Asthma: An anesthetic update

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The effective management of the patient with asthma continues to represent a significant challenge in modern anesthesia practice. The prevalence of asthma is increasing worldwide and is the most common chronic disorder among children. Classification and treatment strategies continue to evolve as new therapies emerge. Fortunately, the incidence of bronchospastic episodes under anesthesia has declined in recent years with the development of improved anesthetic drugs and techniques. A thorough understanding of the pathogenesis of asthma will assist in developing anesthetic management plans that are patient specific and use the best treatment pathways currently available.

Key words: Anesthesia, asthma, bronchospasm.

OBJECTIVES
At the completion of this course, the reader should be able to:
1. Discuss how the prevalence of asthma has changed in the United States in the past 20 years.
2. Describe the biochemical mediators and associated pathophysiologic mechanisms involved with the asthmatic episode.
3. Identify common clinical characteristics and associated complications of asthma in the awake and anesthetized patient.
4. Compare the differences between immunologic and non-immunologic asthma classifications.
5. Compare the available treatment regimens for acute and chronic asthma.

Introduction
Asthma is a chronic respiratory illness characterized by bronchial inflammation and hyperresponsiveness resulting in recurrent episodes of bronchoconstriction, airway edema, cough, and wheeze. Complications associated with an asthmatic attack may include hypoxia, pneumothorax, airflow obstruction, and respiratory fatigue. Surgical patients with a history of asthma have a higher incidence of perioperative respiratory complications than do patients without asthma. In a minority of surgical patients, the complications may cause serious perioperative morbidity. Prudence dictates developing rational approaches to minimize risks and avoid catastrophic outcomes in patients with asthma. This article presents the epidemiology, pathogenesis, and pathophysiology of asthma; summarizes treatment options; and outlines a framework for the anesthetic management of patients with asthma.

Epidemiology
Asthma is a relatively common disorder, affecting about 4% to 5% of the US population. The disease disproportionately affects African Americans and those living in urban areas. About half of all patients develop the disease in childhood, but the age of onset can continue into the sixth or seventh decade. Complete remission of asthma occurs in 30% to 70% of patients with childhood-onset disease, but asthma that develops in adulthood rarely subsides.
From 1980 to 1996, the number of Americans with asthma increased by 75%, with children younger than 5 years experiencing the highest rate of increase. The remarkable rise of asthma both in the United States and worldwide has prompted experts to focus attention on possible causes. Reasons for the escalating rates are unclear; many believe that factors related to our modern lifestyle, including greater exposure to environmental and occupational pollutants, may underlie the increasing prevalence. Atopy, the genetically inherited susceptibility to become allergic, is the most important predictor of a person developing asthma. The genetic susceptibility to asthma and the complex interplay between susceptibility and environmental exposure are not well understood. Moreover, the mortality rate for asthma has increased steadily during the past 20 years. The death rate for children 19 years and younger increased by 78% between 1980 and 1993.

In the surgical patient, complication rates associated with asthma have fallen significantly compared with 30 years ago. Today, asthma-related morbidity and mortality are uncommon in the perioperative period, and the risk of significant bronchospasm in patients with stable asthma is low. Improvements in medical therapy, improved anesthetic care, and better perioperative monitoring may account for the reduced risk.

In a retrospective review of 706 surgical patients with asthma, Warner et al reported perioperative bronchospasm in only 1.7% of patients. There were no reported episodes of pneumothorax, unanticipated mechanical ventilation, or death, and only 1 patient experienced refractory bronchospasm. Older patients and those with severe disease (eg, recent therapy in a medical facility, recent antiasthmatic drug use, or recent asthmatic symptoms) had the highest frequency of perioperative complications.

Olsson reviewed 136,929 patients having surgery during an 11-year period in Sweden. Bronchospasm occurred in 1.6 per 1,000 anesthetics, and no deaths were noted. A history of allergy or asthma was not a significant factor in the occurrence of bronchospasm, and most episodes were triggered by mechanical stimulation such as intubation.

A database at the University of Washington Medical Center, Seattle, of 30,654 general anesthetics noted 70 cases of bronchospasm, or a 0.23% incidence. Of these cases, 10% occurred in people with asthma. The American Society of Anesthesiologists Closed Claims Project Database contains 88 cases of significant bronchospasm out of 3,533 claims. Twenty eight (32%) reported a history of asthma. It seems reasonable to conclude from these reports that although asthma-related morbidity is uncommon, occasional serious airway complications may still occur.

