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RESPIRATION AND THE AIRWAY

Perioperative considerations for the patient with asthma and bronchospasm

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The incidence of asthma is increasing worldwide, but morbidity and mortality are decreasing because of improvements in medical care. Although the incidence of severe perioperative bronchospasm is relatively low in asthmatics undergoing anaesthesia, when it does occur it may be life-threatening. The keys to an uncomplicated perioperative course are assiduous attention to detail in preoperative assessment, and maintenance of the anti-inflammatory and bronchodilatory regimens through the perioperative period. Potential trigger agents should be identified and avoided. Many routinely used anaesthetic agents have an ameliorative effect on airway constriction. Nonetheless, acute bronchospasm can still occur, especially at induction and emergence, and should be promptly and methodically managed.

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Asthma is a disorder of variable intensity, typified by sentinel symptoms, airway obstruction, inflammation, and hyperresponsiveness.[1] Worldwide, this condition is estimated to occur in 300 million persons and is implicated in one of every 250 deaths;[38] its prevalence in the USA is 6.7% of the population.[2] The asthmatic patient undergoing surgery is at risk for perioperative morbidity and mortality. A large retrospective review revealed that the incidence of intraoperative bronchospasm and laryngospasm was surprisingly low, ~1.7%, although complications were more frequent in older patients and those with active asthma.[61] A large closed-claim analysis further revealed that although bronchospasm comprised only 2% of the database, ~90% of the claims involved severe brain injury or death.[9] In short, intraoperative bronchospasm—which is most likely to occur at induction—is an uncommon but potentially devastating complication of anaesthesia.

A history of asthma has several implications in the perioperative setting. The patient may present for an anaesthetic poorly optimized, particularly in the setting of urgent or emergent surgery. Because of airway hyperreactivity, bronchospasm may readily be precipitated by instrumentation, a variety of drugs, and perioperative complications such as aspiration, infection, or trauma. Emergence from anaesthesia presents a constant risk of laryngospasm and bronchospasm. Pain, fluid shifts, and delayed mobilization can contribute to an increased risk of postoperative pulmonary complications in these patients. These risks are exacerbated by the co-existence of chronic obstructive pulmonary disease (COPD) or active smoking, but this review will focus on bronchospasm resulting from asthma in the adult.

Pathophysiology

Asthma implies a wide phenotype range of severity, chronicity, persistence, and response to therapy. The severity of the disease process is related to the severity of airway inflammation, which governs hyperresponsiveness, the degree of obstruction, and symptomatology. Bronchoconstriction results from contraction of bronchial smooth muscle induced by a myriad possible stimuli, including intrinsic factors, allergens, exercise, stress, or cold air (Table 1). Vagal and sympathetic factors directly modulate airway tone. Inflammatory oedema and mucous plugging exacerbate airflow limitation and progressively impair the response to bronchodilator therapy. Airway remodelling, thickening, and abnormal communications between the injured airway epithelium and the pulmonary mesenchyme confer resistance to corticosteroid therapy as well.[24] Airway smooth muscle changes have been implicated in chronic, poorly responsive bronchospastic

disease—both as a mechanical and as an inflammatory mediator.[56]

The immunologic-inflammatory pathways involved in the pathogenesis of asthma are complex and include lymphocytes (both Th1 and Th2), immunoglobulin E, eosinophils, neutrophils, mast cells, leucotrienes, and cytokines. These pathways are triggered and modified by extrinsic and environmental factors such as allergens, respiratory infections, smoke, and occupation-related exposure.[1 7] Thus, asthma ultimately represents a dynamic interaction between host and environmental factors (Fig. 1).

