Review Article

Dental management of children with asthma

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Abstract

Asthma affects about 1 in 10 children. The condition is characterized by acute respiratory distress brought on by environmental factors. The condition is treated with medications aimed to reduce reaction to stimulants by the airway. Dental management involves attention to the status of the patient and awareness of stimulants of the reactive airway. Clinical recommendations are provided. (Pediatr Dent 18:363–70, 1996)

Asthma is a chronic airway disease characterized by inflammation and bronchoconstriction. Both genetic and environmental factors are responsible for this disease, which affects approximately 5–10% of children.[1,2] Although the pathophysiology is well understood, morbidity and mortality rates are increasing.[3] Asthma is the leading cause of pediatric hospitalization and accounts for nearly 1% of all U.S. medical expense.[4] The prevalence of childhood asthma necessitates that dental practitioners be familiar with this disease. This paper will review the pathophysiology and medical management of asthma in children and discuss some of the oral problems and behavioral changes associated with this disease.

Clinical manifestations and pathophysiology of asthma

The typical symptoms of asthma are coughing, wheezing, chest tightness, and dyspnea. More severe bronchial obstruction results in labored breathing, tachypnea, tachycardia, pulsus paradoxus (a decline of 10 mm Hg or more in blood pressure during inspiration compared to expiration), and diaphoresis (profuse perspiration).[5] Asthma seems to be a heterogeneous disease, particularly in early childhood.[6] About 75% of asthma in early childhood is mild, with minimal or no daily symptoms, and short-lived exacerbations often secondary to viral respiratory tract infections.[7] Wheezing in the first 3 years of life is generally self-limiting with 60% of these children being symptom-free by age 6 years.[8]

The National Heart, Lung, and Blood Institute (NHLBI) of the National Institutes of Health has recently released an advisory on asthma that categorizes asthma as mild, moderate, or severe, based in part on the frequency and severity of daytime symptoms, exercise tolerance, and night-time symptoms.[9] Children with mild asthma experience wheezing fewer than 2 days per week, lack nocturnal symptoms, and have relatively good exercise tolerance. Those with moderate asthma have wheezing 2–5 days per week with nocturnal symptoms and limited exercise tolerance. Patients with severe asthma have daily wheezing, exercise intolerance, and frequent nocturnal symptoms.

Bronchial inflammation is a major factor in the pathophysiology of childhood asthma. Inflammation potentiates the bronchial hyper-responsiveness to various triggering agents that is characteristic of asthma. The cellular inflammatory response has been shown to include infiltration of eosinophils, mast cells, and lymphocytes (mainly CD4+ T lymphocytes), together with respiratory epithelial cell damage and subepithelial thickening.[10] Patients with acute severe asthma have significant increases of three surface proteins associated with T lymphocyte activation: interleukin 2 receptor (IL-2R, CD_{25}); class II histocompatibility antigen (HLA-DR); and "very late activation" antigen.[11] Lymphocytes and other inflammatory cells are a major source of cytokines, such as interleukins-3 (IL-3), IL-4 and IL-5, and granulocyte-macrophage colony stimulating factor (GM-CSF).[12] Several mediators including histamine, cysteinyl leukotrienes, kinins, and eosinophil breakdown products, such as eosinophil cationic protein (ECP), also can be found in the asthmatic airway.[13] These mediators have potent inflammatory and smooth muscle-constricting properties, which exacerbate the disease.[14]