Pathogenesis and pathophysiology

Our contemporary understanding of asthma is that it is not a single entity, but rather a heterogeneous clinical syndrome characterized by episodic hyperresponsive airways, interspersed with symptom-free periods. Bronchoconstriction is a factor long associated with the asthmatic symptom complex, but asthma is much more than bronchoconstriction. Airway inflammation and a nonspecific hyperirritability of the tracheobronchial tree are now recognized as being central to the pathogenesis of even mild cases of asthma. Permanent changes in airway anatomy, referred to as airway remodeling, magnify the inflammatory response.

Asthma can be classified broadly into 2 groups: allergic asthma and idiosyncratic asthma. Allergic asthma (atopic or immunological disease) is triggered by antigens that provoke a T lymphocyte–generated, immunoglobulin E–mediated immune response. It often is associated with a personal and/or family history of allergic disease.

In susceptible patients, exposure to even minute amounts of an offending agent can cause activation of lymphocytes and cytokine release, setting into motion an immune-mediated inflammatory response. Endobronchial biopsy specimens, even from asymptomatic patients, frequently show an active inflammatory process. Eosinophils, mast cells, neutrophils, and macrophages are prominent features in asthmatic airways, and their activation and degranulation fuel the proinflammatory cascade.

Potent biochemical mediators released from proinflammatory and airway epithelial cells promote vasoconstriction, increased smooth muscle tone, enhanced mucus secretion, submucosal edema, increased vascular permeability, and inflammatory cell chemotaxis. Leukotrienes have been identified as especially potent spasmodic and proinflammatory substances. Released molecules that are toxic to the airway epithelium cause patchy desquamation, exposing cholinergic nerve endings and compounding the bronchoconstrictive and hyperresponsive response.

The asthmatic diathesis creates airways that are inflamed, edematous, and hypersensitive to irritant stimuli, and the degree of airway hyperresponsiveness, and bronchoconstriction seems to parallel the extent of inflammation. When airway reactivity is high, asthmatic symptoms generally are more severe and unrelenting, and the amount of therapy required to control the episode is greater. Table 1 lists some of the chemical mediators involved in the asthmatic episode and their physiologic effects.

The mechanisms underlying idiosyncratic asthma (nonimmunologic disease) are less clearly defined. Nonimmunologic asthma occurs in patients with no...
history of allergy and a normal serum immunoglobulin E level. Asthmatic symptoms typically develop in response to some provocative or noxious stimuli, such as cold air, airway instrumentation or irritation, climate changes, or an upper respiratory illness. Recent upper respiratory infection may precipitate bronchospasm in any patient, but the risk is higher in patients with a history of asthma. The increased bronchomotor tone associated with viral respiratory infections may persist for as long as 5 weeks.22 Children without asthma who have an upper respiratory infection are 2 to 7 times more likely than children with no such infection to experience an adverse event perioperatively and are more prone to postoperative desaturation.23 Children with asthma can be considered to be at a similar if not greater risk. Enhanced parasympathetic nervous system tone can contribute to the airflow obstruction.

Immune mechanisms seem to be causally related or contributory to the development of asthma in more than 50% of cases, but many patients with asthma have disease mechanisms from both categories.15 Asthma that has its onset in childhood tends to have a strong allergic component, whereas asthma that arises in adults tends to be nonallergic or to have mixed causes.15,24 As a general rule, nonallergic mechanisms are more prevalent in the perioperative period.25 Significantly, the clinical features of idiosyncratic asthma are essentially indistinguishable from the immune-mediated response (Figure).

**Clinical features**

- **Awake patient.** Key hallmarks of asthma in the awake patient include the following:4,20
  1. Wheezing
  2. Dyspnea (may parallel the severity of expiratory airflow obstruction)
  3. Cough (productive or nonproductive; frequently

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**Table 1. Inflammatory mediators and asthma***