Cardiopulmonary effects of bronchospasm

Progressive acute bronchoconstriction rapidly leads to increased work of breathing (WOB), decreased airflow, air trapping, dynamic hyperinflation, ventilation–perfusion (V/Q) mismatch, increased pulmonary vascular resistance (PVR), and right ventricular overload.[25] The forced expiratory volume in the first second of expiration (FEV_1) is substantially decreased during active bronchospasm. The forced vital capacity (FVC), expiratory reserve volume (ERV), inspiratory capacity, and forced expiratory flow between 25% and 75% of the FVC ($FEF_{25-75\%}$) are also decreased, whereas residual volume, functional residual capacity, and total lung capacity are increased. All these indices can return towards normal between bronchospastic attacks. The dynamic compliance of the lungs decreases because of air trapping, and accessory muscles (scalene and sternocleidomastoid) are recruited to preserve the tidal volume (V_t) despite increased ERV. Soon, intrinsic and extrinsic recoil of the lungs is unable to overcome airway obstruction, necessitating active exhalation using intercostal, abdominal, and diaphragmatic muscles. The WOB progressively increases, together with oxygen consumption (VO_2) and carbon dioxide production (VCO_2), whereas the O_2 supply and CO_2 elimination diminish. Ultimately, this may result in myocardial oxygen imbalance (discussed later).

Table 1 Triggering factors for bronchospasm. Adopted from Expert Panel Report 3 (EPR-3)[1] and Hepner and Castells[23]

Native
Increased secretions
Vagal-sympathetic tone imbalance
Acute respiratory infection
Exercise
Environmental
Pollen and dust
Animal dander (dog, cat, dust mite, cockroach)
Cleaning and industrial chemicals
Tobacco
Air pollution
Cold air
Medications
Neuromuscular blocking agents
Antibiotics
β-Blockers
Protamine
Non-synthetic opioids
Drug preservatives
Ester local anaesthetics
Hospital materials
Latex
Invasive ventilatory devices

In severe bronchospasm, blood gas abnormalities occur as a consequence of V/Q mismatch.[60] Variable airway obstruction leads to areas of hyperinflation and hypoinflation, which can be matched initially by hypoxic pulmonary vasoconstriction (HPV).[21] However, inflammatory (and therapeutic) vasodilation soon reverses HPV and hypoxaemia results. In the early stages of bronchospasm, hyperventilation leads to respiratory alkalosis. Increasing air trapping and respiratory muscle fatigue lead to increasing CO_2 retention; 'normalization' of the Pa_{CO_2} actually reflects decompensation and indicates a need for immediate substantive therapeutic intervention. Once hypercapnia supervenes, the condition can deteriorate to status asthmaticus, with a high morbidity and mortality. Extreme dynamic air trapping may cause so much hyperinflation that cardiac filling is restricted by 'pulmonary tamponade', ultimately resulting in pulseless electrical activity.

During an acute attack, PVR increases *pari passu* with the increasing airway and alveolar pressure, and is exacerbated by hypoxaemia, acidosis, and endogenous or exogenous catecholamines. Elevated PVR in the setting of abnormal myocardial oxygen balance (see above) exerts strain on the right ventricle that can lead to acute failure, particularly in the presence of pre-existing heart disease. At the same time, central venous pressure, pulmonary artery diastolic pressure, and pulmonary artery occlusion pressure (PAOP) can be misleadingly elevated because the

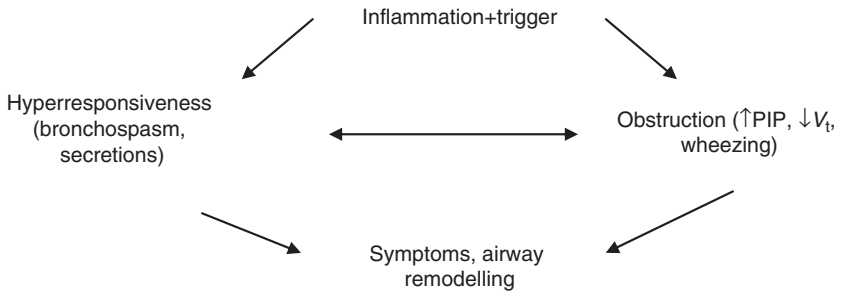

Fig 1 Pathophysiology of asthma and bronchospasm. PIP, peak inspiratory pressure, V_t, tidal volume. Adopted from Expert Panel Report 3 (EPR-3),[1] with permission from Elsevier.

high airway and intrapleural pressures are directly transmitted to the intravascular space. For example, an intrapleural pressure of +10 mm Hg can result in a PAOP reading of 20 mm Hg, whereas the effective transmural pressure is actually 20−10=10 mm Hg. This disparity might become apparent only when neuromuscular block abolishes the high ventilatory muscle tone or the bronchospasm resolves.