Precipitating factors

The most common causes of asthma exacerbations are allergen exposures (e.g., pollens, mold spores, house dust, and insect and animal emanations), viral

or mycoplasmal respiratory tract infections, exposures to nonspecific airway irritants (such as cigarette smoke), and tapering of anti-inflammatory medications either by design or noncompliance. Exercise, especially in cold weather, triggers symptoms in approximately 80% of children with asthma.[15] Cigarette smoke is a nonspecific airway irritant that can increase the frequency and severity of respiratory symptoms in children with asthma. More severe asthma, diminished lung function, and increased episodes of respiratory infections are seen often in children whose parents smoke.[16] Emotional and psychological stress also may trigger asthmatic attacks. Aspirin and beta adrenergic blockers have been shown to trigger bronchospasm by a non-IgE–mediated mechanism. Reactions to food or food additives as well as IgE-mediated insect reactions and adverse responses to allergen desensitization treatments also have been reported.[17] IgE-mediated bronchospasm following ingested or injected agents may occur independently, but typically is part of a more generalized allergic reaction depicted clinically by angioedema, urticaria, and/or anaphylaxis. Epinephrine is indicated in subjects whose acute asthma is a component of such a systemic reaction.

Medical management

Acute and chronic asthma are classified into mild, moderate, and severe according to the severity and frequency of occurrence of the signs and symptoms and according to the degree of airway obstruction as measured with pulmonary function tests (Table 1). The medical management of childhood asthma is determined according to these criteria. However, the severity of either acute or chronic asthma may vary within the same patient over time, and therefore adjustments in treatment frequently are necessary. The goals of asthma therapy according to NHLBI guidelines include:

1. Maintaining normal activity levels (including exercise)
2. Maintaining near normal pulmonary function
3. Preventing chronic symptoms
4. Preventing recurrent exacerbations of asthma
5. Avoiding adverse effects from asthma medication.[9]

Pharmacological management of chronic childhood asthma involves two main categories of drugs: anti-inflammatory agents and bronchodilators (Table 2). Children with mild asthma often are managed only with inhaled β_2 receptor agonist bronchodilators, such as albuterol and terbutaline sulfate. The typical outpatient maintenance dose of albuterol is one to two puffs (90 μg each) every 4 to 6 hr as needed for symptoms. However, excessive use of β_2 agonists (more than 200 inhalations per month) is a sign of poorly controlled asthma. Cromolyn sodium and nedocromil sodium are anti-inflammatory agents that work in part by preventing mast cell release of mediators and are used in patients with moderate asthma. Given prophylactically, cromolyn and nedocromil can prevent allergen-induced early asthmatic responses and late asthmatic responses, and may help reduce airway reactivity.[18] These medications, however, have no significant role in the management of acute asthma attacks.[19] Oral theophylline, which has bronchodilator and some anti-inflammatory effects, is another option for moderate asthma. Lastly, inhaled corticosteroids are very effective anti-inflammatory agents and are recommended for use in children with moderate to severe asthma, although there are more concerns about their long-term safety. Inhaled steroids provide good control for chronic asthma symptoms and are convenient because they can be administered twice daily.[20] In general, asthma that is more than mild in severity requires treatment with an anti-inflammatory agent and with β_2 agonists "as needed", preferably via inhalation.

Acute asthma is a medical emergency and initiation of therapy should not be delayed. The goal in treating acute asthma is to eliminate symptoms and improve lung function as quickly as possible.[21] Initial treatment for acute severe asthma typically involves an inhaled β_2 agonist such as albuterol.[22] Albuterol is a rapid-acting drug whose maximal effects are seen within minutes.[23] Administration in the hospital is via a jet nebulizer driven by 100% oxygen through a face mask[24] or via a metered dose inhaler with a "spacer". Spacers are aerosol holding chambers that help coordinate metered dose inhaler actuation with inhalation; their use also helps minimize oral and enhance lung deposition of the aerosolized drugs. Ipratropium bromide (an anticholinergic) is a less potent bronchodilator used by some patients with moderate or severe acute asthma because when it is administered with albuterol, it provides an additive effect. Moderate doses of systemic corticosteroids (about 2 mg/kg/day of prednisone or equivalent) also are recommended for patients with acute severe asthma.[21] Finally, supportive treatment of acute severe asthma includes supplemental oxygen, fluid and electrolyte maintenance, anxiety relief, and endotracheal intubation and, in extreme situations, mechanical ventilation.[20]

Associated oral problems

A 1993 restrospective study from Sweden reported an increased prevalence in caries in children with moderate to severe asthma.[25] The mechanism for this development may relate to β_2-agonist effects on salivary gland function. Another report found that these agents decrease the secretion of whole saliva by 20% and parotid saliva by 35%, and are associated with an increase in the number of lactobacilli.[26] These adverse changes contribute to an increased caries susceptibility. Due to this risk, asthmatic children should receive special caries prevention attention.

