. Popular sports such 45 as baseball, basketball, soccer, field hockey, softball, wrestling, volleyball, and gymnastics 46 lag far behind in injury protection for girls and boys. Youths participating in leisure 47 activities such as skateboarding, inline or roller skating, and bicycling also benefit from 48 appropriate protective equipment.^{6-8,40}

49 Studies of dental and orofacial athletic injuries are reported throughout the medical and 50 dental literature.^{9,10} A review of literature published over the past 20 years showed that the 51 injury rate varied greatly depending on the size of the sample, the sample's geographic 52 location, the ages of the participants, and the specific sports involved in the study.^{9,10} 53 Although the statistics vary, many studies reported that dental and orofacial injuries 54 occurred regularly and concluded that participation in sports carries a considerable risk of 55 injury .^{5,9-12}

56 Consequences of orofacial trauma for children and their families are substantial because 57 of potential for pain, psychological effects, and economic implications. Children with 58 untreated trauma to permanent teeth exhibit greater impacts on their daily living than those 59 without any traumatic injury.^{13,48} The yearly costs of <u>all</u> injuries, including orofacial injuries, 60 sustained by young athletes have been estimated to be as high as 1.8 billion dollars.⁴ 61 Although the incidence of dental and orofacial trauma is small in comparison to all 62 traumatic injuries from sports-related accidents, the costs incurred are both disproportionate 63 and high.⁵ The National Youth Sports Safety Foundation in 2005 estimated the cost to treat 64 an avulsed permanent tooth and provide follow-up care is between \$5000 and \$20,000 over 65 a lifetime.¹⁴ Traumatic dental injuries have additional indirect costs that include children's

66 hours lost from school and parents' hours lost from work, consequences that

67 disproportionately burdens lower income, minority and non-insured children. 42,43,44,45 68 The majority of sport-related dental and orofacial injuries affect the upper lip, maxilla, 69 and maxillary incisors, with 50-80% of dental injuries involving the maxillary 70 incisors.^{5,9,10,41} Use of a mouthguard may can protect the upper incisors. However, studies 71 have shown that even with a mouthguard in place, up to 25% of dentoalveolar injuries still 72 can occur.15 73 Identifying patients who participate in sports and recreational activities allows the 74 healthcare provider to recommend and implement preventive protocols for individuals at 75 risk for orofacial injuries. In 2000, a predictive index was developed to identify the risk 76 factors involved in various sports. This index is based upon a defined set of risk factors that

77 influence predict the chance of injury including demographic information (age, gender,

78 dental occlusion), protective equipment (type/usage), velocity and intensity of the sport,

79 level of activity and exposure time, level of coaching and type of sports organization,

80 whether the player is a focus of attention in a contact or non-contact sport, history of

81 previous sports-related injury, and the situation (ie, practice vs game).^{12,16} Behavioral risk

82 factors (eg, hyperactivity) also have been associated significantly with injuries affecting the

83 face and/or teeth.17

84 A health professional may be able to modify certain risk factors such as a patient's 85 dental anatomy and occlusion. The frequency of dental trauma is significantly higher for 86 children with increased overjet and inadequate lip coverage.^{18,19} A dental professional may 87 be able to modify these risk factors. Initiating preventive orthodontic treatment in early- to 88 middle-mixed dentition of patients with an overjet >3mm has been proposed has the 89 potential to prevent reduce the severity of traumatic injuries to permanent incisors.¹⁸ 90 Although some sports-related traumatic injuries are unavoidable, most can be 91 prevented.^{12,14,20,21} Helmets, facemasks, and mouthguards have been shown to reduce both

92 the frequency and severity of dental and orofacial trauma.¹² However, few sports have

93 regulations that require their use. The National Federation of State High School

94 Associations mandate mouthguards for only 4 sports: football, ice hockey, lacrosse, and

95 field hockey.²¹ Several states have attempted to increase the number of sports which

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96	mandate mouthguard use with various degrees of success and acceptance. Four New
97	England states have been successful in increasing the number of sports requiring
98	mouthguard use to include sports such as soccer, wrestling, and basketball. ^{21,22}
99	Initially used by professional boxers, the mouthguard has been used as a protective
100	device since the early 1900's. ^{10,23} The mouthguard, also referred to as a gumshield or mouth
101	protector, is defined as a "resilient device or appliance placed inside the mouth to reduce
102	oral injuries, particulary <u>particularly</u> to teeth and surrounding structures."⁵ The
103	mouthguard was constructed to "protect the lips and intraoral tissues from bruising and
104	laceration, to protect the teeth from crown fractures, root fractures, luxations, and avulsions,
105	to protect the jaw from fracture and dislocations, and to provide support for edentulous
106	space." ²⁴ The mouthguard works by "absorbing the energy imparted at the site of impact
107	and by dissipating the remaining energy."25
108	The American Society for Testing and Materials (ASTM) classifies mouthguards by 3
109	categories ²⁶ :
110	1.Type I – Custom-fabricated mouthguards are produced on a dental model of the patient's
111	mouth by either the vacuum-forming or heat-pressure lamination technique. 5.12 The ASTM
112	recommends that for maximum protection, cushioning, and retention, the mouthguard
113	should cover all teeth in one arch, customarily the maxillary arch, less the third molar. $\frac{26}{A}$
114	mandibular mouthguard is recommended in patients with a Class III malocclusion. The
115	custom-fabricated type is superior in retention, protection, and comfort. 5,12,25,27,28 When this
116	type is not available, the mouth-formed mouthguard is preferable to the stock or preformed
117	<u>mouthguard.^{23,29,30}</u>
118	
119	1. Type I – Stock mouthguards are purchased over-the-counter. They are designed for use
120	without any modification and must be held in place by clenching the teeth together <u>to</u>
121	provide a protective benefit. ^{5,12} Clenching a stock-mouthguard in place can interfere with
122	breathing and speaking, and for this reason, stock-mouthguards are considered by many
123	to be less protective. <u>5,24,39,46,47</u> Despite these shortcomings of the stock-mouthguard, they
124	could be the only option possible for patients with particular clinical presentations such as

- 125 <u>use of orthodontic brackets and appliances and \ or periods of rapidly changing occlusion</u>
 126 <u>during mixed dentition.</u>
- 127 2.Type II Mouth-formed, also known as <u>"boil-and-bite"</u>, mouthguards are made from a
- 128 thermoplastic material adapted to the mouth by finger, tongue, and biting pressure after
- 129 immersing the appliance in hot water.⁵ Available commercially at department and sporting
- 130 good stores, these are the most commonly used among athletes but vary greatly in
- 131 protection, retention, comfort, and cost.^{12,39}
- 132 <u>3.Type III Stock mouthguards are purchased over-the-counter. They are designed for use</u>
- 133 without modification and must be held in place by clenching the teeth together to provide a
- 134 protective benefit.^{5,12} Clenching a stock-mouthguard in place can interfere with breathing
- 135 and speaking, and for this reason, stock-mouthguards are considered by many to be less
- 136 protective. <u>5.24,39,46</u> Despite these shortcomings of the stock-mouthguard, they could be the
- 137 <u>only option possible for patients with particular clinical presentations such as use of</u>
- 138 <u>orthodontic brackets and appliances and \ or periods of rapidly changing occlusion during</u>
- 139 <u>mixed dentition.</u>
- 140 <u>3. Type III Custom fabricated mouthguards are produced on a dental model of the</u>
- 141 patient's mouth by either the vacuum-forming or heat-pressure lamination technique.^{5,12}
- 142 The ASTM recommends the custom mouthguard be fabricated for the maxillary arch for
- 143 Class I and II occlusions, and on the mandibular arch for Class III malocclusions.²⁶
- 144 This<u>The custom-fabricated</u> type is superior in retention, protection, and comfort.^{5,12,25,27,28}
- 145 When this type is not available, the mouth-formed mouth-guard <u>mouthguard is</u>
- 146 preferable to the stock or preformed mouth-guard.^{23,29,30}
- 147 The Academy for Sports Dentistry "recommends the use of a properly fitted
- 148 mouthguard; encourages the use of a custom fabricated mouthguard made over a dental
- 149 cast and delivered under the supervision of a dentist; and supports a mandate for use of a
- 150 properly fitted mouthguard in all collision and contact sports."³¹<u>During fabrication of the</u>
- 151 <u>mouthguard, it is recommended to establish proper anterior occlusion of the maxillary and</u>
- 152 mandibular arches, since this will prevent or reduce injury by better absorbing and
- 153 <u>distributing the force of impact.⁴¹ The practitioner should also consider the patient's vertical</u>
- 154 <u>dimension of occlusion, personal comfort and breathing ability.³⁹ By providing cushioning</u>

155 between the maxilla and mandible, mouthguards also may reduce the incidence or severity 156 of condylar displacement injuries, as well as the potential for concussions.39,47 48 157 Due to the continual shifting of teeth in orthodontic therapy, the exfoliation of primary 158 teeth, and the eruption of permanent teeth, a custom-fabricated mouthguard may not fit the 159 voung athlete soon after the impression is obtained.³² Several block-out methods used in 160 both the dental operatory and laboratory may incorporate space to accommodate for future 161 tooth movement and dental development.³² By anticipating required space changes, a 162 custom fabricated mouthguard may be made to endure several sports seasons.³² 163 Parents play an important role in the acquisition of a mouthguard for young athletes. In 164 a 2004 national fee survey, custom mouthguards ranged from \$60 to \$285.³³ In a study to 165 determine the acceptance of the 3 types of mouthguards by 7- and 8-year old children 166 playing soccer, only 24% of parents surveyed were willing to pay \$25 for a custom 167 mouthguard.³⁴ Therefore, cost may be a barrier.³³ 168 Attitudes of officials, coaches, parents, and players about wearing mouthguards 169 influence their usage.³⁵ Although coaches are perceived as the individuals with the greatest 170 impact on whether or not players wear mouthguards, parents view themselves as equally 171 responsible for maintaining mouthguard use.^{35,36} However, parental views about surveys of 172 parents regarding the indications for mouthguard usage reveal a lack of complete 173 understanding of the benefits of mouthguard use.35 Players' perceptions of mouthguard 174 usage use and comfort largely determine their compliance and enthusiasm.^{25,34} Therefore, 175 the dental profession needs to influence and educate all stakeholders about the risk of 176 sports-related orofacial injuries and <u>available</u> preventive strategies.^{23,33,37} Routine dental 177 visits can be an opportunity to initiate patient/parent education and make appropriate 178 recommendations for use of a properly-fitted athletic mouthguard.¹² 179 180 **Policy statement** 181 The AAPD recommends: 182 1. dentists play an active role in educating the public in the use of mouthguards and 183 protective equipment for the prevention of orofacial injuries during sporting and

184 <u>recreational</u> activities to prevent orofacial injuries;

- 185 2. continuation of preventive practices instituted in youth, high school and college186 football, lacrosse, field hockey and ice hockey;
- 187 3. for youth participating in organized baseball and softball activities, an ASTM-certified
- 188 face protector be required (according to the playing rules of the sport);
- 4. mandating the use of properly fitted mouthguards in other organized sporting activities
 with that carry risk of orofacial injury;
- 191 5. prior to initiating practices for a sporting season, coaches/administrators of organized
- 192 sports consult a dentist with expertise in orofacial injuries for recommendations for
- 193 immediate management of sports-related injuries (eg, avulsed teeth);
- 194 6. continuation of research in development of a comfortable, efficacious, and cost-effective
- sports mouthguard to facilitate more widespread use of this proven protective device;
- 196 <u>7. dentists of all specialties, including pediatric and general dentists, provide</u>
- 197 <u>education to parents and patients regarding prevention of orofacial injuries as part of the</u>
- 198 <u>anticipatory guidance discussed in dental visits;</u>
- 199 <u>8. dentists should prescribe, fabricate or provide an appropriate referral for mouthguard</u>
- 200 protection for patients at increase risk for orofacial trauma;
- 201 <u>9. that third party payors realize the benefits of mouthguards for the prevention and</u>
- 202 protection from orofacial sports-related injuries and encourages them to improve access
- 203 <u>these services;</u>
- 204 7.<u>10.</u> the Academy for Sports Dentistry and the International Association of Dental
- Traumatology be consulted as valuable resources for the professions and public.
- 206

207 References

- 208 1. Castaldi CR. Sports-related oral and facial injuries in the young athlete: A new challenge
 209 for the pediatric dentist. Pediatr Dent 1986;8(4):311-6.
- 2102. Castaldi CR. Athletic mouthguards: History and present status. Sports Med Digest 1988;10:1-2.
- 3. Barron M, Powell J. Fundamentals of injury prevention in youth sports. J Pediatr Dent Care 2005;11(2):10-2.
- 4. Adirim T, Cheng T. Overview of injuries in the young athlete. Sports Med 2003;33(1):7581.
- 5. Newsome P, Tran D, Cooke M. The role of the mouth-guard mouthguard in the prevention of sports-related dental injuries: A review. Int J Paediatr Dent 2001;11(6):396-404.