Therapeutic considerations

Historically, medications for asthma have been classified according to mechanism and target. More recently, a short- *vs* long-term relief schema has become popular, particularly as newer drugs defy the old classification.[13] This paradigm is considered to be more amenable to patient compliance and simplifies the perioperative approach to therapy (Table 2).

Quick-acting drugs

Quick-acting β_2-selective adrenergic agonists provided by metered-dose inhalers (MDIs) are the mainstay for fast relief of bronchoconstriction. Examples of this class of drugs are albuterol (salbutamol), levalbuterol (levosalbutamol), and pirbuterol. Their onset of action occurs within 5 min, peak effect is within 1 h, and their duration of action is 4−6 h. Patients with poor inhalation technique should use a valved holding chamber (spacer). These drugs are recommended only for short-term relief of symptoms or before known triggers such as exercise; β_2-agonists are not contraindicated in patients taking β-blockers for cardiac disease. Side-effects such as tremor, anxiety, palpitations, and tachycardia occur but are not common at standard doses. Levalbuterol, the *R*-enantiomer of albuterol, has been touted to have fewer side-effects than its parent, but differences in tachycardia have not been observed in critical care patients.[33] Hypokalaemia and hypomagnesaemia occasionally result from stimulation of the Na^+/K^+-ATPase cellular pump (and can be an effective therapeutic modality in acute renal failure). Paradoxical bronchospasm has been reported with excessive use of both albuterol and levalbuterol.[47] Parenteral administration of short-acting β_2-agonists is discouraged because of slow onset time, diminished potency, and considerably greater systemic adverse effects.

Long-acting drugs

Long-acting β_2-selective agonists, such as arformoterol, formoterol, and salmeterol, provide bronchodilation for >12 h and are largely free of side-effects. Unfortunately, some of the bronchoprotective effect of the long-acting β_2-selective agonists decreases with time; short-acting β_2-selective agonist effect is not blunted by this phenomenon. The long-acting agents do not suppress inflammation and should not be used without anti-inflammatory treatment for the control of asthma.

Table 2 Therapeutic options in asthma and bronchospasm. Adapted in part from Fanta[13]

Drug type	Mechanism of action	Examples	Comment
Acute therapy (treatment of acute bronchospasm in the operating theatre or intensive care unit)			
Inhaled short-acting β-agonist	β_2-Agonist	Albuterol, levalbuterol, pirbuterol	Not for maintenance therapy
Parenteral corticosteroids	Multiple anti-inflammatory effects	Methylprednisolone, hydrocortisone, prednisone	For difficult to control exacerbations
Volatile anaesthetic agents	Multiple pathways including smooth muscle dilation, vagal block, and anti-inflammatory actions	Isoflurane, sevoflurane, halothane	Desflurane can worsen bronchoconstriction in smokers
Ketamine	Sympathomimetic and endothelin pathway		Increases secretions
Magnesium	Smooth muscle relaxation		No standard dose; can cause weakness and CNS depression
Helium−oxygen mixture	Reduces inhaled gas viscosity and improves laminar flow	Heliox	$F_{I_{O_2}}$ is <1.0; does not bronchodilate; several helium:oxygen ratio mixtures available
Chronic (maintenance) therapy			
Inhaled long-acting β-agonist	β_2-Agonist	Salmeterol, formoterol	Appropriate for maintenance therapy typically with inhaled corticosteroid; formoterol has more rapid onset of action
Inhaled corticosteroids	Multiple anti-inflammatory effects	Beclomethasone, budesonide, fluticasone, triamcinolone	Primarily for maintenance therapy in persistent asthma
Leucotriene modifiers	Inhibit leucotriene pathway	Montelukast, zafirlukast, pranlukast, zileuton	Not for acute treatment of bronchospasm
Inhaled anticholinergic agents	Muscarinic receptor block	Ipratropium	Onset delayed by 20 min; more appropriate for COPD but can be used for severe asthma or β-agonist intolerant patients
IgE immunotherapy	Binds IgE and reduces circulating levels	Omalizumab	For chronic asthma refractory to standard treatment

