Guideline on Perinatal Oral Health Care

Originating Council
Council on Clinical Affairs

Adopted
2009

Purpose
The American Academy of Pediatric Dentistry (**AAPD**) recognizes that perinatal oral health, along with infant oral health, is one of the foundations upon which preventive education and dental care must be built to enhance the opportunity for a child to have a lifetime free from preventable oral disease. Aware that dentists, physicians, allied health professionals, and community organizations must be involved as partners to achieve this goal, the AAPD proposes recommendations for perinatal oral healthcare, including caries risk assessment, anticipatory guidance, preventive strategies, and appropriate therapeutic interventions, to be followed by the stakeholders in perinatal and pediatric oral health.

Method
This guideline is based on a review of the current dental and medical literature related to perinatal oral health care. An electronic search was conducted using the following parameters: Terms: "early childhood caries", "perinatal", and "perinatal oral health"; Fields: all fields; Limits: within the last 10 years, humans, English, and clinical trials. Papers for review were chosen from the resultant list and from 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 clinicians.

Background
The perinatal period is defined as the period around birth, beginning with the completion of the 20-28th week of gestation and ending 7-28 days after birth.[1-4] Perinatal oral health plays a crucial role in the overall health and well being of pregnant women.[5] It is also essential for the health and well being of their newborn children. Many women do not seek dental care during their pregnancy, and those that do often confront unwillingness by dentists to provide care.[6,7] Many expectant mothers are unaware of the implications of poor oral health for themselves, their pregnancy, and/or their unborn child. Research continues to show links between periodontal disease and adverse outcomes in pregnancy including preterm deliveries, low birth weight babies, and preeclampsia.[8-14] Furthermore, mothers with poor oral health and high levels of cariogenic oral bacteria are at greater risk for infecting their children with the bacteria and increasing their children's caries risk at an early age.[15] Dental caries in infants is a disease that is, by and large, preventable. Determining those mothers at highest risk for transferring cariogenic bacteria to their children improves opportunities for preventive intervention.

The primary goal of perinatal oral health care, with regard to caries transmission, is to lower the numbers of cariogenic bacteria in an expectant mother's mouth so that Mutans streptococci (**MS**) colonization of the infant can be delayed as long as possible.[16] Timely delivery of educational information and preventive therapies to these parents can help prevent the later need for dental rehabilitation in their children. Physicians, nurses, and other health care professionals are far more likely to see expectant or new mothers and their infants than are dentists. Therefore, it is essential that these providers be aware of the infectious etiology and associated risk factors of caries and early childhood caries (**ECC**), make appropriate decisions regarding timely and effective interventions for pregnant women, and facilitate the establishment of a dental home.[17-19]

Perinatal Oral Health
Caries
Caries results from an overgrowth of specific organisms that are part of the normally-occurring human oral flora.[20] MS is considered to be a principal indicator group of bacterial organisms responsible for dental caries.[21] MS colonization of an infant has been shown to occur from the time of birth.[22-28] While colonization had been thought to occur after dental eruption (as teeth provide non-shedding surfaces for adherence), current data show that other surfaces also may harbor MS.[26,29-30] The furrows of the tongue appear to be an important ecological niche in harboring the bacteria in predentate infants.[27,29]

Vertical colonization of MS from mother to infant is well documented;[31-33] genotypes of MS in infants appear identical to those present in mothers in approximately 71% of mother-infant pairs.[34] The higher the levels of maternal salivary MS, the greater the risk of the infant being colonized.[35] The success of transmission and resulting colonization of the mother's salivary MS may be related to several factors including the magnitude of the inoculum, frequency of inoculation, and a minimum infective dose.[36-38] Along with maternal salivary levels of MS, the mother's oral hygiene, periodontal disease, snack frequency, and socioeconomic status also are associated with infant colonization.[30] Recent reports have indicated that horizontal transmission (ie, transmission between members of a group) may be of concern.[39-41] Horizontal sources may include siblings of similar age or children in a daycare center.