- 6. Tesini DA, Soporowski NJ. Epidemiology of orofacial sports-related injuries. Dent Clin
 North Am 2000;44 (1):1-18.
- 7. Ranalli DN. Prevention of sports-related dental traumatic injuries. Dent Clin North Am 2000;44(1):35-51.
- 8. Finnoff JT, Laskowski ER, Altman KC, Diehl NW. Barriers to bicycle helmet use.
 Pediatrics 2001;108(1):4-10.
- 9. Kumamoto D, Maeda Y. Global trends and epidemiology of sports injuries. J Pediatr
 Dent Care 2005;11(2):15-25.
- 227 10. Kumamoto D, Maeda Y. A literature review of sports-related orofacial trauma. Gen Dent 2004;52(3);270-80.
- 229 11. Gassner R, Tuli T, Hachl O, Rudisch A, Ulmer H. Craniomaxillofacial trauma: A 10 year
 230 review of 9,543 cases with 21,067 injuries. J Craniomaxillofac Surg 2003;31:51-61.
- 231 12. Ranalli DN. Sports dentistry in general practice. Gen Dent 2000;48(2):158-64.
- 232 13. Cortes M, Marcenes W, Sheiham A. Impact of traumatic injuries to the permanent teeth
 233 on the oral health-related quality of life in 12-14-year old children. Comm Dent and Oral
 234 Epidemiol 2002;30(3):193-8.
- 14. National Youth Sports Safety Foundation, Inc. 20052009: "www.nyssf.org". Accessed:
 336 July 16, 2005. August 15, 2009.
- 237 15. Onyeaso C, Adegbesan O. Knowledge and attitudes of coaches of secondary school
 238 athletes in Ibadan, Nigeria regarding orofacial injuries and mouthguard use by the
 239 athletes. Dent Traumatol 2003;19(5):204-8.
- 240 16. Fos P, Pinkham JR, Ranalli DN. Prediction of sports-related dental traumatic injuries.
 241 Dent Clin North Am 2000;44(1):19-33.
- 242 17. Lalloo R. Risk factors for major injuries to the face and teeth. Dent Traumatol
 243 2003;19(1):12-14.
- 244 18. Bauss O, Rohling J, Schwestka-Polly R. Prevalence of traumatic injuries to the
 245 permanent incisors in candidates for orthodontic treatment. Dent Traumatol
 246 2004;20(2):61-6.
- 247 19. Forsberg C, Tedestam G. Etiological and predisposing factors related to traumatic
 248 injuries to permanent teeth. Swed Dent J 1993;17(5):183-90.
- 249 20. 1st World Congress of Sports Injury Prevention Abstracts. Br J Sports Med 2005;39:373250 408.
- 251 21. Mills S. Can we mandate prevention? J Pediatr Dent Care 2005;11(2):7-8.
- 252 22. Kumamoto D. Establishing a mouthguard program in your community. Gen Dent 2000;48:160-4.
- 23. Patrick DG, van Noort R, Found MS. Scale of protection and the various types of sports
 mouthguard. Br J Sports Med 2005;39(5):278-81.
- 24. Biasca N, Wirth S, Tegner Y. The avoidability of head and neck injuries in ice hockey: A historical review. Br J Sports Med 2002;36(6):410-27.
- 25. McClelland C, Kinirons M, Geary L. A preliminary study of patient comfort associated
 with customised mouthguards. Br J Sports Med 1999;33(3):186-9.
- 26. American Society for Testing and Materials. Standard practice for care and use of
 261 mouthguards <u>athletic mouth protectors</u>. ASTM F697-86 F697-00. Philadelphia, Pa:
 262 American Society for Testing and Materials; 1986 Reapproved 2006.

- 263 27. Warnet L, Greasley A. Transient forces generated by projectiles on variable quality
 264 mouthguards monitored by instrumented impact testing. Br J Sports Med
 265 2001;35(4):257-62.
- 28. Greasley A, Imlach G, Karet B. Application of a standard test to the in vitro performance of mouthguards. Br J Sports Med 1998;32(1):17-9.
- 268 29. Bureau of Dental Health Education and Bureau of Economic Research and Statistics.
 269 Evaluation of mouth protectors used by high school football players. J Am Dent Assoc
 270 1964;68:430-42.
- 271 30. DeYoung AK, Robinson E, Godwin WC. Comparing comfort and wearability: Custom272 made vs self-adapted mouthguards. J Am Dent Assoc 1994;125(8):1112-8.
- 273 31. Academy for Sports Dentistry 20052009: "www.sportsdentistry-asd.org". Accessed:
 274 March 7, 2006October 24, 2009.
- 275 32. Croll T, Castaldi CR. Custom sports mouthguard modified for orthodontic patients and children in the transitional dentition. Pediatr Dent 2004;26(5):417-20.
- 33. Walker J. Parents plus: Getting mouthguards into kids' mouths. J Pediatr Dent Care
 2005;11(2):39-40.
- 34. Walker J, Jakobsen J, Brown S. Attitudes concerning mouthguard use in 7- to 8-year-old children. J Dent Child 2002;69(2):207-11.
- 35. Gardiner D, Ranalli DN. Attitudinal factors influencing mouthguard utilization. Dent
 Clin North Am 2000;44(1): 53-65.
- 283 36. Diab N, Mourino A. Parental attitudes toward mouthguards. Pediatr Dent 1997;19(8):455-60.
- 285 37. Woodmansey K. Athletic mouth guards prevent orofacial injuries: A review. Gen Dent 1999;47(1):64-9..
- 287 <u>38. Rodd HD, Chesham DJ. Sports-related oral injury and mouthguard use among Sheffield</u>
 288 <u>school children. Community Dent Health 1997;14(1):25-30.</u>
- 39. ADA Council on Access, Prevention and Interprofessional Relations and ADA Council
 on Scientific Affairs. Using mouthguards to reduce the incidence and severity of sports related oral injuries. J Am Dent Assoc 2006;137(12):1712-20.
- 40. Fasciglione D, Persic R, Pohl Y, Fillippi A. Dental injuries in inline skating level of information and prevention. Dent Traumatol 2007; 23(3);143-8.
- <u>41. Takeda T, Ishigami K, Nakajima K, Naitoh K, Kurokawa K, Handa J, Shomura M,</u>
 <u>Regner CW.Are all mouthguards the same and safe to use? Part 2. The influence of</u>
 <u>anterior occlusion against a direct impact on maxillary incisors.Dent Traumatol 2008;</u>
 <u>24(3); 360-5.</u>
- 42. Sane J, Ylipaavalniemi P, Turtola L, Niemi T, Laaka V. Traumatic injuries among
 university students in Finland. J Am Coll Health 1997; 46(1);21-4
- 300 <u>43. Ngyuyen PM, Kenny DJ, Barret EJ. Socio-economic burden of permanent incisor</u>
 301 <u>replantation on children and parents. Dent Traumatol 2004; 20(3);123-33.</u>
- <u>44. Gift HC, Reisine ST, Larach DC. The social impact of dental problems and visits. Am J</u>
 <u>Public Health 1992;82(12);1663-8.</u>
- 304 <u>45. McIntyre JD, Lee JY, Trope M, Vann WF. Elementary school staff knowledge about</u>
 305 <u>dental injuries. Dent Traumatol 2008; 24(3);289-98.</u>
- <u>46. Ranalli DN. Prevention of craniofacial injuries in football. Dent Clin North Am</u>
 <u>1991;35(4);627-45.</u>

308	<u>47. Biasca N, Wirth S, Tegner Y. The avoidability of head and neck injuries in ice hockey: a</u>
309	historical review. Br J Sports Med 2002; 36(6);410-27.
310	4847. Waliko T, Bir C, Godwin W, King A. Relationship between temporomandibular joint
311	dynamics and mouthguards: feasibility of a test method. Dent Traumatol 2004;20(5);255-
312	<u>60.</u>
313	48. Berger TD, Kenny DJ, Casas MJ, Barrett EJ, Lawrence HP. Effects of severe dentoalveolar
314	trauma on the quality-of-life of children and parents. Dent Traumatol 2009;25(5);462-9.

1	Policy on Tobacco Use					
2 3 4 5	Originating Council Council on Clinical Affairs					
5 6 7 8 9 10	Review Council Council on Clinical Affairs					
	Adopted 2000					
11 12 13 14 15	Revised 2003, 2006 <u>, 2010</u>					
16	Purpose					
17	The American Academy of Pediatric Dentistry (AAPD), in order to reduce the incidence of					
18	pain, chronic disease, disability, and death caused by nicotine addiction, recommends					
19	routine screening for tobacco use, treating tobacco dependence, preventing tobacco use					
20	among children and adolescents, and educating the public on the enormous health and					
21	societal costs of tobacco.					
22						
23	Methods					
24	This policy revision is based upon on a review of current dental, medical, and public health					
25	literature related to tobacco use. A MEDLINE An electronic search was conducted using the					
26	terms "tobacco", "teen tobacco use", "tobacco use in children", and "smoking", <u>"smokeless</u>					
27	tobacco", "smokeless tobacco and oral disease", "pregnancy and smoking", "pregnancy and					
28	tobacco", "secondhand smoke" and "caries and smoking". Web sites for the American					
29	Lung Association, American Cancer Society, Centers for Disease Control and Prevention,					
30	Environmental Protection Agency, Campaign for Tobacco Free Kids and US Department of					
31	Health and Human Services were reviewed. FIELD: All fields; LIMITS: within the last 10					
32	years; humans; English; clinical trials; birth through age 19. 316 articles matched these					
33	criteria. When data did not appear sufficient or were inconclusive, recommendations were					
34	based upon expert and /or consensus opinion by experienced researchers and clinicians.					
35						

36 Background

37 Tobacco use, principally cigarette smoking, remains the leading preventable cause of 38 disease and premature death in the US and imposes substantial health-related and economic 39 costs to society.¹⁻⁵ Approximately one third of all tobacco users in this country will die 40 prematurely because of their dependence on tobacco and their addiction to nicotine.¹ 41 Significant oral, dental, and systemic health consequences associated with all forms of 42 tobacco use (ie, cigarettes, cigars, smokeless [spit] tobacco, pipes) are well documented in 43 the literature. Such consequences include oral cancer, periodontal disease, cardiovascular disease, pulmonary diseases, and lung cancer.⁵⁻⁸ The 2004 US Surgeon General report on 44 45 health effects of smoking identified a number of diseases caused by, but not previously 46 causally associated with, smoking. The list included cancers of the stomach, uterus, cervix, 47 pancreas, and kidney; acute myeloid leukemia; pneumonia; abdominal aortic aneurysm; 48 cataract; and periodontitis.9 Smoking during pregnancy is associated with adverse 49 outcomes, including low birth-weight, intrauterine growth retardation, and infant 50 morbidity and mortality, as well as negative consequences for child health and 51 development.^{1,5,10-12} Recent studies have concluded exposure to environmental tobacco 52 smoke (ETS; eg, second-hand or sidestream smoke, passive smoking) also presents serious 53 health hazards including cancer and heart disease in healthy nonsmokers.^{2,4,5,13,14} Infants and 54 children exposed to ETS have higher rates of lower respiratory illness, middle car infections, 55 asthma, and caries in the primary dentition and are at increased risk for sudden infant death 56 syndrome (SIDS).2,4,5,12-16 57 58 adolescence. One third of all smokers had their first cigarette by age 14, and 28% of high 59 school students report using some type of tobacco.^{17,18} The earlier that children and

60 adolescents begin using tobacco, the more likely they will become highly addicted and

61 continue using as adults.² If current tobacco use patterns continue in the US, an estimated

62 6.4 million persons now under the age of 18 will die prematurely from a tobacco-related

63 illness.^{5,19} Each year in the US, tobacco kills more citizens than alcohol, cocaine, heroin,

64 homicide, suicide, car accidents, fire, and autoimmune deficiency syndrome (AIDS)

65 combined.²

66	Tobacco is a risk factor for 6 of the 8 leading causes of deaths in the world and kills up to
67	one-half of its users. ¹ In the US, the Surgeon General Report states that smoking is the
68	single greatest avoidable cause of death. ² This report concludes that even in nonsmokers,
69	secondhand smoke exposure causes disease and death.
70	The Center for Disease Control (CDC) has conducted a National Youth Tobacco Survey
71	(NYTS) for the years 1999, 2000, 2002, 2004 and 2006 as part of the Healthy People 2010
72	objectives on tobacco use. ³ While middle school students showed a decrease in the use of
73	cigarettes, cigars and bidis (unfiltered cigarettes from India) ⁴ , they did not show a change in
74	the use of smokeless tobacco, pipes or kreteks (unfiltered cigarettes from India) ⁴ between
75	2004 and 2006. ⁴⁵ Unfortunately during this same period, no significant change was seen in
76	the tobacco use of high school students. ⁴⁵ Tobacco use among high school students is 20.0%
77	or 3.5 million, while 19.8% of adults smoke ⁵⁶ Smokeless tobacco is seen in 13.4% of male
78	high school students and 2.3% of females. ⁵ <u>6</u> Each day approximately 3,600 youth between
79	<u>12 – 17 years of age try smoking with 1,100 a day becoming regular daily users.⁶⁷</u>
80	Risk factors for tobacco use include: 440,000 deaths per year from smoking and an
81	additional 50,000 deaths per year from secondhand smoke ⁵ . Other catastrophic risk factors
82	are: cardiovascular disease; reproductive effects; pulmonary disease; cancers of the cervix,
83	kidney, pancreas, stomach, lung, larynx, bladder and esophagus; leukemia; cataracts;
84	abdominal aortic aneurysm; bronchitis; and other lung diseases including pneumonia. ⁷⁸
85	<u>Smoking and tobacco use is linked to cancer of the oral cavity.² This includes smokeless</u>
86	tobacco that also carries the risk of oral cancer. ⁸
87	Secondhand exposure to tobacco smoke imposes significant risks as well.
88	Cardiovascular disease and lung cancer are increased by 25-30% in nonsmokers who inhale
89	secondhand smoke. ² Infants and children who are exposed to smoke are at risk for Sudden
90	Infant Death Syndrome (SDS), acute respiratory infections, middle ear infections, bronchitis,
91	pneumonia, asthma ¹⁰ , allergies ^{11,12} , and infections during infancy. ¹³ Caries in the primary
92	dentition is also related to secondhand smoke exposure. ^{14,15,16} Enamel hypoplasia in both
93	the primary and permanent dentition is also seen in children exposed to cigarette smoke. ¹⁷
94	A new term, "thirdhand" smoke, has been proposed to describe the particulate residual
95	toxins that are deposited in layers all over the home after a cigarette has been

