

Report of the Council on Clinical Affairs

Proposed changes/additions to oral health policies and clinical recommendations of the American Academy of Pediatric Dentistry

3/27/2019

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NOTICE TO ACTIVE AND LIFE MEMBERS

(1) Reference Committee Hearing and Reports &

(2) General Assembly Meeting

Constitution and Bylaws amendments and proposed changes/additions to oral health policies and clinical recommendations 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 recommendations were posted as a Members-only document on the AAPD website (*www.aapd.org*) no later than sixty (60) days prior to the General Assembly. All members will be alerted to this availability via *AAPD E-News*.

The Reference Committee hearing will take place on Saturday, May 25, 2019, from 10 to 11 a.m., in the Waldorf Room at the Hilton Chicago. 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 recommendations, and other business to come before the General Assembly.

Reference Committee Reports will be available in the back of the Waldorf Room at the Hilton Chicago beginning at 8:30 a.m., on Sunday morning May 26, 2019, prior to the beginning of the General Assembly and Awards Recognition at 9:30 a.m. If available in time, copies will also be provided at District Caucuses on Saturday, May 25, 2019, from 1 to 2 p.m.

The Awards Recognition and General Assembly will take place on Sunday, May 26, 2019, from 9:30 to 11:30 a.m., in the Waldorf Room of the Hilton Chicago. 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. An agenda for the General Assembly meeting will be posted on the AAPD website (*www.aapd.org*) approximately one month prior to the meeting. All members will be alerted to this availability via *AAPD E-News*.



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1 Definition of Medically-Necessary Care

- 2
- 3
- 4 Review Council
- 5 Council on Clinical Affairs
- 6 Latest Revision Reaffirmation
- 7 2015 <u>2019</u>
- 8

9 Medically-necessary care (MNC) is the reasonable and essential diagnostic, preventive, and treatment 10 services (including supplies, appliances, and devices) and follow-up care as determined by qualified 11 health care providers in treating any condition, disease, injury, or congenital or developmental 12 malformation to promote optimal health, growth, and development. MNC includes all supportive 13 health care services that, in the judgment of the attending dentist, are necessary for the provision of 14 optimal quality therapeutic and preventive oral care. These services include, but are not limited to, 15 sedation, general anesthesia, and utilization of surgical facilities. MNC must take into account the patient's age, developmental status, and psychosocial well-being, in addition to the setting appropriate 16 17 to meet the needs of the patient and family. 18 19 Dental care is medically-necessary to prevent and eliminate orofacial disease, infection, and pain, to 20 restore the form and function of the dentition, and to correct facial disfiguration or dysfunction.

21

- 22 This definition was originally developed by the Council on Clinical Affairs and adopted in 1997. This
- 23 document is an update <u>a reaffirmation</u> of the previous version, revised in 2011 2015 version.

¹ Policy on a Patient's Bill of Rights and Responsibilities^{*}

- 2
- 3 Review Council
- 4 Council on Clinical Affairs
- 5 Reaffirmed
- 6 2014
- 7 Latest Revision
- 8 <u>2019</u>
- 9
- 10 Purpose
- 11 The American Academy of Pediatric Dentistry (AAPD) recognizes that pediatric oral health care
- 12 should be rendered under conditions that are acceptable to both patient and dentist. The expectation is
- 13 that oral health care providers, their staff, and patients, and parents of patients will support this policy,
- 14 thereby enhancing patient care.
- 15

16 Methods

- 17 This policy was adopted in 2009. The updated document used electronic database and hand searches
- 18 of the articles in the medical and the dental literature using the following parameters: Terms:: patient
- 19 freedoms, patient's Bill of Rights, Bill of Rights, Consumer Bill of Rights; Fields: all; Limits: within
- 20 the last 10 years and English; birth through age 18. Additionally, when data did not appear sufficient
- 21 or were inconclusive, recommendations were based upon expert and/or consensus opinion by
- 22 experienced organizations relating to the concept of patient rights and responsibilities.
- 23 This policy was adopted in 2009, and is based on a systematic literature search of the PubMed-
- 24 /MEDLINE database using the terms: patient freedoms, patient's Bill of Rights, Bill of Rights,
- 25 Consumer Bill of Rights; fields: all; limits: within the last 10 years and English. Eighty one articles-
- 26 met these criteria. Papers for review were chosen from this list and from the references within-
- 27 selected articles. Documents of health care and public policy organizations relating to the concept of
- 28 patient rights and responsibilities also were reviewed.(AAPD Vision, AHA 1992, President's-
- 29 Advisory Commission 1998)

* ABBREVIATION

AAPD: American Academy Pediatric Dentistry.

30	

31 Background

The AAPD is the leader in representing the oral health interests of infants, children, adolescents, and 32 persons with special health care needs.¹Effective oral health care requires collaboration between 33 34 pediatric dentists, their patients/parents and pediatric dentists and other health care professionals. 35 Optimal patient care requires honest and open communication between provider and patient, mutual respect for personal and professional values, and sensitivity to cultural differences.² Open and honest-36 communication, respect for personal and professional values, and sensitivity to differences are 37 38 integral to optimal patient care. 39 40 Often, the delivery of contemporary pediatric oral health care many times can be confusing to parents. It is normal for parents to have expectations about their child's proposed care. It is important that 41 parents have realistic expectations in order for them to have a clear understanding of their 42 43 responsibilities in the delivery of care to their children. whose children have planned oral health care-44 treatment to have a set of expectations about the proposed care. Likewise, it is desirable for these parents to have a clear understanding of their responsibilities in the delivery of care to their children. 45 46 Patient's Bill of Rights and Responsibilities A Patient's Bill of Rights-is a statement of the rights to 47 48 which patients are entitled as recipients of medical/dental care. These rights can be exercised on the 49 patient's behalf by a parent or legal guardian if the patient is a minor, lacks decision-making capacity, 50 or is legally incompetent. It articulates the positive rights that health care providers and facilities 51 should provide patients, thereby providing information, offering fair treatment, and granting them 52 autonomy over medical decisions. 53 54 The collaborative nature of health care requires that patients, or their families/surrogates, participate 55 in their care. The effectiveness of care and patient satisfaction with the course of treatment depend, in 56 part, on the patient's fulfilling certain responsibilities. As such, the AAPD proposes this Policy on a 57 Patient's Bill of Rights and Responsibilities in the planning and delivery of pediatric oral health care. 58 The AAPD encourages oral health care providers to tailor this Bill of Rights and Responsibilities to

- their patient community by translating and/or simplifying it as may be necessary to ensure that
- 60 patients and their families understand their rights and responsibilities.³
- 61

62 Bill of rights

- 63 these rights can be exercised on the patient's behalf by a parent or legal guardian if the patient is a
- 64 minor, lacks decision-making capacity, or is legally incompetent. The patient has the right to:
- 65 Patient's Rights include:
- <u>Receive treatment at a</u> dental home that provides comprehensive, considerate, and respectful
 care.
- Have oral health diagnoses made by a dentist.
- Know the identity, education, and training of the providers involved in his/her care, as well as
 when those involved are students, residents, or other trainees.⁴
- Choose an A choice of oral health care provider. The parent has a right to designate a pediatric
 dentist as a primary oral health care provider for their child.
- Participate fully in all the decisions related to his/her care.
- Receive accurate, relevant, current, and easily-understood information concerning diagnosis,
 treatment, and prognosis.
- The patient is entitled to the opportunity to Discuss and request information related to the
 specific procedures and/or treatments, <u>including accompanying risks and benefits</u>, and the risks involved, and the medically reasonable. alternatives and their accompanying risks and benefits.
 Life threatening emergency care could be an exception.
- Make decisions about the plan of care prior to and during the course of treatment, to refuse a
 recommended treatment or plan of care to the extent permitted by law, and to be informed of
 the health consequences of this action refusal. In case of such refusal, the patient is entitled to
 other appropriate care and services that the pediatric dentist offers or to transfer to another
 dentist.⁵
- Consent to or decline to participate in proposed research studies affecting care and treatment or
 requiring direct patient involvement
- and to Have those studies explained fully prior to consent. A patient who declines to participate
 in research is entitled to the most effective care that the pediatric dentist can otherwise provide.
- Expect reasonable continuity of care.
- 90 <u>Receive</u> emergency care <u>as needed</u> for acute dental trauma and odontogenic infections, as needed.
 91 needed.
- 92 Know the identity, education, and training of the providers involved in his/her care, as well as
 93 when those involved are students, residents, or other trainees.
- Know the immediate and long-term financial implications of treatment choices, insofar as they

- 95 are known by the provider. The patient has the right to be informed of the charges for services96 and available payment methods.
- Be informed of the provider's policies and practices that relate to patient care, treatment, and
 responsibilities. <u>This includes patient has the right to be informed of</u> available resources for
 resolving disputes, grievances, and conflicts, such as ethics committees, patient representatives,
 or other mechanisms available in an organization.
- Have privacy considered in every interaction. Every consideration of privacy. Case discussion,
 consultation, examination, and treatment should be conducted <u>in such a way that best protects</u>
 so as to protect each patient's privacy.
- Expect that all communications and records pertaining to his/her care will be treated as
 confidential, except in cases <u>where reporting is permitted or required by law</u>, such as suspected
 abuse and public health hazards. when reporting is permitted or required by law. The patient
 has the right to expect that the provider will emphasize the confidentiality of information
- 108 released to other parties entitled to review this information.
- 109 Advise staff regarding specific privacy concerns or questions.³
- Review the records pertaining to his/her medical care and to have the information explained or
 interpreted as necessary, except when restricted by law. The patient has the right to request
 amendments to his/her record.
- Ask and be informed of the existence of business relationships among institutions, other health
 care providers, or payers that may influence the patient's treatment and care.
- 115
- 116 Patient's Bill of r-Responsibilities:
- 117 The responsibilities can be exercised on the patient's behalf by a parent or legal guardian if the patient118 is a minor, lacks decision-making capacity, or is legally incompetent. <u>The patient is responsible for:</u>
- for p-Providing, to the best of his/her knowledge, accurate and complete information about past
 illnesses, hospitalizations, medications, and other <u>health status-related</u> matters. related to
 his/her health status.
- The patient must take responsibility for r Requesting additional information or clarification
 about his/her health status or treatment when he/she does not fully understand information and
 instructions.
- The patient is responsible for <u>H</u>is/her actions if he/she refuses treatment or does not follow the instructions of the provider.³ It is the patient's responsibility to inform dentists and other

127	caregivers of anticipated problems in following prescribed treatment, including follow-up
128	treatment instructions.
129	• The patient has a responsibility to $k \underline{K}$ eeping appointments and when unable to do so, to notify
130	the dental office as soon as possible.
131	• The patient is responsible for b-Being considerate of the rights of other patients and health care
132	workers. ⁶ This includes not and for not interfering with the general functioning of the facility,
133	nor using profane or derogatory behavior, and minimizing noise. ⁷
134	• <u>The patient is responsible for h His/her conduct with staff. The patient must resolve conflicts</u>
135	using available institutional mechanisms. Verbal and physical abuse of staff is prohibited. ⁸
136	• The patient is responsible for p Providing accurate insurance information and for accepting the
137	financial obligations associated with the services rendered.
138	• Following HIPAA guidelines including not taking videos/photographs of people and/or
139	protected health information.
140	
141	References
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¹ Policy on Infection Control^{*}

- 2
- 3 Review Council
- 4 Council on Clinical Affairs
- 5 Latest Revision
- 6 <u>2014</u> <u>2019</u>
- 7
- 8 Purpose
- 9 The American Academy of Pediatric Dentistry (AAPD) recognizes the importance of infection
- 10 control policies, procedures, and practices in dental health care settings in order to prevent disease
- 11 transmission from patient to care provider, from care provider to patient, and from patient to patient.
- 12

13 Methods

- 14 This policy was originally developed by the Infectious Disease Control Subcommittee and adopted in
- 15 1989. This document is a revision of the previous version, revised in 2014. The revision of this policy
- 16 is based upon a review of current dental and medical literature related to infection control, expert
- 17 <u>opinion, and best current practices.</u> an update from the last revision in 2009. Literature searches of
- 18 PubMed[®] and Google Scholar databases were An electronic database search was conducted using the
- 19 terms: dentistry infection control AND health care, and infection control AND dental; fields: all;
- 20 limits: within the last 10 years; English; humans; comparative study, meta-analysis, multi-center
- study, systematic reviews, and validation study. The search returned 352 34 articles that matched the
- 22 criteria. The articles were evaluated by title and/or abstract and relevance to dental care for children
- and adolescents. <u>Twenty-five</u> Three <u>articles</u> eitations were chosen from this method and from
- 24 references within selected articles.
- 25

26 Background

- 27 The application of standard universal precautions regarding infection control during dental treatment
- 28 is paramount. The environment in which dental care is delivered impacts both patient and provider
- 29 safety. Health care professionals should remain knowledgeable in how to reduce exposure and
- 30 contamination risks to infectious materials. This would include body substances, contaminated

^{*} ABBREVIATION

AAPD: American Academy Pediatric Dentistry.

- 31 supplies, equipment, environmental surfaces, water, and air. Some infection control practices
- 32 routinely used by healthcare professionals cannot be rigorously evaluated by clinical trials for ethical
- and logistical reasons. $\frac{1}{(Kohn et al 2003)}$
- 34
- 35 Many resources are available to aid dental providers in creating checklists, standard operating
- 36 procedures or other quality assurance mechanisms for use in daily practice. All patient care,
- 37 <u>laboratory procedures and equipment management should be carried out in an environment with</u>
- 38 <u>techniques consistent with guidelines set forth by Center for Disease Control and Prevention (CDC)</u>,
- 39 the Occupational Safety and Health Administration (OSHA), as well as state and local regulatory
- 40 boards. Providers should consult such organizations as the CDC, the OSHA, state dental boards and
- 41 <u>other dental organizations for current infection control recommendations.²⁻⁹</u>
- 42
- 43 Dental practices should stay abreast of updated infection control requirements issued by official
- 44 regulatory organizations. Standard precautions includes hand washing, personal protective equipment
- 45 (PPE) such as gloves, gowns, eyewear with safety side shields, and masks whenever touching or
- 46 <u>exposure to patients' body fluids is likely.</u>
- 47
- 48 The possibility of contamination within the internal components of dental handpieces has led to the
- 49 more recent recommendation that all dental handpieces, including low-speed motors and removable
- 50 prophylaxis angles, undergo heat sterilization between patients. Providers should verify that the
- 51 instructions for sterilization provided by the manufacturer of their reusable equipment comply with
- 52 <u>current standards.²⁻⁴</u>
- 53
- 54 Dental practices should be cognizant of potential infections associated with waterlines. It is important
- 55 to follow manufacturer guidelines to disinfect waterlines, monitor water quality, use point-of-use
- 56 water filters, and eliminate dead ends in plumbing where stagnant water can enable biofilm
- 57 formation.^{4,5,10} There have been cases of disease transmission from waterlines.^{10,11} In 2015, an
- 58 <u>outbreak of *Mycobacterium abscessus* odontogenic infections was reported in children receiving</u>
- 59 pulpotomy treatment from a pediatric dental clinic. The source of the *Mycobacterium* was
- 60 contaminated water from dental unit waterlines.^{5,11} A practitioner may consider use of sterile water or
- 61 <u>saline when irrigating pulpal tissue.¹¹</u>
- 62
- 63 <u>Although no adverse health effects have been reported with use of saliva ejectors, CDC cautions the</u>

64	<u>dental l</u>	nealth care providers to be aware of the possibility of a potential backflow, where suctioned				
65	fluids in tubing can flow back into patient's mouth. This can happen when: 1) Pressure in patient's					
66	mouth, as a result of closing their lips and forming a seal around the tip of the ejector, is lower than					
67	the pres	ssure in saliva ejector; 2) Suction tubing attached to the ejector is positioned above patient's				
68	mouth;	or 3) Saliva ejector is used at same time with other high-volume suctions. Clinicians are				
69	recomn	nended to take all necessary precautions to prevent potential backflow associated with use of				
70	<u>saliva e</u>	pjectors. ²				
71						
72	Policy	statement				
73	The AA	APD <u>:</u> acknowledges				
74	<u>•</u>	Acknowledges the Centers for Disease Control and Prevention's Statement on Reprocessing				
75		Dental Handpieces- 2018 ³ , Guidelines for Infection Control in the Dental Health-Care				
76		Setting-2003 ¹ , Guidelines for Disinfection and Sterilization in Healthcare Facilities-2008 ⁷				
77		and Updated CDC Recommendations for the Management of Hepatitis B Virus-infected				
78		Health Care Providers and Students—2012 ⁶ as in-depth reviews of infection control				
79		measures for dental settings and supports the strategies therein.				
80	<u>•</u>	Aware that some Some recommendations are based only on suggestive evidence or				
81		theoretical rationale, and because many concerns regarding infection control in the dental-				
82		setting remain unresolved, the AAPD encourages dental practitioners to follow current-				
83		literature and consider carefully infection control measures in their practices so as to-				
84		minimize the risk of disease transmission.				
85	<u>•</u>	Encourages providers to follow CDC recommendations to heat sterilize all dental handpieces,				
86		including low-speed motors and removable prophylaxis angles, between patients. ³				
87	<u>•</u>	Encourages providers and their dental teams to be proactive in addressing infection control				
88		concerns. Staff may benefit from additional training to better answer questions from parents				
89		regarding the infection control practices at their practice.				
90						
91						
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11

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133	

¹ Policy on the Use of Dental Bleaching for Child and Adolescent

2 Patients*

- 3
- 4 Review Council
- 5 Council on Clinical Affairs
- 6 Latest Revision
- 7 2014 2019
- 8

9 Purpose

10 The American Academy of Pediatric Dentistry (AAPD) recognizes that the desire for dental whitening in

11 pediatric and adolescent patients has increased. This policy is intended to help professionals and patients

12 make informed decisions about the indications, efficacy, and safety of internal and external bleaching of

13 primary and young permanent teeth and incorporate such care into a comprehensive treatment plan.

14

15 Methods

16 This policy was originally developed by the Council on Clinical Affairs and adopted in 2004. This

17 document is an update from the last revision in 2009.2014. This revision included a new literature search

18 of the PubMed®/MEDLINE database using the terms: dental bleaching, dental whitening, and tooth

19 bleaching; fields: all; limits: within the last 10 years, humans, English, and birth through age 18. 260-

20 articles matched these criteria. Papers for review were chosen from this list and from the references-

21 within selected articles. Articles were selected and reviewed. Additional information was obtained from

22 reviewing references within selected articles. When data did not appear sufficient or were inconclusive,

recommendations were based upon expert and/or consensus opinion by experienced researchers and

- 24 clinicians.
- 25

26 Background

- 27 Through news stories and advertisements, the public has become more aware of advances in cosmetic-
- 28 dentistry. The desire for improved dental esthetics has fueled innovations in dental materials and the
- 29 <u>development of new resources</u>. Increased demand for bleaching materials and services has affected both-
- 30 the variety and availability of dental bleaching products on the market. Both the variety and availability

* ABBREVIATION

AAPD: American Academy of Pediatric Dentistry

- 31 of bleaching products on the market. have increased. Consequently Patients, parents and the news media
- 32 <u>continue to request information on dental whitening for children and adolescents with increasing</u>
- 33 frequency. <u>In addition, increased demand for bleaching materials and services has affected both the</u>
- 34 <u>variety and availability of dental bleaching products on the market.</u>
- 35
- 36 <u>Discoloration of teeth is classified by etiology.</u>¹ Clinical indications for internal or external dental
- 37 whitening for individual teeth may include discoloration resulting from a traumatic injury (i.e., calcific
- 38 metamorphosis, darkening with devitalization), irregularities in enamel coloration of a permanent tooth
- 39 due to trauma or infection of the related primary tooth, or intrinsic discoloration/staining (e.g., enamel
- 40 hypoplasia, fluorosis, tetracycline staining).²⁻⁷ <u>Teeth staining from metals (e.g., iron supplements) or</u>
- 41 <u>consumption of tea, coffee, soft drinks, alcohol and certain foods is extrinsic and easier to treat compared</u>
- 42 to intrinsic factors which are congenital or acquired. Severe discolorations may be best treated with
- 43 <u>microabrasion and subsequent bleaching to achieve desirable results.⁸ A positive self-image due to a</u>
- 44 discolored tooth or teeth can have serious consequences on adolescents and could be considered an-
- 45 indication for bleaching. (Donly 2003) Due to the difference in the thickness of enamel of primary and
- 46 permanent teeth, tooth coloration within a dental arch may vary significantly during the mixed dentition.
- 47 Full arch cosmetic bleaching during this developmental stage, however, would result in mismatched
- 48 dental appearance once the permanent dentition is reached. <u>As adolescents present with unique dental</u>
- 49 <u>needs, the impact of tooth discoloration on an adolescent's self-image could be considered an indication</u>
- 50 for bleaching.⁸ Tooth whitening has been successful in adolescent patients using typical bleaching
- 51 agents,⁸ but research is lacking on the effects of bleaching on the primary dentition.
- 52
- 53 Dental whitening may be accomplished by using either professional or at-home bleaching modalities.
- 54 Advantages of in-office whitening or whitening products dispensed and monitored by a dental
- 55 professional include:
- An initial professional examination to help identify causes of discoloration and clinical concerns
 with treatment (e.g., existing restorations, side effects).
- Professional control and soft-tissue protection.
- 59 <u>Patient compliance</u>.
- 60 Rapid results.
- 61 <u>Immediate attention to teeth sensitivity and other adverse effects</u>
- 62
- 63 The pretreatment professional assessment helps identify pulp pathology that may be associated with a

64 single discolored tooth. This examination also identifies restorations that are faulty or could be affected by the bleaching process, and the associated costs for replacing such restorations to maximize esthetic 65 66 results.⁸⁻¹² By using photographs and/or a shade guide, the dentist can document the effectiveness of treatment. In addition to providing in-office bleaching procedures, a dentist may fabricate custom travs 67 68 for at-home use of a bleaching product. Custom trays ensure intimate fit and fewer adverse gingival effects.¹³ Over-the-counter products for at-home use include bleaching gels, whitening strips, brush-on 69 agents, pens, toothpastes, mints, chewing gum, and mouth rinses. Their main advantages include patient 70 71 convenience and lower associated costs. 72 73 Peroxide-containing whiteners or bleaching agents improve the appearance of a tooth by changing its 74 intrinsic color. The professional-use products usually range from 10 percent carbamide peroxide 75 (equivalent to about 3.5 percent hydrogen peroxide) to 38 percent carbamide peroxide (equivalent to approximately 13 percent hydrogen peroxide). In-office bleaching products require isolation with a rubber 76 dam or a protective gel to shield the gingival soft tissues. Home-use bleaching products contain lower 77 concentrations of hydrogen peroxide or carbamide peroxide.^{2-4,14} Efficacy and long-term outcomes of 78 home whitening products will vary according the concentration of peroxide used and the severity of the 79 initial tooth discoloration.¹⁵⁻¹⁸ Many whitening toothpastes contain polishing or chemical agents to 80 improve tooth appearance by removing extrinsic stains through gentle polishing, chemical chelation, or 81 other non-bleaching action.^{10,19} 82 83 84 Side effects from bleaching vital and non-vital teeth have been documented. It should be noted that most of the research on bleaching has been performed on adult patients, with only a limited number of 85 published research studies using child or adolescent patients.^{2,4,8,14,17,20-23} The more common side effects 86 87 associated with bleaching vital teeth are tooth sensitivity and tissue irritation. Tooth sensitivity associated with vital teeth bleaching may be due to permeation of enamel and dentin by hydrogen peroxide and a 88 subsequent mild, transient inflammatory response.²⁴⁻²⁶ Sensitivity will be affected by the concentration of 89 90 whitening agent and the amount of time that the teeth are exposed to the bleaching product²⁷ Between 8 91 and 66 percent of patients experience post-bleaching sensitivity, most often during the early stages of 92 treatment.^{7,10,14,17,20,23} Over-treatment has been shown to harm tooth structure, which is of particular concern when bleaching products are used excessively by overzealous teens and young adults.²⁸⁻³⁰ 93 94 95 In most cases, tissue irritation results from an ill-fitting tray rather than the bleaching agents and resolves once a more accurately fitted tray is used. Both sensitivity and tissue irritation are usually temporary and 96

97	cease with	discontinuatio	on of treatm	ent. ^{6,7,14,17,19,20,23, 32}	² Additional	risks may	v include	erosion.	mineral

- 98 <u>degradation</u>, pulpal damage and increased marginal leakage of existing restorations.^{14,33} When used
- 99 correctly, however, teeth bleaching has been proven to be safe and causes no irreversible tooth structure
- 100 <u>damage.²⁸</u>
- 101
- 102 Internal bleaching for non-vital endodontically treated teeth in young patients can be performed in the
- same way as for adults.²⁸ The more common side effects from internal bleaching of nonvital teeth are
- 104 external root resorption³⁴⁻³⁷ and ankylosis. With external bleaching of nonvital teeth, the most common
- side effect is increased marginal leakage of an existing restoration.^{3,38-42} One of the degradation
- 106 byproducts of hydrogen peroxide or carbamide peroxide is a hydroxyl-free radical. <u>This byproduct has</u>
- 107 <u>been associated with periodontal tissue damage and root resorption. Due to concern for hydroxyl free</u>
- 108 $radical damage^{43-45}$ and the potential side effects of dental bleaching, minimizing exposure to the lowest

109 effective concentration of hydrogen peroxide or carbamide peroxide is recommended. Providers should

110 use caution when bleaching primary anterior teeth, as the underlying permanent teeth are in jeopardy of

- 111 <u>developmental disturbance from intramedullary inflammatory changes.^{28,46}</u>
- 112
- 113 Of growing concern is the preponderance of non-dental professionals offering teeth whitening services to
- 114 the public.⁴⁷ Tooth whitening is defined as any process to whiten, lighten, or bleach teeth.⁴⁷
- 115 <u>Teeth whitening kiosks, retail and beauty salons are providing whitening services and dispensing teeth</u>
- 116 whitening agents.^{19,48} Dental organizations throughout the nation have supported state regulations that
- 117 restrict the practice of providing bleaching services to only dentists or other qualified dental staff under
- 118 the direct supervision of a dentist. The use of over-the-counter whitening products remain exempt from
- 119 this regulation. Legislation defining the scope of practice by non-dentists offering whitening treatment

120 vary from state to state and should be examined when these services are being provided.⁴⁸⁻⁵⁰

- 121 <u>For Current literature and clinical studies support the use of sodium perborate mixed with water for</u>
- 122 bleaching nonvital teeth.28,29 Studies have shown higher incidences of root resorption when hydrogen-
- 123 peroxide is mixed with sodium perborate21-24 or any mixture of sodium perborate is heated.29-
- 124 Therefore, the use of hydrogen peroxide and heating any mixture of sodium perborate are not-
- 125 recommended.
- 126
- 127 Policy statement
- 128 <u>Teeth whitening procedures have been shown to be safe and may be beneficial for children and</u>
- 129 adolescents. Their use should follow the safety and efficacy standards as defined by clinical research and

130	best	practice. Young patients undergoing dental bleaching should be supervised by an adult and under the
131	guid	ance of a dentist. Although the use of whitening agents can successfully improve teeth esthetics and
132	<u>enha</u>	nce a person's self-esteem, proper treatment planning with objectives should be conducted prior to
133	enga	ging in any bleaching protocol.
134		
135	The	AAPD encourages:
136	•	The judicious use of bleaching for vital and non-vital teeth.
137	•	Patients to consult their dentists to determine appropriate methods for and the timing of dental
138		whitening within the context of an individualized, comprehensive, and sequenced treatment plan.
139	•	Dental professionals and consumers to consider side effects when contemplating dental bleaching
140		for child and adolescent patients.
141	•	Further research of dental whitening agents in children.
142		
143	The	AAPD discourages full-arch cosmetic bleaching for patients in the mixed and primary dentition.
144		
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263

¹ Policy on Third-Party Payor Audits, Abuse, and Fraud^{*}

- 2
- 3 Originating Review Council
- 4 Council on Clinical Affairs
- 5 Adopted
- 6 2014
- 7 Latest Revision
- 8 <u>2019</u>
- 9

10 Purpose

11 One of the aims of the Deficit Reduction Act,¹ approved by the U.S. Congress in 2005, was to prevent

12 Medicaid fraud and abuse through an audit process. Despite the good intentions of this law, experts

- 13 predicted health care providers will would see more investigations, enforcement actions, and
- 14 whistleblower cases, and will would need to devote more resources toward compliance.² Pediatric dentists
- 15 play a critical role in the Medicaid program, and there will be negative impact on access to care if honest
- 16 providers are burdened with excessive regulations and unfounded audits. The American Academy of
- 17 Pediatric Dentistry (AAPD) supports efforts to eliminate Medicaid abuse. The AAPD cautions, however,
- 18 against ill-informed or misguided investigations that may discourage dental provider participation in the
- 19 program.² The AAPD is opposed to any of its dentist members committing abuse and fraud as it relates to
- 20 their relationship with third party payors. Such behavior is unprofessional conduct and could result in loss
- of membership status in AAPD.³ This policy is intended to help AAPD members understand the audit
- 22 process, both internal and external audits.

23

24 Methods

- 25 This policy is based upon a review of current dental and medical literature, including a literature search of
- the PubMed® electronic database using the terms: dental audits, dental abuse and fraud, peer review,
- 27 provider profiling, practice management, EPSDT; field: all; limits: within the last 10 years; human;
- 28 English. Nineteen articles match these criteria. Papers for review were chosen from this list as well as
- 29 references within the selected articles. When data did not appear sufficient or were inconclusive,

* ABBREVIATIONS

AAPD: American Academy Pediatric Dentistry. **CMS**: Centers for Medicare and Medicaid Services. **EPSDT**: Early and periodic screening, diagnosis, and treatment. **MNC**: Medically Necessary care. **RAC**: Recovery audit contractor.

- 30 recommendations were based upon expert and/or consensus opinion by experienced researchers and-
- 31 clinicians.
- 32

33 Background

34 External audits are increasingly common for a full range of health care providers. AAPD members Dentists are no exception, as some of our members pediatric dentists have experienced. If a provider 35 36 requests payment from third-party payors, the claims may be subject to review by a recovery audit contractor (RAC), a private entity that reviews paid claims and, in some cases, earns contingency fees for 37 38 improper payments it retrieves. Private and public third-party payors use audits as a mechanism to recoup 39 over-payments, inspect for potential improper behavior, and possibly guide health care providers to 40 control utilization and costs.⁴ Notably, there can be serious financial and even criminal penalties 41 associated with billing errors.⁵ 42 In 2012-2017, an estimated \$19 37 billion, or seven ten percent, of the federal Medicaid funds were 43 44 absorbed by improper payments, which include fraud and abuse as well as unintentional mistakes such as 45 paper errors.⁶ Improper payments totaled an estimated \$11 billion, or nine percent of states' Medicaid 46 budgets in 2010, the most recent year for which data is available.(CMS Medicaid Improper Payment) Improper payments can occur when funds go to the wrong recipient, the recipient receives the incorrect 47 amount of funds (either an underpayment or overpayment), documentation is not available to support a 48 49 payment, or the recipient uses the funds in an improper manner.⁶ 50 51 The AAPD recognizes the concern its members have regarding these external audits. The AAPD 52 encourages its members to develop internal self-audit programs to address these challenges. Internal 53 audits are used in order to preemptively detect discrepancies before the external authorities can discover 54 them and impose penalties.⁴ Given the heightened concern for compliance to avoid an external audit, 55 internal audits have taken on importance. A compliance program generally will incorporate a credible 56 internal audit system, which means that it must be prepared to respond to an external audit by various 57 authorities. In addition, some pediatric dentists have discovered that an internal audit system can be 58 developed so that it not only addresses the external audit, but also serves other quality of care and performance improvement purposes.⁴ 59

60

61 **Definitions**

62 *Abuse:* "provider practices that are inconsistent with sound fiscal, business, or medical practices, and

63	result in an unnecessary cost to the Medicaid program, or reimbursement for services that are not
64	medically necessary or that fail to meet the professionally recognized standards for healthcare. It also
65	includes recipient practices that result in unnecessary cost to the Medicaid program." ⁷ The AAPD
66	supports medically necessary care (MNC) and recognizes that dental care is medically necessary for the
67	purpose of preventing and eliminating orofacial disease, infection, and pain, restoring the form and
68	function of the dentition, and correcting facial disfiguration or dysfunction. ⁸
69	
70	Audit: "planned and documented activity performed by qualified personnel to determine by investigation,
71	examination, or evaluation of objective evidence, the adequacy and compliance with established
72	procedures, or applicable documents, and the effectiveness of implementation".9 After receiving a notice
73	of an impending audit from a third-party payor, the dentist should ascertain the type and scope of audit to
74	be conducted <u>in writing</u> . ^{10,11}
75	
76	Fraud: "an intentional deception or misrepresentation made by a person with the knowledge that the
77	deception could result in some unauthorized benefit to him or some other person."7
78	
79	Third party payor: "an organization other than the patient (which would be the first party) or health care-
80	provider (also known as the second party) involved in the financing of health care services."(Oberman-
81	2010)-"An organization other than the patient (first party) or health care provider (second party) involved
82	in financing of personal health services." ¹²
83	
84	Recovery audit contractor: RAC's review claims on a post-payment basis. The RACs detect and correct
85	past improper payments so that CMS and Carriers, FIs, and MACs can implement actions that will
86	prevent future improper payments. ¹³
87	
88	
89	Credentials of auditors. The Affordable Care Act required that each state Medicaid program use at least
90	one RAC beginning in 2011.14 Some states have started employing the RACs to aid in recovery of
91	improper payments. ¹⁵ The AAPD strongly believes that, while audits are a part of third-party payment
92	contracts and are necessary to protect the integrity of these programs, such audits must be completed by
93	those who have credentials on par with the dental provider being audited. For example, pediatric dentists
94	must be audited by a dentist who specializes in pediatric dentistry and who understands the clinical

95 guidelines and standards of care which have been adopted and followed by their specialty. The AAPD is

- adamantly opposed to auditors receiving financial incentives for any money recuperated through these
- 97 audits. This represents a conflict of interest.
- 98

99 **Provider profiling.** The AAPD is opposed to "provider profiling," a strategy that may be used by health 100 plans to assess efficiency among providers and believes that dentist providers selected for audits should 101 be chosen randomly or with compelling evidence that makes them an outlier compared to peers practicing in similar geographic areas, on similar populations of patients, and within the same specialty. Claims-102 based data used for provider profiling are not collected exclusively for performance assessment and, as a 103 result, may be irrelevant or inadequate for profiling.¹⁶ Claims data may be unable to properly and fully-104 characterize an episode of care and may fail to reveal a patient's baseline status.14 In addition, codes-105 106 contained in claims data do not articulate "patients' compliance, their desire for care, or their-107 socioeconomic status". Furthermore, the procedure codes included in claims-based data cannot fully characterize the nature of a particular episode of care, and may fail to account for variations in baseline 108 109 status, socioeconomic considerations, compliance with treatment and access to care.¹⁶ 110 111 Peer review as part of audit outcomes. The AAPD supports peer review as a way to offer information 112 and support to dentists who need to review best practices regarding chart documentation, coding, and 113 billing practices related to third party payors. This should be offered in lieu of financial penalties when an 114 audit shows that no intent to fraud was present, but that the dentists need education to improve their practice systems. It provides practicing dentists a means to preserve their reputation and good standing in 115 116 the community, and fosters risk management, accountability and self-regulation among dental professionals.¹⁷ This model would be consistent with the peer review practices that occur when clinical 117 decision making is in question. The intent of peer review is to resolve discrepancies between the dentists 118 119 and third-party payors expeditiously, fairly, and in a confidential manner.¹⁷ 120 121 Best practices for chart documenting, coding, and billing. The AAPD supports the education of pediatric dentistry residents, pediatric dentists, and their staff to ensure good understanding of appropriate 122 coding and billing practices. The AAPD, therefore, supports the creation of educational resources and 123 124 programs that promote best practices, which may include: 125 Programming-Programs offered at the AAPD's Annual Session or other AAPD-sponsored 126 continuing education course. 127 Programs offered to by the pediatric dentistry state unit and district organizations. ٠ 128 The creation of a web-based tutorial for dentists and their staff, including the states' Dental Medicaid •

- 129 <u>Provider Manuals and frequently asked questions regarding Medicaid.</u>
- Partnering with other public/private organizations and agencies to distribute 'Medicaid Updates' that
 can be received via e-mail, and building open *Medicaid Compliance for the Dental Professional*
- 132 webinars offered jointly by AAPD and Centers for Medicare and Medicaid Services (CMS)¹⁸
- The development of a third party payor submission compliance program.
- 134

135 Medicaid policies that conflict with AAPD elinical practice guidelines <u>clinical recommendations</u>.

- 136 The AAPD is opposed to Medicaid programs that have policies which are in direct conflict with AAPD
- 137 clinical practice guidelines recommendations and are of detriment to patient care. For example, iIn several
- 138 states, children are not receiving appropriate dental treatment covered by Early and Periodic Screening,
- 139 Diagnosis, and Treatment (**EPSDT**) because there is a refusal to reimburse providers for EPSDT-covered
- 140 dental services.¹⁹ It is in the best interest of the public to have EPSDT dental periodicity schedules readily
- 141 available on the Internet. Such availability would also improve compliance by health care professionals-
- 142 and EPSDT staff members with federal EPDST requirements.(Hom et al 2013) In addition, aAccording to
- 143 CMS, "federal law also requires that states inform all families about EPSDT coverage"²⁰ The and AAPD
- 144 recommends that <u>supports</u> this requirement be followed to enable caregivers to seek necessary dental
- treatment for their children.
- 146

147 Policy statement

- 148 Dental care is medically necessary to prevent and eliminate orofacial disease, infection, and pain, to 149 restore the form and function of the dentition, and to correct facial disfiguration or dysfunction. MNC is 150 based upon current preventive and therapeutic practice guidelines formulated by professional 151 organizations with recognized clinical expertise. Expected benefits of MNC outweigh potential risks of 152 treatment or no treatment. Early detection and management of oral conditions can improve a child's oral 153 health, general health and well-being, school readiness, and self-esteem. Early recognition, prevention, 154 and intervention could result in savings of health care dollars for individuals, community health care 155 programs, and third party payors. Because a child's risk for developing dental disease can change over 156 time, continual professional reevaluation and preventive maintenance are essential for good oral health. 157 Value of services is an important consideration, and all stakeholders should recognize that cost-effective care is not necessarily the least expensive treatment.⁸ 158
- 159

160 The AAPD:

• Encourages it members and all third-party payors to support efforts to eliminate Medicaid abuse.

- Opposes any of its dentist members committing abuse and fraud as it relates to their relationship
 with third-party payors.
- Recognizes the concern its members have regarding these external audits.
- Encourages its members to develop internal self-audit programs to address these challenges.
- Strongly believes that, while audits are a part of third-party payment contracts and are necessary to
 protect the integrity of these programs, such audits must be completed by those who have credentials
 on par with the dental provider being audited.
- Adamantly opposes auditors receiving financial incentives for any money recuperated through audits.
- Opposes provider profiling and believes that dentist providers selected for audits should be chosen
 randomly or with compelling evidence that makes them an outlier as compared to their peers who
 practice in similar geographic areas, on similar populations of patients, and within the same
 specialty.
- Supports peer review in lieu of financial penalties when an audit shows that no intent to fraud was
 present, as a way to offer information and support to dentists who need to re-acquaint themselves on
 best practices regarding chart documentation, coding, and billing practices relating to third-party
 payors.
- Supports the education of pediatric dentistry residents, pediatric dentists, and their staff to ensure a
 good understanding of appropriate coding and billing practices.
- Supports the creation of educational resources and programs that promote appropriate coding and
 billing practices.
- Opposes Medicaid programs that have policies in direct conflict with AAPD clinical practice
 guidelines recommendations and are of detriment to patient care.
- Endorses the enforcement of the "federal law that requires that states inform all families about
 EPSDT coverage"²⁰ to enable caregivers to seek necessary dental treatment for their children.
- 187

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- ¹ Policy on Medically-Necessary Care^{*}
- 2
- 3 Review Council
- 4 Council on Clinical Affairs
- 5 Latest Revision
- 6 2015 <u>2019</u>
- 7
- 8 Purpose

9 The American Academy of Pediatric Dentistry (AAPD) recognizes that dental care is medically-

- 10 necessary for the purpose of preventing and eliminating orofacial disease, infection, and pain, restoring
- 11 the form and function of the dentition, and correcting facial disfiguration or dysfunction.
- 12

13 Methods

- 14 This document was originally developed by the Council on Clinical Affairs and adopted in 2007. This
- 15 document is an update from the last revision in 2014<u>5</u>. It includes an electronic search with Scopus and
- 16 PubMed /MEDLINE® using the terms: medically-necessary care, systemic disease and oral disease,
- 17 dentistry as medically-necessary care, periodontal disease and cardiovascular disease, oral health and
- 18 pregnancy, oral health and respiratory illness, <u>oral health and quality of life</u>, pediatric dentistry general
- 19 anesthesia, and nutritional deficiency cognitive development; fields: all; limits: within the last 15 years,
- 20 human, English. The reviewers agreed upon the inclusion of <u>59-78</u> articles that met the defined criteria.
- 21 When data did not appear sufficient or were inconclusive, recommendations were based upon expert
- 22 and/or consensus opinion by experienced researchers and clinicians.
- 23

24 Background

- 25 The AAPD defines medically-necessary care (MNC) as "the reasonable and essential diagnostic,
- 26 preventive, and treatment services (including supplies, appliances, and devices) and follow-up care as
- 27 determined by qualified health care providers in treating any condition, disease, injury, or congenital or
- 28 developmental malformation to promote optimal health, growth, and development. MNC includes all
- 29 supportive health care services that, in the judgment of the attending dentist, are necessary for the
- 30 provision of optimal quality therapeutic and preventive oral care. These services include, but are not

^{*} ABBREVIATIONS

AAPD: American Academy of Pediatric Dentistry. CC: Chronic condition. ECC: Early childhood caries. MNC: Medically-necessary care

- 31 limited to, sedation, general anesthesia, and utilization of surgical facilities. MNC must take into account
- 32 the patient's age, developmental status, and psychosocial well-being, in addition to the clinical setting
- appropriate to meet the needs of the patient and family."¹
- 34

35 MNC is based upon current preventive and therapeutic practice guidelines formulated by professional

36 organizations with recognized clinical expertise. Such recommendations ideally are evidence based but, in

- the absence of conclusive evidence, may rely on expert opinion and clinical observations. Expected
- 38 benefits of care should outweigh potential risks. MNC increases the probability of good health and well-
- 39 being and decreases the likelihood of an unfavorable outcome. Value of services is an important
- 40 consideration, and all stakeholders should recognize that cost-effective care is not necessarily the least
- 41 expensive treatment.²
- 42

Dental care is medically-necessary to prevent and eliminate orofacial disease, infection, and pain, to
restore the form and function of the dentition, and to correct facial disfiguration or dysfunction. Following
the U.S. Surgeon General's report³ emphasizing that oral health is integral to general health, the U.S.
Department of Health and Human Services recommended changing perceptions of the public, policy
makers, and healthcare providers so that oral health becomes an accepted component of general health.^{4,5}
Oral diseases can have a direct and devastating impact on overall health, especially for those with certain
systemic health problems or conditions.

50

Caries is the most common chronic disease of childhood.^{3,6} Approximately 60 percent of children 51 52 experience caries in their primary teeth by age five.⁷ Between 1988-1994 and 1999-2004, prevalence of 53 caries in primary teeth increased for youths aged two to 11 years, with a significant increase noted for 54 those in the two to five year age range.⁶ By 17 years of age, 78 percent of children in the U.S. have experienced caries.⁵ As much as 90 percent of all caries in school-aged children occurs in pits and 55 fissures. Caries, periodontal diseases, and other oral conditions, if left untreated, can lead to pain, 56 infection, and loss of function. These undesirable outcomes can adversely affect learning, communication, 57 nutrition, and other activities necessary for normal growth and development.⁸ Rampant caries is 58 59 associated with insufficient development in children who have no other medical problems.⁹(Acs et al-1992) Children with early childhood caries (ECC) may be severely underweight because of the associated 60 61 pain and disinclination to eat. Nutritional deficiencies during childhood can impact cognitive development.10,11 62

63

Other oral conditions also can impact general health and well-being. Gingivitis is nearly universal in 64 children and adolescents, and children can develop severe forms of periodontitis.¹²(AAPerio 2003) There 65 66 exists may exist a relationship between periodontal disease and cardiovascular disease¹³⁻¹⁵ as well as periodontal disease and adverse pregnancy outcomes.^{16,17} including pregnancy hypertension.¹⁸ An 67 association between oral health and respiratory diseases has been recognized.^{18,19} Oral health, oral 68 69 microflora, and bacterial pneumonia, especially in populations at high risk for respiratory disease, have 70 been linked. The mouth can harbor respiratory pathogens that may be aspirated, resulting in airway 71 infections.²⁰) Furthermore, dental plaque may serve as a reservoir for respiratory pathogens in patients who are undergoing mechanical ventilation.²¹. Problems of esthetics, form, and function can affect the 72 73 developing psyche of children, with life-long consequences in social, educational, and occupational environments.^{22,23} Self-image, self-esteem, and self-confidence are unavoidable issues in society, and an 74 acceptable orofacial presentation is a necessary component of these psychological concepts.^{24,25} 75 76 77 Congenital or acquired orofacial anomalies (e.g., ectodermal dysplasia, cleft defects, cysts, tumors) and 78 malformed or missing teeth can have significant negative functional, esthetic, and psychological effects

79 on individuals and their families.^{26,27} Patients with craniofacial anomalies often require specialized oral

80 health care as a direct result of their craniofacial condition. These services are an integral part of the

81 rehabilitative process.²⁶ Young children benefit from esthetic and functional restorative or surgical

techniques and readily adapt to appliances that replace missing teeth and improve function, appearance,

83 and self-image. During the period of facial and oral growth, appliances require frequent adjustment and

84 have to be remade as the individual grows.

85

Professional care is necessary to maintain oral health,^{3,4} and risk assessment is an integral element of
contemporary preventive care for infants, children, adolescents, and persons with special health care
needs.²⁸ The goal of caries risk assessment is to prevent disease by identifying and minimizing causative
factors (e.g., microbial burden, dietary habits, dental morphology) and optimizing protective factors (e.g.,
fluoride exposure, personal oral hygiene, sealants).^{29,30} Ideally, risk assessment and implementation of
preventive strategies would occur before the disease process has been initiated.

92

Infants and young children have unique caries-risk factors such as ongoing establishment of oral flora and
 host defense systems, susceptibility of newly erupted teeth, and development of dietary habits and

95 childhood food preferences. Children are most likely to develop caries if mutans streptococci is acquired

at an early age.³¹⁻³³ High-risk dietary practices are multi-factorial³⁴ and food preferences appear to be

CCA.f. P_MedicallyNecessaryCare

- 97 established early, probably by 12 months of age, and are maintained throughout early childhood.^{35,36}
- 98 Adolescence can be a time of heightened caries activity and periodontal disease due to an increased intake
- 99 of cariogenic substances and inattention to oral hygiene procedures.³⁷⁻³⁹
- 100

An analysis of caries risk includes determination of protective factors, such as fluoride exposure. More 101 102 than one-third of the U.S. population does not benefit from community water fluoridation.³ Fluoride 103 contributes to the prevention, inhibition, and reversal of caries.⁴⁰ Therefore, early determination of a 104 child's systemic and topical fluoride exposure is important. Children experiencing caries as infants and 105 toddlers have a much greater probability of subsequent caries in both the primary and permanent dentitions.¹⁰ An individualized preventive plan based on a caries risk assessment is the key component of 106 107 caries prevention. Because any risk assessment tool may fail to identify all infants at risk for developing ECC, early establishment of the dental home is the ideal approach for disease prevention.⁴¹ Early 108 109 diagnosis and timely intervention, including necessary referrals, can prevent the need for more extensive and expensive care often required when problems have gone unrecognized and/or untreated.42-44 110

111

112 When very young children have not been the beneficiaries of adequate preventive care and subsequently

develop ECC, therapeutic intervention should be provided by a practitioner with the training, experience,

and expertise to manage both the child and the disease process. Because of the aggressive nature of ECC,

restorative treatment should be definitive yet specific for each individual patient. Conventional restorative

approaches may not arrest the disease.⁴⁵ Areas of demineralization and hypoplasia can cavitate rapidly.

117 The placement of stainless steel crowns may be necessary to decrease the number of tooth surfaces at risk

118 for new or secondary caries. Stainless steel crowns are less likely than other restorations to require

retreatment.^{45,46} Low levels of compliance with follow-up care and a high recidivism rate of children

requiring additional treatment also can influence a practitioner's decisions for management of ECC⁴⁷ and

121 may decrease success of a disease management approach to ECC.⁴⁸

122

Sealants are particularly effective in preventing pit and fissure caries and providing cost savings if placed on the teeth of patients during periods of greatest risk.⁴⁹ Children with multiple risk factors and tooth morphology predisposed to plaque retention (i.e., developmental defects, pits and fissures) benefit from having such teeth sealed prophylactically. A child who receives sealants is 72 percent less likely to receive restorative services over the next three years than children who do not.⁵⁰ Sealants placement on primary molars in young children is a cost-effective strategy for children at risk for caries, including those

insured by state Medicaid programs.^{51,52} Although sealant retention rates initially are high, sealant loss

- does occur.⁵³ It is in the patient's interest to receive periodic evaluation of sealants. With follow-up care,
 the success rate of sealants may be 80 to 90 percent, even after a decade.⁵³
- 132

142

Sealants are safe and effective, yet their use continues to be low.⁵³⁻⁵⁵ Initial insurance coverage for 133 sealants often is denied, and insurance coverage for repair and/or replacement may be limited.^{55,56} While 134 135 all Medicaid programs reimburse dentists for placement of sealants on permanent teeth, only one in three reimburses for primary molar sealants.⁵⁷ While some third party carriers restrict reimbursement for 136 137 sealants to patients of certain ages, it is important to consider that timing of dental eruption can vary 138 widely. Furthermore, caries risk may increase at any time during a patient's life due to changes in habits (e.g., dietary, home care), oral microflora, or physical condition, and previously unsealed teeth 139 subsequently might benefit from sealant application.^{53,58} 140 141

affect the practitioner's behavior guidance approaches. The success of restorations may be influenced by 143 the child's response to the chosen behavior guidance technique. To perform treatment safely, effectively, 144 145 and efficiently, the practitioner caring for a pediatric patient may employ advanced behavior guidance techniques such as protective stabilization and/or sedation or general anesthesia.^{59,60} The patient's age, 146 dental needs, disabilities, medical conditions, and/or acute situational anxiety may preclude the patient's 147 148 being treated safely in a traditional outpatient setting.^{61,62} For some infants, children, adolescents, and persons with special health care needs, treatment under sedation or general anesthesia in a hospital, 149 150 outpatient facility, or dental office or clinic represents the only appropriate method to deliver necessary

The extent of the disease process, as well as the patient's developmental level and comprehension skills,

151 oral health care.^{59,63} Failure by insurance companies to cover general anesthesia costs, hospital fees,

152 <u>and/or sedation costs can expose the patient to multiple ineffective, potentially unsafe and/or</u>

153 <u>psychologically traumatic in-office experiences.</u> The impact of chronic conditions (CC) status and CC

severity increases the odds of receiving dental treatment under general anesthesia.⁶⁴ Although general

anesthesia may provide optimal conditions to perform restorative procedures, it can add significantly to

the cost of care.⁶⁵ General anesthesia may be required in the hospital setting due to the extent of

157 treatment, the need to deliver timely care, or the patient's medical history/CC (such as cardiac defects,

158 severe bleeding disorders, limited opening due to orofacial anomalies). General anesthesia, under certain

159 circumstances, may offer a cost-saving alternative to sedation for children with ECC.^{66,67}

160

161 Reimbursement issues defined by the concept of MNC have been a complicated topic for dentistry.

162 Pediatric dental patients may be denied access to oral health care when insurance companies refuse to

CCA.f. P_MedicallyNecessaryCare

provide reimbursement for sedation/general anesthesia and related facility services. Most denials cite the 163 procedure as "not medically-necessary".⁶⁸ This determination appears to be based on arbitrary and 164 inconsistent criteria.⁶⁹⁻⁷⁴ For instance, medical policies often provide reimbursement for sedation/general 165 anesthesia or facility fees related to myringotomy for a three-year-old child, but deny these benefits when 166 167 related to treatment of dental disease and/ or dental infection for the same patient. American Dental 168 Association Resolution 1989-546 states that insurance companies should not deny benefits that would otherwise be payable "solely on the basis of the professional degree and licensure of the dentist or 169 170 physician providing treatment, if that treatment is provided by a legally qualified dentist or physician operating within the scope of his or her training and licensure."74 171 172 173 Patients with craniofacial anomalies often are denied third party coverage for initial appliance 174 construction and, more frequently, replacement of appliances as the child grows. The distinction between 175 congenital anomalies involving the orofacial complex and those involving other parts of the body is often arbitrary and unfair. Often medical insurance companies interpret dental appliance construction to be 176 solely esthetic, without taking into consideration the restorative function. For instance, health care 177

178 policies may provide reimbursement for the prosthesis required for a congenitally missing extremity and

- 179 its replacement as the individual grows, but deny benefits for the initial prosthesis and necessary periodic
- 180 replacement for congenitally missing teeth. Third-party payers frequently will refuse to pay for oral health
- 181 care services even when they clearly are associated with the complete rehabilitation of the craniofacial
- **182** condition.^{75,76}
- 183

184 Policy statement

185 Dental care is medically-necessary to prevent and eliminate orofacial disease, infection, and pain, to 186 restore the form and function of the dentition, and to correct facial disfiguration or dysfunction. MNC is 187 based upon current preventive and therapeutic practice guidelines formulated by professional organizations with recognized clinical expertise. Expected benefits of MNC outweigh potential risks of 188 189 treatment or no treatment. Early detection and management of oral conditions can improve a child's oral health, general health and well-being, school readiness, and self-esteem. Early recognition, prevention, 190 191 and intervention could result in savings of health care dollars for individuals, community health care 192 programs, and third party payors. Because a child's risk for developing dental disease can change over 193 time, continual professional reevaluation and preventive maintenance are essential for good oral health. 194 Value of services is an important consideration, and all stakeholders should recognize that cost-effective 195 care is not necessarily the least expensive treatment.