Caries risk assessment can be performed to determine the patient's relative risk for caries. Its goal is to prevent disease by identifying and minimizing causative factors (eg, microbial burden, dietary habits, plaque accumulation) and optimizing protective factors (eg, fluoride exposure, oral hygiene, sealants).[42] Caries risk assessment tools can aid in the identification of reliable predictors and allow health care professionals to identify and refer high-risk patients.[43] The early establishment of a dental home provides time-critical opportunities to implement preventive oral health practices, including a caries risk assessment, and reduces the risk of preventable oral/dental conditions or disease for children.[44]

Anticipatory guidance [45]

Anticipatory guidance for the mother and/or other caregivers can help delay the onset and reduce the impact of MS colonization of the infant. Modification of the mother's oral hygiene and diet and the use of topical chlorhexidine and/or fluoride can have a significant effect on MS levels and, correspondingly, the child's caries rate.[46-48] Perinatal anticipatory guidance includes the following:

- Oral health education: The perinatal period is an opportune time to educate and perform dental treatment on expectant mothers.[7,10,49,50] Pregnancy offers an opportunity to educate women regarding oral health by providing a "teachable moment" in self-care and future child-care.[51] Early intervention and counseling during the perinatal period from all health care providers (eg, physicians, dentists, nurses) are essential to ensure good oral health for the mother and infant.
- Oral hygiene: Tooth-brushing and flossing by the mother on a daily basis are important to help dislodge food and reduce bacterial plaque levels. Systematic literature reviews suggest an association between periodontal disease and an increased risk of adverse pregnancy outcomes, including preterm deliveries and low birth weight babies.[52,53] Periodontal infections, which can be a reservoir for inflammatory mediators, can pose a threat to the placenta and fetus which can increase the likelihood of preterm delivery.[54,55] Mothers with severe periodontitis have high levels of prostaglandin in their gingival crevicular fluid and blood. In turn, these increased levels of prostaglandins may be associated with uterine contractions leading to preterm deliveries.[54,56] Fortunately, research shows that scaling and root planning during pregnancy can reduce the likelihood of preterm deliveries and low birth weight babies.[8,57-61] These data thus emphasize the need for perinatal intervention.

  The effects of pregnancy negatively may affect oral health behaviors among pregnant women. Nausea and vomiting may lead to avoidance of tooth brushing, resulting in an increased caries rate. For a pregnant woman experiencing frequent vomiting, rinsing with a cup of water containing a teaspoon of baking soda and waiting an hour before brushing can help minimize dental erosion.[7] Using a fluoridated toothpaste, chewing sugarless or xylitol-containing gum, and eating small amounts of nutritious food through out the day can help minimize their caries risk.[7]
- Diet: Important components of the mother's diet need to be discussed fully. A healthy diet is necessary to provide adequate amounts of nutrients for the mother-to-be and unborn child. Food cravings may lead to the consumption of foods that increase the mother's caries risk. The caries potential of the mother's diet, (ie, cariogenicity of certain foods, beverages, medicines) as well as its effect on her child, should be addressed. The frequency of consumption of cariogenic substances and resulting demineralization process also are important discussion topics.
- Fluoride: Daily use of a fluoridated toothpaste approved by the American Dental Association and rinsing nightly with an alcohol-free, over-the-counter mouth rinse containing 0.05% sodium fluoride have been suggested to help reduce plaque levels and help promote enamel remineralization.[16]
- Professional oral health care: Routine professional dental care for the mother can help optimize her oral health. Every pregnant woman should have an oral evaluation, be counseled on proper oral hygiene, and be referred for preventive and therapeutic oral health care.[7,10] Removal of active caries with subsequent restoration is important to suppress maternal MS reservoirs and has the potential to minimize the transfer of MS to the infant, thereby decreasing the infant's risk of developing ECC.[7] The safest time to perform dental treatment during pregnancy is in the second trimester, or the 14th–20th weeks.[7,10,51,62] The risk of pregnancy loss is lower compared to that in the first trimester, and organogenesis is complete.[7] Even though the second trimester is usually optimal, dental treatment can be accomplished safely at any time during pregnancy.