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96	extinguished. ¹⁸ These volatile compounds are deposited and "off gas" into the air over
97	months.19.20 Since children inhabit these low-lying contaminated areas and because the dust
98	ingestion rate in infants is more than twice that of an adult, they are even more susceptible
99	to thirdhand smoke. Studies have shown that these children have associated cognitive
100	defects in addition to the other associated risks of secondhand smoke exposure. ²¹
101	Tobacco use can result in oral disease. Oral cancer ² , periodontitis, 22-25 compromised
102	wound healing, a reduction in the ability to smell and taste, smoker's palate and melanosis,
103	coated tongue, staining of teeth and restorations, implant failure and leukoplakia ^{26,27} are all
104	seen in tobacco users. Smokeless tobacco is a risk factor for periodontal conditions.28-30 and
105	<u>oral cancer³¹</u>
106	Initiation of tobacco use begins before age 19 for 90% of adult smokers. ³² In fact, most
107	studies show that people who do not use tobacco as a teen never use it.32 Aggressive
108	marketing of tobacco products by manufacturers ^{33,34} , smoking by parents ³⁵ , peer influence, a
109	functional belief in the benefits and normalcy of tobacco ³⁶ , availability and price of tobacco
110	products, low socioeconomic status, low academic achievement, lower self image and a lack
111	of behavioral skills to resist tobacco offers all contribute to the initiation of tobacco use
112	during childhood and adolescence ³⁷ . Teens who use tobacco are more likely to use alcohol
113	and other drugs and engage in high risk sexual behaviors.37
114	The monetary costs of this addiction and resultant morbidity and mortality is
115	staggering. Annually, cigarette smoking costs the U.S. \$193 billion, based on lost
116	productivity (more than \$97 billion) and health care expenditures (more than \$96 billion).38
117	<u>Health care cost from the exposure to second hand smoke is about \$10 billion annually.³⁸</u>
118	Contrast this with tobacco industry expenditures on advertising and political influence of
119	<u>\$13.11 billion in 2005.³⁹</u>
120	Current trends indicate that tobacco use will cause more than 8 million deaths a year by
121	2030.40 It is incumbent on the healthcare community to reduce the burden of tobacco related
122	morbidity and mortality by supporting preventive measures, educating the public about the
123	risks of tobacco and screening for tobacco use and nicotine dependence.
124	

125 Policy statement

126	The AAPD opposes the use of all forms of tobacco including cigarettes, pipes, cigars, <u>bidis</u> ⁴ ,
127	<u>kreteks</u> ⁴ , and smokeless tobacco and alternative nicotine delivery systems (ANDS), such as
128	tobacco lozenges, nicotine water, nicotine lollipops, or "heated tobacco" cigarette
129	substitutes [‡] . The AAPD supports national, state, and local legislation that eliminates tobacco
130	advertising and promotions that appeal to or influence children, adolescents, or special
131	groups. The AAPD supports prevention efforts through merchant education and
132	enforcement of state and local laws prohibiting tobacco sales to minors. As environmental
133	tobacco smoke (ETS) is a "known human carcinogen" and there is no evidence to date of a
134	"safe" exposure level to <u>ETS (</u> second-hand <u>or passive</u> smoke), ^{13 40} the AAPD also supports
135	the enactment and enforcement of state and local clean indoor air and/or smoke-free
136	policies or ordinances prohibiting smoking in public places.
137	
138	Furthermore, the AAPD encourages its members to:
139	1. promote and establish policies that ensure dental offices, clinics, and/or health care
140	facilities, including property grounds, are tobacco free;
141	2. support tobacco free school laws and policies as advocated by the American Dental
142	Association; ^{41,42}
143	3. serve as role models by not using tobacco and urging staff members who use tobacco to
144	stop;
145	4. routinely examine patients for oral signs <u>and changes associated with</u> of tobacco use;
146	5. determine and document tobacco use by patients and smoking status of their parents,
147	guardians, and caregivers;
148	6. educate patients, parents, and guardians on the serious health consequences of tobacco
149	use and exposure to ETS in the home;
150	7. provide both prevention and cessation services using evidence-based interventions
151	identified as "best practice" for treating tobacco use and nicotine addiction;
152	8. work to ensure all third-party payors include "best practice" tobacco cessation
153	counseling and pharmacotherapeutic treatments as benefits in health packages;
154	9. work with school boards to increase tobacco-free environments for all school facilities,
155	property, vehicles, and school events;

156	10. work on the national level and within their state and community to organize and
157	support anti-tobacco campaigns and to prevent the initiation of tobacco use among
158	children and adolescents, eliminate cigarette sales from vending machines, and increase
159	excise tax on tobacco products to reduce demand;
160	11. work with legislators, community leaders and healthcare organizations to ban tobacco
161	advertising, promotion and sponsorships;
162	10. <u>11.</u> organize and support efforts to pass national, state, and local legislation prohibiting
163	smoking in businesses such as day-care centers where children routinely visit and other
164	establishments where adolescents frequently are employed;
165	11. 12. establish and support education/training activities and prevention/cessation
166	services throughout the community;
167	12. 13. recognize the US Public Health Service Clinical Practice Guideline "Treating
168	Tobacco Use and Dependence" ³⁵ as a valuable resource.
169 170	References
171	1. US Dept of Health and Human Services. Treating Tobacco Use and Dependence: A
172	Clinical Practice Guideline. US Dept of Health and Human Services. US Government
173	Printing Office-Washington, DC. 20402-9328. ISBN 1-58763-007-9. June 2000.
174	-2. US Dept of Health and Human Services. Preventing Tobacco Use Among Young People:
175	Report of the Surgeon General. Atlanta, Ga: US Dept of Health and Human Services,
176	Public Health Service, CDC, National Center for Chronic Disease Prevention and Health
177	Promotion, Office on Smoking and Health; 1994.
178	-3. CDC. Annual smoking-attributable mortality, years of potential life lost, and economic
179	costs-United States 1995-1999. MMWR Morb Mortal Wkly Rep 2002;51(14): 300-3.
180	-4. CDC. Guidelines for school health programs to prevent tobacco use and addiction.
181	MMWR Recomm Rep1994;43(RR-2):1-18.
182	-5. CDC. Targeting tobacco use: The nation's leading cause of death-At a glance. 2005.
183	Available at: "http://www.cdc.gov/nccdphp/agg/agg/osh.htm". Accessed February
184	6, 2006.
185	-6. American Cancer Society, National Cancer Institute, National Institutes of Health. How
186	to help your patients stop using tobacco: A National Cancer Institute manual for the
187	oral health team. Bethesda, Md: National Institutes of Health; 1998. Publication No. 98-
188	3191.
189	-7. American Dental Association. Summary of policy and recommendations regarding
190	
101	tobacco 1964-present. ADA Resolution 1H-1992. In: ADA Transactions 1992. Chicago,
191	Ill: American Dental Association; 1993:598.

193 of Health, CDC; Nov 8, 1999.

194 -9. US Dept of Health and Human Services. The health consequences of smoking: A report 195 of the Surgeon General. US Dept of Health and Human Services, CDC, National Center 196 for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health. 197 2004. Washington, DC. 198 10. Matthews TJ. Smoking during pregnancy in the 1990s. National vital statistics report. 199 Hvattsville, Md: National Center for Health Statistics: 2001:49:7. CDC. DHHS 200 Publication No. (PHHS)2001-1120; PRS 01-0539 (8/2001). 201 11. US Dept of Health and Human Services, CDC, National Center for Chronic Disease 202 Prevention and Health Promotion Office on Smoking and Health. Report of the Surgeon 203 General-Women and smoking: Tobacco use and reproductive outcomes - Fact sheet. US 204 Public Health Service. Office of the Surgeon General. 2001. Available at: 205 "http://www.cdc.gov/tobacco/sgr/sgr_forwomen/factsheet_outcomes.htm". 206 Accessed Feb. 6, 2006. 207 12. World Health Organization. International consultation on environmental tobacco smoke 208 (ETS) and child health-Consultation report. Geneva, Switzerland: World Health 209 Organization; 1999. 210 13. US Dept of Health and Human Services. The report on carcinogens. 9th ed. 2000. 211 Available at: "http://www.nih.gov/news/pr/may2000/niehs-15.htm". Accessed Feb. 212 6, 2006. 213 14. US Environmental Protection Agency. Fact Sheet: Res-piratory health effects of passive 214 smoking. January 1993. Environmental Protection Agency document No. 43-F-93-003. 215 Available at: "http://www.epa.gov/iaq/ets/pubs/etsfs.html". Accessed February 6, 216 2006. 217 15. Aligne CA, Moss ME, Auinger P, Weitzman M. Association of pediatric dental caries 218 with passive smoking. JAMA 2003;289(10):1258-64. 219 16. US Dept of Health and Human Services. Healthy people 2010: Tobacco use and healthy 220 people 2010 objectives-Tobacco priority area. Washington, DC. Available at: 221 "http://www.healthypeople.gov/document/html/Volume2/27tobacco.htm". 222 Accessed Feb. 6, 2006. 223 17. American Cancer Society. Child and teen tobacco use. Available at: 224 "www.cancer.org/docroot/PED/content/PED_10_2X_Child_And_Teen_Tobacco_Use. 225 ASP?sitearea=PED". Accessed Feb. 6, 2006. 226 18. American Lung Association. Smoking and teen fact sheet. Nov 2004. Available at: 227 "www.lungusa.org/site/pp.asp?c=dvLUK9O0E&b=39871". Accessed Feb. 6, 2006. 228 19. CDC. Cigarette smoking -Attributable morbidity - United States, 2000. MMWR Morb 229 Mortal Wkly Rep 2003;52(35):842-4. Available at: 230 "http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5235a4.htm". Accessed Feb. 6, 231 2006. 232 233 1. World Health Organization. Tobacco key facts. Available at: 234 www.who.int/topics/tobacco/facts/en/index.html Accessed Aug 6, 2009. 235 2. U.S. Department of Health and Human Services. The health consequences of 236 smoking: a report of the Surgeon General. U.S. Department of Health and Human 237 Services, Centers for Disease Control and Prevention, National Center of Chronic 238 Disease Prevention and Health Promotion, Office on Smoking and Health, 2004.

239		Available at: http://www.surgeongeneral.gov/library/smokingconsequences/
240		Accessed Aug 6, 2009.
241	3.	U.S. Department of Health and Human Services. Healthy people 2010: Tobacco use
242		and healthy people 2010 objectives-Tobacco priority area. Washington, DC. Available
243		at: http://www.healthypeople.gov/document/HTML/Volume2/27Tobacco.htm
244	4.	http://www.cdc.gov/tobacco/data_statistics/fact_sheets/tobacco_industry/bidis_k
245		<u>reteks/</u> Accessed Nov 8, 2009
246	5.	CDC. 2006 National Youth Tobacco Survey and Key Prevalence Indicators. Available
247		at: http://www.cdc.gov/tobacco/data_statistics/surveys/nyts/index.htm
248		Accessed Aug 6, 2009.
249	6.	CDC. Youth and tobacco use: current estimates. Updated Feb. 2009. Available at:
250		http://www.cdc.gov/tobacco/data_statistics/fact_sheets/youth_data/tobacco_use/
251		index.htm Accessed Aug 6, 2009.
252	7.	Toll of Tobacco in the United States of America. Tobacco use in the USA. Campaign
253		for Tobacco-Free Kids, Dec. 8, 2008. Available at:
254		http://www.tobaccofreekids.org/research/factsheets/ Accessed Aug 6, 2009.
255	8.	<u>CDC. Smoking and Tobacco Use. Health Effects of Cigarette Smoking. Updated Jan.</u>
256		2008. Available at:
257		http://www.cdc.gov/tobacco/data_statistics/fact_sheets/health_effects/effects_cig
258	_	_smoking. Accessed Aug 6, 2009.
259	9.	U.S. Department of Health and Human Services. The Health Consequences of
260		Involuntary Exposure to Tobacco Smoke: A Report of the Surgeon Gerneral. U.S.
261		Department of Health and Human Services, Centers for Disease Control and
262		Prevention, National Center for Chronic Disease Prevention and Health Promotion,
263		Office on Smoking and Health, 2006. Available at:
264		http://www.surgeongeneral.gov/library/secondhandsmoke/report/ Accessed Aug
265	10	<u>12, 2009.</u>
266	10.	Dietert RR, Zelikoff JT. Early-life environment, developmental immunotoxicology,
267		and the risk of pediatric allergic disease including asthma. Birth Detects Res B Dev
268	44	<u>Reprod Toxicol 2008; 83(6):547-60.</u>
269	11.	Goodwin RD, Cowles RA. Household smoking and childhood asthma in the United
270	4.0	States: a state-level analysis. J Asthma 2008;45(7):607-10.
271	12.	Lannerö E, Wickman M, van Hage M, Bergström A, Pershagen G, Nordvall L.
272		Exposure to environmental tobacco smoke and sensitisation in children. Thorax
273	10	2008;63(2):172-6.
274	13.	Ladomenou F, Kafatos A, Galanakis E. Environmental tobacco smoke exposure as a
275	4.4	risk factor for infections in infancy. Acta Paediatr 2009; 98(7):1137-41.
276	14.	Leroy R, Hoppenbrouwers K, Jara A, Declerck D. Parental smoking behavior and
277		caries experience in preschool children. Community Dent Oral Epidemiol
278	4 -	<u>2008;36(3):249-57.</u>
219	15.	Hanioka I, Nakamura E, Ojima M, Tanaka K, Aoyama H. Dental caries in 3-year-old
∠ŏU 204	1/	children and smoking status of parents. Paediatr Perinat Epidemiol 2008;22(6):546-50.
201	16.	Aligne CA, Moss ME, Auinger P, Weitzman M. Association of pediatric dental caries
ΖԾΖ		with passive smoking. JAMA 2003;289(10):1258-64.