Inhaled and parenteral corticosteroids

Inhaled corticosteroids, for example, beclomethasone, budesonide, fluticasone, and triamcinolone, are the cornerstone of therapy to stabilize and improve persistent asthma. Their consistent use has probably contributed to the decreased morbidity and mortality observed in asthma, at the same time that the disease has become more widespread. Some formulations combine a steroid and a long-acting β_2-selective agonist, for example, budesonide–formoterol and fluticasone–salmeterol. Nonetheless, inhaled corticosteroids are suppressive rather than curative. No clinically important adrenal suppression has been found with their administration in low to moderate doses. No significant therapeutic differences appear to exist between different formulations.

Parenteral steroids remain a mainstay of the treatment of acute asthma. However, their beneficial effect on airway mechanics can take 4–6 h in acute bronchospasm, a noteworthy point in the poorly controlled asthmatic requiring urgent or emergent surgery.[14] Adrenal suppression, infection, delayed healing, hyperglycaemia, and fluid retention are common complications of prolonged therapy. However, delayed wound healing and increased infection have not been observed in asthmatic patients treated with perioperative systemic steroids.[28 44] Patients who have been taking systemic corticosteroids for >2 weeks during the prior 6 months should be considered at risk for adrenal suppression in the setting of severe acute disease, trauma, or major surgery.

Leucotriene modifiers

Leucotriene modifiers, such as montelukast, zafirlukast, pranlukast, and zileuton, inhibit the leucotriene pathway, a mediator of bronchoconstriction. Evidence of their beneficial effect on inflammation is conflicting. They are not useful for acute treatment of bronchospasm. Zileuton use has been associated with hepatic injury, and leucotriene-receptor antagonist use with Churg–Strauss vasculitis, especially when steroids dosage is decreased. In short, leucotriene modifiers are second-line to steroids in the management of chronic asthma.

Anticholinergic agents

Anticholinergic bronchodilators such as ipratropium have a more limited role in the therapy of acute asthmatic bronchospasm than β_2-selective agonists. They act by inhibiting cyclic guanosine monophosphate formation and block vagus nerve-mediated bronchoconstriction, which is an important component of bronchospasm in patients with COPD. Indications for their use include, as implied, patients with COPD, but also patients who are intolerant of β_2-selective agonists, or who are severely asthmatic, or have β-blocker-induced bronchospasm. Inhaled glycopyrrolate can be bronchoprotective in asthma and COPD.[19] Anticholinergic agents dry airway secretions. Although controversy exists as to whether this improves inflammation or worsens inspissation, it certainly decreases airway hyperresponsiveness, an important consideration for the anaesthetist.

Other therapies

Mucolytic agents such as *N*-acetylcysteine are not recommended. Not only do they increase secretions, which worsen airway hyperreactivity, but they are irritating to the airway and can themselves provoke bronchospasm. Theophylline has a narrow therapeutic dose range and a wide array of side-effects and has been replaced by inhaled corticosteroids and long-acting β_2-agonists. Mast cell stabilizers such as cromolyn are of limited benefit, require several weeks for action, and have been replaced by inhaled steroids and leucotriene modifiers. Anti-IgE therapy, specifically omalizumab, is reserved for patients with moderate to severe persistent asthma who do not respond to standard treatment. It is costly and has no role in the acute management of bronchospasm.[1 13]

Preoperative evaluation

Preoperative assessment and intervention is the key to successful management of the patient with asthma. When asthma is well controlled, it probably confers no additional risk for perioperative complications; when it is poorly controlled, it almost always does.[55] Unfortunately, the nature and scope of anaesthetic practice is such that the timeframe for preoperative intervention is all too often short. Moreover, the practitioner can be misled by the variable nature of the disease. Symptoms may be completely absent before operation with no less a potential for intraoperative bronchospasm. Anti-inflammatory therapy can be suboptimal even when the diagnosis is well established.