CCA.f. P_MedicallyNecessaryCare

196		
197	The A	APD encourages:
198	1.	Oral health care to be included in the design and provision of individual and community-based
199		health care programs to achieve comprehensive health care.
200	2.	Establishment of a dental home for all children by 12 months of age in order to institute an
201		individualized preventive oral health program based upon each patient's unique caries risk
202		assessment.
203	3.	Healthcare providers who diagnose oral disease to either provide therapy or refer the patient to a
204		primary care dentist or dental/medical specialist as dictated by the nature and complexity of the
205		condition. Immediate intervention is necessary to prevent further dental destruction, as well as
206		more widespread health problems.
207	4.	Evaluation and care provided for an infant, child, or adolescent by a cleft lip/palate, orofacial, or
208		craniofacial deformities team as the optimal way to coordinate and deliver such complex services.
209	5.	The dentist providing oral health care for a patient to determine the medical indication and
210		justification for treatment. The dental care provider must assess the patient's developmental level
211		and comprehension skills, as well as the extent of the disease process, to determine the need for
212		advanced behavior guidance techniques such as sedation or general anesthesia.
213		
214	Furthe	more the AADD encourages third party payors to:
215		rmore, the AAPD encourages third party payors to:
	1.	Recognize malformed and missing teeth are resultant anomalies of facial development seen in
216	1.	
216 217	1.	Recognize malformed and missing teeth are resultant anomalies of facial development seen in
	1. 2.	Recognize malformed and missing teeth are resultant anomalies of facial development seen in orofacial anomalies and may be from congenital defects. Just as the congenital absence of other
217		Recognize malformed and missing teeth are resultant anomalies of facial development seen in orofacial anomalies and may be from congenital defects. Just as the congenital absence of other body parts requires care over the lifetime of the patient, so will these.
217 218		Recognize malformed and missing teeth are resultant anomalies of facial development seen in orofacial anomalies and may be from congenital defects. Just as the congenital absence of other body parts requires care over the lifetime of the patient, so will these. Include oral health care services related to these facial and dental anomalies as benefits of health
217 218 219		Recognize malformed and missing teeth are resultant anomalies of facial development seen in orofacial anomalies and may be from congenital defects. Just as the congenital absence of other body parts requires care over the lifetime of the patient, so will these. Include oral health care services related to these facial and dental anomalies as benefits of health insurance without discrimination between the medical and dental nature of the congenital defect.
217 218 219 220	2.	Recognize malformed and missing teeth are resultant anomalies of facial development seen in orofacial anomalies and may be from congenital defects. Just as the congenital absence of other body parts requires care over the lifetime of the patient, so will these. Include oral health care services related to these facial and dental anomalies as benefits of health insurance without discrimination between the medical and dental nature of the congenital defect. These services, optimally provided by the craniofacial team, include, but are not limited to, initial
217 218 219 220 221	2.	Recognize malformed and missing teeth are resultant anomalies of facial development seen in orofacial anomalies and may be from congenital defects. Just as the congenital absence of other body parts requires care over the lifetime of the patient, so will these. Include oral health care services related to these facial and dental anomalies as benefits of health insurance without discrimination between the medical and dental nature of the congenital defect. These services, optimally provided by the craniofacial team, include, but are not limited to, initial appliance construction, periodic examinations, and replacement of appliances.
217 218 219 220 221 222	2.	Recognize malformed and missing teeth are resultant anomalies of facial development seen in orofacial anomalies and may be from congenital defects. Just as the congenital absence of other body parts requires care over the lifetime of the patient, so will these. Include oral health care services related to these facial and dental anomalies as benefits of health insurance without discrimination between the medical and dental nature of the congenital defect. These services, optimally provided by the craniofacial team, include, but are not limited to, initial appliance construction, periodic examinations, and replacement of appliances. End arbitrary and unfair refusal of compensation for oral health care services related to orofacial
217 218 219 220 221 222 223	2. 3.	Recognize malformed and missing teeth are resultant anomalies of facial development seen in orofacial anomalies and may be from congenital defects. Just as the congenital absence of other body parts requires care over the lifetime of the patient, so will these. Include oral health care services related to these facial and dental anomalies as benefits of health insurance without discrimination between the medical and dental nature of the congenital defect. These services, optimally provided by the craniofacial team, include, but are not limited to, initial appliance construction, periodic examinations, and replacement of appliances. End arbitrary and unfair refusal of compensation for oral health care services related to orofacial and dental anomalies.
217 218 219 220 221 222 223 223	2. 3.	Recognize malformed and missing teeth are resultant anomalies of facial development seen in orofacial anomalies and may be from congenital defects. Just as the congenital absence of other body parts requires care over the lifetime of the patient, so will these. Include oral health care services related to these facial and dental anomalies as benefits of health insurance without discrimination between the medical and dental nature of the congenital defect. These services, optimally provided by the craniofacial team, include, but are not limited to, initial appliance construction, periodic examinations, and replacement of appliances. End arbitrary and unfair refusal of compensation for oral health care services related to orofacial and dental anomalies. Recognize the oral health benefits of dental sealants and not base coverage for sealants on
217 218 219 220 221 222 223 224 225	2. 3. 4.	Recognize malformed and missing teeth are resultant anomalies of facial development seen in orofacial anomalies and may be from congenital defects. Just as the congenital absence of other body parts requires care over the lifetime of the patient, so will these. Include oral health care services related to these facial and dental anomalies as benefits of health insurance without discrimination between the medical and dental nature of the congenital defect. These services, optimally provided by the craniofacial team, include, but are not limited to, initial appliance construction, periodic examinations, and replacement of appliances. End arbitrary and unfair refusal of compensation for oral health care services related to orofacial and dental anomalies. Recognize the oral health benefits of dental sealants and not base coverage for sealants on permanent and primary teeth on a patient's age.

- 229 6. Regularly consult the AAPD with respect to the development of benefit plans that best serve the 230 oral health interests of infants, children, adolescents, and persons with special health care needs, 231 especially those with craniofacial or acquired orofacial anomalies. 232 233 References 234 1. American Academy of Pediatric Dentistry. Definition of medically-necessary care. Pediatr Dent 235 20185;340(6)7(special issue):1515. American Academy of Pediatrics. Policy statement: Model Essential contractual language for 236 2. 237 medical necessity for children. Pediatrics 201305;13216(21):261-2398-401. 238 3. U.S. Dept of Health and Human Services. Oral Health in America: A Report of the Surgeon 239 General. Rockville, Md.: U.S. Dept of Health and Human Services, National Institute of Dental and 240 Craniofacial Research, National Institutes of Health; 2000. Available at: 241 https://www.nidcr.nih.gov/sites/default/files/2017-10/hck1ocv.%40www.surgeon.fullrpt.pdf. 242 Accessed 09/11/2018. Institute of Medicine, National Research Council. Improving Access to Oral Health Care for 243 4. 244 Vulnerable and Underserved Populations. Washington, D.C.: The National Academies Press; 2011. Available at: "http://books.nap.edu/openbook.php?record id=13116". Accessed June 12, 2015. 245 https://www.nap.edu/read/13116/chapter/1. Accessed September 19, 2018. 246 247 5. US Dept of Health and Human Services. National Call to Action to Promote Oral Health. Rockville, Md.: U.S. Dept of Health and Human Services, Public Health Service, National Institute 248 of Health, National Institute of Dental and Craniofacial Research; NIH Publication No. 03-5303, 249 250 May, 2003. Available at: https://www.ncbi.nlm.nih.gov/books/NBK47472/. Accessed 09/11/2018. 251 Dye BA, Tan S, Smith V, et al. Trends in oral health status: United States, 1988-1994 and 1999-6. 252 2004. National Center for Health Statistics. Vital Health Stat 11 (248). Hyattsville, Md.; 2007. Available at: https://www.cdc.gov/nchs/data/series/sr_11/sr11_248.pdf. Accessed on 09/11/2018. 253 254 https://www.ncbi.nlm.nih.gov/pubmed/17633507, Accessed on 12/10/18. 7. Crall JJ. Development and integration of oral health services for preschool-age children. Pediatr 255 256 Dent 2005; 27(4):323-30. 257 8. American Academy of Pediatric Dentistry. Definition of dental neglect. Pediatr Dent 258 20185;4037(6)(special issue):1313. 259 Khanh LN, Ivey SL, Sokal-gutierrez K, et al., Early childhood caries, mouth pain, and nutritional 9. 260 threats in Vietnam. Amer J Pub Health 2015; 105(12): 2510-17.
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444					

- 1 Policy on Workforce Issues and Delivery of Oral Health Care Services in
- 2 a Dental Home
- 3
- 4 Originating Council
- 5 Council on Clinical Affairs
- 6 Review Council
- 7 Council on Clinical Affairs
- 8 Adopted
- 9 2011
- 10 Revised
- 11 2014, <u>2019</u>
- 12
- 13 Purpose
- 14 The American Academy of Pediatric Dentistry (AAPD) advocates optimal oral health and health care
- 15 services for all children, including those with special health care needs. Strategies for improving
- 16 access to dental care, the most prevalent unmet health care need for disadvantaged U.S. children, and
- 17 increasing utilization of available services should include, but not be limited to, workforce
- 18 considerations. This policy will address workforce issues with an emphasis on the benefits of oral
- 19 health care services delivered within a dentist-directed dental home.
- 20

21 Methods

- 22 In 2008, the AAPD created a Task Force on Workforce Issues (TFWI) which was charged, in part,
- 23 with investigating the problem of access to oral health care services by children in the U.S. and
- 24 analyzing the different auxiliary delivery systems available. The TFWI's findings and
- recommendations were summarized in a report¹ presented to the AAPD Board of Trustees in 2009.
- 26 That report serveds as the basis for the original version of this policy.- This document was originally
- 27 developed by the Council on Clinical Affairs and adopted in 2011. This document is an update of the
- 28 previous version, revised in 2014.
- 29

30 Background

- 31 Access to oral health care for children is an important concern that has received considerable attention
- 32 since publication of Oral Health in America: A Report of the Surgeon General in 2000.² The report

This draft does not constitute an official AAPD health oral policy or clinical recommendation until approval by the General Assembly. Circulation is limited to AAPD members.

identified "profound and consequential disparities in the oral health of our citizens" and that dental 33 34 disease "restricts activities in school, work, and home, and often significantly diminishes the quality 35 of life." It concluded that for certain large groups of disadvantaged children there is a "silent 36 epidemic" of dental disease. This report identified dental caries as the most common chronic disease 37 of children in the U.S., noting that 80 percent of tooth decay is found in 20 to 25 percent of children, large portions of whom live in poverty or low-income households and lack access to an on-going 38 39 source of quality dental care. The latest #Research on the topic has shown that the distribution of these disparities may vary by age group.³ 40

41

42 The mission of the AAPD, the membership organization representing the specialty of pediatric-

43 dentistry, is "to advocate policies, guidelines, and programs that promote optimal oral health and oral-

44 health care for infants and children through adolescence, including those with special health care-

45 needs."(AAPD Mission Statement) is "to advance optimal oral health for all children by delivering

46 <u>outstanding service that meets and exceeds the needs and expectations of our members, partners, and</u>

47 <u>stakeholders."</u>⁴ AAPD has long focused its efforts on addressing the disparities between children who

48 are at risk of having high rates of dental caries and the millions of U.S. children who enjoy access to

49 quality oral health care and unprecedented levels of oral health. AAPD's advocacy activities take

50 place within the broader health care community and with the public at local, regional, and national

51 levels. Access to care issues extend beyond a shortage or maldistribution of dentists or, more

52 specifically, dentists who treat Medicaid or State Children's Health Insurance Program (CHIP)

recipients. Health care professionals often elect to not participate as providers in these programs due

to low reimbursement rates, administrative burdens, and the frequency of failed appointments by

patients whose treatment is publicly funded.⁵⁻⁸ Nevertheless, American Dental Association (ADA)

survey data reveals that pediatric dentists report the highest percentage of patients insured through

57 public assistance among all dentists.⁹ <u>Medicaid-enrolled children living in areas with more pediatric</u>

58 dentists are more likely to utilize preventive dental care.¹⁰ However, Especially when considering

59 the disincentives of participating as Medicaid/CHIP providers, more dentists and/or non-dentist oral

60 health care providers cannot be considered the panacea for oral health disparities.

61

62 Inequities in oral health can result from underutilization of services. Lack of health literacy, limited

63 English proficiency, and cultural and societal barriers can lead to difficulties in utilizing available

64 services. Financial circumstances and as well as geographical and /transportational considerations

also can impede

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- 66 access to care. Eliminating such barriers will require a collaborative, multi-faceted approach.^{11,12}
- 67 Systematic policy and environmental changes that improve living conditions and alleviate poverty are
- 68 <u>needed to directly address the social determinants of health.¹³ All the while, stakeholders must</u>
- 69 promote education and primary prevention so that disease levels and the need for therapeutic services
- 70 decrease.
- 71
- 72 All AAPD advocacy efforts are based upon the organization's core values (AAPD Core Values)
- 73 <u>strategic objectives.⁴ which include:</u>
- 74 1. Health and health care equity.
- 75 2. An effective dental workforce.
- 76 3. Effective public programs.
- 77 4. Oral health promotion.
- 78 5. Child and adolescent welfare.
- 79 6. Science, education, research, and evidence-based care.
- 80

81 A major component of AAPD's advocacy efforts is development of oral health policies, and 82 evidence based clinical practice guidelines, and best practices that promote access to and delivery of 83 safe, high quality comprehensive oral health care for all children, including those with special health 84 care needs, within a dental home. A dental home is the ongoing relationship between the dentist and 85 the patient, inclusive of all aspects of oral health care delivery, in a comprehensive, continuously 86 accessible, coordinated, and family-centered way.¹⁴ Such care takes into consideration the patient's 87 age, developmental status, and psychosocial well-being and is appropriate to the needs of the child and family. This concept of a dental home was detailed in a 2001 AAPD oral health policy¹⁵ and is 88 derived from the American Academy of Pediatrics' (AAP) model of a medical home.^{16,17} Children-89 who have a dental home are more likely to receive appropriate preventive and therapeutic oral health-90 91 care. The AAPD, AAP, ADA, and Academy of General Dentistry support the establishment of a dental home as early as six months of age and no later than 12 months of age.^{15,17-19} This provides 92 93 time-critical opportunities to provide education on preventive health practices and reduce a child's risk of preventable dental/oral disease when delivered within the context of an ongoing relationship. 94 95 Prevention can be customized to an individual child's and/or family's risk factors. Growing evidence supports the effectiveness of early establishment of a dental home visits in reducing early childhood 96 caries.²¹⁻²² Each child's dental home should include the capacity to refer to other dentists or medical 97

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- 98 care providers when all medically necessary care cannot be provided within the dental home. The
- AAPD strongly believes a dental home is essential for ensuring optimal oral health for all children.²³
 100
- 101 Central to the dental home model is dentist-directed care. The dentist performs the examination, 102 diagnoses oral conditions, and establishes a treatment plan that includes preventive services, and all 103 services are carried out under the dentist's supervision. The dental home delivery model implies 104 direct supervision (i.e., physical presence during the provision of care) of allied dental personnel by the dentist. The allied dental personnel [e.g., dental hygienist, expanded function dental 105 106 assistant/auxiliary (EFDA), dental assistant] work under direct supervision of the dentist to increase 107 productivity and efficiency while preserving quality of care. This model also allows for provision of 108 preventive oral health education by EFDAs and preventive oral health services by a dental hygienist allied dental personnel under general supervision (i.e., without the presence of the supervising dentist 109 110 in the treatment facility) following the examination, diagnosis, and treatment plan by the licensed, supervising dentist. Furthermore, the dental team can be expanded to include auxiliaries who go into 111 112 the community to provide education and coordination of oral health services. Utilizing allied 113 personnel to improve oral health literacy could decrease individuals' risk for oral diseases and 114 mitigate a later need for more extensive and expensive therapeutic services. 115 116 In addition to promoting quality advancing optimal oral health eare for all children through its by developing policies and guidelines, AAPD advocacy efforts, in part, include: 117 118 1. Improving perinatal and infant oral health by training pediatric and general dentists to performinfant oral health examinations. Working closely with legislators, professional associations and 119 120 health care professionals to implement research opportunities in pediatric oral health and educate pediatric dentists, health care providers and the public regarding pediatric oral health. 121 2. Representing pediatric dentists on an advisory committee to the Bureau of Health Professions, 122 promoting Convening an annual Advocacy Conference in Washington, D.C. to advocate for 123 124 funding for pediatric and general dentistry residency programs and faculty loan repayment. 3. Conducting annual workshops which train pediatric dentists from across the country to educate 125 legislators on strategies to improve access to pediatric dental care. 126 127 4.3. Working with the ADA to identify non-financial barriers to oral health care and develop recommendations to improve access to care for Medicaid recipients.^{24,25} 128 129 5.4. Partnering with federally funded agencies to develop strategies to improve children's oral
- health.²⁶

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- 6.5. Utilizing a Task Force on Workforce Issues (2008-2009) to examine the Investigating various
 non-dentist (also known as mid-level) provider models that exist and/or are being proposed to
 address the access to care issues.²⁷
- 134

135 The AAPD Task Force TFWI reported that a number of provider models to improve access to care for 136 disadvantaged children have been proposed and, in some cases, implemented following the Surgeon General's report.¹ At the heart of the issue with each non-dentist provider proposal is ensuring 137 ongoing access to dental care for the underserved. Therefore, practice location and retention of 138 139 independent non-dentist providers are important considerations. When providers are government employees (e.g, Indian Health Services, National Health Services Corps), they are assigned to high-140 need areas. assignment to areas of greatest need is possible. The dental therapy model has been shown 141 to improve use of dental care services in Alaska.^{28,29} However, the current U.S. proposed models are 142 private practice/non-government employee models, providing no assurances that independent 143 providers will locate in underserved areas. Recent case studies of private practices in Minnesota 144 describe the impact of dental therapists on production. Their findings suggest that while a therapist 145 146 joining a dentist in a located practice may increase the dentist's efficiency, it does not expand geographic access to dental care characteristic of the Alaska initiative or of the international model of 147 148 therapists.³⁰⁻³² Moreover, evidence from several developed countries that have initiated mid-level 149 provider programs suggests that, when afforded an opportunity, those practitioners often gravitate 150 toward private practice settings in less remote areas, thereby diminishing the impact on care for the 151 underserved.33 152 153 In all existing and proposed non-dentist provider models, the clinician receives abbreviated levels of education compared to the educational requirements of a dentist. For example, the dental health aid 154

- therapist model in Alaska is a two-year certificate program with a pre-requisite high school
- education,^{34,35} the educational requirement for licensure as a dental therapist in Minnesota is a
- 157 baccalaureate from a dental therapy program (Minnesota 2008), and proposed legislation for dental-
- 158 therapists in Vermont requires a two year curriculum including at least 100 hours of dental therapy-
- 159 clinical practice under the general supervision of a licensed dentist (Vermont 2011). The level of
- 160 educational training varies from state to state,³⁶⁻³⁸ and none of the current programs are approved by
- 161 <u>CODA. In contrast, b</u>Building on their college education, dental students generally spend four years
- 162 learning the biological principles, diagnostic skills, and clinical techniques to distinguish between
- 163 health and disease and to manage oral conditions while taking into consideration a patient's general

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health and well-being. The clinical care they provide during their doctoral education is under direct
supervision. Those who specialize in pediatric dentistry must spend an additional 24 or more months
in a full time post-doctoral program that provides advanced didactic and clinical experiences.³⁹ The
skills that pediatric dentists develop are applied to the needs of children through their ever-changing
stages of dental, physical, and psychosocial development, treating conditions and diseases unique to
growing individuals.

170

171 While most pediatric dental patients can be managed effectively using communicative behavioral

172 guidance techniques, many of the disadvantaged children who exhibit the greatest levels of dental

173 disease require advanced techniques (e.g., sedation, general anesthesia).^{40,41} Successful behavior

guidance enables the oral health team to perform quality treatment safely and efficiently and to

175 nurture a positive dental attitude in the pediatric patient.⁴² Accurate diagnosis of behavior and safe

and effective implementation of advanced behavior guidance techniques necessitate specialized

177 knowledge and experience.

178

179 Studies addressing the technical quality of restorative procedures performed by non-dentist providers have found, in general, that within the scope of services and circumstances to which their practices 180 181 are limited, the technical quality is comparable to that produced by dentists.^{43,44} There is, however, no 182 evidence to suggest that they deliver any expertise comparable to a dentist in the fields of diagnosis, pathology, trauma care, pharmacology, behavioral guidance, treatment plan development, and care of 183 184 patients with special health care needs patients. It is essential that policy makers recognize that evaluations which demonstrate comparable levels of technical quality merely indicate that individuals 185 186 know how to provide certain limited services, not that those providers have the knowledge and 187 experience necessary to determine whether and when various procedures should be performed or to manage individuals' comprehensive oral health care, especially with concurrent conditions that may 188 complicate treatment or have implications for overall health. Technical competence cannot be equated 189 190 with long-term outcomes.

191

192 The AAPD continues to work diligently to ensure that the dental home is recognized as the

193 foundation for delivering oral health care of the highest quality to infants, children, and adolescents,

194 including those with special health care needs. The AAPD envisions that many new and varied

delivery models will be proposed to meet increasing demands on the infrastructure of existing oral

196 health care services in the U.S.. New Zealand, known for utilizing dental therapists since the 1920s

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and frequently referenced as a workforce model for consideration in the U.S., recently completed its-

198 first nationwide oral health status survey in over 20 years. makes D dental care is available at no cost

199 for children up to 18 years of age, with most public primary schools having a dental clinic and many

200 regions operating mobile clinics.⁴⁵ In New Zealand's most recent nationwide oral health status

201 <u>survey</u>, Ooverall, one in two children in New Zealand aged two-17 years was caries-free. The caries

rate for five-year-olds and eight-year-olds in 2009 was 44.4 percent and 47.9 percent respectively.⁴⁶

203 These caries rates, which are higher than the U.S., United Kingdom, and Australia, help refute a

204 presumption that utilization of non-dentist providers will overcome the disparities.

205

As technology continues to improve, proposed models may suggest dentist supervision of services

207 outside the primary practice location via electronic communicative means to be comparable in safety

and effectiveness to services provided under direct supervision by a dentist. Health care already has

209 witnessed benefits of electronic communications in diagnostic radiology and other consultative

210 services. The AAPD encourages exploration of new models of dentist-directed health care services

that will increase access to care for underserved populations. But as witnessed through the New

212 Zealand oral health survey, a multi-faceted approach will be necessary to improve the oral health

213 status of our nation's children.

214

215 Policy statement

The American Academy of Pediatric Dentistry remains committed in its core values vision and
mission to address the disparities between children who lack access to quality oral health care and
those who benefit from such services. AAPD believes that all infants, children, and adolescents,
including those with special health care needs, deserve access to high quality comprehensive
preventive and therapeutic oral health care services provided through a dentist-directed dental home.

In the delivery of all dental care, patient safety must be of paramount concern.

222

AAPD encourages the greater use of expanded function dental assistants/auxiliaries and dental
hygienists under direct supervision by a dentist to help increase volume of services provided within a
dental home, based upon their proven effectiveness and efficiency in a wide range of settings.⁴⁴⁻⁵⁰
The AAPD also supports provision of preventive oral health services by a dental hygienist under
general supervision (i.e., without the presence of the supervising dentist in the treatment facility)
following the examination, diagnosis, and treatment plan by the licensed, supervising dentist.

229 Similarly, partnering with other health providers, especially those who most often see children during

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230	the f	irst years of life (e.g., pediatricians, family physicians, pediatric nurses), will expand efforts for			
231	improving children's oral health.				
232					
233	The AAPD strongly believes there should not be a two-tiered standard of care, with our nation's most				
234	vulnerable children receiving services by providers with less education and experience, especially				
235	when	n evidence based research to support the safety, efficiency, effectiveness, and sustainability of			
236	such delivery models is not available.				
237					
238	AAF	PD will continue its efforts to:			
239	1.	Educate families, health care providers, academicians, community leaders, and partnered			
240		governmental agencies on the benefits of early establishment of a dental home.			
241	2.	Forge alliances with legislative leaders that will advance the dental home concept and improve			
242		funding for delivery of oral health care services and dental education.			
243	3.	Expand public-private partnerships to improve the oral health of children who suffer			
244		disproportionately from oral diseases.			
245	4.	Encourage recruitment of qualified students from rural areas and underrepresented minorities			
246		into the dental profession.			
247	5.	Partner with other dental and medical organizations to study barriers to care and			
248		underutilization of available services.			
249	6.	Support scientific research on safe, efficacious, and sustainable models of delivery of dentist-			
250		directed pediatric oral health care that is consistent with AAPD's oral health policies and			
251		clinical practice guidelines recommendations.			
252					
253	Furtl	nermore, AAPD encourages researchers and policy makers to consult with AAPD and its state			
254	units	in the development of pilot programs and policies that have potential for significant impact in			
255	the c	lelivery of oral health care services for our nation's children.			
256					
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Policy on School Absences for Dental Appointments*

2

3 Review Council

- 4 Council on Clinical Affairs
- 5 Latest Revision Reaffirmation
- 6 2015 <u>2019</u>
- 7

8 Purpose

9 The American Academy of Pediatric Dentistry (AAPD) recognizes dental care is medically necessary and

10 that poor oral health can negatively affect a child's ability to learn. This policy is intended to assist public

- 11 health and school education administrators in developing enlightened policies on school absence for
- 12 dental appointments and support parents in seeking medically necessary care for their children.
- 13

14 Methods

- 15 This policy was originally developed by the Council on Clinical Affairs and adopted in 2010. <u>This</u>
- 16 document is a reaffirmation of the previous version, revised in 2015. An electronic database search was
- 17 conducted using the terms: school absences for dental appointments, excused school absences, and
- 18 department of education. Papers for review were chosen from this list and from references within selected
- 19 articles. When data did not appear sufficient or were inconclusive, recommendations were based on
- 20 expert and/or consensus opinion by experienced researchers and clinicians. It is beyond the scope of this
- 21 document to review every state statute and regulation on absences from school for dental appointments.

22

23 Background

- 24 Oral health is integral to general health. Many systemic diseases and conditions have oral manifestations.
- 25 These oral manifestations may be the initial sign of clinical disease and indicate the need for further
- assessment.¹ Oral conditions can interfere with eating and adequate nutritional intake, speaking, self-
- esteem, daily activities, and quality of life.² Dental care is medically necessary to prevent and eliminate
- 28 orofacial disease, infection, and pain. It is also important to restore the form and function of the dentition
- and correct facial disfiguration or dysfunction.³ The public's lack of awareness of the importance of oral
- 30 health is a major barrier to dental care.¹ Unrecognized disease and postponed care result in exacerbated

^{*} ABBREVIATION

AAPD: American Academy Pediatric Dentistry.

- 31 problems, which lead to more extensive and costly treatment needs.³
- 32

33 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 34 healthy and fit physically, mentally, and socially".⁴ Children and adolescents with poorer oral health 35 status are more likely to experience oral pain, miss school, and perform poorly in school compared with 36 their counterparts with better oral health status.⁵ Children with dental pain may be irritable, withdrawn, 37 or unable to concentrate. Pain can affect test performance as well as school attendance.^{1,6} Left untreated, 38 the pain and infection caused by tooth decay can lead to problems in eating, speaking, and learning.⁷ 39 40 The social impact of oral disease in children is substantial. More than 51 million school hours are lost 41 42 each year to dental related illness.¹ On average, children and adolescents with oral health problems are absent one school day per year more than other children or adolescents.⁸ When these problems are treated 43 44 and children no longer are experiencing pain, their learning and school attendance improve.² 45 46 According to the U.S. Surgeon General, "a national public health plan for oral health does not exist".¹ 47 This corresponds with the fact that there is no national policy on excused absences from school for dental

appointments. Some states (e.g., California, Texas) have very specific laws excusing students for dental
 appointments.^{9,10} Other state laws are more general and recognize absences due to doctor's appointments
 or illness.^{11,12}

51

52 Policy statement

Dental care is medically necessary and oral health is integral to general health. Undiagnosed and untreated oral conditions may interfere with a child's ability to eat, sleep, or function well at home or at school due to discomfort or pain. The unaesthetic nature of caries and dental malocclusion may compromise a child's self-esteem and social development. Schools' policies that prevent or discourage legitimate school absence for the purpose of delivery of vital health care services may cause harm to their students.

59

60 Children who have their dental conditions corrected improve learning and attendance in school. State

61 laws and local school district policies are not uniform on absences from school for dental appointments. A

62 uniform policy that recognizes the negative effect of chronic truancy on academic performance would be

63 useful. Such policies should not restrict necessary health care delivery.

CCA.h. P_SchoolAbsences

64

- 65 The AAPD supports state law or school policy that allows the absence for legitimate health care delivery,
- 66 including that of oral health services, and encourages parents, school administrators, and dentists to work
- 67 together to ensure that children receive dental care while minimizing school absences.
- 68

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100

1 [Best Practices]

2 Antibiotic Prophylaxis for Dental Patients at Risk for Infection*

- 3
- 4 Review Council
- 5 Council on Clinical Affairs
- 6 Latest Revision
- 7 2014 <u>2019</u>
- 8
- 9 Purpose

10 The American Academy of Pediatric Dentistry (AAPD) recognizes that numerous medical conditions

11 predispose patients to bacteremia-induced infections. Because it is not possible to predict when a

12 susceptible patient will develop an infection, prophylactic antibiotics are recommended when these

13 patients undergo procedures that are at risk for producing bacteremia. This guideline is intended to

14 help practitioners make decisions regarding antibiotic prophylaxis for dental patients at risk.

15

16 Methods

17 This guideline was originally developed by the Council on Clinical Affairs and adopted in 1990. This 18 document is a revision of the previous version, last revised in 2011/2014, and based on a review of 19 current dental and medical literature pertaining to post procedural bacteremia-induced infections. This 20 document included database searches using key terms: infective endocarditis (IE), bacteremia, 21 antibiotic prophylaxis, and dental infection. Articles were evaluated by title and/or abs-tract and 22 relevance to dental care for children, adolescents, and those with special health care needs. Thirty-one 23 citations were chosen from this method and from references within selected articles. When data did 24 not appear sufficient or were inconclusive, recommendations were based upon expert and/or 25 consensus opinion by experienced researchers and clinicians. In addition, "Prevention of infective endocarditis: Guidelines from the American Heart Association,"^{1,2} "Infective Endocarditis in 26 27 Childhood: 2015 Update: A Scientific Statement From the American Heart Association,"³ and the

28 ADA report on "The use of prophylactic antibiotics prior to dental procedures in patients with

* ABBREVIATIONS

AAOS: American Academy of Orthopedic Surgeons. **AAPD**: American Academy Pediatric Dentistry. **ADA**: American Dental Association. **AHA**: American Heart Association. **CHD**: Congenital heart disease. **CIED**: Cardiovascular implantable electronic device. **IE**: Infective endocarditis.

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- 29 <u>prosthetic joints⁴</u> "Prevention of orthopedic implant infection in patients undergoing dental-
- 30 procedures: Guidelines from the American Academy of Orthopedic Surgeons (AAOS) and American
- 31 Dental Association (ADA)"(Watters et al 2013, AAOS 2013) were reviewed.
- 32

33 Background

- 34 Bacteremia, bacteria in the bloodstream, is anticipated following invasive dental procedures and can
- 35 lead to complications in an immunodeficient patient.^{5,6} Patients with high risk cardiac disease,
- 36 <u>immunosuppression and immunodeficiencies may compromise one's ability to fight simple infection.</u>
- 37 <u>The rationale for antibiotic prophylaxis was to reduce or eliminate transient bacteremia caused by</u>
- 38 <u>invasive dental procedures.</u>⁷ <u>Infective endocarditis is an uncommon but life threatening complication</u>
- 39 resulting from bacteremia. The incidence of infections such as IE ranges from 5.0 to 7.9 per 100,000-
- 40 person-years with a significant increasing trend among women.(de Sa et al 2010)_Only a limited-
- 41 number of bacterial species have been implicated in resultant postoperative infections. Viridans group
- 42 streptococci, *Staphylococcus aureus*, enterococcus, pseudomonas, serratia, and candida are some of
- 43 the microorganisms implicated with IE.(Wilson et al 2007-JADA, Wilson et al 2007-Circulation) The
- 44 vast majority of cases of IE caused by oral microflora can result from bacteremia associated with
- 45 routine daily activities such as toothbrushing, flossing, and chewing.(Wilson et al 2007 JADA,
- 46 Wilson et al 2007-Circulation) However, antibiotic Antibiotic prophylaxis is recommended with-
- 47 certain dental procedures.(Wilson et al 2007-JADA, Wilson et al 2007-Circulation, Lockhart et al-
- 48 2004, Roberts et al 2006) An effective antibiotic regimen should be directed against the most likely-
- 49 infecting organism, with antibiotics administered shortly before the procedure. When procedures-
- 50 involve infected tissues or are performed on a patient with a compromised host response, additional-
- 51 doses or a prescribed postoperative regimen of antibiotics may be necessary.
- 52
- 53 Antibiotic usage may result in the development of resistant organisms.^{1,2,5,6,8-10} Utilization of
- 54 antibiotic prophylaxis for patients at risk does not provide absolute prevention of infection. Post-
- 55 procedural symptoms of acute infection (e.g., fever, malaise, weakness, lethargy) may indicate
- 56 antibiotic failure and need for further medical evaluation.
- 57
- 58 The decision to use antibiotic prophylaxis should be made on an individual basis. Some medical
- 59 conditions that may predispose patients to post-procedural infections are discussed below. This is not
- 60 intended to be an exhaustive list; rather, the categorization should help practitioners identify children
- 61 who may be at increased risk. If a patient reports a syndrome or medical condition with which the

- 62 practitioner is not familiar, it is appropriate to contact the child's physician to determine susceptibility
- 63 to bacteremia-induced infections.
- 64
- 65 In 2007, the American Heart Association (AHA) released its newly revised guidelines for the
- 66 prevention of IE and reducing the risk for producing resistant strains of bacteria.(Wilson et al 2007-
- 67 JADA, Wilson et al 2007-Circulation) The AAPD, acknowledging the AHA's expertise and efforts to
- 68 produce evidenced based recommendations, continues to endorse the AHA guideline for antibiotic-
- 69 prophylaxis, entitled "Prevention of Infective Endocarditis".
- 70

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71 The significant reasons for the revision include (Wilson et al 2007-JADA, Wilson et al 2007-
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- 72 Circulation):
- **IE is much more likely to result from frequent exposure to random bacteremias associated
 with daily activities than from bacteremia caused by a dental, GI tract, or GU tract
- whith during derivities than from bacterennia eaused by a dental, of frace, of 00 frace
- 75 procedure." (Wilson et al 2007-JADA) (Daily activities would include toothbrushing,
- 76 flossing, chewing, using toothpicks, using water irrigation devices, and other activities.)
- **Prophylaxis may prevent an exceedingly small number of cases of IE if any, in individuals
 who undergo a dental, GI tract, or GU tract procedure.
- The risk of antibiotic associated adverse events exceeds the benefit, if any, from
 prophylactic antibiotic therapy.
- Maintenance of optimal oral health and hygiene may reduce the incidence of bacteremia
- 82 from daily activities and is more important than prophylactic antibiotics for a dental
- 83 procedure to reduce the risk of IE." (Wilson et al 2007-JADA)
- 84
- 85 The 2007 AHA revision was intended to clarify when antibiotic prophylaxis is/is not recommended-
- 86 and to provide more uniform global recommendations. Major changes from the 1997 version (Wilson-

87	at al 2007 IADA	Wilson et al 2007	Circulation Da	viani at al 1007) include.
07	ct al 2007-311D11 ,	wilson et al 2007	Circulation, De	ijani et al 1777	7 menuae.

- * "The Committee concluded that only an extremely small number of cases of infective
 endocarditis might be prevented by antibiotic prophylaxis for dental procedures even if such-
- 90 prophylactic therapy were 100 percent effective.
- 91 Infective endocarditis prophylaxis for dental procedures is reasonable only for patients with
- 92 underlying cardiac conditions associated with the highest risk of adverse outcome from 93 infective endocarditis.

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94	 For patients with these underlying cardiac conditions, prophylaxis is reasonable for all-
95	dental procedures that involve manipulation of gingival tissue or the periapical region of
96	teeth or perforation of the oral mucosa.
97	Prophylaxis is not recommended based solely on an increased lifetime risk of acquisition of
98	infective endocarditis." (Wilson et al 2007-JADA)
99	
100	Recommendations-
101	To date, the evidence base supporting the efficacy and use of antibiotic prophylaxis is limited,
102	especially in the pediatric population. Many of the indications are based on consensus. ^{3,11-13} The
103	conservative use of antibiotics is indicated to minimize the risk of developing resistance to current
104	antibiotic regimens. ^{1,2,8,14,15} Given the increasing number of organisms that have developed resistance
105	to current antibiotic regimens, as well as the potential for an adverse anaphylactic reaction to the drug
106	administered, it is best to be judicious in the use of antibiotics for the prevention of $IE^{1,2}$ and or other
107	distant-site infections. ^{8.,10,16}
108	
109	Recommendations
110	Antibiotic prophylaxis is recommended with certain dental procedures ^{1-3,5,7,17} but this should be
111	directed against the most likely infecting organism. When procedures involve infected tissues or are
112	performed on a patient with a compromised host response, additional doses or a prescribed pre- and
113	postoperative regimen of antibiotics may be necessary. Emphasis should be placed on the prevention
114	of disease, establishment of good oral health care habits and routine oral health assessments through a
115	dental home. to This may prevent the frequent need for the use of antibiotic therapy and, thus,
116	decrease the risks of resistance and adverse events relation to its use. ^{7,18,19}
117	
118	
119	Patients with cardiac conditions
120	Infective endocarditis is an example of an uncommon but life-threatening complication resulting from
121	bacteremia. The incidence pediatric admissions due to infective endocarditis was between 0.5 and
122	0.12 cases per 1000 admissions from 2003-2010. ³ Only a limited number of bacterial species have
123	been implicated in resultant postoperative infections. Viridans group streptococci, Staphylococcus
124	aureus, Enterococcus species are the main microorganisms implicated in IE. ¹⁻³ Enterococcal and other
125	organisms such as Haemophilus species, Aggregatibacter species, Cardiobacterium hominis,

CCA.i. BP_AntibioticProphylaxis

126	Eikenella corrodens and Kingella species are less common. ³ Routine daily activities such as			
127	toothbrushing, flossing, and chewing contribute more to the incidence of bacteremia when compared			
128	to dental procedures. ^{1,2} There has been a shift of focus from antibiotic prophylaxis to an emphasis on			
129	oral hygiene and the prevention of oral diseases. ^{3,7,12,13,17,19}			
130				
131	In 2007, the American Heart Association (AHA) revised its guidelines for the prevention of IE and			
132	reducing the risk for producing resistant strains of bacteria. ^{1,2} The significant reasons for the revision			
133	include: ^{1,2}			
134	• <u>"IE is much more likely to result from frequent exposure to random bacteremias associated</u>			
135	with daily activities than from bacteremia caused by a dental, GI tract, or GU tract			
136	procedure." (Daily activities would include toothbrushing, flossing, chewing, using			
137	toothpicks, using water irrigation devices, and other activities.)			
138	• <u>Prophylaxis may prevent an exceedingly small number of cases of IE if any, in individuals</u>			
139	who undergo a dental, GI tract, or GU tract procedure.			
140	• The risk of antibiotic-associated adverse events exceeds the benefit, if any, from			
141	prophylactic antibiotic therapy.			
142	• <u>Maintenance of optimal oral health and hygiene may reduce the incidence of bacteremia</u>			
143	from daily activities and is more important than prophylactic antibiotics for a dental			
144	procedure to reduce the risk of IE."			
145				
146	The AHA guidelines focus on antibiotic prophylaxis prior to certain dental procedures for patients in			
147	the highest risk group (See Table 1). ^{1-3,5} Globally, there is still a lack of consensus with regards to the			
148	benefit of antibiotic prophylaxis for prevention of infective endocarditis. Since the change in			
149	recommendations, the rate and incidence of IE has been low. ³			
150				
151	The AHA recommends that children with cyanosis with specific periodontal concerns may have an			
152	increased risk of IE, which makes optimum oral hygiene very important. Dental practitioners should			
153	consider prophylactic measures to minimize the risk of IE in patients with underlying cardiac-			
154	conditions. The risk of developing IE can arise from a combination of high-risk patients and dental-			
155	procedures. However, at At-risk patients with poor oral hygiene and gingival bleeding after routine			
156	activities (e.g., toothbrushing) also have shown an increased incidence of bacteremia as a measure for			
157	risk potential for developing complications of IE. ^{1,2, 21,22} The focus should be on maintaining good oral			

158 <u>hygiene</u>, routine dental reviews examinations, infection control to reduce bacteremia and

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- 159 <u>discouraging tattooing or piercing rather than relying on antibiotic prophylaxis for patients at</u>
- 160 <u>risk.^{12,13,17-19,22}</u> It, therefore, is recommended to encourage daily good oral hygiene practices to reduce
- 161 gingivitis as part of the prophylactic regimen.(Wilson et al 2007-JADA, Wilson et al 2007-
- 162 Circulation, Lockhart et al 2009, Lockhart et al 2008) These patients and/or their parents need to be
- educated and motivated to maintain personal oral hygiene through daily plaque removal, including
- 164 flossing.^{1,2} Greater <u>There is a shift in the emphasis should be placed</u> on improved access to dental care
- and oral health in patients with underlying cardiac conditions at high risk for IE and less <u>of a focus</u> on
- a dental procedure and antibiotic coverage.³ Professional prevention strategies should be based upon
- the individual's assessed risk for caries and periodontal disease.
- 168

169 Specific recommendations from the 2007 AHA guideline on prevention of IE are included in the

- 170 following tables. The AHA recommends antibiotic prophylaxis only for those whose underlying
- 171 cardiac conditions are associated with the highest risk of adverse outcome (Wilson et al 2007-JADA,
- 172 Wilson et al 2007-Circulation) (see Table 1). Such conditions include prosthetic heart valves or
- 173 prosthetic material used for cardiac valve repair, a previous history of IE unrepaired or incompletely-
- 174 repaired cyanotic congenital heart disease (CHD) including palliative shunts and conduits,
- 175 completely repaired congenital heart defect with prosthetic material or device (whether placed by-
- 176 surgery or by catheter intervention) during the first six months after the procedure, and repaired CHD-
- 177 with residual defects at the site or adjacent to the site of a prosthetic patch or device, and cardiac
- 178 transplantation recipients who develop cardiac valvulopathy such as valve regurgitation due to a-
- 179 structurally abnormal valve.(Wilson et al 2007-JADA, Wilson et al 2007-Circulation, Nishimura et al-
- 180 2008)
- 181 After the 2007 AHA revised guidelines were published, there were concerns by healthcare providers-
- 182 that there may be an increase in cases of viridans group streptococci IE due to the decrease in the
- 183 numbers of patients requiring antibiotic prophylactic coverage for dental procedures.(DeSimone et al-
- 184 2012) A population based review of definitive and possible cases of IE demonstrated no observed
- 185 increase in viridans group streptococci IE after publication of the 2007 AHA endocarditis prevention-
- 186 guidelines.(DeSimone et al 2012) In addition to those diagnoses listed in the AHA guidelines,
- 187 patients with a reported history of injection drug use may be considered at risk for developing IE in
- 188 the absence of cardiac anomalies.²¹ Patients should also be discouraged from tattooing and
- 189 <u>piercing</u>.^{12,13,23} Although quite rare, complications from intraoral tongue piercing can include IE-
- among patients with a pre-existing cardiac valvular condition and/or history of injection drug-
- **191** use.(Lick et al 2005, Akhondi and Rahimi 2002, Tronel et al 2001) Consultation with the patient's

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- 192 physician may be necessary to determine susceptibility to bacteremia-induced infections.
- 193 <u>*</u>Antibiotics are recommended for all dental procedures that involve manipulation of gingival tissue
- 194 or the periapical region of teeth or perforation of the oral mucosa for cardiac patients with the highest
- risk^{1,2} (see Table 1 and 2). Specific antibiotic regimens can be found in Table 3. Practitioners and
- 196 patients/ parents can review the entire AHA guidelines in the AHA Circulation Journal archives,
- 197 "http://circ.ahajournals.org/cgi/ content/full/116/15/1736" for additional background information as
- 198 well as discussion of special circumstances (e.g., patients already receiving antibiotic therapy,
- 199 patients on anti-coagulant therapy).
- 200

201 Patients with compromised immunity

- 202 Patients with a compromised immune system may not be able to tolerate a transient bacteremia-
- 203 following invasive dental procedures. (Lockhart et al 2007) These non-cardiac factors can place a-
- 204 patient with compromised immunity at risk for distant-site infection from a dental-
- 205 procedure.(Lockhart et al 2007) Non-cardiac patients with a compromised immune system may be at
- 206 risk for complications of bacteremia and distant site infection following invasive dental procedures).
- 207 Existing evidence does not support the extensive use of antibiotic prophylaxis and should only be
- 208 limited to immunocompromised patients or those at high risk.¹⁸ Consultation with the patient's
- 209 physician is recommended for management of patients with a compromised immune system. This-
- 210 category Although there is not enough data to support its use, high risk patients who should be
- 211 <u>considered for use of prophylaxis includes</u>, but is not limited to, patients with the following medical
- **212** conditions:^{12,13,24}
- **213** 1. Immunosuppression secondary to:
- a. human immunodeficiency virus (HIV);
- b. severe combined immunodeficiency (SCIDS);
- 216 c. neutropenia;
- d. cancer chemotherapy; and
- e. hematopoietic stem cell or solid organ transplantation.
- 219 2. Head and neck radiotherapy.
- 220 3. Autoimmune disease (e.g., juvenile arthritis, systemic lupus erythematosus).
- **221** 4. Sickle cell anemia.²⁵
- 222 5. Asplenism or status post splenectomy.
- 223 6. Chronic <u>high dose steroid usage</u>.
- 224 7. Diabetes <u>mellitus</u>.

225 8. Bisphosphenate therapy.^{26,27}

- 226 <u>9. Hemodialysis</u>
- 227 Consultation with the child's physician is recommended for management of patients with a

228 compromised immune system. Discussion of antibiotic prophylaxis for patients undergoing

- chemotherapy, irradiation, and hematopoietic cell transplantation appears in a separate AAPD
- 230 guideline.²⁸
- 231

232 Patients with shunts, indwelling vascular catheters, or medical devices

- 233 The AHA recommends that antibiotic prophylaxis for non-valvular devices, including indwelling
- vascular catheters (e.g., central lines) and cardiovascular implantable electronic devices (CIED), is
- 235 indicated only at the time of placement of these devices in order to prevent surgical site
- infection.^{22,24,29} The AHA found no convincing evidence that microorganisms associated with dental
- procedures cause infection of CIED and nonvalvular devices at any time after implantation.^{22,24,29} The
- 238 infections occurring after device implantation most often are caused by *Staphylococcus aureus* and
- 239 coagulase negative staphylococci or other microorganisms that are non-oral in origin but are
- 240 associated with surgical implantation or other active infections.^{22,29,30} The AHA further states that-
- 241 immunosuppression is not an independent risk factor for nonvalvular device infections;
- 242 immunocompromised hosts who have those devices should receive antibiotic prophylaxis as-
- 243 advocated for immunocompetent hosts.(Lockhart et al 2007, Baddour et al 2003, Baddour et al 2010,
- 244 Baddour et al 2011)Consultation with the child's physician is recommended for management of
- 245 patients with nonvalvular devices.

246

- 247 Ventriculoatrial (VA), ventriculocardiac (VC), or ventriculovenus (VV) shunts for hydrocephalus are
- 248 at risk of bacteremia-induced infections due to their vascular access.^{24,31} In contrast,
- 249 ventriculoperitoneal (VP) shunts do not involve any vascular structures and, consequently, do not
- require antibiotic prophylaxis.^{24,31} Consultation with the child's physician is recommended for
- 251 management of patients with vascular shunts.

252

253 Patients with prosthetic joints

- 254 For patients with a history of total joint arthroplasty, deep hematogenous infections can lead to life
- threatening complications such as a loss of the prosthetic joint or even increased morbidity and
- 256 mortality.^{32,33} There is not enough evidence to demonstrate an association between dental procedures
- 257 and prosthetic joint infection. Antibiotic prophylaxis has not shown a significant reduction in the risk

- 258 <u>of developing joint infections subsequent to dental procedures.^{32,34} Given the increasing risk in</u>
- 259 <u>antibiotic resistance and adverse reactions, antibiotic prophylaxis prior to dental procedures is not</u>
- 260 recommended in the prevention of prosthetic joint infections.^{4,7} A 2012 information statement-
- 261 published by the AAOS recommends that dentists consider antibiotic prophylaxis for at risk joint
- 262 replacement patients who are undergoing an invasive procedure.(AAOS-Guideline on prevention of
- 263 orthopaedic implant infection) Patients with an increased risk of hematogenous total joint infection
- are all patients with a prosthetic joint replacement, previous prosthetic joint infection, inflammatory-
- 265 arthropathies (e.g., rheumatoid arthritis, systemic lupus erythematosus), megaprosthesis, hemophilia,
- 266 malnourishment, and compromised immunity (see examples in previous section).(AAOS-Guideline-
- 267 on prevention of orthopaedic implant infection) However, AAOS states that clinical judgment must-
- 268 consider the potential benefit of antibiotic prophylaxis versus the risks of adverse reactions for each
- 269 patient.(Watters et al 2013, AAOS 2013, AAOS Guideline on prevention of orthopaedic implant-
- 270 infection) The AAPD recognizes recommendations from AAOS and the ADA with regards to-
- 271 antibiotic prophylaxis for patients with joint replacement. A joint collaboration of the AAOS and the
- 272 ADA developed evidence based recommendations on antibiotic prophylaxis for patients at a high risk-
- 273 for implant infection undergoing dental procedures (Table 4). (Watters et al 2013, AAOS 2013,
- 274 AAOS-Guideline on prevention of orthopaedic implant infection)
- 275
- 276 Currently, the AAPD endorses the 2012 recommendations of the ADA and the AAOS for-
- 277 management of patients with prosthetic joints.(Watters et al 2013, AAOS 2013, AAOS -Guideline on-
- 278 prevention of orthopaedic implant infection) Antibiotic prophylaxis has not shown a significant
- 279 reduction in the risk of developing joint infections subsequent to dental procedures.(Aminoshariae-
- and Kulild 2010, Berbari et al 2010) Therefore, antibiotic prophylaxis is not indicated for dental
- 281 patients with pins, plates, screws, or other hardware that is not within a synovial joint nor is it
- 282 indicated routinely for most dental patients with total joint replacements.(AAOS Guideline on
- 283 prevention of orthopaedic implant infection, Little et al 2010)
- 284
- 285 Consultation with the child's physician may be necessary for management of at-risk patients as well
 286 as patients with other implanted devices (e.g., Harrington rods, external fixation devices).^{24,32-35}
- 287
- 288 Acceptance by dental practitioners of AHA guidelines for antibiotic prophylaxis-
- 289 The revised 2007 AHA guidelines provided a significant reduction in patients requiring antibiotic-
- 290 prophylaxis prior to invasive dental procedures. In a survey sent to US dentists, 71 percent of the

- 291 respondents reported they were satisfied with the current guidelines.(Lockhart et al 2013) The survey-
- 292 also demonstrated that 70 percent of the dentists reported a majority of patients previously receiving-
- 293 antibiotic prophylaxis no longer required prophylaxis.(Lockhart et al 2013) However, the same-
- 294 percentage of respondents also indicated that a significant number of these patients were still-
- 295 receiving antibiotic prophylaxis regardless of the AHA revised guidelines.(Lockhart et al 2013)
- 296

Table 1.CARDIAC CONDITIONS ASSOCIATED WITH THE HIGHEST RISK
OF ADVERSE OUTCOME FROM ENDOCARDITIS FOR WHICH
PROPHYLAXIS WITH DENTAL PROCEDURES IS REASONABLE

Prosthetic cardiac valve or prosthetic material used for cardiac valve repair

Previous infective endocarditis

Congenital heart disease (CHD)*

Unrepaired cyanotic CHD, including palliative shunts and conduits

Completely repaired congenital heart defect with prosthetic material or device, whether placed by surgery or by catheter intervention, during the first six months after the procedure †

Repaired CHD with residual defects at the site or adjacent to the site of a prosthetic patch or prosthetic device (which inhibit endothelialization)

Cardiac transplantation recipients who develop cardiac valvulopathy

* Except for the conditions listed above, antibiotic prophylaxis is no longer recommended for any other form of CHD.

[†] Prophylaxis is reasonable because endothelialization of prosthetic material occurs within six months after the procedure.

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Table 2. DENTAL PROCEDURES FOR WHICH ENDOCARDITIS PROPHYLAXIS IS REASONABLE FOR PATIENTS IN TABLE 1

All dental procedures that involve manipulation of gingival tissue or the periapical region of teeth or perforation of the oral mucosa**

** The following procedures and events do not need prophylaxis: routine anesthetic injections through non-infected tissue, taking dental radiographs, placement of removable prosthodontic or orthodontic appliances, adjustment of orthodontic appliances, placement of orthodontic brackets, shedding of deciduous teeth, and bleeding from trauma to the lips or oral mucosa.

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Table 3. REGIMENS FOR A I	DENTAL PROCEDURE				
Regime	Regimen: Single Dose 30 to 60 min Before Procedure				
Situation	Agent	Adults	Children		
Oral	Amoxicillin	2 g	50 mg/kg		
Unable to take oral medication	Ampicillin OR	2 g IM or IV	50 mg/kg IM or IV		
	Cefazolin or ceftriaxone	1 g IM or IV	50 mg/kg IM or IV		
Allergic to penicillins or	Cephalexin*† OR	2 g	50 mg/kg		
amipicillin—oral	Clindamycin OR	600 mg	20 mg/kg		
	Azithromycin or clarithromycin	500 mg	15 mg/kg		
Allergic to penicillin or amipicillin and unable to take oral	Cefazolin or ceftriaxone† OR	1 g IM or IV	50 mg/kg IM or IV		
medication	Clindamycin	600 mg IM or IV	20 mg/kg IM or IV		

IM indicates intramuscular; IV, intravenous.

* Or other first-or second-generation oral cephalosporin in equivalent adult or pediatric dosage.

[†] Cephalosporins should not be used in an individual with a history of anaphylaxis, angioedema, or urticaria with penicillins or ampicillin.

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Table 4. AAOS/ADA PREVENTION OF ORTHOPAEDIC IMPLANT INFECTION CLINICAL PRACTICE GUIDELINE PROTOCOL RECOMMENDATIONS-(Watters et al 2013, AAOS 2013).

Recommendation 1

The practitioner might consider discontinuing the practice of routinely prescribing prophylactic antibiotics for patients with hip and knee prosthetic joint implants undergoing dental procedures.

Grade of Recommendation: Limited

A Limited recommendation means the quality of the supporting evidence that exists isunconvincing, or that well-conducted studies show little clear advantage to one approach versusanother. Practitioners should be cautious in deciding whether to follow a recommendationclassified as Limited, and should exercise judgment and be alert to emerging publications thatreport evidence. Patient preference should have a substantial influencing role.

Recommendation 2

We are unable to recommend for or against the use of topical oral antimicrobials in patients with prosthetic joint implants or other orthopaedic implants undergoing dental procedures.

Grade of Recommendation: Inconclusive

An Inconclusive recommendation means that there is a lack of compelling evidence resulting in an unclear balance between potential benefits and potential harm. Practitioners should feel little constraint in deciding whether to follow a recommendation labeled as Inconclusive and should exercise judgment and be alert to future publications that clarify existing evidence fordetermining balance of benefits versus potential harm. Patient preference should have a substantial influencing role.

Recommendation 3

In the absence of reliable evidence linking poor oral health to prosthetic joint infection, it is the opinion of the work group that patients with prosthetic joint implants or other orthopaedic-implants maintain appropriate oral hygiene.

Grade of Recommendation: Consensus

A Consensus recommendation means that expert opinion supports the guideline recommendation even though there is no available evidence that meets inclusion criteria. Practitioners should be flexible in deciding whether to follow a recommendation classified as Consensus, although they may set boundaries on alternatives. Patient preference should have a substantial influencing role.

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301 302

304 (NE Comment: Recommend new table "Management of patients with prosthetic joints 305 undergoing dental procedures" – request table from ADA. See sample below)

Management of patients with prosthetic joints undergoing dental procedures

Clinical Recommendation:

In general, for patients with prosthetic joint implants, prophylactic antibiotics are **not** recommended prior to dental procedures to prevent prosthetic joint infection.

For patients with a history of complications associated with their joint replacement surgery who are undergoing dental procedures that include gingival manipulation or mucosal incision, prophylactic antibiotics should only be considered after consultation with the patient and orthopedic surgeon.* To assess a patient's medical status, a complete health history is always recommended when making final decisions regarding the need for antibiotic prophylaxis.

Clinical Reasoning for the Recommendation:

- There is evidence that dental procedures are not associated with prosthetic joint implant infections.
- There is evidence that antibiotics provided before oral care do not prevent prosthetic joint implant infections.
- There are potential harms of antibiotics including risk for anaphylaxis, antibiotic resistance, and opportunistic infections like Clostridium difficile.
- The benefits of antibiotic prophylaxis may not exceed the harms for most patients.
- The individual patient's circumstances and preferences should be considered when deciding whether to prescribe prophylactic antibiotics prior to dental procedures.

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ADA. Center for Evidence-Based Dentistry"

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- 1 [Best Practices]
- ² Caries-risk Assessment and Management for Infants, Children,
- 3 and Adolescents^{*}
- 4
- 5 Review Council
- 6 Council on Clinical Affairs
- 7 Latest Revision
- 8 2014 <u>2019</u>
- 9
- 10 Key words: Dental caries, risk assessment; practice guidelines
- 11

12 Purpose

- 13 The American Academy of Pediatric Dentistry (AAPD) recognizes that caries-risk assessment and
- 14 management protocols, <u>also called care pathways</u>, can assist clinicians with decisions regarding treatment
- based on <u>child's age</u>, caries risk and patient compliance: and are essential elements of contemporary
- 16 clinical care for infants, children, and adolescents. This guideline is intended to educate health care
- 17 providers and other interested parties on the assessment of caries risk in contemporary pediatric dentistry
- 18 and aid in clinical decision making regarding <u>evidence- and risk-based</u> diagnostic, fluoride, dietary, and
- 19 restorative protocols.
- 20

21 Methods

- 22 This guideline best practices document was originally developed by the Council on Clinical Affairs and
- adopted in 2002 <u>and last revised in 2014</u>. This document is an update of AAPD's Policy on Use of a
- 24 Caries-risk Assessment Tool (CAT) for Infants, Children, and Adolescents, revised in 2006 that includes-
- 25 the additional concepts of dental caries management protocols. The update used electronic and hand
- 26 searches of English written articles in the medical and dental literature within the last 10 years using the
- 27 search terms caries risk assessment, caries management, and caries clinical protocols. From this search,
- 28 1,909 articles were evaluated by title or by abstract. Information from 75 articles was used to update this
- 29 document. To update this best practices, an electronic search of systematic reviews/meta-analyses or

* ABBREVIATIONS

AAPD: American Academy Pediatric Dentistry. **ADA**: American Dental Association. **CDC**: Centers for Disease Control and Prevention. **MS**: Mutans streptococci.