283	17.	Ford D, Seow WK, Kazoullis S, Holcombe T, Newman B. A controlled study of risk
284		factors for enamel hypoplasia in the permanent dentition. Pediatr Dent
285		2009;31(5):382-8.
286	18.	Winickoff JP, Friebely J, Tanski SE, Sherrod C, Matt GE, Hovell MF, McMillen RC.
287		Beliefs about the health effects of "thirdhand" smoke and home smoking bans.
288		Pediatrics 2009;123(1):e74-9.
289	19.	Matt GE, Quintana PJ, Hovell MF, et al. Households contaminated by environmental
290		tobacco smoke: sources of infant exposures. Tob Control 2004;13(1):29-37.
291	20.	Singer BC, Hodgson AT, Guevarra KS, Hawley EL, Nazaroff WW. Gas-phase
292		organics in environmental tobacco smoke. 1. Effects of smoking rate, ventilation, and
293		furnishing level on emission factors. Environ Sci Technol 2002;36(5):846-53.
294	21.	Yolton K, Dietrich K, Auinger P, Lanphear BP, Hornung R. Exposure to
295		environmental tobacco smoke and cognitive abilities among U.S. children and
296		adolescents. Environ Health Perspect 2005;113(1):98-103.
297	22.	Johnson GK, Hill M. Cigarette smoking and the periodontal patient. J Periodontol
298		2004;75(2):196-209.
299	23.	Bergström J, Eliasson S, Dock J. A 10-year prospective study of tobacco smoking and
300		periodontal health. J Periodontol 2000;71(8):1338-47.
301	24.	Albandar JM, Streckfus CF, Adesanya MR, Winn DM. Cigar, pipe, and cigarette
302		smoking as risk factors for periodontal disease and tooth loss. J Periodontol
303		2000;71(2):1874-81.
304	25.	Johnson GK, Slach NA. Impact of tobacco use on periodontal status. J Dent Educ
305		<u>2001;65(4):313-21.</u>
306	26.	Vellappally S, Fiala Z, Smejkalová J, Jacob V, Somanathan R. Smoking related
307		systemic and oral diseases. Acta Medica 2007;50(3):161-6.
308	27.	Reibel J. Tobacco and oral diseases. Update on the evidence, with recommendations.
309		Med Princ Pract 2003;12 Suppl 1:22-32.
310	28.	Montén U, Wennström JL, Ramberg P. Periodontal conditions in male adolescents
311		using smokeless tobacco (moist snuff). J Clin Periodontol 2006;33(12):863-8.
312	29.	Kallischnigg G, Weitkunat R, Lee PN. Systematic review of the relation between
313		smokeless tobacco and non-neoplastic oral diseases in Europe and the United States.
314		<u>BMC Oral Health 2008;8:13-33.</u>
315	30.	Bergström J, Keilani H, Lundholm C, Rådestad U. Smokeless tobacco (snuff) use and
316		periodontal bone loss. J Clin Periodontol 2006;33(8):549-54.
317	31.	Rodu B, Jansson C. Smokeless tobacco and oral cancer: a review of the risks and
318		determinants. Crit Rev Oral Biol Med 2004;15(5):252-63.
319	32.	American Cancer Society. Child and Teen Tobacco Use. Available at:
320		http://www.cancer.org/docroot/PED/content/PED_10_2X_Child_and_Teen_Tobac
321		<u>co_Use.asp?sitearea=PED.</u> Accessed Mar. 28, 2009.
322	33.	<u>CDC.</u> Cigarette brand preference among middle and high school students who are
323		established smokers - United States, 2004 and 2006. MMWR Morb Mortal Wkly Rep
324		<u>2009;58(5):112-5.</u>
325	34.	Lavoto C, Linn G, Stead LF, Best A. Impact of tobacco advertising and promotion on
326		increasing adolescent smoking behaviours. Cochrane Database Syst Rev
327		<u>2003;(4):CD003439.</u>

328	35.	Gilman SE, Rende R, Boergers J, et al. Parental smoking and adolescent smoking
329		initiation: an intergenerational perspective on tobacco control. Pediatrics
330		<u>2009;123(2):e274-81.</u>
331	36.	Song AV, Morrell HE, Cornell JL, et al. Perceptions of smoking-related risks and
332		benefits as predictors of adolescent smoking initiation. Am J Public Health
333		<u>2009;99(3):487-92.</u>
334	37.	U.S. Department of Health and Human Services. Reducing Tobacco Use: A Report of
335		the Surgeon General. Atlanta: U.S. Dept of Health and Human Services, Centers for
336		Disease Control and Prevention, Coordinating Center for Health Promotion, National
337		Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and
338		Health, 2000. Available at:
339		www.cdc.gov/mmwr/preview/mmwrhtml/rr4916a1.htm Accessed Aug 6, 2009.
340	37.	Centers for Disease Control and Prevention. Best Practices for Comprehensive
341		Tobacco Programs-2007. Atlanta; US Dept. of Health and Human Services, Centers
342		for Disease Control and Prevention, National Center for Chronic Disease Prevention
343		and Health Promotion, Office on Smoking and Health; October 2007. (cited 2008 Mar
344		21). Available at:
345	ł	http://www.cdc.gov/tobacco/tobacco_control_programs/stateandcommunity/best
346		practices/ Accessed Aug 6, 2009.
347	38.	Fiore MC, Jaén CR, Baker TB, et al. Treating tobacco use and dependence: 2008
348		Update. Clinical Practice Guideline. Rockville, MD: U.S. Department of Health and
349		Human Services. Public Health Service. Available at:
350		www.ct.gov/dph/lib/dph/rfp_2009-0916.pdf May 2008. Access Aug 6, 2009.
351	39.	U.S. Dept of Health and Human Services. Treating Tobacco Use and Independence:
352		Clinical Practice Guidelines. 2008 Update. Available at:
353		ncbi.nlm.nih.gov/books/bv.fcgi?rid=hstat2.chapter.28163 Accessed Aug 6, 2009.
354	40.	http://www.surgeongeneral.gov/library/secondhandsmoke/factsheets/factsheet7.h
355		tml Accessed Nov 8, 2009.
356	41.	http://www.ada.org/prof/resources/positions/statements/tobac.asp_Accessed
357		<u>Nov 11, 2009.</u>
358	42.	http://www.ada.org/prof/resouces/positions/statements/smokeless.asp_Accessed
359		Nov 11, 2009.

1	
2	Policy on Use of a Caries-risk Assessment Tool (CAT) for Infants, Children, and
3	Adolescents Guideline on Caries Risk Assessment and Management
4 5	Originating Council
6	Council on Clinical Affairs
7	
8	Review Council
9 10	Council on Clinical Affairs
11	Adopted
12	2002
13	
14 15	Revised 2006-2010
16	2000, 2010
17	
18	Purpose
19	The American Academy of Pediatric Dentistry (AAPD) recognizes that caries-risk assessment
20	and guidelines can assist clinicians with decisions regarding treatment based on caries risk and
21	patient compliance; and is an essential element of contemporary clinical care for infants,
22	children, and adolescents, and persons with special health care needs. These is guidelines policy
23	is are intended to educate healthcare providers and other interested parties on the assessment of
24	caries risk in contemporary pediatric dentistry and aid in clinical decision making regarding
25	diagnostic, fluoride, dietary and restorative protocols.
26	
27	Methods
28	This policy revision is based on a review of the current dental and medical literature related to
29	caries-risk assessment tools and methodologies. A MEDLINE search was conducted using the
30	terms "caries risk", "caries assessment", and "caries management tool". Expert opinions and
31	best current practices also were relied upon for this policy.
32	
33	This guideline is an update of the previous document, Policy on Use of a Caries-risk
34	Assessment Tool (CAT) for Infants, Children, and Adolescents", revised in 2006, as well as
35	including the additional concepts of dental caries protocols. The update used electronic and
36	hand searches of English written articles in the medical and dental literature within the last ten

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- 37 years using the search terms, "caries risk assessment", "caries management", and "caries
- 38 <u>clinical protocols</u>". From this search 1,909 articles were evaluated by title or by abstract.
- 39 Information from 75 articles were used to update this guideline. When data did not appear
- 40 <u>sufficient or were inconclusive, recommendations were based upon expert and/or consensus</u>
- 41 <u>opinion by experienced researchers and clinicians</u>.
- 42

43 Background Caries Risk Assessment

- 44 <u>Risk assessment procedures currently used in medical practice normally have sufficient data</u>
- 45 to accurately quantitate disease susceptibility of a person and allow for preventive measures. 1
- 46 Even though caries risk data in dentistry still are not sufficient to quantitate the models, the
- 47 process of determining risk should be a necessary component in the clinical decision making
- 48 process.² Risk assessment fosters the treatment of the disease process instead of treating the
- 49 outcome of the disease; it gives an understanding of the disease factors for a specific patient and
- 50 <u>aids in individualizing preventive discussions with patients; it individualizes and selects and</u>
- 51 <u>determines frequency of preventive and restorative treatment for a patient; and it anticipates</u>
- 52 <u>caries progression or stabilization.</u>
- 53

54 Caries risk assessment models currently involves a combination of factors including diet, 55 fluoride exposure, a susceptible host, and microflora and that interplay with a variety of social, 56 cultural, and behavioral factors.³⁻⁶ Most young children appear to acquire some cariogenic 57 microbes [eg, mutans Sstreptococci (MS)] from their mothers or primary caregivers.⁶ 58 Traditionally, multifactorial caries-risk studies have focused on evaluation of biological, 59 demographic, and dietary factors and have used cavitation of a carious lesion (prevalence or 60 incidence) as the outcome variable.⁸ Caries risk assessment is the determination of the 61 likelihood of the incidence of caries (i.e., the number of new cavitated or incipient lesions) 62 during a certain time period, ⁷ or . It also involves the likelihood that there will be a change in 63 the size or activity of lesions already present. With the ability to detect caries in its earliest 64 stages (i.e., white spot lesions), health care providers can help prevent cavitation.⁸⁻¹⁰ 65

66 Strategies for managing caries increasingly have emphasized the concept of risk assessment
 67 as evidenced by incorporating such principles into guidelines for pit and fissure sealants,

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68 professional topical fluoride and radiographic criteria -19 In 2002, while recognizing that 69 assessment of caries risk undoubtedly would benefit from emerging science and technologies, 70 the AAPD took a first step toward incorporating available evidence into a framework for 71 classifying caries risk in infants, children, and adolescents.²⁰ This tool was based on a set of 72 physical, environmental, and general health factors and intended to be a dynamic instrument 73 that would be evaluated and revised periodically as new evidence warranted.²¹⁻²⁴ 74 75 Risk assessment is a necessary component in the clinical decision making process.²⁵ Caries 76 risk indicators are variables that either currently are thought to cause the disease directly (eg, 77 microflora) or have been shown useful in predicting it (eg, socioeconomic status). These risk 78 factors may vary with race, culture, and ethnicity²⁶⁻³² and may be useful in the clinical 79 management of caries by helping to determine if additional diagnostic procedures are required, 80 identify subjects who require caries control measures, assess the impact of caries control 81 measures, guide in treatment planning decisions, and determine the timing of recall 82 appointments.16,33-35 83 84 Since the etiology of caries is multi-factorial, it has been suggested that risk assessment 85 should be directed at the evaluation of all factors involved with the disease.^{36,37} Studies have 86 indicated that for the success of a caries risk assessment model, 1 or more social, behavioral, 87 microbiologic, environmental, and clinical variables should be included.^{31,33,38} However, 88 requiring an oral examination can hamper the utility of this process in population subgroups 89 that have not sought dental care (eg, many preschool children, especially those from minority 90 populations). 91 A systematic review of literature concerning caries risk indicators concluded that, for caries 92 93 prediction in primary teeth, previous caries experience was the best predictor,²⁵ followed by

94 level of parental education³⁹ and socioeconomic status.⁴⁰ While previous caries experience may

95 be the best indicator of future disease, using it to identify children at high risk comes too late to

96 prevent caries initiation. Most studies do not report the presence of noncavitated lesions,^{25,41,42}

97 although such lesions have been shown to have predictive value.^{43,44} Another important risk

98 factor in young children is the age of MS colonization. The earlier in infancy that high levels of

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99	MS colonization occur, the more severe the caries in the primary dentition.45 47 Early childhood
100	caries is an infectious process that too frequently requires expensive and extensive intervention.
101	Identifying factors that determine those individuals at highest risk—either prior to or very
102	shortly after teeth begin to erupt—is imperative to allow for possible preventive
103	intervention. ^{25,48} Once identified, these factors should be assessed using a reliable and valid tool
104	that is useable by both dental practitioners and trained nondental health professionals.49
105	
106	Caries risk indicators are variables that either currently are thought to cause the disease
107	directly (e.g., microflora) or have been shown useful in predicting it (e.g., socioeconomic status),
108	and includes those variables that may be considered protective factors. Currently, there are no
109	caries risk factors or combination of factors that have achieved high levels of both positive and
110	negative predictive values. ² Although the best tool to predict future caries is past caries
111	experience $\frac{2}{2}$ it is not particularly useful in young children since it is important to determine
112	caries risk before the disease is manifest. Children with white spot lesions should be considered
113	at high caries risk since white spot lesions are precavitated lesions that are indicative of caries
114	activity in the mouth. 11_Plaque accumulation also is strongly associated with caries
115	development in young children. ^{12,13} As a corollary to the presence of plaque 14, a child's mutans
116	streptococci levels, ³ and the age that a child becomes colonized with cariogenic flora 15-16 are
117	valuable in risk assessment, especially in preschool children.
118	
119	While there is no question that fermentable carbohydrates are a necessary link in the
120	causal chain for dental caries, a systematic study of sugar consumption and caries risk has
121	concluded that the relationship between sugar consumption and caries is much weaker in the
122	modern age of fluoride exposure than previously thought. ¹⁷ However, there is evidence that
123	night-time use of the bottle, especially when it is prolonged, may be associated with early
124	childhood caries. ¹⁸ Despite the fact that normal salivary flow is an extremely important
125	intrinsic host factor providing protection against caries, there is little data about the prevalence
126	of low salivary flow in children. ^{19,20}
127	
128	Sociodemographic factors have been extensively studied to determine their effect on