<table>
<thead>
<tr>
<th>Inflammatory mediator</th>
<th>Source</th>
<th>Effects</th>
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<tbody>
<tr>
<td>Histamine</td>
<td>Mast cells</td>
<td>Bronchoconstriction</td>
</tr>
<tr>
<td>Leukotrienes (LTB₁, LTC₄, LTD₄, LTE₅)</td>
<td>Mast cells, basophils, eosinophils, neutrophils, macrophages, monocytes</td>
<td>Bronchoconstriction, Chemotaxis, mucus hypersecretion, edema</td>
</tr>
<tr>
<td>Platelet-activating factor</td>
<td>Mast cells, basophils, eosinophils, neutrophils, macrophages, monocytes, platelets</td>
<td>Eosinophil recruitment, bronchoconstriction, airway hyperresponsiveness</td>
</tr>
<tr>
<td>Proteases</td>
<td>Enzymes</td>
<td>Bronchial hyperresponsiveness</td>
</tr>
<tr>
<td>Prostaglandins (PGF₂α, PGD₂, PGF₂, PGI₂)</td>
<td>Mast cells, endothelial cells</td>
<td>Bronchoconstriction, mucus secretion</td>
</tr>
<tr>
<td>Major basic protein</td>
<td>Eosinophils</td>
<td>Epithelial damage</td>
</tr>
<tr>
<td>Thromboxanes (TXA₂)</td>
<td>Macrophages, monocytes, platelets</td>
<td>Bronchoconstriction, mucus secretion</td>
</tr>
</tbody>
</table>

*Adapted from Bigby and Wasserman4 and Blake.21

Figure. **Model of the pathogenesis of asthma***

- **Allergic**
  - Dust
  - Pollen
  - Animal dander
  - Molds
  - Medications
  - Generation of immunoglobulin E
  - Inflammatory cell chemotaxis
  - Release of biochemical mediators from proinflammatory cells
  - Leukotrienes
  - Histamine
  - Prostaglandins F₂α, D₂
  - Thromboxane
  - Platelet-activating factor
  - Proteases

- **Idiosyncratic**
  - Respiratory infection
  - Environmental pollution
  - Cold air, exercise
  - Airway instrumentation or irritation
  - Immune response
  - Inflamed, hyperirritable airways
  - Bronchospasm
  - Microvascular leakage
  - Edema
  - Mucus secretion
  - Denuded epithelium
  - Neurogenic response


at night or early morning)

4. Labored respirations with accessory muscle use
5. Tachypnea (a respiratory rate of > 30 breaths per minute and a heart rate of 120 suggests severe bronchospasm)
6. Chest tightness
7. Prolonged expiratory phase of respiration
8. Fatigue

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Dioxide elimination is relatively well preserved until ventilation/perfusion abnormalities are severe. An increased arterial carbon dioxide tension may indicate impending respiratory failure in a patient with acute asthma. Chronic asthma eventually may lead to irreversible lung destruction, loss of lung elasticity, pulmonary hypertension, and lung hyperinflation.

- **Anesthetized patient.** In the anesthetized patient, prominent manifestations of the asthmatic episode are wheezing, mucus hypersecretion, high inspiratory pressures, a blunted expiratory carbon dioxide waveform, and hypoxemia. Mechanical ventilation and positive airway pressure are associated with a higher incidence of air trapping and lung hyperinflation, and the associated barotrauma can result in a pneumothorax. In addition, alveolar overdistention may lead to decreased venous return and diminution of cardiac output. The combination of impaired ventilation and hypoxia can precipitate increased pulmonary

<table>
<thead>
<tr>
<th>Step 1</th>
<th>Mild intermittent asthma</th>
<th>Signs and symptoms ≤2 times per week</th>
<th>Short-acting bronchodilator, as needed: inhaled β₂ agonists are the first-line selection</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Generally asymptomatic with normal peak flow between exacerbations</td>
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<tr>
<td></td>
<td></td>
<td>Exacerbations brief, although intensity may vary</td>
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<tr>
<td></td>
<td></td>
<td>Nighttime symptoms occur ≤2 times per month; FEV₁ ‡ or PEFR ‡ ≥80% of predicted value</td>
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</table>

| Step 2 | Mild persistent asthma | Signs and symptoms more than twice per week but less than once per day | Long-term anti-inflammatory medication: inhaled corticosteroid (low dose) |
|        |                        | Exacerbations may affect activity | Cromolyn or nedocromil, particularly in children |
|        |                        | Nighttime symptoms occur more than twice per month | Sustained-release theophylline is an alternative |
|        |                        | FEV₁ or PEFR to 80% or more of predicted value | Zafirlukast or zileuton may be considered for patients ≥12 years old |