- 31 fluoride, radiology, non-restorative treatment, caries risk assessment, active surveillance, caries
- 32 prevention. There were four systematic reviews that informed this update on caries risk assessment $^{1-4}$.
- 33 <u>There were 10 systematic reviews and clinical practice guidelines that inform this update on care</u>
- 34 <u>pathways for caries⁵⁻¹⁴</u>. When data did not appear sufficient or were inconclusive, recommendations were
- based upon expert and/or consensus opinion by experienced researchers and clinicians.
- 36

37 Background

38 Caries-risk assessment

39 Risk assessment procedures used in medical practice normally generally have sufficient data to accurately

- 40 quantitate a person's disease susceptibility and allow for preventive measures.(Lauer and Fontanarosa-
- 41 2001) However, in dentistry Even though caries risk tools data in dentistry there still is a lack of are no

42 <u>sufficiently</u> validated multivariate screen tools to determine which children are at higher risk for dental

43 caries ³⁻⁴. sufficient to validity predict individuals caries risk such procedures have value in that they:-

44 there is quantitate the models, the process of determining risk should be a component in the clinical-

- 45 decision-making process. <u>Nevertheless</u>, caries risk assessment:
- 46 1. Fosters the treatment of the disease process instead of treating the outcome of the disease.
- 47 2. Gives an understanding of the disease factors for a specific patient and aids in individualizing48 preventive discussions.
- 49 3. Individualizes, selects, and determines frequency of preventive and restorative treatment for a50 patient.
- 51 4. Anticipates caries progression or stabilization.
- 52

Caries-risk assessment models currently involve a combination of factors including diet, fluoride
exposure, a susceptible host, and microflora that interplay with a variety of social, cultural, and behavioral
factors. Caries risk assessment is the determination of the likelihood of the <u>increased</u> incidence of caries
(i.e., the number of new cavitated or incipient lesions) during a certain time period, or the likelihood that
there will be a change in the size or activity of lesions already present. With the ability to detect caries in
its earliest stages (i.e., <u>non-cavitated or</u> white spot lesions), health care providers can help prevent
cavitation ¹.

61 Caries risk indicators are variables that are thought to cause the disease directly (e.g., microflora) or have

62 been shown useful in predicting it (e.g., <u>life-time poverty</u>, low health literacy socioeconomic status) and

63 include those variables that may be considered protective factors. <u>The most commonly used caries risk</u>

- 64 indicators include presence of caries lesions, low salivary flow, visible plaque on teeth, high frequency
- 65 <u>sugar consumption</u>, presence of appliance in the mouth, health challenges, socio-demographic factors,
- 66 <u>access to care, cariogenic microflora¹</u>. Protective factors in caries risk factors include a child receiving
- 67 optimally-fluoridated water, having teeth brushed daily with fluoridated toothpaste, receiving topical
- 68 fluoride from a health professional, and a child having regular dental care¹.
- 69
- 70 Currently, there are no caries-risk factors or combinations of factors that have achieved high levels of-
- 71 both positive and negative predictive values. Some issues with the current risk indicators include:
- 72 Although the best tool to predict future caries is Past caries experience is not particularly useful in young
- raise children, and due to the importance of determining caries risk before the disease is manifest. activity of
- reasonable reasonable
- 75 not be relevant in young children¹⁵. Frequent sugar consumption is hard to quantitate. Socio-demographic
- 76 factors are just a proxy for various exposures/behaviors which may affect caries risk. Predictive ability of
- various risk factors across the life-span and how risk changes with age has not been determined¹.
- 78 <u>Furthermore, genome-level risks factors may account for substantial variation in caries risk ¹</u>. Children-
- 79 with white spot lesions should be considered at high risk for caries since these are precavitated lesions-
- 80 that are indicative of caries activity. Plaque accumulation also is strongly associated with caries-
- 81 development in young children. As a corollary to the presence of plaque, a child's Mutans Streptococci-
- 82 (MS) levels and the age at which a child becomes colonized with cariogenic flora are valuable in-
- 83 assessing risk, especially in preschool children.
- 84
- 85 While there is no question that fermentable carbohydrates are a necessary link in the causal chain for-
- 86 dental caries, a systematic study of sugar consumption and caries risk has concluded that the relationship-
- 87 between sugar consumption and caries is much weaker in the modern age of fluoride exposure than
- 88 previously thought. However, there is evidence that night-time use of the bottle, especially when it is-
- 89 prolonged, may be associated with early childhood caries. Despite the fact that normal salivary flow is an
- 90 extremely important intrinsic host factor providing protection against caries, there is little data about the
- 91 prevalence of low salivary flow in children.

- 93 Sociodemographic factors have been studied extensively to determine their effect on caries risk. Children-
- 94 with immigrant backgrounds have three times higher caries rates than non-immigrants. Most consistently,
- 95 an inverse relationship between socioeconomic status and caries prevalence is found in studies of children

- 96 less than six years of age. Perhaps another type of sociodemographic variable is the parents' history of
- 97 cavities and abscessed teeth; this has been found to be a predictor of treatment for early childhood caries.98
- 99 Teeth of children who reside in a fluoridated community have been shown to have higher fluoride content
- 100 than those of children who reside in suboptimally fluoridated communities. Additionally, both pre- and-
- 101 post-eruption fluoride exposure maximize the caries-preventive effects. For individuals residing in non-
- 102 fluoridated communities, fluoride supplements have shown a significant caries reduction in primary and
- 103 permanent teeth. With regard to fluoridated toothpaste, studies have shown consistent reduction in caries-
- 104 experience. Professional topical fluoride applications performed semiannually also reduce caries, and
- 105 fluoride varnishes generally are equal to that of other professional topical fluoride vehicles.
- 106

107 The effect of sugar substitutes on caries rates have been evaluated in several populations with high caries

- 108 prevalence. Studies indicate that xylitol can decrease MS levels in plaque and saliva and can reduce
- 109 dental caries in young children and adults, including children via their mothers. With regard to-
- 110 toothbrushing, there only is a weak relationship between frequency of brushing and decreased dental-
- 111 caries, which is confounded because it is difficult to distinguish whether the effect is actually a measure
- 112 of fluoride application or whether it is a result of mechanical removal of plaque. The dental home or
- 113 regular periodic care by the same practitioner is included in many caries risk assessment models because-
- 114 of its known benefit for dental health.
- 115

116 Risk assessment tools can aid in the identification of reliable predictors and allow dental practitioners, 117 physicians, and other nondental health care providers to become more actively involved in identifying and 118 referring high-risk children. Tables 1 and 2, and 3 incorporate available evidence into practical tools to 119 assist dental practitioners, physicians, and other non-dental health care providers in assessing levels of 120 risk for caries development in infants, children, and adolescents. As new evidence emergences, these tools 121 can be refined to provide greater predictably of caries in children prior to disease initiation. Furthermore, the evolution of caries-risk assessment tools and care pathways protocols can assist in providing evidence 122 for and justifying periodicity of services, modification of third-party involvement in the delivery of dental 123 124 services, and quality of care with outcomes assessment to address limited resources and work-force

- 125 issues.
- 126

127 <u>Care pathways for caries management Caries management protocols</u>

128 Clinical management Care pathways protocols are documents designed to assist in clinical decision-

129 making. They provide criteria regarding diagnosis and treatment and lead to recommended courses of 130 action⁶. The pathways protocols are based on evidence from current peer-reviewed literature and the 131 considered judgment of expert panels, as well as clinical experience of practitioners. Care pathways for caries management in children 0-2 and 3-5 years-old were first introduced in 2011¹⁶. The protocols 132 133 should be Care pathways are updated frequently as new technologies and evidence develop. 134 135 Historically, the management of dental caries was based on the notion that it was a progressive disease 136 that eventually destroyed the tooth unless there was surgical/restorative intervention. Decisions for 137 intervention often were learned from unstandardized dental school instruction, and then refined by 138 clinicians over years of practice. Little is known about the criteria dentists use when making decisions-139 involving restoration of carious caries lesions. 140 It is now known that surgical intervention of dental caries alone does not stop the disease process. 141 142 Additionally, many lesions do not progress, and tooth restorations have a finite longevity. Therefore, modern management of dental caries should be more conservative and includes early detection of non-143 144 cavitated lesions, identification of an individual's risk for caries progression, understanding of the disease 145 process for that individual, and active surveillance to apply preventive measures and monitor carefully for signs of arrestment or progression. 146 147 Caries management protocols Care pathways for children further refine the decisions concerning 148 149 individualized treatment and treatment thresholds based on a specific patient's risk levels, age, and 150 compliance with preventive strategies (Tables <u>3 and 4</u>, 5, 6). Such <u>clinical pathways</u> protocols should yield greater probability of success and better cost effectiveness of treatment greater probability of 151 152 success, fewer complications and more efficient use of resources than less standardized treatment ⁶. Additionally, caries management protocols free practitioners of the necessity for repetitive high level-153 154 treatment decisions, standardize decision making and treatment strategies, eliminate treatment 155 uncertainties, and guarantee more correct strategies. 156 157 Content of the present caries management protocol is based on results of clinical trials, and of systematic 158 reviews and expert panel recommendations that give better understanding of and recommendations for 159 diagnostic, preventive, and restorative treatments. Systemic fluoride protocols are based on the Centers-160 for Disease Control and Prevention's (CDC) recommendations for using fluoride.(CDC 2001); 161 Guidelines for the use of fluoridated toothpaste topical fluoride treatment are based on the three two

systematic reviews^{7,9,10}; and dietary fluoride supplements are based on the CDC's fluoride guidelines¹⁷;

- 163 professionally applied and prescription strength home-use topical fluoride are based on two systematic
- 164 reviews^{8,10}; the use of silver diamine fluoride to arrest caries lesions also is based on two systematic
- 165 <u>reviews^{11,12}</u>.
- 166
- 167 <u>Radiographic diagnostic guidelines are based on the latest uniform guidelines of three national</u>
- 168 <u>organizations⁵</u>. Guidelines for pit and fissure sealants, especially regarding primary teeth, are based on the
- 169 ADA's Council on Scientific Affairs' systematic review of recommendations for the use of pit-and-
- 170 fissure sealants¹³. Dietary interventions are based on a systematic review of strategies to reduce sugar
- 171 <u>sweetened beverages¹⁴</u>. <u>Caries risk is assessed at both the individual level and tooth level. Treatment of</u>
- 172 caries with interim therapeutic restorations is based on AAPD Best Practices^{18.19}. Guidelines for the use of
- 173 xylitol are based on the AAPD's oral health policy on use of xylitol in caries prevention, as well-executed
- 174 clinical trial on high caries-risk infants and toddlers, and two evidence based reviews. Active surveillance
- 175 (prevention therapies and close monitoring) of enamel lesions is based on the concept that treatment of
- 176 disease may only be necessary if there is disease progression²⁰, <u>and that caries can arrest without</u>
- 177 <u>treatment²¹, and that caries progression has diminished over recent decades and that the majority of</u>
- 178 proximal lesions, even in dentin, are not cavitated.
- 179

180 Other approaches to the assessment and treatment of dental caries will emerge with time and, with

- 181 evidence of effectiveness, may be included in future guidelines on caries-risk assessment and
- 182 management care pathways. protocols. For example, there are emerging trends to use calcium and
- 183 phosphate remineralizing solution to reverse dental caries. Other fluoride compounds, such as silver-
- 184 diamine fluoride and stannous fluoride, may be more effective than sodium fluoride for topical-
- 185 applications. There has been interest in antimicrobials to affect the caries rates, but evidence from caries-
- 186 trials is still inconclusive. However, some other proven methods, such as prescription fluoride drops and
- 187 tablets, may be removed from this protocol in the future due to attitudes, risks, or compliance.
- 188

189 Recommendations

- Dental caries-risk assessment, based on a child's age, <u>social/biological factors</u>, protective factors,
 and clinical findings, should be a routine component of new and periodic examinations by oral
- health and medical providers.
- 193 2. While there is not enough information at present to have quantitative caries-risk assessment194 analyses, estimating children at low, moderate, and high caries risk by a preponderance of risk and

- 195 protective factors will enable a more evidence-based approach to medical provider referrals, as well
- as establish periodicity and intensity of diagnostic, preventive, and restorative services.
- 197 3. Clinical management Care pathways protocols, based on a child's age and caries risk, and level of
- 198 patient/parent cooperation, provide health providers with criteria and protocols for determining the
- 199 types and frequency of diagnostic, preventive, and restorative care for patient-specific management
- of dental caries.
- 201
- 202

(For Physicians and Other Non-Dental Health Care Prov	viders)	
Factors	High Risk	Low Risk
Risk Factors, Social/Biological		
Mother/primary caregiver has active cavities	Yes	
Parent/caregiver has life time of poverty, low health literacy has low- socioeconomic status-	¥es	
Child has <u>frequent exposure</u> (>3 times/day) between meal sugar containing- snacks or beverages-	¥es	
<u>Continued bottle use</u> 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 <u>Factors</u>		
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	

Circling those conditions that apply to a specific patient helps the health care worker and parentunderstand the factors that contribute to or protect from caries. Risk assessment categorization of low orhigh is based on preponderance of factors for the individual. However, clinical judgment may justify theuse of one factor (e.g., frequent exposure to sugar containing snacks or beverages, visible cavities) indetermining overall risk.

Overall assessment of the child's dental caries risk: High ______

-(For Dental Providers)			
Factors	High Risk	Moderate Risk	Low Risk
Risk Factors, Social/Biological			
Mother/primary caregiver has active dental caries cavities	Yes		
Parent/caregiver <u>has life-time of poverty, low health literacy</u> has- low socioeconomic status	Yes		
Child has <u>frequent exposure</u> (>3 times/day) between meal sugar- containing snacks or beverages	Yes		
Bottle or non-spill cup Child is put to bed with a bottle containing natural or added sugar used frequently, between meals and/or at bedtime	Yes		
Child has special health care needs	<u>Yes*</u>	Yes	
Child is a recent immigrant		Yes	
Protective <u>Factors</u>			
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
<u> Clinical Findings Risk Factors, Clinical</u>			
Child has >1 decayed/missing/filled surfaces	Yes		
Child has non-cavitated (<u>incipient/</u> white spot) lesions caries or enamel defects	Yes		
Child has visible cavities or fillings or missing teeth due to caries	Yes		
Child has visible plaque on teeth	<u>Yes</u>	Yes	
* Adapted from ^{22,23}			
Circling those conditions that apply to a specific patient helps the pra factors that contribute to or protect from caries. Risk assessment cates is based on preponderance of factors for the individual. However, clin of one factor (e.g., frequent exposure to sugar-containing snacks or be determining overall risk.	gorization of nical judgme	low, moderat	te, or high the use

205 206

 ${\sf CCA.j. BP_CariesRiskAssessment}$

Factors	High Risk	Moderate Risk	Low Risk
Risk Factors, Social/Biological			
Patient <u>has life-time of poverty, low health literacy</u> is of low- socioeconomic status	Yes		
Child has <u>frequent exposure</u> (>3 times/day) between meal sugar- containing snacks or beverages	Yes		
Patient has special health care needs	Yes*	Yes	
Patient is a recent immigrant		Yes	
Protective <u>Factors</u>			
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 (eg xylitol, MI-paste, antimicrobial)			Yes
Patient has dental home/regular dental care			Yes
Clinical Findings			
Patient has ≥ 1 interproximal lesions	Yes		
Patient has active <u>non-cavitated</u> (white spot)-lesions <u>caries</u> or enamel defects	Yes		
Patient has low salivary flow	Yes		
Patient has defective restorations	Yes	Yes	
Patient wearing an intraoral appliance	Yes	Yes	
*Adapted from ^{23,24} .			

Overall assessment of the dental caries risk: High Moderate Low

Table 4. Example of a Caries Management <u>Pathways</u> Protocol for 1-2 Year Olds				
Risk-Category Diagnostics		Interventions	Restorative	
		Fluoride	Diet	
Low Risk	 Recall every six to12 months Baseline MS[#] 	 Twice daily brushing with fluoridated toothpaste^β 	Counseling	- Surveillance [‡]
Moderate risk Parent engaged	 Recall every six months Baseline MS[#] 	 Twice daily brushing with fluoridated toothpaste^β Fluoride supplements^δ Professional topical treatment every six months 	Counseling	<u>Active surveillance⁶ of</u> incipient lesions
Moderate risk Parent not engaged	 Recall every six months Baseline MS[¢] 	 Twice daily brushing with fluoridated toothpaste^β Professional topical treatment every six months 	Counseling, with- limited- expectations	<u>Active surveillance⁶ of</u> incipient lesions
High risk Parent engaged	 Recall every three months Baseline and follow up MS^{&} 	 Twice daily brushing with- fluoridated toothpaste^B Fluoride supplements³Optimize- dietary F levels Professional topical treatment- every three months SDF on cavitated lesions 	Counseling	 Active surveillance^e of incipient lesions Restore cavitated lesions-with ITR[#]-or definitive-restorations
High risk Parent not engaged	 Recall every three months Baseline and follow up MS[#] 	 Twice daily brushing with fluoridated toothpaste^B Professional topical treatment every three months 	Counseling, with limited- expectations	 Active surveillance⁶ of incipient lesions Restore cavitated lesions- with ITR[#] or definitive- restorations

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Table 3 5. Example of a Caries Management Pathways Protocol for 0 to 6 <6 3-5-Year-Olds					
Risk Category	Diagnostics	Interventions			Restorative
Kisk Category	Diagnosues	Fluoride	Diet	Sealants ²	Restorative
Low Risk	 Recall every six to12 months Radiographs every 12 to 24 months Baseline MS^{&} 	 <u>Drink optimally</u> <u>fluoridated water</u> Twice daily brushing with fluoridated toothpaste⁴ 	No <u>Yes</u>	Yes <u>No</u>	- SurveillanceX
Moderate risk Patient/Parent engaged	 Recall every six months Radiographs every six to 12 months Baseline MS^α 	 <u>Drink optimally</u> <u>fluoridated water</u> Twice daily brushing with fluoridated toothpaste? Fluoride supplements^δ Professional topical treatment every six months 	Counseling	Yes	 Active surveillance^E of <u>non-cavitated</u> (white spot) incipient lesions Restoration of cavitated or enlarging lesions
Moderate risk Parent not- engaged	 Recall every six- months Radiographs every six- to 12 months Baseline MS^G 	 Twice daily brushing with fluoridated toothpaste⁴ Professional topical treatment every six- months 	Counseling, with limited expectations	¥es	Active surveillance ⁶ - of incipient lesions Restoration of cavitated or- enlarging lesions
High risk Parent engaged	 Recall every three months Radiographs every six months Baseline and follow up-MS[#] 	 Brushing with 0.5 percent- fluoride (with caution) Twice daily brushing with fluoridated toothpaste Drink optimally fluoridated water Optimize dietary F levels Professional topical treatment every three months 	Counseling	Yes	Active surveillance ^e of <u>non-cavitated</u> (white spot) incipient lesions- <u>caries</u> SDF on cavitated <u>lesions</u> Restoration of cavitated or enlarging lesions
High risk Parent not- engaged	 Recall every three- months Radiographs every six- months Baseline and follow up- MS[#] 	 Brushing with 0.5 percent- fluoride (with caution) Professional topical- treatment every three- months 	Counseling, with limited expectations	Yes	 Restore incipient, cavitated, or enlarging lesions

213

Disk Catagory	Diagnostics	Iı	terventions		Restorative
Risk Category	Diagnostics	Fluoride	Diet	Sealants	Kestorative
Low Risk	 Recall every six to12 months Radiographs every 12 to 24 months 	 <u>Drink optimally</u> <u>fluoridated water</u> Twice daily brushing with fluoridated toothpaste[†] 	No <u>Yes</u>	Yes	- Surveillance¥
Moderate risk Patient/Parent- engaged	 Recall every six months Radiographs every six to 12 months 	 Drink optimally fluoridated water Twice daily brushing with fluoridated toothpasteμ Fluoride supplementsδ Professional topical treatment every six months 	Counseling	Yes	Active surveillance ^E of <u>non-cavitated</u> (white spot) incipient lesions <u>caries</u> Restoration of cavitated or enlarging lesions <u>caries</u>
Moderate risk Patient/Parent- not-engaged	 Recall every six- months Radiographs every six- to 12 months 	 Twice daily brushing with fluoridated toothpastet[#] Professional topical- treatment every six- months 	Counseling, with limited- expectations	Yes	Active surveillance ⁶ of incipient lesions Restoration of eavitated or- enlarging lesions
High risk Patient/Parent engaged	 Recall every three months Radiographs every six months 	 Drink optimally fluoridated water Brushing with 0.5 percent fluoride Professional topical treatment every three months SDF on cavitated lesions 	 Counseling, with limited expectations Xylitol 	Yes	Active surveillance ^E of <u>non-cavitated</u> (white spot) incipient lesions Restoration of cavitated or enlarging lesions
High risk Patient/Parent- not engaged	 Recall every three- months Radiographs every six- months 	Brushing with 0.5 percent- fluoride- Professional topical- treatment every three- months	 Counseling, with limited- expectations Xylitol 	Yes	- Restore incipient, cavitated, or enlarging lesions

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216

217 <u>Notes for caries management pathways tables:</u>

Twice daily brushing: Parental supervision of a "smear" amount of fluoridated toothpaste twice daily for

219 <u>children under age 3; "pea size" amount for children, ages 3-6.</u>

- 220 **Optimize dietary fluoride levels:** Ideally by consuming optimally fluoridated water; alternatively by
- 221 <u>dietary fluoride supplements for children at high caries risk.</u>
- 222 <u>Surveillance and active surveillance: Periodic monitoring for signs of caries progression, and active</u>
- 223 <u>measures by parents and oral health professionals to reduce cariogenic environment and monitor</u>
 224 <u>possible caries progression.</u>
- 225 SDF: Use of 38% silver diamine fluoride to assist in arresting caries lesions. Parental consent because of
 226 staining of lesions.
- **ITR:** Interim therapeutic restorations, (also may be called "protective restorations" (Policy of ITR, 2017).

228	Sealants: Although studies report unfavorable cost/benefit ratio for sealant placement in low caries risk
229	children, expert opinion favors sealants in permanent teeth of low risk children based on possible changes
230	in risk over time and differences in tooth anatomy. The decision to seal primary and permanent molars
231	should account for both the individual level and tooth level risk.
232	
233	
234	
235	Legends for Tables 4-6
236	α Salivary mutans streptococci bacterial levels. ϕ Interim therapeutic restoration.
237	(AAPD P_ITR)
238	χ Periodic monitoring for signs of caries progression. γ Parental supervision of a "pea sized"
239	amount of toothpaste.
240	β Parental supervision of a "smear" amount of toothpaste. λ Indicated for teeth with deep fissure
241	anatomy or developmental
242	δ Need to consider fluoride levels in drinking water. defects.
243	ϵ Careful monitoring of caries progression and μ Less concern about the quantity of
244	
245	

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- 1 [Best Practices]
- 2 Management of the Developing Dentition and Occlusion in
- 3 Pediatric Dentistry*
- 4
- 5 Review Council
- 6 Council on Clinical Affairs
- 7 Latest Revision
- 8 2014 2019
- 9

10 Purpose

11 The American Academy of Pediatric Dentistry (**AAPD**) recognizes the importance of managing the

12 developing dentition and occlusion and its effect on the well-being of infants, children, and adolescents.

13 Management includes the recognition, diagnosis, and appropriate treatment of dentofacial abnormalities.

14 These recommendations are intended to set forth objectives for management of the developing dentition

- 15 and occlusion in pediatric dentistry.
- 16

17 Methods

18 Recommendations on management of the developing dentition and occlusion were developed by the

19 Clinical Affairs Committee–Developing Dentition Subcommittee and adopted in 1990. This document is

20 a revision of the previous version, last revised in 2014. This revision is based upon a new

- 21 PubMed®/MEDLINE search using the terms: tooth ankylosis, Class II malocclusion, Class III
- 22 malocclusion, interceptive orthodontic treatment, evidence-based, dental crowding, ectopic eruption,
- 23 dental impaction, obstruction sleep apnea syndrome (OSAS), occlusal development, craniofacial
- 24 development, craniofacial growth, airway, facial growth, oligodontia, oral habits, occlusal wear and
- 25 dental erosion, anterior crossbite, posterior crossbite, space maintenance, third molar development, and
- tooth size/arch length discrepancy; fields: all; limits: within the last 10 years, humans, English, and birth
- through age 18. Papers for review were chosen from these searches and from references within selected
- 28 articles. When data did not appear sufficient or were inconclusive, recommendations were based upon

* ABBREVIATIONS

AAPD: American Academy Pediatric Dentistry. **AP**: Anteroposterior. **CBCT**: Cone-beam computed tomography. **EE**: Ectopic eruption. **OSAS**: Obstruction sleep apnea syndrome.

- 29 expert and/or consensus opinion by experienced researchers and clinicians.
- 30

31 Background

32 Guidance of eruption and development of the primary, mixed, and permanent dentitions is an integral 33 component of comprehensive oral health care for all pediatric dental patients. Such guidance should 34 contribute to the development of a permanent dentition that is in a stable, functional, and esthetically acceptable occlusion and normal subsequent dentofacial development. Early diagnosis and successful 35 treatment of developing malocclusions can have both short-term and long- term benefits while achieving 36 the goals of occlusal harmony and function and dentofacial esthetics.¹⁻⁴ Dentists have the responsibility to 37 38 recognize, diagnose, and manage or refer abnormalities in the developing dentition as dictated by the 39 complexity of the problem and the individual clinician's training, knowledge, and experience.⁵ 40 Many factors can affect the management of the developing dental arches and minimize the overall success 41 42 of any treatment. The variables associated with the treatment of the developing dentition that will affect 43 the degree to which treatment is successful include, but are not limited to: 1. chronological/mental/emotional age of the patient and the patient's ability to understand and 44 45 cooperate in the treatment. intensity, frequency, and duration of an oral habit. 46 2. 3. 47 parental support for the treatment. 48 4. compliance with clinician's instructions. 5. 49 craniofacial configuration. 50 6. craniofacial growth. 51 7. concomitant systemic disease or condition. 52 8. accuracy of diagnosis. 9. 53 appropriateness of treatment. 54 10. timing of treatment. 55 56 A thorough clinical examination, appropriate pretreatment records, differential diagnosis, sequential 57 treatment plan, and progress records are necessary to manage any condition affecting the developing 58 dentition. 59 60 Clinical examination should include: 61 1. Facial analysis to:

CCA.k. BP_DevelopingDentition

62		a.	identify adverse transverse growth patterns including asymmetries (maxillary and mandibular);		
63		b.	identify adverse vertical growth patterns;		
64		c.	identify adverse sagittal (anteroposterior) growth patterns and dental anteroposterior (AP)		
65			occlusal disharmonies; and		
66		d.	assess esthetics and identify orthopedic and orthodontic interventions that may improve		
67			esthetics and resultant self-image and emotional development.		
68	2.	Int	raoral examination to:		
69		a.	assess overall oral health status; and		
70		b.	determine the functional status of the patient's occlusion.		
71	3.	Fu	nctional analysis to:		
72		a.	determine functional factors associated with the malocclusion;		
73		b.	detect deleterious habits; and		
74		c.	detect temporomandibular joint dysfunction, which may require additional diagnostic		
75			procedures.		
76					
77	Diagnostic records may be needed to assist in the evaluation of the patient's condition and for				
78	documentation purposes. Prudent judgment is exercised to decide the appropriate records required for				
79	diagr	osis	s of the clinical condition. ⁶		
80					
81	Diagnostic orthodontic evaluations records fall into three major categories of evaluation: (1) health of the				
82	teeth and oral structures, (2) alignment and occlusal relationships of the teeth, and (3) facial and jaw				
83	proportions. ⁶ which includes both cephalometric radiographs and facial photographs. Digital images,				
84	including cone-beam computed tomographic images (CBCT), are supplementing and/or replacing film as-				
85	records, especially in cases of impacted teeth.				
86					
87	Diagnostic records may include:				
88	1.	Ext	traoral and intraoral photographs to:		
89		a.	supplement clinical findings with oriented facial and intraoral photographs; and		
90		b.	establish a database for documenting facial changes during treatment.		
91	2.	Dia	agnostic dental casts to:		
92		a.	assess the occlusal relationship;		
93		b.	determine arch length requirements for intraarch tooth size relationships;		
94		c.	determine arch length requirements for interarch tooth size relationships; and		

95		d. determine location and extent of arch asymmetry.			
96	3.	Intraoral and panoramic radiographs to:			
97		a. establish dental age;			
98		b. assess eruption problems;			
99		c. estimate the size and presence of unerupted teeth; and			
100		d. identify dental anomalies/pathology.			
101	4.	Lateral and AP cephalograms to:			
102		a. produce a comprehensive cephalometric analysis of the relative dental and skeletal components			
103		in the anteroposterior, vertical, and transverse dimensions;			
104		b. establish a baseline growth record for longitudinal assessment of growth and displacement of			
105		the jaws; and			
106		c. determine dental maturity relative to skeletal maturity and chronological age.			
107	5.	Other diagnostic views (e.g., magnetic resonance imaging, CBCT) for hard and soft tissue imaging			
108		as indicated by history and clinical examination.			
109					
110	A di	A differential diagnosis and diagnostic summary are completed to:			
111	1.	Establish the relative contributions of the soft tissue and dental and skeletal structures			
112		to the patient's malocclusion.			
113	2.	Prioritize problems in terms of relative severity.			
114	3.	Detect favorable and unfavorable interactions that may result from treatment options for each			
115		problem area.			
116	4.	Establish short-term and long-term objectives.			
117	5.	Summarize the prognosis of treatment for achieving stability, function, and esthetics.			
118					
119		quential treatment plan will:			
120	1.	Establish timing priorities for each phase of therapy.			
121	2.	Establish proper sequence of treatments to achieve short-term and long-term objectives.			
122	3.	Assess treatment progress and update the biomechanical protocol accordingly on a regular basis.			
123	~				
124	Stages of development of occlusion				
125		eral considerations and principles of management: The stages of occlusal development include:			
126	1.	Primary dentition: Beginning in infancy with the eruption of the first tooth, usually about six			
127		months of age, and complete from approximately three to six years of age when all primary teeth			

128 are erupted. 129 2. Mixed dentition: From approximately age six to 13, primary and permanent teeth are present in the 130 mouth. This stage can be further divided into early mixed and late mixed dentition. 3. Adolescent dentition: All primary teeth have exfoliated, All succedaneous teeth have erupted, 131 132 second permanent molars may be erupted or erupting, and third molars have not erupted. 4. Adult dentition: All permanent teeth are present.⁷⁻¹⁰ and eruptive growth is complete. 133 134 These stages may further be divided and referenced as early and late (e.g., early primary, late primary, 135 136 early mixed, late mixed).(Dale and Dale 2012, Ferguson 2011, McNamara and Brudon 2001, Proffit 137 2012-Later stages-138 139 Evaluation and treatment of occlusal and skeletal disharmonies may be initiated at various stages of dental arch development, depending on the problems, growth, parental involvement, risks and benefits of 140 141 treatment and of deferring treatment.(AAPD P_Ethics)-142 143 Historically, orthodontic treatment was provided mainly for adolescents. Interest continues to be 144 expressed in the concept of interceptive (early) treatment as well as in adult treatment. Treatment and timing options for the growing patient, especially in the mixed dentition and early permanent dentition, 145 have increased and continue to be evaluated by the research community.^{9,11,12} Many clinicians seek to 146 modify skeletal, muscular, and dentoalveolar abnormalities before the eruption of the full permanent 147 148 dentition.7 149 150 A thorough knowledge of craniofacial growth and development of the dentition, as well as orthodontic 151 treatment, must be used in diagnosing and reviewing possible interceptive treatment options before 152 recommendations are made to parents.⁹ Treatment is beneficial for many children, but may not be 153 indicated for every patient with a developing malocclusion. 154 **Treatment considerations:** The developing dentition should be monitored throughout eruption. This 155 156 monitoring at regular clinical examinations should include, but not be limited to, diagnosis of missing, 157 supernumerary, developmentally defective, and fused or geminated teeth; ectopic eruption; space and tooth loss secondary to caries and periodontal and pulpal health of the teeth. 158 159 Radiographic examination, when necessary¹³ and feasible, should accompany clinical examination. 160

161	Diag	nosis of anomalies of primary or permanent tooth development and eruption should be made to
162	infor	m the patient's parent and to plan and recommend appropriate intervention. This evaluation is on-
163	going	g throughout the developing dentition, at all stages. ⁶⁻¹⁰
164	1.	Primary dentition stage: Anomalies of primary teeth and eruption may not be evident/diagnosable
165		prior to eruption, due to the child's not presenting for dental examination or to a radiographic
166		examination not being possible in a young child due to age or behavior. Evaluation, however,
167		should be accomplished when feasible. The objectives of evaluation include identification of:
168		a. all anomalies of tooth number and size (as previously noted);
169		b. anterior and posterior crossbites;
170		c. presence of habits along with their dental and skeletal sequelae;-and
171		d. airway problems.
172		e <u>. openbite</u>
173		Radiographs are taken with appropriate clinical indicators or based upon risk assessment/history.
174	2.	Early mixed dentition stage: The objectives of evaluation continue as noted for the primary
175		dentition stage. Palpation for unerupted teeth should be part of every examination. Panoramic,
176		occlusal, and periapical radiographs, as indicated at the time of eruption of the lower incisors and
177		first permanent molars, provide diagnostic information concerning:
178		a. unerupted teeth;
179		b. missing, supernumerary, fused, and geminated teeth;
180		c. tooth size and shape (e.g., peg or small lateral incisors);
181		d. positions (e.g., ectopic first permanent molars);
182		e. developing skeletal discrepancies; and
183		f. periodontal health.
184		Space analysis can be used to evaluate arch length /crowding at the time of incisor eruption.
185	3.	Mid-to-Late mixed dentition stage: The objectives of the evaluations remain consistent with the
186		prior stages, with an emphasis on evaluation for ectopic tooth positions, especially canines,
187		bicuspids, and second permanent molars.
188	4.	Adolescent dentition stage: If not instituted earlier, orthodontic diagnosis and treatment should be
189		planned for Class I crowded, Class II, and Class III malocclusions as well as posterior and anterior
190		crossbites. Third molars should be monitored as to position and space, and parents should be
191		informed of the dentist's observations.
192	5.	Early adult dentition stage: Third molars should be evaluated. If orthodontic diagnosis has not been
193		accomplished, recommendations should be made as necessary.

194

194		
195	Trea	tment objectives: At each stage, the objectives of intervention/treatment include: managing adverse
196	grow	th, correcting dental and skeletal disharmonies, improving esthetics of the smile and the
197	acco	mpanying positive effects on self-image, and improving the occlusion.
198	1.	Primary dentition stage: Habits and crossbites should be diagnosed and, if predicted not likely to be
199		self-correcting, they should be addressed as early as feasible to facilitate normal occlusal
200		relationships. Parents should be informed about findings of adverse growth and developing
201		malocclusions. Interventions/ treatment can be recommended if diagnosis can be made, treatment is
202		appropriate and possible, and parents are supportive and desire to have treatment done.
203	2.	Early-to-mid mixed dentition stage: Treatment consideration should address:
204		a. habits;
205		b. arch length shortage;
206		c. intervention for crowded incisors;
207		d. intervention for ectopic teeth;
208		e. holding of leeway space;
209		f. crossbites <u>:</u>
210		g. openbite;
211		g. <u>h.</u> surgical needs; and
212		h. <u>i.</u> adverse skeletal growth.
213		Intervention for ectopic teeth may include extractions of primary teeth and space
214		maintenance/regaining to aid eruption of succedaneous erupting teeth and reduce the risk of the
215		need for permanent tooth extraction or surgical bracket placement for orthodontic traction.
216		Treatment should take advantage of high rates of growth the child's growth and should be aimed at
217		prevention of adverse dental relationships and skeletal growth.
218	3.	Late mixed dentition stage: Intervention for treatment of skeletal disharmonies and crowding may
219		be instituted at this stage.
220	4.	Adolescent dentition stage: In full permanent dentition, final orthodontic diagnosis and treatment
221		can to provide the most functional, stable, and esthetic occlusion.
222	5.	Early adult dentition stage: Third molar position or space can be evaluated and, if indicated, the
223		tooth/ teeth removed. Full orthodontic treatment should be recommended if needed.
224		

- 225 Recommendations
- 226 Oral habits

227 General considerations and principles of management: The habits of nonnutritive sucking, bruxing,

tongue thrust swallow and abnormal tongue position, self-injurious/self-mutilating behavior, and OSAS

- are discussed in this guideline these recommendations.
- 230

Oral habits may apply <u>negative</u> forces to the teeth and dentoalveolar structures. The relationship between oral habits and unfavorable dental and facial development is associational rather than cause and effect.¹⁴⁻¹⁶ Habits of sufficient frequency, duration, and intensity may be associated with dentoalveolar or skeletal deformations such as increased overjet, reduced overbite, <u>openbite</u>, posterior crossbite, or <u>increased long</u> facial height. The duration of force is more important than its magnitude; the resting pressure from the lips, cheeks, and tongue has the greatest impact on tooth position, as these forces are maintained most of the time.^{17,18}

238

239 Nonnutritive sucking behaviors are considered normal in infants and young children. Prolonged-

240 nonnutritive sucking habits have been associated with decreased maxillary arch width, increased overjet,

241 decreased overbite, anterior open bite, and posterior crossbite. Long term non-nutritive sucking habits

242 (e.g., pacifier use, thumb/finger sucking) have been associated with anterior openbite and posterior

243 <u>crossbite.^{15,17-21} Some As preliminary</u> evidence indicates that some changes resulting from sucking habits

244 persist past the cessation of the habit; therefore, it has been suggested that early dental visits provide

245 parents with anticipatory guidance to help their children stop sucking habits by age 36 months or

- **246** younger.^{15,17,18}
- 247

248 Bruxism, defined as the habitual nonfunctional and forceful contact between occlusal surfaces, can occur 249 while awake or asleep. The etiology is multifactorial and has been reported to include central factors (e.g., emotional stress,²² parasomnias,²³ traumatic brain injury,²⁴ neurologic disabilities²⁵) and morphologic 250 factors (e.g., malocclusion,²⁶ muscle recruitment²⁷). The occlusal wear that may result from bruxism is 251 252 important to differentiate from other forms of occlusal loss of enamel (e.g., erosion caused by diet or 253 gastroesophageal reflux).²⁸ Reported complications of bruxism include dental attrition, headaches, 254 temporomandibular dysfunction, and soreness of the masticatory muscles.²² Evidence indicates that juvenile bruxism is self-limiting and does not persist in adults.²⁹ The spectrum of bruxism management 255 256 ranges from patient/parent education, occlusal splints, and psychological techniques to

257 medications.^{22,24,30,31}

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258

259 Tongue thrusting, an abnormal tongue position and deviation from the normal swallowing pattern, may be 260 associated with anterior open bite, abnormal speech, and anterior protrusion of the maxillary 261 incisors.³²There is no evidence that intermittent short-duration pressures, created when the tongue and lips contact the teeth during swallowing or chewing, have significant impact on tooth position.^{17,32} If the 262 resting tongue posture is forward of the normal position, incisor displacement is likely, but if resting 263 264 tongue posture is normal, a tongue thrust swallow has no clinical significance.¹⁷ 265 266 Self-injurious or self-mutilating behavior (i.e., repetitive acts that result in physical damage to the individual) is extremely rare in the normal child.³³ Such behavior, however, has been associated with 267 developmental delay or disabilities, psychiatric disorders, traumatic brain injuries, and some 268 269 syndromes.^{33,34} The spectrum of treatment options for developmentally disabled individuals includes pharmacologic management, behavior modification, and physical restraint.³⁵ Reported dental treatment 270 271 modalities include, among others, lip-bumper and occlusal bite appliances, protective padding, and extractions.³³ Some habits, such as lip-licking and lip-pulling, are relatively benign in relation to an effect 272 on the dentition.³³ More severe lip- and tongue-biting habits may be associated with profound 273 neurodisability due to severe brain damage.³⁵ Management options include monitoring the lesion, 274 275 odontoplasty, providing a bite-opening appliance, or extracting the teeth.³⁵ 276 277 Research on the relationship between malocclusion and mouth breathing suggests that impaired nasal 278 respiration may contribute to the development of increased facial height, anterior open bite, increased 279 overjet, and narrow palate, but it is not the sole or even the major cause of these conditions.³⁶ 280 281 OSAS may be associated with narrow maxilla, crossbite, low tongue position, vertical growth, increased overjet and open bite.³⁷⁻³⁹ History associated with OSAS may include snoring, observed apnea, restless 282 sleep, daytime neurobehavioral abnormalities or sleepiness, and bedwetting. Physical findings may 283 include growth abnormalities, signs of nasal obstruction, adenoidal facies, and/ or enlarged tonsils.^{36,40,41} 284 285 286 The identification of an abnormal habit and the assessment of its potential immediate and long-term 287 effects on the craniofacial complex and dentition should be made as early as possible. The dentist should evaluate habit frequency, duration, and intensity in all patients with habits. Intervention to terminate the 288 289 habit should be initiated if indicated and parents should be provided with information regarding consequences of a habit and tools to help in elimination of the habit.^{15,16} 290

291

- 292 Patients and their parents should be provided with information regarding consequences of a habit. Parents-293 may play a negative role in the correction of an oral habit as nagging or punishment may result in an 294 increase in habit behaviors; change in the home environment may be necessary before a habit can be 295 overcome.(Warren et al 2001) 296 297 Treatment considerations: Management of an oral habit is indicated whenever the habit is associated 298 with unfavorable dentofacial development or adverse effect on child health or when there is a reasonable 299 indication that the oral habit will result in unfavorable sequelae in the developing permanent dentition. 300 Any treatment must be appropriate for the child's development, comprehension, and ability to cooperate. 301 Habit treatment modalities include patient/parent counseling, behavior modification techniques, 302 myofunctional therapy, appliance therapy (extraoral and/or intraoral), or referral to other providers including, but not limited to, orthodontists, psychologists, myofunctional therapists, or otolaryngologists. 303 304 Use of an appliance to manage oral habits is indicated only when the child wants to stop the habit and would benefit from a reminder. The child's desire to stop the habit is beneficial for managing oral habits.¹⁶ 305 306 307 **Treatment objectives:** Treatment is directed toward decreasing or eliminating the habit and minimizing 308 potential deleterious effects on the dentofacial complex. 309 **Disturbances in number** 310 311 Congenitally missing teeth General considerations and principles of management: Hypodontia, the congenital absence of one or 312 more permanent teeth, has a prevalence of 3.5 to 6.5 percent.⁴² Excluding third molars, the most 313 frequently missing permanent tooth is the mandibular second premolar followed by the maxillary lateral 314 incisor.⁴² In the primary dentition, hypodontia occurs less (0.1 percent to 0.9 percent prevalence) and 315
- almost always affects the maxillary incisors and first primary molars.⁴³ The chance of familial occurrence
- of one or two congenitally missing teeth is to be differentiated from missing lateral incisors in cleft
- 118 lip/palate⁴⁴ and multiple missing teeth (six or more) due to ectodermal dysplasia or other syndromes⁴⁵ as
- the treatment usually differs. A congenitally missing tooth should be suspected in patients with cleft
- 320 lip/palate, certain syndromes, and a familial pattern of missing teeth. In addition, patients with
- 321 asymmetric eruption sequence, over-retained primary teeth, or ankylosis of a primary mandibular second
- 322 molar may have a congenitally missing tooth.^{44,46,47}
- 323

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Treatment considerations: With congenitally missing permanent maxillary incisor(s) or mandibular 324 325 second premolar(s), the decision to extract the primary tooth and close the space orthodontically versus 326 opening the space orthodontically and placing a prosthesis or implant depends on many factors. For maxillary laterals, the dentist may move the maxillary canine mesially and use the canine as a lateral 327 incisor or create space for a future lateral prosthesis or implant.^{16,48} 328 329 Factors that influence the decision are: (1) patient age; (2) canine size and shape; (3) canine position; (4) 330 child's occlusion and amount of crowding; (5) bite depth; and (6) profile (7) smile line and (8) quality and 331 quantity of bone in the edentulous area.^{48,49} Early extraction of the primary canine and/or lateral may be 332 needed.⁴⁸ Opening space for a prosthesis or implant requires less tooth movement, but the space needs to 333 be maintained with an interim prosthesis, especially if an implant is planned.^{45,48} Moving the canine into 334 335 the lateral position produces little facial change, but the resultant tooth size discrepancy often does not allow a canine guided occlusion.^{47,48} Parents generally prefer space closure over implants⁴⁹ 336 337 For a congenitally missing premolar, the primary molar either may either be maintained or extracted with 338 subsequent placement of a prosthesis, or autotransplantation, ⁵⁰⁻⁵² followed by orthodontic treatment or the 339 primary molar may be extracted and the space closed orthodontically.^{48,53,54} orthodontically orthodontic-340 341 space closure closing the space. Maintaining the primary second molar may cause occlusal problems due to its larger mesiodistal diameter, compared to the second premolar.⁴⁸ Reducing the width of the second 342 primary molar is a consideration, but root resorption and subsequent exfoliation may occur.^{16,48} In 343 344 crowded arches or with multiple missing premolars, extraction of the primary molar(s) can be considered, especially in mild Class III cases.^{16,48,53} For a single missing premolar, if maintaining the primary molar is 345 not possible, placement of a prosthesis, autotransplantation or implant should be considered.^{16,49,53} 346 Preserving the primary tooth may be indicated in certain cases. However, maintaining a 347 348 submerged/ankylosed tooth may increase the likelihood of an alveolar defect which can compromise later implant success.^{53,54} Consideration for extraction and space maintenance may be indicated.^{53,54} 349 350 Consultation with an orthodontist and/or prosthodontist may be considered. 351 Treatment objectives: Treatment is directed toward an esthetically pleasing occlusion that functions well 352 353 for the patient. 354 355 *Supernumerary teeth (primary, permanent, and mesiodens)* 356 General considerations and principles of management: Supernumerary teeth, or hyperdontia, can

- 357 occur in the primary or permanent dentition but are five times more common in the permanent.⁵⁵
- Prevalence is reported in the primary <u>dentition from 0.3-0.8%</u> and mixed dentitions from 0.52 to 23
- percent.⁵⁵⁻⁵⁸ Between 80 and 90 percent of all supernumeraries occur in the maxilla, with half in the
- 360 anterior area and almost all in the palatal position.⁵⁵ A supernumerary primary tooth is followed by a
- 361 supernumerary permanent tooth in one third of the cases.⁵⁹ Supernumerary teeth are classified according
- to their form and location.^{55,60}
- 363

364 During the early mixed dentition, 79 to 91 percent of anterior permanent supernumerary teeth are unerupted.^{47,56} While more erupt with age, only 25 percent of all mesiodens (a permanent supernumerary 365 incisor located at the midline) erupt spontaneously.⁵⁵ Mesiodens can prevent or cause ectopic eruption of 366 367 a central incisor. Less frequently, a mesiodens can cause dilaceration or resorption of the permanent 368 incisor's root. Dentigerous cyst formation involving the mesiodens, in addition to eruption into the nasal cavity, has been reported.⁵⁵ If there is an asymmetric eruption pattern of the maxillary incisors, delayed 369 370 eruption, an over-retained primary incisor, or ectopic eruption of an incisor, a supernumerary can be suspected.^{43,44,56} Panoramic, occlusal, and periapical radiographs all can reveal a supernumerary tooth-371 the best way to locate the supernumerary is two periapical or occlusal films reviewed by the parallax-372 rule.(Russell and Folwarczna 2003). To determine the supernumerary tooth's position either a cone beam 373 radiograph or two periapical or occlusal films reviewed by the parallex rule are recommended. 55,57 374

375

376 **Treatment considerations:** Management and treatment of hyperdontia differs if the tooth is primary or 377 permanent. Primary supernumerary teeth normally are accommodated into the arch and usually erupt and exfoliate without complications.⁵⁹ Extraction of an unerupted supernumerary tooth during the primary-378 379 dentition usually is not done to allow it to erupt; Surgical extraction of unerupted supernumerary teeth during the primary dentition can displace or damage the permanent incisor.⁵⁵ Removal of an erupted 380 381 mesiodens or other permanent supernumerary incisor results in eruption of the permanent adjacent normal incisor in 75 percent of the cases.⁵⁵ Extraction of an unerupted supernumerary during the early mixed 382 dentition, at age 6-7 when the permanent crown has completely formed and the root length is less than the 383 crown height allows for a normal eruptive force and eruption of the adjacent normal permanent incisor.⁵⁵⁻ 384 ^{57,61} Later removal of the mesiodens reduces the likelihood that the adjacent normal permanent incisor 385 will erupt on its own, especially if the apex is completed.⁵⁵ Inverted conical supernumeraries can be 386 harder to remove if removal is delayed, as they can migrate deeper into the jaw.⁵⁶ After removal of the 387 388 supernumerary, clinical and radiographic follow-up is indicated in six months to determine if the normal 389 incisor is erupting. If there is no eruption after six to 12 months and sufficient space exists, surgical

- 390 exposure and orthodontic extrusion is may be needed. 55,62,63
- 391
- 392 Treatment objectives: Removal of supernumerary teeth should facilitate eruption of permanent teeth and
- 393 encourage normal alignment. In cases where normal alignment or spontaneous eruption does not occur,
- 394 further orthodontic treatment is indicated.
- 395

396 Localized disturbances in eruption

- 397 *Ectopic eruption*
- **398** General considerations and principles of management: Ectopic eruption (EE) of permanent first
- 399 molars occurs due to the molar's abnormal mesioangular eruption path, resulting in an impaction at the
- 400 distal prominence of the primary second molar's crown.⁶⁴⁻⁶⁵ EE can be suspected if asymmetric eruption
- 401 is observed or if the mesial marginal ridge is noted to be under the distal prominence of the second
- 402 primary molar.⁶⁴⁻⁶⁵
- 403
- 404 Ectopic Eruption of Molars
- EE of permanent molars can be diagnosed from bitewing or panoramic radiographs in the early mixed
- 406 dentition.^{64,65} This condition occurs in up to three percent of the population.^{64,65} This condition permanent
- 407 first molar eruption has been associated with transverse and sagittal crowding, is more common in the
- 408 <u>maxillary arch, and in children with cleft lip and palate.⁶⁵⁻⁶⁷ Ectopic eruption of permanent second molars</u>
- 409 occur infrequently.⁶⁸ EE of permanent molars is classified into two types. There are those that self-correct
- 410 and others that remain impacted. Previous data suggested Ssixty-six percent of EE permanent molars self-
- 411 <u>correct by age seven;^{47,65}- however, a recent cohort study demonstrated that 71% self-correct by age</u>
- 412 <u>nine.⁶⁹ Increased magnitude of impaction is the most reliable predictor of irreversible impaction (Dabbagh</u>
- 413 <u>2017</u>). In some cases, definitive treatment is indicated to manage and/or avoid early loss of the primary
- 414 second molar and space loss.^{64,65} Increase magnitude of impaction, increased resorption of the primary
- 415 tooth and bilateral occurrence were positively associated with irreversible ectopic eruption and may
- 416 indicate the need for early intervention.⁶⁹
- 417
- 418 <u>**Treatment considerations:**</u> Treatment depends on how severe the impaction appears clinically and
- 419 <u>radiographically. For mildly impacted first permanent molars, where little of the tooth is impacted under</u>
- 420 the primary second molar, elastic or metal orthodontic separators can be placed to wedge the permanent
- 421 first molar distally.⁶⁴ For more severe impactions, distal tipping of the permanent molar is required.⁶⁴
- 422 <u>Tipping action can be accomplished with brass wires, removable appliances using springs, fixed</u>

- 423 <u>appliances such as sectional wires with open coil springs,⁷⁰ sling shot-type appliances,⁷¹ or a Halterman</u>
- 424 <u>appliance.⁷²</u>
- 425
- 426 Ectopic Eruption of Canines
- 427 The maxillary canine appears in an impacted position in 1.5 2 percent of the population, $\frac{73}{3}$ while
- 428 <u>Maxillary canine impaction should be suspected when the canine bulge is not palpable, asymmetric</u>
- 429 <u>canine eruption is evident, or maxillary lateral incisor agenesis or peg shaped lateral incisors are</u>
- 430 present.⁷³⁻⁷⁷ Panoramic radiographs may demonstrate that the canine has an abnormal inclination and/or
- 431 overlaps the lateral incisor root. Additional potential radiographic signs of maxillary canine impaction
- 432 <u>include: enlarged follicular sac, lack of root resorption of primary canines, and presence of premolar</u>
- 433 <u>impaction.^{75,76,78}</u>
- 434
- 435 <u>Treatment Considerations: Early diagnosis and treatment of impacted maxillary canines can lessen the</u>
- 436 severity of the impaction and may stimulate eruption of the canine. Extraction of the primary canine is
- 437 <u>indicated when the canine bulge cannot be palpated in the alveolar process and there is radiographic</u>
- 438 <u>overlapping of the canine with the formed root of the lateral during the mixed dentition.</u>^{73,79,80} The use of
- 439 <u>rapid maxillary expansion alone^{81,82} or with cervical pull headgear⁸³ in the early mixed dentition has been</u>
- 440 shown to increase the potential for eruption of palatally displaced maxillary cuspids. When the impacted
- 441 <u>canine is diagnosed at a later age (11 to 16-years-old), if the canine is not horizontal, extraction of the</u>
- 442 primary canine lessens the severity of the permanent canine impaction and 75 percent will erupt.⁸⁴
- 443 Extraction of the first primary molar also has been reported to allow eruption of first bicuspids and to
- 444 <u>assist in the eruption of the cuspids.⁸⁵ The need can be determined from a panoramic radiograph,^{86,87}</u>
- 445 <u>although CBCT will provide greater localization of the impacted canine.⁸⁸ Bonded orthodontic treatment</u>
- 446 <u>normally is required to create space or align the canine. Long-term periodontal health of impacted canines</u>
- 447 after orthodontic treatment is similar to nonimpacted canines and there is insufficient data to conclude the
- 448 <u>best type of surgical technique.^{89,90}</u>
- 449
- 450 <u>Ectopic Eruption of Incisors</u>
- 451 <u>mMaxillary incisors can erupt ectopically or be impacted from supernumerary teeth in up to two percent</u>
- 452 of the population.⁶⁰ Incisors also can have altered eruption due to pulp necrosis (following trauma or
- 453 caries) or pulpal treatment of the primary incisor.⁹¹ <u>EE of permanent incisors can be suspected after</u>
- 454 trauma to primary incisors, with pulpally-treated primary incisors, with asymmetric eruption, or if a
- 455 <u>supernumerary incisor is diagnosed.</u>^{73,77}