129 <u>caries risk. Children with immigrant backgrounds have three times higher caries rates than non</u>

130	immigrants. ²¹ Most consistently, an inverse relationships between socio-economic status and
131	caries prevalence is found in studies of children less than six years of age. 22 Perhaps another
132	type of sociodemographic variable is parents' history of cavities and abscessed teeth which has
133	been found to be a predictor of treatment for early childhood caries.23.24
134	
135	The most studied factors that are protective of dental caries include systemic and topical
136	fluoride, sugar substitutes, and tooth brushing with fluoridated toothpaste. Teeth of children
137	who reside in a fluoridated community have been shown to have higher fluoride content than
138	those of children who reside in suboptimal fluoridated communities. 25 Additionally, both pre-
139	and post-eruption fluoride exposure maximize the caries preventive effects. 26.27 For those
140	individuals that reside in non-fluoridated communities fluoride supplements have shown a
141	significant caries reduction in primary and permanent teeth.28 With regard to fluoridated
142	toothpaste, studies have shown consistent reduction in caries experience. ²⁹ Professional topical
143	fluoride applications performed semiannually also reduce caries, <u>30</u> and fluoride varnishes
144	generally are equal to that of other professional topical fluoride vehicles. ³¹
145	
146	The effect of sugar substitutes on caries rates have been evaluated in several populations
147	with high caries prevalence. ³² Studies indicate that xylitol can decrease mutans streptococci
148	levels in plaque and saliva and can reduce dental caries in young children and mothers. ³³ With
149	regard to tooth-brushing there only is a weak relationship between frequency of brushing and
150	decreased dental caries, which is confounded because it is difficult to distinguish whether the
151	effect is actually a measure of fluoride application or whether it is a result of mechanical
152	removal of plaque. ³⁴ Currently, the "dental home" or regular periodic care by the same
153	practitioner, is included in many caries risk assessment models because of its known benefit for
154	dental health. ³⁵
455	
155	
156	Kisk assessment tools can aid in the identification of reliable predictors and allow <u>dental</u>
157	practitioners, and trained nondental health care professionals physicians and other non-dental

159 <u>Tables 1, 2, and 3</u> The following table_incorporates-available evidence into a concise, practical

- 160 tools to assist both dental practitioners, and nondental health care providers physicians and
- 161 <u>other non-dental providers</u> in assessing levels of risk for caries development in infants, children,
- 162 and adolescents. As new evidence emergences, <u>these</u> this tools can be refined to provide and aid
- 163 in providing greater predictably of caries in children prior to disease initiation. Furthermore,
- 164 the evolution of <u>caries assessment tools and protocols</u> and CAT can assist in providing evidence
- 165 for and justifying periodicity of services, modification of third-party involvement in the
- 166 delivery of dental services, and quality of care with outcomes assessment to address limited
- 167 resources and workforce issues.
- 168
- 169 Individuals using this tool should:
- 170 <u>1. be able to visualize adequately a child's teeth and mouth and have access to a reliable</u>
- 171 historian for non-clinical data elements;
- 172 2. be familiar with footnotes that clarify use of individual factors in this instrument;
- 173 3. understand that each child's ultimate risk classification is determined by the highest risk
- 174 category where a risk indicator exists (ie, the presence of a single risk indicator in any area of
- 175 the "high-risk" category is sufficient to classify a child as being at "high risk"; the presence of at
- 176 least 1 "moderate-risk" indicator and no "high-risk" indicators results in a "moderate-risk"
- 177 classification; and a child designated as "low risk" would have no "moderate-risk" or "high-
- 178 risk" indicators).
- 179
- 180 Users of CAT must understand the following caveats:
- 181 1. CAT provides a means of classifying caries risk at a point in time and, therefore, should
- 182 be applied periodically to assess changes in an individual's risk status.
- 183 <u>2. CAT is intended to be used when clinical guidelines call for caries-risk assessment.</u>
- 184 Decisions regarding clinical management of caries, however, are left to qualified dentists
- 185 (ideally, the dentist responsible for the child's dental home).
- 186 <u>- 3. CAT can be used by both dental and nondental personnel. It does not render a</u>
- 187 diagnosis. However, indiduals using CAT must be familiar with the clinical presentation of
- 188 dental caries and factors related to caries initiation and progression.

189	
190	instrument, advanced technologies (ie, radiographic assessment and microbiologic testing) have
191	been included but are not essential for using this tool.
192	
193	
194	minimal, ^{15,49} as are guidelines for frequency of caries risk reevaluation. Since the carious process
195	is a fluctuating continuum, periodicity of reassessment should be based on risk status (ie,
196	greater frequency for children at high risk).
197	
198	Policy Statement
199	The AAPD:
200	-1. encourages both dental and non-dental health care providers to use CAT in the care of
201	infants, children, and adolescents and to provide basic preventive counseling;
202	-2. recommends that non-dental health care providers refer all children, especially those at
203	moderate or high risk, to a dentist for oral health care (ie, establish a dental home);
204	-3. encourages dentists to use advanced technologies such as radiographic assessment and
205	microbiologic testing with CAT when assessing an individual's caries risk;
206	-4. recognizes the need to evaluate CAT periodically and revise the tool as new science and
207	technologies warrant.
208	
209	The AAPD also encourages the scientific community to:
210	-1. identify additional predictors of caries experience (eg, survey parent for self-perception of
211	health and determine correlation to child's health);
212	-2. research genetic factors that contribute to an individual's susceptibility or resistance to
213	caries;
214	-3. develop technology to detect and quantify early carious lesions and to assess directly
215	carious lesion status (active vs inactive);
216	-4. provide evidence to establish clinical applications (eg, customized periodicity schedules,
217	preventive regimens, and / or treatment strategies) of CAT.
218	_
219	

- 220
- 221
- 222

223 Table legends

- 224 A Children with special health care needs are those who have a physical, developmental, mental,
- 225 sensory, behavioral, cognitive, or emotional impairment or limiting condition that requires
- 226 medical management, health care intervention, and/or use of specialized services. The
- 227 condition may be developmental or acquired and may cause limitations in performing daily
- 228 self-maintenance activities or substantial limitations in a major life activity. Health care for
- 229 special needs patients is beyond that considered routine and requires specialized knowledge,
- 230 increased awareness and attention, and accommodation.⁵⁰
- 231 ^B-Alteration in salivary flow can be the result of congenital or acquired conditions, surgery,
- 232 radiation, medication, or age-related changes in salivary function. Any condition, treatment, or
- 233 process known or reported to alter saliva flow should be considered an indication of risk unless
- 234 proven otherwise.
- 235 COrthodontic appliances include both fixed and removable appliances, space maintainers, and
- 236 other devices that remain in the mouth continuously or for prolonged time intervals and which
- 237 may trap food and plaque, prevent oral hygiene, compromise access of tooth surfaces to
- 238 fluoride, or otherwise create an environment supporting caries initiation.
- 239 ^D-National surveys have demonstrated that children in low-income and moderate-income
- 240 households are more likely to have caries and more decayed or filled primary teeth than
- 241 children from more affluent households. Also, within income levels, minority children are more
- 242 likely to have caries. Thus, socioeconomic status should be viewed as an initial indicator of risk
- 243 that may be offset by the absence of other risk indicators.
- 244 Examples of sources of simple sugars include carbonated beverages, cookies, cake, candy,
- 245 cereal, potato chips, French fries, corn chips, pretzels, breads, juices, and fruits. Clinicians using
- 246 caries-risk assessment should investigate individual exposures to sugars known to be involved
- 247 in caries initiation.

- 248 ^FOptimal systemic and topical fluoride exposure is based on use of a fluoride dentifrice and
- 249 American Dental Association/American Academy of Pediatrics guidelines for exposure from
- 250 fluoride drinking water and/or supplementation.
- 251 ^GUnsupervised use of toothpaste and at-home topical fluoride products are not recommended
- 252 for children unable to expectorate predictably.
- 253 ^HAlthough microbial organisms responsible for gingivitis may be different than those primarily
- 254 implicated in caries, the presence of gingivitis in an indicator of poor or infrequent oral hygiene
- 255 practices and has been associated with caries progression.
- 256 ⁺Tooth anatomy and hypoplastic defects (eg, poorly formed enamel, developmental pits) may
- 257 predispose a child to develop caries.
- 258 ^J-Advanced technologies such as radiographic assessment and microbiologic testing are not
- 259 essential for using this tool.
- 260
- 261 <u>Caries Management Protocols</u>
- 262 <u>Clinical management protocols are documents designed to assist in clinical decision</u>
- 263 making by providing criteria regarding diagnosis and treatment and that lead to recommended
- 264 <u>courses of action</u>. They are based on evidence from current peer-reviewed literature, the
- 265 <u>considered judgment of expert panels, as well as clinical experience of practitioners. These</u>
- 266 protocols should be updated frequently as new technologies and evidence develop.
- 267
- 268 Historically, the management of dental caries was based on the notion that it was a
- 269 progressive disease that eventually destroyed the tooth unless there was surgical and
- 270 restorative intervention. Decisions for intervention often were learned from unstandardized
- 271 <u>dental school instruction, and then refined by clinicians over years of practice. Still little is</u>
- 272 known about the criteria dentists use when making decisions involving restoration of carious
- 273 <u>lesions.³⁶</u>
- 274
- 275 It is now known that surgical intervention of dental caries alone does not stop the
 276 disease process. Additionally, many lesions do not progress, and tooth restorations have a

277	finite longevity. Therefore, modern management of dental caries should be more conservative
278	and includes early detection of noncavitated lesions, identification of an individual's risk for
279	caries progression, understanding of the disease process for that individual, and "active
280	surveillance" to apply preventive measures and carefully monitor for signs of arrestment or
281	progression.
282	
283	Caries management protocols for children further refine the decisions concerning
284	individualized treatment of caries and treatment thresholds based on a specific patient's risk
285	levels, age, and compliance with preventive strategies (Tables 4, 5, 6). Such protocols should
286	yield greater probability of success and better cost effectiveness of treatment than less
287	standardized treatment. Additionally, caries management protocols free practitioners of the
288	necessity for repetitive high level treatment decisions, standardize the decision making and
289	treatment strategies 36-38 eliminate treatment uncertainties, and guarantee more correct
290	strategies. ³⁹
291	
292	Current content of the present caries management protocol are based on results of
293	clinical trials, systematic reviews, and expert panel recommendations that give better
294	understanding to, and recommendations for, diagnostic, preventive and restorative treatments.
295	The radiographic diagnostic guidelines are based on the latest guidelines from the ADA. $\frac{40}{2}$
296	Systemic fluoride protocols are base on the CDC's recommendations for using fluoride. ²⁹
297	Guidelines for the use of topical fluoride treatment are based on the ADA's Council on Scientific
298	Affairs' recommendations for professionally applied topical fluoride, 41 the Scottish
299	Intercollegiate Guideline Network guideline for the management of caries in pre-school
300	children, ⁴² a Maternal and Child Health Bureau Expert Panel, ⁴³ and the CDC's fluoride
301	guidelines. ²⁹ Guidelines for pit and fissure sealants are based on ADA's Council on Scientific
302	Affairs recommendations for the use of pit-and-fissure sealants. ⁴⁴ Guidelines on diet
303	counseling to prevent caries are based on two review papers. 45.46 Guidelines for the use of
304	xylitol are based on the the AAPD's Oral Health Policy on Xylitol, ³² a well executed clinical trial
305	on high caries risk infants and toddlers, 47 and on two evidence-based reviews. 48,49 Active
306	surveillance (prevention therapies and close monitoring) of enamel lesions is based on the

307 <u>concept that treatment of disease may only be necessary if there is disease progression ;⁵⁰ that</u>

308	caries progression has diminished over recent decades, 51 and that the majority of proximal
309	lesions, even in dentin, are not cavitated.52
310	
311	Other approaches to the assessment and treatment of dental caries will emerge with
312	time, and with evidence of effectiveness, may be included in future guidelines on caries risk
313	assessment and management protocols. For example, there are emerging trends to use calcium
314	and phosphate remineralizing solution to reverse dental caries. ⁵³ Other fluoride compounds,
315	such as silver diamine fluoride, ⁵⁴ and stannous fluoride ⁵⁵ may be more effective than sodium
316	fluoride for topical applications. There has been interest in antimicrobials to affect the caries
317	rates, but evidence from caries trials still are inconclusive. ⁵⁶ However, some other proven
318	methods, such as prescription fluoride drops and tablets, may be removed from this protocol in
319	the future due to attitudes, risks or compliance.57.58
320	
321 322 323 324	References
325	2001;285:2486-2497.
326 327	2. Zero D, Fontana M, Lennon AM. Clinical applications and outcomes of using indicators of risk in caries management. J Dent Educ 2001;65:1126-32.
328 329	3. Litt MD, Reisine S, Tinanoff N. Multidimensional causal model of dental caries development in low- income preschool children. Public Health Reports 1995;110:607-617.
330 331 332	<u>4. Nicolau B, Marcenes W, Bartley M, Sheiham A. A life course approach to assessing causes of dental caries experience: The relationship between biological, behavioural, socio-economic and psychological conditions and caries in adolescents. Caries Res 2003;37(5):319-26.</u>
333 334	5. Featherstone JD. The caries balance: Contributing factors and early detection. J Calif Dent Assoc 2003;31:129-33.
335 336	<u>6. Featherstone JD. The caries balance: The basis for caries management by risk assessment.</u> Oral Health Prev Dent 2004;2(Suppl 1):259-64.
337	7. Reich E, Lussi A, Newbrun E. Caries-risk assessment. Int Dent J 1999;49:15-26.
338 339	<u>8. Ismail AI, Nainar SM, Sohn W. Children's first dental visit: Attitudes and practices of US pediatricians and family physicians. Pediatr Dent 2003;25:425-430.</u>
340 341	9. Tsang P, Qi F, Shi W. Medical approach to dental caries: Fight the disease, not the lesion. Pediatr Dent 2006;28:188-198.