  Treatment options may include diagnostic x-rays,[7,63-67] dental prophylaxis, periodontal treatment, and restorations[68-73] with the administration of local anesthetics containing epinephrine.[7,74] Amalgam may be considered as a restorative material in pregnant women. There is no evidence that fetal exposure to mercury releases from the mother's existing amalgam restorations causes any adverse effects.[68-70,72] Since mercury vapor released during removal and placement of amalgam restoration may be absorbed into the blood stream and cross the placental barrier, the use of rubber dam and high speed evacuation is recommended.[72] Antibiotics and analgesics for treating infection and controlling pain may be administered.[7] Acute conditions, such as pain and swelling, should be treated as soon as possible.[7,75] Delay in necessary treatment could result in significant risk to the mother and indirectly to the fetus.[7] The consequences of not treating an active infection during pregnancy outweigh the possible risks presented by most of the medications required for dental treatment.[76] Due to patient positioning, comfort is a consideration for treatment during the third trimester. In these cases, elective treatment sometimes is best deferred until after delivery.

- Delay of colonization: Reducing maternal MS reservoirs, avoiding or delaying MS transmission, and implementing preventive practices for the child can help delay the colonization process.[77-79] Maternal MS reservoirs can be suppressed by dietary counseling, reducing the frequency of simple carbohydrate intake, applying topical chlorhexidine and/or fluoride, removing and restoring active caries, and chewing xylitol-containing chewing gum.[7] Evidence suggests that the use of xylitol chewing gum (4 pieces per day by the mother) has a significant impact on decreasing the child's caries rate.[48,80,81] Avoidance or delay of MS transmission can be accomplished by educating the mother or caregiver on behaviors that directly pass saliva to the child (eg, sharing utensils or cups, cleaning a dropped pacifier by mouth).[77,82,83] Routine preventive efforts should include toothbrushing, optimizing the child's fluoride exposure, and limiting the child's frequency of carbohydrate intake.

Recommendations

The AAPD recommends:

1. Every expectant mother receive a comprehensive oral health evaluation from a dentist and, if not previously accomplished, establish a dental home as early as possible during pregnancy. The first visit should consist of review of medical, dental, and dietary histories, risk assessment for caries and periodontal disease, diagnosis of disease and other conditions, oral health education, and anticipatory guidance. Radiographic assessment and treatment of oral conditions should be performed with attention to maternal and fetal safety and patient comfort.
2. All primary health care professionals who serve pregnant women provide education on the etiology and prevention of ECC. Oral health counseling and referral for a comprehensive oral examination and treatment during pregnancy is especially important for the mother.
3. The curriculum of all medical, nursing, and allied health professional programs include education in perinatal oral health, including the infectious etiology of ECC, methods of oral health risk assessment, anticipatory guidance, and the need for early establishment of a dental home.
4. Parents/caregivers establish a dental home for infants by 12 months of age.
5. Legislators, policy makers and third party payors be educated about the benefits of perinatal intervention in order to support efforts that improve access to oral health care for pregnant women, including more frequent and comprehensive services.

