The Bronchospastic Patient

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I. INCIDENCE

A. Prevalence and mortality
Affects 14.9 million people in U.S.
Causes 500,000 hospitalizations and > 5,500 deaths annually
Costs $11.3 billion annually
Low socioeconomic status/blacks at higher risk


B. Occurrence under Anesthesia

One case per 634 anesthetics in a retrospective Scandinavian study (Olsson, 1987). These events were serious enough to note in the medical record.
Most cases were unpredictable and had no known history of allergy or asthma prior to the anesthetic.
Triggering factor was most often mechanical (bronchoscopy, mediastinoscopy).


ASA Closed Claims Project Database – 88 claims (2.5%) in which bronchospasm was the damaging event or mechanism of patient injury (3,533 closed claims between 1975-1994)
28 (32%) had history of asthma; 10 (11%) had history of COPD.


Overall, the incidence of perioperative pulmonary complications in asthmatic patients appears to approach that of the general population. In a retrospective study by Warner et al., 1.7% of asthmatic patients (n = 708) had bronchospasm during surgery or in the PACU. An additional 2.4% received supplemental bronchodilators.


II. PATHOPHYSIOLOGY

A. Mechanisms in asthma and bronchoconstriction

Asthma- is an episodic, variable airflow obstruction with increased responsiveness of the airways to a variety of stimuli. Asthma is associated with marked submucosal inflammation.

1. Trigger (antigens and nonspecific stimuli)
2. Increased cellular activity
   Mast cell degranulation
   Eosinophils, macrophages
3. Mucosal edema; increased vascular permeability
4. Mucus secretion and cellular infiltrates
5. Airway smooth muscle contraction secondary to mast cell-derived mediators
6. Airway remodeling and fibrosis

B. Physiologic Effects

1. Airway narrowing.
2. Dynamic hyperinflation and auto-PEEP; increased RV.
3. Local hypoxemia and HPV.
4. V/Q mismatch; potential worsening of hypoxemia with vasodilators.
5. Dynamic hyperinflation and decreased venous return.

III. PREOPERATIVE ASSESSMENT

A. Signs and symptoms

Certain aspects of the patient’s history suggests an increased risk of perioperative complications:

1. Frequent nocturnal awakenings from asthma (increased airway hyper-responsiveness).
2. Frequent or continuous use of systemic corticosteroids.
3. Recent hospitalizations or emergency room visits because of asthma.
5. Large amounts of sputum production.
7. Alternative diagnoses (e.g., pulmonary edema, COPD, anaphylaxis, foreign body, airway obstruction).

B. Spirometry

Forced expiratory volume (FEV1), and/or peak expiratory flow rate (PEFR) compare with the patient’s best value in the recent weeks:

80-100% of baseline: Normal
50-80% of baseline: Moderate exacerbation
< 50% of baseline: Severe episode


IV. THERAPY FOR BRONCHOSPASM

A. Beta-2-adrenergic agonists

Treatment of choice for bronchospasm:
• Directly relaxes bronchial smooth muscle
• Decreases vascular permeability
• May modulate mediator release from mast cells and basophils

Choices:
• Inhaled agonists (Albuterol, salbutamol, etc.).
• No clear advantage to levalbuterol (isomer of albuterol).
• Subcutaneous epinephrine (0.3 mg subcutaneously at 20 min intervals).
• Intravenous epinephrine (caution).
• Terbutaline (0.25 mg subcutaneously).


B. Concomitant use of beta-blocking agents

The use of beta-adrenergic antagonists may precipitate or worsen bronchospasm in patients with reactive airway diseases. The incidence of this complication is reduced but not eliminated when «cardioselective» beta blockers are used. Nevertheless, when β-1 adrenergic blockade is desirable in a patient with bronchospasm, the use of a more selective agent such as labetalol or esmolol is reasonable. These agents are unlikely to produce clinically significant worsening of pulmonary function.


C. Anticholinergics

Bronchoconstriction, especially secondary to mechanical manipulation of the airway, can occur via cholinergically mediated pathways. Anticholinergic drugs cause bronchodilation directly and blunt bronchoconstriction resulting from cholinergically-mediated triggers.

Ipratropium (Atrovent) is an inhaled atropine derivative available as a metered-dose inhaler or as a solution for nebulization. Atrovent causes bronchodilation and enhances the action of β-2-adrenergic agents. Inhaled atropine and glycopyrrolate are also effective, used frequently, but are not FDA approved. Glycopyrrolate has the advantage of few, if any, systemic side-effects.