116

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456

456	
457	Treatment Considerations: Treatment of ectopically erupting incisors depends on the etiology.
458	Extraction of necrotic or over-retained pulpally-treated primary incisors is indicated in the early mixed
459	dentition.91 Removal of supernumerary incisors in the early mixed dentition will lessen ectopic eruption
460	of an adjacent permanent incisor. ⁵⁵ After incisor eruption, orthodontic treatment involving removable or
461	banded therapy may be needed.
462	
463	EE of permanent molars is classified into two types. There are those that self-correct and others that
464	remain impacted. Sixty six percent of EE permanent molars self-correct by age seven:.(Robertson and-
465	Mohlin 2000, Barberia Leache et al 2005) A permanent molar that presents with part of its occlusal-
466	surface clinically visible and part under the distal of the primary second molar usually does not self-
467	correct and is the impacted type.(Yaseen et al 2011, Barberia Leache et al 2005) After the age of seven,
468	definitive treatment is indicated to manage and/or avoid early loss of the primary second molar and space
469	loss.(Yaseen et al 2011, Barberia-Leache et al 2005)
470	
471	Maxillary canine impaction should be suspected when the canine bulge is not palpable, asymmetric
472	canine eruption is evident, or peg shaped lateral incisors are present. (Richardson and Russell 2000,
473	Sachan and Chatunedi 2012) Panoramic radiographs may demonstrate that the canine has an abnormal-
474	inclination and/or overlaps the lateral incisor root. EE of permanent incisors can be suspected after trauma
475	to primary incisors, with pulpally treated primary incisors, with asymmetric eruption, or if a
476	supernumerary incisor is diagnosed. (Richardson and Russell 2000, Sachan and Chatunedi 2012)
477	
478	Treatment considerations: Treatment depends on how severe the impaction appears clinically and
479	radiographically. For mildly impacted first permanent molars, where little of the tooth is impacted under-
480	the primary second molar, elastic or metal orthodontic separators can be placed to wedge the permanent
481	first molar distally.(Warren et al 2001, Yaseen et al 2011) For more severe impactions, distal tipping of-
482	the permanent molar is required.(Yaseen et al 2011) Tipping action can be accomplished with brass wires,
483	removable appliances using springs, fixed appliances such as sectional wires with open coil-
484	springs <u>(Seerah 2011)</u> , sling shot type appliances,(Gehm and Crespi 1997) or a Halterman
485	appliance,(Halterman 1982) or surgical uprighting.(Terry and Hegtvedt 1992)
486	
487	Early diagnosis and treatment of impacted maxillary canines can lessen the severity of the impaction and
488	may stimulate eruption of the canine. Extraction of the primary canine is indicated when the canine bulge

- 489 cannot be palpated in the alveolar process and there is radiographic overlapping of the canine with the
- 490 formed root of the lateral during the mixed dentition.(Richardson and Russell 2000, Bedoya and Park-
- 491 2009, Litsas and Acar 2011) The use of rapid maxillary expansion in the early mixed dentition has been
- 492 shown to increase the rate of eruption of palatally displaced maxillary cuspidsWhen the impacted canine-
- 493 is diagnosed at a later age (11 to 16), if the canine is not horizontal, extraction of the primary canine-
- 494 lessens the severity of the permanent canine impaction and 75 percent will erupt.(Olive 2002) Extraction-
- 495 of the first primary molar also has been reported to allow eruption of first bicuspids and to assist in the
- 496 eruption of the cuspids. The need can be determined from a panoramic radiograph. (D'Amico et al 2003,
- 497 Bonetti et al 2011). Bonded orthodontic treatment normally is required to create space or align the canine.
- 498 Long-term periodontal health of impacted canines after orthodontic treatment is similar to nonimpacted-
- 499 canines (Andreasen and Andreasen 1994-Avulsion injuries).
- 500
- 501 Treatment of ectopically erupting incisors depends on the etiology. Extraction of necrotic or over retained
- 502 pulpally-treated primary incisors is indicated in the early mixed dentition. (Coll and Sadrian 1996)
- 503 Removal of supernumerary incisors in the early mixed dentition will lessen ectopic eruption of an-
- 504 adjacent permanent incisor. (Russell and Folwarczna 2003) After incisor eruption, orthodontic treatment-
- 505 involving removable or banded therapy may be needed.
- 506

507 Treatment objectives: Management of ectopically erupting molars, canines, and incisors should result in
 508 improved eruptive positioning of the tooth. In cases where normal alignment does not occur, subsequent
 509 comprehensive orthodontic treatment may be necessary to achieve appropriate arch form and

- 510 intercuspation.
- 511
- 512 *Ankylosis*

General considerations and principles of management: Ankylosis is a condition in which the cementum of a tooth's root fuses directly to the surrounding bone.⁹² The periodontal ligament is replaced with osseous tissue, rendering the tooth immobile to eruptive change.⁹² An <u>ankylosed tooth stays at the same vertical level; however, in a growing child appears to submerge as the other teeth continue to erupt.⁹³ Ankylosis can occur in the primary and permanent dentitions, with the most common incidence involving primary molars. The incidence is reported to be between seven and 14 percent in the primary dentition.⁹⁴ In the permanent dentition, ankylosis occurs most frequently following luxation injuries.^{95,96}</u>

521 Ankylosis is common in anterior teeth following trauma and is referred to as replacement resorption.

522	Periodontal ligament cells are destroyed and the cells of the alveolar bone perform most of the healing.
523	Over time, normal bony activity results in the replacement of root structure with osseous tissue.94,95
524	Ankylosis can occur rapidly or gradually over time, in some cases as long as five years post trauma. It
525	also may be transient if only a small bony bridge forms then is resorbed with subsequent osteoclastic
526	activity. ⁹⁷
527	
528	Ankylosis can be verified by clinical and radiographic means. Submergence of the tooth is the primary
529	recognizable sign, but the diagnosis also can be made through percussion and palpation. ⁹⁸ Radiographic
530	examination also may reveal the loss of the periodontal ligament and bony bridging.92
531	
532	Treatment considerations: With ankylosis of a primary molar, exfoliation usually occurs normally.
533	Extraction is recommended if prolonged retention of the primary molar is noted. If a severe marginal
534	ridge discrepancy develops, extraction should be considered to prevent the adjacent teeth from tipping
535	and producing space loss ^{3,98} or vertical occlusal discrepancies. ⁹⁹ Replacement resorption of permanent
536	teeth usually results in the loss of the involved tooth. ⁹⁴
537	
538	Mildly to moderately ankylosed primary molars without permanent successors may be retained and
539	restored to function in arches without crowding. ⁹⁹ Extraction of these molars can assist in resolving
540	crowded arches in complex orthodontic cases. ¹⁰⁰ Surgical luxation of ankylosed permanent teeth with
541	forced orthodontic eruption has been described as an alternative to premature extraction. ^{101,102}
542	
543	Treatment objectives: Treatment of ankylosis should result in the continuing normal development of the
544	permanent dentition. In the case of replacement resorption of a permanent tooth, appropriate prosthetic
545	replacement should be planned.
546	
547	
548	Primary failure of eruption
549	
550	General considerations and management: Primary failure of eruption (PFE) is an eruption disorder
551	characterized by partial or complete non-eruption of permanent teeth in the absence of any mechanical
552	obstruction or syndrome. ¹⁰³ Failure in eruptive mechanisms prevent permanent successors from following
553	the eruption path after the exfoliation of deciduous teeth. ¹⁰⁴ Posterior teeth are most commonly affected
554	and one or all four quadrants may be involved ¹⁰⁵ Although typically associated with permanent teeth,

555	examples in the primary dentition have been noted. ¹⁰⁶ Two main phenotypes of PFE have been identified:
556	(1) All teeth distal to the most mesial non-erupted tooth are affected, or (2) unerupted teeth do not follow
557	the pattern that all teeth distal to the most mesial involved tooth are also affected. ¹⁰⁷ Hallmark features of
558	PFE include: posterior open bite in the presence of normal vertical growth, infraocclusion of affected
559	teeth, and the inability to move affected teeth orthodontically. ¹⁰⁸
560	
561	The reported incidence of PFE is between 0.01 and 0.06 percent; ^{109,110} however, some data suggests PFE
562	may be misdiagnosed as infra-occlusion or ankylosis. ^{111,112} PFE differs from ankylosis in that eruption
563	fails to occur due to an imbalance in resorptive and appositional factors related to tooth eruption. ^{113,114}
564	Teeth with PFE are not initially ankylosed but may become ankylosed when orthodontic forces are
565	applied. ¹¹⁵
566	
567	A systematic review demonstrated 85% of patients with PFE have another family member with the
568	condition. ¹⁰⁵ PFE has variable expression and has been associated with mutations in the autosomal
569	dominant parathyroid hormone receptor (PTH1R) gene. ¹¹⁵⁻¹¹⁸ A DNA sample of blood or saliva can be
570	used to test for mutations in PTH1R. ^{117,119}
571	
572	Treatment considerations: Diagnosis of PFE should be based on a combination of clinical, radiographic,
573	and genetic information. ¹¹³⁻¹¹⁵ A positive family history also supports a diagnosis of PFE. ¹⁰⁷ Other than a
574	few anecdotal reports, PFE is strongly associated with the failure of orthodontically assisted eruption or
575	tooth movement. ^{113,114} To that point early orthodontic intervention of the affected teeth should be
576	avoided. ^{108,113,114,119} To date there are no established mechanotherapeutic methods of modifying
577	dentoalveolar growth for these patients. ^{108,113,114,119} Space maintenance, up-righting adjacent teeth that
578	have tipped into the sites, prevention of supra-eruption in opposing arch or modification of lateral tongue
579	thrust habits may be additional considerations. ^{108,119} Once growth is complete, multidisciplinary treatment
580	options such as single tooth or segmental osteomies with immediate traction, or selective extractions
581	followed by implants can be considered to create a functioning occlusion. ¹⁰⁸ Early extraction of first
582	molars allowing the second molars to drift forward have also been suggested. ¹⁰⁸
583	
584	Treatment objectives: Since best available evidence does not support early orthodontic intervention,
585	treatment objectives of PFE should involve reassurance and education about the eruption disorder and
586	preparation for future prosthetic rehabilitation. ¹⁰⁸ In some cases, early extraction can improve normal
587	development of the alveolus and permanent dentition. ¹⁰⁸ Objectives include space and intra-arch

- 588 <u>maintenance in preparation for future implants, prosthetic rehabilitation, or corticotomy-assisted tooth</u>
- 589 <u>movement.¹⁰⁸</u>
- 590
- 591

592 Tooth size/arch length discrepancy and crowding

593 General considerations and principles of management: Arch length discrepancies include inadequate 594 arch length and crowding of the dental arches, excess arch length and spacing, and tooth size discrepancy, often referred to as a Bolton discrepancy.¹²⁰ These arch length discrepancies may be found in conjunction 595 596 with complicating and other etiological factors including missing teeth, supernumerary teeth, and fused or 597 geminated teeth. Inadequate arch length with resulting incisor crowding is a common occurrence with various negative sequelae and is particularly common in the early mixed dentition.¹²¹⁻¹²⁴ Studies of arch 598 599 length in today's children compared to their parents and grandparents of 50 years ago indicate less arch length, more frequent incisor crowding, and stable tooth sizes.¹²⁵⁻¹²⁷ This implies that the problem of 600 601 incisor crowding and ultimate arch length discrepancies may be increasing in numbers of patients and in

- amount of arch length shortage.^{125,126,128}
- 603

Arch length and especially crowding must be considered in the context of the esthetic, dental, skeletal,

- and soft tissue relationships. Mandibular incisors have a high relapse rate in rotations and crowding.^{121,123}
- 606 Growth of the aging skeleton causes further crowding and incisor rotations.¹²⁹ Functional contacts are
- 607 diminished where rotations of incisors, canines, and bicuspids exist.¹³⁰ Occlusal harmony and

temporomandibular joint health are impacted negatively by less functional contacts.¹³⁰

609

610 Initial assessment may be done in early mixed dentition, when mandibular incisors begin to erupt.¹²¹

611 Evaluation of available space and consideration of making space for permanent incisors to erupt may be

done initially utilizing appropriate radiographs to ascertain the presence of permanent successors.

613 Comprehensive diagnostic analysis is suggested, with evaluation of maxillary and mandibular skeletal

relationships, direction and pattern of growth, facial profile, facial width, muscle balance, and dental and

occlusal findings including tooth positions, arch length analysis, and leeway space.

616

617 Derotation of teeth just after emergence in the mouth implies correction before the transseptal fiber

618 arrangement has been established.^{121,130} It has been shown that the transseptal fibers do not develop until

619 the cementoenamel junction of erupting teeth pass the bony border of the alveolar process: $\frac{130}{100}$ therefore,

620 long-term stability of aligned incisors may be increased.¹³¹

CCA.k. BP_DevelopingDentition

621	
622	Treatment considerations: Treatment considerations may include, but are not limited to:
623	1. gaining space for permanent incisors to erupt and become straight naturally through primary canine
624	extraction and space/arch length maintenance with holding arches. Extraction of primary or
625	permanent teeth with the aim of alleviating crowding should not be undertaken without a
626	comprehensive space analysis and a short and long term orthodontic treatment plan.
627	2. orthodontic alignment of permanent teeth as soon as erupted and feasible, expansion and correction
628	of arch length as early as feasible.
629	3. utilizing holding arches in the mixed dentition until all permanent bicuspids and canines have
630	erupted.
631	4. maintaining patient's original arch form. ¹³⁰
632	5. interproximal stripping (enamel removal) the mandibular primary canines to allow alignment of
633	crowded lower permanent lateral incisors. ¹³²
634	
635	Additional treatment modalities may include, but are not limited to: (1) interproximal reduction; (2)
636	restorative bonding; (3) veneers; (4) crowns; (5) implants; and (6) orthognathic surgery.
637	
638	Treatment objectives: Well-timed intervention can:
639	1. prevent crowded incisors.
640	2. increase long-term stability of incisor positions.
641	3. decrease ectopic eruption and impaction of permanent canines.
642	4. reduce orthodontic treatment time and sequelae.
643	5. improve gingival health and overall dental health. ^{121,133,134}
644	
645	Space maintenance
646	General considerations and principles of management: The premature loss of primary teeth due to
647	caries, infection, trauma, ectopic eruption, or crowding deviates from the normal exfoliation pattern and
648	may lead to loss of arch length. other causes may lead to undesirable tooth movements of primary and/or-
649	permanent teeth including loss of arch length. Arch length deficiency can produce or increase the severity
650	of malocclusions with crowding, rotations, ectopic eruption, crossbite, excessive overjet, excessive
651	overbite, and unfavorable molar relationships. ¹³⁵ Whenever possible, restoration of carious primary teeth
652	should be attempted to avoid malocclusions that could result from their extraction. ¹³⁶ The use of space
653	maintainers to reduce the prevalence and severity of malocclusion following premature loss of primary

- teeth should be considered.^{16,137,138} is recommended. Space maintenance may be a consideration in the
- 655 primary dentition after early loss of a maxillary incisor when the child has an active digit habit. An-
- 656 intense habit may reduce the space for the erupting permanent incisor.
- 657
- 658 Adverse effects associated with space maintainers include:
- (1) dislodged, broken, and lost appliances; (2) plaque accumulation; (3) caries; (4) damage or
- 660 interference with successor eruption; (5) undesirable tooth movement; (6) inhibition of alveolar growth;
- (7) soft tissue impingement; and (8) pain and (9) increase in microorganisms and increase in periodontal
- 662 <u>index scores.^{135,139-145}</u> Premature loss of a primary tooth of any type, especially in crowded dentitions, has
- the potential to cause loss of space available for the succeeding permanent tooth, but there is a lack of
- 664 consensus <u>or evidence</u> regarding the effectiveness of space maintainers in preventing or reducing the
- 665 severity of malocclusion.^{135,140,141,146-155}
- 666
- 667 Treatment considerations: It is prudent to consider space maintenance when primary teeth are lost
 668 prematurely. Factors to consider include: (1) specific tooth lost; (2) time elapsed since tooth loss; (3) pre-
- 669 existing occlusion and space assessment; (4) favorable space analysis dental age; (5) presence and root
- 670 development of permanent successor; (6) <u>root development and amount of alveolar bone covering</u>
- permanent successor; (7) patient's health <u>history and medical</u> status; (8) patient's cooperative ability; (9)
- active oral habits; and (10) oral hygiene.^{16,135,156}
- 673
- The literature pertaining to the use of space maintainers specific to the loss of a particular primary tooth
- type include expert opinion, case reports, and details of appliance design.^{16,137,138} Space maintainers can be
- 676 designed as fixed unilateral (band and loop, crown and loop, distal shoe), fixed bilateral (lower lingual
- 677 <u>holding arch, Nance appliance, transpalatal arch) or removable (partial dentures, Hawley type appliance),</u>
- 678 Variations of these appliances have been described. Unilateral space maintainer kits as well as direct
- 679 <u>bonded techniques eliminate laboratory involvement and allow for single visit delivery; however, the</u>
- 680 literature describes mixed results on the longevity of these options compared to success rates of custom
- 681 <u>appliances.¹⁵⁷⁻¹⁶⁰</u> Treatment modalities may include, but are not limited to:
- Fixed appliances (e.g., band and loop, crown and loop, passive lingual arch, distal shoe, Nance appliance, transpalatal arch).
- 684 2. Removable appliances (e.g., partial dentures, Hawley appliance). (Bell et al 2011, Ngan et al 1999,
 685 Terlaje and Donly 2001)
- 686

- 687 The placement and retention of space maintaining appliances requires ongoing compliant patient
- behavior. Follow-up of patients with space maintainers is necessary to assess integrity of cement and to
- evaluate and clean the abutment teeth.¹⁴⁴ The appliance should function until the succedaneous teeth have
- 690 erupted into the arch; however, a<u>djustments or new appliances may be necessary with continued</u>
- 691 <u>development and changes in the dentition.</u>
- 692
- 693 Treatment objectives: The goal of space maintenance is to prevent loss of arch length, width, and
- 694 perimeter by maintaining the relative position of the existing dentition.^{16,137}
- 695
- 696 The AAPD recognizes the need for controlled randomized clinical trials to determine efficacy of space
- 697 maintainers as well as analysis of costs and side effects of treatment
- 698
- 699 Space regaining
- General considerations and principles of management: Some of the more common causes of space
 loss within an arch are (1) primary teeth with interproximal caries; (2) ectopically erupting teeth; (3)
- alteration in the sequence of eruption; (4) ankylosis of a primary molar; (5) dental impaction; (6)
- transposition of teeth; (7) loss of primary molars without proper space management; (8) congenitally
- missing teeth; (9) abnormal resorption of primary molar roots; (10) premature and delayed eruption of
- permanent teeth; and (11) abnormal dental morphology.^{16,135,138,161,162} Therefore, loss of space in the dental
- arch that interferes with the desired eruption of the permanent teeth may require evaluation.
- 707

708 The degree to which space is affected varies according to the arch, site in the arch, and time elapsed since

- tooth loss.¹⁶³ The quantity and incidence of space loss are dependent upon which adjacent teeth are
- 710 present in the dental arch and their status.^{16,135}. The amount of crowding or spacing in the dental arch will
- 711 determine the consequence of space loss.^{1,162}
- 712
- 713 **Treatment considerations:** Space can be maintained or regained with removable or fixed
- appliances.^{135,137}Some examples of fixed space regaining appliances are active holding arches, pendulum
- appliances, <u>Halterman type appliances</u> and Jones jig. Examples of removable space regaining appliances
- 716 are Hawley appliance with springs, lip bumper, and headgear.¹³⁷ If space regaining is planned, a
- comprehensive analysis should be completed prior to any treatment decisions. Some factors that should
- be considered in the analysis include: dentofacial development, age at time of tooth loss, tooth that has
- **719** been lost, space available, and space needed.^{1,135,137}

124

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720 721 Treatment objectives: The goal of space regaining intervention is the recovery of lost arch width and 722 perimeter and/or improved eruptive position of succedaneous teeth. Space regained should be maintained 723 until adjacent permanent teeth have erupted completely and/or until a subsequent comprehensive 724 orthodontic treatment plan is initiated. 725 726 *Crossbites (dental, functional, and skeletal)* General considerations and principles of management: Crossbites are defined as any abnormal buccal-727 lingual relation between opposing incisors, molars, or premolars in centric relation.¹⁶⁴⁻¹⁶⁶ If the mid-lines 728 undergo a compensatory or habitual shift when the teeth occlude in crossbite, this is termed a functional 729 shift.¹⁶² A crossbite can be of dental or skeletal origin or a combination of both.¹⁶² 730 731 732 A simple anterior crossbite is of dental origin if the molar occlusion is Class I and the malocclusion is the 733 result of an abnormal axial inclination of maxillary and or mandibular anterior teeth. This condition should be differentiated from a Class III skeletal malocclusion where the crossbite is the result of the 734 basal bone position.¹⁶⁴ Posterior crossbites may be the result of bilateral or unilateral lingual position of 735 the maxillary teeth relative to the mandibular posterior teeth due to tipping or alveolar discrepancy, or a 736 737 combination. Most often, unilateral posterior crossbites are the manifestation of a bilateral crossbite with a functional mandibular shift.¹⁶⁶ Dental crossbites may be the result of tipping or rotation of a tooth or 738 teeth. In this case, the condition is localized and does not involve the basal bone. In contrast, skeletal 739 740 crossbites involve disharmony of the craniofacial skeleton.^{166,167} Aberrations in bony growth may give rise to crossbites in two ways: 741 1. adverse transverse growth of the maxilla and mandible. 742 2. disharmonious or adverse growth in the sagittal (AP) length of the maxilla and mandible.^{165,168} 743 744 Such growth aberrations can be due to inherited growth patterns, trauma, or functional disturbances that 745 alter normal growth.166-168 746 747 748 Treatment considerations: Crossbites should be considered in the context of the patient's total treatment 749 needs. Anterior crossbite correction can: (1) reduce dental attrition; (2) improve dental esthetics; (3) redirect skeletal growth; (4) improve the tooth-to-alveolus relationship; and (5) increase arch perimeter 750 (6) avoid periodontal damage, and (7) prevent the potential for temporomandibular joint disfunction.^{167, 169} 751 752 If enough space is available, a simple anterior crossbite can be aligned as soon as the condition is noted.

753	Treatment options include acrylic incline planes, acrylic retainers with lingual springs, or fixed appliances		
754	with springs. If space is needed, an expansion appliance also is an option. ¹⁶⁵ Posterior crossbite correction		
755	can accomplish the same objectives and can improve the eruptive position of the succedaneous teeth.		
756	Early correction of unilateral posterior crossbites with a mandibular functional shift has been shown to		
757	improve functional conditions significantly and largely eliminate morphological and positional		
758	asymmetries of the mandible. ^{32,170,171} . Contemporary evidence indicates a need for long-term studies to		
759	assess the possibility for spontaneous crossbite correction, as current proof is conflicting. ¹⁷² Functional		
760	shifts should be eliminated as soon as possible with early correction ¹⁶⁸ to avoid temporomandibular joint		
761	dysfunction and/or asymmetric growth. ^{166,172} Treatment can be completed with:		
762	1. equilibration.		
763	2. appliance therapy (fixed or removable).		
764	3. extractions.		
765	4. a combination of these treatment modalities to correct the alveolar constriction. ¹⁷³		
766			
767	Skeletal expansion with fixed or removable palatal expanders can be utilized until midline suture fusion		
768	occurs. ^{162,164} Treatment decisions depend on the:		
769	1. amount and type of movement (tipping vs. bodily movement, rotation, or dental vs. orthopedic		
770	movement).		
771	2. space available.		
772	3. AP, transverse, and vertical skeletal relationships.		
773	4. growth status.		
774	5. patient cooperation.		
775			
776	Patients with crossbites and concomitant Class III skeletal patterns and/or skeletal asymmetry should		
777	receive comprehensive treatment as covered in the Class III malocclusion section.		
778			
779	Treatment objectives: Treatment of a crossbite should result in improved intramaxillary alignment and		
780	an acceptable interarch occlusion and function. ¹⁷⁰		
781			
782	Class II malocclusion		
783	General considerations and principles of management: Class II malocclusion (distocclusion) may be		
784	unilateral or bilateral and involves a distal relationship of the mandible to the maxilla or the mandibular		
785	teeth to maxillary teeth. This relationship may result from dental (malposition of the teeth in the arches),		

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- skeletal (mandibular retrusion and/or maxillary protrusion), or a combination of dental and skeletal
 factors.⁶
- 788

789	Results of randomized clinical trials indicate that Class II malocclusion can be corrected effectively with
790	either a single or two-phase regimen. ¹⁷⁴⁻¹⁷⁷ Growth-modifying effects in some studies did not show an
791	influence on the Class II skeletal pattern, ^{176,178,179} while other studies dispute these findings. ^{180,181} There is
792	substantial variation in treatment response to growth modification treatments (headgear or functional
793	appliance) and no reliable predictors for favorable growth response have been found. ^{174,180} Some reports
794	state interceptive treatment does not reduce the need for either premolar extractions or orthognathic
795	surgery, ^{175,176} while others disagree with these findings. ¹⁸² Two-phase treatment results in significantly
796	longer treatment time, 168,175,183 although the time spent in full bonded appliance therapy in the permanent
797	dentition can be significantly less. ¹⁸⁴
798	
799	Clinicians may decide to provide interceptive treatment based on other factors. ^{175, 180} Evidence suggests
800	that, for some children, interceptive Class II treatment may improve self-esteem and decreases negative
801	social experiences, although the improvement may not be different longterm. ^{180,185} Early Class II
802	correction may improve facial convexity and /or reduce incidence of maxillary anterior tooth trauma.186-
803	¹⁹¹ Incisor injury is associated with overjet greater than three millimeters. An overjet in excess of three
804	millimeters is associated with an increased risk of incisor injury, with large overjets (>8mm) resulting in
805	trauma in more than 40% of children. ^{192,193} Further, when injury is more severe than simple enamel-
806	fractures, increased overjet and prognathic position of the maxilla are more strongly associated (Kania et-
807	al 1996) Some studies indicate interceptive treatment for Class II malocclusions can be initiated,
808	depending upon patient cooperation and management. (Baccetti et al 1997)
809	
810	Treatment considerations: Factors to consider when planning orthodontic intervention for Class II
811	malocclusion are: (1) facial growth pattern; (2) amount of AP discrepancy; (3) patient age; (4) projected
812	patient compliance; (5) space analysis; (6) anchorage requirements; and (7) patient and parent desires.
813	
814	Treatment modalities include: (1) extraoral appliances headgear; (2) functional appliances; (3) fixed
815	appliances; (4) tooth extraction and interarch elastics; and (5) orthodontics with orthognathic surgery. ¹⁶²
816	
817	Treatment objectives: Treatment of a developing Class II malocclusion should result in an improved
818	overbite, overjet, and intercuspation of posterior teeth and an esthetic appearance and profile compatible

- 819 with the patient's skeletal morphology.
- 820
- 821 Class III malocclusion

General considerations and principles of management: Class III malocclusion (mesio-occlusion)
involves a mesial relationship of the mandible to the maxilla or mandibular teeth to maxillary teeth. This
relationship may result from dental factors (malposition of the teeth in the arches), skeletal factors
(asymmetry, mandibular prognathism, and/or maxillary retrognathism), anterior functional shift of the
mandible, or a combination of these factors.¹⁹⁴

827

The etiology of Class III malocclusions can be hereditary, environmental, or both. Hereditary factors can
include clefts of the alveolus and palate as well as other craniofacial anomalies that are part of a genetic
syndrome.^{195,196} Some environmental factors are trauma, oral/digital habits, caries, and early child- hood

831 OSAS.¹⁹⁷

832

Treatment considerations: Treatment of Class III malocclusions is indicated to provide psychosocial 833 834 benefits for the child patient by reducing or eliminating facial disfigurement and to reduce the severity of malocclusion by promoting compensating growth.¹⁹⁸ Interceptive Class III treatment has been proposed 835 for years and has been advocated as a necessary tool in contemporary orthodontics; initiation in the 836 primary-early mixed dentition is recommended.¹⁹⁹⁻²⁰⁸ Factors to consider when planning orthodontic 837 intervention for Class III malocclusion are: (1) facial growth pattern; (2) amount of AP discrepancy; (3) 838 839 patient age; (4) projected patient compliance and (5) space. analysis; (6) anchorage headgear; (7)functional appliances; fixed appliances; (9) tooth extraction; (10) interarch elastics; and (11) orthodontics-840 with orthognathic surgery. (Proffit and Sarver 2012). 841 842 843 **Treatment objectives:** Interceptive Class III treatment may provide a more favorable environment for growth and may improve occlusion, function, and esthetics.²⁰⁹ Although interceptive treatment can 844 minimize the malocclusion and potentially eliminate future orthognathic surgery, this is not always 845 possible. Typically, Class III patients tend to grow longer and more unpredictably and, therefore, surgery 846 847 combined with orthodontics may be the best alternative to achieve a satisfactory result for some patients, especially if they exhibit facial characteristics as follows: mandible forward to cranial base, increased 848 mandibular length, short ramal length or obtuse gonial angle.^{162,210-212} 849

850

851 Treatment of a Class III malocclusion <u>can be achieved using several modalities including: protraction</u>

- 852 therapy with or without rapid palatal expansion, functional appliances, intermaxillary elastics with
- 853 modified miniplates, or chin cup therapy.^{199,200-202,210,213-218} These interventions in a growing patient should
- result in improved overbite, overjet, and intercuspation of posterior teeth and an esthetic appearance and
- profile compatible with the patient's skeletal morphology.
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1 [Best Practices]

2 Pediatric Restorative Dentistry*

- 3
- 4 Review Council
- 5 Council on Clinical Affairs
- 6 Latest Revision
- 7 2016^{†‡} <u>2019</u>
- 8

9 Purpose

10 The American Academy of Pediatric Dentistry (AAPD) intends this guideline these recommendations to

- 11 help practitioners make decisions regarding restorative dentistry, including when it is necessary to treat
- 12 and what the appropriate materials and techniques are for restorative dentistry in children and adolescents.
- 13

14 Methods

- 15 A thorough review of the scientific literature in the English language pertaining to restorative dentistry in
- 16 primary and permanent teeth was completed to revise the previous guideline. Electronic database and
- hand searches, for the most part between the years 1995-2013 <u>2000-2018</u>, were conducted using the
- 18 terms: restorative treatment decisions, caries diagnosis, caries excavation, dental amalgam, glass
- 19 ionomers, resin modified glass ionomers, conventional glass ionomers, atraumatic/ alternative restorative
- 20 technique (ART), interim therapeutic restoration (ITR), resin infiltration, dental composites, pit and
- 21 fissure sealants, resin based sealants, glass ionomer sealants, resin based composite, dental composites,
- 22 compomers, stainless steel crowns (SSC), primary molar, preformed metal crown, strip crowns, pre-
- veneered crowns, esthetic restorations, clinical trials and, randomized controlled clinical trials.

* ABBREVIATIONS

AAPD: American Academy of Pediatric Dentistry. **ART**: Alternative restorative technique. **BPA**: Bisphenol A. **FDA**: Food and Drug Administration. **GIC**: Glass ionomer cement. **HT**: Hall technique. **ITR**: Interim therapeutic restoration. **RMGIC**: Resin modified glass ionomer cements. **SSC**: Stainless steel crowns.

⁺ The 2016 revision is limited to the addition of Hall technique for preformed metal crowns.

[‡] The AAPD, in conjunction with the American Dental Association, published in 2016 a separate document, Evidence based Clinical Practice Guideline for the Use of Pit and Fissure Sealants (available at: "http://www.aapd.org/media/Policies_Guidelines/G_EBD-Sealants.pdf"). The clinical guidance in that document supersedes any conflicting recommendations which may be found in this section.

24

25	Those papers that were used to evaluate clinical efficacy on specific restorative dentistry topics (e.g.,
26	amalgam, resin-based composite) initially were evaluated by abstract by two individuals. Criteria for
27	evaluation included if the paper fulfilled the qualification of a controlled clinical trial, meta-analysis, or
28	systematic review. Full evaluation and abstraction included examination of the research methods and
29	potential for study bias (e.g., patient recruitment, randomization, blinding, subject loss, sample size
30	estimates, conflicts of interest, statistics). Research that was considered deficient or had high bias was
31	eliminated. In those topic areas for which there were rigorous meta-analyses or systematic reviews
32	available, only those clinical trial articles that were not covered by the reviews were subjected to full
33	evaluation and abstraction. This strategy yielded 35 meta-analyses/systemic reviews and 62 randomized
34	controlled clinical trials that primarily made up the evidence for this guideline.
35	
36	The assessment of evidence for each topic was based on a modification of the American Dental
37	Association's grading of recommendations: strong evidence (based on well-executed randomized control
38	trials, meta-analyses, or systematic reviews); evidence in favor (based on weaker evidence from clinical
39	trials); and expert opinion (based on retrospective trials, case reports, in vitro studies, and opinions from
40	clinical researchers). ¹
41	
42	This guideline was These recommendations were originally developed by the Restorative Dentistry
43	Subcommittee of the Clinical Affairs Committee and adopted in 1991. The last comprehensive revision of
44	this document was in 2014.
45	
46	When to restore
47	Historically, the management of dental caries was based on the belief that caries was a progressive
48	disease that eventually destroyed the tooth unless there was surgical and restorative intervention. ² It is
49	now recognized that restorative treatment of dental caries alone does not stop the disease process ³ and
50	restorations have a finite lifespan. Conversely, some carious lesions may not progress and, therefore, may
51	not need restoration. Consequently, eContemporary management of dental caries includes identification
52	of an individual's risk for caries progression, understanding of the disease process for that individual, and
53	active surveillance to assess disease progression and manage with appropriate preventive services,
54	supplemented by restorative therapy when indicated. ⁴
55	
56	With the exception of reports of dental examiners in clinical trials, studies of reliability and

- 57 reproducibility of detecting dental caries are not conclusive.⁵ There also is minimal information regarding
- validity of caries diagnosis in primary teeth,² as primary teeth may require different criteria due to thinner
- 59 enamel and dentin and broader proximal contacts.⁶ Furthermore, indications for restorative therapy only
- 60 have been examined superficially because such decisions generally have been regarded as a function of
- 61 clinical judgment.⁷ Decisions for when to restore carious lesions should include at least clinical criteria of
- 62 visual detection of enamel cavitation, visual identification of shadowing of the enamel, and/ or
- 63 radiographic recognition of enlargement of lesions.^{4,8,9} over time.
- 64
- 65 The benefits of restorative therapy include: removing cavitations or defects to eliminate areas that are
- susceptible to caries; stopping the progression of tooth demineralization; restoring the integrity of tooth
- 67 structure; preventing the spread of infection into the dental pulp; and preventing the shifting of teeth due
- to loss of tooth structure. The risks of restorative therapy include lessening the longevity of teeth by
- 69 making them more susceptible to fracture, recurrent lesions, restoration failure, pulp exposure during
- caries excavation, future pulpal complications, and iatrogenic damage to adjacent teeth.¹⁰⁻¹² Primary teeth
- 71 may be more susceptible to restoration failures than permanent teeth.¹³ Additionally, before restoration of
- 72 primary teeth, one needs to consider the length of time remaining prior to tooth exfoliation
- 73

74 *Recommendations:*

- Management of dental caries includes identification of an individual's risk for caries progression,
 understanding of the disease process for that individual, and active surveillance to assess disease
- progression and manage with appropriate preventive services, supplemented by restorative therapywhen indicated.
- 2. Decisions for when to restore carious lesions should include at least clinical criteria of visual
- 80 detection of enamel cavitation, visual identification of shadowing of the enamel, and/or
- 81 radiographic recognition of enlargement of lesions over time progression of lesions.
- 82

83 Deep caries excavation and restoration

- 84 Among the objectives of restorative treatment are to repair or limit the damage from caries, protect and
- 85 preserve the tooth structure, and maintain pulp vitality whenever possible. The AAPD Guideline on Pulp-
- 86 Therapy for Primary and Immature Permanent Teeth Use of Vital Pulp Therapies in Primary Teeth with
- 87 <u>Deep Caries Lesions and Best Practices for Pulp Therapy for Primary and Immature Permanent Teeth</u>
- states the treatment objective for a tooth affected by caries is to maintain pulpal vitality, especially in
- 89 immature permanent teeth for continued apexogenesis.¹⁴

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91	With regard to the treatment of deep caries, three methods of caries removal have been compared to
92	complete excavation, where all carious dentin is removed. Stepwise excavation is a two-step caries
93	removal process in which carious dentin is partially removed at the first appointment, leaving caries over
94	the pulp, with placement of a temporary filling. At the second appointment, all remaining carious dentin
95	is removed and a final restoration placed. ¹⁵ Partial, or one-step, caries excavation removes part of the
96	carious dentin, but leaves caries over the pulp, and subsequently places a base and final restoration. ^{16,17}
97	No removal of caries before restoration of primary molars in children aged three to 10 years also has been
98	reported. ¹⁸
99	
100	Evidence from randomized controlled trials and a systematic review shows that pulp exposures in primary
101	and permanent teeth are significantly reduced using incomplete caries excavation compared to complete
102	excavation in teeth with a normal pulp or reversible pulpitis. Two trials and a Cochrane review found that
103	partial excavation resulted in significantly fewer pulp exposures compared to complete excavation. ¹⁹⁻²¹
104	Two trials of step-wise excavation showed that pulp exposure occurred more frequently from complete
105	excavation compared to step-wise excavation. ^{15,20} There also is evidence of a decrease in pulpal
100	complications and post-operative pain after incomplete caries excavation compared to complete
106	complete carles excavation complete to complete
108	excavation in clinical trials, ^{15,20,22,23} summarized in a meta-analysis. ²⁴
107	
107 108	excavation in clinical trials, ^{15,20,22,23} summarized in a meta-analysis. ²⁴
107 108 109	excavation in clinical trials, ^{15,20,22,23} summarized in a meta-analysis. ²⁴ Additionally, a meta-analysis found the risk for permanent restoration failure was similar for incompletely
107 108 109 110	excavation in clinical trials, ^{15,20,22,23} summarized in a meta-analysis. ²⁴ Additionally, a meta-analysis found the risk for permanent restoration failure was similar for incompletely and completely excavated teeth. ²⁴ With regard to the need to reopen a tooth with partial excavation of
107 108 109 110 111	excavation in clinical trials, ^{15,20,22,23} summarized in a meta-analysis. ²⁴ Additionally, a meta-analysis found the risk for permanent restoration failure was similar for incompletely and completely excavated teeth. ²⁴ With regard to the need to reopen a tooth with partial excavation of caries, one randomized controlled trial that compared partial (one-step) to stepwise excavation in
107 108 109 110 111 112	excavation in clinical trials, ^{15,20,22,23} summarized in a meta-analysis. ²⁴ Additionally, a meta-analysis found the risk for permanent restoration failure was similar for incompletely and completely excavated teeth. ²⁴ With regard to the need to reopen a tooth with partial excavation of caries, one randomized controlled trial that compared partial (one-step) to stepwise excavation in permanent molars found higher rates of success in maintaining pulp vitality with partial excavation,
107 108 109 110 111 112 113	excavation in clinical trials, ^{15,20,22,23} summarized in a meta-analysis. ²⁴ Additionally, a meta-analysis found the risk for permanent restoration failure was similar for incompletely and completely excavated teeth. ²⁴ With regard to the need to reopen a tooth with partial excavation of caries, one randomized controlled trial that compared partial (one-step) to stepwise excavation in permanent molars found higher rates of success in maintaining pulp vitality with partial excavation, suggesting there is no need to reopen the cavity and perform a second excavation. ¹⁶ Interestingly, two
107 108 109 110 111 112 113 114	excavation in clinical trials, ^{15,20,22,23} summarized in a meta-analysis. ²⁴ Additionally, a meta-analysis found the risk for permanent restoration failure was similar for incompletely and completely excavated teeth. ²⁴ With regard to the need to reopen a tooth with partial excavation of caries, one randomized controlled trial that compared partial (one-step) to stepwise excavation in permanent molars found higher rates of success in maintaining pulp vitality with partial excavation, suggesting there is no need to reopen the cavity and perform a second excavation. ¹⁶ Interestingly, two randomized controlled trials suggest that no excavation can arrest dental caries so long as a good seal of
107 108 109 110 111 112 113 114 115	excavation in clinical trials, ^{15,20,22,23} summarized in a meta-analysis. ²⁴ Additionally, a meta-analysis found the risk for permanent restoration failure was similar for incompletely and completely excavated teeth. ²⁴ With regard to the need to reopen a tooth with partial excavation of caries, one randomized controlled trial that compared partial (one-step) to stepwise excavation in permanent molars found higher rates of success in maintaining pulp vitality with partial excavation, suggesting there is no need to reopen the cavity and perform a second excavation. ¹⁶ Interestingly, two randomized controlled trials suggest that no excavation can arrest dental caries so long as a good seal of
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107 108 109 110 111 112 113 114 115 116 117	excavation in clinical trials, ^{15,20,22,23} summarized in a meta-analysis. ²⁴ Additionally, a meta-analysis found the risk for permanent restoration failure was similar for incompletely and completely excavated teeth. ²⁴ With regard to the need to reopen a tooth with partial excavation of caries, one randomized controlled trial that compared partial (one-step) to stepwise excavation in permanent molars found higher rates of success in maintaining pulp vitality with partial excavation, suggesting there is no need to reopen the cavity and perform a second excavation. ¹⁶ Interestingly, two randomized controlled trials suggest that no excavation can arrest dental caries so long as a good seal of the final restoration is maintained. ^{18,25} <i>Recommendations</i>
107 108 109 110 111 112 113 114 115 116 117 118	excavation in clinical trials, ^{15,20,22,23} summarized in a meta-analysis. ²⁴ Additionally, a meta-analysis found the risk for permanent restoration failure was similar for incompletely and completely excavated teeth. ²⁴ With regard to the need to reopen a tooth with partial excavation of caries, one randomized controlled trial that compared partial (one-step) to stepwise excavation in permanent molars found higher rates of success in maintaining pulp vitality with partial excavation, suggesting there is no need to reopen the cavity and perform a second excavation. ¹⁶ Interestingly, two randomized controlled trials suggest that no excavation can arrest dental caries so long as a good seal of the final restoration is maintained. ^{18,25} <i>Recommendations</i> 1. There is evidence from randomized controlled trials and systematic reviews that incomplete caries
107 108 109 110 111 112 113 114 115 116 117 118 119	excavation in clinical trials, ^{15,20,22,23} summarized in a meta-analysis. ²⁴ Additionally, a meta-analysis found the risk for permanent restoration failure was similar for incompletely and completely excavated teeth. ²⁴ With regard to the need to reopen a tooth with partial excavation of caries, one randomized controlled trial that compared partial (one-step) to stepwise excavation in permanent molars found higher rates of success in maintaining pulp vitality with partial excavation, suggesting there is no need to reopen the cavity and perform a second excavation. ¹⁶ Interestingly, two randomized controlled trials suggest that no excavation can arrest dental caries so long as a good seal of the final restoration is maintained. ^{18,25} <i>Recommendations</i> 1. There is evidence from randomized controlled trails and systematic reviews that incomplete caries excavation in primary and permanent teeth with normal pulps or reversible pulpitis, either partial

- is no higher after incomplete rather than complete caries excavation.
- 124 3. There is evidence that partial excavation (one-step) followed by placement of final restoration leads
- to higher success in maintaining pulp vitality in permanent teeth than stepwise (two-step)
- excavation.
- 127

128 Pit and fissure sealants

- 129 Pit and fissure caries account for approximately 80 to 90 percent of all caries in permanent posterior teeth-
- 130 and 44 percent in primary teeth.(Beauchamp et al 2008) Pit and fissure sealant has been described as a
- 131 material placed into the pits and fissures of caries susceptible teeth that micromechanically bonds to the
- 132 tooth preventing access by cariogenic bacteria to their source of nutrients, (Simonsen 1978) thus reducing-
- 133 the risk of caries in those susceptible pits and fissures.
- 134
- 135 With regard to evidence of effectiveness, a Cochrane review found that sealants placed on the occlusal-
- 136 surfaces of permanent molars in children and adolescents reduced caries up to 48 months when compared-
- 137 to no sealant.(Ahovuo Saloranta et al 2013) According to a meta analysis of 24 studies, the overall-
- 138 effectiveness of auto-polymerised fissure sealants in preventing dental decay was 71 percent.(Llodra et al-
- 139 1993) Another Cochrane review calculated that placement of resin-based sealant in children and
- 140 adolescent reduces caries incidence of 86 percent after one year and 57 percent at 48 to 54-
- 141 months.(Ahovuo-Saloranta et al 2004) Sealants must be retained on the tooth and should be monitored to-
- 142 be most effective. Studies incorporating recall and maintenance have reported sealant success levels of 80-

143 to 90 percent after 10 or more years.(Simonsen 1991, Romcke et al 1990)

- 144
- 145 There are many systematic reviews and clinical trials regarding optimizing the effectiveness of dental-
- 146 sealants. Sealants are more cost effective in children with caries risk and generally are recommended to-
- 147 be placed only in those children at caries risk. (AAPD BP_Caries risk, Beauchamp et al 2008, Weintraub-
- 148 2001) The best evaluation of high caries risk is done by an experienced clinician using indicators of low-
- 149 socio economic status, high frequency of sugar consumption, prior caries, active white spot lesions and
- 150 enamel defects, and low salivary flow.(AAPD BP_Caries risk)
- 151
- 152 Pit-and-fissure sealants lower the number of viable bacteria, including *Streptococcus mutans* and
- 153 lactobacilli, by at least 100 fold and reduced the number of lesions with any viable bacteria by about 50-
- 154 percent.(Griffin et al 2009) This evidence supports recommendations to seal sound surfaces and non-
- 155 cavitated enamel lesions.(Beauchamp et al 2008, Griffin et al 2009)

156

- 157 Evidence based reviews have found that caries risk for sealed teeth that have lost some or all sealant does-
- 158 not exceed the caries risk for never sealed teeth. Therefore, it has been recommended to provide sealants-
- 159 to children even if follow-up cannot be ensured.(Griffin et al 2009)
- 160
- 161 Systematic reviews and clinical trials have evaluated techniques for placement of sealants. According to a
- 162 systematic review, isolation of the tooth is an important aspect of sealant placement and use of rubber-
- 163 dam improves the retention rates of light cured resin based sealants.³⁴ Moisture control systems (IsoliteTM,
- 164 VacuEjectorTM, <u>DrySheild</u>TM) produce sealant retention rates comparable to cotton roll isolation or rubber
- 165 dam, while decreasing procedure time. (Wood et al 1989, Collette et al 2010) Another systematic review-
- 166 has shown that four handed technique has been associated with higher retention of resin based
- 167 sealants.(Griffin et al 2008) Two systematic reviews have shown that teeth cleaned prior to sealant
- 168 application with a tooth brush prophylaxis exhibited similar or higher success rate compared to those-
- 169 sealed after hand piece prophylaxis.(Griffin et al 2008, Gray et al 2009) Additionally, there is limited and
- 170 conflicting evidence to support mechanical preparation with a bur prior to sealant placement, and it is not
- 171 recommended.(Beauchamp et al 2008) There is evidence that mechanical preparation may make a tooth-
- 172 more prone to caries in case of resin-based sealant loss.(Dhar et al 2012)
- 173

174 With regard to primer placement before sealant application, there is one randomized clinical trial that

- 175 suggests that acetone or ethanol solvent based primers, especially the single bottle system, enhanced the
- 176 retention of sealants, whereas water-based primers were found to drastically reduce the retention of
- 177 sealants.(Feigal 2000) With regard to self etch bonding agents that do not involve a separate step for-
- 178 etching, a systematic review found that self etch bonding agents may not provide as good retention as-
- 179 acid etch technique;(Muller-Bolla et al 2006) however, one recent randomized clinical trial reported-
- 180 similar retention rates of self-etch system compared to acid etch group.(Maher et al 2013)
- 181
- 182 Based on a systematic review and clinical trials, there is substantial data regarding the use of resin based
- 183 and glass ionomer-based sealants. One meta analysis and a Cochrane review show high retention rates of
- 184 resin-based sealants compared to glass ionomer-based sealants.(Ahovuo Saloranta 2013, Kühnisch et al-
- 185 2012) However, glass ionomer sealants exhibited good short term retention comparable with resin-
- sealants at one year, and they may be used as an interim preventive agent when resin-based sealant cannot
- 187 be placed as moisture control may compromise such placement.(Beauchamp et al 2008, Ahovuo-
- 188 Saloranta 2013) Another systematic review of the caries-preventive effects of glass ionomer and resin-

189 based fissure sealants suggests no difference between these two products.(Mickenautsch and Yengopal-190 2011) 191 192 There is insufficient data to support use of fissure sealants in primary teeth. One trial reported retention-193 rate of 76.5 percent for light polymerized fissure sealants in the follow-up time of 2.8 years.(Hotuman et-194 al 1998) Another randomized clinical trial studied effectiveness of glass ionomer sealants in primarymolars and found retention rate as low as 18.7 percent in 1.38 years and no statistically significant caries 195 reduction.(Chadwick et al 2005) 196 197 198 Recommendations: 1. Based on a meta-analysis and Cochrane reviews, sealants should be placed on pit and fissure-199 200 surfaces judged to be at risk for dental caries or surfaces that already exhibit incipient, non-201 cavitated carious lesions to inhibit lesion progression. 202 2. According to a systematic review and a randomized clinical trial, sealant placement methods should include careful cleaning of the pits and fissures without mechanical tooth preparation. 203 204 3. Based on a systematic review, resin based sealants require placement in a moisture controlled 205 environment, often facilitated by four-handed technique. 4. There is evidence from a randomized clinical trial that a low-viscosity hydrophilic material bonding 206 207 layer, as part of or under the actual sealant, is better for long-term retention and effectiveness. 5. There is evidence from a Cochrane review and a systematic review that resin based materials 208 209 achieve better retention and, therefore, may be preferred as dental sealants, but glass ionomersealants could be used as transitional sealants when moisture control is not possible. 210 211 212 **Resin infiltration** Resin infiltration is an innovative approach utilized primarily to arrest the progression of non-cavitated 213 interproximal caries lesions.^{26,27} The aim of the resin infiltration technique is to allow penetration of a low 214 viscosity resin into the porous lesion body of enamel caries.²⁶ 215 216 217 Most randomized clinical trials done on resin infiltration had industrial support with potential of conflict of interest. One such trial evaluated infiltration and sealants versus placebo and found significant 218 differences between infiltration versus placebo with lesion progression 32 percent versus 70 percent 219 220 respectively.²⁸ Another randomized clinical trial reported significant difference between infiltration (7 percent) versus placebo (37 percent) in the percentage of progression in lesion depth.²⁶ A systematic 221

- review on randomized clinical trials on resin infiltration rated the quality score to be low to moderate. The 222 223 review concluded that resin infiltration has a potential consistent benefit in slowing the progression or 224 reversing non-cavitated carious lesions.²⁹ 225 226 An additional use of resin infiltration has been suggested to restore white spot lesions formed during 227 orthodontic treatment. Based on a randomized clinical trial, resin infiltration significantly improved the clinical appearance of such white spot lesions and visually reduced their size.³⁰ 228 229 230 *Recommendation:* 1. From randomized controlled trials, tThere is low to moderate³¹ evidence in favor of resin 231 infiltration as a treatment option for small, non-cavitated interproximal carious lesions in permanent 232 233 teeth. 234 235 **Dental amalgam** Dental amalgam has been the most commonly used restorative material in posterior teeth for over 150 236 years and is still widely used throughout the world today.³² Amalgam contains a mixture of metals such as 237 silver, copper, and tin, in addition to approximately 50 percent mercury.³³ Dental amalgam has declined in 238 239 use over the past decade,³² perhaps due to the controversy surrounding perceived health effects of 240 mercury vapor, environmental concerns from its mercury content, and increased demand for esthetic alternatives. 241 242 With regard to safety of dental amalgam, a comprehensive literature review of dental studies published 243 between 2004 and 2008 found insufficient evidence of associations between mercury release from dental 244 amalgam and the various medical complaints.³⁴ Two independent randomized controlled trials in children 245 have examined the effects of mercury release from amalgam restorations and found no effect on the 246 central and peripheral nervous systems and kidney function.^{35,36} However, on July 28, 2009, the Food and 247 Drug Administration (FDA) issued a final rule that reclassified dental amalgam to a Class II device 248 249 (having some risk) and designated guidance that included warning labels regarding: (1) possible harm of 250 mercury vapors; (2) disclosure of mercury content; and (3) contraindications for persons with known
- 251 mercury sensitivity. Also in this final rule, the FDA noted that there is limited information regarding
- dental amalgam and the long-term health outcomes in pregnant women, developing fetuses, and children
- **253** under the age of six.³³
- 254

255 With regard to clinical efficacy of dental amalgam, results comparing longevity of amalgam to other

restorative materials are inconsistent. The majority of meta-analyses, evidence-based reviews, and

- 257 randomized controlled trials report comparable durability of dental amalgam to other restorative
- 258 materials,³⁷⁻⁴² while others show greater longevity for amalgam.^{43,44}The comparability appears to be
- especially true when the restorations are placed in controlled environments such as university settings.³⁷
- 260

261 Class I amalgam restorations in primary teeth have shown in a systematic review and two randomized

- controlled trials to have a success rate of 85 to 96 percent for up to seven years, with an average annual
- failure rate of 3.2 percent.^{13,41,44} Efficacy of Class I amalgam restorations in permanent teeth of children
- has been shown in two independent randomized controlled studies to range from 89.8 to 98.8 percent for
- up to seven years. 41,43
- 266

With regard to Class II restorations in primary molars, a 2007 systematic review concluded that amalgam should be expected to survive a minimum of 3.5 years and potentially in excess of seven years.⁴⁵ For Class II restorations in permanent teeth, one meta-analysis and one evidence-based review conclude that the mean annual failure rates of amalgam and composite are equal at 2.3 percent.^{37,40} The meta-analysis comparing amalgam and composite Class II restorations in permanent teeth suggests that higher replacement rates of composite in general practice settings can be attributed partly to general practitioners' confusion of marginal staining for marginal caries and their subsequent premature

replacements. Otherwise, this meta-analysis concludes that the median success rate of composite and

- amalgam are statistically equivalent after ten years, at 92 percent and 94 percent respectively.³⁷
- 276

277 The limitation of many of the clinical trials that compare dental amalgam to other restorative materials is

that the study period often is short (24 to 36 months), at which time interval all materials reportedly

279 perform similarly.⁴⁶⁻⁵⁰ Some of these studies also may be at risk for bias, due to lack of true

- randomization, inability of blinding of investigators, and in some cases financial support by the
- 281 manufacturers of the dental materials being studied.
- 282
- 283 *Recommendation:*
- There is strong evidence that dental amalgam is efficacious in the restoration of Class I and Class II
 cavity restorations in primary and permanent teeth.
- 286
- 287 Composites

288 Resin-based composite restorations were introduced in dentistry about a half century ago as an esthetic restorative material,^{51,52} and composites are increasingly used in place of amalgam for the restoration of 289 290 carious lesions.⁵³ Composites consist of a resin matrix and chemically bonded fillers.³⁷ They are classified 291 according to their filler size, because filler size affects polishability/esthetics, polymerization depth, 292 polymerization shrinkage, and physical properties. Hybrid resins combine a mixture of particle sizes for improved strength while retaining esthetics.⁵⁴ The smaller filler particle size allows greater polishability 293 294 and esthetics, while larger size provides strength. Flowable resins have a lower volumetric filler percentage than hybrid resins.⁵⁵ 295 296 297 Several factors contribute to the longevity of resin composites, including operator experience, restoration size, and tooth position.⁴³ Resins are more technique sensitive than amalgams and require longer 298 299 placement time. In cases where isolation or patient cooperation is in question, resin-based composite may not be the restorative material of choice.⁵⁶ 300 301 302 Bisphenol A (**BPA**) and its derivatives are components of resin-based dental sealants and composites. 303 Trace amounts of BPA derivatives are released from dental resins through salivary enzymatic hydrolysis and may be detectable in saliva up to three hours after resin placement.⁵⁷ Evidence is accumulating that 304 305 certain BPA derivatives may pose health risks attributable to their estrogenic properties. BPA exposure 306 reduction is achieved by cleaning filling surfaces with pumice, cotton roll, and rinsing. Additionally, potential exposure can be reduced by using a rubber dam.⁵⁷ Considering the proven benefits of resin based 307 308 dental materials and minimal exposure to BPA and its derivatives, it is recommended to continue using

- 309 these products while taking precautions to minimize exposure.⁵⁷
- 310

There is strong evidence from a meta-analysis of 59 randomized clinical trials of Class I and II composite
 and amalgam restorations showing an overall success rate about 90 percent after 10 years for both
 materials, with rubber dam use significantly increasing restoration longevity.³⁷ Other isolation techniques
 <u>may be used (e.g., Isolite®, Dry ShieldTM)</u>. Strong evidence from randomized controlled trials comparing
 composite restorations to amalgam restorations showed that the main reason for restoration failure in both
 materials was recurrent caries.^{41,43,58}

317

318 In primary teeth, there is strong evidence that composite restorations for Class I restorations are

successful.^{13, 41} There is only one randomized controlled trial showing success in Class II composite

320 restorations in primary teeth that were expected to exfoliate within two years.⁴⁸ In permanent molars,

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- 321 composite replacement after 3.4 years was no different than amalgam,⁴¹ but after seven to 10 years the
- 322 replacement rate was higher for composite.⁵⁶ Secondary caries rate was reported as 3.5 times greater for
- 323 composite versus amalgam.⁴³
- 324
- 325 There is evidence from a meta-analysis showing that etching and bonding of enamel and dentin
- 326 significantly decreases marginal staining and detectable margins in composite restorations.³⁷ Regarding
- 327 different types of composites (packable, hybrid, nano, macro, and micro filled) there is strong evidence
- 328 showing similar overall clinical performance for these materials.⁵⁹⁻⁶²
- 329

330 *Recommendations:*

- In primary molars, there is strong evidence from randomized controlled trials that composite resins
 are successful when used in Class I restorations. For Class II lesions in primary teeth, there is one
 randomized controlled trial showing success of composite resin restorations for two years.
- In permanent molars, there is strong evidence from meta-analyses that composite resins can be used
 successfully for Class I and II restorations.
- 336 3. Evidence from a meta-analysis shows enamel and dentin bonding agents decrease marginal staining337 and detectable margins for the different types of composites.
- 338

339 Glass ionomer cements

Glass ionomers cements have been used in dentistry as restorative cements, cavity liner/base, and luting 340 341 cement since the early 1970s.⁶³ Originally, glass ionomer materials were difficult to handle, exhibited poor wear resistance, and were brittle. Advancements in conventional glass ionomer formulation led to 342 better properties, including the formation of resin-modified glass ionomers. These products showed 343 improvement in handling characteristics, decreased setting time, increased strength, and improved wear 344 resistance.^{64,65}All glass ionomers have several properties that make them favorable for use in children 345 including: chemical bonding to both enamel and dentin; thermal expansion similar to that of tooth 346 structure; biocompatibility; uptake and release of fluoride; and decreased moisture sensitivity when 347 compared to resins. 348

349

350 Fluoride is released from glass ionomer and taken up by the surrounding enamel and dentin, resulting in

- teeth that are less susceptible to acid challenge.^{66,67} One study has shown that fluoride release can occur
- for at least one year.⁶⁸ Glass ionomers can act as a reservoir of fluoride, as uptake can occur from
- dentifrices, mouth rinses, and topical fluoride applications.^{69,70} This fluoride protection, useful in patients

at high risk for caries, has led to the use of glass ionomers as luting cement for SSCs, space maintainers,
 and orthodontic bands.⁷¹

356

357 Regarding use of conventional glass ionomers in primary teeth, one randomized clinical trial showed the overall median time from treatment to failure of glass ionomer restored teeth was 1.2 years.⁴⁴ Based on 358 findings of a systematic review and meta-analysis, conventional glass ionomers are not recommended for 359 Class II restorations in primary molars.^{72,73} Conventional glass ionomer restorations have other drawbacks 360 such as poor anatomical form and marginal integrity.^{74,75} Composite restorations were more successful 361 than glass ionomer cements where moisture control was not a problem.⁷³ 362 363 Resin modified glass ionomer cements (**RMGIC**), with the acid-base polymerization supplemented by a 364 second resin light cure polymerization, has been shown to be efficacious in primary teeth. Based on a 365 meta-analysis, RMGIC is more successful than conventional glass ionomer as a restorative material.⁷³ A 366

367 systematic review supports the use of RMGIC in small to moderate sized Class II cavities.⁷² Class II

368 RMGIC restorations are able to withstand occlusal forces on primary molars for at least one year.⁷³

369 Because of fluoride release, RMGIC may be considered for Class I and Class II restorations of primary

molars in a high caries risk population.⁷⁵ There is also some evidence that conditioning dentin improves

the success rate of RMGIC.⁷² According to one randomized clinical trial, cavosurface beveling leads to

high marginal failure in RMGIC restorations and is not recommended.⁵⁸

373

374 With regard to permanent teeth, a meta-analysis review reported significantly fewer carious lesions on single-surface glass ionomer restorations in permanent teeth after six years as compared to restorations 375 376 with amalgam.⁷⁵ Data from a meta-analysis shows that RMGIC is more caries preventive than composite resin with or without fluoride.⁷⁶ Another meta-analysis showed that cervical restorations (Class V) with 377 glass ionomers may have a good retention rate, but poor esthetics.⁷⁷ For Class II restorations in permanent 378 teeth, one randomized clinical trial showed unacceptable high failure rates of conventional glass 379 380 ionomers, irrespective of cavity size. However, a high dropout rate was observed in this study limiting its significance.⁷⁸ In general, there is insufficient evidence to support the use of RMGIC as long-term 381 382 restorations in permanent teeth.