History

Patients can be asymptomatic at the time of evaluation. However, key clues to severe disease include a history of frequent exacerbations, hospital visits, and, most importantly, prior tracheal intubation and mechanical ventilation to deal with a severe attack. Prior perioperative exacerbation of respiratory disease is also significant.[58 61] The patient should be interrogated regarding their most common triggering agents—they will usually be acutely aware of them. Type, dose, frequency, and degree of benefit of therapy provide important clues to the severity and control of the disease. This applies especially to steroid therapy: inhaled *vs* systemic use, duration of exposure, and side-effects should be elicited. Respiratory infections, including sinus infections, can trigger an asthmatic attack; thus, any recent fever, change in cough or sputum, and other evidence of respiratory infection should

raise concern. Airway hyperreactivity can remain for several weeks after resolution of infectious symptoms. Interestingly, however, a frequently cited retrospective study found no increase in incidence of intraoperative bronchospasm in the asthmatic patient with a recent upper respiratory infection.[61]

Patients with moderate or severe asthma can be provided with a peak expiratory flow rate (PEFR) meter for home assessment. The normal range (200–600 litre min^{-1}) varies widely depending on age, gender, height, and weight; what is relevant is how the PEFR varies from the patient's own baseline. The American Lung Association has suggested green (80% of usual), yellow (50–80% of usual), and red zones (<50% of usual) to alert patients about fluctuations in their condition, and when to get medical attention.

Physical examination

The preoperative physical examination should focus on detecting signs of acute bronchospasm or active lung infection (which should defer elective surgery), chronic lung disease, and right heart failure. When expiratory airflow is markedly decreased, breath sounds are diminished or inaudible. A simple screening test for prolonged exhalation is the forced expiratory time (FET), which can be assessed by listening over the trachea while the patient exhales forcibly and fully. An FET >6 s correlates with a substantially lowered FEV_1/FVC ratio and should initiate further investigation.

Investigations

Laboratory studies are guided by the history and physical examination. Formal pulmonary function tests (PFTs) help detect chronic residua of acute asthma and help stratify the severity of the disease. However, in many patients, PFTs normalize between attacks, so normal values do not guarantee an uncomplicated perioperative course.[54] Arterial blood gas (ABG) analysis is important during asthmatic attacks, but can be normal at baseline. The ECG can show right atrial or ventricular hypertrophy, acute strain, right axis deviation, and right bundle branch block. Chest radiographs reveal flattened diaphragms if the lungs are hyperinflated, and are useful to evaluate for pulmonary congestion, oedema, or infiltrate.

The anaesthetic plan

The anaesthetic plan should balance suppression and avoidance of bronchospasm with the usual goals of patient safety, comfort, and a quiet surgical field. Choice of anaesthetic method must be tailored to the patient, the procedure, clinical assessment, and the preferences of all involved. No definitive evidence shows that one method is generally superior to another. It seems prudent to avoid direct instrumentation of the airway if at all possible, but anxiety or pain during regional anaesthesia (peripheral nerve or neuraxial block) could themselves precipitate an attack of bronchospasm. Clearly, the highest risk cases are those in which the airway itself is the subject of surgery, or surgery involving the thorax or upper abdomen where tracheal intubation cannot be avoided.