D. Corticosteroids

Glucocorticoids are useful in patients with asthma and chronic obstructive lung disease who have not responded adequately to β-2-adrenergic agonists. While the reason for the efficacy of steroids is unclear, possible mechanisms of action include reduction of inflammation and histamine release and inhibition of arachidonic acid metabolism. Steroids may also increase the sensitivity of the airway to bronchodilation by β2-adrenergic agents and reduce airway hyperreactivity induced by propranolol.

For severe acute exacerbations, the equivalent of 60 to 80 mg of methylprednisolone can be administered intravenously every 6 to 8 hours and then tapered. For chronic administration, alternate-day dosage schedules or inhaled steroids, which reduce the incidence and severity of side effects, are often effective.

Patients who have received steroids within the last six months are assumed to have suppressed adrenal cortical function and probably should receive glucocorticoid coverage on the day of surgery. The equivalent of 100 mg hydrocortisone every 8 hours is traditional, but usually represents an overdosage. Postoperatively, steroids should be discontinued or quickly tapered.


E. Leukotriene receptor antagonists

Montelukast (Singulair 10 mg po qd) - leukotriene receptor (CysLT1) antagonist Inhibits activity of LTD4. For long-term treatment of mild to moderate asthma. Not for acute exacerbations asthma or bronchospasm. Continue perioperatively.
Metabolized by cytochrome P450 enzymes. Rarely, systemic eosinophilia and vasculitis (Churg-Strauss syndrome) may occur.

F. Magnesium


A Cochrane review concluded that IV magnesium sulfate improved pulmonary function and reduced hospital admissions, especially for patients with severe asthma.

Inhaled magnesium sulfate, nebulized and co-administered with albuterol, may also improve pulmonary function in acute asthma.


A randomized placebo controlled trial of 40 mg/kg iv magnesium sulfate for moderate to severe asthmatic exacerbation in pediatric (6 to 18 yr old) ER patients demonstrated improved PFT’s at 20 and 100 minutes; patients treated with magnesium were more likely to be discharged home (8/16 vs 0/14).


G. Theophylline

Limited usefulness in the acute setting; less potent bronchodilator than adrenergic drugs; and toxic. In chronic asthma and COPD, theophylline decreases the frequency and severity of asthmatic symptoms and decreases steroid requirements in some patients. Its true mechanism of action is unknown. Theophylline inhibits cyclic AMP phosphodiesterase activity only at toxic concentrations. Alternative explanations are that theophylline acts as an adenosine receptor antagonist or decreases the availability of intracellular calcium.

At least in dogs, theophylline produces bronchodilation by increasing the release of endogenous catecholamines. Halothane blocks this effect.

No longer recommended for acute therapy.


A. Conservative measures

1. Avoid triggers.

Environmental allergens and irritants (endotracheal intubation, cold air, latex products) may cause direct bronchoconstriction. Preoperative exposure to known irritants should be minimized. Ideally, elective surgery should be postponed during acute exacerbations. Be on the lookout for latex and aspirin sensitive patients.

2. Respiratory therapy.

Asthmatics and patients with chronic obstructive lung disease often have trouble clearing secretions postoperatively. Adequate perioperative hydration and humidification of inspired gases reduce drying of the airway and improve mobilization of secretions. Cold inspired gases may directly cause bronchoconstriction.

3. Treat infections.

Eradication of acute pulmonary infections prior to surgery may reduce the likelihood of postoperative pulmonary complications. Elective surgery should be postponed until acute infections are cleared. Broad spectrum prophylactic antibiotics are not indicated, as they may produce overgrowth of resistant organisms. Instead, antibiotic therapy should be directed at infections documented by sputum Gram stain and culture.


B. Premedication

Preanesthetic medication should reduce anxiety and relieve the discomfort of transport, line placement, etc. Benzodiazepines are effective in allaying anxiety in most patients. Narcotics may be used to provide preoperative analgesia but should be titrated carefully in patients with CO2 retention. While
narcotics can cause systemic histamine release, there is no evidence to suggest that narcotics, in the doses used for premedication, cause clinically significant bronchoconstriction. Avoid aspirin and non-steroidal anti-inflammatory drugs in patients with severe asthma, nasal polyps, or aspirin sensitivity.

C. Regional anesthesia

While it seems reasonable that regional anesthesia should avoid many of the pulmonary complications of general anesthesia and reduce the incidence of postoperative pulmonary complications, this hypothesis has been difficult to prove. Local or regional anesthesia for surgery on the extremities, eye operations, and other peripheral procedures appears, in general, to be the best choice for patients with asthma. Spinal or epidural anesthesia with levels of sensory anesthesia higher than T6 decreases functional residual capacity, expiratory reserve volume, and the patient’s ability to cough. Airway reactivity, however, actually decreases, probably due to systemic absorption of the anesthetic.