383

Other applications of glass ionomers where fluoride release has advantages are for interim therapeutic
 restorations (ITR) and the atraumatic/alternative restorative technique (ART). These procedures have

386 similar techniques but different therapeutic goals. ITR may be used in very young patients,⁷⁹

- 387 uncooperative patients, or patients with special health care needs⁴² for whom traditional cavity
- 388 preparation and/or placement of traditional dental restorations are not feasible or need to be postponed.
- 389 Additionally, ITR may be used for caries control in children with multiple open carious lesions, prior to
- 390 definitive restoration of the teeth.⁸⁰ In vitro, leaving caries-affected dentin does not jeopardize the
- bonding of glass ionomer cements to the primary tooth dentin.⁸¹ ART, endorsed by the World Health
- 392 Organization and the International Association for Dental Research, is a means of restoring and
- preventing caries in populations that have little access to traditional dental care and functions as definitive
- treatment.
- 395
- 396 According to a meta-analysis, single surface ART restorations showed high survival rates in both primary
- and permanent teeth.⁸² One randomized clinical trial supported single surface restorations irrespective of
- the cavity size and also reported higher success in non-occlusal posterior ART compared to occlusal
- 399 posterior ART.⁸³ With regard to multi-surface ART restorations, there is conflicting evidence. Based on a
- 400 meta-analysis, ART restorations presented similar survival rates to conventional approaches using
- 401 composite or amalgam for Class II restorations in primary teeth.⁸⁴ However, another meta-analysis
- 402 showed that multi-surface ART restorations in primary teeth exhibited high failure rates.⁸²
- 403

404 *Recommendations:*

- 1. There is evidence in favor of glass ionomer cements for Class I restorations in primary teeth.
- 406 2. From a systematic review, there is strong evidence that resin-modified glass ionomer cements for
- 407 Class I restorations are efficacious, and expert opinion supports Class II restorations in primary408 teeth.
- 3. There is insufficient evidence to support the use of conventional or resin-modified glass ionomercements as long-term restorative material in permanent teeth.
- 411 4. From a meta-analysis, there is strong evidence that interim therapeutic restoration/atraumatic
- 412 restorative technique (ITR/ART) using high viscosity glass ionomer cements has value as single
- 413 surface temporary restoration for both primary and permanent teeth. Additionally, ITR may be used
- 414 for caries control in children with multiple open carious lesions, prior to definitive restoration of the415 teeth.
- 416

417 Compomers

- 418 Polyacid-modified resin-based composites, or compomers, were introduced into dentistry in the mid-
- 419 1990s. They contain 72 percent (by weight) strontium fluorosilicate glass and the average particle size is

- 420 2.5 micrometers.⁸⁵ Moisture is attracted to both acid functional monomer and basic ionomer-type in the
- 421 material. This moisture can trigger a reaction that releases fluoride and buffers acidic environments.^{86,87}
- 422 Considering the ability to release fluoride, esthetic value, and simple handling properties of compomer, it
- 423 can be useful in pediatric dentistry.⁸⁵
- 424
- 425 Based on a recent randomized clinical trial, the longevity of Class I compomer restorations in primary
- teeth was not statistically different compared to amalgam, but compomers were found to need
- 427 replacement more frequently due to recurrent caries.⁴¹ In Class II compomer restorations in primary teeth,
- 428 the risk of developing secondary caries and failure did not increase over a two-year period in primary
- 429 molars.^{49,88} Compomers also have reported comparable clinical performance to composite with respect to
- 430 color matching, cavosurface discoloration, anatomical form, and marginal integrity and secondary
- 431 caries.^{89,90} Most randomized clinical trials showed that compomer tends to have better physical properties
- 432 compared to glass ionomer and resin modified glass ionomer cements and in primary teeth, but no
- 433 significant difference was found in cariostatic effects of compomer compared to these materials.^{44,88,91}
- 434

435 *Recommendations:*

- 436 1. Componers can be an alternative to other restorative materials in the primary dentition in Class I437 and Class II restorations.
- 438 2. There is not enough data comparing compomers to other restorative materials in permanent teeth of439 children.
- 440

441 Preformed metal crowns

442 Preformed metal crowns (also known as SSCs) are prefabricated metal crown forms that are adapted to

- 443 individual teeth and cemented with a biocompatible luting agent. Preformed metal crowns have been
- indicated for the restoration of primary and permanent teeth with extensive caries, cervical decalcification
- and/or developmental defects (eg, hypoplasia, hypocalcification), when failure of other available
- restorative materials is likely (eg, interproximal caries extending beyond line angles, patients with
- 447 bruxism), following pulpotomy or pulpectomy, for restoring a primary tooth that is to be used as an
- 448 abutment for a space maintainer, for the intermediate restoration of fractured teeth, for definitive
- 449 restorative treatment for high caries-risk children, and used more frequently in patients whose treatment is
- 450 performed under sedation or general anesthesia.⁹²
- 451
- 452 There are very few prospective randomized clinical trials comparing outcomes for preformed metal

crowns to intracoronal restorations.^{93,94} A Cochrane review and two systematic reviews conclude that the 453 454 majority of clinical evidence for the use of preformed metal crowns has come from nonrandomized and 455 retrospective studies.^{13,95-97} However, this evidence suggests that preformed metal crowns showed greater 456 longevity than amalgam restorations,¹³ despite possible study bias of placing SSCs on teeth more damaged by caries.^{95,96,98} Five studies which retrospectively compared Class II amalgam to preformed 457 metal crowns showed an average five year failure rate of 26 percent for amalgam and seven percent for 458 preformed metal crowns.96 459

- 460
- 461 A two-year randomized control trial regarding restoration of primary teeth that had undergone a
- pulpotomy procedure found a non-significant difference in survival rate for teeth restored with preformed 462
- metal crowns (95 percent) versus resin modified glass ionomer/composite restoration (92.5 percent).⁹³ In 463
- 464 another prospective study, significantly less restoration failure and improved calcium hydroxide
- pulpotomy success was found with preformed metal crowns (79.7 percent) versus amalgam restorations 465
- 466 (60 percent) after one year.⁹⁹ However, a systematic review did not show strong evidence that preformed
- metal crowns were superior over other restorations for pulpotomized teeth.¹⁰⁰ 467
- 468

469 With regards to gingival health adjacent to preformed metal crowns, a one year randomized controlled 470 trial showed no difference in gingival inflammation between preformed metal crowns and composite restorations after pulpotomy.⁹⁵ Yet, a two year randomized clinical study showed more gingival bleeding 471 for preformed metal crowns vs. composite/glass ionomer restorations.⁹³ Inadequately contoured crown 472 473 and residues of set cement remaining in contact with the gingival sulcus are suggested as reasons for gingivitis associated with preformed metal crowns, and a preventive regime including oral hygiene 474 475 instruction is recommended to be incorporated into the treatment plan.96

476

477 There is one randomized control trial on preformed metal crowns versus cast crowns placed on permanent teeth.¹⁰¹ and this report found no difference between the two restoration types for quality and longevity 478 479 after 24 months. The remaining evidence is case reports and expert opinion concerning indications for use 480 of preformed metal crowns on permanent molars. The indications include teeth with severe 481 genetic/developmental defects, grossly carious teeth, traumatized teeth, along with tooth developmental 482 stage or financial considerations that require semi-permanent restoration instead of a permanent cast restoration.^{97,98,101} The main reasons for pre-formed metal crown failure reportedly are crown loss^{13,99,102} 483 484 and perforation.¹⁰²

485

Hall Technique (HT). The Hall technique calls for cementation of a SSC over a caries-affected primary
molar without local anesthetic, caries removal or tooth preparation. It is a less invasive caries

- 488 management procedure for treating carious primary teeth, which involves the concept of caries control by
- 489 managing the activity of the biofilm.¹⁰³This technique was developed for use when delivery of ideal
- 490 treatment was not feasible. Crowns fitted using the Hall Technique may reduce discomfort from local
- 491 <u>anesthetic and caries removal at the time of treatment compared to fillings</u>,¹⁰⁴ but may add the discomfort
- 492 of placement of separator bands prior to the SSC/HC, as well as the pain from biting the SSC/HC into
- 493 <u>place.¹⁰⁵ While The HT has gained some popularity in the United Kingdom (U.K.),¹⁰⁴ primarily from use</u>
- 494 by general dentists, who provide the majority of care for young children.¹⁰⁶ the technique is highly
- 495 controversial in the U.S. All prospective investigations on the effectiveness of HT have been by general
- 496 dentists in the U.K. who provide care for the majority of young children and comparison groups include
- 497 restorative treatment as traditionally provided in those settings, where. Traditional use of SSCs to restore
- 498 caries in primary teeth has not been a popular or a frequently used technique. in the U.K., This is in spite
- 499 of the existence of guidelines and policy statements from the British Society of Paediatric Dentistry that
- 500 SSCs are the restoration of choice for primary molars with multisurface lesions or extensive caries, or
- 501 when pulp treatment has been performed.¹⁰⁷⁻¹¹⁰
- 502

Results of a 2003 repeat questionnaire of general dentists in the U.K. showed that the use of amalgam had

- declined with an increase in the use of glass ionomer cement (GIC) and very little change in the use of
- 505 SSCs.¹⁰⁷ Placement of GIC restorations or observation without treatment was the management approach
- of choice, and the use of local anesthesia to provide dental care to children was infrequent.¹⁰⁸ A recent-
- 507 systematic review indicates that HT is more effective when compared to GIC restorations in-
- 508 compromised, caries affected primary molars,(Seale and Randall 2015) but it points out that reliable
- 509 Given the differences in treatment approaches in health care settings and systems between countries, the
- 510 Hall technique has not been widely adapted in the US, and it is usually limited to individual situations
- 511 where proven methods of caries management cannot be used. sStudies that compare this technique to
- traditionally placed SSCs using radiographic assessment and caries removal are needed.¹¹¹
- 513
- 514 <u>A recent retrospective study for cost-effectiveness combined with a cross-sectional evaluation of patient</u>
- 515 acceptance showed that 95.8% of primary teeth restored using the HT remained asymptomatic after a
- 516 <u>follow-up period of up to 77 months; compared to 95.3% in the conventional methods (caries removal</u>
- 517 and placing SSC or other restorative material)l, but does not report a breakdown by follow-up time.
- 518 Although both approaches had similar successful outcomes, using the Hall Technique was also associated

- 519 with reduced treatment costs, and both approaches were accepted favorably by the children and care
- 520 providers.¹¹².
- 521
- 522 <u>SSCs continue to offer the advantages of full coverage to combat recurrent caries and provide strength as</u>
- 523 well as long term durability with minimal maintenance, both desirable outcomes for caries management
- 524 <u>on high-risk children.</u>
- 525

526 *Recommendations:*

- 527 1. There is evidence from retrospective studies showing greater longevity of preformed metal crown
- 528 restorations compared to amalgam <u>or resin-based</u> restorations for the treatment of carious lesions in
- 529 primary teeth. <u>Therefore, its use is supported on high risk children with large or multiple-surface</u>
- 530 lesions on primary molars, especially when children require general anesthesia for the provision of
 531 restorative dental care.
- 532 2. There is evidence from case reports and one randomized controlled trial supporting the use of
 533 preformed metal crowns in permanent teeth as a semi-permanent restoration for the treatment of
 534 severe enamel defects or grossly carious teeth.
- 535

536 Anterior esthetic restorations in primary teeth

537 Despite the continuing prevalence of dental caries in primary maxillary anterior teeth in children, the 538 esthetic management of these teeth remains problematic.¹¹³ Esthetic restoration of primary anterior teeth 539 can be especially challenging due to: the small size of the teeth; close proximity of the pulp to the tooth 540 surface; relatively thin enamel; lack of surface area for bonding; and issues related to child behavior.¹¹³

541

There is little scientific support for any of the clinical techniques that clinicians have utilized for many years to restore primary anterior teeth, and most of the evidence is regarded as expert opinion. While a lack of strong clinical data does not preclude the use of these techniques, it points out the strong need for well designed, prospective clinical studies to validate the use of these techniques.¹¹⁴ Additionally, there is limited information on the potential psychosocial impact of anterior caries or unaesthetic restorations in primary teeth.¹¹³

548

549 Class III (interproximal) restorations of primary incisors are often <u>can be</u> prepared with labial or lingual

- 550 dovetails to incorporate a large surface area for bonding to enhance retention.¹¹⁵ Resin-based restorations
- are appropriate for anterior teeth that can be adequately isolated from saliva and blood. Resin-modified

- 552 glass ionomer cements have been suggested for this category, especially when adequate isolation is not
- 553 possible.^{116,117} It has been suggested that patients considered at high-risk for future caries may be better
- served with placement of full tooth coverage restorations.¹¹⁷
- 555
- 556 Class V (cervical) cavity preparations for primary incisors are similar to those in permanent teeth. Due to
- the young age of children treated and associated behavior management difficulty, it is sometimes
- 558 impossible to isolate teeth for the placement of composite restorations. In these cases, glass ionomer
- cement or resin-modified glass ionomer cement is suggested.^{116,117}
- 560
- 561 Full coronal restoration of carious primary incisors may be indicated when: (1) caries is present on
- 562 multiple surfaces, (2) the incisal edge is involved, (3) there is extensive cervical decalcification, (4) pulpal
- therapy is indicated, (5) caries may be minor, but oral hygiene is very poor, or (6) the child's behavior
- 564 makes moisture control very difficult.¹¹⁵ Successful full-coronal restorations of extensively decayed
- 565 primary anterior teeth have been reported; however, due to the lack of available clinical studies, it is
- 566 difficult to determine whether certain techniques of restoring carious primary anterior teeth are
- 567 effective.^{114,118} A retrospective study showed that 80 percent of strip crowns were completely retained
- after three years, and 20 percent were partially retained, with none being completely lost.¹¹⁹ Another
- retrospective study, with 24-74 months follow-up, reported 80 percent retention of strip crowns.¹²⁰
- 570
- 571 Pre-veneered stainless steel crowns also are among the options of restoring primary anterior teeth with
- 572 full coronal coverage. Three retrospective studies report excellent clinical retention of these types of
- 573 crowns, yet with a high incidence of partial or complete loss of the resin facings.^{113,121,122} <u>The pre-veneer</u>
- 574 <u>stainless crowns have the concerns of color stability and surface roughness changes</u>,¹²³ so long term
- 575 <u>clinical studies are required to establish their comparative effectiveness.</u> Pre-formed stainless steel crowns
- 576 and open-faced stainless steel crowns are other options; however, there appears to be no published data on
- 577 the use of either crown on primary anterior teeth.(Roshan et al 2003) still an option for treatment on
- 578 primary anterior teeth, but published studies reporting their effectiveness and use are sparse,¹⁰⁷ given the
- 579 more esthetic and easier to use alternatives.
- 580
- 581 <u>Preformed pediatric zirconia crowns are another option for esthetic full coronal coverage</u>
- 582 restoration.¹²⁴As they require a passive fit, the amount of tooth reduction is greater than that required for
- 583 stainless steel crowns (minimum of 1.5-2.0 mm), and technique for tooth preparation does vary
- 584 <u>significantly among different brands.¹²⁵ There are several preformed pediatric zirconia crowns available</u>

585	on the market and each brand has very different material composition, fabrication, surface treatment,			
586	retentive feature and cementation methods. Although an RCT with a follow-up of only six months			
587	suggests that zirconia crowns gave significantly better results than the others with regard to gingival			
588	health and crown fractures (reference 1), a systematic review on the topic (reference 2) concluded that			
589	due to the small number of RCTs on this topic and their risk of bias, future RCTs with better study			
590	design are required to compare differences between the different types of pediatric preformed zirconia			
591	crowns and between other esthetic treatment options.			
592				
593	Recommendations:			
594	1. There is expert opinion that suggests the use of resin-based composites as a The use of resin-			
595	modified glass ionomer cement is a viable treatment option for Class III and Class V restorations in			
596	the primary and permanent dentition. (Based on expert opinion)			
597	2. There is expert opinion that suggests tThe use of resin-modified glass ionomer cement as a is a			
598	viable treatment option for Class III and Class V restorations for primary teeth, particularly in			
599	circumstances where adequate isolation of the tooth to be restored is difficult. (Based on expert			
600	opinion)			
601	3. There is expert opinion that suggests that sStrip crowns, pre-veneered stainless steel crowns,			
602	preformed stainless steel crowns, and open-faced stainless steel crowns and zirconia crowns are a			
603	viable treatment options for full coronal coverage restorations in primary anterior teeth. (Based on			
604	expert opinion)			
605				
606	Posterior esthetic restorations in primary teeth			
607	Scientific studies that evaluate esthetic options for large carious lesion of requiring full coverage			
608	restoration for primary posterior teeth are not widely reported in the literature. The most popular options			
609	are open-faced stainless-steel crowns, pre-veneered stainless-steel crowns or zirconia crowns. The interest			
610	in esthetic options from the clinicians as well as patients is increasing. ^{128,129} The indications for the			
611	preformed esthetic crowns are generally the same as those of the preformed stainless-steel crowns but			
612	with consideration of esthetics. ¹³⁰ The amount of tooth reduction and technique for tooth preparation does			
613	vary significantly. ¹²⁵ There is need for more circumferential tooth reduction requirements for proper fit			
614	and placement for zirconia crowns compared to SSC. ¹²⁵ SSCs have comparatively better retention, but a			
615	recent study demonstrates that the gingival health plaque accumulation around a zirconia crowns is better			
616	than SSC. ^{130,131}			

617

618

Table 1. EVIDENCE OF EFFICACY OF VARIOUS DENTAL MATERIALS/TECHNIQUES IN PRIMARY TEETH WITH REGARD TO CAVITY PREPARATION CLASSIFICATIONS

Strong evidence – based on well executed randomized control trials, meta-analyses, or systematic reviews; Evidence in favor – based on weaker evidence from clinical trials; Expert opinion – based on retrospective trials, case reports, in vitro studies and opinions from clinical researchers; Evidence against – based on randomized control trials, meta-analysis, systematic reviews.

	Class I	Class II	Class III	Class IV	Class V
Amalgam	Strong evidence	Strong evidence	No data	No data	Expert opinion
Composite	Strong evidence	Expert opinion- Strong Evidence	Expert opinion	No data	Evidence in favor
Glass ionomer	Strong evidence α	Evidence against $^{\beta}$	Evidence in favor $\boldsymbol{\gamma}$	No data	Expert opinion γ
RMGIC	Strong evidence	Expert opinion ^ɛ	Expert opinion	No data	Expert opinion
Compomers	Evidence in favor	Evidence in favor	No data	No data	Expert opinion
SSC	Evidence in favor δ	Evidence in favor $^{\delta}$	No data	No data	No data
Anterior ^φ crowns	N/A	N/A	Expert opinion	Expert opinion	Expert opinion

619

620 RMGIC = resin modified glass ionomer cement.

- **621** α Evidence from ART trials.
- **622** β Conflicting evidence for multisurface ART restorations.

SSC = stainless steel crown.

 γ Preference when moisture control is an issue.

162

- $\phi \qquad Strip \ crowns, \ stainless \ steel \ crowns \\$
- with/without facings.
- Small restorations; life span 1-2 years.
- 624 625

623

3

δ Large lesions.

Table 2.EVIDENCE OF EFFICACY OF VARIOUS DENTAL MATERIALS/TECHNIQUES IN PERMANENT
TEETH WITH REGARD TO CAVITY PREPARATION CLASSIFICATIONS

	Class I	Class II	Class III	Class IV	Class V
Amalgam	Strong evidence	Strong evidence	No data	No data	No data
Composite	Strong evidence	Evidence in favor	Expert opinion	No data	Evidence in favor
Glass ionomer	Strong evidence α	Evidence against	Evidence in favor $^{\beta}$	No data	Expert opinion β
RMGIC	Strong evidence	No data	Expert opinion	No data	Evidence in favor
Compomers	Evidence in favor ϕ	No data	Expert opinion	No data	Expert opinion
SSC	Evidence in favor γ	Evidence in favor γ	No data	No data	No data
Anterior ^δ crowns	N/A	N/A	No data	No data	No data

626

627

620	DI	ACIC main modified along innerner comment	
628	KIV	AGIC = resin modified glass ionomer cement.	
629	α	Evidence from ART trials.	1
630			
631	β	Preference when moisture control is an issue.	č
632			

633 ϕ Evidence from studies in adults.

- SSC = stainless steel crown.
- γ For children and adolescents with gross caries or severely hypoplastic teeth.
- $\delta \qquad Strip \ crowns, \ stainless \ steel \ crowns \ with/without \ facings.$

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634		
635		
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1 [Best Practices]

² Use of Antibiotic Therapy for Pediatric Dental Patients^{*}

- 3 Review Council
- 4 Council on Clinical Affairs
- 5 Latest Revision
- 6 2014 2019
- 7
- 8 Purpose
- 9 The American Academy of Pediatric Dentistry (AAPD) recognizes the increasing prevalence of
- 10 antibiotic-resistant micro-organisms and potential for adverse drug reactions and interactions. This
- 11 guideline is intended to provide guidance in the proper and judicious use of antibiotic therapy in the
- 12 treatment of oral conditions.(Wilson et al 2007)
- 13

14 Methods

- 15 This guideline was originally developed by the Council on Clinical Affairs and adopted in 2001. This
- 16 document is a revision of the previous version, last revised in <u>20092014</u>. The <u>last</u> revision was based upon
- a new systematic literature search of the PubMed®/MEDLINE database using the terms: antibiotic
- 18 therapy, antibacterial agents, antimicrobial agents, dental trauma, oral wound management, orofacial
- 19 infections, periodontal disease, viral disease, and oral contraception; fields: all; limits: within the last 10
- 20 years, humans, English, clinical trials, birth through age 18. One hundred sixty-five articles matched these
- 21 criteria. Papers for review were chosen from this search and from hand searching. When data did not
- 22 appear sufficient or were inconclusive, recommendations were based upon expert and/or consensus
- 23 opinion by experienced researchers and clinicians. <u>This update is based on a review of current literature.</u>
- 24

25 Background

- 26 Antibiotics are beneficial in patient care when prescribed and administered correctly for bacterial
- 27 infections. However, the widespread use of antibiotics has permitted common bacteria to develop
- resistance to drugs that once controlled them.^{1.2.3} (Wilson et al 2007) Drug resistance is prevalent
- throughout the world.³ In the United States, at least 2 million people are infected by antibiotic-resistant
- 30 <u>bacteria per year.²</u> Some microorganisms may develop resistance to a single antimicrobial agent, while

* ABBREVIATION

AAPD: American Academy Pediatric Dentistry.

31	others develop multidrug-resistant strains.3 (CDC Antibiotic/Antimicrobial resistance) To diminish the
32	rate at which resistance is increasing, health care providers must be prudent in the use of antibiotics. ²
33	(Wilson et al 2007) Conservative use of antibiotics is indicated to minimize the risk of developing
34	resistance to current antibiotic regimens. ^{2,3} Adverse events such as allergic reactions, development of C.
35	difficile, or drug interactions and side effects can occur. ⁴ The Centers for Disease Control and Prevention
36	report that every year there are 140,000 emergency department visits for reactions to antibiotics, and that
37	antibiotics are the most common cause of emergency department visits for adverse drug events in children
38	under the age of 18 years. ⁴ Practitioners should adhere to the following general principles when
39	prescribing antibiotics for the pediatric population.
40	
41	The use of antibiotic prophylaxis for dental patients at risk for infection is addressed in a separate
42	document. ⁵
43	
44	For a description of useful antibiotics, please see "Useful Medications for Oral Conditions" in the
45	Resources Section. ⁶
46	
47	Recommendations
48	Conservative use of antibiotics is indicated to minimize the risk of developing resistance to current-
49	antibiotic regimens.(CDC Antibiotic/Antimicrobial resistance, Costelloe et al 2010) Practitioners should
50	adhere to the following general principles when prescribing antibiotics for the pediatric population.
51	
52	Oral wound management Oral Wounds
53	Factors related to host risk (e.g., age, systemic illness, co-morbidities, malnutrition) and type of wound
54	(e.g., laceration, puncture) must be evaluated when determining the risk for infection and subsequent need
55	for antibiotics. Wounds can be classified as clean, potentially contaminated, or contaminated/dirty. Facial
56	puncture wounds and lacerations may require topical antibiotic agents. ⁷ Intraoral puncture wounds and
57	lacerations that appear to have been contaminated by extrinsic bacteria, debris (dirt, soil, gravel), foreign
58	body, open fractures, and joint injury have an increased risk of infection and should be covered managed
59	with systemic antibiotics. ⁷ Tetanus immunization status should be determined. If it is determined that
60	antibiotics would be beneficial to the healing process, the timing of the administration of antibiotics is
61	critical to supplement the natural host resistance in bacterial killing. The drug should be administered as
62	soon as possible for the best result. The most effective route of drug administration (intravenous vs.
63	intramuscular vs. oral) must be considered. The clinical effectiveness of the drug must be monitored. The

64	minimal duration of drug therapy should be five days beyond the point of substantial improvement or
65	resolution of signs and symptoms; this is usually a five- to seven-day course of treatment dependent upon
66	the specific drug selected. ⁸⁻¹⁰ In light of the growing problem of drug resistance, the clinician should
67	consider altering or discontinuing antibiotics following determination of either ineffectiveness or cure
68	prior to completion of a full course of therapy. ¹¹ If the infection is not responsive to the initial drug
69	selection, a culture and <u>sensitivitysusceptibility</u> testing of a <u>swab</u> isolates from the infective site <u>or, in</u>
70	some cases, a blood microbiology, culture and sensitivity may be indicated.
71	
72	Special conditions
73	Pulpitis/apical periodontitis/draining sinus tract/localized intra- oral swelling
74	Bacteria can gain access to the pulpal tissue through caries, exposed pulp or dentinal tubules, cracks into
75	the dentin, and defective restorations. If a child presents with acute symptoms of pulpitis, treatment (i.e.,
76	pulpotomy, pulpectomy, or extraction) should be rendered. Antibiotic therapy usually is not indicated nor
77	effective if the dental infection is contained within the pulpal tissue or the immediate surrounding tissue.
78	In this case, the child will have no systemic signs of an infection (i.e., no fever and no facial swelling). ^{1}
79	(Maestre-Vera 2004, Keenan et al 2006)
80	
81	Consideration for use of antibiotics should be given in cases of advanced non-odontogenic bacterial
82	infections such as staphylococcal mucositis, tuberculosis, gonococcal stomatitis, and oral syphilis. If
83	suspected, it is best to refer patients for microbiology, culture and sensitivity, biopsy, or other laboratory
84	tests for documentation and definitive treatment.
85	
86	Acute facial swelling of dental origin
87	A child presenting with a facial swelling or facial cellulitis secondary to an odontogenic infection should
88	receive prompt dental attention. In most situations, immediate surgical intervention is appropriate and
89	contributes to a more rapid cure. ¹² The clinician should consider age, <u>cooperation</u> , the ability to obtain
90	adequate anesthesia (local vs. general), the severity of the infection, the medical status, and any social
91	issues of the child. ^{12,13} Signs of systemic involvement and septicemia (i.e., e.g., fever, malaise,
92	asymmetry, facial swelling, lymphadenopathy, trismus, tachycardia, dysphagia, respiratory distress)
93	warrant emergency treatment. Additional testing such as a complete blood exam, c-reactive protein, blood
94	cultures, and bacterial culture and sensitivity can aide in assessment and diagnosis. Intravenous antibiotic
95	therapy and/or referral for medical management is may be indicated. ^{12,13} (Maestre-Vera 2004, Keenan et-
96	al 2006) Penicillin derivatives remains the empirical choice for odontogenic infections; however,

- 97 consideration of additional adjunctive antimicrobial therapy (i.e., metronidazole) can be given where
- 98 there is anaerobic bacterial involvement.^{11,14} <u>Cephalosporins could be considered as an alternative choice</u>
- 99 <u>for odontogenic infections.¹⁴</u>
- 100
- 101 Dental <u>trauma</u>
- 102 Systemic antibiotics have been recommended as adjunctive therapy for avulsed permanent incisors with
- 103 an open or closed apex.¹⁵⁻¹⁷ (Andreasen and Andreasen 2007) Tetracycline (doxycycline twice daily for
- seven days) is the drug of choice, but consideration of the child's age must be exercised in the systemic
- use of tetracycline due to the risk of discoloration in the developing permanent dentition. (Rega et al-
- 106 2006, Andreasen and Andreasen 2007) Penicillin V or amoxicillin can be given as an alternative in
- 107 patients under 12 years of age.^{15,17}(Andreasen and Andreasen 2007) The use of topical antibiotics
- 108 (minocycline or doxycycline) to enhance induce pulpal revascularization and periodontal healing in
- 109 immature non-vital traumatized teeth has shown some potential.^{15,17,18} (Andreasen and Andreasen 2007)
- 110 However, further randomized clinical trials are needed.^{19,20} (Thibodeau et al 2007) For luxation injuries in
- 111 the primary dentition, antibiotics generally are not indicated.^{15,21} Antibiotics can be warranted in cases of
- 112 concomitant soft tissue injuries (see **Oral wound management**) and when dictated by the patient's
- 113 medical status.
- 114

115 *Pediatric periodontal diseases*

- 116 <u>Gingival inflammation due to the presence of bacterial plaque accumulation is a key factor in the</u>
- development of periodontal disease and must be controlled.²² However, a distinction must be made
- 118 <u>between a site of gingival inflammation versus a gingival case, diagnosed at the patient level, using</u>
- 119 specific criteria, including bleeding on probing.²³ Periodontal diseases are now classified as necrotizing
- 120 periodontal disease, periodontitis as manifestation of systemic diseases, and periodontitis.²⁴ The specifics
- 121 of these new classifications are addressed in a future document.²⁵ Dental plaque-induced gingivitis is
- 122 managed by appropriate local therapeutic interventions²² including professional oral hygiene and re-
- 123 <u>enforcement of brushing twice daily for at last 2 minutes.²⁶ does not require antibiotic therapy</u>. Treatment-
- 124 recommendations have not yet been made for these new periodontal classifications. Based on previous
- 125 definitions of periodontal diseases, Ppediatric patients with aggressive periodontal diseases may require
- 126 adjunctive antimicrobial therapy in conjunction with localized treatment.^{27,28} In pediatric periodontal
- 127 diseases associated with systemic disease (e.g., severe congenital neutropenia, Papillon-Lefèvre
- syndrome, leukocyte adhesion deficiency), the immune system is unable to control the growth of
- 129 periodontal pathogens and, in some cases, treatment may involve antibiotic therapy.^{27, 28} The use of

130	systemic antibiotics has been recommended as adjunctive treatment to mechanical debridement in patients
131	with aggressive periodontal disease. (AAPerio 2003, Schmidt et al 2013 In severe and refractory cases,
132	extraction is indicated. ^{27,28} Culture and susceptibility testing of isolates from the involved sites are helpful
133	in guiding the drug selection. ^{27,28}
134	
135	Viral diseases
136	Conditions of viral origin such as acute primary herpetic gingivostomatitis should not be treated with
137	antibiotic therapy. ⁴ unless there is strong evidence to indicate that a secondary bacterial infection-
138	exists.(AAP 2003).
139	
140	Salivary gland infections
141	For acute salivary gland swellings of bacterial nature, antibiotic therapy is indicated. ²⁹ If the patient does
142	not improve in 24 – 48 hours on antibiotics alone, incision and drainage may be warranted. ²⁹ Many-
143	salivary gland infections, following confirmation of bacterial etiology, will respond favorable to antibiotic
144	therapy. Acute bacterial parotitis has two forms: hospital acquired and community acquired.(Carlson-
145	2009) Both can be treated with antibiotics. Amoxicillin/clavulanate is used as empirical therapy to cover
146	both staphylococcal and streptococcal species as most bacterial infections of the salivary glands originate
147	from oral flora. ²⁹ Clindamycin is appropriate for penicillin allergic patients. ²⁹ The most common
148	inflammatory salivary gland disorder in the United States is juvenile recurrent parotitis (JRP), with first
149	onset of symptoms between ages 3 – 6 years old, continuing to puberty. ²⁹ Although JRP is self-limiting,
150	administration of B-lacatam antibiotics can shorten symptom duration. ²⁹ Hospital acquired usually-
151	requires intravenous antibiotics; oral antibiotics are appropriate for community acquired. Chronic-
152	recurrent juvenile parotitis generally occurs prior to puberty. Antibiotic therapy is recommended and has
153	been successful.(Carlson 2009) For both acute bacterial submandibular sialadenitis and chronic recurrent
154	submandibular sialadenitis, antibiotic therapy is included as part of the treatment. ³⁰
155	
156	Oral contraceptive use
157	Although caution is advised with the concomitant use of antibiotics and oral contraceptives, ^{31,32} a 2018
158	systematic review of drug interactions between non-rifamycin antibiotics and hormonal contraception
159	found that most women can expect no reduction in hormonal contraceptive effect with the concurrent use
160	of non-rifamycin antibiotics. ³³ The World Health Organization (WHO), in 2015, also reported that most
161	broad-spectrum antibiotics do not affect the contraceptive effectiveness of combined oral contraceptives,
162	combined contraceptive patch, or the combined contraceptive vaginal ring. ³⁴ In addition, no differences in

- 163 <u>ovulation were found when oral contraceptives were combined with ampicillin, doxycycline,</u>
- 164 temafloxacin, ofloxacin, ciprofloxacin, clarithromycin, roxithromycin, dirithromycin, or metronidazole.³³
- 165 <u>Women should be encouraged to take oral contraceptives correctly and consistently at all times, including</u>
- 166 <u>during periods of illness.³³</u>
- 167
- 168 <u>Rifamcyin antibiotics, such as rifampin or rifabutin, induce hepatic enzymes that are required for</u>
- 169 hormonal contraceptive metabolism, which could compromise the contraceptive or antibiotic effect.^{33,34}
- 170 Use of other contraceptives should be advised with long-term use of these medications.³⁴ Whenever an-
- 171 antibiotic is prescribed to a female patient taking oral contraceptives to prevent pregnancy, the patient-
- 172 must be advised to use additional techniques of birth control during antibiotic therapy and for at least one-
- 173 week beyond the last dose, as the antibiotic may render the oral contraceptive ineffective.(DeRossi and
- 174 Hersh 2002, Becker 2011) Rifampicin has been documented to decrease the effectiveness of oral-
- 175 contraceptives. (DeRossi and Hersh 2002, Becker 2011) Other antibiotics, particularly tetracycline and
- 176 penicillin derivatives, have been shown to cause significant decrease in the plasma concentrations of
- 177 ethinyl estradiol, causing ovulation in some individuals taking oral contraceptives. (DeRossi and Hersh-
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- 1 [Best Practices]
- 2 Informed Consent*
- 3
- 4 Review Council
- 5 Council on Clinical Affairs
- 6 Latest Revision
- 7 2015 2019
- 8

9 Purpose

10 The American Academy of Pediatric Dentistry (AAPD) recognizes that informed consent is essential

11 in the delivery of health care. The informed consent process allows the patient or, in the case of

12 minors, the parent[†] to participate in and retain autonomy over the health care received. Informed

13 consent also may decrease the practitioner's liability from claims associated with miscommunication.

14 This guideline recognizes that Informed consent is governed by the statutes and case laws of

15 individual states; oral health care providers should review the applicable laws and regulations of their

- 16 state.
- 17

18 Methods

19 This guideline was Originally developed by the Council on Clinical Affairs and adopted in 2005,

20 This this document is a revision of the previous version. last Last revised in 2014 2015, This this

- 21 revision included a systematic literature search of the PubMed®/MEDLINE database using the terms:
- 22 informed consent, pediatric consent, pediatric informed consent, consent, informed refusal, cultural
- 23 background informed consent, linguistic background informed consent, and interpreters informed
- 24 consent: fields: all; limits: within the last 10 years, humans, English, review of legal cases. One-
- 25 hundred fifty four One hundred forty two articles matched these criteria. Papers for review were

* ABBREVIATIONS

ADA: American Dental Association. AAPD: American Academy Pediatric Dentistry.

⁺ In all AAPD oral health care policies and clinical practice guidelines, the term "parent" has a broad meaning encompassing a natural/biological father or mother of a child with full parental legal rights, a custodial parent who in the case of divorce has been awarded legal custody of a child, a person appointed by a court to be the legal guardian of a minor child, or a foster parent (a noncustodial parent caring for a child without parental support or protection who was placed by local welfare services or a court order). American Academy of Pediatric Dentistry. Reference Manual: Introduction. Pediatr Dent 2015;37(special issue):2-3.

- 26 chosen from this list and from references within selected articles. When data did not appear sufficient
- 27 or were inconclusive, recommendations were based upon expert and/ or consensus opinion by
- 28 experienced researchers and legal practitioners.
- 29

30 Background

- 31 Informed consent is the process by which a health care provider gives relevant information
- 32 concerning diagnosis and treatment needs to a patient of providing that the patient or, in the case of a
- 33 minor or incompetent adult, the parent, so with relevant information regarding diagnosis and
- 34 treatment needs so that the patient or parent can make a an voluntary, educated decision regarding to
- 35 accept or refuse treatment. can be made by the patient or parent. Minor children are legally unable to
- 36 give informed consent, and intellectually disabled adults lack capacity to give consent. Parents are
- 37 authorized to grant or decline permission for treatment with assent or agreement from the child or
- 38 incompetent adult whenever possible.¹⁻⁴ All requirements of informed consent apply when the parent
- 39 is acting on behalf of the child.^{1,3}
- 40
- 41 Informed consent involves both ethical and legal obligations of the health care provider to the patient.

42 The American Dental Association (ADA) states that dentists are "required to provide information to

- 43 patients/parents about the dental health problems the dentist observes, the nature of any proposed
- 44 treatment, the potential benefits and risks associated with that treatment, any alternatives to the
- 45 treatment proposed, and the potential risks and benefits of alternative treatments, including no
- 46 treatment."⁵Following the informed consent discussion, an assessment of patient/parental
- 47 understanding should be made, and any confusion about the treatment should be clarified by the
- 48 provider before consent is granted.^{2,6}
- 49

50 State laws and court decisions determine the criteria for informed consent. (De Bord 2014) (Sfikis-

51 2003) Autonomy over healthcare decisions is a patient's right. A In 1914, a New York state court

- 52 ruled that "every human being of adult years and sound mind has a right to determine what shall be
- 53 done with his own body...."7-Although most cases have involved other health professionals, oral-
- 54 health care providers should follow the rulings established by these cases. Additionally, Ruling
- 55 ruling from the Supreme Court of North Dakota found that laws pertaining to a physician's duty to
- obtain informed consent also pertained to dentists.⁸ As court rulings and laws differ in each state, it is 56
- 57 difficult to develop an inclusive guideline.

58

50	
59	The law generally has several criteria for selecting information to provide to a patient/parent as part
60	of an informed consent. Some states follow a patient-oriented standard-that information which a
61	reasonably prudent patient/parent in same or similar circumstances would wish to know.9,10,11 Other
62	states follow a practitioner-oriented standard-that information which a health care provider,
63	practicing within the standard of care, would reasonably provide to a patient/parent in the same
64	circumstances.9,10,12 A hybrid approach, combining the patient-oriented and practitioner-oriented
65	standards, is followed by some states. ^{10,12} Finally, a subjective person standard requires the practioner
66	to give information that the particular patient in question would want to know. ^{2,9}
67	
68	Regardless of the standard a state has chosen to follow, the treating practitioner must disclose
69	information that he/she considers material to the patient's/parent's decision-making process and
70	provide a warning of death or serious bodily injury where that is a known risk of the procedure. ^{10,13}
71	The informed consent process generally excludes adverse consequences associated with a simple
72	procedure if the risk of occurrence is considered remote and when such circumstances commonly are
73	understood by the profession to be so.
74	
/ 4	
75	It generally is understood that the person granting consent is the patient of the age of majority. For-
	It generally is understood that the person granting consent is the patient of the age of majority. For- Patients under the age of majority or adults with diminished mental capacity, informed consent
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75 76	Patients under the age of majority or adults with diminished mental capacity, informed consent
75 76 77	Patients under the age of majority or adults with diminished mental capacity, informed consent – permission should be obtained from a parent. (Sfikis 2003, LeBlang et al 2004) The ADA code of
75 76 77 78	<u>Patients</u> under the age of majority or adults with diminished mental capacity, informed consent – <u>permission</u> should be obtained from a parent. (Sfikis 2003, LeBlang et al 2004) <u>The ADA code of</u> <u>ethics recommends that dentist provide information "in a manner that allows the patient to become</u>
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91 may not be allowed to consent to medical procedures, according to individual state law. It is advisable 92 that the oral health care provider obtain a copy of court orders appointing a guardian to verify who is 93 authorized to consent for medical treatment for the patient.¹⁶(Sfikis 2003) One option to consider is 94 obtaining a parent's authorization via a consent by proxy or power of attorney agreement for any other individual to make dental treatment decisions for a child.^{10,16} In situations where individuals 95 96 other than the parent regularly bring the child to the dental office, this can help eliminate doubt as to 97 whether such individual has the legal authority to provide informed consent. Practitioners, however, 98 should consult their own attorney in deciding whether to utilize such a form in their own practice. 99 Another option for obtaining authorization for treatment is a telephone conversation with the 100 parent.^{16,17} (Australian Capital Territory Government Health Directorate 2012) The parent should be 101 told there are two people on the telephone and asked to verify the patient's name, date of birth, and 102 address and to confirm he/she has responsibility for the patient.¹⁷ (Australian Capital Territory-103 Government Health Directorate 2012) The parent is presented with all elements of a valid informed consent followed by documentation in the patient's chart with signatures.^{16,17} (Australian Capital-104

- 105 Territory Government Health Directorate 2012)
- 106

Written consent is required by some most states before treatment of a patient.^{10,} (Sfikis 2003) Even if 107 108 not mandated by state law, written consent is advisable as it may decrease the liability from 109 miscommunication.¹⁷ A patient's or parent's signing a consent form should not preclude a thorough 110 discussion. Studies have shown that even when seemingly adequate information has been presented to 111 patients/parents, their ability to fully understand the information may be limited.^{6,9} Dentists should be 112 aware of the cultural and linguistic backgrounds of their patients and families and take care to ensure 113 that information is available in culturally and linguistically competent formats to help parents in the 114 decision-making process.¹⁹(Australian Capital Territory Government Health Directorate 2012) Also, 115 to assure a person who is deaf or hearing impaired can consent, a dentist carefully should consider the 116 patient's self-assessed communication needs before any treatment. Practitioners may need to provide 117 access to translation services (e.g., in person, by telephone, by subscription to a language line) and sign language services.^{1,19} (Chen et al 2007) Practitioners who receive federal funding, as well as 118 119 those in a significant number of states, are mandated to provide these services at no cost to the 120 patient.^{1,19} (Chen et al 2007) Supplements such as informational booklets, or wideos, or models may be 121 helpful to the patient in understanding a proposed procedure. The oral discussion between provider 122 and patient, not the completion of a form, is the important issue of informed consent. The consent 123 form should document the oral discussion of the proposed therapy, including risks, benefits, and

- possible alternative therapy, as well as no treatment.^{3,5,19} (Australian Capital Territory Government-124
- 125 Health Directorate 2012)
- 126

127 Informed refusal occurs when the patient/parent refuses the proposed and alternative treatments.^{5,17} 128 (Australian Capital Territory Government Health Directorate 2012) The dentist must inform the 129 patient/parent about the consequences of not accepting the proposed treatment and obtain a signed-130 informed refusal. It is recommended by the ADA that informed refusal be documented in the chart 131 and that the practitioner should attempt to obtain an informed refusal signed by the parent for 132 retention in the patient record. An informed refusal, however, does not release the dentist from the 133 responsibility of providing a standard of care.⁵ If the dentist believes the informed refusal violates 134 proper standards of care, he/she should recommend the patient seek another opinion and/or dismiss 135 the patient from the practice.⁵ If the dentist suspects dental neglect, appropriate authorities should be informed.18 136 137 138 When a consent form is utilized, it is best to use simple words and phrases. - avoiding technical terms, 139 so that it may be easily understood. A modified or customized consent form is preferred over a 140 standard form and should be in a format written so that is readily understandable to a lay person.^{3,5,17,19} (Australian Capital Territory Government Health Directorate 2012, Tait et al 2005)

141

Overly broad statements such as "any and all treatment deemed necessary..." or "all treatment which 142

143 the doctor in his/her best medical judgment deems necessary, including but not limited to..." should

144 be avoided. Courts have determined it to be so broad and unspecific that it does not satisfy the duty of

145 informed consent. Informed consent discussion, when possible, should occur on a day separate from

146 the treatment and the practitioner should avoid downplaying the risks involved with the proposed

147 therapy.⁶Items that should appear on a consent form are listed under Recommendations.

148

Informed consent and informed refusal forms²⁰ should be procedure specific, with multiple forms 149

150 likely to be used. Dentists should consult their own attorney and the state dental association as-

151 informed consent laws vary by state. For example, risks associated with restorative procedures will

152 differ from those associated with an extraction. Separate forms, or separate areas outlining each

153 procedure on the same form, would be necessary to accurately advise the patient regarding each

154 procedure.⁵ Consent for sedation, general anesthesia, or behavior guidance techniques such as

155 protective stabilization (i.e., immobilization) should be obtained separately from consent for other

procedures.^{4,21} Consent may need to be updated or changed accordingly as changes in treatment plans 156

- 157 occur. When a primary tooth originally planned for pulp therapy is determined to be nonrestorable at
- 158 the time of treatment, consent will need to be updated to reflect the change in treatment. <u>Depending</u>
- 159 on state laws, this update may be in oral or written form. Dentists should consult their own attorney
- 160 and state dental association as informed consent laws vary by state.⁵
- 161

162 Recommendations

- 163 Informed consent is the process of providing the patient or, in the case of a minor or incompetent-
- 164 adult, the parent with relevant information regarding diagnosis and treatment needs so that an
- 165 educated decision regarding treatment can be made by the patient. In the case of a minor or
- 166 intellectually disabled adult, the parent gives informed permission with assent or agreement from the
- 167 <u>child whenever possible. The oral discussion between provider and patient or parent, not the</u>
- 168 <u>completion of a form, is the important issue of informed consent. A written consent form serves as</u>
- 169 documentation of the consent process and is required by most states. Other states allow the oral
- 170 <u>discussion to be documented in the patient record</u>. Dentists should be aware of the cultural and
- 171 linguistic backgrounds of their patients and families, and take care to ensure that information is
- available in culturally and linguistically competent formats to help <u>patients and</u> parents in the decision
- 173 making process.
- 174
- 175 Statutes and case law of individual states govern informed consent. Some states allow oral-

176 discussions, which should be documented in the medical record, while others may require written

- 177 consent. Oral health practitioners should review applicable state laws to determine their level of
- 178 compliance. Consent forms should be procedure specific, utilize simple terms, and avoid overly broad
- 179 statements. When a practitioner utilizes an informed consent form, the following should be included:
- 180 1. Legal name and date of birth of pediatric patient.
- 181 2. Legal name and relationship to the pediatric patient/legal basis on which the person is
 182 consenting granting permission on behalf of the patient.
- 183 3. Patient's diagnosis.
- 184 4. Nature and purpose of the proposed treatment in simple terms.
- 185 5. Potential benefits and risks associated with that treatment.
- 186 6. Professionally-recognized or evidence-based alternative treatment including no treatment –
 187 to recommended therapy and risk(s).
- 188 7. Place for parent to indicate that all questions have been asked and adequately answered.