Preoperative preparation

If evaluated far enough in advance, the patient should be advised to stop smoking at least 2 months before surgery.[55] This will allow the greatest recovery of endobronchial cilial mucus clearance. Lung function should be improved to baseline or to as near baseline as possible by optimizing medications and compliance, or considering a short course of oral corticosteroids. Oral methylprednisolone 40 mg for 5 days before surgery has been shown to decrease post-intubation wheezing in newly diagnosed or poorly compliant patients with reversible airway obstruction.[53] If the patient is first evaluated immediately before operation and steroids are indicated, then i.v. corticosteroids may be useful. Steroid-induced suppression of perioperative adrenal function is unlikely to occur unless the patient has been on systemic steroids for >2 weeks within the prior 6 months and is undergoing major stress or surgery. These patients should be prescribed a short-acting steroid such as hydrocortisone (e.g. 100 mg i.v. every 8 h) during the perioperative period.[1] Short-acting bronchodilator therapy given prophylactically has likely benefit; MDI or nebulizer delivery is equivalent if proper technique is used. Elective surgery should not be performed in the presence of active bronchospasm, and the cause (e.g. a new respiratory infection) and symptoms should be actively treated until the patient is back to baseline status.

An optimal premedication allays anxiety, improves work of breathing, and possibly averts the induction of bronchospasm, while eschewing oversedation and respiratory depression. No ideal drug or drug combination exists for this. The α-2 agonist dexmedetomidine has a favourable profile, including anxiolysis, sympatholysis, and drying of secretions without respiratory depression. Although there are numerous reports of its benefits in awake intubation and anaesthetic emergence,[18] there are no data on its role in the asthmatic patient. By drying secretions and suppressing upper airway vagal responses, anticholinergic agents such as atropine or glycopyrrolate can decrease airway reactivity and should be considered. The patient's own MDIs should be brought to the operating theatre (discussed later).

Choice of monitoring should be geared towards assessment of airway mechanics (volumes, pressures, airway flows, *I*:*E* ratio, compliance, respiratory waveforms if available). It is very useful to augment the end-tidal CO_2 monitoring (E'_{CO_2}) with a visible waveform so that flattening

of the capnogram can be used as an index of expiratory airway flow. However, as bronchospasm worsens, the E'_{CO_2} to Pa_{CO_2} gradient widens. Consideration should always be given to placing an arterial line in high-risk cases to facilitate ABG measurement.

Intraoperative management

Bronchospasm can be provoked by laryngoscopy, tracheal intubation, airway suctioning, cold inspired gases, and tracheal extubation. Airway tone is increased by vagal stimulation caused by endoscopy, peritoneal, or visceral stretch. Application of excessive levels of PEEP can worsen incipient or increasing air trapping.

A number of perioperative medications can induce bronchospasm through histamine release, muscarinic activity, or by provoking allergic reactions. Neuromuscular blocking agents are the most common medications to cause allergic reactions in the operating theatre.[23] Some, such as mivacurium (no longer available in the USA) and atracurium, have histamine-releasing effects. Cisatracurium does not cause histamine release or bronchospasm; rocuronium is useful for the asthmatic who needs a rapid sequence intubation.[57] Rapacuronium, a promising rapid-onset neuromuscular blocker, was removed from the market because of its muscarinic receptor-mediated bronchospastic effects.[26 27]

There is always a concern that non-synthetic opioids such as morphine can induce bronchospasm through histamine release. There are few objective data to support this, and even some evidence that morphine suppresses bronchoconstriction in mild asthmatics.[12] In general, i.v. induction agents are safe.

Propofol appears to be superior to thiopental and etomidate in constraining increases in airway resistance, but there have been case reports of its association with bronchospasm in susceptible patients.[11 29 42 45] In heavy smokers undergoing anaesthesia, a propofol formulation that uses metabisulphite induces higher airway resistance than that preserved with calcium edetate,[48] an observation that should be taken into consideration in asthmatics as well. Ketamine has excellent induction characteristics and induces bronchodilation, possibly by interfering with the endothelin pathway.[52] Given its propensity to increase secretions and cause dysphoria, antisialogogue and benzodiazepine are useful adjuncts. Like many other anaesthetic agents, the evidence in favour of ketamine is largely based on animal studies and case reports rather than randomized, controlled trials.[5 10 34 41] Lidocaine can prevent bronchospasm by attenuating sensory responses to airway instrumentation or irritation. However, inhalation of lidocaine is itself irritating and can precipitate or worsen bronchospasm.[17] Intravenous injection of lidocaine quickly achieves adequate airway anaesthesia, but even when administered by this route bronchial tone can be increased.[6 8 37]