- 342 <u>10. Crall JJ. Development and integration of oral health services for preschool-age children.</u>
- **343** <u>Pediatr Dent 2005;27:323-330.</u>
- 344 <u>11. Vadiakas G. Case definition, aetiology and risk assessment of early childhood caries (ECC):</u>
 345 <u>A revisited review. European Arch Paed Dent 2008;9:114-125.</u>
- 346 <u>12. Alaluusua S, Malmivirta R. Early plaque accumulation a sign for caries risk in young</u>
 347 children. Community Dent Oral Epidemiol 1994; 22:273-227.
- 348 <u>13. Roeters J, Burgesdijk R, Truin GJ van't Hof M. Dental caries and its determinants in 2- to 5-</u>
 349 <u>year old children. J Dent Child 1995;62:401-408.</u>
- <u>14. Lee CL, Tinanoff N, Minah G, Romberg E. Effect of mutans streptococci colonization on</u>
 <u>plaque formation and regrowth.</u> J. Pub Hlth Dentistry 2008; 68:57-60.
- 352 <u>15. Thibodeau EA, O'Sullivan DM, Tinanoff N. Mutans streptococci and caries prevalence in</u>
 353 preschool children. Community Dent Oral Epidemiol 1993;21:288-291.
- 354 <u>16. Grindefjord M, Dahllof G, Nilsson B, Modeer T. Prediction of dental caries development in</u>
- 355 <u>1-year old children</u>. Caries Res 1995;29:343-348.
- 356 <u>17. Burt BA Satishchandra P. The relationship between low birthweight and subsequent</u>
 357 <u>development of caries: A systematic review. J Dent Ed 2001;65:1017-1023.</u>
- 358 <u>18. Reisine S Douglass JM. Psychosocial and behavioral issues in early childhood caries</u>
 359 <u>Community Dent Oral Epidemiol. 1998;26(1 Suppl):45-48.</u>
- 360 <u>19. Cataldo WL Oppenheim FG. Physical and chemical aspects of saliva as indicators of risk</u>
 361 <u>for dental caries in humans. J Dent Ed. 2001;65:1054-1062.</u>
- 362 <u>20. Vanobbergen J Martens L Lesaffre E Bogaerts K Declerck D. The value of a baseline caries</u>
 363 <u>risk assessment model in the primary dentition for the prediction of caries increment in the</u>
 364 <u>permanent dentition. Caries Res 2001;35:442-450.</u>
- 365 <u>21. Nunn ME Dietrich T Singh HK Henshaw MM Kressin NR. Prevalence of early childhood</u>
 366 <u>caries among very young urban Boston children compared with US Children. Am Assoc Pub</u>
 367 Hlth Dent 2009;69:156-162.
- 368 <u>22. Vargas CM Crall JS Schneider DA. Sociodemographic distribution of pediatric dental</u>
 369 <u>caries: NHANES III, 1988-1994. JADA 1998;129:1229-1238.</u>
- 370 <u>23. Southward LH Robertson A Edelstein BL, et al. Oral health of young children in Mississippi</u>
- 371 Delta child care centers: A second look at early childhood caries risk assessment. J Pub Hlth Dent
 372 2008;68:188-195.
- 373 <u>24. Thitasomakul S, Piwat A, Thearmontree O, et al. Risks for early childhood caries analyzed</u>
 374 by negative binomial models. J Dent Res 2009;88:137-141.
- 375 <u>25. Weatherell J, Deutsch D, Robinson C, Hallsworth AS. Assimilation of fluoride by enamel</u>
- 376 <u>throughout the life of the tooth. Caries Res 1977;11:85-115.</u>

377 378 379	26. Backer Dirks O, Houwink B, Kwant GW. The results of 6 ½ years of artificial fluoridation of drinking water in The Netherlands – The Tiel Cumemborg experiment. Arch Oral Biol 1961;5:284-300.
380 381	27. Singh KA, Spencer AJ and Armfield JM. Relative effects of pre-and posteruption water fluoride on caries experience of permanent first molars. J Pub Heath Dent 2003;63:11-19.
382 383	28. Murray JJ, Naylor MN. Fluorides and dental caries. In, Prevention of Oral Disease. ed. Murray, JJ. Oxford University Press, Oxford. 1996;pp. 32-67.
384 385	29. CDC. Recommendations for using fluoride to prevent and control dental caries in the United State. MMWR Morb Mortal Wkly Rep 2001;50:1-42.
386 387 388	30. Ripa LW. A critique of topical fluoride methods (dentifrice, mouthrinses, operator-, and self- applied gels) in an era of decreased caries and increased fluorosis prevalence. J Pub Health Dent 1991;51:23-41.
389 390	31. Beltran-Aguilar ED, Goldstein JW, Lockwood SA. Fluoride varnishes: a review of their clinical use, cariostatic mechanism, efficacy and safety. JADA 2000;131:589-596.
391 392	32. Pediatric Dentistry Reference Manual. Policy on use of xylitol in caries prevention. Pediatr Dent 2008-2009; 31:36-37.
393 394	33. Ka L Milgrom P Rothen M. Xylitol, sweeteners, and dental caries. Ped Dent 2006;28:154- 163.
395 396	34. Reisine ST, Psoter W. Socioeconomic status and selected behavioral determinants and risk factor for dental caries. J Dent Ed 2001;65:1009-1016.
397 398	35. Nowak AJ Casamassimo PS. The dental home. A primary care oral health concept. JADA 2002;133:93-98.
399 400	36. Bader JD Shugars DA. What do we know about how dentists make caries-related treatment decisions. Community Dent Oral Epidemiology 1997; 25:97-103.
401 402	37. Anusavice K. Management of dental caries as a chronic infectious disease. J Dent Ed <u>1998;62:791-802.</u>
403 404	38. Benn DK Clark TD Dankel DD Kostewicz SH. Practical approach to evidence-based management of caries. J Am Coll Dent 1999;66:27-35.
405 406	39. White BA Maupome G. Clinical decision-making for dental caries management. JDent Ed 2001; 65:1121-1125.
407 408	40. ADA Council on Scientific Affairs. The use of dental radiographs. Update and recommendations. JADA 2006;137:1304-1312.
409	41. ADA Council on Scientific Affairs. Professionally applied topical fluoride: Evidence-based

410 <u>clinical recommendations. JADA 2006;137:1151-1159.</u>

411 42. Scottish Intercollegiate Guideline Network (SIGN). 2005. Prevention and Management of 412 dental decay in the pre-school child. http://www.sign.ac.uk/pdf/sign83.pdf. 413 43. Maternal and Child Health Bureau Expert Panel. Topical fluoride recommendations for 414 high-risk children: Development of decision support matrix. October 22–23, 2007, Altarum 415 Institute, Washington, DC. 416 44. American Dental Association Council on Scientific Affairs. Evidence-based clinical 417 recommendations for the use of pit-and-fissure sealants. JADA 2008;139:257-267. 418 45. Tinanoff, N. Association of diet with dental caries in preschool children. Dental Clin North 419 Am 2005;49:725-727. 420 46. Burt BA Pai S. Sugar consumption and caries risk: A systematic review. J Dent Ed. 421 2001;65:1017-1023. 422 47. Milgrom P Ly KA Tut OK Mancl L Roberts MC Briand K Gancio MJ. Xylitol pediatric 423 topical oral syrup to prevent dental caries. Arch Pediatr Adolesc Med 2009;163:601-607. 424 48. Maguire A Rugg-Gunn AJ. Xylitol and caries prevention – Is it a magic bullet? British Dent 425 J 2003;194:429-426. 426 49. Haves C. The effect of non-cariogenic sweeteners on the prevention of dental caries: a 427 review of the evidence. J Dent Ed 2001;65:1106-1109. 428 50. Parker C. Active surveillance: Toward a new paradigm in the management of early prostate 429 cancer. Lancet Oncol 2004;5:101-106. 430 51. Warren JJ Levy SM Broffitt B Kanellis MJ. Longitudinal study of non-cavitated carious 431 lesion progression in the primary dentition J Pub Hlth Dent 2006;66:83-87. 432 52. Anusavice KJ. Present and future approaches for the control of caries J Dent Ed 2005; 433 69:538-554. 434 53. Hicks J Garcia-Godoy F Flaitz C. Biological factors in dental caries: Role of remineralizatin and fluoride in the dynamic process of demineralization and remineralization. I Clin Ped Dent 435 436 2004;28:203-214. 437 54. Rosenblatt A, Stamford TCM, Niederman R. Silver diamine fluoride: A caries "silver-438 fluoride bullet". J Dent Res 2009;88:116-125. 439 55. Tinanoff N. Progress regarding the use of stannous fluoride in clinical dentistry. J Clinical 440 Dent 1995: 6: 37-40. 441 55. Twetman S. Prevention of early childhood caries (ECC). Review of literature published 442 1998-2007. Europ Archives Paed Dent 2008;9:12-18. 443 56. Caufield PW Desanayke AP Li Y. The antimicrobial approach to caries management. J Dent 444 Ed 2001,65:1091-1095.

- 445 <u>57. Ismail AI Hassen H. Fluoride supplements, dental caries and fluorosis. A systematic</u>
- 446 review. JADA 2008;139:1457-1468.
- 447 <u>58. Tinanoff N. Use of Fluorides, in Early Childhood Oral Health, eds, Berg J and Slayton RA,</u>
 448 <u>Wiley-Blackwell, pp 92-109, 2009.</u>
- 449
- 450 Alaluusua S. Longitudinal study of salivary IgA in children from 1 to 4 years old with reference
 451 to dental caries. Scand J Dent Res 1983;91(3):163-8.
- 452 American Academy of Pediatric Dentistry. Definition of persons with special health care needs.
 453 Pediatr Dent 2005;27(suppl):15.
- 454 American Academy of Pediatric Dentistry. Guideline on periodicity of examination, preventive
- 455 dental services, anticipatory guidance, and oral treatment for children. Pediatr Dent
 456 2005;27(suppl):84-6.
- 457 American Academy of Pediatric Dentistry. Policy on early childhood caries: Classifications,
- 458 consequences, and preventive strategies. Pediatr Dent 2005;27(suppl):31-3.
- 459 American Academy of Pediatrics. Oral health risk assessment: Timing and establishment of the
 460 dental home. Pediatrics 2003;111(5Pt1):1113-6.
- 461 American American Academy of Pediatric Dentistry. Policy on use of a caries-risk assessment
- 462 tool (CAT) in infants, children, and adolescents. Pediatric Dent 2002;24(suppl):15-7.
- 463 American Dental Association, US Dept of Health and Human Services. The selection of patients
- 464 for dental radiographic examinations 2004. Available at:
- 465 "http://www.ada.org/prof/resources/topics/radiography.asp". Accessed March 22, 2006.
- 466 Anderson MH, Shi W. A probiotic approach to caries management. Pediatr Dent 2006;28(2):151 467 3; discussion 192-8.
- 468 Beck JD, Kohout F, Hunt RJ. Identification of high caries risk adults: Attitudes, social factors and
 469 diseases. Int Dent J 1988;38(4):231-8.
- 470 Beck JD. Risk revisited. Comm Dent Oral Epidemiol 1998;26(4):220-5.
- 471 Brambilla E, Garcia-Godoy F, Strohmenger L. Principles of diagnosis and treatment of high472 caries-risk subjects. Dent Clin North Am 2000;44(3):507-40, vi.
- 473 Casamassimo P. Bright futures in practice: Oral health. Arlington, Va: National Center for
 474 Education in Maternal and Child Health; 1996.
- 475 CDC. Recommendations for using fluoride to prevent and control dental caries in the United
 476 States. MMWR-Recomm Rep August 17, 2001;50(RR14):1-42.
- 477 Demers M, Brodeur JM, Mouton C, Simard PL, Trahan L, Veilleux G. A multivariate model to
- 478 predict caries increment in Montreal children aged 5 years. Comm Dent Health 1992;9(3):273-81.
- 479 Disney JA, Graves RC, Stamm JW, Bohannan HM, Abernathy JR, Zack DD. The University of
- 480 North Carolina Caries Risk Assessment study: Further developments in caries risk prediction.
- 481 Comm Dent Oral Epidemiol 1992;20(2):64-75.