- 189 8. Places for signatures of the parent or legal guardian, dentist, and an office staff member as a 190 witness.
- 191
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- 1 [Best Practices]
- 2 Acquired Temporomandibular Disorders in Infants, Children, and
- 3 Adolescents*
- 4
- 5 Review Council
- 6 Council on Clinical Affairs
- 7 Latest Revision
- 8 2015 <u>2019</u>
- 9
- 10 Key words: pediatric, adolescent, temporomandibular joint, temporomandibular disorders,
- 11 temporomandibular joint dysfunction syndrome
- 12
- 13

14 Methods

- 15 This guideline document was originally developed by the Clinical Affairs Committee –
- 16 Temporomandibular Joint Problems in Children Subcommittee and adopted in 1990. This document is a
- 17 revision of the previous version, last revised in 2010. The update included an electronic search using the
- 18 terms: temporomandibular disorder, TMJ dysfunction, TMD AND adolescents, TMD AND gender
- 19 differences, TMD AND occlusion, TMD AND treatment; fields: all fields; limits: within the last 15 years,
- 20 humans, English, clinical trials. The reviewers agreed upon the inclusion of 78-102 references to support
- 21 this documentguideline. When data did not appear sufficient or were inconclusive, recommendations were
- 22 based upon expert and/or consensus opinion by experienced researchers and clinicians.
- 23

24 Background

25 Definition of TMD

- 26 TMD is a collective term for a group of musculoskeletal and neuromuscular conditions which includes
- 27 several clinical signs and symptoms involving the muscles of mastication, the TMJ, and associated
- 28 structures.¹ While TMD has been defined as "functional disturbances of the masticatory system", $\frac{2}{2}$ others
- 29 include masticatory muscle disorders,³ degenerative and inflammatory TMJ disorders,⁴ and TMJ disk

* ABBREVIATIONS

AAPD: American Academy Pediatric Dentistry. **CBCT**: Cone-beam computed tomography. **TMD**: Temporomandibular disorder. **TMJ**: Temporomandibular joint. **DC**: Diagnostic criteria

- 30 displacements⁵ under the umbrella of TMD.
- 31
- 32 In a recent update, the American Academy of Orofacial Pain divided TMD in two broad categories: TMJ-
- 33 disorders and masticatory muscle disorders.
- 34

35 Prevalence of TMD in children and adolescents

- 36 <u>TMDs have been identified as a major cause of nonodontogenic pain in the orofacial region⁶ (Bells Oral-</u>
- 37 <u>and Facial Pains 2014</u>). The reported prevalence of TMD in infants, children, and adolescents varies
- $38 widely in the literature.^{7-12} This variation may be due to differences in populations studied, diagnostic$
- 39 criteria, examination methods, and inter- and/or intra-rate<u>r-d</u> variations of examining practitioners. $\frac{13.14}{\text{The}}$
- 40 Diagnostic Criteria (DC) TMD examination protocol is used in research settings to decrease variability in
- 41 <u>diagnosis; however, few pediatric studies use this methodology.^{15,16} One study using the DC-TMD criteria</u>
- 42 <u>demonstrated an 11.9 percent prevalence of TMD in adolescents.^{16.} Most data suggests the Pprevalence</u>
- 43 of signs and symptoms <u>of TMD</u> increases with age.^{12,16,17} One study reported thethat TMD-related
- 44 symptoms were rare in three- and five-year-olds_whereas five to nine percent of 10- and 15-year-olds
- 45 reported more severe symptoms.¹⁷ Another <u>study</u> found that 4.2 percent of adolescents aged 12-19 years
- 46 reported TMD pain.¹⁴ A study of <u>children in</u> the primary dentition reported found that 34 percent of
- 47 patients <u>have with</u> signs and/or symptoms of TMD.¹⁸ This could, in part, be due to inclusion of muscular
- 48 signs <u>such as tenderness to palpation which can be difficult to assess in young children</u>.^{11,18} versus-
- 49 symptoms. An epidemiological study of 4,724 children aged five through 17 years reported 25 percent-
- 50 with symptoms. Clicking was seen in 2.7 percent of children in the primary dentition and 10.1 percent in-
- 51 late mixed dentition, and further increased to 16.6 percent in patients with permanent dentition.(Thilander-
- 52 et al 2002) <u>A systematic review and meta-analysis of intra-articular TMD in children and adolescents</u>
- 53 found a 16 percent prevalence of clinical signs and a 14 percent prevalence of TMJ sounds.¹¹ Headaches-
- 54 appear to be independently and highly associated with TMD in adolescents, with headaches most
- 55 commonly occurring before the onset of TMD jaw pain (odds ratio 9.4).(Nilsson et al 2013) Although
- 56 TMD pain in children increases with age in both boys and girls, recent surveys have indicated a
- 57 significantly higher prevalence of symptoms and greater need for treatment in girls than boys.^{12,19} with t
- 58 <u>The development of symptomatic TMD has been correlated with the onset of puberty in girls.²⁰ For ages</u>
- 59 16-19 years, 32.5 percent of girls compared to 9.7 percent of boys reported school absences and analgesic
- 60 consumption due to TMD<u>-related</u> pain.¹⁹ <u>Headaches appear to be independently and highly associated</u>
- 61 with TMD in adolescents, with most occurring before the onset of jaw pain.²¹
- 62

63 Etiology of TMD

- 64 Temporomandibular disorders have multiple etiological factors.²² <u>There is Research is insufficient</u>
- 65 <u>evidence</u> to reliably predict which patients will or will not develop TMD.²³ as most published
- 66 investigations evaluate static, morphologic variables rather than study the dynamic relationship between-
- 67 the joint and the teeth during function.(Howard 2013) There are pPredisposing (or risk) factors,
- 68 precipitating (or initiating) factors, and perpetuating (or sustaining) factors that contribute to the
- 69 development of TMDs.²³ Many studies show a The available evidence base suggests a poor correlation
- 70 between any single etiological factor and resulting signs (i.e., findings identified by the dentist during the
- examination) and symptoms (i.e., findings reported by the child or parent).²⁴ Alterations in any one or a
- 72 combination of teeth, periodontal ligament, the TMJ, or the muscles of mastication may lead to $TMD.^{24}$
- 73 Furthermore, systemic and psychosocial factors may reduce the adaptive capacity of the masticatory
- 74 system and contribute to $TMD.^{1}$
- 75

76 Etiologic factors suggested as contributing to the development of TMD are:

- 77 1. Macrotrauma: This would include impact injuries such as trauma to the chin. A common 78 occurrence in childhood because of falling, chin trauma is reported to be a factor in the development of TMD in pediatric patients.²⁵⁻²⁸ Additional macrotraumatic injuries occur due to 79 80 motor vehicle accidents, sports, physical abuse, forceful intubation, and third molar extraction.^{25,29} 81 Unilateral and bilateral intracapsular or subcondylar fractures are the most common mandibular 82 fractures in children.³⁰ Closed reduction and prolonged immobilization can result in ankylosis.^{31,32} 83 Improperly treated fractures may result in facial asymmetry.^{31,32} Traumatic brain injury (TBI) may accompany mandibular fracture and other types of jaw injuries.²⁵ Indirect trauma such as flexion-84 extension (whiplash) injuries may alter pain processing and lead to TMD symptoms; however, a 85 direct relationship between TMD and indirect trauma has yet to be established.¹ 86
- 87

2. Microtrauma from parafunctional habits: Bruxism, clenching, hyperextension, and other repetitive 88 habitual behaviors are thought to contribute to the development of TMD by joint overloading that 89 90 leads to cartilage breakdown, synovial fluid alterations, and other changes within the joint.³³ 91 Bruxism may occur while the patient is asleep or awake; sleep bruxism is a different entity from daytime bruxism. Sleep bruxism has been classified as a sleep-related movement disorder.³⁴ A 92 study of 854 patients younger than 17 years old found the prevalence of bruxism to be 38 percent, $\frac{35}{2}$ 93 94 but studies generally do not distinguish between sleep or daytime bruxism. The literature on the association between parafunction and TMD in pediatric patients is contradictory. $\frac{36,37,38}{100}$ However, 95

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96		childhood parafunction was found to be a predictor of the same parafunction 20 years later. ³⁹ Other
97		studies found correlations a significant association between reported bruxism and TMD. ^{40.41} with a
98		3.4 odds ratio Children who grind their teeth were found to complain more often of pain and muscle
99		tenderness when eating. ⁴² Other examples of microtrauma include repetitive strain such as playing a
100		wind instrument, fingernail biting, ²³ or another activity in which the mouth is held open outside of
101		rest position.
102	3.	Anatomical factors (skeletal and occlusal) and orthodontic treatment: There is a relatively low weak
103		association of skeletal and occlusal factors and the development of TMD. ^{1.43,44,45} It is reasonable-
104		that some occlusal factors may place greater adaptive demands on the masticatory system. Current-
105		literature Furthermore, the available data does not support that the development of TMD is caused
106		or improved by orthodontic treatment, 46,47,48,49,50 regardless of whether premolars were extracted-
107		prior to treatment. ²⁹ Changes in freeway dimension of the rest position (normally two to four
108		millimeters) may be impinged by occlusal changes, disease, muscle spasms, nervous tension, and/or
109		restorative prosthetics. ² While most children and adolescents may be able to compensate without
110		problem, in others, failure of the masticatory system to adapt may lead to greater risk of
111		dysfunction. In a study of 4,724 children aged five 17 years grouped by stage of dental
112		development, the following malocclusions were found to be associated with TMD: (Thilander et al-
113		2002) Although there is little evidence to implicate skeletal or occlusal factors with TMD the
114		following have some association across studies:
115		• Skeletal anterior open bite. ^{51,52}
116		• Steep articular eminence of the temporal bone $\frac{1}{2}$
117		• Overjet greater than six to seven millimeters. 51,52,53,54
118		• <u>Skeletal class II profile⁵⁵</u>
119		• <u>Hyperdivergent growth pattern⁵⁵</u>
120		• Class III malocclusion. <u>51, 52</u>
121		• <u>Unilateral</u> Pposterior crossbite ^{43,52.}
122		• Loss of posterior support ⁴³
123		
124		Craniocervical posture has been suggested to be associated with occlusion and with dysfunction of
125		the TMJ, including abnormalities of the mandibular fossa, condyle, ramus, and disc. ⁵⁶ Cervical pain
126		and dysfunction can be a result of poor posture. ⁵⁷ Cervical pain is frequently referred to orofacial
127		structures and can be misinterpreted as TMD. ⁶⁾
128	4.	Psychosocial factors: Psychosocial factors may play a part in the etiology of TMD. ^{58,59} Behavioral

129 factors such as somatization, anxiety, obsessive-compulsive feelings, and psychologic stress were predictors of TMD onset.⁵⁸- Emotional stress predisposes to clenching and bruxism which in turn 130 contribute to orofacial pain.⁶⁰ Results from a case-control study indicate that management of stress 131 132 and anxiety can mitigate the signs and symptoms of TMD.⁵⁹ Depression, anxiety, post-traumatic stress disorder, psychologic distress, and sleep dysfunction may influence TMD prognosis and 133 symptoms.⁶¹ Behavioral factors such as somatization and depression influence TMD pain to a larger 134 degree in girls than in boys. Higher pain intensity in the orofacial region correlated with greater 135 impact on quality of life including difficulty with prolonged jaw opening, eating hard/soft foods. 136 and sleeping. $\frac{61}{2}$ 137 5. Systemic and pathologic factors: Systemic factors contributing to TMD include connective tissue 138 139 diseases such as rheumatoid arthritis, systemic lupus erythematosus, juvenile idiopathic arthritis, and psoriatic arthritis.^{62,63,23} These systemic diseases occur as a result of imbalance of pro-140 inflammatory cytokines which causes oxidative stress, free radical formation, and ultimately joint 141 142 damage.⁶⁴ Other systemic factors may include joint hypermobility, genetic susceptibility, and hormonal fluctuations. Generalized joint laxity or hypermobility (e.g., Ehler Danlos syndrome) has 143 been cited but has a weak association with TMD.^{65,66}Pathologic destructive and overgrowth 144 processes such as post radiation therapy deformity, post traumatic tumor resection defect, condylar 145 146 hyperplasia, and condylar tumors represent a unique category of TMDs.⁶³ 147 6. Genetic and hormonal factors: There is little research in regard to the genetic susceptibility for development of TMD. Recently, study of catechol-O-methyl-transferase (COMT) haplotypes found 148 149 that the presence of one low pain sensitivity haplotype decreased the risk of developing TMD.⁵⁸ 150 The role of hormones in the etiology of TMD is debatable. Randomized controlled trials indicate 151 that estrogen does not play a role in the etiology of TMD, whereas cohort and case-controlled 152 studies show the opposite.¹ Although the biological basis for gender-based disparity in TMD is unclear, the time course of symptoms is of note in females. Additional studies have shown that TMJ 153 pain and other symptoms vary in relation to phases of the menstrual cycle.⁶⁷ The suggestion of a 154 hormonal influence in development of TMD is supported clinically by a study of 3,428 patients 155 who sought treatment for TMD. This study revealed that 85.4 percent of patients seeking treatment 156 were female and the peak age for treatment seeking was 33.8 years.⁶⁷ In a similar study of 157 adolescents,⁶⁸ 15.1 percent of all patients evaluated for TMD were less than 20 years of age and 158 159 girls accounted for 89.9 percent of patients aged 15-19 seeking care and 75.5 percent of patient six-160 14 years of age.

162 Diagnosing TMD

163	All comprehensive dental examinations should include a screening evaluation of the TMJ and
164	surrounding area. ^{69,70} Diagnosis of TMD is based upon a combination of historical information, clinical
165	examination, and/or craniocervical and TMJ imaging.1 The findings are classified as symptoms and
166	signs. ⁶⁹ These symptoms may include pain, headache, TMJ sounds, TMJ locking, and ear pain. ⁷¹ Certain
167	medical conditions are reported to occasionally mimic TMD. Among these differential diagnoses are
168	trigeminal neuralgia, central nervous system lesions, odontogenic pain, sinus pain, otological pain,
169	developmental abnormalities, neoplasias, parotid diseases, vascular diseases, myofascial pain, cervical
170	muscle dysfunction, and Eagle's syndrome. ⁶ Other common medical conditions (e.g., otitis media,
171	allergies, airway congestion, rheumatoid arthritis) can cause symptoms similar to TMD. ⁷¹
172	
173	Clinical and physical assessment of the patient may include history and determination of joint sounds,
174	evaluation of mandibular range of motion, appraisal of pain, evaluation for signs of inflammation, and
175	select radiographic examination. ⁷¹
176	
177	A screening history, as part of the health history, may include questions such as: ^{1,23}
178	• Do you have difficulty opening your mouth?
179	• Do you hear noises within your jaw joint?
180	• Do you have pain in or around your ears or your cheeks?
181	• Do you have pain when chewing, talking, or using your jaws?
182	• Do you have pain when opening your mouth wide or when yawning?
183	• Has your bite felt uncomfortable or unusual?
184	• Does your jaw ever lock or go out?
185	• Have you ever had an injury to your jaw, head, or neck? If so, when? How was it treated?
186	• Have you previously been treated for a temporomandibular disorder? If so, when? How was it
187	treated?
188	
189	Physical assessment should include the following: ^{1,23,71}
190	1. Palpation of the muscles of mastication and cervical muscles for tenderness, pain, or pain referral
191	patterns. (American Academy of Orofacial Pain Orofacial Pain: Guidelines)
192	2. Palpation of the lateral capsule of the TMJs.(Howard 2014)
193	3. Mandibular function and provocation tests. (American Academy of Orofacial Pain Orofacial Pain:-
194	Guidelines, Howard 2014)

195	4.	Palpation and auscultation for TMJ sounds.(Howard 2014)
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- 197
- 198 Evaluation of jaw movements including assessment of mandibular range of motion using a millimeter
- 199 ruler (i.e., maximum unassisted opening, maximum assisted opening, maximum lateral excursion,
- 200 maximum protrusive excursion) and mandibular opening pattern (i.e., symmetrical vs. asymmetrical) may
- 201 <u>be helpful in the diagnosis of TMD</u>. <u>In addition, b</u>Both limited and excessive mandibular range of motion
- 202 may be seen in TMD. 1,23
- 203
- 204 TMJ imaging is recommended when there is a recent history of trauma or developing facial asymmetry,
- or when hard-tissue grinding or crepitus is detected.⁷² Imaging should also be considered in patients that
 have failed to respond to conservative TMD treatment.³³ <u>TMJ imaging Radiographic</u> assessment may
- 207 include:
- Panoramic <u>radiographor full mouth periapical films;</u>
- Lateral cephalogram;
 - Mandible radiographs including oblique views
- <u>Conventional computed tomography (CT) or cone-beamed CT</u>
- TMJ tomography;
- Magnetic resonance imaging (both open and closed mouth to view disc position); or
- <u>Ultrasound</u>
- Cone beam computed tomography (CBCT).
- 216

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- 217 TMJ arthography is not recommended as a routine diagnostic procedure.^{73,74,75} The readily available
- 218 panoramic radiograph is reliable for evaluating condylar head morphology and angulation but does not
- 219 permit evaluation of the joint space, soft tissues, or condylar motion.²³ The panograph panoramic
- 220 <u>radiograph</u> may indicate osseous changes, but negative findings do not rule out TMJ pathology.⁷⁶ The
- 221 CBCT can be used to detect boney abnormalities and fractures and to assess asymmetry,^{74,75,76} but
- 222 generates a much higher radiation burden than the panoramic image. Magnetic resonance imaging
- provides visualization of soft tissues, specifically the position and contours of the TMJ disc, and can be
- used to detect inflammation.^{72, 23,75} Ultrasound is a noninvasive imaging method for viewing superficial
- **225** lateral aspects of the TMJ.⁷⁷
- 226
- 227 TMD has been divided into two broad categories, TMJ disorders and masticatory muscles disorders, 1

^{196 5.} Mandibular range of motion.(Howard 2014)

228	whicl	h are	listed below.
229	I.	TM	J disorders:
230		a.	Joint pain:
231			1. Aarthralgia (synovitis, capsulitis, and retro-discitis)
232			2. Arthritis
233		b.	Joint disorders:
234			1. Disc-condyle complex disorders (disc displacement with reduction, disc displacement
235			with reduction with intermittent locking, disc displacement without reduction, with
236			limited opening, disc displacement without reduction without limited opening).
237			2. Hypomobility disorders (intra-articular ankylosis, bony ankylosis, fibrous adhesions,
238			ankylosis)-
239			3. Hypermobility disorders (subluxation, luxation)-
240		c.	Joint diseases:
241			1. Osteoarthritis also known as [degenerative joint disease(DJD), condylysis/idiopathic_
242			condylar resorption, osteochondritis dissecans, osteonecrosis].
243			2. Systemic arthritides such as rheumatoid arthritis, <u>JIA</u> idiopathic juvenile arthritis,
244			spondyloarthropathies, psoriatic arthritis, infections arthritis, Reiter syndrome, and crystal
245			induced disease-
246			3. Neoplasms .
247			4. Fractures (open and closed condylar and subcondylar)-
248	II.	Mas	sticatory muscle disorders:
249		a.	Muscle pain limited to orofacial region (local myalgia, myofascial pain with spreading,
250			myofascial pain with referral, tendonitis, myositis, spasm)-
251		b.	Muscle pain due to systemic/central disorders (centrally mediated myalgia, fibromyalgia).
252		c.	Movement disorders (dyskinesia, dystonia).
253		d.	Other muscle disorders (contracture, hypertrophy, neoplasm)-
254			
255	Trea	tmen	t of TMD
256	The g	goals	of TMD treatment include restorational of function, decreased pain, decreased aggravating or
257	<u>contr</u>	ibutir	ng factors, and improved return of quality of life. ^{78,79} Few studies document success or failure of
258	speci	fic tre	eatment modalities for TMD in infants, children, and adolescents on a long-term basis. It has
259	been	sugge	ested that simple, conservative, and reversible types of therapy are effective in reducing most
260	TMD	sym	ptoms in children. ^{79,80} The focus of treatment should be to find a balance between active and

- 261 passive treatment modalities. Active modalities include participation of the patient whereas passive
- 262 modalities may include wearing a stabilization splint. In a randomized trial, adolescents undergoing
- 263 occlusal appliance therapy combined with information attained a clinically significant improvement on
- the pain index.⁸¹ Combined approaches may be more successful in treating TMD than single treatment
- 265 modalities.⁷⁹
- 266

283

293

267	Treatment of TMD can be divided into reversible and irreversible treatment. Reversible therapies may
268	include:

- 269 • Patient education (e.g., explanation in clear and simple terms describing the nature of the disorder, the significance of predisposing, precipitating, and perpetuating factors, anatomy of 270 the TMJ, management options, and goals of therapy).^{1,79} 271 relaxation training, developing behavior coping strategies, modifying inadequate perceptions-272 273 about TMD, patient awareness of clenching and bruxing habits, if present).(Michelotti et al-274 2004) 275 • Physical therapy [e.g., jaw exercises or transcutaneous electrical nerve stimulation (TENS), 276 ultrasound, iontophoresis, massage, TMJ distraction and mobilization, thermotherapy, coolant therapy)].79,1,82,33,83,84 277 • Behavioral therapy (e.g., biofeedback, relaxation training, cognitive behavioral therapy (CBT) 278 279 for developing behavior coping strategies and modifying perceptions about TMD, habit 280 reversal and awareness of daytime clenching and bruxing, avoiding excessive chewing of hard foods or gum, voluntary avoidance of stressors, habit reversal; decreasing stress, treatment of 281 282 co-morbid behavioral health conditionsanxiety, and/or depression obtaining adequate,
- Prescription medication (e.g., non-steroidal anti- inflammatory drugs, anxiolytic agents, muscle relaxers). While antidepressants have proved to be beneficial, they should be prescribed by a practitioner familiar with pain management.^{79,1,33,85,}
- Occlusal splints. The goal of an occlusal appliance is to provide orthopedic stability to the TMJ. These alter the patient's occlusion temporarily and may be used to decrease parafunctional activity and pain.^{86,81,87,88} Occlusal splints may be made of hard or soft acrylic.
 The stabilization type of splint covers all of the teeth on either the maxillary or mandibular arch and is balanced so that all teeth are in occlusion when the patient is closed and the jaw is in a musculoskeletally stable position.^{6,33}
 - Additional reversible therapies may include TMJ arthrocentesis, TMJ injections, nerve blocks,

uninterrupted sleep).79,33,84

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294	acupuncture, trigger point injections, and off-label use of Botulinum toxin A injections. ⁸⁹⁻⁹³
295	
296	Irreversible therapies can include:
297	• Occlusal adjustment (i.e., permanently altering the occlusion or mandibular position by
298	selective grinding or full mouth restorative dentistry).94 A systematic review and meta-
299	analysis demonstrated that occlusal alteration seems to have no effect on TMD 95
300	• Orthodontics. This may include mandibular positioning devices designed to alter the growth or
301	permanently reposition the mandible (e.g., headgear, functional appliances). There is little
302	evidence that orthodontic treatment can prevent or relieve TMD. ^{1,96,97}
303	• Surgery. Surgical interventional includes orthognathic surgery, open joint TMJ surgery to
304	removed diseased synovium, and TMJ reconstruction. ⁶³ Data suggests surgery is limited in
305	most situations to cases of severe joint degeneration or destruction following trauma or tumor
306	resection 98,63,79
307	Botulinum toxin A injections. Although recently approved for use in adults to provide-
308	masticatory muscle relaxation, this modality has not been approved for use in children.(Dym-
309	and Israel 2012)
310	
311	Controversy surrounds the significance of signs and symptoms in this age group children and adolescents,
312	the value of certain diagnostic procedures, and what constitutes appropriate therapy. ^{100,55,99} It is not clear
313	whether these signs and symptoms constitute normal variation, preclinical features, or manifestations of a
314	disease state. ¹⁰¹ Whether these signs and symptoms warrant treatment as predictors of TMD in adulthood
315	is questionable. ^{101,39)}
316	
317	Referral should be made to other health care providers, including those with expertise in TMD, oral-
318	surgery, or pain management, when the diagnostic and/or treatment needs are beyond the treating-
319	dentist's scope of practice.
320	
321	Recommendations
322	Every comprehensive dental history and examination should include a TMJ history and assessment. ⁷⁰ The
323	history should include questions concerning the presence of head and neck pain and mandibular
324	dysfunction, previous orofacial trauma, and history of present illness with an account of current
325	symptoms. ¹⁰⁰ In the presence of a positive history and/or signs and symptoms of TMD, a more
326	comprehensive examination (e.g., palpation of masticatory and associated muscles and the TMJs,

327	docu	imentation of joint sounds, occlusal analysis, and assessment of range of mandibular movements			
328	inclu	uding maximum opening, protrusion, and lateral excursions) should be performed. ¹⁰⁰ A referral may-			
329	be considered. Joint imaging may be recommended in some cases(Hammer 2018) by other specialists to				
330	investigate joint sounds in the absence of other TMD signs and symptoms. Referral should be made to				
331	othe	r health care providers, including those with expertise in TMD, oral surgery, or pain management,			
332	whe	n the diagnostic and/or treatment needs are beyond the treating dentist's scope of practice. ¹			
333					
334					
335	The	rapeutic modalities to prevent TMD in the pediatric population are yet to be supported by controlled			
336	stud	ies. Reversible therapies should be considered fFor children and adolescents with signs and			
337	sym	ptoms of TMD, reversible therapies should be considered. 81,102 Because of inadequate data regarding			
338	their	effectiveness usefulness, irreversible therapies should be avoided. ^{81,94,97} Referral to a medical			
339	spec	ialist may be indicated when primary headaches, otitis media, allergies, abnormal posture, airway			
340	cong	gestion, rheumatoid arthritis, connective tissue disease, psychiatric disorders, or other medical			
341	cond	litions are suspected.			
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- 4
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- 9
- 10 The American Academy of Pediatric Dentistry (AAPD), in its efforts to promote optimal health for
- 11 children with cleft lip/ palate and other craniofacial anomalies, endorses the current statements of the

12 American Cleft Palate-Craniofacial Association (ACPA).¹

13

14 A child born with cleft lip/palate or other craniofacial anomalies has multiple and complex problems,

- 15 including early feeding and nutritional concerns, middle ear disease, hearing deficiencies, deviations
- 16 in speech and resonance, dentofacial and orthodontic abnormalities, and psychosocial adjustment
- 17 problems.
- 18
- Reports by the U.S. Surgeon General^{2,3} on children with special needs issued in 1987 and 2005 19 20 stressed that the care of these children should be comprehensive, coordinated, culturally sensitive, 21 specific to the needs of the individual, and readily accessible. Recognizing that children with clefts 22 and other craniofacial anomalies have special needs, the Maternal and Child Health Bureau in 1991 23 provided funding to ACPA to develop parameters of care for these patients through a series of consensus conferences among a multidisciplinary group of specialists.¹ In addition, the ACPA joined 24 with the Cleft Palate Foundation to create standards for approval of teams to ensure that care is 25 26 provided in a coordinated and consistent manner, including an appropriate sequence of evaluations and treatment for the patient's overall developmental, medical, and psychological needs.⁴ 27
- 28

ABBREVIATIONS

AAPD: American Academy Pediatric Dentistry. ACPA: American Cleft Palate-Craniofacial Association.

- As part of the parameters¹ and standards,⁴ several fundamental principles were identified as critical to
 optimal cleft/ craniofacial care. These principles are:
- Management of patients with craniofacial anomalies is best provided by an interdisciplinary
 team of specialists.¹ These teams are composed of qualified health professionals from medical,
- 33 surgical, dental, and allied health fields working together in a coordinated system. A
- designated patient care coordinator should be included in the team to assist in coordinated care
 for patients and their families/caregivers.⁴
- Optimal care for patients with craniofacial anomalies is provided by teams that see sufficient
 numbers of these patients each year to maintain clinical expertise in diagnosis and treatment.
- 38 3. The optimal time for the first evaluation is within the first few weeks of life and, whenever
 39 possible, within the first few days. However, referral for team evaluation and management is
 40 appropriate for patients of any age.¹
- 4. From the time of first contact with the child and family, every effort must be made to assist the
 family in adjusting to the birth of a child with a craniofacial anomaly and the consequent
 demands and stress placed upon that family.¹
- Parents/caregivers must be given information about recommended treatment procedures,
 options, risk factors, benefits, and costs to assist them in: (1) making informed decisions on
- 46 the child's behalf, and (2) preparing the child and themselves for all recommended procedures.
- 47 The team should actively solicit family participation and collaboration in treatment planning.^{1,4}
- 48 When the child is mature enough to do so, he or she should also participate in treatment
- 49 decisions.¹
- 50 6. Treatment plans should be developed and implemented on the basis of team
 51 recommendations.¹
- 52 7. Care should be coordinated by the team, but should be provided at the local level whenever
 53 possible; however, complex diagnostic or surgical procedures should be restricted to major
 54 centers with appropriate treatment facilities and experienced care providers.
- 8. It is the responsibility of each team to be sensitive to linguistic, cultural, ethnic, psychosocial,
 economic, and physical factors that affect the dynamic relationship between the team, the
 patient, and his/her family.¹
- 58 9. It is the responsibility of the team to monitor both short-term and long-term outcomes. Thus,
 59 longitudinal follow up of patients, including appropriate documentation and record-keeping, is
 60 essential.¹
- 61 10. Evaluation of treatment outcomes must take into account the satisfaction and psychosocial

62

well-being of the patient, as well as effects on growth, function, and appearance.¹

63

64 Patients with craniofacial anomalies require dental care throughout life as a direct result of their 65 condition and as an integral part of the treatment process. A dental home should be established within 66 six months of eruption of the first tooth and no later than 12 months of age. It includes oral health 67 examinations, caries control, and preventive, restorative, and prosthetic dental treatment as needed. 68 Patients should be closely monitored for periodontal disease and anomalies in dentition and eruption. 69 The condition of the developing dentition and supporting tissues, with counseling regarding early oral 70 hygiene and prevention of early childhood caries is essential. Prosthetic appliances such as an 71 obturator may help to close a fistula or aid in speech. Orthodontic treatment is also an integral part of 72 the rehabilitative process and often takes place in phases. The skeletal and dental components should 73 be regularly evaluated. When indicated, orthodontic treatment prepares a child for alveolar bone grafting of the cleft maxilla, correcting malocclusions, and preparation for jaw surgery.¹ As members 74 of the interdisciplinary team of physicians, dentists, speech-language pathologists, and other allied 75 76 health professionals, pediatric dentists should provide dental services in close cooperation with their orthodontic, oral and maxillofacial surgery, and prosthodontic colleagues.^{1,4} All dental specialists 77 78 should ensure that¹: 79 1. Consult with an appropriate dental specialist for cleft lip taping and or pre-surgical orthopedics 80 including but not limited to nasal alveolar molding (NAM). A craniofacial orthodontist (or appropriately trained clinician) who can discuss with the family the types of infant orthopedic 81 82 services available and the rationale for using infant orthopedics prop initial cleft lip repair is 83 necessary 84 +2. Dental radiographs, cephalometric radiographs, and other imaging modalities as indicated 85 should be utilized to evaluate and monitor dental and facial growth and development. 23. Diagnostic records, including properly occluded dental study models, should be collected at 86 87 Diagnostic records, including properly occluded dental study models, should be collected at 88 appropriate intervals for patients at risk for developing malocclusion or maxillary-mandibular 89 discrepancies. 34. As the primary dentition erupts, the team evaluation should include a dental examination and, if 90 91 such services are not already being provided, referral to appropriate providers for caries control, 92 preventive measures, restorative care, and space management. 93 45. Before the primary dentition has completed eruption, the skeletal and dental components should 94 be evaluated to determine if a malocclusion is present or developing.

95	<u>56</u> .	Depending upon the specific goals to be accomplished and also upon the age at which the
96		patient is initially evaluated, orthodontic management of the malocclusion may be performed in
97		the primary, mixed, or permanent dentition. In some cases, orthodontic treatment may be
98		necessary in all three stages.
99	<u>67</u> .	While continuous active orthodontic treatment from early mixed dentition to permanent
100		dentition should be avoided, each stage of orthodontic therapy may be followed by retention
101		and regular observation. Orthodontic retention for the permanent dentition may extend into
102		adulthood.
103	7 <u>8</u> .	For some patients with craniofacial anomalies, functional orthodontic appliances may be
104		indicated.
105	<u>89</u> .	For patients with craniofacial anomalies, orthodontic treatment may be needed in conjunction
106		with surgical correction (and/or distraction osteogenesis) of the facial deformity.
107	<u>910</u>	. Congenitally missing teeth may be replaced with a removable appliance, fixed restorative
108		bridgework, or osseointegrated implants.
109	1011. Patients should be closely monitored for dental and periodontal disease.	
110	44 <u>12</u> . Prosthetic obturation of palatal fistulae may be necessary in some patients.	
111	<u>12</u> 1	<u>3</u> . A prosthetic speech device may be used to treat velopharyngeal inadequacy in some patients.
112		
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Monitoring and Management of Pediatric Patients Before, During, and After Sedation for Diagnostic and Therapeutic Procedures: Update 2016

Developed and Endorsed by

American Academy of Pediatric Dentistry and American Academy of Pediatrics

Latest Revision*

20162019¹

Abbreviations

AAP: American Academy of Pediatrics
AAPD: American Academy of Pediatric Dentistry
ASA: American Society of Anesthesiologists
BIS: Bispectral index
CPAP: Continuous positive airway pressure
ECG: Electrocardiography
EEG: Electroencephalogram/electroencephalography.
EMS: Emergency medical services
LMA: Laryngeal mask airway
MRI: Magnetic resonance imaging
OSA: Obstructive sleep apnea
PALS: Pediatric advanced life support

Abstract

The safe sedation of children for procedures requires a systematic approach that includes the following: no administration of sedating medication without the safety net of medical/dental supervision, careful presedation evaluation for underlying medical or surgical conditions that would place the child at increased risk from sedating medications, appropriate fasting for elective procedures and a balance between the depth of sedation and risk for those who are unable to fast because of the urgent nature of the procedure, a focused airway examination for large (kissing) tonsils or anatomic airway abnormalities that might increase the potential for airway obstruction, a clear understanding of the medication's pharmacokinetic and pharmacodynamic effects and drug interactions, appropriate training and skills in airway management to allow rescue of the patient, age- and size-appropriate equipment for airway management and venous access, appropriate medications and reversal agents, sufficient numbers of staff to both carry out the procedure and monitor the patient, appropriate

¹ 2019 revision limited to Personnel section. Approved by AAP Board December 2018

physiologic monitoring during and after the procedure, a properly equipped and staffed recovery area, recovery to the presedation level of consciousness before discharge from medical/dental supervision, and appropriate discharge instructions. This report was developed through a collaborative effort of the American Academy of Pediatrics and the American Academy of Pediatric Dentistry to offer pediatric providers updated information and guidance in delivering safe sedation to children.

Introduction

The number of diagnostic and minor surgical procedures performed on pediatric patients outside of the traditional operating room setting has increased in the past several decades. As a consequence of this change and the increased awareness of the importance of providing analgesia and anxiolysis, the need for sedation for procedures in physicians' offices, dental offices, subspecialty procedure suites, imaging facilities, emergency departments, other inpatient hospital settings, and ambulatory surgery centers also has increased markedly.¹⁻⁵² In recognition of this need for both elective and emergency use of sedation in nontraditional settings, the American Academy of Pediatrics (AAP) and the American Academy of Pediatric Dentistry (AAPD) have published a series of guidelines for the monitoring and management of pediatric patients during and after sedation for a procedure. $^{53-58}$ The purpose of this updated report is to unify the guidelines for sedation used by medical and dental practitioners; to add clarifications regarding monitoring modalities, particularly regarding continuous expired carbon dioxide measurement; to provide updated information from the medical and dental literature; and to suggest methods for further improvement in safety and outcomes. This document uses the same language to define sedation categories and expected physiologic responses as The Joint Commission, the American Society of Anesthesiologists (ASA), and the AAPD.^{56,57,59-61}

This revised statement reflects the current understanding of appropriate monitoring needs of pediatric patients both during and after sedation for a procedure.^{3,4,11,18,20,21,23,24,33,39,41,44,47,51,62–73} The monitoring and care outlined may be exceeded at any time on the basis of the judgment of the responsible practitioner. Although intended to encourage high-quality patient care, adherence to the recommendations in this document cannot guarantee a specific patient outcome. However, structured sedation protocols designed to incorporate these safety principles have been widely implemented and shown to reduce morbidity.^{11,23,24,27,30–33,35,39,41,44,47,51,74–84} These practice recommendations are proffered with the awareness that, regardless of the intended level of sedation or route of drug administration, the sedation of a pediatric patient represents a continuum and may result in respiratory depression, laryngospasm, impaired airway patency, apnea, loss of the patient's protective airway reflexes, and cardiovascular instability.^{38,43,45,47,48,59,62,63,85–112}

Procedural sedation of pediatric patients has serious associated risks.^{2,5,38,43,45,47,48,62,63,71,83,85,88–105,107–138} These adverse responses during and after sedation for a diagnostic or therapeutic procedure may be minimized, but not completely eliminated, by a careful preprocedure review of the patient's underlying medical conditions and consideration of how the sedation process might affect or be affected by these conditions: for example, children with developmental disabilities

have been shown to have a threefold increased incidence of desaturation compared with children without developmental disabilities.^{74,78,103} Appropriate drug selection for the intended procedure, a clear understanding of the sedating medication's pharmacokinetics and pharmacodynamics and drug interactions, as well as the presence of an individual with the skills needed to rescue a patient from an adverse response are critical.^{42, 48,62,63,92,97,99,125–127,132,133,139–158} Appropriate physiologic monitoring and continuous observation by personnel not directly involved with the procedure allow for the accurate and rapid diagnosis of complications and initiation of appropriate rescue interventions.^{44,63,64,67,68,74,90,96,110,159–174} The work of the Pediatric Sedation Research Consortium has improved the sedation knowledge base, demonstrating the marked safety of sedation by highly motivated and skilled practitioners from a variety of specialties practicing the above modalities and skills that focus on a culture of sedation safety.^{45,83,95,128–} ¹³⁸ However, these groundbreaking studies also show a low but persistent rate of potential sedation-induced life-threatening events, such as apnea, airway obstruction, laryngospasm, pulmonary aspiration, desaturation, and others, even when the sedation is provided under the direction of a motivated team of specialists.¹²⁹ These studies have helped define the skills needed to rescue children experiencing adverse sedation events.

The sedation of children is different from the sedation of adults. Sedation in children is often administered to relieve pain and anxiety as well as to modify behavior (e.g., immobility) so as to allow the safe completion of a procedure. A child's ability to control his or her own behavior to cooperate for a procedure depends both on his or her chronologic age and cognitive/ emotional development. Many brief procedures, such as suture of a minor laceration, may be accomplished with distraction and guided imagery techniques, along with the use of topical/local anesthetics and minimal sedation, if needed.^{175–181} However, longer procedures that require immobility involving children younger than 6 years or those with developmental delay often require an increased depth of sedation to gain control of their behavior.^{86,87,103} Children younger than 6 years (particularly those younger than 6 months) may be at greatest risk of an adverse event.¹²⁹ Children in this age group are particularly vulnerable to the sedating medication's effects on respiratory drive, airway patency, and protective airway reflexes.^{62,63} Other modalities, such as careful preparation, parental presence, hypnosis, distraction, topical local anesthetics, electronic devices with age-appropriate games or videos, guided imagery, and the techniques advised by child life specialists, may reduce the need for or the needed depth of pharmacologic sedation.^{29,46,49,182-211}

Studies have shown that it is common for children to pass from the intended level of sedation to a deeper, unintended level of sedation,^{85,88,212,213} making the concept of rescue essential to safe sedation. Practitioners of sedation must have the skills to rescue the patient from a deeper level than that intended for the procedure. For example, if the intended level of sedation is "minimal," practitioners must be able to rescue from "moderate sedation"; if the intended level of sedation is "moderate," practitioners must have the skills to rescue from "deep sedation"; if the intended level of sedation is "moderate," practitioners must have the skills to rescue from "deep sedation"; if the intended level of sedation is "deep," practitioners must have the skills to rescue from a state of "general anesthesia." The ability to rescue means that practitioners must be able to recognize the various levels of sedation and have the skills and age- and size-appropriate equipment necessary to provide appropriate cardiopulmonary support if needed.

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These guidelines are intended for all venues in which sedation for a procedure might be performed (hospital, surgical center, freestanding imaging facility, dental facility, or private office). Sedation and anesthesia in a nonhospital environment (e.g., private physician's or dental office, freestanding imaging facility) historically have been associated with an increased incidence of "failure to rescue" from adverse events, because these settings may lack immediately available backup. Immediate activation of emergency medical services (EMS) may be required in such settings, but the practitioner is responsible for life-support measures while awaiting EMS arrival.^{63,214} Rescue techniques require specific training and skills.^{63,74,215,216} The maintenance of the skills needed to rescue a child with appeal larvngospasm, and/or airway obstruction include the ability to open the airway, suction secretions, provide continuous positive airway pressure (CPAP), perform successful bag-valve-mask ventilation, insert an oral airway, a nasopharyngeal airway, or a laryngeal mask airway (LMA), and, rarely, perform tracheal intubation. These skills are likely best maintained with frequent simulation and team training for the management of rare events.^{128,130,217–220} Competency with emergency airway management procedure algorithms is fundamental for safe sedation practice and successful patient rescue (see Figs. 1, 2, and 3).^{215,216,221–223}

Practitioners should have an in-depth knowledge of the agents they intend to use and their potential complications. A number of reviews and handbooks for sedating pediatric patients are available.^{30,39,65,75,171,172,201,224–233} There are specific situations that are beyond the scope of this document. Specifically, guidelines for the delivery of general anesthesia and monitored anesthesia care (sedation or analgesia), outside or within the operating room by anesthesiologists or other practitioners functioning within a department of anesthesiology, are addressed by policies developed by the ASA and by individual departments of anesthesiology.²³⁴ In addition, guidelines for the sedation of patients undergoing mechanical ventilation in a critical care environment or for providing analgesia for patients postoperatively, patients with chronic painful conditions, and patients in hospice care are beyond the scope of this document.

Goals of sedation

The goals of sedation in the pediatric patient for diagnostic and therapeutic procedures are as follows: (1) to guard the patient's safety and welfare; (2) to minimize physical discomfort and pain; (3) to control anxiety, minimize psychological trauma, and maximize the potential for amnesia; (4) to modify behavior and/or movement so as to allow the safe completion of the procedure; and (5) to return the patient to a state in which discharge from medical/dental supervision is safe, as determined by recognized criteria (Supplemental Appendix 1).

These goals can best be achieved by selecting the lowest dose of drug with the highest therapeutic index for the procedure. It is beyond the scope of this document to specify which drugs are appropriate for which procedures; however, the selection of the fewest number of drugs and matching drug selection to the type and goals of the procedure are essential for safe practice. For example, analgesic medications, such as opioids or ketamine, are indicated for painful procedures. For nonpainful procedures, such as computed tomography or MRI, sedatives/hypnotics are preferred. When both sedation and analgesia are desirable (e.g., fracture

reduction), either single agents with analgesic/sedative properties or combination regimens are commonly used. Anxiolysis and amnesia are additional goals that should be considered in the selection of agents for particular patients. However, the potential for an adverse outcome may be increased when 2 or more sedating medications are administered.^{62,127,136,173,235} Recently, there has been renewed interest in noninvasive routes of medication administration, including intranasal and inhaled routes (e.g., nitrous oxide; see below).²³⁶

Knowledge of each drug's time of onset, peak response, and duration of action is important (e.g., the peak EEG effect of intravenous midazolam occurs at ~ 4.8 minutes, compared with that of diazepam at ~ 1.6 minutes $^{237-239}$). Titration of drug to effect is an important concept; one must know whether the previous dose has taken full effect before administering additional drugs.²³⁷ Drugs that have a long duration of action (e.g., intramuscular pentobarbital, phenothiazines) have fallen out of favor because of unpredictable responses and prolonged recovery. The use of these drugs requires a longer period of observation even after the child achieves currently used recovery and discharge criteria.^{62,238–241} This concept is particularly important for infants and toddlers transported in car safety seats; re-sedation after discharge attributable to residual prolonged drug effects may lead to airway obstruction.^{62,63,242} In particular, promethazine (Phenergan; Wyeth Pharmaceuticals, Philadelphia, Pa.) has a "black box warning" regarding fatal respiratory depression in children younger than 2 years.²⁴³ Although the liquid formulation of chloral hydrate is no longer commercially available, some hospital pharmacies now are compounding their own formulations. Low-dose chloral hydrate (10–25 mg/kg), in combination with other sedating medications, is used commonly in pediatric dental practice.

General guidelines

Candidates

Patients who are in ASA classes I and II are frequently considered appropriate candidates for minimal, moderate, or deep sedation (Supplemental Appendix 2). Children in ASA classes III and IV, children with special needs, and those with anatomic airway abnormalities or moderate to severe tonsillar hypertrophy present issues that require additional and individual consideration, particularly for moderate and deep sedation.^{68,244–249} Practitioners are encouraged to consult with appropriate subspecialists and/ or an anesthesiologist for patients at increased risk of experiencing adverse sedation events because of their underlying medical/surgical conditions.

Responsible person

The pediatric patient shall be accompanied to and from the treatment facility by a parent, legal guardian, or other responsible person. It is preferable to have 2 adults accompany children who are still in car safety seats if transportation to and from a treatment facility is provided by 1 of the adults.²⁵⁰

Facilities

The practitioner who uses sedation must have immediately available facilities, personnel, and equipment to manage emergency and rescue situations. The most common serious complications

of sedation involve compromise of the airway or depressed respirations resulting in airway obstruction, hypoventilation, laryngospasm, hypoxemia, and apnea. Hypotension and cardiopulmonary arrest may occur, usually from the inadequate recognition and treatment of respiratory compromise.^{42,48,92,97,99,125,132,139–155} Other rare complications also may include seizures, vomiting, and allergic reactions. Facilities providing pediatric sedation should monitor for, and be prepared to treat, such complications.

Back-up emergency services

A protocol for immediate access to back-up emergency services shall be clearly outlined. For nonhospital facilities, a protocol for the immediate activation of the EMS system for life-threatening complications must be established and maintained.⁴⁴ It should be understood that the availability of EMS does not replace the practitioner's responsibility to provide initial rescue for life-threatening complications.

On-site monitoring, rescue drugs, and equipment

An emergency cart or kit must be immediately accessible. This cart or kit must contain the necessary age-and size appropriate equipment (oral and nasal airways, bag-valve-mask device, LMAs or other supraglottic devices, laryngoscope blades, tracheal tubes, face masks, blood pressure cuffs, intravenous catheters, etc.) to resuscitate a nonbreathing and unconscious child. The contents of the kit must allow for the provision of continuous life support while the patient is being transported to a medical/dental facility or to another area within the facility. All equipment and drugs must be checked and maintained on a scheduled basis (see Supplemental Appendices 3 and 4 for suggested drugs and emergency life support equipment to consider before the need for rescue occurs). Monitoring devices, such as electrocardiography (**ECG**) machines, pulse oximeters with size-appropriate probes, end-tidal carbon dioxide monitors, and defibrillators with size-appropriate patches/ paddles, must have a safety and function check on a regular basis as required by local or state regulation. The use of emergency checklists is recommended, and these should be immediately available at all sedation locations; they can be obtained from *http://www.pedsanesthesia.org/*.

Documentation

Documentation prior to sedation shall include, but not be limited to, the following recommendations:

- 1. Informed consent: The patient record shall document that appropriate informed consent was obtained according to local, state, and institutional requirements.^{251,252}
- 2. Instructions and information provided to the responsible person: The practitioner shall provide verbal and/or written instructions to the responsible person. Information shall include objectives of the sedation and anticipated changes in behavior during and after sedation.^{163,253–255} Special instructions shall be given to the adult responsible for infants and toddlers who will be transported home in a car safety seat regarding the need to carefully observe the child's head position to avoid airway obstruction. Transportation in a car safety seat poses a particular risk for infants who have received medications known to have a long half-life, such as chloral hydrate, intramuscular pentobarbital, or

phenothiazine because deaths after procedural sedation have been reported.^{62,63,238,242,256,257} Consideration for a longer period of observation shall be given if the responsible person's ability to observe the child is limited (e.g., only 1 adult who also has to drive). Another indication for prolonged observation would be a child with an anatomic airway problem, an underlying medical condition such as significant obstructive sleep apnea (**OSA**), or a former preterm infant younger than 60 weeks' postconceptional age. A 24-hour telephone number for the practitioner or his or her associates shall be provided to all patients and their families. Instructions shall include limitations of activities and appropriate dietary precautions.

Dietary precautions

Agents used for sedation have the potential to impair protective airway reflexes, particularly during deep sedation. Although a rare occurrence, pulmonary aspiration may occur if the child regurgitates and cannot protect his or her airway.^{95,127,258} Therefore, the practitioner should evaluate preceding food and fluid intake before administering sedation. It is likely that the risk of aspiration during procedural sedation differs from that during general anesthesia involving tracheal intubation or other airway manipulations.^{259,260} However, the absolute risk of aspiration during elective procedural sedation is not yet known; the reported incidence varies from ~1 in 825 to ~1 in 30037.^{95, 127,129,173,244,261} Therefore, standard practice for fasting before elective sedation generally follows the same guidelines as for elective general anesthesia; this requirement is particularly important for solids, because aspiration of clear gastric contents causes less pulmonary injury than aspiration of particulate gastric contents.^{262,263}

For emergency procedures in children undergoing general anesthesia, the reported incidence of pulmonary aspiration of gastric contents from 1 institution is ~ 1 in 373 compared with ~ 1 in 4544 for elective anesthetics.²⁶² Because there are few published studies with adequate statistical power to provide guidance to the practitioner regarding the safety or risk of pulmonary aspiration of gastric contents during procedural sedation, 95,127,129,173,244,259-261,264-268 it is unknown whether the risk of aspiration is reduced when airway manipulation is not performed/ anticipated (e.g., moderate sedation). However, if a deeply sedated child requires intervention for airway obstruction, apnea, or laryngospasm, there is concern that these rescue maneuvers could increase the risk of pulmonary aspiration of gastric contents. For children requiring urgent/emergent sedation who do not meet elective fasting guidelines, the risks of sedation and possible aspiration are as-yet unknown and must be balanced against the benefits of performing the procedure promptly. For example, a prudent practitioner would be unlikely to administer deep sedation to a child with a minor condition who just ate a large meal; conversely, it is not justifiable to withhold sedation/analgesia from the child in significant pain from a displaced fracture who had a small snack a few hours earlier. Several emergency department studies have reported a low to zero incidence of pulmonary aspiration despite variable fasting periods^{260,264,268}; however, each of these reports have, for the most part, clearly balanced the urgency of the procedure with the need for and depth of sedation.^{268,269} Although emergency medicine studies and practice guidelines generally support a less restrictive approach to fasting for brief urgent/ emergent procedures, such as care of wounds, joint dislocation, chest tube placement, etc., in healthy children, further research in many thousands of patients would be desirable to better define the relationships between various fasting intervals and sedation complications.^{262–270}

Before elective sedation

Children undergoing sedation for elective procedures generally should follow the same fasting guidelines as those for general anesthesia (Table 1).²⁷¹ It is permissible for routine necessary medications (e.g., antiseizure medications) to be taken with a sip of clear liquid or water on the day of the procedure.

For the emergency patient

The practitioner must always balance the possible risks of sedating nonfasted patients with the benefits of and necessity for completing the procedure. In particular, patients with a history of recent oral intake or with other known risk factors, such as trauma, decreased level of consciousness, extreme obesity (BMI \geq 95% for age and sex), pregnancy, or bowel motility dysfunction, require careful evaluation before the administration of sedatives. When proper fasting has not been ensured, the increased risks of sedation must be carefully weighed against its benefits, and the lightest effective sedation should be used. In this circumstance, additional techniques for achieving analgesia and patient cooperation, such as distraction, guided imagery, video games, topical and local anesthetics, hematoma block or nerve blocks, and other techniques advised by child life specialists, are particularly helpful and should be considered.^{29,49,182–201,274,275} The use of agents with less risk of depressing protective airway reflexes, such as ketamine, or moderate sedation, which would also maintain protective reflexes, may be preferred.²⁷⁶ Some emergency patients requiring deep sedation (e.g., a trauma patient who just ate a full meal or a child with a bowel obstruction) may need to be intubated to protect their airway before they can be sedated.

Use of immobilization devices (protective stabilization)

Immobilization devices, such as papoose boards, must be applied in such a way as to avoid airway obstruction or chest restriction.^{277–281} The child's head position and respiratory excursions should be checked frequently to ensure airway patency. If an immobilization device is used, a hand or foot should be kept exposed, and the child should never be left unattended. If sedating medications are administered in conjunction with an immobilization device, monitoring must be used at a level consistent with the level of sedation achieved.

Documentation at the time of sedation

Health evaluation: Before sedation, a health evaluation shall be performed by an appropriately licensed practitioner and reviewed by the sedation team at the time of treatment for possible interval changes.²⁸² The purpose of this evaluation is not only to document baseline status but also to determine whether the patient has specific risk factors that may warrant additional consultation before sedation. This evaluation also facilitates the identification of patients who will require more advanced airway or cardiovascular management skills or alterations in the doses or types of medications used for procedural sedation. An important concern for the practitioner is the widespread use of medications that may interfere with drug absorption or metabolism and therefore enhance or shorten the effect time of sedating medications. Herbal medicines (e.g., St. John's wort, ginkgo, ginger, ginseng, garlic) may alter drug pharmacokinetics through

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inhibition of the cytochrome P450 system, resulting in prolonged drug effect and altered (increased or decreased) blood drug concentrations (midazolam, cyclosporine, tacrolimus).^{283–292} Kava may increase the effects of sedatives by potentiating γ aminobutyric acid inhibitory neurotransmission and may increase acetaminopheninduced liver toxicity.^{293–295} Valerian may itself produce sedation that apparently is mediated through the modulation of γ -aminobutyric acid neurotransmission and receptor function.^{291,296–299} Drugs such as erythromycin, cimetidine, and others may also inhibit the cytochrome P450 system, resulting in prolonged sedation with midazolam as well as other medications competing for the same enzyme systems.^{300–304} Medications used to treat HIV infection, some anticonvulsants, immunosuppressive drugs, and some psychotropic medications (often used to treat children with autism spectrum disorder) may also produce clinically important drug-drug interactions.^{305–314} Therefore, a careful drug history is a vital part of the safe sedation of children. The practitioner should consult various sources (a pharmacist, textbooks, online services, or handheld databases) for specific information on drug interactions.^{315–319} The U.S. Food and Drug Administration issued a warning in February 2013 regarding the use of codeine for postoperative pain management in children undergoing tonsillectomy, particularly those with OSA. The safety issue is that some children have duplicated cytochromes that allow greater than expected conversion of the prodrug codeine to morphine, thus resulting in potential overdose; codeine should be avoided for postprocedure analgesia.³²⁰⁻³²⁴

2. Prescriptions. When prescriptions are used for sedation, a copy of the prescription or a note describing the content of the prescription should be in the patient's chart along with a description of the instructions that were given to the responsible person. Prescription medications intended to accomplish procedural sedation must not be administered without the safety net of direct supervision by trained medical/dental personnel. The administration of sedating medications at home poses an unacceptable risk, particularly for infants and preschool-aged children traveling in car safety seats because deaths as a result of this practice have been reported.^{63,257}

The health evaluation should include the following:

- age and weight (in kg) and gestational age at birth (preterm infants may have associated sequelae such as apnea of prematurity); and
- health history, including (1) food and medication allergies and previous allergic or adverse drug reactions; (2) medication/drug history, including dosage, time, route, and site of administration for prescription, over-the-counter, herbal, or illicit drugs; (3) relevant diseases, physical abnormalities (including genetic syndromes), neurologic impairments that might increase the potential for airway obstruction, obesity, a history of snoring or OSA,^{325–328} or cervical spine instability in Down syndrome, Marfan syndrome, skeletal dysplasia, and other conditions; (4) pregnancy status (as many as 1% of menarchal females presenting for general anesthesia at children's hospitals are pregnant)^{329–331} because of concerns for the potential adverse effects of most sedating and anesthetic drugs on the fetus^{329,332–338}; (5) history of prematurity (may be associated with subglottic stenosis or propensity to apnea after sedation); (6) history of any seizure disorder; (7) summary of previous relevant hospitalizations; (8) history of sedation or

general anesthesia and any complications or unexpected responses; and (9) relevant family history, particularly related to anesthesia (e.g., muscular dystrophy, malignant hyperthermia, pseudocholinesterase deficiency).

The review of systems should focus on abnormalities of cardiac, pulmonary, renal, or hepatic function that might alter the child's expected responses to sedating/analgesic medications. A specific query regarding signs and symptoms of sleep disordered breathing and OSA may be helpful. Children with severe OSA who have experienced repeated episodes of desaturation will likely have altered mu receptors and be analgesic at opioid levels one-third to one-half those of a child without OSA^{325–328,339,340}; lower titrated doses of opioids should be used in this population. Such a detailed history will help to determine which patients may benefit from a higher level of care by an appropriately skilled health care provider, such as an anesthesiologist. The health evaluation should also include:

- vital signs, including heart rate, blood pressure, respiratory rate, room air oxygen saturation, and temperature (for some children who are very upset or noncooperative, this may not be possible and a note should be written to document this circumstance);
- physical examination, including a focused evaluation of the airway (tonsillar hypertrophy, abnormal anatomy [e.g., mandibular hypoplasia], high Mallampati score [i.e., ability to visualize only the hard palate or tip of the uvula]) to determine whether there is an increased risk of airway obstruction^{74,341–344};
- physical status evaluation (ASA classification [see Appendix 2]); and
- name, address, and telephone number of the child's home or parent's, or caregiver's cell phone; additional information such as the patient's personal care provider or medical home is also encouraged.

For hospitalized patients, the current hospital record may suffice for adequate documentation of presedation health; however, a note shall be written documenting that the chart was reviewed, positive findings were noted, and a management plan was formulated. If the clinical or emergency condition of the patient precludes acquiring complete information before sedation, this health evaluation should be obtained as soon as feasible.

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Documentation during treatment

The patient's chart shall contain a time-based record that includes the name, route, site, time, dosage/ kilogram, and patient effect of administered drugs. Before sedation, a "time out" should

be performed to confirm the patient's name, procedure to be performed, and laterality and site of the procedure.⁵⁹ During administration, the inspired concentrations of oxygen and inhalation sedation agents and the duration of their administration shall be documented. Before drug administration, special attention must be paid to the calculation of dosage (i.e., mg/kg); for obese patients, most drug doses should likely be adjusted lower to ideal body weight rather than actual weight.³⁴⁵ When a programmable pump is used for the infusion of sedating medications, the dose/kilogram per minute or hour and the child's weight in kilograms should be doublechecked and confirmed by a separate individual. The patient's chart shall contain documentation at the time of treatment that the patient's level of consciousness and responsiveness, heart rate, blood pressure, respiratory rate, expired carbon dioxide values, and oxygen saturation were monitored. Standard vital signs should be further documented at appropriate intervals during recovery until the patient attains predetermined discharge criteria (Appendix 1). A variety of sedation scoring systems are available that may aid this process.^{212,238 346–348} Adverse events and their treatment shall be documented.

Documentation after treatment

A dedicated and properly equipped recovery area is recommended (see Appendices 3 and 4). The time and condition of the child at discharge from the treatment area or facility shall be documented, which should include documentation that the child's level of consciousness and oxygen saturation in room air have returned to a state that is safe for discharge by recognized criteria (see Appendix 1). Patients receiving supplemental oxygen before the procedure should have a similar oxygen need after the procedure. Because some sedation medications are known to have a long half-life and may delay a patient's complete return to baseline or pose the risk of re-sedation^{62,104,256, 349,350} and because some patients will have complex multiorgan medical conditions, a longer period of observation in a less intense observation area (e.g., a step-down observation area) before discharge from medical/dental supervision may be indicated.²³⁹ Several scales to evaluate recovery have been devised and validated.^{212, 346–348, 351, 352} A simple evaluation tool may be the ability of the infant or child to remain awake for at least 20 minutes when placed in a quiet environment.²³⁸

Continuous quality improvement

The essence of medical error reduction is a careful examination of index events and root-cause analysis of how the event could be avoided in the future.^{353–359} Therefore, each facility should maintain records that track all adverse events and significant interventions, such as desaturation; apnea; laryngospasm; need for airway interventions, including the need for placement of supraglottic devices such as an oral airway, nasal trumpet, or LMA; positive-pressure ventilation; prolonged sedation; unanticipated use of reversal agents; unplanned or prolonged hospital admission; sedation failures; inability to complete the procedure; and unsatisfactory sedation, analgesia, or anxiolysis.³⁶⁰ Such events can then be examined for the assessment of risk reduction and improvement in patient/family satisfaction.

Preparation for sedation procedures

Part of the safety net of sedation is using a systematic approach so as to not overlook having an important drug, piece of equipment, or monitor immediately available at the time of a developing emergency. To avoid this problem, it is helpful to use an acronym that allows the same setup and checklist for every procedure. A commonly used acronym useful in planning and preparation for a procedure is SOAPME, which represents the following:

S = Size-appropriate suction catheters and a functioning suction apparatus (e.g., Yankauer-type suction).

O= an adequate Oxygen supply and functioning flow meters or other devices to allow its delivery.

A = size-appropriate Airway equipment (e.g., bag-valve-mask or equivalent device [functioning]), nasopharyngeal and oropharyngeal airways, LMA, laryngoscope blades (checked and functioning), endotracheal tubes, stylets, face mask.