Ester local anaesthetics carry a low but appreciable incidence of allergic reactions, whereas antibiotics frequently act as allergens. Vancomycin can induce hypotension, erythema, and bronchospasm through histamine release, the so-called 'red-man syndrome'.[59] Protamine sulphate can induce bronchospasm as a component of type I anaphylaxis. Risk is greater in patients receiving protamine–insulin who develop IgE and IgG antibodies.[62] Anaphylactic reactions to polymethylmethacrylate[63] and i.v. contrast agents[40] can cause bronchospasm. Bronchospasm is an important component to anaphylaxis induced by latex allergy.

Inhalation anaesthetic induction should be considered if circumstances allow. Sevoflurane is well tolerated as an inhalational induction agent and has good bronchodilatory effect.[50] Halothane has been favoured in the past, but now is not as available, is more blood-soluble leading to longer induction times, and in the setting of hypoxaemia or acidaemia could potentiate arrhythmias. Warm, humidified gases should be provided at all times. Rapid sequence or standard induction should be performed as indicated as long as adequate anaesthesia is assured; succinylcholine is not contraindicated.

The decision whether to intubate the trachea, provide anaesthesia by mask, or use a laryngeal mask airway (LMA) is a clinical one. However, there is evidence that tracheal intubation causes reversible increases in airway resistance not observed with placement of an LMA.[32] Inadequate depth of anaesthesia at any point can allow bronchospasm to be precipitated. Anaesthetic maintenance with a volatile agent such as isoflurane or sevoflurane confers protective bronchodilation. However, there is evidence that desflurane provokes bronchoconstriction in smokers.[15]

In selecting a ventilatory mode, attention should be given to providing an adequately long expiratory time to avoid the build-up of intrinsic or auto-PEEP. This can be facilitated by using higher inspiratory flow rates or smaller tidal volumes than usual.[30] Patients should be kept adequately hydrated as usual, but fluid overload, pulmonary congestion, and oedema can precipitate bronchospasm ('cardiac asthma').

Acute intraoperative bronchospasm

Signs of airway obstruction consistent with bronchospasm include elevation of the peak inspiratory pressure, prolonged expiratory phase, and visible slowing or lack of chest fall. The patient should be turned to 100% inspired oxygen and manual bag ventilation should immediately be instituted to directly assess compliance; the bag will not fill on exhalation if the bronchospasm is severe. The chest (and, if not accessible, the expiratory limb of the anaesthetic circuit) should be auscultated to confirm wheezing. Diminished or absent breath sounds can be an ominous sign suggesting critically low airflow. The differential diagnosis includes mucous plugging of the artificial or

native airway and pulmonary oedema. A unilateral wheeze suggests the possibility of endobronchial intubation, foreign body obstruction such as a dislodged tooth, or even a tension pneumothorax.

If none of these conditions exists, or if bronchospasm persists after they have been corrected, a treatment algorithm for acute intraoperative bronchospasm should be instituted. This should include frequent monitoring of ABGs to evaluate hypoxaemia and hypercarbia (see above). The adverse effects on the circulation can add a metabolic component to the respiratory acidosis. Hypercarbia, hypoxaemia, and acidaemia promote arrhythmias and impair the response to bronchodilator therapy.