References

1. Center for Cancer Education, University of Newcastle upon Tyne. Dictionary of Cell and Molecular Biology. Available at: "http://cancerweb.ncl.ac.uk/cgi-bin/omd?query=perinatal". Accessed June 11, 2009.
2. Datasegment.com. Online dictionary. Available at: "http://onlinedictionary.datasegment.com/word/perinatal". Accessed June 11, 2009.
3. The Free Encyclopedia Wikimedia Foundation Inc. Wikipedia. Available at: "http://www.en.wikipedia.org/wiki/Perinatal". Accessed June 11, 2009.
4. Merriam-Webster Online Dictionary. Available at: "http://www.merriam-webster.com/dictionary/perinatal". Accessed June 11, 2009.
5. Brown A. Access or Oral Health Care During the Perinatal Period: A Policy Brief. National Maternal and Child Oral Health Resource Center. Georgetown University, Washington, DC; 2008. Available at: "http://www.mchoralhealth.org/PDFs/PerinatalBrief.pdf". Accessed August 30, 2009.
6. Gaffield ML, Gilbert BJ, Malvitz DM. Oral Health during pregnancy: An analysis of information collected by the pregnancy risk assessment monitoring system. J Am Dent Assoc 2001;132(7):1009-16.
7. New York State Department of Health. Oral health care during pregnancy and early childhood: Practice Guidelines. August, 2006. Available at: "http://www.health.state.ny.us/publications/0824.pdf". Accessed December 29, 2008.
8. Dasanayake AP, Gennaro S, Hendricks-Muñoz KD, Chhun N. Maternal periodontal disease, pregnancy, and neonatal outcomes. MCN Am J Matern Child Nurs 2008;33(1):45-9.
9. Sacco G, Carmagnola D, Abati S, et al. Periodontal disease and preterm birth relationship: A review of the literature. Minerva Stomatol 2008;57(5):233-50.
10. Silk H, Douglass AB, Douglass JM, Silk L. Oral health during pregnancy. Am Fam Physician 2008;77(8):1139-44.
11. Xiong X, Buekens P, Fraser WD, Beck J, Offenbacher S. Periodontal disease and adverse pregnancy outcomes: A systematic review. BJOG 2006;113(2):135-43.
12. Siqueira FM, Cota LO, Costa JE, Haddad JP, Lana AM, Costa FO. Maternal periodontitis as a potential risk variable for preeclampsia: A case-control study. J Periodontol 2008;79(2):207-15.
13. Oettinger-Barak O, Barak S, Ohel G, et al. Severe pregnancy complication (preeclampsia) is associated with greater periodontal destruction. J Periodontol 2005;76(1):134-7.
14. McKeown D. The link between periodontal disease and adverse birth outcomes. Toronto Staff Report 2006. Available at: "http://www.toronto.ca/legdocs/2006/agendas/committees/hl/hl060227/it002.pdf". Accessed June 23, 2006.
15. Ramos-Gomez FJ, Weintraub JA, Gansky SA, Hoover CI, Featherstone JD. Bacterial, behavioral and environmental factors associated with early childhood caries. J Clin Pedi Dent 2002;26(2):165-73.
16. American Academy of Pediatrics. Policy on oral health risk assessment timing and establishment of the dental home. Pediatrics 2003;111(5Pt1):1113-6.
17. Nowak AJ, Warren JJ. Infant oral health and oral habits. Pediatr Clin North Am 2000;47(5):1043-66.
18. Lewis CW, Grossman DC, Domoto PK, Deyo RA. The role of the pediatrician in the oral health of children: A national survey. Pediatrics 2000;106(6):E84.