- 482 Ekstrand KR, Bruun G, Bruun M. Plaque and gingival status as indicators for caries progression
- 483 on approximal surfaces. Caries Res 1998;32(1):41-5.
- 484 Ekstrand KR, Ricketts DN, Kidd EA, Qvist V, Schou S. Detection, diagnosing, monitoring and
- 485 logical treatment of occlusal caries in relation to lesion activity and severity: An in vivo
- 486 examination with histological validation. Caries Res 1998;32(4):247-54.
- 487 Featherstone JD. Caries prevention and reversal based on the caries balance. Pediatr Dent
 488 2006;28(2):128-32;discussion 192-8.
- 489 Filstrup SL, Briskie D, da Fonseca M, Lawrence L, Wandera A, Inglehart MR. Early childhood
- 490 caries and quality of life: Child and parent perspectives. Pediatr Dent 2003;25(5):431-40.
- Huntington NL, Kim IJ, Hughes CV. Caries-risk factors for Hispanic children affected by early
 childhood caries. Pediatr Dent 2002;24(6):536-42.
- 493 Ismail AI, Sohn W. A systematic review of clinical diagnostic criteria of early childhood caries. J
- 494 Public Health Dent 1999;59(3):171-91.
- 495 Isokangas P, Alanen P, Tiekso J. The clinician's ability to identify caries risk subjects without
 496 saliva tests A pilot study. Comm Dent Oral Epidemiol 1993;21(1):8-10.
- 497 Kaste LM, Drury TF, Horowitz AM, Beltran E. An evaluation of NHANES III estimates of early
 498 childhood caries. J Public Health Dent 1999;59(3):198-200.
- 499 Klock B, Krasse B. A comparison between different methods for prediction of caries activity.
 500 Scand J Dent Res 1979;87(2):129-39.
- 501 Lee JY, Bouwens TJ, Savage MF, Vann WF, Jr. Examining the cost-effectiveness of early dental
 502 visits. Pediatr Dent 2006;28(2):102-5; discussion 192-8.
- 503 Li Y, Caufield PW. The fidelity of initial acquisition of mutans streptococci by infants from
 504 their mothers. J Dent Res 1995;74(2):681-5.
- 505 Linke HA, Kuyinu EO, Ogundare B, et al. Microbiological composition of whole saliva and
 506 caries experience in minority populations. Dent Clin North Am 2003;47(1):67-85, ix.
- 507 Mundorff SA, Billings RJ, Leverett DH, et al. Saliva and dental caries risk assessment. Ann NY
 508 Acad Sci 1993;694:302-4.
- 509 Ng MW. Multicultural influences on child-rearing practices: Implications for today's pediatric
 510 dentist. Pediatr Dent 2003;25(1):19-22.
- 511 Okunseri C, Badner V, Kumar J, Cruz GD. Dental caries prevalence and treatment need among
- 512 racial/ethnic minority schoolchildren. NY State Dent J 2002;68(8):20-3.
- 513 Pitts NB, Stamm JW. International Consensus Workshop on Caries Clinical Trials (ICW-CCT) -
- 514 Final / consensus statements: Agreeing where the evidence leads. J Dent Res 2004;83(Spec No 515 C):C125-8.
- 516 Punwani IC. Our multicultural society: Implications for pediatric dental practice. Pediatr Dent
- 517 2003;25(1):9-10.
- 518 Shiboski CH, Gansky SA, Ramos-Gomez F, Ngo L, Isman R, Pollick HF. The association of
- 519 early childhood caries and race/ethnicity among California preschool children. J Public Health
 520 Dent 2003;63(1):38-46.
- 521 Steiner M, Helfenstein U, Marthaler TM. Dental predictors of high caries increment in children.
 522 J Dent Res 1992;71(12):1926-33.
- 523 <u>Tinanoff N, Kanellis MJ, Vargas CM. Current understanding of the epidemiology, mechanisms,</u>
- 524 and prevention of dental caries in preschool children. Pediatr Dent 2002;24(6):543-51.
- 525 Trahan L, Soderling E, Drean MF, Chevrier MC, Isokangas P. Effect of xylitol consumption on
- the plaque-saliva distribution of mutans streptococci and the occurrence and long-term survival
 of xylitol-resistant strains. J Dent Res 1992;71(11):1785-91.
- 528 US Dept of Health and Human Services. Oral health in America: A report of the Surgeon
- 529 General. Rockville, Md: US Dept of Health and Human Services, National Institute of Dental
- 530 and Craniofacial Research, National Institutes of Health; 2000.
- 531 Weinstein P. Provider versus patient-centered approaches to health promotion with parents of
- 532 young children: What works/does not work and why. Pediatr Dent 2006;28(2):172-6; discussion
 533 192-8.
- Workshop on Guidelines for Sealant Use. Recommendations. J Pub Health Dent 1995;55(5 Spec
 No):263-73.
- 536 Young DA. Lasers in modern caries management Part II: CAMBRA. Pract Proced Aesthet
- 537 Dent 2005;17(1):65-6, 68.

Table 1: Physicians and Other Non-Dental Providers Caries Risk Assessment Form for 0-3 Year Olds**

	High Risk	Moderate	Protective
Biological Factors	Factors	Risk Factors	Factors
Mother/primary caregiver has active cavities	Yes		
Parent/caregiver has low socioeconomic status	Yes		
Child has >3 between meal sugar containing snacks or beverages per day	Yes		
Child is put to bed with a bottle containing natural or added sugar	Yes		
Child has special health care needs		Yes	
Child is a recent immigrant		Yes	
Protective Factors			
Child receives optimally fluoridated drinking water or fluoride supplements			Yes
Child has teeth brushed daily with fluoridated toothpaste			Yes
Child receives topical fluoride from health professional			Yes
Child has dental home/regular dental care			Yes
Clinical Findings			
Child has white spot lesions or enamel defects	Yes		
Child has visible cavities or fillings	Yes		
Child has plaque on teeth		Yes	

** Modified from Ramos-Gomez et al., CDA Journal 2007;35:687-702; and ADA Caries Risk Assessment Forms. <u>www.ada.org/prof/resources/topics/topics_caries_under6.doc and over6.doc</u>. Accessed Feb. 30, 2009.

Circling those conditions that apply to a specific patient helps the health care worker and parent understand the factors that contribute to or protect from caries. Risk assessment categorization of low, moderate or high is based on preponderance of factors for the individual. However, clinical judgment may justify the use of one factor in determining overall risk, for instance, frequent exposure to sugar containing snacks or beverages, or visible cavities.

Overall assessment of the child's dental caries risk High Moderate Low

Table 2: Dental Providers Caries Risk Assessment Form for 0-5 Year Olds**

	High Risk	Moderate	Protective
Biological Factors	Factors	Risk Factors	Factors
Mother/primary caregiver has active caries	Yes		
Parent/caregiver has low socioeconomic status	Yes		
Child has >3 between meal sugar containing snacks or beverages per day	Yes		
Child is put to bed with a bottle containing natural or added sugar	Yes		
Child has special health care needs		Yes	
Child is a recent immigrant		Yes	
Protective Factors			
Child receives optimally fluoridated drinking water or fluoride supplements			Yes
Child has teeth brushed daily with fluoridated toothpaste			Yes
Child receives topical fluoride from health professional			Yes
Child has dental home/regular dental care			Yes
Clinical Findings			
Child has more than one dmfs	Yes		
Child has active white spot lesions or enamel defects	Yes		
Child has elevated mutans streptococci	Yes		
Child has plaque on teeth		Yes	

** Modified from Ramos-Gomez et al., CDA Journal 2007;35:687-702; and ADA Caries Risk Assessment Forms. <u>www.ada.org/prof/resources/topics/topics_caries_under6.doc and over6.doc</u>. Accessed Feb. 30, 2009.

Circling those conditions that apply to a specific patient helps the practitioner and parent understand the factors that contribute to or protect from caries. Risk assessment categorization of low, moderate or high is based on preponderance of factors for the individual. However, clinical judgment may justify the use of one factor in determining overall risk, for instance, frequent exposure to sugar containing snacks or beverages, or more than one dmfs.

Overall assessment of the child's dental caries risk High
Moderate
Low

Table 3: Dental Providers Caries Risk Assessment Form for >6 Years Olds **

	High Risk	Moderate	Protective
Biological Factors	Factors	Risk Factors	Factors
Patient has >3 between meal sugar containing snacks or beverages per day	Yes		
Patient is of low socioeconomic status	Yes		
Patient has special health care needs		Yes	
Patient is a recent immigrant		Yes	
Protective Factors			
Patient receives optimally fluoridated drinking water			Yes
Patient brushes teeth daily with fluoridated toothpaste			Yes
Patient receives topical fluoride from health professional			Yes
Additional home measures (xylitol, MI paste, antimicrobial)			Yes
Patient has dental home/regular dental care			Yes
Clinical Findings			
Patient has one or more interproximal lesions	Yes		
Patient has active white spot lesions or enamel defects	Yes		
Patient has low salivary flow	Yes		
Patient has defective restorations		Yes	
Patient wearing an intraoral appliance		Yes	

** Modified from Featherstone et al., CDA Journal 2007;35:703—713; and ADA Caries Risk Assessment Forms. <u>www.ada.org/prof/resources/topics/topics_caries_under6.doc and over6.doc</u>. Accessed Feb. 30, 2009.

Circling those conditions that apply to a specific patient helps the practitioner and patient understand the factors that contribute to or protect from caries. Risk assessment categorization of low, moderate or high is based on preponderance of factors for the individual. However, clinical judgment may justify the use of one factor in determining overall risk, for instance, one or more interproximal lesion or low salivary flow.

Overall assessment of the dental caries risk High Moderate Low

Table 4: Example of a Caries Management Protocol for 1-2 Year Olds

Risk Category	Diagnostics	Interventions	Restorative	
		Fluoride	Diet	
Low Risk	Recall every 6-12 months Baseline MS α	Twice daily brushing with F toothpaste eta	Counseling	Surveillance χ
Moderate Risk	Recall every six months	Twice daily brushing with F toothpaste	Counseling	
parent engaged	Baseline MS	Fluoride supplements ${f \delta}$ Professional topical treatment every 6 mo.		Active surveillance ɛ
Moderate Risk	Recall every six months	Twice daily brushing with F toothpaste	Counseling, with limited	
parent not engaged	Baseline MS	Professional topical treatment every 6 mo.	expectations	Active surveillance
High Risk	Recall every three months	Twice daily brushing with F toothpaste	Counseling	Active surveillance
parent engaged	Baseline and follow up MS	Fluoride supplements		Restore cavitated lesions with ITR $ igoplus$ or
		Professional topical treatment every 3 mo.		definitive restorations
High Risk	Recall every three months	Twice daily brushing with F toothpaste	Counseling, with limited	Active surveillance of incipient lesions
parent not engaged	Baseline and follow up MS	Professional topical treatment every 3 mo.	expectations	Restore cavitated lesions with ITR or definitive restorations

Table 5: Example of a Caries Management Protocol for 3-5 Year Olds

Risk Category	Diagnostics	Interventions			Restorative
		Fluoride	Sealants	Diet	
Low Risk	Recall every 6-12 months Radiographs 12-24 months Baseline MS	Twice daily brushing with F toothpaste γ	(Yes) λ	No	Surveillance
Moderate Risk parent engaged	Recall every six months Radiographs 6-12 months Baseline MS	Twice daily brushing with F toothpaste Fluoride supplements Professional topical treatment every 6 mo.	Yes	Counseling	Active surveillance of incipient smooth surface lesions Restoration of cavitated or enlarging lesions
Moderate Risk parent not engaged	Recall every six months Radiographs 6-12 months Baseline MS	Twice daily brushing with F toothpaste Professional topical treatment every 6 mo.	Yes	Counseling, with limited expectations	Active surveillance of incipient smooth surface lesions Restoration of cavitated or enlarging lesions
High Risk parent engaged	Recall every three months Radiographs, six months Baseline and follow up MS	Brushing with .5% F (with caution) Fluoride supplements Professional topical treatment every 3 mo.	Yes	Counseling	Active surveillance of incipient smooth surface lesions Restoration of cavitated or enlarging lesions
High Risk parent not engaged	Recall every three months Radiographs, six months Baseline and follow up MS	Professional topical treatment every 3 mo.	Yes	Counseling, with limited expectations	Restore incipient, cavitated or enlarging lesions

Table 6: Example of a Caries Management Protocol for >6 Year-Olds

Risk Category	Diagnostics	Interventions			Restorative
		Fluoride	Sealants	Diet	
Low Risk	Recall every 6-12 months Radiographs 12-24 months	Twice daily brushing with F toothpaste μ	(Yes) λ	No	Surveillance
Moderate Risk patient engaged	Recall every six months Radiographs 6-12 months	Twice daily brushing with F toothpaste Fluoride supplements Professional topical treatment every 6 mo.	Yes	Counseling	Active surveillance of incipient smooth surface lesions Restoration of cavitated or enlarging lesions
Moderate Risk patient not engaged	Recall every six months Radiographs 6-12 months	Twice daily brushing with F toothpaste Professional topical treatment every 6 mo.	Yes	Counseling, with limited expectations	Active surveillance of incipient smooth surface lesions Restoration of cavitated or enlarging lesions
High Risk patient engaged	Recall every three months Radiographs, six months	Brushing with .5% F Fluoride supplements Professional topical treatment every 3 mo.	Yes	Counseling Xylitol	Active surveillance of incipient smooth surface lesions Restoration of cavitated or enlarging lesions
High Risk patient not engaged	Recall every three months Radiographs, six months	Brushing with .5% F Professional topical treatment every 3 mo.	Yes	Counseling, with limited expectations Xylitol	Restore incipient, cavitated or enlarging lesions

Legends, Tables 4-6

- α Salivary mutans streptococci bacterial levels
- $\beta\,$ Parental supervision of a "smear" amount of tooth paste
- χ Surveillance = periodic monitoring for signs of caries progression
- δ Need to consider fluoride levels in drinking water
- ϵ Active Surveillance = careful monitoring of caries progression and prevention program
- ♦ ITR Interim Therapeutic Restoration (AAPD Reference Manual, 2008)
- γ Parental supervision of a "pea sized" amount of tooth paste
- $\boldsymbol{\lambda}$ indicated for teeth with deep fissure anatomy or developmental defects
- $\boldsymbol{\mu}$ Less concern about the quantity of tooth paste

Fig 1: Clinical Management of Avulsed Permanent Incisors with an Open Apex





Follow-up: 1 w, 1 m, 3 m, 6 m, 12 m, and annually for 5 y

Fig 2: Clinical Management of Avulsed Permanent Incisors with a Closed Apex



Follow-up: 1 w, 1 m, 3 m, 6 m, 12 m, and annually for 5 y

1	AMERICAN ACADEMY OF PEDIATRIC DENTISTRY						
2	LOGO (AAPD)						
3							
4	A Message to Parents and School Administrators						
5	Regarding School Absence for Dental Appointments						
6							
/	Tooth decay in children can be painful, just as it is in adults. Although largely preventable with						
8	proper home care and regular dental visits, untreated tooth decay diminishes the child's ability						
9	to eat, sleep, and function well at home or at school. The unesthetic nature of tooth decay and						
10	dental malocclusion also may compromise the child's self-esteem and social development.						
11	Prevention, early diagnosis, and treatment of oral health conditions require professional care.						
12							
13	The American Academy of Pediatric Dentistry recognizes the importance of regular school						
14	attendance and the effect of chronic absence on academic performance. We encourage our						
15	members to work with parents and educators to minimize school absence whenever possible. It						
16	is not always possible or appropriate to provide all the care children need during non-school						
17	hours. School policies that prevent legitimate school absence for the purpose of dental visits						
18	may have a negative impact on the health and well being of children.						
19							
20	The American Academy of Pediatric Dentistry urges parents, school administrators, and						
21	member dentists to work together to ensure that children receive needed health care while						
22	minimizing school absences. The American Academy of Pediatric Dentistry encourages						
23	parents to return their children to school for the remainder of the day after their dental visit						
24	when possible. In the case of an appointment later in the day, the child should attend school						
25	and be excused prior to their dental appointment.						
26							
27	School Attendance Release						
28							
29	had an appointment						
30	in this office on from to						
31	for necessary oral health care.						