 \mathbf{P} = Pharmacy: all the basic drugs needed to support life during an emergency, including antagonists as indicated.

 \mathbf{M} = Monitors: functioning pulse oximeter with size-appropriate oximeter probes,^{361,362} end-tidal carbon dioxide monitor, and other monitors as appropriate for the procedure (e.g., noninvasive blood pressure, ECG, stethoscope).

E = special Equipment or drugs for a particular case (e.g., defibrillator).

Specific guidelines for intended level of sedation

Minimal sedation

Minimal sedation (old terminology, "anxiolysis") is a drug induced state during which patients respond normally to verbal commands. Although cognitive function and coordination may be impaired, ventilatory and cardiovascular functions are unaffected. Children who have received minimal sedation generally will not require more than observation and intermittent assessment of their level of sedation. Some children will become moderately sedated despite the intended level of minimal sedation; should this occur, then the guidelines for moderate sedation apply.^{85,363}

Moderate sedation

Moderate sedation (old terminology, "conscious sedation" or "sedation/analgesia") is a druginduced depression of consciousness during which patients respond purposefully to verbal commands or after light tactile stimulation. No interventions are required to maintain a patent airway, and spontaneous ventilation is adequate. Cardiovascular function is usually maintained. The caveat that loss of consciousness should be unlikely is a particularly important aspect of the definition of moderate sedation; drugs and techniques used should carry a margin of safety wide enough to render unintended loss of consciousness unlikely. Because the patient who receives moderate sedation may progress into a state of deep sedation and obtundation, the practitioner should be prepared to increase the level of vigilance corresponding to what is necessary for deep sedation.⁸⁵

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Personnel

The practitioner. The practitioner responsible for the treatment of the patient and/or the administration of drugs for sedation must be competent to use such techniques, to provide the level of monitoring described in these guidelines, and to manage complications of these techniques (i.e., to be able to rescue the patient). Because the level of intended sedation may be exceeded, the practitioner must be sufficiently skilled to rescue a child with apnea, laryngospasm, and/or airway obstruction, including the ability to open the airway, suction secretions, provide CPAP, and perform successful <u>bag-valve-mask</u> ventilation should the child progress to a level of deep sedation. Training in, and maintenance of, advanced pediatric airway skills is required (e.g., pediatric advanced life support [**PALS**]); regular skills reinforcement with simulation is strongly encouraged.^{79,80,128,130,217–220,364}

Support personnel. The use of moderate sedation shall include the provision of a person, in addition to the practitioner, whose responsibility is to monitor appropriate physiologic parameters and to assist in any supportive or resuscitation measures, if required. This individual may also be responsible for assisting with interruptible patient-related tasks of short duration, such as holding an instrument or troubleshooting equipment.⁶⁰ This individual should be trained in and capable of providing advanced airway skills (e.g., PALS). The support person shall have specific assignments in the event of an emergency and current knowledge of the emergency cart inventory. The practitioner and all ancillary personnel should participate in periodic reviews, simulation of rare emergencies, and practice drills of the facility's emergency protocol to ensure proper function of the equipment and coordination of staff roles in such emergencies.^{133,365–367} It is recommended that at least 1 practitioner be skilled in obtaining vascular access in children.

Monitoring and documentation

Baseline.

Before the administration of sedative medications, a baseline determination of vital signs shall be documented. For some children who are very upset or uncooperative, this may not be possible, and a note should be written to document this circumstance.

During the procedure. The physician/dentist or his or her designee shall document the name, route, site, time of administration, and dosage of all drugs administered. If sedation is being directed by a physician who is not personally administering the medications, then recommended practice is for the qualified health care provider administering the medication to confirm the dose verbally before administration. There shall be continuous monitoring of oxygen saturation and heart rate; when bidirectional verbal communication between the provider and patient is appropriate and possible (i.e., patient is developmentally able and purposefully communicates), monitoring of ventilation by (1) capnography (preferred) or (2) amplified, audible pretracheal stethoscope (e.g., BluetoothTM technology)³⁶⁸⁻³⁷¹ or precordial stethoscope is strongly recommended. If bi-directional verbal communication is not appropriate or not possible, monitoring of ventilation by capnography (preferred), amplified, audible pretracheal stethoscope, or precordial stethoscope is required. Heart rate, respiratory rate, blood pressure, oxygen saturation, and expired carbon dioxide values should be recorded, at minimum, every 10 minutes in a time-based record. Note that the exact value of expired carbon dioxide is less important than simple assessment of continuous respiratory gas exchange. In some situations, in which there is

excessive patient agitation or lack of cooperation or during certain procedures such as bronchoscopy, dentistry, or repair of facial lacerations capnography may not be feasible, and this situation should be documented. For uncooperative children, it is often helpful to defer the initiation of capnography until the child becomes sedated. Similarly, the stimulation of blood pressure cuff inflation may cause arousal or agitation; in such cases, blood pressure monitoring may be counterproductive and may be documented at less frequent intervals (e.g., 10-15 minutes, assuming the patient remains stable, well oxygenated, and well perfused). Immobilization devices (protective stabilization) should be checked to prevent airway obstruction or chest restriction. If a restraint device is used, a hand or foot should be kept exposed. The child's head position should be continuously assessed to ensure airway patency.

After the procedure. The child who has received moderate sedation must be observed in a suitably equipped recovery area, which must have a functioning suction apparatus as well as the capacity to deliver >90% oxygen and positive-pressure ventilation (bag-valve mask) with an adequate oxygen capacity as well as age- and size-appropriate rescue equipment and devices. The patient's vital signs should be recorded at specific intervals (e.g., every 10–15 minutes). If the patient is not fully alert, oxygen saturation and heart rate monitoring shall be used continuously until appropriate discharge criteria are met (see Appendix 1). Because sedation medications with a long half-life may delay the patient's complete return to baseline or pose the risk of re-sedation, some patients might benefit from a longer period of less intense observation (e.g., a step-down observation area where multiple patients can be observed simultaneously) before discharge from medical/ dental supervision (see section entitled "Documentation Before Sedation" above).^{62,256,349,350} A simple evaluation tool may be the ability of the infant or child to remain awake for at least 20 minutes when placed in a quiet environment.²³⁸ Patients who have received reversal agents, such as flumazenil or naloxone, will require a longer period of observation, because the duration of the drugs administered may exceed the duration of the antagonist, resulting in re-sedation.

Deep sedation/General anesthesia

"Deep sedation" ("deep sedation/ analgesia") is a drug-induced depression of consciousness during which patients cannot be easily aroused but respond purposefully after repeated verbal or painful stimulation (e.g., purposefully pushing away the noxious stimuli). Reflex withdrawal from a painful stimulus is not considered a purposeful response and is more consistent with a state of general anesthesia. The ability to independently maintain ventilatory function may be impaired. Patients may require assistance in maintaining a patent airway, and spontaneous ventilation may be inadequate. Cardiovascular function is usually maintained. A state of deep sedation may be accompanied by partial or complete loss of protective airway reflexes. Patients may pass from a state of deep sedation to the state of general anesthesia. In some situations, such as during MRI, one is not usually able to assess responses to stimulation, because this would defeat the purpose of sedation, and one should assume that such patients are deeply sedated.

"General anesthesia" is a drug-induced loss of consciousness during which patients are not arousable, even by painful stimulation. The ability to independently maintain ventilatory function is often impaired. Patients often require assistance in maintaining a patent airway, and

positive-pressure ventilation may be required because of depressed spontaneous ventilation or drug-induced depression of neuromuscular function. Cardiovascular function may be impaired.

Personnel

During deep sedation, there must be 1 person whose only responsibility is to constantly observe the patient's vital signs, airway patency, and adequacy of ventilation and to either administer drugs or direct their administration. This individual must, at a minimum, be trained in PALS and capable of assisting with any emergency event. At least 1 individual must be present who is trained in and capable of providing advanced pediatric life support and who is skilled to rescue a child with apnea, laryngospasm, and or airway obstruction. Required skills include the ability to open the airway, suction secretions, provide CPAP, insert supraglottic devices (oral airway, nasal trumpet, LMA), and perform successful bag valve-mask ventilation, tracheal intubation, and cardiopulmonary resuscitation.

During office-based deep sedation/general anesthesia of a pediatric patient, there must be at least two individuals, an independent observer (licensed anesthesia provider) and the operating dentist; present with the patient throughout the extent of the procedure, including recovery. Both individuals, at a minimum, must maintain current training and certification in pediatric advanced life support (PALS) or advanced pediatric life support (APLS). One of these two must remain an independent observer, independent of performing or assisting with the dental procedure. This individual's sole responsibility is to administer pharmacologic agents or direct their delivery and to continuously monitor and record the patient's physiologic vital signs, cardiopulmonary and neurologic status, airway patency and adequacy of ventilation, while assuming the lead role during the management of any perioperative emergencies. Because the intended level of sedation may exceed the practitioner's objective, the practitioner must be skilled and trained to establish intravenous access, rescue a child with apnea, laryngospasm, airway obstruction, hypotension, anaphylaxis, or cardiopulmonary arrest including the ability to suction, open and maintain the airway. He/She must be experienced in placement of endotracheal tubes or supraglottic devices (oral airway, nasal trumpet, laryngeal mask airway, or iGel[©]), providing constant positive airway pressure (CPAP) using bag-valve-mask ventilation, cardiopulmonary resuscitation, and the administration of rescue medications. As permitted by state regulation, the anesthesia provider may be a dentist or physician anesthesiologist, certified registered nurse anesthetist (CRNA), or a second oral and maxillofacial surgeon.

During deep sedation/general anesthesia of a pediatric patient in a hospital or ambulatory surgical center setting, at least two individuals must be present with the patient throughout the extent of the procedure experienced in in patient rescue and with current PALS or APLS certification. One of these individuals may either administer pharmacologic agents or direct their delivery by a skilled independent observer. The abilities of the individual directing or administering sedation/anesthetic medications must include those described in the previous paragraph. Providers who may fulfill the role of the skilled independent observer in a hospital or ambulatory surgical center, as permitted by state regulation, must be a dentist anesthesiologist, an oral and maxillofacial surgeon, physician with sedation training and advanced airway skills such as, but not limited to, an anesthesiologist, or other medical specialists with the requisite licensure, training and competencies; a CRNA, or certified anesthesiology assistant (CAA); a

nurse with advanced emergency management skills such as several years of experience in the emergency room, pediatric recovery room, or intensive care setting (i.e. nurses who are experienced with assisting the individual administering or directing sedation with patient rescue during life-threatening emergencies).

Equipment

In addition to the equipment needed for moderate sedation, an ECG monitor and a defibrillator for use in pediatric patients should be readily available.

Vascular access

Patients receiving deep sedation should have an intravenous line placed at the start of the procedure or have a person skilled in establishing vascular access in pediatric patients immediately available.

Monitoring

A competent individual shall observe the patient continuously. Monitoring shall include all parameters described for moderate sedation. Vital signs, including heart rate, respiratory rate, blood pressure, oxygen saturation, and expired carbon dioxide, must be documented at least every 5 minutes in a time-based record. Capnography should be used for almost all deeply sedated children because of the increased risk of airway/ ventilation compromise. Capnography may not be feasible if the patient is agitated or uncooperative during the initial phases of sedation or during certain procedures, such as bronchoscopy or repair of facial lacerations, and this circumstance should be documented. For uncooperative children, the capnography monitor may be placed once the child becomes sedated. Note that if supplemental oxygen is administered, the capnograph may underestimate the true expired carbon dioxide value; of more importance than the numeric reading of exhaled carbon dioxide is the assurance of continuous respiratory gas exchange (i.e., continuous waveform). Capnography is particularly useful for patients who are difficult to observe (e.g., during MRI or in a darkened room).^{64,67,72,90,96,110,159–162,164–166,167–170,372–375}

The physician/dentist or his or her designee shall document the name, route, site, time of administration, and dosage of all drugs administered. If sedation is being directed by a physician who is not personally administering the medications, then recommended practice is for the nurse administering the medication to confirm the dose verbally before administration.

The inspired concentrations of inhalation sedation agents and oxygen and the duration of administration shall be documented.

Postsedation care

The facility and procedures followed for postsedation care shall conform to those described under "moderate sedation." The initial recording of vital signs should be documented at least every 5 minutes. Once the child begins to awaken, the recording intervals may be increased to 10 to 15 minutes. Table 2 summarizes the equipment, personnel, and monitoring requirements for moderate and deep sedation.

Special considerations

Neonates and former preterm infants

Neonates and former preterm infants require specific management, because immaturity of hepatic and renal function may alter the ability to metabolize and excrete sedating medications,³⁷⁶ resulting in prolonged sedation and the need for extended post-sedation monitoring. Former preterm infants have an increased risk of postanesthesia apnea,³⁷⁷ but it is unclear whether a similar risk is associated with sedation, because this possibility has not been systematically investigated.³⁷⁸

Other concerns regarding the effects of anesthetic drugs and sedating medications on the developing brain are beyond the scope of this document. At this point, the research in this area is preliminary and inconclusive at best, but it would seem prudent to avoid unnecessary exposure to sedation if the procedure is unlikely to change medical/dental management (e.g., a sedated MRI purely for screening purposes in preterm infants).^{379–382}

Local anesthetic agents

All local anesthetic agents are cardiac depressants and may cause central nervous system excitation or depression. Particular weight-based attention should be paid to cumulative dosage in all children.^{118,120,125,383–386} To ensure that the patient will not receive an excessive dose, the maximum allowable safe dosage (e.g., mg/kg) should be calculated before administration. There may be enhanced sedative effects when the highest recommended doses of local anesthetic drugs are used in combination with other sedatives or opioids (see Tables 3 and 4 for limits and conversion tables of commonly used local anesthetics).^{118,125,387-400} In general, when administering local anesthetic drugs, the practitioner should aspirate frequently to minimize the likelihood that the needle is in a blood vessel; lower doses should be used when injecting into vascular tissues.⁴⁰¹ If high doses or injection of amide local anesthetics (bupivacaine and ropivacaine) into vascular tissues is anticipated, then the immediate availability of a 20% lipid emulsion for the treatment of local anesthetic toxicity is recommended (Tables 3 and 5).^{402–} ⁴⁰⁹ Topical local anesthetics are commonly used and encouraged, but the practitioner should avoid applying excessive doses to mucosal surfaces where systemic uptake and possible toxicity (seizures, methemoglobinemia) could result and to remain within the manufacturer's recommendations regarding allowable surface area application.⁴¹⁰⁻⁴¹⁵

Pulse oximetry

Newer pulse oximeters are less susceptible to motion artifacts and may be more useful than older oximeters that do not contain updated software.^{416–420} Oximeters that change tone with changes in hemoglobin saturation provide immediate aural warning to everyone within hearing distance. The oximeter probe must be properly positioned; clip-on devices are easy to displace, which may produce artifactual data (under-or overestimation of oxygen saturation).^{361,362}

Capnography

Expired carbon dioxide monitoring is valuable to diagnose the simple presence or absence of respirations, airway obstruction, or respiratory depression, particularly in patients sedated in less-

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accessible locations, such as in MRI machines or darkened rooms.^{64,66,67,72,90,96,110,159–162,164–170,372–375,421–427} In patients receiving supplemental oxygen, capnography facilitates the recognition of apnea or airway obstruction several minutes before the situation would be detected just by pulse oximetry. In this situation, desaturation would be delayed due to increased oxygen reserves; capnography would enable earlier intervention.¹⁶¹ One study in children sedated in the emergency department found that the use of capnography reduced the incidence of hypoventilation and desaturation (7% to 1%).¹⁷⁴ The use of expired carbon dioxide monitoring devices is now required for almost all deeply sedated children (with rare exceptions), particularly in situations in which other means of assessing the adequacy of ventilation are limited. Several manufacturers have produced nasal cannulae that allow simultaneous delivery of oxygen and measurement of expired carbon dioxide values.^{421,422,427} Although these devices can have a high degree of false-positive alarms, they are also very accurate for the detection of complete airway obstruction or apnea.^{164,168,169} Taping the sampling line under the nares under an oxygen face mask or nasal hood will provide similar information. The exact measured value is less important than the simple answer to the question: Is the child exchanging air with each breath?

Processed EEG (Bispectral Index)

Although not new to the anesthesia community, the processed EEG (bispectral index [**BIS**]) monitor is slowly finding its way into the sedation literature.⁴²⁸ Several studies have attempted to use BIS monitoring as a means of noninvasively assessing the depth of sedation. This technology was designed to examine EEG signals and, through a variety of algorithms, correlate a number with depth of unconsciousness: that is, the lower the number, the deeper the sedation. Unfortunately, these algorithms are based on adult patients and have not been validated in children of varying ages and varying brain development. Although the readings correspond quite well with the depth of propofol sedation, the numbers may paradoxically go up rather than down with sevoflurane and ketamine because of central excitation despite a state of general anesthesia or deep sedation.^{429,430}

Opioids and benzodiazepines have minimal and variable effects on the BIS. Dexmedetomidine has minimal effect with EEG patterns, consistent with stage 2 sleep.⁴³¹ Several sedation studies have examined the utility of this device and degree of correlation with standard sedation scales.^{347,363,432–435} It appears that there is some correlation with BIS values in moderate sedation, but there is not a reliable ability to distinguish between deep sedation and moderate sedation or deep sedation from general anesthesia.⁴³² Presently, it would appear that BIS monitoring might provide useful information only when used for sedation with propofol³⁶³; in general, it is still considered a research tool and not recommended for routine use.

Adjuncts to airway management and resuscitation

The vast majority of sedation complications can be managed with simple maneuvers, such as supplemental oxygen, opening the airway, suctioning, placement of an oral or nasopharyngeal airway, and bag-mask-valve ventilation. Rarely, tracheal intubation is required for more prolonged ventilatory support. In addition to standard tracheal intubation techniques, a number of supraglottic devices are available for the management of patients with abnormal airway anatomy or airway obstruction. Examples include the LMA, the cuffed oropharyngeal airway, and a variety of kits to perform an emergency cricothyrotomy.^{436,437}

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The largest clinical experience in pediatrics is with the LMA, which is available in multiple sizes, including those for late preterm and term neonates. The use of the LMA is now an essential addition to advanced airway training courses, and familiarity with insertion techniques can be life-saving.⁴³⁸⁻⁴⁴² The LMA can also serve as a bridge to secure airway management in children with anatomic airway abnormalities.^{443,444} Practitioners are encouraged to gain experience with these techniques as they become incorporated into PALS courses.

Another valuable emergency technique is intraosseous needle placement for vascular access. Intraosseous needles are available in several sizes; insertion can be life-saving when rapid intravenous access is difficult. A relatively new intraosseous device (EZ-IO Vidacare, now part of Teleflex, Research Triangle Park, N.C.) is similar to a hand-held battery powered drill. It allows rapid placement with minimal chance of misplacement; it also has a low-profile intravenous adapter.^{445–450} Familiarity with the use of these emergency techniques can be gained by keeping current with resuscitation courses, such as PALS and advanced pediatric life support.

Patient simulators

High-fidelity patient simulators are now available that allow physicians, dentists, and other health care providers to practice managing a variety of programmed adverse events, such as apnea, bronchospasm, and laryngospasm.^{133,220,450–452} The use of such devices is encouraged to better train medical professionals and teams to respond more effectively to rare events.^{128, 131,451,453–455} One study that simulated the quality of cardiopulmonary resuscitation compared standard management of ventricular fibrillation versus rescue with the EZ-IO for the rapid establishment of intravenous access and placement of an LMA for establishing a patent airway in adults; the use of these devices resulted in more rapid establishment of vascular access and securing of the airway.⁴⁵⁶

Monitoring during MRI

The powerful magnetic field and the generation of radiofrequency emissions necessitate the use of special equipment to provide continuous patient monitoring throughout the MRI scanning procedure.^{457–459} MRI-compatible pulse oximeters and capnographs capable of continuous function during scanning should be used in any sedated or restrained pediatric patient. Thermal injuries can result if appropriate precautions are not taken; the practitioner is cautioned to avoid coiling of all wires (oximeter, ECG) and to place the oximeter probe as far from the magnetic coil as possible to diminish the possibility of injury. ECG monitoring during MRI has been associated with thermal injury; special MRI-compatible ECG pads are essential to allow safe monitoring.^{460–463} If sedation is achieved by using an infusion pump, then either an MRI-compatible pump is required, or the pump must be situated outside of the room with long infusion tubing so as to maintain infusion accuracy. All equipment must be MRI compatible, including laryngoscope blades and handles, oxygen tanks, and any ancillary equipment. All individuals, including parents, must be screened for ferromagnetic materials, phones, pagers, pens, credit cards, watches, surgical implants, pacemakers, etc., before entry into the MRI suite.

Nitrous oxide

Inhalation sedation/analgesia equipment that delivers nitrous oxide must have the capacity of

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delivering 100% and never less than 25% oxygen concentration at a flow rate appropriate to the size of the patient. Equipment that delivers variable ratios of nitrous oxide >50% to oxygen that covers the mouth and nose must be used in conjunction with a calibrated and functional oxygen analyzer. All nitrous oxide-to-oxygen inhalation devices should be calibrated in accordance with appropriate state and local requirements. Consideration should be given to the National Institute of Occupational Safety and Health Standards for the scavenging of waste gases.⁴⁶⁴ Newly constructed or reconstructed treatment facilities, especially those with piped-in nitrous oxide and oxygen, must have appropriate state or local inspections to certify proper function of inhalation sedation/analgesia systems before any delivery of patient care.

Nitrous oxide in oxygen, with varying concentrations, has been successfully used for many years to provide analgesia for a variety of painful procedures in children.^{14,36,49,98,465–493} The use of nitrous oxide for minimal sedation is defined as the administration of nitrous oxide of \leq 50% with the balance as oxygen, without any other sedative, opioid, or other depressant drug before or concurrent with the nitrous oxide to an otherwise healthy patient in ASA class I or II. The patient is able to maintain verbal communication throughout the procedure. It should be noted that although local anesthetics have sedative properties, for purposes of this guideline they are not considered sedatives in this circumstance. If nitrous oxide in oxygen is combined with other sedating medications, such as chloral hydrate, midazolam, or an opioid, or if nitrous oxide is used in concentrations >50%, the likelihood for moderate or deep sedation increases.^{107,197,492,494,495} In this situation, the practitioner is advised to institute the guidelines for moderate or deep sedation, as indicated by the patient's response.⁴⁹⁶

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Supplemental information

Appendix 1. Recommended Discharge Criteria

- 1. Cardiovascular function and airway patency are satisfactory and stable.
- 2. The patient is easily arousable, and protective reflexes are intact.
- 3. The patient can talk (if age appropriate).
- 4. The patient can sit up unaided (if age appropriate).
- 5. For a very young or handicapped child incapable of the usually expected responses, the presedation level of responsiveness or a level as close as possible to the normal level for that child should be achieved.
- 6. The state of hydration is adequate.

Appendix 2. ASA Physical Status Classification

Class I A normally healthy patient.

Class II A patient with mild systemic disease (e.g., controlled reactive airway disease).

Class III A patient with severe systemic disease (e.g., a child who is actively wheezing).

Class IV A patient with severe systemic disease that is a constant threat to life (e.g., a child with status asthmaticus).

Class V A moribund patient who is not expected to survive without the operation (e.g., a patient with severe cardiomyopathy requiring heart transplantation).

Appendix 3. Drugs* That May Be Needed to Rescue a Sedated Patient⁴⁴

Albuterol for inhalation Ammonia spirits Atropine Diphenhydramine Diazepam Epinephrine (1:1000, 1:10 000) Flumazenil Glucose (25 percent or 50 percent) Lidocaine (cardiac lidocaine, local infiltration)

Lorazepam Methylprednisolone Naloxone Oxygen Fosphenytoin Racemic epinephrine Rocuronium Sodium bicarbonate Succinylcholine

* The choice of emergency drugs may vary according to individual or procedural needs.

Appendix 4. Emergency Equipment [†] That May Be Needed to Rescue a Sedated Patient [‡]

Intravenous Equipment

Assorted IV catheters (e.g., 24-, 22-, 20-, 18-, 16-gauge) Tourniquets Alcohol wipes Adhesive tape Assorted syringes (e.g., 1-, 3-, 5-, 10-mL) IV tubing Pediatric drip (60 drops/mL) Pediatric burette Adult drip (10 drops/mL) Extension tubing 3-way stopcocks IV fluid Lactated Ringer solution Normal saline solution D₅ 0.25 normal saline solution Pediatric IV boards Assorted IV needles (e.g., 25-, 22-, 20-, and 18-gauge) Intraosseous bone marrow needle Sterile gauze pads

Airway Management Equipment

Face masks (infant, child, small adult, medium adult, large adult) Breathing bag and valve set Oropharyngeal airways (infant, child, small adult, medium adult, large adult) Nasopharyngeal airways (small, medium, large) Laryngeal mask airways (1, 1.5, 2, 2.5, 3, 4, and 5) Laryngoscope handles (with extra batteries) Laryngoscope blades (with extra light bulbs) 2019 proposed changes/additions to oral health policies and clinical recommendations of the American Academy of Pediatric Dentistry

This draft does not constitute an official AAPD health oral policy or clinical recommendation until approval by the General Assembly. Circulation is limited to AAPD members.

Straight (Miller) No. 1, 2, and 3 Curved (Macintosh) No. 2 and 3

Endotracheal tubes (2.5, 3.0, 3.5, 4.0, 4.5, 5.0, 5.5, and 6.0 uncuffed and 6.0, 7.0, and 8.0 cuffed) Stylettes (appropriate sizes for endotracheal tubes) Surgical lubricant Suction catheters (appropriate sizes for endotracheal tubes) Yankauer-type suction Nasogastric tubes Nebulizer with medication kits Gloves (sterile and nonsterile, latex free)

† The choice of emergency equipment may vary according to individual or procedural needs.‡ The practitioner is referred to the SOAPME acronym described in the text in preparation for sedating a child for a procedure.

Use of Anesthesia Providers in the Administration of Office-based Deep Sedation/General Anesthesia to the Pediatric Dental Patient

Review Council Council on Clinical Affairs

Latest Revision $\frac{2018}{2019^{1}}$

Abbreviations

AAPD: American Academy of Pediatric Dentistry ASA: American Society of Anesthesiologists CAA: Certified anesthesiologist assistant CO₂: Carbon dioxide CRNA: Certified registered nurse anesthetist

Purpose

The American Academy of Pediatric Dentistry (**AAPD**) recognizes that there are pediatric dental patients for whom routine dental care using nonpharmacologic behavior guidance techniques is not a viable approach.¹ The AAPD intends this guideline to assist the dental practitioner who elects to use a licensed anesthesia provider for the administration of deep sedation/general anesthesia for pediatric dental patients in a dental office or other facility outside of an accredited hospital or ambulatory surgical center. This document discusses personnel, facilities, documentation, and quality assurance mechanisms necessary to provide optimal and responsible patient care.

Methods

Recommendations on the use of anesthesia providers in the administration of office-based deep sedation/general anesthesia were developed by the Clinical Affairs Committee – Sedation and General Anesthesia Subcommittee and adopted in 2001. This document is a revision of the previous version, last revised in 2017. The revision is based upon a review of current dental and medical literature pertaining to deep sedation/general anesthesia of dental patients, including a search of the PubMed[®]/MEDLINE database using the terms: office-based general anesthesia, pediatric sedation, deep sedation, sleep dentistry, and dental sedation; fields: all; limits: humans, all children from birth through age 18, English, clinical trials, and literature reviews. The search

¹ 2019 revision limited to Personnel section.

returned 69 articles; the reviewers agreed upon the inclusion of 12 articles that met the defined criteria. When data did not appear sufficient or were inconclusive, recommendations were based upon expert and/or consensus opinion by experienced researchers and clinicians.

Background

Pediatric dentists seek to provide oral health care to infants, children, adolescents, and persons with special health care needs in a manner that promotes excellence in quality of care and concurrently induces a positive attitude in the patient toward dental treatment. Behavior guidance techniques have allowed most pediatric dental patients to receive treatment in the dental office with minimal discomfort and without expressed fear. Minimal or moderate sedation has allowed others who are less compliant to receive treatment. Some children and individuals with special care needs who have extensive oral healthcare needs, acute situational anxiety, uncooperative age-appropriate behavior, immature cognitive functioning, disabilities, or medical conditions require deep sedation/general anesthesia to receive dental treatment in a safe and humane fashion.² Access to hospital-based anesthesia services may be limited for a variety of reasons, including restriction of coverage of by third-party payors.^{2,3} Pediatric dentists and others who treat children can provide for the administration of deep sedation/ general anesthesia by utilizing properly trained and currently licensed anesthesia providers in their offices or other facilities outside of the traditional surgical setting.

Office-based deep sedation/general anesthesia can provide benefits for the patient and the dental team. Such benefits may include:

- improved access to care;
- improved ease and efficiency of scheduling;
- decreased administrative procedures and facility fees when compared to a surgical center or hospital;
- minimized likelihood of patient's recall of procedures;
- · decreased patient movement which may optimize quality of care; and
- use of traditional dental delivery systems with access to a full complement of dental equipment, instrumentation, supplies, and auxiliary personnel.

The use of licensed anesthesia providers to administer deep sedation/general anesthesia in the pediatric dental population is an accepted treatment modality.⁴⁻⁸ Caution must be used in patients younger than two years of age. Practitioners must always be mindful of the increased risk associated with <u>office-based</u> deep sedation/general anesthesia in the infant and toddler populations. This level of pharmacologic behavioral modification should only be used when the risk of orofacial disease outweighs the benefits of monitoring, interim therapeutic restoration, or arresting medicaments to slow or stop the progression of caries. The AAPD supports the provision of deep sedation/general anesthesia when clinical indications have been met and additional properly-trained and credentialed personnel and appropriate facilities are used.^{1,3,4} In many cases, the patient may be treated in an appropriate outpatient facility (including the dental

office) because the extensive medical resources of a hospital may not be deemed necessary for delivering routine health care.

Recommendations

Clinicians may consider using deep sedation or general anesthesia in the office to facilitate the provision of oral health care. Practitioners choosing to use these modalities must be trained in rescue emergency procedures and be familiar with their patient's medical history, as well as the regulatory and professional liability insurance requirements needed to provide this level of pharmacologic behavior management. This guideline does not supersede, nor is it to be used in deference to, federal, state, and local credentialing and licensure laws, regulations, and codes.

Personnel

Deep sedation/general anesthesia techniques in the dental office require <u>the presence of</u> at least <u>two three of the following</u> individuals <u>throughout the extent of the procedure</u>:

- independently practicing and currently licensed anesthesia provider independent <u>licensed</u> anesthesia provider, who is independent of performing or assisting with the dental procedure.
- operating dentist.
- support personnel.

The anesthesia care provider's responsibilities are to administer drugs or direct their administration and to continuously monitor the patient's vital signs, airway patency, cardiovascular and neurological status, and adequacy of ventilation. Both the surgical and anesthesia teams are responsible for maintaining optimal patient positioning, such as keeping the head and neck aligned and supported while padding all pressure points. Additional attention should be placed on moving extremities during long procedures so as to avoid the possibility of complications secondary to prolonged immobility (e.g., peripheral neuropathy).

It is the exclusive responsibility of treating practitioners the operating dentist, when employing anesthesia providers to administer deep sedation/general anesthesia, to verify and carefully review their credentials and experience. Significant pediatric training, including anesthesia care of the very young, and experience in a dental setting are important considerations, especially when caring for young pediatric and special needs populations. In order to provide anesthesia services in an office-based setting:

- the <u>licensed</u> anesthesia care provider must be a licensed dental and/or medical practitioner with current state certification to independently administer deep sedation/general anesthesia in a dental office. He/She must be in compliance with state and local laws regarding anesthesia practices. Laws vary from state to state and may supersede any portion of this document.
- if state law permits a certified registered nurse anesthetist (CRNA) or certified anesthesiologist assistant (CAA) to function under the direct supervision of a dentist, the

dentist is required to have completed training in deep sedation/general anesthesia and be licensed or permitted for that level of pharmacologic management, appropriate to state law. Furthermore, to maximize patient safety, the dentist supervising the CRNA or CAA would not simultaneously be providing dental treatment. The CRNA or CAA must be licensed with current state certification to administer deep sedation/general anesthesia in a dental office. He/She must be in compliance with state and local laws regarding anesthesia practices. Laws vary from state to state and may supersede any portion of this document.

The dentist and anesthesia care provider must be compliant with the American Academy of Pediatrics/AAPD's Guideline on Monitoring and Management of Pediatric Patients Before, During, and After Sedation for Diagnostic and Therapeutic Procedures: Update 2016⁴ or other appropriate guideline(s) of the American Dental Association (ADA), American Society of Dentist Anesthesiologists (ASDA), American Society of Anesthesiologists (ASA), and other organizations with recognized professional expertise and stature. The recommendations in this document may be exceeded at any time if the change involves improved safety and/or is superseded by state law. The dentist and licensed anesthesia provider must collaborate to enhance patient safety. Continuous and effective perioperative communication and appropriately timed interventions are essential in mitigating adverse events or outcomes. The dentist introduces the concept of deep sedation/general anesthesia to the parent, justifies its necessity, and provides appropriate preoperative instructions and informational materials. The dentist or his/her designee coordinates medical consultations when necessary and conveys pertinent information to the anesthesia care provider. The anesthesia care provider explains potential risks and obtains informed consent for sedation/anesthesia. Office staff should understand their additional roles and responsibilities and special considerations (e.g., loss of protective reflexes) associated with office-based deep sedation/general anesthesia.

Both the licensed anesthesia provider and the operating dentist, individuals, at a minimum, must at a minimum, maintain current certification and training in pediatric advanced life support (PALS) or advanced pediatric life support (APLS), patient rescue, and be capable of managing potential airway complications related to the delivery of all levels of sedation.

The licensed anesthesia provider's sole responsibility is to administer pharmacologic agents or direct their administration and to continuously monitor and record the patient's depth of sedation, physiologic vital signs, cardiopulmonary and neurologic status, airway patency and adequacy of ventilation, while assuming the lead role during the management of any perioperative emergencies. Because the intended level of sedation may exceed the practitioner's objective, the practitioner must be skilled to establish intravenous access, rescue a child with apnea, laryngospasm, airway obstruction, hypotension, anaphylaxis, or cardiopulmonary arrest including the ability to suction and open the airway.⁴ He/She must be experienced in placement of endotracheal tubes or supraglottic devices (oral airway, nasal trumpet, laryngeal mask airway, or *i*Gel[©]), providing constant positive airway pressure (CPAP), using bag-valve-mask

ventilation, cardiopulmonary resuscitation, and the administration of rescue medications. As permitted by state regulation, the anesthesia provider may be one of the following:

- <u>dentist or physician anesthesiologist</u>
- <u>certified registered nurse anesthetist, or</u>
- an oral and maxillofacial surgeon.

It is the responsibility of the anesthesia provider to ensure that the operating dentist and supportive staff is capable of providing him/her with skilled assistance support and have an established emergency and transport protocol in place in the event of an adverse incident.

Advanced training in recognition and management of pediatric emergencies is critical in providing safe sedation and anesthetic care. During deep sedation/general anesthesia in the dental setting, there must be at least two individuals present with the skills in patient rescue and pediatric advanced life support (e.g., PALS) and capable of managing any emergency event.⁴ One of the two must be an independent observer who sole responsibility is to constantly observe the patient's vital signs, levels of sedation, airway patency, and adequacy of ventilation. The independent observer must be capable of recognizing the depth of sedation as well as be skilled to establish intravenous access and draw up and administer rescue medications. This provider must have management skills to rescue the non-breathing child, a child with airway obstruction, and a child with hypotension, anaphylaxis, or cardiac arrest; this would include the ability to open the airway, suction secretions, provide continuous positive airway pressure (CPAP), insert supraglottic devices (oral airway, nasal trumpet, laryngeal mask airway [LMA]), and perform successful bag-valve-mask ventilation, tracheal intubation, and cardiopulmonary resuscitation.⁴ The independent observer must be one of the following: (1) a physician anesthesiologist, (2) a dental anesthesiologist, (3) a certified registered nurse anesthetist, (4) an oral and maxillofacial surgeon. The second skilled individual (e.g., the responsible dental practitioner) must be trained in and capable of providing pediatric advanced life support and skilled in assisting the independent observer with the rescue of a child with any of the adverse events described above.

Personnel experienced in post anesthetic recovery care and trained in advanced resuscitative techniques (e.g., PALS) must be in attendance and provide continuous respiratory and cardiovascular monitoring during the recovery period.⁴ The supervising anesthesia provider, not the operating dentist, shall determine when the patient exhibits respiratory and cardiovascular stability and appropriate discharge criteria⁴ have been met. The operating dentist and his/her clinical staff must be well-versed in emergency recognition, rescue, and emergency protocols including maintaining cardiopulmonary resuscitation certification for healthcare providers.⁶ In addition, it is highly recommended that the operating dentist be trained in advanced resuscitative techniques. Contact numbers for local emergency medical and ambulance services must be readily available, and a protocol for immediate access to backup emergency services must be clearly outlined.⁴ Emergency preparedness must be updated and practiced on a regular (e.g., semi-annual) basis [see Table 1], so as to keep all staff members up to date on established protocols.⁹

Facilities

A continuum exists that extends from wakefulness across all levels of sedation. Often these levels are not easily differentiated, and patients may drift among them.¹⁰ When anesthesia care providers are utilized for office-based administration of deep sedation or general anesthesia, the facilities in which the dentist practices must meet the guidelines and appropriate local, state, and federal codes for administration of the deepest possible level of sedation/anesthesia. Facilities must be in compliance with applicable laws, codes, and regulations pertaining to controlled drug storage, fire prevention, building construction and occupancy, accommodations for the disabled, occupational safety and health, and disposal of medical waste and hazardous waste.⁴ The treatment room must accommodate the dentist and auxiliaries, the patient, the anesthesia care provider, the dental equipment, and all necessary anesthesia delivery equipment along with appropriate monitors and emergency equipment. Expeditious access to the patient, anesthesia machine (if present), and monitoring equipment should be available at all times.

It is beyond the scope of this document to dictate equipment necessary for the provision of deep sedation/general anesthesia, but equipment must be appropriate for the technique used and consistent with the guidelines for anesthesia providers, in accordance with governmental rules and regulations. Because laws and codes vary from state to state, *Guidelines for Monitoring and Management of Pediatric Patients Before, During, and After Sedation for Diagnostic and Therapeutic Procedures: Update 2016*⁴ should be followed as the minimum requirements.

For deep sedation/general anesthesia, there must be continuous monitoring of the patient's level of consciousness and responsiveness, heart rate, blood pressure, respiratory rate, expired carbon dioxide (**CO**₂) values, and oxygen saturation.⁴ When adequacy of ventilation is difficult to observe using capnography, use of an amplified, audible precordial stethoscope (e.g., Bluetooth technology) is encouraged.⁴ In addition, an electrocardiographic monitor and a defibrillator capable of delivering an attenuated pediatric dose are required for deep sedation/general anesthesia.⁴ Emergency equipment must be readily accessible and should include Yankauer suction, drugs necessary for rescue and resuscitation (including 100 percent oxygen capable of being delivered by positive pressure at appropriate flow rates for up to one hour), and age-/size-appropriate equipment to resuscitate and rescue a non-breathing and/or unconscious pediatric dental patient and provide continuous support while the patient is being transported to a medical facility.^{4,5} The licensed practitioners are responsible for ensuring that medications, equipment, and protocols are available to treat malignant hyperthermia when triggering agents are used.¹¹ Recovery facilities must be available and suitably equipped. Backup power sufficient to ensure patient safety should be available in case of emergency power outage.⁴

Documentation

Prior to delivery of deep sedation/general anesthesia, patient safety requires that appropriate documentation shall address rationale for sedation/general anesthesia, anesthesia and procedural informed consent, instructions to parent, dietary precautions, preoperative health evaluation, and any prescriptions along with the instructions given for their use.⁴ Because laws and codes vary from state to state, *Guidelines on Monitoring and Management of Pediatric Patients Before, During, and After Sedation for Diagnostic and Therapeutic Procedures: Update 2016*⁴ should be followed as minimum requirements for a time-based anesthesia record.

- Vital signs: Pulse and respiratory rates, blood pressure, heart rhythm, oxygen saturation, and expired CO₂ must be continuously monitored and recorded on a time-based record throughout the procedure, initially every five minutes and then, as the patient awakens, at 10-15 minute intervals until the patient has met documented discharge criteria.⁴
- Drugs: Name, dose, route, site, time of administration, and patient effects (e.g., level of consciousness, patient responsiveness) of all drugs, including local anesthesia, must be documented.⁴ When anesthetic gases are administered, inspired concentration and duration of inhalation agents and oxygen shall be documented.⁴
- Recovery: The condition of the patient, that discharge criteria have been met, time of discharge, and into whose care the discharge occurred must be documented. Requiring the signature of the responsible adult to whom the child has been discharged, verifying that he/she has received and understands the post-operative instructions, is encouraged.⁴

Various business/legal arrangements may exist between the treating dentist and the anesthesia provider. Regardless, because services were provided in the dental facility, the dental staff must maintain all patient records, including time-based anesthesia records, so that they may be readily available for emergency or other needs. The dentist must assure that the anesthesia provider also maintains patient records and that they are readily available.

Risk management and quality assurance

Dentists who utilize office-based anesthesia care providers must take all necessary measures to minimize risk to patients. The dentist must be familiar with the ASA physical status classification.¹² Knowledge, preparation, and communication between professionals are essential. Prior to subjecting a patient to deep sedation/general anesthesia, the patient must undergo a preoperative health evaluation by an appropriate and currently licensed medical or anesthesia provider.^{4,6} High-risk patients should be treated in a facility properly equipped and staffed to provide for their care.^{4,6} The dentist and anesthesia care provider must communicate during treatment to share concerns about the airway or other details of patient safety. Furthermore, they must work together to develop and document mechanisms of quality assurance.

Untoward and unexpected outcomes must be documented and reviewed to monitor the quality of services provided. This will decrease risk, allow for open and frank discussions, document risk analysis and intervention, and improve the quality of care for the pediatric dental patient.^{4,5}

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Table 1. CONSIDERATIONS IN FREQUENCY OF CONDUCTING EMERGENCY EXERCISES ⁹	
Changesinplans	Changes in the emergency response plan need to be disseminated and practiced.
Changesinpersonnel	New staff members need training in their emergency response roles. Emergency roles left by former staff members need to be filled.
Changes in property	Infrastructure changes can affect how the plan is implemented. New equipment may require training for their use.
Foreseen problems	Protocols for newly identified problems must be established, practiced and implemented.

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- 1 [Best Practices]
- 2 Classification of Periodontal Diseases in Infants, Children,
- Adolescents, and Individuals with Special Health Care Needs
- 4
- 5 Review Council
- 6 Council on Clinical Affairs
- 7 Adopted
- 8 2019
- 9
- 10 Keywords: classification, periodontal health, gingivitis, periodontitis, plaque.
- 11
- 12 Purpose

The American Academy of Pediatric Dentistry (AAPD) recognizes that although the prevalence
of destructive forms of periodontal disease is low among children and adolescents, this

15 population can develop several forms of periodontal diseases and conditions most frequently

associated with an underlying systemic or immunologic disorder.¹⁻⁴ In addition, current and early

17 studies show that gingivitis occurs in half of the population by age of 4 or 5 years and peaks

18 nearly to 100% at puberty.³ The prevalence of gingivitis can be similar to or greater than dental

19 caries during childhood.¹ Nevertheless, when compared to dental caries, gingivitis in children

- 20 has received much less attention in understanding the long-term impact that chronic
- 21 inflammation of the periodontal tissues in childhood may have on overall health of the

22 periodontium throughout life.¹ Therefore, it is critical that pediatric dental patients receive a

23 periodontal assessment as part of their routine dental visits. Early diagnosis ensures the greatest

24 opportunity for successful treatment, primarily by reducing etiological factors, establishing

appropriate therapeutic measures, and developing an effective periodic maintenance protocol.²

26

In 2017, the American Academy of Periodontology and the European Federation of
 Periodontology co-sponsored the World Workshop on the Classification of Periodontal and Peri implant Diseases and Conditions. The objective of the workshop was to update the previous

30 disease classification established at the 1999 International Workshop for Classification of

31 Periodontal Diseases and Conditions.⁵ One of the major highlights included the recategorization

32 of three forms of periodontitis, the development of a multidimensional staging and grading

- 33 system for periodontitis, and the new classification for peri-implant diseases and conditions.⁶
- 34

35 The intent of this best practices document is to present an abbreviated overview of the new classification of periodontal and peri-implant diseases and conditions, including gingivitis. 36 In addition, this document aims to emphasize the key role dentists have in diagnosing, treating 37 and/or referring pediatric patients and those medically compromised or with special health care 38 needs affected by periodontal problems. A comprehensive review of the 2017 World Workshop 39 on the Classification of Periodontal and Peri-implant Diseases and Conditions including the 40 rationale, criteria, and implementation of the new classifications, is available at the Journal of 41 Periodontology in June 2018, (Table 1).⁶⁻²⁸ 42

43

44 Methods

This document presents an abbreviated overview of the new classification of periodontal and 45 peri-implant diseases and conditions.⁶⁻²⁸ In addition to reviewing the proceeding papers from the 46 2017 World Workshop, an electronic search was conducted using PubMed® using the terms: 47 health, periodontal health, gingival disease and children and adolescents, periodontal disease and 48 children and adolescents, gingivitis, periodontitis, gingival disease and prevalence, periodontal 49 disease and prevalence, gingivitis and prevalence, periodontitis and prevalence, dental plaque, 50 drug-influenced gingival enlargements/overgrowth/hyperplasia, necrotizing periodontitis, 51 52 systemic disease, vitamin C, scurvy, puberty, pregnancy, menstrual cycle, oral contraceptives, 53 hyperglycemia, leukemia, malnutrition, smoking; fields: all; limits: within the last 10 years, humans, English, and clinical trials. From this search, 158 articles matched these criteria and 54 were evaluated by title and/or abstract. Information from 61 papers for review was chosen from 55 this list and from references within selected articles. When data did not appear sufficient or were 56 57 inconclusive, recommendations were based upon expert and/or consensus opinion by experienced researchers and clinicians. 58

59

60 Background

61 Periodontal health, gingival diseases and conditions

62 *Periodontal health*

The World Health Organization (WHO) defines health as "a state of complete physical, 63 mental and social well-being and not merely the absence of disease or infirmity".²⁹ Following 64 this framework, periodontal health is defined as the absence of clinical inflammation associated 65 with gingivitis, periodontitis, or any other periodontal conditions, and may include patients who 66 have had a history of successfully treated gingivitis or periodontitis, or other periodontal 67 conditions, and who have been and are able to maintain their dentition without signs of clinical 68 gingival inflammation.¹¹ According to the WHO health framework,²⁹ the absence of 69 inflammatory periodontal disease allows an individual to function normally and avoid the 70 consequences (mental or physical) associated to present or past disease.¹¹ 71

72

Assessing periodontal health is important to establish a common reference point for 73 diagnosing disease and determining therapy outcomes by practitioners.^{11,21} Four levels of 74 periodontal health have been proposed, depending on whether (1) the periodontium (attachment 75 and bone level) is structurally and clinically sound or reduced, (2) the ability to control local and 76 systemic modifying factors, as well as (3) the relative treatment outcomes. These levels are: (1) 77 pristine periodontal health, characterized by total absence of clinical inflammation, and 78 physiological immune surveillance on a periodontium with normal support; (2) *clinical* 79 *periodontal health*, characterized by an absence or minimal levels of clinical inflammation in a 80 periodontium with normal support; (3) periodontal disease stability, characterized as a state in 81 which the periodontitis has been successfully treated and clinical signs of the disease do not 82 83 appear to worsen in extent or severity despite the presence of a reduced periodontium; and (4) periodontal disease remission/control, characterized as a period in the course of disease when 84 symptoms become less severe but may not be fully resolved with a reduced periodontium (Table 85 2).^{6, 21} It should be noted that "pristine periodontal health" characterized by no attachment loss, 86 87 no bleeding on probing (**BoP**), no sulcular probing >3 mm in the permanent dentition and no redness, clinical swelling/edema or pus is a rare entity, especially among adults.²¹ Therefore, 88

- minimal levels of clinical inflammation observed in "clinical periodontal health" is compatiblewith a patient classified as periodontally healthy.
- 91

92 Monitoring gingival health or inflammation is best documented by the parameter of BoP 93 since it is considered the primary parameter to set thresholds for gingivitis, and the most reliable for monitoring patients longitudinally in clinical practice.^{6,21} Clinicians are encouraged to start 94 probing regularly when the first permanent molars are fully erupted and the child is able to 95 cooperate for this procedure in order to establish a baseline, detect early signs of periodontal 96 97 disease and prevent its progression. . Probing prior to the eruption of the first permanent molars is encouraged in the presence or suspicion of any clinical and/or radiographic signs of 98 periodontal disease. While probing, clinicians should rule out the presence of pseudo pockets 99 100 associated, for example, with tooth exfoliation or partially erupted teeth. For patients with special health care needs receiving dental treatment under sedation and/or general anesthesia, clinicians 101 are encouraged to take this opportunity and perform the periodontal probing. The probing force 102 should not exceed 0.25 Newton (light probing) in order to rule out the confounding issue of BoP 103 induced by too much pressure, as well as unnecessary bleeding resulting from trauma.²¹ When 104 probing positioning and pressure into the sulcus/pocket are performed correctly, the patient 105 106 should not feel discomfort. With regards to periodontal probing depth (PPD), there is strong 107 evidence that deep pockets are not necessarily consistent with disease. Deep pockets may remain stable and uninflamed, especially in cases where patients receive long term careful supportive 108 periodontal care, and are referred to as "healthy pockets". PPD or probing attachment levels 109 alone should not be used as evidence of gingival health or disease; however, be considered in 110 conjunction with other important clinical parameters such as BoP, as well as modifying and 111 112 predisposing factors. Radiographic assessment is a critical component of clinical assessment of the periodontal tissues. Radiographically, a normal, anatomically intact periodontium would 113 present an intact lamina dura, no evidence of bone loss in furcation areas, and a 2 mm distance, 114 on average, from the most coronal portion of the alveolar bone crest to the cementoenamel 115 116 junction varying between 1.0 and 3.0 mm. While analyzing radiographic findings in children, it is important that clinicians do not focus only on diagnosing interproximal carious lesions, but 117

- also evaluate the periodontal status, especially as the child grows older. Tooth mobility is not
- recommended to be used as a clinical parameter of either periodontal health or disease status.²¹
 120

Important main differences between periodontal disease stability and periodontal disease 121 122 remission/control are the ability to control for any modifying factors and the therapeutic 123 response. Stability is characterized by minimal inflammation (< 10% in BoP sites), optimal 124 therapeutic response (no probing depths > 4mm), lack of progressive periodontal destruction, while controlling for risk factors. Remission/control is characterized by a significant decrease in 125 126 inflammation, some improvement in other clinical parameters, and stabilization of disease 127 progression. Stability is the major treatment goal for periodontitis; however, remission/control may be the more realistically achievable therapeutic goal when it is not possible to fully control 128 for modifying factors.^{11,19,22,28} 129

130

There are three major determinants of clinical periodontal health. These include (1) 131 microbiological determinants (e.g., supragingival plaque and subgingival biofilm compositions), 132 (2) host determinants (local predisposing factors: e.g., periodontal pockets, dental restorations, 133 root anatomy, tooth position and crowding; systemic modifying factors: e.g., host immune 134 function, systemic health, genetics), and (3) environment determinants (e.g., smoking, 135 136 medications, stress, nutrition). In order to attain or maintain clinical periodontal health, clinicians should not underestimate predisposing and modifying factors for each patient, and recognize 137 when these factors can be fully controlled or not. Predisposing factors are any agent or condition 138 that contributes to the accumulation of dental plaque (e.g., tooth anatomy, tooth position, 139 140 restorations), while modifying factors are any agent or condition that alters the way in which an individual responds to subgingival plaque accumulation (e.g., smoking, systemic conditions, 141 medications). Many factors are determined controllable (e.g., removal of overhangs, smoking 142 cessation, good diabetes control) while others are not (e.g., genetics, immune status, use of 143 critical medications).²¹ 144

145

146 *Gingival health*

279

CCA.s. BP_ClassificationPeriodontalDiseases

- Gingival health is usually associated with an inflammatory infiltrate and host response in
 relatively stable equilibrium.²¹ Gingival health in a patient with intact periodontium is diagnosed
 by (1) no probing attachment loss, (2) no radiographic bone loss, (3) ≤3mm of PPD, and (4)
 <10% BoP.¹¹Gingival health can be restored following treatment of gingivitis and periodontitis.
 A patient with a current gingival health status who has a history of successful treated and stable
 periodontitis remains at an increased risk of recurrent periodontitis; therefore, the patient should
 be monitored closely to ensure optimal disease management.
- 154

The features of clinical gingival health are the same on an intact periodontium followed or not by treatment of gingivitis, that is, absence of BoP, erythema and edema, patient symptoms, and attachment and bone loss (physiological bone levels range from 1.0 to 3.0 mm apical to the cemento-enamel junction). The same gingival health features are also observed on a reduced periodontium following successful treatment of periodontitis.

160

161 *Gingival diseases and conditions*

Gingivitis is a reversible disease characterized by an inflammation of the gingiva that does not 162 result in clinical attachment loss (CAL).³⁰ Gingivitis of varying severity is highly prevalent 163 among children and adolescents^{11,21} and necessary prerequisite for the development of 164 periodontitis and progressive connective tissue attachment and bone loss.^{6,22,28} Controlling 165 gingival inflammation is considered the primary preventive strategy for periodontitis, as well as 166 the secondary preventive strategy for recurrence of periodontitis. Even though there is a 167 predilection of attachment loss to occur at inflamed sites of the gingiva, not all affected areas are 168 169 destined to progress to periodontitis. This is because the inter-relationship between health, 170 gingivitis and periodontitis is highly dependent on the host's susceptibility and immuneinflammatory response. Nevertheless, clinicians must understand their crucial role in ongoing 171 management of gingivitis for their patients of all ages with and/or without a history of 172 periodontal disease. There are broadly two categories of gingival disease and conditions: dental 173 174 plaque biofilm-induced gingivitis and non-dental plaque-induced gingival disease.

175

176 Dental plaque biofilm-induced gingivitis

During the 2017 World Workshop on the Classification of Periodontal and Peri-implant Diseases and Conditions, revisions of the 1999 classification system⁵ for dental plaque-induced gingival diseases included four components: (1) description of the extent and severity of the gingival inflammation; (2) description of the extent and severity of gingival enlargements; (3) a reduction in gingival disease taxonomy; and (4) discussion of whether mild localized gingivitis should be considered a disease or variant of health.²² These four components are addressed in this review.