The role of impaired nasorespiratory function as an etiologic factor in the development of certain dentofacial deformities has been suggested.[27,28] Bresolin

TABLE 1. CATEGORIZATION OF ACUTE AND CHRONIC ASTHMA IN CHILDREN

Asthma Type	*Mild*	*Moderate*	*Severe*
Acute			
Symptoms			
Respiratory rate above mean	Normal to 30% above mean	30–50% above mean	> 50% above mean
Dyspnea	Mild if present severe	Present but not speech	Fragmented
Color/level of consciousness	Good/normal	Pale/normal	Maybe cyanotic/ maybe decreased
Retractions intercostal	None to slight SCM retraction	Intercostal and and nasal flaring	Increasing effort
Auscultation wheezes	End-expiratory atory wheezes	Inspiratory/expi- sounds	Decreased breath
PEFR* (% of baseline)	70–90%	50–70%	< 50%
SaO_2† in room air	> 95%	90–95%	< 90%
Treatment			
β2-agonist	Inhaled	Inhaled subcutaneous	Inhaled or
Oxygen	No	Yes	Yes
Systemic steroids	± Oral 4–5 days	Oral 4–5 days	IV
Usual outcome	Discharged	± Hospitalized	Hospitalized, ± ICU
Chronic			
History of ER/ hospital use	None	Occasional ER hospital	ER/occasional
Symptoms			
Frequency	< 2 days/week	≥ 2 days/week	Daily
Duration	< 1/2 hour	May last few days	Almost continuous
PEFR (% baseline)	≥ 80%	60–80%	< 60%
Treatment			
β2-agonists	p.r.n.	p.r.n.	Daily
Anti-inflammatory	None; dose inhaled steroids	Cromolyn or low dose inhaled steroids plus theophylline	High dose
Other	None; rarely oral steroids 4–5 days	Theophylline, oral steroids	Frequent or daily
Dental management	Routine examination, cleaning, simple operative procedures	Routine examination and cleaning	Defer dental visit until controlled

* PEFR (peak expiratory flow rate): the highest expiratory flow rate that can be achieved during a maximally forceful exhalation that starts at total lung capacity. This flow rate correlates well with the degree of bronchial obstruction and can be measured easily in the home or office with relatively inexpensive hand-held devices.

† SaO_2: transcutaneous oxygen saturation of hemoglobin.

TABLE 2. A LIST OF COMMONLY USED DRUGS IN THE TREATMENT OF ASTHMATIC CHILDREN

Category	*Generic Name*	*Common Trade Names*	*Comments*	*Side Effects*
Bronchodilator				
Inhaled β2				
Short acting	Albuterol•	Ventolin™, Proventil™	First line drugs; recommended use "as needed" for symptoms. Effects last 4–6 hr	Transient tachycardia, tremor, nausea; less frequently nervousnes, palpitations
	Terbutaline• Bitolterol Pirbuterol	Brethaire™ Tornalate™ Maxair Autohaler™		
	Metaproternol•	Alupent™, Metaprel™		
Long acting	Salmeterol	Serevent™	Used as maintenance therapy with anti-inflammatory drugs	Same as short acting
Inhaled Anticholinergic				
	Ipratropium•	Atrovent™	Not used as first line drug in children	Cough, nervousness nausea
Oral				
	Theophylline	Theodur™, Slobid™	Used as maintenance therapy, may need to monitor blood levels. Fever, erythromycin and cimetidine increase blood levels	Nausea, vomiting, epigastric pain. Less frequently caffeine-like CNS effects
Anti-inflammatory				
Inhaled Steroids	Beclomethasone	Beclovent™, Vanceril™	Most effective inhaled anti-inflammatory agents. Impact on growth in children remains controversial	Oropharyngeal candidiasis, hoarseness, throat irritation. Very high doses may cause adrenal suppression
	Triamcinolone Flunisolide	Azmacort™ Aerobid™		
Other Inhaled Anti-inflammatory Agents				
	Cromolyn•	Intal™	Safest inhaled anti-inflammatory agent, first choice in children	Cough, wheezing, throat irritation
	Nedocromil	Tilade™	Similar to cromolyn	Bad taste, cough, wheezing