19. Harrison R. Oral health promotion for high-risk children: Case studies from British Columbia. J Can Dent Assoc 2003;69(5):292-6.
20. Loesche WJ. Clinical and microbiological aspects of chemotherapeutic agents used according to the specific plaque hypothesis. J Dent Res 1979;58(12):2404-12.
21. Ge Y, Caufield PW, Fisch GS, Li Y. *Streptococcus mutans* and *Streptococcus sanguinis* colonization correlated with caries experience in children. Caries Res 2008;42(6):444-8.
22. Stiles HM, Meyers R, Brunnelle JA, Wittig AB. Occurrence of *Streptococcus mutans* and *Streptococcus sanguis* in the oral cavity and feces of young children. In: Stiles M, Loesch WJ, O'Brien T, eds. Microbial Aspects of Dental Caries. Washington, DC: Information Retrieval; 1976:187.
23. Loesche WJ. Microbial Adhesion and Plaque. In: Dental Caries: A Treatable Infection. 2nd ed. Grand Haven, Mich. Automated Diagnostic Documentation, Inc; 1993:81-116.
24. Berkowitz RJ, Jordan HV, White G. The early establish-ment of *Streptococcus mutans* in the mouth of infants. Arch Oral Biol 1975;20(3):171-4.
25. Wan AK, Seow WK, Purdie DM, Bird PS, Walsh LJ, Tudehope DI. A longitudinal study of *Streptococcus mutans* colonization in infants after tooth eruption. J Dent Res 2003;82(7):504-8.
26. Wan AK, Seow WK, Walsh LJ, Bird P, Tudehope DI, Purdie DM. Association of *Streptococcus mutans* infection and oral developmental nodules in pre-dentate infants. J Dent Res 2001;80(10):1945-8.
27. Berkowitz RJ. Mutans streptococci: Acquisition and transmission. Pediatr Dent 2006;28(2):106-9; discussion 192-8.
28. Law V, Seow WK, Townsend G. Factors influencing oral colonization of mutans streptococci in young children. Aust Dent J 2007;52(2):93-100; quiz 159.
29. Tanner AC, Milgrom PM, Kent R Jr, et al. The microbiota of young children from tooth and tongue samples. J Dent Res 2002;81(1):53-7.
30. Wan AK, Seow WK, Purdie DM, Bird PS, Walsh LJ, Tudehope DI. Oral colonization of *Streptococcus mutans* in six-month-old predentate infants. J Dent Res 2001;80 (12):2060-5.
31. Davey AL, Rogers AH. Multiple types of the bacterium *Streptococcus mutans* in the human mouth and their intrafamily transmission. Arch Oral Biol 1984;29(6):453-60.
32. Berkowitz R, Jones P. Mouth-to-mouth transmission of the bacterium *Streptococcus mutans* between mother and child. Arch Oral Biol 1985;30(4):377-9.
33. Douglass JM, Li Y, Tinanoff N. Association of Mutans streptococci between caregivers and their children. Pediatr Dent 2008;29(5):375-87.
34. Li Y, Caufield PW. The fidelity of initial acquisition of Mutans streptococci by infants from their mothers. J Dent Res 1995;74(2):681-5.
35. American Academy of Pediatric Dentistry. Policy on early childhood caries (ECC): Classifications, consequences, and prevention strategies. Pediatr Dent 2008;29(suppl):39-41.