| Step 3 | Moderate persistent asthma | Daily symptoms | Long-term control medications: medium-dose inhaled corticosteroids or low- to medium-dose inhaled corticosteroids plus long-acting bronchodilator (inhaled or oral β₂ agonist, sustained-release theophylline), especially for nocturnal symptoms |
|        | Daily use of short-acting β₂ agonist | Exacerbations that affect activity occur ≥2 times a week and may last for days | FEV₁ or PEFR to 80% or more of predicted value |
|        | Nighttime symptoms occur more than once per week | Sustained-releasetheophylline | Zafirlukast or zileuton may be considered for patients ≥12 years old |
|        | FEV₁ or PEFR 60% to 80% of predicted value |                                |                                |

| Step 4 | Severe persistent asthma | Continuous signs and symptoms, frequently exacerbated | High-dose inhaled corticosteroids |
|        | Frequent nighttime symptoms | Long-acting bronchodilators, as indicated in step 3 |                                |
|        | Limited physical activity | Systemic corticosteroids (eg, prednisone) |                                |
|        | FEV₁ or PEFR ≤60% of predicted value | **Notes:** Adapted from National Heart, Lung, and Blood Institute and from Galant SP. Pharmacotherapeutic options for the management of asthma: Formulary. 1998;33:343-356; and Kaiser Permanente Care Management Program. Asthma Successful Practice Guidelines, Pasadena, Calif: Kaiser Permanente; 2001:1-14. For all severity steps, β₂ agonists are used for quick relief of acute symptoms. FEV₁ indicates forced expiratory volume in 1 second; PEFR indicates peak expiratory flow rate. |

The clinical classification of asthma is given in Table 2. Typically, most attacks are short-lived, lasting minutes to hours. Between attacks the person with asthma may be entirely symptom-free; however, underlying airway remodeling is still evident. Severe obstruction persisting for days or weeks is known as status asthmaticus. Use of accessory muscles of respiration and the increased work of breathing associated with a protracted asthmatic episode may result in respiratory muscle fatigue and respiratory failure. During exacerbations, pulmonary function tests may reflect acute expiratory airflow obstruction. Viscid mucus secretion may compound the airway narrowing and produce airway collapse.

The asthmatic episode produces not only airflow obstruction but also gas exchange abnormalities. The resulting low ventilation/perfusion state produces arterial oxygen desaturation. Hypoxemia is common, but in most patients with acute bronchospasm, carbon
vascular resistance, enhanced right ventricular afterload, and, finally, hemodynamic collapse.

The onset of an asthmatic episode may occur abruptly in the surgical patient. Airway manipulation, acute exposure to allergens, or the stress of surgery can provoke wheezing in a patient who previously was asymptomatic. The lability of the disease makes assiduous observation crucial.

Wheezing often suggests potentially reversible bronchoconstriction, but the extent or degree of wheezing is a notoriously poor indicator of the degree of airway obstruction. In addition, care must be taken to differentiate wheezing of asthmatic origin from other causes of wheezing, such as pneumothorax, endotracheal tube obstruction, endobronchial intubation, anaphylaxis, pulmonary edema, or pulmonary aspiration.

Treatment

Drugs from several categories, given alone or in combination, are used for asthma therapy and are listed in Table 3. Doses and combinations are chosen according to the severity of symptoms. Inhaled β2 agonists are the most effective agents for treatment of acute bronchospasm, and inhaled corticosteroids are most important for treatment of any form of persistent asthma. In cases of mild intermittent asthma, the occasional use of an inhaled β2 agonist may suffice. In more persistent mild to moderate asthma, defined by β2 agonist use more than twice a week, an inhaled corticosteroid should be added. The leukotriene modifiers, mast cell degranulation inhibitors, and theophylline are reserved for prophylactic long-term therapy, as they have little efficacy for treatment of acute symptoms. A suggested asthma treatment plan based on symptom severity is given in Table 2.

Anesthesia management

Several important anesthetic considerations and risk-reduction strategies have been reported.