•Also available in nebulizer solution.

and coworkers evaluated 45 children with chronic rhinitis and mouth breathing and found that they presented with an increased upper anterior and total anterior facial height, higher palatal vaults, greater overjets, and higher prevalence of posterior crossbites.[29] In another controlled study, Venetikidou reported a greater incidence of posterior crossbite in asthmatic children.[30]

Inhaled steroids are being used increasingly as first-line therapy in children with moderate asthma.[31] However, inhaled steroid therapy has potential side effects including adrenal suppression, growth impairment,[32] throat irritation, dysphonia, dryness of the mouth, oropharyngeal candidiasis,[33] and rarely, tongue enlargement.[34]

Associated behavior problems

Psychological disturbances have been shown to be more common in patients with severe asthma than in healthy children.[35, 36] For example, Mrazek found that 25% of severe asthmatics suffered from emotional disturbances.[37] Some authors suggest a "vicious cycle": asthma contributes to the development of behavioral problems, which, in turn, trigger or exacerbate asthmatic symptoms.[38, 39]

Asthma medications also have been associated with behavioral, affective, and neuropsychological changes in children. Patients were found to be easily tired, argumentative, irritable, and sad when receiving steroids.[40] High-dose prednisone therapy, commonly used to treat acute severe asthma, may result in anxiety or depression, particularly for children with a history of emotional difficulty.[40] Oral β_2 agonists have been reported to cause psychotic reactions in adult patients. In children, inhaled albuterol frequently induces a short-term hand tremor.[41] The National Asthma Education Program Expert Panel Report: *Guidelines for the Diagnosis and Management of Asthma* has designated theophylline as a "second line drug", or a therapeutic alternative to cromolyn or inhaled corticosteroid in the treatment of moderately severe childhood asthma.[42] Like caffeine, theophylline is a CNS stimulant and its side effects, which tend to increase with increasing plasma levels, include gastrointestinal discomfort, headache, nausea, vomiting, nervousness, insomnia, and rarely, seizures.[17]

Studies have found that parents of asthmatic children also have a higher anxiety level than parents of healthy children.[43, 44] Parental and child anxiety may result in overprotection and interfere with the child's ability to develop autonomy.[39] The fear of precipitating an asthma attack may result in parental failure to set appropriate limitations on behavior. Another common family dysfunction problem is child neglect. In a study of neglected children with chronic illness from low-income families, 16% were found to be asthmatic.[45]

Dental management

Safe dental management of children with asthma depends on the pulmonary status of the patient at the time of the dental intervention. For patients with asthma, the practitioner should consider the following to determine how well the disease is controlled: 1) the frequency of asthmatic attacks, 2) the type of medications used chronically and during acute attacks, and 3) the length of time since the child was last seen emergently with acute asthma. Physical examination may include auscultation of the lungs, observation of the rate and depth of respiration, use of accessory muscles for respiration, shortness of breath, and coughing. For a severe asthmatic, consultation with the patient's primary care physician is recommended.