36. Acs G, Lodolini G, Kaminshy S, Cisneros GJ. Effect of nursing caries on body weight in pediatric populations. Pediatr Dent 1992;14(5):302-5.
37. Clarke M, Locker D, Berall G, Pencharz P, Kenny DJ, Judd P. Malnourishment in a population of young children with severe early childhood caries. Pediatr Dent 2006;28 (3):254-9.
38. Ismail AI. Prevention of early childhood caries. Comm Dent Oral Epidemiol 1998;26(suppl):49-61.
39. Mattos-Graner RO, Li Y, Caufield PW, Duncan M, Smith JD. Genotypic diversity of Mutans streptococci in Brazilian nursery children suggests horizontal transmission. J Clin Microbiol 2001;39(6):2313-6.
40. Van Loveren C, Buijs JF, ten Cate JM. Similarity of bacteriocin activity profiles of Mutans streptococci within the family when the children acquire strains after the age of 5. Caries Res 2000;34(6):481-5.
41. Redmo Emanuelsson IM, Wang XM. Demonstration of identical strains of Mutans streptococci within Chinese families by genotyping. Eur J Oral Sci 1998;106(3):778-94.
42. American Academy of Pediatric Dentistry. Guideline on periodicity of examination, preventive dental services, anticipatory guidance, and oral treatment for infants, children, and adolescents. Pediatr Dent 2009;31(special issue):118-25.
43. American Academy of Pediatric Dentistry. Policy on the use of a caries-risk assessment tool (CAT) for infants, children, and adolescents. Pediatr Dent 2009;30(suppl):29-33.
44. American Academy of Pediatric Dentistry. Policy on the dental home. Pediatr Dent 2009;30(suppl):22-23.
45. Nowak AJ, Casamassimo PS. Using anticipatory guidance to provide early dental intervention. J Am Dent Assoc 1995;126(8):1156-63.
46. Köhler B, Andréen I, Jonsson B. The effects of caries-preventive measures in mothers on dental caries and the oral presence of the bacteria *Streptococcus mutans* and lactobacilli in their children. Arch Oral Biol 1984;29(11):879-83.
47. Brambilla E, Felloni A, Gagliani M, Malerba A, García-Godoy F, Strohmenger L. Caries prevention during pregnancy: Results of a 30-month study. J Am Dent Assoc 1998;129(7):871-7.
48. Isokangas P, Söderling E, Pienihäkkinen K, Alanen P. Occurrence of dental decay in children after maternal consumption of xylitol chewing gum: A follow-up from 0 to 5 years of age. J Dent Res 2000;79(11):1885-9.
49. Boggess KA, Society for Maternal-Fetal Medicine Publications Committee. Maternal oral health in pregnancy. Obstet Gynecol 2008;111(4):976-86.
50. De La Cruz GG, Rozier RG, Slade G. Dental screening and referral of young children by pediatric primary care providers. Pediatrics 2004;114(5):e642-52.
51. Boggess KA, Edelstein BL. Oral health in women during preconception and pregnancy: Implications for birth outcomes and infant oral health. Matern Child Health J 2006;10(5 suppl):S169-74.
52. Xiong X, Buekens P, Vastardis S, Yu SM. Periodontal disease and pregnancy outcomes: State-of-the-science. Obstet Gynecol Surv 2007;62(9):605-15.