The advantage of hand-ventilation in assessing lung compliance and exhalation has already been mentioned. It can be impossible for a conventional ventilator to achieve the rapid inspiratory flows needed to facilitate a prolonged expiratory time. Increased concentrations of a volatile anaesthetic (sevoflurane and isoflurane) are often helpful. If this is not effective, a rapid-acting β_2-selective agonist should be administered via a nebulizer or, if an MDI is available, via an airway adaptor. Because of rain-out in the tracheal tube, a considerably larger dose (8–10 puffs) must be given to achieve adequate therapeutic levels. Consideration should be given to high-dose steroids, for example, 125 mg i.v. methylprednisolone, with the understanding that it will take 4–6 h before they exert their beneficial effect. If bronchospasm remains refractory, epinephrine 5–10 mg can be administered i.v., although this has a high risk of exacerbating tachycardia and tachyarrhythmias. Alternatively, a continuous epinephrine infusion of 0.5–2 $\mu g\ min^{-1}$ in adults can provide maintenance bronchodilation with less adverse effect. Helium–oxygen mixtures (heliox) have been used to maintain laminar flow in acute bronchospasm,[31] but reports on its use perioperatively are limited.[36] A major limitation is that heliox mixtures can provide only 21–30% oxygen.[49] Helium facilitates ventilation but does not reverse the underlying bronchospasm, but it can provide an important bridge until corticosteroids take effect. Magnesium sulphate (1.2–2 g i.v.) can be helpful in difficult cases; it is cheap, available, and can suppress tachyarrhythmias. However, high doses induce muscle weakness and central nervous system depression.[3 51] Nitroglycerin has been reported anecdotally to reverse acute bronchospasm, probably through direct smooth muscle relaxation.[4 16 39]

Emergence and postoperative care

Airway obstruction, laryngospasm, bronchospasm, poor ventilation, and hypoxaemia are major hazards of the emergence phase. Suctioning of the airways must be rendered cautiously, if at all. Aspiration can not only cause injury in its own right but also trigger severe bronchospasm. Prophylactic use of anti-emetic agents, gastric motility agents, antacids, and gastric suctioning before emergence should be considered. Respiratory mechanics must be fully assessed before extubation and emergence.

If acute bronchospasm persists at the end of the case or if it has been severe, or if the patient has a difficult airway, trauma, or a full stomach, consideration should be given to a period of postoperative mechanical ventilation to avoid having to reverse neuromuscular block and to allow time for airway recovery. Repeat administration of a β_2-agonist such as albuterol before emergence is advised. When emergence does occur, it should be with adequate analgesia in place, whether i.v. or neuraxial. Dexmedetomidine can be a useful ancillary agent for the reasons already discussed. Reversal of neuromuscular block has a number of hazards. Neostigmine increases bronchospasm risk because of its muscarinic and pro-secretory effects.[22] These can be blunted by co-administration of atropine or glycopyrrolate, but the duration of action of neostigmine can outlast that of the vagolytic agent, especially in the presence of renal insufficiency.

'Deep extubation' (tracheal extubation while still deeply anaesthetized) has been practised for many years, especially in children, but it has its own inherent hazards. It mandates full reversal of neuromuscular block. Even if tracheal extubation is smooth, emergence through the arousal stage can initiate severe bronchospasm with an unprotected airway. The risk of regurgitation and aspiration is ever-present.

The keys to minimizing postoperative pulmonary complications are vigilance for bronchospasm and its causes; good pain control, be it by the neuraxial route or patient-controlled analgesia;[35] bronchodilator therapy; incentive spirometry, deep breathing exercises, and early mobilization.[46] Control of gastro-oesophageal reflux is beneficial in asthma.[20] Non-invasive positive pressure ventilation is an option in some asthmatics who have persistent bronchospasm after tracheal extubation.[43]

Summary and guidelines

The incidence of asthma is increasing worldwide, but morbidity and mortality are decreasing because of improvements in medical care. Although the incidence of severe perioperative bronchospasm is relatively low in asthmatics undergoing anaesthesia, when it does occur it can be life-threatening. The keys to an uncomplicated perioperative course are assiduous attention to detail in preoperative assessment, and maintenance of the anti-inflammatory and bronchodilatory regimen through the perioperative period. Potential trigger agents should be identified and avoided. Many routinely used anaesthetic agents have a salutary effect on airway constriction. Nonetheless, acute bronchospasm can still occur, especially at induction and emergence, and should be promptly and methodically managed.

Several guidelines on the management of asthma are available.[1 38 46] Although they focus largely on assessment

and management in the chronic outpatient setting, much of their advice also applies to the preoperative holding area, operating theatre, and post-anaesthetic care unit or intensive care unit. Given its prevalence and often subclinical nature, a routine and standard approach to acute bronchospasm enables quick response and good outcome.

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