Dental procedures may be accomplished in the clinic setting for the asymptomatic or well-controlled asthmatic. A wheezing or poorly controlled patient should be reappointed. If a patient has been or is currently using a metered dose inhaler bronchodilator, it should be brought to each dental appointment. Anxiety is a trigger in children with asthma, and the dental environment is a common site for an acute asthmatic attack.[46] A calm and confident approach by the dental staff may help alleviate anxiety. If conscious sedation is required, hydroxyzine (Vistaril™, Pfizer Labs, NY, NY), which has antihistaminic and sedative effects, and benzodiazepines, which are anxiolytic and do not induce bronchoconstriction, are usually recommended.[47, 48] Barbiturates and narcotics (especially morphine and meperidine) should be avoided in children with asthma because of their potential for stimulating histamine release, which can lead to bronchospasm.[49] The analgesic and anxiolytic properties of nitrous oxide (N_2O), as well as the supplemental oxygen received during N_2O administration, are thought to help manage children with asthma.[49] According to Malamed,[50] the use of N_2O in children with mild to moderate asthma can effectively prevent acute symptoms. However, because N_2O is somewhat irritating to the airway, its use in children with severe asthma is contraindicated, and medical consultation is recommended prior to N_2O use in these children.[49, 50]

IV sedation should be used with extreme caution as asthmatics have limited control of their airways. Ketamine, a dissociative anesthetic with sedative, analgesic, and bronchodilating properties,[51, 52] has been used safely in asthmatic patients.[53, 54] However, ketamine's sympathetic activation is of concern for patients with a history of cardiovascular or hypertensive heart disease. Patients who have anything more than mild asthma should have procedures performed where standard monitors (pulse oximetry, end-tidal $SaCO_2$, EKG, and blood pressure cuff) and intubation equipment are available. Patients with asthma who are prone to abrupt and severe episodes of airway obstruction are best treated in a hospital.

Children with asthma may be at risk for significant adverse reactions to medications commonly used in dental practice. Nearly 4% of patients with asthma are

allergic to aspirin and other nonsteroidal anti-inflammatory agents.[55] Thus, acetaminophen usually is recommended for these children. Patients taking theophylline preparations should not receive erythromycin, because it interferes with the metabolism of theophylline and raises its blood level into the toxic range.[49] Historically, dentists have been warned not to use local anesthetics with vasoconstrictors in asthmatic patients because vasoconstrictors contain sodium metabisulfite, a highly allergenic substance.[56,57] Despite this warning, local anesthetics with vasoconstrictors have been used safely.[58] However, from another point of view, local anesthetics with vasoconstrictors should be used with caution since they may add to the effects of β_2-agonists, resulting in palpitations, increased blood pressure, and arrhythmias.[48]

Asthmatic children exposed to systemic glucocorticoids (GC) may be at risk for developing adrenal insufficiency during major dental procedures or general anesthesia. They also have a greater risk (up to three times) than children without asthma for developing anesthesia-related complications postoperatively.[59] Children with asthma on maintenance systemic GC (daily or every other day) are adrenally suppressed and need to be supplemented on the day of the dental procedure by doubling the patient's usual daily dose.[59] Children at risk for developing adrenal insufficiency with major dental procedures or general anesthesia include those who have had four or more brief courses (4–5 days/course)[60] or a continuous 10–14 day course of systemic GC for acute asthma within the previous year, and those who have taken systemic GC within 30 days.[59] These children probably need to receive stress replacement doses of steroids (60 mg hydrocortisone/m^2/dose) 6–8 hr before, and again 1 hr before the procedure,[61] although this is not generally agreed to by all authors.[59,62] Children with asthma who do not fit these categories, and those who do but are undergoing only minor dental procedures, such as routine examination, cleaning, and simple operative procedures, do not require supplemental steroids.[59]

Children with severe asthma require an anesthesiology evaluation to avoid the risk of developing peri- and postoperative complications. A review of the patient's history, a physical examination, and in selected patients, a determination of pulmonary function (chest radiograph and peak expiratory flow or spirometry) may be necessary to identify those who require adjustment of their asthma therapy prior to dental treatment. Pulmonary function tests can identify some patients who are asymptomatic but are significantly obstructed and have a below-normal FEV_1 (the forced expiratory volume in 1 sec, a common measure of airway obstruction). Poorly controlled patients and those with nocturnal wheezing, frequent severe attacks, uncontrolled exercise-induced bronchospasm, or poor pulmonary function, should have dental treatment postponed until the asthma is controlled.