53. Khader YS, Ta'ani Q. Periodontal diseases and the risk of preterm birth and low birth weight: A meta-analysis. J Periodontol 2005;76(2):161-5.
54. Offenbacher S, Katz V, Fertik G, et al. Periodontal infections as a possible risk factor for preterm low birth weight. J Periodontol 1996;67(suppl 10):1103-13.
55. Jeffcoat MK, Geurs NC, Reddy MS, Cliver SP, Goldenberg RL, Hauth JC. Periodontal infection and preterm birth: Results of a prospective survey. J Am Dent Assoc 2001;132(7):875-80.
56. Offenbacher S, Jared HL, O'Reily PG, et al. Potential pathogenic mechanisms of periodontitis associated pregnancy complications. Ann Periodontol 1998;3(1):233-50.
57. López NJ, Da Silva I, Ipinza J, Gutiérrez J. Periodontal therapy reduces the rate of preterm low birth weight in women with pregnancy-associated gingivitis. J Periodontol 2005;76(11 suppl):2144-53.
58. Jeffcoat MK, Hauth JC, Geurs NC, et al. Periodontal disease and preterm birth: Results of a pilot intervention study. J Periodontal 2003;74(8):1214-8.
59. Scannapieco FA, Bush RB, Paju S. Periodontal disease as a risk factor for adverse pregnancy outcomes. A systemic review. Ann Periodontol 2003;8(1):70-8.
60. Dasanayake AP, Li Y, Wiener H, Ruby JD, Lee, MJ. Salivary *actinomyces naeslundii* genospecies 2 and *lactobacillus casei* levels predict pregnancy outcomes. J Periodontol 2005;76(2):171-7.
61. López NJ, Smith PC, Gutiérrez J. Periodontal therapy may reduce the risk of preterm low birth weight in women with periodontal disease: A randomized controlled trial. J Periodontol 2002;73(8):911-24.
62. Gajendra S, Kumar JV. Oral health and pregnancy: A review. NY State Dent J 2004;70(1):40-4.
63. American College of Obstetrics and Gynecology. Committee Opinion, Number 299, September 2004 Guidelines for diagnostic imaging during pregnancy. Obstet Gynecol 2004;104(3):647-51.
64. National Council on Radiation Protection and Measurement. Report No. 54: Medical radiation exposure of pregnant and potentially pregnant women. Bethesda, Md; 1977.
65. Toppenberg KS, Hill DA, Miller DP. Safety of radiographic imaging during pregnancy. Am Fam Physician 1999;59(7):1813-8, 1820.
66. Matteson SR, Joseph LP, Bottomley W, et al. The report of the panel to develop radiographic selection criteria for dental patients. Gen Dent 1991;39(4):264-70.
67. American Dental Association, US Dept of Health and Human Services. The selection of patients for x-ray examinations: Dental radiographic examinations. Rockville, Md. Food and Drug Administration, 2004; HHS Publication Number 88-8273. Available at: "http://www.ada.org/prof/resources/topics/radiography.asp#radiographs". Accessed December 30, 2008.
68. Life Sciences Research Office. Review and analysis of the literature on the potential adverse effects of dental amalgam. Bethesda, Md. December 9, 2004. Available at: "http://www.lsro.org/presentation_files/amalgam/amalgam_press-release.pdf". Accessed June 15, 2009.
69. US Food and Drug Administration, Center for Devices and Radiological Health Consumer Information. Consumer Update: Dental Amalgam. Available at: "http://www.fda.gov/cdrh/consumer/amalgams.html". Accessed December 30, 2008.
70. Hujoel PP, Lydon-Rochelle M, Bollen AM, Woods JS, Geurtsen W, del Aguila MA. Mercury exposure from dental filling replacement during pregnancy and low birth weight risk. Am J Epidemiol 2005;161(8):734-40.
71. March of Dimes. During your pregnancy: Things to avoid: Mercury. 2008. Available at: "http://www.marchofdimes.com/pnhec/159_15759.asp". Accessed December 30, 2008.
72. Whittle KW, Whittle JG, Sarll DW. Amalgam fillings during pregnancy. Br Dent J 1998;185(10):500.
73. Olea N, Pulgar R, Perez P, et al. Estrogenicity of resin-based composites and sealants used in dentistry. Environ Health Perspect 1996;104(3):298-305.
74. Rosen MA. Management of anesthesia for the pregnant surgical patient. Anesthesiol 1999;91(4):1159-63.
75. Chiodo GT, Rosenstein DI. Dental treatment during pregnancy: A preventive approach. J Am Dent Assoc 1985; 110(3).
76. Moore PA. Selecting drugs for the dental patient. J Am Dent Assoc 1998;129(9):1281-6.
77. Berkowitz RJ. Causes, treatments and prevention of early childhood caries: A microbiologic perspective. J Can Dent Assoc 2003;69(5):304-7.
78. Kohler B, Andreen I. Influence of caries-preventive measures in mothers on cariogenic bacteria and caries experience in their children. Arch Oral Biol 1994;39(10): 907-11.
79. Li Y, Dasanayake AP, Caufield PW, Elliot RR. Butts JT III. Characterization of maternal Mutans streptococci transmission in an African American population. Dent Clin North Am 2003;47(1):87-101.
80. Söderling E, Isokongas P, Pienihäkkien K, Tenovuo J, Alanen P. Influence of maternal xylitol consumption on mother-child transmission of Mutans streptococci: 6-year follow-up. Caries Res 2001;35(3):173-7.
81. Gomez SS, Weber AA. Effectiveness of a caries preventive program in pregnant women and new mothers on their offspring. Int J Paediatr Dent 2001;11(2):117-22.
82. Berkowitz RJ. Acquisition and transmission of Mutans streptococci. J Calif Dent Assoc 2003;31(2):135-8.
83. Caufield PW, Wannemuehler YM, Hansen JB. Familial clustering of *Streptococcus mutans* cryptic plasmid strain in a dental clinic population. Infect Immun 1982;38(2): 907-11.