D. General anesthesia

1. Induction

Induction may be performed with intravenous or inhalational agents, remembering that patients with bronchospasm have increased functional residual capacities and prolonged induction and emergence times. Evidence suggests that induction of general anesthesia with propofol greatly reduces the incidence of wheezing after endotracheal intubation, compared with barbiturate induction, in both asthmatic (0% vs 45% incidence) and non-asthmatic patients (3% vs 16% incidence). Thiobarbiturates may release histamine and could worsen bronchospasm in the asthmatic. Induction with methohexital, an oxybarbiturate, has been associated with less histamine release. Ketamine or an inhalational agent also may be used.


Intravenous lidocaine, 0.5-1.0 mg/kg, decreases airway reflexes and is a helpful adjunct to general anesthesia in bronchospastic patients.


Nebulized lidocaine can cause bronchoconstriction in some patients.


Avoid airway manipulations (tracheal intubation) when possible. Use of a laryngeal mask airway, when indicated, is an excellent alternative to endotracheal intubation in patients with increased airway responsiveness.


2. Effects of anesthetics

a. Block parasympathetic irritant pathways.

b. Direct relaxation of bronchial smooth muscle.

c. Augment β-2-adrenergic responses (ketamine).


3. Inhalational anesthetics depress airway reflexes, cause bronchodilation, and are recommended for maintenance of general anesthesia in patients with bronchospasm. Enflurane, halothane, and isoflurane and sevoflurane all have bronchodilating properties. Of the commonly used volatile agents,
average, sevoflurane appears to have the greatest effect on airway resistance.


4. Bronchospastic patients may be extubated while still under deep anesthesia, if they do not require airway protection or postoperative ventilatory support. This practice reduces reflex bronchoconstriction during emergence, since the continued presence of the endotracheal tube during light planes of anesthesia can increase airway reactivity.

5. Epidural local anesthetics and narcotics may be used to supplement a general anesthetic and provide postoperative analgesia as well.

E. Mechanical ventilation of the asthmatic

1. Controlled ventilation using slow rates and prolonged expiratory times reduces the bronchospastic lung’s tendency towards air-trapping. Monitoring chest movements, inspiratory pressure, expiratory time, the end-tidal CO2, and the expiratory CO2 waveform is invaluable.

2. “Auto PEEP” - In patients with severe obstructive disease, expiratory flow rates may be very prolonged and/or respiratory rates increased. As a result, inhalation begins before the previous exhalation is complete. This traps gas in the lungs, often under surprisingly high pressure. This «auto-PEEP», like applied PEEP, can produce profound decreases of venous return and cardiac output. In extreme cases, electro-mechanical dissociation, leading to death, may occur!


Wiener C. Ventilatory management of respiratory failure in asthma. JAMA 1993;269:2128-2131.

3. Overdistention and auto-PEEP may be reduced by limiting tidal volume and respiratory rate. Prolonged expiratory times may be required. In these cases, the PaCO2 is allowed to rise passively—a strategy called permissive hypercapnia. The goal of permissive hypercapnia is simply to avoid regional or global overdistention of the lung.


Quinlan JJ, Buffington CW. Deliberate hypoventilation in a patient with air trapping during lung transplantation. Anesthesiology 1993;78:1177-1181.

4. Some Guidelines for Mechanical Ventilation of the Asthmatic:

a. Monitor expiratory airflow (physical exam, ET-CO2, spirogram)
b. Ventilate Slowly! Decrease respiratory rate to limit auto-PEEP
c. Provide adequate time for exhalation
d. Allow PaCO2 to rise, if necessary
e. Limit peak alveolar pressure (end-insp. plateau pressure) < 30 cm H2O
f. Use low levels of PEEP (5 to 15 cm H2O)

VI. ADDITIONAL TOPICS

A. Heliox

Heliox is a blend of helium and oxygen (usually an 80:20 or 70:30 mix) which is less dense than air and can decrease airway resistance in patients with airway obstruction and asthma. While breathing heliox, inspiratory and expiratory resistance may decrease, resulting in a decrease of PaCO2. Evidence for the efficacy of heliox in the asthmatic remains largely anecdotal.


B. General Anesthesia for Treatment of Status Asthmaticus

Halothane was the classic drug of choice. The possibility of hepatotoxicity, arrhythmias and high bromide levels with prolonged administration decreases the utility of the drug. Both enflurane and isoflurane have been used to treat status asthmaticus. The main benefit of general anesthesia is sedation and the control of the patient’s respiratory pattern. Total intravenous anesthesia (propofol, ketamine, etc.) is a more acceptable and practical alternative.