The following steps should be taken to manage an acute asthmatic attack in the dental office:

1. Discontinue the dental procedure and allow the patient to sit or lie down in a comfortable position
2. Keep the airway open and administer β_2-agonists with inhaler or nebulizer
3. Administer oxygen via face mask, nasal hood, or cannula
4. If no improvement takes place and the patient is worsening, administer epinephrine subcutaneously (1:1000 solution, 0.01 mg/kg of body weight to a maximum dose of 0.3 mg), and summon medical assistance.[50]

It is very important to continue the therapy and keep a satisfactory oxygen saturation level until the patient is free of wheezing or until medical transportation is available to take the patient to an emergency room. The episode should be documented in detail and reported to the child's primary care physician.

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1. Cropp GJA: Special features of asthma in children. Chest 87:[Suppl 55s–62s], 1985.
2. McCarthy TP, Lenney W: Management of asthma in preschool children. Br J Gen Pract 42:429–34, 1992.
3. Sly RM: Changing asthma mortality. Ann Allergy 73:259–68, 1994.
4. Weiss KB, Gergen PJ, Hodgson TA: An economic evaluation of asthma in the United States. N Engl J Med 326:862–66, 1992.
5. Casey KR, Winterbauer RH: Acute severe asthma: How to recognize and respond to a life-threatening attack. Postgrad Med 97:71–78, 1995.
6. Wilson NM: Wheezy bronchitis revisited. Arch Dis Child 64:1194–99, 1989
7. Clough J: Asthma in the very young. Practitioner 239:198–202, 1995.
8. Martinez FD, Wright AL, Taussig LM, Holberg CJ, Halonen M, Morgan WJ: Asthma and wheezing in the first six years of life. The Group Health Medical Associates. N Engl J Med 332:133–38, 1995. [Comment 332:181–82, 1995]
9. National Asthma Advisory Panel Guidelines for the Diagnosis and Treatment of Asthma. J Allergy Clin Immunol 88:425–34, 1991.
10. Djukanovic R, Roche WR, Wilson JW, Beasley CR, Twentyman OP, Howarth RH, Holgate ST: Mucosal inflammation in asthma. Am Rev Respir Dis 142:434–57, 1990.