- Preoperative considerations and strategies. A careful preoperative history and physical examination are essential to discern the current disease status and medication profile. Frequent nocturnal awakenings from respiratory difficulty, recent increases in medication use, and signs of viral infection may signal an increased likelihood of intraoperative difficulties.
patient undergoing an elective procedure who has significant respiratory symptoms, the procedure should be postponed and the patient’s condition should be normalized as much as possible.34

The predictive value of routine pulmonary function testing remains controversial.32,35,36 Forced expiratory volume and peak expiratory flow rate, which can be measured with inexpensive handheld devices, may be helpful for assessing current respiratory status. Values that fall 30% to 50% below the expected baseline represent a moderate episode of bronchoconstriction. Values below 50% of normal indicate a severe episode.32

Pretreatment with systemic corticosteroids has been advocated for patients with asthma who are undergoing surgical procedures. Kabalin and associates37 studied the administration of corticosteroids to asthmatic surgical patients. Of the 89 subjects in the study, 86 had no postoperative wheezing when given either prednisone or hydrocortisone preoperatively.37 Complications of corticosteroid therapy, such as delayed wound healing, infection, and adrenocortical insufficiency were not noted. Ensuring that a patient who is currently receiving inhaled or systemic corticosteroids receives them immediately before surgery is a prudent course.

Routine preoperative medications should be given to allay anxiety. The anticholinergics atropine and glycopyrrolate exhibit mild bronchodilating effects, and, as noted earlier, people with asthma experience increased parasympathetic tone. They are most effective as prophylactic drugs given 20 to 30 minutes preoperatively rather than for treatment of an acute episode.38 Caution must be exercised when administering narcotics to patients whose respiratory difficulties are evident or when using narcotics associated with histamine release, such as morphine. Fentanyl and the other phenylpiperidine analogues commonly used in anesthesia have been widely used and are safe.32,38,39 The use of histamine 2 receptor blocking agents, such as cimetidine and ranitidine to reduce gastric volume and acidity, should be avoided.32,38 Bronchospasm following their use has been reported, possibly due to loss of inhibitory feedback control via presynaptic histamine 2 autoreceptor blockade resulting in increased histamine release. Some suggested risk-reduction strategies for use in the perioperative period are listed in Table 4.

- Intraoperative considerations and strategies. Despite a lack of definitive controlled clinical studies, regional anesthesia generally is thought to be safer than general anesthesia.32,40,41 Spinal or epidural anesthetic levels to the midthoracic area or higher, however, decrease functional residual capacity, expiratory reserve volume, and the ability to cough and should be avoided.32

All of the common induction drugs, thiopental, propofol, etomidate, and ketamine have been used successfully in patients with asthma; however, some differences exist. Ketamine is the only induction drug with bronchodilating properties that makes it the agent of choice for a patient with active asthmatic symptoms requiring emergency surgery. Thiopental and the other barbiturates may cause histamine release in a small percentage of patients; however, when higher doses are used, the rapidly produced deep levels of anesthesia are a primary factor in blunting this problem. Pizov et al42 compared thiopental, thiamylal, methohexital, and propofol in a double-blind, randomized study in patients with and without asthma. None of the patients with asthma who received propofol exhibited wheezing 2 and 5 minutes after intubation. This is contrasted with wheezing after intubation in 26% to 43% of the patients who received 1 of the 3 barbiturates. These authors suggested that propofol is advantageous for routine induction in patients with asthma. Generic propofol contains sulfites, however, and use should be avoided for sulfite-sensitive patients.

The potent inhalation agents produce bronchial relaxation, and all have been used successfully in patients with asthma following administration of an intravenous induction drug. Isoflurane and desflurane, however, are both mild respiratory irritants, which may be a consideration during emergence. It is common practice to blunt this effect with the administration of opiates. Although halothane has been shown to have the most prominent

### Table 4. Risk-reduction strategies for anesthetizing patients with asthma*

<table>
<thead>
<tr>
<th>Preoperative</th>
<th>Intraoperative</th>
<th>Postoperative</th>
</tr>
</thead>
<tbody>
<tr>
<td>Encourage cessation of cigarette smoking for at least 8 weeks.</td>
<td>Limit duration of surgery to less than 3 hours.</td>
<td>Encourage deep-breathing exercises or incentive spirometry.</td>
</tr>
<tr>
<td>Aggressively treat airflow obstruction.</td>
<td>Use regional anesthesia when possible.</td>
<td>Use continuous positive airway pressure.</td>
</tr>
<tr>
<td>Administer antibiotics and delay surgery if respiratory infection is present.</td>
<td>Avoid the use of long-acting neuromuscular blocking agents.</td>
<td>Use intercostal nerve blocks and local anesthesia infiltration of incisional area for pain when appropriate.</td>
</tr>
</tbody>
</table>