Dental plaque biofilm-induced gingivitis is usually regarded as a localized inflammation 184 185 initiated by microbial biofilm accumulation on teeth and considered one of the most common human inflammatory diseases (Table 2).^{6,19} When dental plaque is not removed, gingivitis may 186 initiate as a result of loss of symbiosis between the biofilm and the host's immune-inflammatory 187 response. The common features of plaque-induced gingivitis include (1) clinical signs and 188 symptoms of inflammation confined to the free and attached gingiva that do not extend to the 189 periodontal attachment (cementum, periodontal ligament and alveolar bone); (2) reversibility of 190 the inflammation achieved by biofilm removal at and apical to the gingiva margin; (3) presence 191 192 of a high bacterial plaque burden needed to initiate the inflammation; and (4) stable attachment levels on a periodontium, which may or may not have experienced a loss of attachment or 193 alveolar bone (Table 3).^{11,22,28} The diagnostic criteria for gingivitis is based on clinical features. 194 195 Radiographs and probing attachment level analysis should not be used to diagnose gingivitis since they usually do not indicate loss of supporting structures. Clinical signs of *inflammation* 196 include erythema, edema, pain (soreness), heat, and loss of function. Clinical signs of gingivitis 197 include swelling (loss of knife-edged gingival margin and blunting of papillae), redness, bleeding 198 199 and discomfort on gentle probing. Patient symptoms may include bleeding gums, metallic/altered taste, pain/soreness, halitosis, difficulty eating, appearance of swollen red gums, and reduced 200 oral health-related quality of life.¹¹Although there are no objective clinical criteria for defining 201 gingivitis severity, the extent of gingivitis referred as "mild, moderate, and severe" can be used 202 as a patient communication tool. The definitions of mild, moderate, and severe gingivitis 203 204 continue to be a matter of professional opinion. Practitioners may define gingivitis as percentages of BoP sites (e.g. mild = < 10%, moderate = 10%-30\%, severe = >30% sites) or 205 based on grading (e.g. grade 1 to 5 in 20% quintiles for % sites BoP).¹⁰ The gingival index 206

207described by Löe³¹ can also be used to describe the intensity of gingival inflammation as *mild*208(area with a minor change in color and little change in the texture of the tissue); *moderate* (area209with glazing, redness, edema, enlargement, and bleeding upon probing); and *severe* (area of210overt redness and edema with a tendency toward bleeding when touched rather than probed).211Lastly, the extent or the number of gingival sites exhibiting gingival inflammation can be212described as either *localized* when < 30% of the teeth are affected or *generalized* when ≥30% of213the teeth are affected.²²

214

As mentioned above, one revision from the 1999 classification system⁵ was the proposal to introduce the term "incipient gingivitis"..."where, by definition, only a few sites are affected by mild inflammation, expressed as mild redness and/or a delayed and broken line of bleeding rather than edema or an immediate unbroken line of bleeding on probing. Incipient gingivitis may be regarded as a condition that is part of a spectrum of "clinical health," but may rapidly become localized gingivitis if untreated. "²²

221

222 The severity, extent and progression of plaque-induced gingivitis at specific sites or at the entire mouth varies between individuals and can be influenced by local (predisposing factors) 223 and systemic factors (modifying factors). Local oral factors that exacerbate plaque-induced 224 225 gingivitis are those that can influence the initiation or progression of gingival inflammation by facilitating accumulation of bacterial plaque at a specific site, inhibiting daily mechanical plaque 226 removal, and/or creating a biological niche that encourages increased plaque accumulation. 227 Examples of plaque-induced gingivitis exacerbated by plaque biofilm retention are prominent 228 subgingival restoration margins and certain tooth anatomies that contribute with plaque 229 230 accumulation increasing the risk for gingivitis and, consequently, compromising the gingival health. Oral dryness is a clinical condition frequently associated with symptoms of xerostomia, 231 which in turn is a symptom caused by a decrease in the salivary flow (hyposalivation). 232 Hyposalivation interferes with plaque removal increasing the risk of caries, halitosis, and 233 234 gingival inflammation among other oral conditions. Xerostomia may occur as a side effect of medications such as antidepressants, antihistamines, decongestants, and antihypertensive 235

- medications. In addition, health diseases/conditions such as Sjögren's syndrome, anxiety, poorly
 controlled diabetes may cause xerostomia due to hyposalivation.^{11,22}
- 238

Systemic risk factors (modifying factors) can modify the host immune inflammatory
response in the presence of dental plaque biofilm resulting in exaggerated inflammatory
response. Examples of systemic conditions include: (1) sex steroid hormones (e.g. puberty,
pregnancy, menstrual cycle, and oral contraceptives); (2) hyperglycemia; (3) leukemia; (4)
malnutrition; and (5) smoking.^{11,22}

244

Elevations in sex steroid hormones, especially, during puberty and pregnancy may 245 modify the gingival inflammatory response and result in an exaggerated gingival inflammation in 246 the presence of even relatively small amounts of plaque. Other factors that predispose to 247 gingivitis in both male and female adolescents to consider are dental caries, mouth breathing, 248 crowding and eruption of teeth. As for the use of oral contraceptives, exaggerated gingival 249 inflammatory responses to plaque is not reported in current oral contraceptive formulations with 250 lower dosages as previously observed with first generation high-dose oral contraceptives.³²⁻³⁴ 251 Although modest gingival inflammation changes have been reported during ovulation,³⁵⁻³⁷ most 252 women with gingival inflammation associated with menstrual cycles will present with non-253 detectable clinical signs of the condition.³⁸⁻⁴⁰ 254

255

Hyperglycemia, hematologic malignancies (e.g. leukemia), and nutritional deficiencies 256 are also significant systemic conditions that can negatively affect the gingival tissues. Increased 257 incidence of chronic gingivitis and risk of periodontitis among children with poorly controlled 258 type 1 diabetes mellitus have been reported.⁴¹⁻⁴³ The severity of gingival inflammation may be 259 more associated with the level of glycemic control rather than the quality of plaque control.³⁶⁻⁴⁰ 260 Hyperglycemia can alter the immune system, have a negative direct effect on periodontal cells 261 and neutrophil activity, as well as an indirect adverse effect by stimulating immune system cells 262 to release inflammatory cytokines.^{44,45} Early diagnosis of periodontal problems among children 263 264 and adolescents with poorly controlled diabetes through periodic periodontal screenings, as well 265 as prevention of periodontal diseases among this population is of fundamental importance. It is

also worth mentioning that in addition to gingivitis and periodontitis, xerostomia and candida 266 infections are also associated with diabetes.⁴⁵ Certain hematologic malignancies such as 267 leukemia are associated with signs of excess gingival inflammation inconsistent with levels of 268 dental plaque biofilm accumulation. Oral manifestations include gingival enlargement/bleeding, 269 270 petechia, oral ulcerations/infections, and cervical lymphadenopathy. Signs of gingival inflammation include swollen, glazed, and spongy tissues that are red to deep purple in 271 appearance.^{11,22,46,47} These oral manifestations may be either the result of direct gingiva 272 infiltration of leukemic cells or thrombocytopenia and/or clotting-factor deficiencies. Both 273 274 gingival bleeding and hyperplasia have been reported as initial oral signs and symptoms of patients with acute and chronic leukemias.^{22,46,47} Through periodic clinical exams, dentists have 275 an opportunity for early diagnosis of such malignant diseases, as well as timely referral and, 276 subsequently, increased chances for improvement of patient treatment outcomes. 277

278

The literature lacks information regarding the exact role of nutrition in the initiation 279 and/or progression of periodontal diseases. However, the role of vitamin C in supporting 280 periodontal tissues due to its essential function in collagen synthesis is well-documented.^{10,19} 281 Vitamin C deficiency, or scurvy, compromises antioxidant micronutrient defenses to oxidative 282 stress and collagen synthesis leading to weakened capillary blood vessels, consequently 283 increasing the predisposition to gingival bleeding.⁴⁸ Nevertheless, gingival inflammation due to 284 Vitamin C deficiency may be difficult to detect clinically and indistinguishable from plaque-285 induced gingivitis.²² Scurvy may occur in certain populations of pediatric interest such as infants 286 and children from low socioeconomic families.²² 287

288

One major change in the 2017 classification of dental plaque–induced gingival diseases was to simplify the system for the clinician and condense the catalog to include only conditions affecting the gingiva that could be clinically identified. Therefore, terms previously used such as "menstrual cycle–associated gingivitis," "oral contraceptive–associated gingivitis," and "ascorbic acid–associated gingivitis" were eliminated from the classification system because the clinical signs of these conditions were not clinically evident to the dentist.¹¹

295

Smoking is one of the major lifestyle and behavioral risk factors for periodontitis, mostly 296 attributed to alterations in the microflora and/or host response.^{11,22} Increased pocket depth 297 measurements, attachment loss and alveolar bone loss are more prevalent in smokers than non-298 smokers.⁴⁹ Tobacco use is no longer classified as a habit, but as a dependence to nicotine and a 299 chronic relapsing medical disorder.⁵⁰ Smoking and smokeless tobacco use almost always are 300 initiated and established in adolescence.⁵¹⁻⁵⁷ The most common tobacco products used by middle 301 4school and high school students are reported to be e-cigarettes, cigarettes, cigars, smokeless 302 tobacco, hookahs, pipe tobacco, and bidis (unfiltered cigarettes from India).⁵² However, the 303 exposure to cannabis (marijuana) among children and adolescents has increased in the United 304 States due to its legalization in many states.⁵⁵ Frequent cannabis use has been associated with 305 deeper probing depths, more CAL, and increased risk of severe periodontitis.⁵⁵ Periodontitis, 306 visible plaque, and gingival bleeding has also been reported among crack cocaine users.⁵⁶ 307 Clinical signs associated with smokeless tobacco may include increased gingival recession and 308 attachment loss, particularly at the sites adjacent to mucosal lesion associated with the habit.⁵⁵ 309 Health professionals who treat adolescents and young adults should be aware of the signs of 310 tobacco use, and be able to provide counseling (or referral to an appropriate provider) regarding 311 the serious health consequences of tobacco and drug use, as well as use brief interventions for 312 encouragement, support, and positive reinforcement for cessation when the habit is identified. 313

314

Drug-influenced gingival enlargements occur as a side effect in patients treated with 315 anticonvulsant drugs (e.g., phenytoin and sodium valproate), certain calcium channel-blocking 316 drugs (e.g., nifedipine, verapamil, diltiazem, amlodipine, felodipine), immune-regulating drugs 317 (e.g., cyclosporine), and high-dose oral contraceptives.^{11,57} For drug-influenced gingival 318 conditions to occur, the presence of plaque bacteria is needed to produce a gingival response. 319 The onset of this condition may occur within 3 months of the drug use,¹¹ but not all individuals 320 taking these medications are susceptible and will develop gingival overgrowth. Reports show 321 that approximately half of the people who take phenytoin, nifedipine, or cyclosporin are affected 322 with this condition.⁵⁷ A major consideration during the 2017 workshop was to select an easy and 323 324 appropriate clinical assessment to define the extent and severity of the drug-influenced 325 overgrowth. The extent of gingival enlargements were defined as either *localized* (enlargement

limited to the gingiva in relation to a single tooth or group of teeth) or *generalized* (enlargement involves the gingiva throughout the mouth).²² *Mild* gingival enlargement involves enlargement of the gingival papilla; *moderate* gingival enlargement involves enlargement of the gingival papilla and marginal gingiva, and *severe* gingival enlargement involves enlargement of the gingival papilla, gingival margin, and attached gingiva.²² Drug-influenced gingival enlargement is not associated with attachment loss or tooth mortality.

332

333 Non-dental plaque-induced gingival diseases

The gingiva and oral tissues may demonstrate a variety of non-dental plaque-induced gingival 334 lesions that are not caused by plaque and usually do not resolve after plaque removal (Table 2).⁶ 335 However, the severity of the clinical manifestations of these lesions are often dependent upon 336 plaque accumulation and subsequent gingival inflammation. These lesions may often be 337 manifestations of a systemic condition or medical disorder. They may also represent pathologic 338 339 changes confined to the gingiva. Because oral health and systemic health are strongly interrelated, it is important that dentists and other health care providers collaborate to adequately 340 diagnose, educate the patient about their condition, treatment plan, treat, or refer to a specialist 341 for treatment. The current classification of non-dental plaque-induced gingival conditions is 342 343 based on the etiology of the lesions. These include: genetic/developmental disorders (e.g. 344 hereditary gingival fibromatosis); specific infections of bacterial (e.g. necrotizing periodontal 345 diseases, Streptococcal gingivitis), viral (e.g. hand-foot-and-mouth disease, primary herpetic gingivostomatitis) and fungal (e.g. candidosis) origins; inflammatory and immune conditions and 346 347 lesions (e.g. hypersensitivity reactions, autoimmune disease of skin and mucous membranes); reactive processes (e.g. epulides); premalignant neoplasms (e.g. leukoplakia); malignant 348 349 neoplasms (e.g. leukemia, lymphoma); traumatic lesions (e.g. physical, chemical and thermal 350 insults); endocrine, nutritional and metabolic diseases (e.g. vitamin deficiencies); and gingival 351 pigmentation (e.g. amalgam tattoo). The major difference between the 1999 and 2017 classifications is the development of a more comprehensive nomenclature of non-plaque induced 352 gingival diseases and conditions based on the primary etiology, as well as the inclusion of the 353 International Statistical Classification of Diseases and Related Health Problems (ICD)-10 354 355 diagnostic codes (e.g. ICD – 10 code for primary herpetic gingivostomatitis is B00.2).^{6,11,19}

Several of these conditions may occur in pediatric patients, as well as in those with special health care needs; therefore, they are of great interest to pediatric dentists. For a comprehensive review on this topic, the reader is encouraged to review the position paper on non-dental plaque-induced gingival diseases by Holmstrup et al¹⁹ and the workshop consensus report by Chapple et al.¹¹

360

361 Classification of periodontal diseases

362 The new classification of periodontal disease proposed in the 2017 workshop defines three

distinct forms: (1) *periodontitis* (single category grouping the two forms of the disease formerly

recognized as "aggressive" or "chronic"); (2) necrotizing periodontitis; and (3) periodontitis as a

365 *manifestation of systemic conditions*. The new periodontitis classification was further

366 characterized based on a multi-dimensional staging and grading framework system. The former

indicates the disease severity and complex management, while the latter estimates the rate and

368 likelihood of the disease progression and/or response to standard periodontal therapy taking into

369 consideration the patient's biological features. 6,24,26 An individual case of periodontitis should be

further defined using a simple matrix that describes the stage and grade of the disease²⁴ as seen

in Table 4.

372

373 Periodontitis

Currently, there is insufficient evidence to support the notion that chronic and aggressive 374 periodontitis are two pathophysiologically distinct diseases. Due to concerns from clinicians. 375 researchers, educators, and epidemiologists regarding their ability to properly distinguish 376 between chronic and aggressive periodontitis, the 2017 World Workshop members proposed 377 grouping these two previously forms of periodontitis into a single category simply referred to as 378 *periodontitis*.^{24,27} The clinical entity previously referred to as "aggressive periodontitis" due to 379 its rapid rate of progression is now categorized as "Grade C" periodontitis and represents the 380 381 extreme end of a continuum of disease rates.

382

Periodontitis is a multifactorial, microbially-associated, host-mediated inflammatory
 disease characterized by progressive destruction of the periodontal attachment apparatus. Loss of
 periodontal tissue support is the primary feature of periodontitis, which is detected as clinical

386	attachment loss (CAL) by circumferential assessment of erupted teeth with a standardized
387	periodontal probe with reference to the cemento-enamel junction. Clinically, a patient is
388	characterized as a "periodontitis case" if: (1) interdental CAL is detectable at ≥ 2 non-adjacent
389	teeth; or (2) buccal or oral CAL \geq 3 mm with pocketing $>$ 3 mm is detectable at \geq 2 teeth.
390	Furthermore, the CAL cannot be attributed to non-periodontal causes such as: (1) gingival
391	recession of traumatic origin; (2) dental caries extending in the cervical area of the tooth; (3) the
392	presence of CAL on the distal aspect of a second molar and associated with malposition or
393	extraction of a third molar; (4) an endodontic lesion draining through the marginal periodontium;
394	and (5) the occurrence of a vertical root fracture. ^{24,27}
395	
396	In the context of the 2017 world workshop, three clearly different forms of periodontitis
397	have been identified based on pathophysiology: (1) necrotizing periodontitis; (2) periodontitis as
398	a direct manifestation of systemic diseases; and (3) periodontitis. Differential diagnosis is based
399	on the history and the specific signs and symptoms of necrotizing periodontitis and the presence
400	or absence of an uncommon systemic disease that definitively modify the host immune
401	response. ^{6,24,27}
402	
403	Evidence supports <i>necrotizing periodontitis</i> as a separate disease entity based on (1)
404	distinct pathophysiology characterized by prominent bacterial invasion and ulceration of
405	epithelium; (2) rapid and full thickness destruction of the marginal soft tissue resulting in
400	here derived in the set of the se

characteristic soft and hard tissue defects; (3) obvious symptoms; and (4) faster resolution in 406 response to specific antimicrobial treatment.²⁷ This painful and infectious condition should be 407 diagnosed primarily based on its typical clinical features, which includes necrosis and ulceration 408 in the interdental papilla, gingival bleeding, pseudomembrane formation, and halitosis.^{18,24} In 409 severe cases, bone sequestrum may also occur.⁵⁸ Pain and halitosis is less often observed among 410 children, while systemic conditions such as fever, adenopathy, and sialorrhea (hypersalivation) 411 are more frequently observed.^{18,59} Necrotizing periodontal diseases are strongly associated with 412 413 impairment of the host immune system. Predisposing factors include inadequate oral hygiene, chronic gingivitis, human immunodeficiency virus and acquired immune deficiency syndrome 414 415 (HIV/AIDS), malnutrition, tobacco/alcohol consumption, psychological stress and insufficient

sleep among others.²⁴ Among children, higher risk of necrotizing periodontitis are observed in 416 those with HIV/AIDS, severe malnutrition, living in extreme conditions (e.g. substandard 417 418 accommodations, limited access to potable water, poor sanitary disposal system), and exposed to severe (viral) infections (e.g. measles, chicken pox, malaria).^{18,24} Although the prevalence of 419 necrotizing periodontitis is low, it is a severe condition leading to very rapid tissue destruction 420 that can be life-threating among compromised children.¹⁸ For a more in-depth review of 421 necrotizing periodontitis, readers are directed to the positional papers by Herrera et al¹⁸ and 422 Tonetti et al,²⁷ as well as to the consensus report by Papapanou et al.²⁴ 423

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425 Systemic disease is defined as a disease that affects multiple organs and tissues or that affects the body as a whole.⁶⁰ Several systemic disorders and conditions can affect the course of 426 periodontal diseases or have a negative impact on the periodontal attachment apparatus 427 independently of dental biofilm-induced inflammation.^{7,20} For some cases, the periodontal 428 problems may be among the first signs of the disease. These disorders or conditions are grouped 429 as periodontitis as a manifestation of systemic disease and classification should be based on and 430 follow the classification of the primary systemic disease according to the respective ICD codes.⁶ 431 Moreover, they can be grouped into broad categories such as genetic disorders that affect the 432 433 host immune response (e.g. down syndrome, Papillon-Lefèvre, Histiocytosis) or affect the 434 connective tissues (e.g. Ehlers-Danlos syndrome, systemic lupus erythematosus); metabolic and endocrine disorders (e.g. hypophosphatasia, hypophosphatemic rickets); inflammatory conditions 435 (e.g. epidermolysis bullosa acquisita, inflammatory bowel disease); as well as other systemic 436 disorders (e.g. obesity, emotional stress and depression, diabetes mellitus, Langerhans Cell 437 Histiocytosis (LCH), neoplasms). For a more comprehensive review of classifications, case 438 definitions and diagnostic considerations, the reader is encouraged to read the positional paper 439 and consensus report by Albandar et al⁷ and Jepsen et al.²⁰ respectively. 440

441

The remaining clinical cases of periodontitis that do not present with the local
characteristics of necrotizing periodontitis or the systemic characteristics of a rare immune
disorder with a secondary manifestation of periodontitis should be diagnosed as *periodontitis* and

be further characterized using the staging and grading system that describes clinical
 presentation,^{6,7,18,20,24,27} (Table 4).

447

The concept of staging is adopted from the field of oncology that classifies staging of 448 449 tumors based on baseline clinical observations of size or extent and whether it has metastasized or not.⁶¹ Understanding the stage of the periodontal disease helps the clinician communicate with 450 451 the patient the current severity and extent of the disease (localized or generalized), assess the complexities of disease management, develop a prognosis, and design an individualized 452 453 treatment plan for the patient. Staging is determined by a number of variables such as probing 454 depth, CAL, amount and percentage of bone loss, presence and extent of angular bony defects and furcation involvement, tooth mobility, and tooth loss due to periodontitis.²⁷ Staging involves 455 four categories: Stage I (Initial Periodontitis), Stage II (Moderate Periodontitis), Stage III (Severe 456 Periodontitis – potential for tooth loss), and *Stage IV* (Severe Periodontitis – potential for loss of 457 dentition). Grading assesses the future risk of the periodontitis progression and anticipated 458 treatment outcomes, but also estimates the positive or negative impact that periodontitis and its 459 460 treatment have on the overall health status of the patient. Grading also allows the clinician to incorporate the individual patient risk factors (e.g. smoking, uncontrolled Type II diabetes, etc.) 461 462 into the diagnosis, which may influence the comprehensive case management. Grading includes 463 three levels: Grade A (Low Risk of Progression), Grade B (Moderate Risk of Progression), and Grade C (High Risk of Progression). Tables 4 shows the framework for staging and grading of 464 periodontitis, as well as the criteria for periodontitis stage and grade, respectively.²⁷ Table 5 465 presents the three steps to staging and grading a patient with periodontitis.²⁷ For a more 466 comprehensive description of staging and grading of periodontitis, the reader is encouraged to 467 review an outcome workshop paper by Tonetti et al²⁷ and the workshop consensus report by 468 Papapanou et al.²⁴ 469

470

471 Other conditions affecting the periodontium

472 *Peridontal abscesses and endodontic-periodontal lesions*

473 Both *periodontal abscesses* and *endodontic-periodontal lesions* share similar

474 characteristics that differentiate them from other periodontal conditions. These include pain and

discomfort requiring immediate emergency treatment, rapid onset and destruction of periodontal

tissues, negative effect on the prognosis of the affected tooth, and possible severe systemic

- 477 consequences.
- 478

479 Periodontal abscesses (PA) are defined as acute lesions characterized by localized 480 accumulation of pus within the gingival wall of the periodontal pocket, initiated by either bacterial invasion or foreign body impaction.^{18,24} The most prominent sign associated with PA is 481 the presence of an ovoid elevation in the gingiva along the lateral part of the root. Other signs 482 483 and symptoms may include pain, tenderness and swelling of the gingiva, bleeding and 484 suppuration on probing, deep periodontal pocket, bone loss observed radiographically, and increased tooth mobility.^{18,24} Facial swelling, elevated body temperature, malaise, regional 485 lymphadenopathy or increased blood leukocytes are less commonly observed.¹⁸ Etiology factors 486 such as pulp necrosis, periodontal infections, pericoronitis, trauma, surgery, or foreign body 487 impaction may explain the development of PAs. PA can develop in both periodontitis and non-488 periodontitis patients. Of interest to pediatric dentists, PA can occur in healthy sites due to 489 impaction of foreign bodies (e.g. dental floss, orthodontic elastic, popcorn hulls); harmful habits 490 (e.g. nail biting, clenching), inadequate orthodontic forces, gingival enlargement, and alterations 491 of the root surface (e.g. invaginated tooth, alterations, enamel pearls, iatrogenic perforations, 492 493 vertical root fracture, external root resorption).

494

495 Endodontic-periodontal lesions (EPL) are pathological communications between the endodontic and periodontal tissues at a given tooth that occur in either an acute or a chronic form 496 497 and are classified according to the signs and symptoms that have direct impact on their prognosis 498 and treatment (e.g. presence or absence of fractures and perforations, presence or absence of 499 periodontitis and the extent of periodontal destruction around the affected teeth). The primary signs associated with EPL are deep periodontal pockets reaching or close to the apex and/or 500 negative or altered response to pulp vitality tests. Other signs and symptoms may include 501 502 radiographic evidence of bone loss in the apical or furcation region, spontaneous pain or pain on 503 palpation and percussion, purulent exudate or suppuration, tooth mobility, sinus tract/fistula, crown and/or gingival color alterations.^{18,24} Signs observed in endo-periodontal lesions 504

associated with traumatic and/or iatrogenic factors may include root perforation,

- 506 fracture/cracking, or external root resorption, commonly associated with the presence of an
- abscess accompanied by pain. In periodontitis patients, EPL usually presents low and chronic
- 508 progression without evident symptoms. For further review on the classification,
- pathophysiology, microbiology, and histopathology of both PA and EPL, readers are directed to
- the positional paper by Herrera et al^{18} and the consensus report by Papapanou et al^{24} .
- 511

512 *Mucogingival deformities and conditions*

513 Normal mucogingival condition is defined as the absence of pathosis such as gingival recession, gingivitis, and periodontitis. Mucogingival deformities, including gingival recession, are a group 514 of conditions that affect a large number of patients that are observed more frequently in adults 515 516 and have a tendency to increase with age independent of the patient oral hygiene status. 517 Recession is defined as an apical shift of the gingival margin caused by different conditions and pathologies that is associated with CAL in any surface (buccal/lingual/interproximal) of the 518 teeth.²⁰ Although, gingival thickness has been referenced in the literature as "gingival biotype", 519 520 the 2017 World Workshop group strongly suggested the adoption of the term "periodontal phenotype", which is determined by gingival phenotype (gingival thickness, keratinized tissue 521 522 width) and bone morphotype (thickness of the buccal bone plate). Periodontal phenotype can be 523 assessed by measuring the gingival thickness through the use of a periodontal probe. Thin *phenotype* is classified when the periodontal probe shining through the gingival tissue is $\leq 1 \text{ mm}$ 524 visible after being inserted into the sulcus, and classified as *thick phenotype* when the probe is > 525 1mm visible.²⁰ The development and progression of gingival recession is not associated with 526 527 increased tooth mortality. However, this condition is often associated with patient esthetic 528 concerns, dentinal hypersensitivity and carious/non-carious cervical lesions on the exposed root surface.^{12,20} While lack of keratinized tissue is a predisposing factor for gingival recession and 529 530 inflammation, periodontal health can be maintained despite the lack of keratinized tissues in most patients with optimal home care and professional maintenance. Conversely, patients with 531 532 thin periodontal phenotypes, inadequate oral hygiene, and requiring cervical restorative and/or orthodontic treatment are at an increased risk for gingival recession.^{12,20} Monitoring specific 533 534 gingival recession sites is considered a proper approach in the absence of any pathosis. However,

- 535 mucogingival surgical interventions may be necessary in the presence of esthetic concerns,
- dentin hypersensitivity, cervical lesions, thin gingival biotypes and mucogingival deformities.
- 537

538 Traumatic occlusal forces and occlusal trauma

539 Traumatic occlusal force is defined as any occlusal force that causes an injury to the teeth and/or 540 the periodontal attachment apparatus. It may be indicated by one or more of the following: fremitus (visible tooth movement upon occlusal force), tooth mobility, thermal sensitivity, 541 excessive occlusal wear, tooth migration, discomfort/pain on chewing, fractured teeth, 542 543 radiographically widened periodontal ligament space, root resorption, and hypercementosis.²⁰ 544 Occlusal trauma is a lesion in the periodontal ligament, cementum and adjacent bone caused by traumatic occlusal forces. It may be indicated by one or more of the following: progressive tooth 545 mobility, adaptive tooth mobility (fremitus), radiographically widened periodontal ligament 546 space, tooth migration, discomfort/pain on chewing, and root resorption.²⁰ Traumatic occlusal 547 forces and occlusal trauma can be classified as: (1) primary occlusal trauma; (2) secondary 548 occlusal trauma; and (3) orthodontic forces. Primary and secondary occlusal trauma have been 549 defined as injuries resulting in tissue changes from traumatic occlusal forces, the former when 550 applied to a tooth or teeth with normal periodontal support and the latter when applied to a tooth 551 552 or teeth with reduced support.

553

554 There is either little or no evidence that traumatic occlusal forces can cause periodontal attachment loss, inflammation of the periodontal ligament, non-carious cervical lesions, 555 abfraction or gingival recession.^{14,20} Traumatic occlusal forces lead to adaptive mobility in teeth 556 557 with normal support and are not progressive, while in teeth with reduced support they lead to progressive mobility usually requiring splinting. Although, there is evidence that traumatic 558 occlusal forces may be associated periodontitis, there is no evidence that these forces can 559 accelerate the progression of periodontitis in humans.²⁰ Moreover, there is insufficient clinical 560 evidence regarding the impact that elimination of traumatic occlusal forces may have on the 561 562 response to periodontal therapies. With regards to orthodontic forces, observational studies suggest that orthodontic treatment has minimal adverse effects to the periodontal supporting 563 apparatus, especially in patients with good plaque control and healthy periodontium.^{14,20} 564

- However, non-controlled orthodontic forces can have adverse effects on the periodontium suchas pulpal disorders, root and alveolar bone resorptions.
- 567

568 Dental prostheses and tooth-related factors

569 Several conditions associated with the fabrication and presence of dental restorations and fixed 570 prostheses, placement of orthodontic appliances, as well as tooth-related factors may facilitate 571 the development of gingivitis and periodontitis, especially in individuals with poor compliance 572 with homecare plaque control and attendance to periodic maintenance visits.^{13,20}

573

Tooth anatomic factors (e.g. cervical enamel projections, enamel pearls, developmental 574 grooves); root proximity, abnormalities, and traumatic dental injuries potentially altering the 575 576 local anatomy of both hard and soft tissues; as well as tooth relationships in the dental arch and with the opposing dentition are associated with dental plaque-biofilm induced gingivitis and 577 periodontitis. Placement of restoration margins infringing within the junctional epithelium and 578 supracrestal connective tissue attachment (biological width) can also be associated with gingival 579 inflammation and, potentially, recession. Tooth-supported and/or tooth-retained restorations and 580 their design, fabrication, delivery, and materials have often been associated with plaque retention 581 582 and loss of periodontal supporting tissues. However, optimal restoration margins located within 583 the gingival sulcus do not cause gingivitis if patients are compliant with self-performed plaque control and periodic maintenance care.^{13,20} 584

585

The available evidence does not support that optimal removable and fixed dental 586 prostheses are associated with periodontitis when patients perform adequate plaque control and 587 588 attend maintenance appointments. However, there is evidence to suggest that removable dental prostheses can serve as plaque retentive factors, be associated with gingivitis/periodontitis, 589 increased mobility and gingival recession in patients with poor compliance. Moreover, there is 590 evidence to suggest that design, fabrication, delivery and materials used for fixed dental 591 592 prostheses procedures can be associated with plaque retention, gingival recession and loss of supporting periodontal tissues.^{13,20} 593

594

595 Lastly, it is important to point out that dental materials, including commonly used 596 appliances (e.g., stainless steel crowns, space maintainers, ortho appliances) may be associated 597 with hypersensitivity reactions observed clinically as a localized inflammation. If the hypersensitivity does not resolve with adequate measures of plaque control, additional treatment 598 599 may be required, including removal of material or appliance. However, it appears that adequate 600 periodontal assessment and treatment, appropriate instructions, and motivation in self-performed plaque control and compliance to periodic maintenance protocols are the most important factors 601 to limit or avoid the potential negative effects on the periodontium caused by fixed and 602 603 removable prostheses when hypersensitivity reactions are not suspected.¹³ 604 **Peri-implant diseases and conditions** 605 The 2017 World Workshop members developed a new classification for *peri-implant health*, 606 *peri-implant mucositis* and *peri-implantitis*. The case definitions were developed based on a 607 608 review of the evidence applicable for diagnostic considerations for use by clinicians for both individual case management and population studies.^{6,25} Because the majority of pediatric dentists 609 are not the ones responsible for the placement of osseointegrated dental implants, the reader is 610 encouraged to review the positional paper by Renvert et al.²⁵ and the consensus report by 611 Berglundh et al⁹ for more comprehensive information about the rationale, criteria and 612 implementation of the new classification. Nevertheless, it is important that all clinicians are able 613 to diagnose potential problems, complications, and failures associated with dental implants in 614 order to either provide proper treatment or refer the patient to a specialist. Case definitions and 615 clinical criteria of these conditions are presented below. 616

617

618 *Peri-implant health*

Clinically, peri-implant health is characterized by an absence of visual signs of inflammation
such as redness, swelling, profuse BoP, as well as an absence of further additional bone loss
following initial healing. Peri-implant health can occur around implants with normal or reduced
bone support.^{6,25}

- 623
- 624 Peri-implant mucositis

625 Peri-implant mucositis is characterized by visual signs of inflammation such as redness,

- swelling, line or drop of bleeding within 30 seconds following probing, combined with no
- additional bone loss following initial healing. There is strong evidence that peri-implant
- mucositis is caused by plaque, while very limited evidence for non-plaque induced peri-implant
- 629 mucositis. Peri-implant mucositis can be reversed with dental plaque removal measures.^{6,25}
- 630

631 Peri-implantitis

- 632 Peri-implantitis is defined as a plaque-associated pathologic condition occurring in the tissue
- around dental implants, characterized by signs of inflammation in the peri-implant mucosa,
- radiographic evidence of bone loss following initial healing, increasing probing depth as
- 635 compared to probing depth values after the implant placement, and subsequent progressive loss
- of supporting bone. In the absence of baseline radiographs, radiographic bone level ≥ 3 mm in
- 637 combination with BoP and probing depths $\geq 6mm$ is indicative of peri-implantitis. Peri-
- 638 implantitis is preceded by peri-implant mucositis.^{6,25}
- 639

640 Recommendations

- Periodontal disease in children is of great interest in pediatric dentistry and a problem that
 should not be ignored. Therefore, it is critical that pediatric dental patients receive a
 periodontal assessment as part of their routine dental visits.
- Early diagnosis of periodontal diseases ensures the greatest opportunity for successful
 treatment, primarily by reducing etiological factors, establishing appropriate therapeutic
 measures, and developing an effective periodic maintenance protocol.
- 647 3. Clinicians should become familiarized with the most current classification of periodontal
 648 diseases and conditions, including gingivitis, in order to properly diagnose patients
 649 affected by these problems.
- 4. Pediatric dentists are often the front line in diagnosing periodontal conditions and in great
 position to treat, refer, coordinate, collaborate and/or organize the patient care activities
 between two or more health care providers to ensure that the appropriate treatment is
 delivered in a timely fashion.

- 5. Monitoring gingival health or inflammation is best documented by the parameter of 654 655 bleeding on probing since it is considered the primary parameter to set thresholds for gingivitis, and the most reliable for monitoring patients longitudinally in clinical practice. 656 Clinicians are encouraged to start probing regularly when the first permanent molars are 657 658 fully erupted and the child is able to cooperate for this procedure in order to establish a 659 baseline, detect early signs of periodontal disease and prevent its progression. 660 6. Probing prior to the eruption of the first permanent molars is encouraged in the presence or suspicion of any clinical and/or radiographic signs of periodontal disease. For patients 661
- with special health care needs receiving dental treatment under sedation and/or general
 anesthesia, clinicians are encouraged to take this opportunity and perform the periodontal
 probing.
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The intent of this document was to present an abbreviated overview of the proceeding papers from the 2017 World Workshop on the Classification of Periodontal and Peri-implant Diseases and Conditions. Major highlights from the 2017 workshop included the recategorization of three forms of periodontitis, the development of a multidimensional staging and grading system for periodontitis, and the new classification for peri-implant diseases and conditions. A best practice document on periodontal disease therapies will be available in a future publication of the AAPD Reference Manual.

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recommendation until approval by the General Assembly. Circulation is limited to AAPD TABLE 1. 2017 World Workshop on the Classification of Periodontal and Peri-implant Diseases and Conditions (Adapted from Caton et al⁶) **Periodontal Diseases and Conditions** Periodontitis **Other Conditions Affecting the** Periodontal Health, Gingival Periodontium **Diseases and Conditions** Papapanou, Sanz et al. 2018 Consensus Rept Jepsen, Caton et al. Consensus Rept Jepsen, Caton et al. 2018 Consensus Rept Chapple, Mealey, et al. 2018 Rept Tonetti, Greenwell, Komman. 2018 Case Papapanou, Sanz et al. 2018 Consensus Rept Trombelli et al. 2018 Case Definitions Definitions Periodontal Health, Gingival Diseases **Necrotizing Periodontal Diseases** Systemic Diseases or Conditions Herrera et al. 2018 Affecting the Periodontal Supporting and Conditions Lang & Bartold 2018 Tissues 1. Necrotizing Gingivitis Albandar et al. 2018 1. Clinical gingival health on an intact 2. Necrotizing Periodontitis 3. Necrotizing Stomatitis periodontium 2. Clinical gingival health on a reduced periodontium Periodontal Abscesses and **Endodontic-Periodontal Lesions** a. Stable periodontitis patient Periodontitis as Manifestations of Papapanou, Sanz et al. 2018 b. Non-periodontitis patient Systemic Diseases Herrera et al. 2018 Jepsen, Caton et al. 2018 Consensus Rept./Albandar et al. 2018 Classification of these conditions should **Mucogingival Deformities** be based on the primary systemic **Gingivitis - Dental Biofilm-induced** and Conditions disease according to the International Murakami et al. 2018 Cortellini & Bissada 2018 Statistical Classification of Diseases and 1. Associated with dental biofilm Related Health Problems (ICD) codes 1. Gingival phenotype alone 2. Gingival/soft tissue recession 2. Mediated by systemic or local risk 3. Lack of gingiva factors 4. Decreased vestibular depth 3. Drug-influenced gingival 5. Aberrant frenum/muscle position Periodontitis enlargement 6. Gingival excess Fine et al. 2018/Needleman et al. 7. Abnormal color 2018/Billings et al. 2018 8. Condition of the exposed root 1. Stages: Based on Severity and surface Complexity of Management Gingival diseases - Non-Dental Stage I: Initial Periodontitis **Biofilm-induced** Stage II: Moderate Periodontitis Holmstrup et al. 2018 Stage III: Severe Periodontitis with **Traumatic Occlusal Forces** 1. Genetic/developmental disorders potential for additional tooth loss Fan & Caton 2018 2. Specific infections Stage IV: Sever Periodontitis with 3. Inflammatory and immune potential for loss of the dentition 1. Primary occlusal trauma conditions 2. Extent and distribution: localized; 2. Secondary occlusal trauma 4. Reactive processes generalized; molar-incisor 3. Orthodontic forces 5. Neoplasms distribution 6. Endocrine, nutritional & metabolic 3. Grades: Evidence or risk of rapid progression, anticipated treatment diseases **Tooth and Prosthesis-related Factors** 7. Traumatic lesions response Ercoli & Caton 2018 8. Gingival pigmentation a. Grade A: Slow rate b. Grade B: Moderate rate of 1. Localized tooth-related factors progression 2. Localized dental prostheses-related c. Grade C: Rapid rate of factors progression **Peri-Implant Diseases and Conditions** Berglundh, Armitage et al. 2018 Consensus Rept CCA s BP ClassificationPeri dontalDiseases Peri-Implant Health **Peri-Implant mucositis Peri-implantitis** Peri-Implant soft and hard tissue deficiencies Araujo & Lindhe 2018 Heitz-Mayfield & Salvi 2018 Hammerle & Tarnow 2018

Schwarz et al. 2018

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863 864 865 866	TABLE 2. Classification	of Gingival Health and Gingival Diseases (Adapted from Chapple et al ¹¹)	and Conditions
867	Periodontal Health	Gingivitis — Dental Plaque- induced	Gingival Disease — Non-dental Plaque-induced
868			· · · · · · · · · · · · · · · · · · ·
868 869 870 871 872 873 874 875 876 876 877 878 879 880 881	 Clinical health on an intact periodontium Clinical gingival health on a reduced periodontium a. Stable periodontitis patient b. Non-periodontitis patient 	 Associated with biofilm alone Mediated by systemic or local risk factors Systemic risk factors (modifying factors) Smoking Hyperglycemia Nutritional factors Pharmacological agents (prescription, non-prescription and recreational) Sex steroid hormones (puberty, menstrual cycle, pregnancy, oral 	 Genetic/developmental disorders Hereditary gingival fibromatosis Specific infections a. Bacterial origin Viral origin Viral origin Fungal origin Inflammatory and immune conditions Autoimmune diseases of skin and mucous membranes Granulomatous inflammatory lesions (orofacial granulomatosis) Reactive processes Epulides Neoplasms
882 883 884 885 886 887 888 888 889		contraceptives) - Hematological conditions b. Local risk factors	 a. Premalignancy b. Malignancy c. Endocrine, nutritional and metabolic diseases a. Vitamin deficiencies 7. Traumatic lesions a. Physical/mechanical trauma b. Chemical (toxic) burn c. Thermal insults 8. Gingival pigmentation
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TABLE 3. Diagnostic look-up table for gingival health or dental plaque-induced gingivitis in clinical practice (Adapted from Chapple et al¹¹)

Intact periodontium Gingivitis	Health	Gingivitis
Probing attachment loss	No	No
Probing pocket depths (assuming no pseudo pockets) <u>*</u>	≤3 mm	≤3 mm
Bleeding on probing	<10%	Yes (≥ 10%)
Radiological bone loss	No	No
<u>*on fully erupted teeth</u>		
Reduced periodontium Non-periodontitis patient	Health	Gingivitis
Probing attachment loss	Yes	Yes
Probing pocket depths (all sites & assuming no pseudo pockets)*	≤3 mm	≤3 mm
Bleeding on probing	<10%	Yes (≥ 10%)
Radiological bone loss	Possible	Possible
Successfully treated stable periodontitis patient	Health	Gingivitis in a patient with a history of periodontitis
Probing attachment loss	Yes	Yes
Probing pocket depths (all sites & assuming no pseudo pockets)*	\leq 4 mm (no site \geq 4 mm with BOP)	≤3 mm
Bleeding on probing	<10%	Yes (≥ 10%)
Radiological bone loss	Yes	Yes

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TABLE 4. Periodontitis Staging and Grading

Framework	-	ntitis				Diseas	e Severity and Complexity	of Management	
staging and	graaing		Stage I Initial periodo		Stage II: Moderate periodontit	tis	Stage III: Severe periodontitis with potential for additional to loss		
Evidence or risk		Grade A					1	I	
progression, and treatment respon	nse, and	Grade B			Individual Stage and Grade Assignment				
effects on system	mic health	Grade C							
Periodontiti	s stage	Stage 1	I Stage II		Stage III		Stage IV		
Severity	Interdental CAL at site of greatest los	1 to 2 m	ım	$3 \text{ to } 4 \text{ mm}$ $\geq 5 \text{ mm}$		ım	≥5 mm		
	Radiographic bone loss	Coronal (<15%)			Coronal third (<15% to 33%)		nding to mid-third of root beyond	Extending to mid-third of root and beyond	
	Tooth loss	No toot	h loss due to periodontitis		Tooth loss due to periodontitis of \leq 4 teeth		Tooth loss due to periodontitis of ≥ 5 teeth		
Complexity Local Maximum probing dep ≤4 mm Mostly horizontal bone loss		depth depth ≤5 mm Mostly horizontal bone loss al		In addition to stage II complexity: Probing depth ≥6 mm Vertical bone loss ≤3 mm Furcation involvement Class II or III Moderate ridge defect		 In addition to stage III complexity: Need for complex rehabilitation due to Masticatory dysfunction Secondary occlusal trauma (tooth mobility degree ≥2) Severe ridge defect Bite collapse, drifting, flaring Less than 20 remaining teeth (10 opposing pairs) 			
Extent and distributionAdd to stage as descriptorFor each			h stage, d	lescribe e	xtent as local	ized (<	30% of teeth involved), ge	neralized, or molar/incisor pattern	
Periodontitis grade				Grade A: Slow rate of progression			Grade B: Moderate rate of progression	Grade C: Rapid rate of progression	
Primary Criteria	Direct eviden of progression		dinal dat aphic bo CAL)		Evidence of no loss over 5 years		<2 mm over 5 years	$\geq 2 \text{ mm over 5 years}$	
	Indirect evidence of	% bone	% bone loss/age <		<0.25		0.25 to 1.0	>1.0	
	progression	Case ph	dep		eavy biofilm eposits with low vels of destruction		Destruction commensurate with biofilm deposits	Destruction exceeds expectation given biofilm deposits; specific clinical patterns suggestive of periods of rapid progression and/or early onset disease (e.g., molar/incisor pattern; lack of expected response to standard bacterial control therapies)	
Grade modifiers	Risk factors	Smokin	g Non-smoker			Smoker <10 cigarettes/day	Smoker ≥10 cigarettes/day		

		Diabetes	Normoglycemic/no diagnosis of diabetes	HbAlc <7.0% in patients with diabetes	HbAlc \geq 7.0% in patients with diabetes			
896 897 898 899	TA		to Staging and Grading a Patient with Periodontitis Adapted from Tonetti et al ²⁷)					
900 901 902			Screen: • Full mouth • Full mouth	n probing n radiographs				
903	Initial Case	ep 1 Overview to Disease	• Missing tee	eth	ically be either Stage I or Stage II			
904 905			-		typically be either Stage III or g IV			
906		L]			
907				rate periodontitis (typi linical attachment loss	bically Stage I or Stage II):			
908 909			 Rule out non-periodontitis causes of CAL (e.g., cervical restorations or caries, root fractures, CAL due to traumatic causes) 					
910 911		ep 2 ish Stage	 Determine maximum CAL or radiographic bone loss (RBL) Conform RBL patterns For moderate to severe periodontitis (typically Stage III or Stage IV): 					
912			• Confirm R	maximum CAL or R BL patterns				
913 914				A	ontitis rs (e.g., severe CAL frequency,			
915		L		<u>6</u> /	J			
916 917 [RBL (% of root length k factors (e.g., smokin	h x 100) divided by age ng, diabetes)			
917 918	Stej Establisl		Measure reAssess exp	esponse to scaling and pected rate of bone los	d root planning and plaque control ss			
919				etailed risk assessmer or medical and system	nt nic inflammatory considerations			
920		L						

1 Policy on Management of the Frenulum in Pediatric Dental

2 Patients

- 3
- 4 Originating Council
- 5 Council on Clinical Affairs
- 6 Adopted
- 7 2019
- 8
- 9 Purpose
- 10 Evidence suggests that the prevalence of frenotomy/frenectomies is increasing, with reports
- 11 indicating as much as 90 percent increase in recent years.^{1,2} American Academy of Pediatric
- 12 Dentistry (AAPD) recognizes a policy on frenula would make the information and
- 13 recommendations more accessible to dentists, physicians, and other allied health professionals in
- 14 an evidence-based format.
- 15

16 Methods

17 This policy is a review of current dental and medical literature and sources of recognized

- 18 professional expertise and stature, including both the academic and practicing health communities
- 19 related to frenula/frenotomies. In addition, literature searches of PubMed/MEDLINE and Google
- 20 Scholar databases were conducted using the terms: ankyloglossia, breast-feeding, frenotomy,
- 21 systematic review, lip-tie, super labial frenulum, maxillary lip-tie, breastfeeding cessation,
- 22 frenulum, frenum, tongue-tie, speech articulation, frenuoplasty, midline diastema, lactation,
- 23 nipple pain, Hazelbaker, IBFAT, LATCH, mandibular labial frenulum, periodontal; fields: all;
- 24 limits: within the last 15 years, English. One hundred seventeen articles matched these criteria.
- 25 Papers for review were chosen from this list and from references within selected articles. Expert
- 26 and/or consensus opinion by experienced researchers and clinicians also was considered.

27

28 Background

- 29 Frenulum attachments and their impact on oral motor function and development have become a
- 30 topic of emerging interest among the community as well as various specialties of healthcare
- 31 providers. Studies have shown differences in treatment recommendations among pediatricians,

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- 32 otolaryngologists, lactation consultants, speech pathologists, surgeons, and dental
- 33 specialists. ^{3,4,5,6,7,8,9,10} Clear indications and timing of surgical treatment remain controversial due
- 34 to lack of consensus regarding accepted anatomical and diagnostic criteria for degree of
- restriction and relative impact on growth, development, feeding, or oral motor function. ^{3,4,5,6,7,8,9,10}
- 36 Although the etiology of this condition remains unknown, there appears to be a higher
- 37 predilection in male towards anomalies of frenulum attachments, whether it is ankyloglossia or
- 38 hypertrophic/restrictive maxillary labial frenum.^{4,9,11,12,13} There are several frenulum that are
- 39 usually present in the oral cavity, most notable the maxillary labial frenulum, the mandibular
- 40 labial frenulum, and the lingual frenulum.¹⁴ Their primary function is to provide stability of the
- 41 upper lip, lower lip, and tongue, respectively.¹⁵
- 42

43 Maxillary frenulum

- 44 A prominent maxillary frenulum in infants, children, and adolescents, although a common
- 45 finding, is often a concern to the parents. The maxillary labial frenal attachment can be classified
- 46 with respect to its anatomical insertion level:
- 47 1. Mucosal (frenal fibers are attached up to the mucogingival junction).
- 48 2. Gingival (fibers are inserted within the attached gingiva).
- 49 3. Papillary (fibers are extending into the interdental papilla).
- 4. Papilla penetrating (fibers cross the alveolar process and extend up to the palatine
 papilla).¹⁴
- 52

53 The most commonly observed types are mucosal and gingival.⁹ However, it is also reported that a 54 maxillary frenulum is a dynamic structure that presents changes in position of insertion, structure, 55 and shape during growth and development.⁹ Infants have the highest prevalence of papillary 56 penetrating phenotype.⁹ In severe instances, a restrictive maxillary frenulum attachment has been 57 associated with breastfeeding difficulties among newborns.^{16,17} It has been suggested that a 58 restrictive maxillary frenulum may inhibit an airtight seal on the maternal breast through 59 "flanging" of both lips.^{12,17} With the lack of knowledge surrounding the function of the upper 60 frenulum, the ubiquity of its presence, and level of attachment in most infants, the release of the 61 maxillary frenulum based on appearance alone cannot be endorsed at this time.¹⁸ Hyperplastic 62 labial frenulum that inserts into free or marginal gingiva has been suggested to interfere with 63 proper oral hygiene measures and potentially lead to facial-cervical caries as well as initiation and

progression of gingival/periodontal disease due to interference with adequate oral hygiene.¹⁹ 64 65 There is no evidence to support this conclusion although antidotal speculation persists. When 66 treatment is considered due to higher caries risk, anticipatory guidance and other preventive 67 measures should be emphasized. However, further research is required to substantiate the cause-68 and-effect relationship.Surgical removal of maxillary midline frenulum is also related to presence 69 or prevention of midline diastema formation, prevention of post orthodontic relapse, esthetics, 70 and psychological considerations.^{7,8,9,20} Treatment options and sequence of care vary with patient 71 age and can include orthodontics, restorative dentistry, surgery, or a combination of these.²⁰ 72 Treatment is suggested when the attachment exerts a traumatic force on the gingiva causing the 73 papilla to blanch when the upper lip is pulled or if it causes a diastema wider than two 74 millimeters, which is known to rarely close spontaneously during further development.^{9.20,21} 75 When a diastema is present, the objectives for treatment involve managing both the diastema of 76 permanent teeth and its etiology.²⁰ If orthodontic treatment is indicated, the need for frenotomy 77 should be assessed and coordinated with orthodontic closure of the diastema to achieve stable results.^{20,21,22} There is general agreement between pediatric dentists and orthodontist that a 78 79 frenectomy should not be performed before the permanent canines erupt and that the operation 80 should follow orthodontic closure of the space.²³

81

82 Mandibular labial frenulum

83 A high frenulum sometimes can present on the labial aspect of the mandibular ridge. This is most 84 often seen in the permanent central incisor area and frequently occurs in individuals where the 85 vestibule is shallow.⁶ The mandibular anterior frenulum, as it is known, occasionally inserts into 86 the free or marginal gingival tissue.⁶ Movements of the lower lip cause the frenulum to pull on 87 the fibers inserting into the free marginal tissue, which creates pocket formation which in turn, 88 can lead to food and plaque accumulation.⁶ Early treatment can be considered to prevent subsequent inflammation, recession, pocket formation, and possible loss of the alveolar bone 89 90 and/or tooth.⁶ However, if factors causing gingival/periodontal inflammation are controlled, the degree of recession and need for treatment decreases.^{3,6} Again, when treatment is considered due 91 92 to higher caries risk, anticipatory guidance and other preventive measures should be emphasized. 93 94 95

96 Lingual Frenum

- 97 World Health Organization (WHO) has recommended mothers worldwide to exclusively
- 98 breastfeed infants for the child's first six months to achieve optimum growth, development and
- 99 health. Thereafter, they should be given complementary foods and continue breastfeeding up to
- 100 the age of two years or beyond. ²⁴ American Academy of Pediatrics (AAP), in 2018, reaffirmed
- 101 its recommendation of exclusive breastfeeding for about six months, followed by continued
- 102 breastfeeding as complementary foods are introduced, with continuation of breastfeeding for one
- 103 year or longer as mutually desired by mother and child. ²⁵ Maxillary and lingual frenulum have
- been associated by some practitioners as impedance to successful breastfeeding leading to
- 105 recommendations for frenotomy.
- 106

107 Ankyloglossia

- 108 Ankyloglossia is a developmental anomaly of the tongue characterized by a short, thick lingual
- 109 frenulum resulting in limitation of tongue movement (partial ankyloglossia) or by the tongue
- appearing to be fused to the floor of the mouth (total ankyloglossia).^{6,26} Studies with different
- 111 diagnostic criteria report prevalence of ankyloglossia between four and 10.7 percent of the
- 112 population.^{3,5} Several diagnostic classifications have been proposed based on anatomical and
- 113 functional criteria, but none has been universally accepted.³
- 114
- 115 Ankyloglossia has been associated with breastfeeding difficulties among neonates, limited tongue
- 116 mobility and speech difficulties, malocclusion, and gingival recession.^{3,4,5,6,7,8,9,10,21} A short
- 117 frenulum can inhibit tongue movement and create deglutition problems .^{3,27,28} During
- 118 breastfeeding, a restrictive frenulum can cause ineffective latch, inadequate milk transfer and
- 119 intake, and persistent maternal nipple pain, all of which can affect feeding
- adversely.^{3,4,5,6,7,8,9,10,11,21,26,27,28,29,30} Systematic literature review articles acknowledge the role of
- 121 frenotomy procedure when there is clear_evidence of frenal constriction in reduction in maternal
- 122 nipple pain when provided in conjunction with support of other allied healthcare
- **123** professionals.^{3,4,5,6,8,31}
- 124
- 125 Limitations in tongue mobility and speech pathology have been associated with ankyloglossia.^{3, 32,}
- ³³ Speech articulation is largely perceptual in nature, and differences in pronunciation are often
- 127 evaluated subjectively. There is very high variability in the speech assessment outcomes among

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128	individuals and specialists from different medical backgrounds. ⁴ The difficulties in articulation
129	are evident for consonants and sounds like /s/, /z/, /t/, /d/, /l/, /j/, /she/, /chi/, /the/, /dg/, and it is
130	especially difficult to roll an r ^{4,32} Speech therapy in conjunction with frenuloplasty or frenotomy
131	can be a treatment option to improve tongue mobility and speech. ^{32,33} There has been varied
132	opinion among health care professionals regarding the correlation between ankyloglossia and
133	speech disorders. Further evidence is needed to determine the benefit of surgical correction of
134	ankyloglossia and its relation to speech pathology as there are many children and individuals with
135	ankyloglossia who do not suffer from speech difficulty. ^{3, 8, 34}
136	
137	There is limited evidence to show that ankyloglossia and abnormal tongue position may affect
138	skeletal development and be associated with Class III malocclusion. ^{28, 35} A complete orthodontic
139	evaluation, diagnosis, and treatment plan are necessary prior to any surgical intervention. ³⁵
140	
141	Localized gingival recession on the lingual aspect of the mandibular incisors has been associated
142	with ankyloglossia in some cases where frenal attachment causes gingival retraction. ^{3, 6} As with
143	most periodontal conditions, elimination of plaque-induced gingival inflammation can minimize
144	gingival recession without any surgical intervention. ³ When recession continues even after oral
145	hygiene management, surgical intervention may be indicated. ^{3,6}
146	
147	Frenotomy procedure
148	Although there is limited evidence in the literature to promote the timing, indication, and type of
149	surgical intervention, frenotomy for functional limitations should be considered on an individual
150	basis. ^{3,4,28,30,33,36} When indicated, frenuloplasty (various methods to release the frenulum and
151	correct the anatomic situation) or frenectomy/frenotomy (simple cutting of the frenulum) may be
152	a successful approach to alleviate the problem. ^{3,4,9,37} Each of these procedures involves surgical
153	incision, establishing hemostasis, and wound management. ³⁸ Dressing placement or the use of
154	antibiotics is not necessary. ³⁸ Post-operative recommendations include maintaining a soft diet,
155	regular oral hygiene, and analgesics as needed. The use of electrosurgery or laser technology for
156	frenotomies has demonstrated a shorter operative working time, a better ability to control
157	bleeding, reduced intra- and post-operative pain and discomfort, fewer postoperative
158	complications (e.g., swelling, infection), no need for suture removal, and increased patient
159	acceptance ³⁹ These procedures require extensive training as well as skillful technique and patient

- 160 management.^{3,4,9,33,37,40,41,42,43} As with all surgical procedures, an informed consent should be
- 161 obtained.
- 162

163 Policy statement

- 164 Recognizing there is limited evidence; AAPD supports additional research on the causative
- association between ankyloglossia and breast feeding difficulties or speech articulation problems.
- 166 Further randomized controlled trails of high methodological quality are necessary to determine
- 167 the effects of frenotomy. With all surgical procedures an informed consent is necessary, which
- 168 includes relevant information regarding diagnosis, nature and purpose of proposed treatment,
- 169 potential benefits and risks, and professionally-recognized or evidence-based alternative
- 170 treatment–including no treatment–to recommended therapy and risk(s).⁴⁴ When treatment is
- 171 considered due to higher caries risk, anticipatory guidance and other preventive measures should
- be emphasized.
- 173

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