11. Corrigan CJ, Hartnell A, Kay AB: T-lymphocyte activation in acute severe asthma. Lancet 21:1129–32, 1988.
12. Broide DH, Paine NM, Firestein GS: Eosinophils express interleukin 5 and granulocyte macrophage-colony-stimulating factor mRNA at sites of allergic inflammation in asthmatics. J Clin Invest 90:1414–24, 1992.
13. Weller PF, Lee CW, Foster DW, Corey EJ, Austen KF, Lewis RA: Generation and metabolism of 5-lipoxygenase pathway leukotrienes by human eosinophils: predominanat production of leukotriene C4. Proc Natl Acad Sci USA 80:7626–30, 1983.
14. Chung KF, Barnes PJ: Role of inflammatory mediators in asthma. Br Med Bull 48:135–48, 1992.
15. Asher MI, Toop L, Mitchell EA: Asthma in children: consensus on preventive management in New Zealand. NZ Med J 107:108–10, 1994.
16. Weitzman M, Gortmaker S, Walker DK, Sobol A: Maternal smoking and childhood asthma. Pediatrics 85:505–11, 1990.
17. Kaliner MA, White MV: Asthma: causes and treatment. Compr Ther 20:645–50, 1994.
18. Wasserman SI: A review of some recent clinical studies with nedocromil sodium. J Allergy Clin Immunol 92:210–15, 1993.
19. Leatherman J: Life-threatening asthma. Clin Chest Med 15:453–79, 1994.
20. McWilliams B: Outpatient management of childhood asthma. Pediatr Ann 22:571–72, 575, 579–81, 1993.
21. Cockcroft DW: Management of acute severe asthma. Annals Allergy Asthma Immunol 75:83–89, 1995. [Quiz 75:90–93, 1995]
22. Fitzgerald JM, Hargreave FE: Acute asthma: emergency department management and prospective evaluation of outcome. Can Med Assoc J 142:591–95, 1990. [Comment 142:1183, 1186–87, 1990]
23. McFadden ER: Beta2 receptor agonist: metabolism and pharmacology. J Allergy Clin Immunol 68:91, 1982.
24. Colacone A, Afilalo M, Wolkove N, Kreisman H: A comparison of albuterol administered by metered dose inhaler (and holding chamber) or wet nebulizer in acute asthma. Chest 104:835–41, 1993.
25. Arnrup K, Lundin S-A, Dahllof G: Analysis of paediatric dental services provided at a regional hospital in Sweden. Dental treatment needs in medically compromised children referred for dental consultation. Swed Dent J 17:255–59, 1993.
26. Ryberg M, Möller C, Ericson T: Saliva composition and caries development in asthmatic patients treated with beta 2-adrenoceptor agonists: a 4-year follow-up study. Scand J Dent Res 99:212–18, 1991.
27. Linder-Aronson S: Adenoids: their effects on mode of breathing and nasal airflow and their relationship to characteristics of the facial skeleton and the dentition. A biometric, rhino-manometric and cephalometro-radiographic study on children with and without adenoids. Acta Otolaryngo Supp 265:1–32, 1970.
28. Woodside DG, Linder-Aronson S, Lundstrom A, McWilliam J: Mandibular and maxillary growth after changed mode of breathing. Am J Orthod Dentofacial Orthop 100:1–18, 1991.
29. Bresolin D, Shapiro PA, Shapiro GG, Chapko MK, Dassel S: Mouth breathing in allergic children: its relationship to dentofacial development. Am J Orthod 83:334–40, 1983.
30. Venetikidou A: Incidence of malocclusion in asthmatic children. J Clin Pediatr Dent 17:89–94,1993.
31. Agertoft L, Pedersen S: Effects on long-term treatment with an inhaled corticosteroid on growth and pulmonary function in asthmatic children. Respir Med 88:373–81, 1994.
32. Priftis K, Everard ML, Milner AD: Unexpected side effects of inhaled steroids: a case report. Eur J Pediatr 150:448–49, 1991.
33. Pingleton WW, Bone RC, Kerby GR, Ruth WE: Oropharyngeal candidiasis in patients treated with trimincolone acetonide aerosol. J Allergy Clin Immunol 60:254–58, 1977.
34. Linder N, Kuint J, German B, et al: Hypertrophy of the tongue associated with inhaled corticosteroid therapy in premature infants. J Pediatr 127:651–53, 1995.
35. Perrin JM, MacLean WE Jr, Perrin EC: Parental perceptions of health status and psychological adjustment of children with asthma. Pediatrics 83:26–30, 1989.