*Adapted from Hurford32 and Smetana.34

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The potent inhalation agents produce bronchial relaxation, and all have been used successfully in patients with asthma following administration of an intravenous induction drug. Isoflurane and desflurane, however, are both mild respiratory irritants, which may be a consideration during emergence. It is common practice to blunt this effect with the administration of opiates. Although halothane has been shown to have the most prominent
bronchodilatory effects, its use is rapidly being supplant by the newer sevoflurane for inhalation induction in children. Several authors have noted sevoflurane's safe administration in these situations. Other anesthesia-related medications that should be avoided for patients with asthma include atracurium, mivacurium, and curare due to histamine release and esmolol and labetalol, which as β receptor blockers, produce bronchoconstriction. Many practitioners avoid long-acting muscle relaxants and the associated possibility of residual muscle weakness in people with asthma. Ketorolac and other nonsteroidal anti-inflammatory agents should be avoided in patients with aspirin-intolerant asthma.

If an episode of bronchospasm occurs during anesthesia, the following steps are recommended. (1) deep anesthetic level with a volatile agent, ketamine, propofol, intravenous lidocaine, or a combination that rapidly increases anesthetic depth; (2) administer 100% oxygen; (3) administer a β2 agonist; (4) in severe cases, give epinephrine intravenously or subcutaneously; (5) administer intravenous corticosteroids; and (6) if long-term postoperative mechanical ventilation is planned, consider intravenous aminophylline. It has little efficacy for the treatment of acute bronchoconstrictive episodes.

A strategy for mechanical ventilation that avoids lung hyperinflation and barotrauma, while allowing for longer expiratory times, should be chosen. A reduction in minute ventilation, by limiting inspiratory times and prolonging expiratory times, and moderate permissive hypercapnia have been suggested. 

• **Emergence.** The primary issue during emergence is when to extubate. Some authors suggest deep extubation to avoid the mechanical stimulation from the endotracheal tube on awakening. Others fear that the loss of a secure airway before patient awakening may present a greater difficulty than the presence of the endotracheal tube. Either way, a judgment must be made about when to extubate the patient with the understanding that the earliest possible time is advantageous to prevent mechanical bronchial stimulation. Administration of intravenous lidocaine and small non–respiratory depressing doses of opiates may help diminish airway sensitivity.

The use of anticholinesterase reversal agents also has been an area of concern. If their administration can be avoided by the use of short-acting nondepolarizing muscle relaxants, this should be considered. When they are necessary, a small increase in the coadministered dose of atropine or glycopyrrolate to ensure total blockade of parasympathomimetic side effects is suggested.

• **Asthma and the pregnant surgical patient.** Treatment for the pregnant patient with asthma undergoing surgery generally is the same as for the nonpregnant

### Table 5. Anesthetic considerations for the pregnant patient with asthma

<table>
<thead>
<tr>
<th>Step</th>
<th>Consideration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Avoid nonemergency surgery until after delivery.</td>
<td></td>
</tr>
<tr>
<td>Postpone semiemergency surgery until after the first trimester.</td>
<td></td>
</tr>
<tr>
<td>Administer higher than normal oxygen concentrations.</td>
<td></td>
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<tr>
<td>Use antacids but not histamine 2 receptor blockers for gastric preparation.</td>
<td></td>
</tr>
<tr>
<td>Use uterine displacement, generous fluid replacement, compression stockings, or leg elevation to minimize hypotension.</td>
<td></td>
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<tr>
<td>If a vasopressor is necessary, use ephedrine.</td>
<td></td>
</tr>
<tr>
<td>Avoid using nitrous oxide.</td>
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</tr>
<tr>
<td>Shield the fetus from radiographic exposure when possible.</td>
<td></td>
</tr>
<tr>
<td>Use inhaled but not parenteral β2 receptor agonists.</td>
<td></td>
</tr>
<tr>
<td>Use fetal monitoring and ultrasound when feasible.</td>
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</table>

patient. Inhaled β agonists, corticosteroids, and theophylline have been used safely in pregnant patients; however, parenteral β agonists should be avoided. Maintaining proper maternal oxygenation is essential to the health of the fetus. Some anesthetic considerations for the pregnant asthma patient are given in Table 5.

**Summary**

Anesthetists frequently encounter patients with asthma, and reactions during anesthesia in this patient group are a continual cause for concern. Thorough understanding of current treatment options, proper preoperative assessment and planning, intraoperative vigilance, prompt recognition, and early treatment of asthmatic episodes are of proven value in moderating morbidity and mortality.

**REFERENCES**