- 1 Policy on Second Opinion
- 2
- 3 Originating Council
- 4 Council on Clinical Affairs
- 5 Adopted
- 6 2010
- 7

8 Purpose

9 The American Academy of Pediatric Dentistry (AAPD) recognizes that second opinions are one avenue for additional information regarding health care issues. Parents frequently seek additional information and/or other opinions in order to address their child's health care needs and make informed decisions. This policy is intended to provide guidance to the pediatric dentist by addressing what should be obtained and documented when providing a second opinion, as well as an understanding of the ethical and legal obligations involved in the process.

15

16 Methods

17 This policy is based on a review of the current dental and medical literature related to the

18 concept of second opinion in dentistry and medicine. An electronic search and a MEDLINE

19 search was conducted using the search terms, "second opinion", "medical second opinion", and

- 20 "dental second opinion". Documents relating to principles of ethics of dental and medical
- 21 organizations were also reviewed.

22

23 Background

24 Patients today are more informed about their medical and dental problems and treatment

options, and are playing an active role in decision making with their doctors. Medical and
dental knowledge and available treatment options are also evolving at a rapid pace and it is
becoming increasingly difficult for health care providers to be fully aware of all the latest
advances and information. As a result, patients and health care providers are seeking second
opinions so that more informed decisions can be made regarding the patient's health care based
on the risks and benefits.

For minor health problems, second opinions are usually unnecessary. However, a second 31 32 opinion is recommended if the patient disagrees with or questions the diagnosis or the 33 treatment plan of the health care provider, has multiple medical problems, or is diagnosed with a life-threatening disease such as cancer. Second opinions are also recommended when there 34 35 is no improvement in the patient's condition, when there is a communication barrier between 36 the patient and the provider and additionally, when extensive oral care needs or high cost may 37 make treatment prohibitive. For the health care practitioner, a second opinion or referral may be warranted if they are unsure of the diagnosis, or the diagnosis/treatment is beyond the 38 39 scope of their expertise. A second opinion may also be necessary when required by a third party 40 payor.

41 There is a debate among health care providers about whether to provide a patient with a "blind" second opinion (ie, when medical/dental records, test results and first provider's 42 43 opinion are not made available to the second provider) or if the patient's diagnostic information 44 and diagnosis should be shared with the provider giving the second opinion. The advantage of 45 the "blind" second opinion is that it cannot be influenced by previous information. The provider will develop their own unbiased opinion. The disadvantage is that the provider performing the 46 47 second opinion may have to repeat diagnostic tests and the patient will incur additional costs. The second provider may also not be able to explain to the patient why his/her opinion is 48 49 different without knowing the patient's previous history and the basis of the first provider's 50 opinion. A third option would be to provide test results, radiographs and other information 51 without the first doctor's written diagnosis and treatment recommendations. This would allow 52 for an unbiased opinion without having the patient incur unnecessary charges or radiation exposure from repeated radiographs. 53

Council on Clinical Affairs 2009-10 Charge 5(a) P-Second Opinion

54 When presented with requests for second opinions, practitioners should consider the legal 55 implications of such request. Health care providers rendering second opinions could be 56 unwarily involved in litigation, either on behalf of the patient or in defending themselves against other practitioners, as a result of the consult. ⁶The fact that one is the second or third 57 58 professional consulted does not mean that the provider is exempt from liability.⁷ A dissatisfied patient could file a lawsuit naming not only the treating doctor, but also the doctor rendering 59 60 the second opinion as defendants. In addition, a colleague who believes his or her professional reputation has been damaged by statements made to a patient during a consultation could file a 61 62 lawsuit for defamation of character. Patients should be advised of their health status without 63 disparaging comments about their prior treatment or previous provider.

64 **Policy statement**

- 65 The AAPD recommends that:
- A patient has a right to a second opinion. A provider who is trained and experienced in
 diagnosing and treating the condition should provide the second opinion. Internet sites or a
 telephone conversation cannot be relied upon and should not constitute a second opinion.¹
- A health care practitioner has the right to request a second opinion from one or multiple
 practitioners/specialists as deemed necessary to facilitate the optimal clinical outcome.²

Educating the patient regarding the diagnosis, available treatment options, their risks and
 benefits should be the goal of a second opinion consultation. Health care providers may
 disagree on the best treatment for an individual patient. Any opinion should be rendered
 only on careful consideration of all the facts and with due attention given to current and
 previous states of the patient. Instances of gross or continual faulty diagnosis or treatment
 by other providers may require that the provider be reported to an appropriate reviewing
 agency as determined by the local component or constituent dental society.²

4. A provider has the ethical obligation on request of either the patient or the patient's new
provider to furnish records, including radiographs or copies of them. These may_be
beneficial for the future treatment of that patient ². HIPAA privacy rules and state laws

- 81 apply to all exchanges of health care information.
- 5. Second opinions may be mandatory by third party payers. The provider should be
 independent and the opinion should be based on best outcomes for the patient and not
 financial incentives.
- 85 6. Most second opinions are voluntary. It is the responsibility of the patient to check with their86 insurance carrier for specific policies and benefits regarding coverage of second opinions.
- 87 7. When presented with requests for second opinions, practitioners should consider the legal
- 88 implications of such request. Patients should be fully advised of their health status without
- 89 disparaging comments about their prior treatment or previous provider.
- 90
- 91
- 92

93 **References**

- 94 1. http://www.womenshealth.gov/tools/secondopinion.cfm. Accessed March 14, 2009
- 95 2. ADA Principles of Ethics and Code of Professional Conduct. Revised January 2009.
- 96 3. http://www.cancer.org/docroot/ETO/content/ETO_1_7X_Should_I_Get_A_Second_O
 97 pinion.asp. Accessed March 14, 2009.
- 98 4. http://www.medicare.gov/publications/pubs/pdf/02173.pdf. Accessed April 27,
 99 2009.
- 100 5. http://www.ynhh.org/choice/secondopinion.html. Accessed March 14, 2009.
- 101 6. Second opinions 8.03.3 AAO
- Machen, D. Legal Aspects of Orthodontic Practice: Risk Management Concepts. Am J
 Orthod Dentofac Orthop; March 1990
- 104

- 1 Policy on Absences from School for Dental Appointments
- 2
- **3** Originating Council
- 4 Council on Clinical Affairs
- 5 Adopted
- 6 2010 7
- 8 Purpose
- 9 The American Academy of Pediatric Dentistry (AAPD) recognizes dental care as medically
- 10 necessary and that poor oral health can negatively affect a child's ability to learn. This policy is
- 11 intended to assist public health and school education administrators developing enlightened
- 12 policies on school absence for dental appointments. It also is intended to assist parents in
- 13 making informed decisions.
- 14

15 Methods

- 16 This policy is based on a review of current dental, medical, educational, and public health
- 17 literature and state statute and regulation related to student absence for dental appointments.
- 18 An electronic search was conducted using the search terms, "school absences for dental
- 19 appointments", "excused school absences", and "department of education". Fields: All fields;
- 20 Limits: within the last 10 years; humans; English. 841 articles matched these criteria. Papers for
- 21 review were chosen from this list and from references within selected articles. When data did
- 22 not appear sufficient or were inconclusive, recommendations were based on expert and/or
- 23 consensus opinion by experienced researchers and clinicians. It is beyond the scope of this
- 24 document to review every state statute and regulation on absences from school for dental
- 25 appointments.
- 26

27 Background

Oral health is integral to general health. Oral conditions can interfere with eating and adequate nutritional intake, speaking, self-esteem and daily activities.¹ Dental care is medically necessary to prevent and eliminate orofacial disease, infection, pain. It is also important to restore the form and function of the dentition, and correct facial disfiguration or dysfunction.² The public's lack of awareness of the importance of oral health is a major barrier to dental care.³

Unrecognized disease and postponed care result in exacerbated problems, which lead to more
 extensive and costly treatment needs.²

The National Association of State Boards of Education recognizes "health and success in
 schools are interrelated. Schools cannot achieve their primary mission of education if students
 and staff are not healthy and fit physically, mentally, and socially".⁴

38 Children with dental pain may be irritable, withdrawn, or unable to concentrate. Pain can affect

39 test performance as well as school attendance.³ Left untreated, the pain and infection caused

40 by tooth decay can lead to problems in eating, speaking, and learning.⁵ In 1996, students aged

41 5 to 17 missed an average of 3.1 days/100 students due to acute dental problems.¹ When these

42 problems are treated and children no longer are experiencing pain, their learning and school

43 attendance improved.¹

According to the US Surgeon General, "a national public health plan for oral health does not exist".³ This corresponds with the fact that there is no national policy on excused absences from school for dental appointments. Some states, for example California and Texas have very specific laws excusing students for dental appointments. ^{6,7} Other states laws are more general and recognize absences due to doctors appointments or illness.^{8,9}

49

51

50 Policy statement

Dental care is medically necessary and oral health is integral to general health.

52 Undiagnosed and untreated oral conditions may interfere with a child's ability to eat, sleep, or

53 function well at home or at school due to discomfort or pain. The unesthetic nature of caries and

54 dental malocclusion may compromise a child's self-esteem and social development. School

55 policies that prevent or discourage legitimate school absence for the purpose of delivery of vital

56 health care services may cause harm to their students.

57 Children who have dental conditions corrected improve their learning and attendance in

58 school. State laws and local school district policies are not uniform on this issue. A uniform

59 policy guideline that recognizes the negative effect of chronic truancy on academic performance

60 would be useful. This policy should not restrict necessary health care delivery.

61 The AAPD:

62	1.	Recommends state law or school policy that allows the absence for legitimate healthcare
63		delivery, including that of oral health services.
64	2.	Encourages parents, school administrators, and dentists to work together to ensure that
65		children receive dental care while minimizing school absences.
66		
67	Refere	ences
68	1.	National Center for Education in Maternal and Child Health and Georgetown
69		University. Fact sheet: Oral health and learning. Arlington, Va: NCEMCH; 2001.
70		http://www.collierhealthdept.org/pdf/dental/oralhealthlearning.pdf. accessed
71		<u>October 21, 2009.</u>
72	2.	American Academy of Pediatric Dentistry. Policy on Medically Necessary Care. Pediatr
73		Dent 2009;31(supl):16-20.
74	3.	US Dept of Health and \underline{H} uman Services. Oral health in America: A report of the
75		Surgeon General. Rockville, Md: US Dept of Health and Human Services, National
76		Institute of Dental and Craniofacial Research, National institutes of Health; 2000:12.
77	4.	Bogden JF, Vega-Matos CA, Fit, healthy, and ready to learn: A school health policy
78		guide, part 1: Physical activity, healthy eating, and tobacco-use prevention. Alexandria,
79		Va: National Association of State Boards of Education; 2000.
80	5.	U.S. General Accounting Office. 2000. Oral Health; Dental Disease is a Chronic Problem
81		Among Low-income and Vulnerable Populations. Washington, DC;U.S. General
82		Accounting Office
83	6.	California Education Code. http://Law.Onecle.com/california/education/48205.html.
84		accessed March 8, 2010.
85	7.	Texas Education Code. http://law.onecle.com/Texas/education/25.087.00.html.
86		Accessed March 8, 2010.
87	8.	Georgia Department of Education.
88		www.doeK12.ga.us/_documents/doe/legalservices/160-5-110pdf. Accessed March 8,
89		2010.

- 90 9. Michigan Department of Education.
- 91 <u>http://www.Michigan.gov/documents/mde/compulsory_attendance_257944_7.pdf.</u>
- **92** <u>Accessed March 8</u>, 2010.
- 93

AMERICAN ACADEMY OF PEDIATRIC DENTISTRY LOGO (AAPD)

A Message to Parents and School Administrators Regarding School Absence for Dental Appointments

Tooth decay in children can be painful, just as it is in adults. Although largely preventable with proper home care and regular dental visits, untreated tooth decay diminishes the child's ability to eat, sleep, and function well at home or at school. The unesthetic nature of tooth decay and dental malocclusion also may compromise the child's self-esteem and social development. Prevention, early diagnosis, and treatment of oral health conditions require professional care.

The American Academy of Pediatric Dentistry recognizes the importance of regular school attendance and the effect of chronic absence on academic performance. We encourage our members to work with parents and educators to minimize school absence whenever possible. It is not always possible or appropriate to provide all the care children need during non-school hours. School policies that prevent legitimate school absence for the purpose of dental visits may have a negative impact on the health and well being of children.

The **American Academy of Pediatric Dentistry** urges parents, school administrators, and member dentists to work together to ensure that children receive needed health care while minimizing school absences. The **American Academy of Pediatric Dentistry** encourages parents to return their children to school for the remainder of the day after their dental visit when possible. In the case of an appointment later in the day, the child should attend school and be excused prior to their dental appointment.

School Attendance Release

in this office on	from	to	

_____ had an appointment

for necessary oral health care.

Council on Clinical Affairs 2009-10 Charge 5(b) P-School Absences for Dental Appointments