36. Hilliard JP, Fritz GK, Lewiston NJ: Goal setting behavior of asthmatic, diabetic and healthy children. Child Psychiatry Hum Dev 13:35–47, 1982.
37. Mrazek DA: Psychiatric complications of pediatric asthma. Ann Allergy 69:285–90, 1992.
38. Lehrer PM, Isenberg S, Hochron SM: Asthma and emotion: a review. J Asthma 30:5–21, 1993. [Comment 30:1–3, 1993]
39. Richards W: Preventing behavior problems in asthma and allergies. Clin Pediatr (Phila) 617–24, 1994, October.
40. Milgrom H, Bender B: Behavioral side effects of medications used to treat asthma and allergic rhinitis. Pediatrics in Review 16:333–35, 1995.
41. Mazer B, Figueroa-Rosario W, Bender B: The effect of albuterol aerosol on fine-motor performance in children with chronic asthma. J Allergy Clin Immunol 86:243–48, 1990.
42. Sheffer AL: The National Asthma Education Program attacks asthma. J Allergy Clin Immunol 87:468–69, 1991. [Editorial]
43. Brook U, Weitzman A, Wigal JK: Parental anxiety associated with a child's bronchial asthma. Pediatr Asthma Allerg Immunol 5:15–20, 1991.
44. Staudenmayer H: Parental anxiety and other psychosocial factors associated with childhood asthma. J Chronic Dis 34:627–36, 1981.
45. Jaudes PK, Diamond LJ: Neglect of chronically ill children. Am J Dis Child 140:655–58, 1986.
46. Fast TB, Martin MD, Ellis TM: Emergency preparedness: a survey of dental practitioners. J Am Dent Assoc 112:499–501, 1986.
47. Mungo RP, Kopel HM, Church JA: Pediatric dentistry and the child with asthma. Special Care in Dent November-December 6:270–73, 1986.
48. Luce EB: Respiratory disease. In: Dentistry in Systemic Disease: Diagnostic and Therapeutic Approach to Patient Management. Redding SW, Montgomery M, Eds. Portland, OR: JBK Publishing, 1990, pp 55–79.
49. Little JW, Falace DA: Pulmonary disease. In: Dental Management of the Medically Compromised Patient, 4th ed. Little JW, Falace DA, Eds. St Louis: CV Mosby Co, 1993, pp 235–41.
50. Malamed SF: Asthma. In: Medical Emergencies in the Dental Office, 4th ed, St Louis: CV Mosby Co, 1993, pp 194–207.
51. White PF, Way WL, Trevor AJ: Ketamine—its pharmacology and therapeutic uses. Anesthesiol 56:119–36, 1982.
52. Öye I, Paulsen O, Maurset A: Effects of ketamine on sensory perception: evidence for a role of N-methyl-D-aspartate receptors. J Pharmacol Exp Ther 260:1209–13, 1992.
53. Haas DA, Harper DG: Ketamine: a review of its pharmacologic properties and use in ambulatory anesthesia. Anesth Prog 39:61–68, 1992.
54. Lökken P, Bakstad OJ, Fonnelop E, Skogedal N, Hellsten K, Bjerkeland CE, Storhaug K, Öye I: Conscious sedation by rectal administration of midazolam or midazolam plus ketamine as alternatives to general anesthesia for dental treatment of uncooperative children. Scand J Dent Res 102:274–80, 1994.
55. Kacso G, Terézhalmy GT: Acetylsalicylic and acetaminophen. Dent Clin North Am 38:633–44, 1994.
56. Simon RA: Sulfite sensitivity. Ann Allergy 56:281–88, 1986.

57. Seng GF, Gay BJ: Dangers of sulfites in dental local anesthetic solutions: warning and recommendations. J Am Dent Assoc 113:769–70, 1986.
58. Pérusse R, Goulet JP, Turcotte JY: Sulfite, asthma and vasoconstrictors. Can Dent Assoc J 55:55–56, 1989.
59. Glick M: Glucocorticosteroid replacement therapy: a literature review and suggested replacement therapy. Oral Surg Oral Med Oral Pathol 67:614–20, 1989.
60. Dolen LM, Kesarwala HH, Holroycle JC, Fischer TJ: Short term, high-dose, systemic steroids in children with asthma: the effect on the hypothalemic-pituitary-adrenal axis. J Allergy Clin Immunol 80:81–87, 1987.
61. Johnson KB: Special drug topics. In: The Harriet Lane Handbook, 13th ed. Green M, Ed. St Louis: CV Mosby Co, 1993, pp 565.
62. Holland EG, Taylor AT: Glucocorticoids in clinical practice. J Fam Pract 32:512–19, 1991.