

MANAGEMENT OF PERIOPERATIVE BRONCHOSPASM

INTRODUCTION

Bronchospasm is an abnormal contraction of the smooth muscle of the bronchi, resulting in an acute narrowing and obstruction of the respiratory airway

Bronchospasm during general anaesthesia can present in isolation or as a component of a more serious underlying pathology such as anaphylaxis. It is characterised by prolonged expiration, wheeze and increased peak airway pressures during Intermittent Positive Pressure Ventilation (IPPV). Untreated it can cause hypoxia, hypotension and increased morbidity and mortality. Suspected bronchospasm during anaesthesia should be assessed and treated promptly.

Etiology of BRONCHOSPASM

Bronchospasm and wheeze are common features of reactive airways disease. Patients with bronchial asthma and some with chronic obstructive pulmonary disease (COPD) show hyperreactive airway responses to mechanical and chemical irritants. In these groups there is a combination of constriction of bronchial smooth muscle, mucosal oedema and mucous hypersecretion with plugging.

Perioperative bronchospasm in patients with reactive airways disease is however relatively uncommon. In patients with well-controlled asthma and COPD the incidence is approximately 2%. The overall incidence of bronchospasm during general anaesthesia is approximately 0.2%.¹

Exposure to tobacco smoke, history of atopy and viral upper respiratory tract infection (URTI) all increase the risk of bronchospasm during anaesthesia. In many patients with bronchospasm during anaesthesia there may be no history of reactive airways disease. Intubation, airway foreign body, light plane of anaesthesia can provoke a broncho spasm.

Causes of wheeze during general anaesthesia
Partial obstruction of tracheal tube (including ETT abutting the carina or endobronchial intubation)
Bronchospasm
Pulmonary oedema
Aspiration of gastric contents
Pulmonary embolism
Tensionpneumothorax
Foreign body in the tracheobronchial tree

Diagnosis of bronchospasm

Key points in diagnosis

1. "tight bag"
2. Reduced or absent breath sounds
3. Fall in oxygen saturation

Bronchospasm during anaesthesia usually manifests as increased airway pressures or prolonged expiration. An associated expiratory wheeze may be auscultated in the chest or heard in the breathing circuit. Wheezing requires movement of gas through narrowed airways and so in severe bronchospasm wheeze may be quiet or absent.

Similarly, breath sounds may be reduced or absent. With IPPV, peak airway pressures are increased, tidal volumes reduced, or both. Bronchospasm is not the only cause of wheeze or increased peak airway pressures during anaesthesia. With capnography, narrowed airways and prolonged expiration result in a delayed rise in end-tidal carbon dioxide, producing a characteristic 'sharkfin' appearance. However, this is not diagnostic, representing an obstruction at some stage in the expiratory pathway. With limitation in air flow, a prolonged period of exhalation is needed for alveolar pressure to normalise. Positive pressure ventilation delivered before exhalation is complete can result in 'breath-stacking' and the development of an intrinsic (or auto) positive end-expiratory pressure (iPEEP or autoPEEP). Intrinsic PEEP can increase intrathoracic pressure, decrease venous return and impair cardiac output.

Causes of increased peak airway pressure during IPPV "TIGHT BAG"	
Anaesthetic equipment	<ol style="list-style-type: none">1.Excessive tidal volume2.High inspiratory flowrates
Airway device	<ol style="list-style-type: none">1.Small diameter tracheal tube2.Endobronchial intubation3.Tube kinked or blocked
Patient	<ol style="list-style-type: none">1.Obesity2. Head down position3. Pneumoperitoneum4.Tensionpneumothorax5. BRONCHOSPASM

DIFFERENTIAL DIAGNOSIS

Bronchospasm occurs most commonly and approximately equally during the induction and maintenance stages of anaesthesia and is less commonly encountered in the emergence and recovery stages.

Bronchospasm during the induction stage is most commonly caused by airway irritation, often related to intubation. During the maintenance stage of anaesthesia, bronchospasm may result from an anaphylactic or serious allergic reaction. Drugs (antibiotics, neuromuscular blockers), blood products (red blood cells, fresh frozen plasma) and other allergens (latex) are the agents commonly responsible. Features of allergic or anaphylactic reaction include cutaneous signs (rash, urticaria, angioedema) and cardiovascular signs (tachy/bradycardia, hypotension, circulatory collapse).

1. Mechanical obstruction

A kinked (see case report in this edition), blocked (mucous plug, cuff herniation) or misplaced (endobronchial, oesophageal) tracheal tube or occlusion in the breathing circuit can mimic severe bronchospasm. Unless rapidly recognised and corrected this can have disastrous consequences

2. Laryngospasm

This should be considered and excluded. In non-intubated patients acute laryngospasm can produce upper airway noise (usually inspiratory), reduced breath sounds and difficulty in ventilation. Laryngospasm can present with signs of airway obstruction including increased respiratory effort, tracheal tug and paradoxical movement of the chest and abdomen ('see-saw' respiration)

3. Bronchial hyperreactivity

If the patient is known to be at increased risk of bronchial hyperreactivity the suspicion of bronchospasm is increased. The main patient groups are those with reactive airways disease, especially poorly controlled asthma and COPD. Bronchial hyperreactivity is also associated with preoperative exposure to tobacco smoke, upper respiratory tract infection (URTI) and a history of atopy. Many of these factors also predispose to laryngospasm.

4. Inadequate depth of anaesthesia

Manipulation of the airway or surgical stimulation under light anaesthesia increases the risk of bronchospasm. Certain surgical procedures have highly stimulating stages that can trigger bronchospasm (and laryngospasm). Examples of these include anal or cervical dilatation, stripping of the long saphenous vein during varicose vein surgery and traction on the peritoneum. These are often predictable and can be prevented or countered by an intravenous bolus of opioid and/or anaesthetic agent such as propofol.

5. Pharmacological

Certain volatile anaesthetic agents (isoflurane, desflurane) if introduced quickly can trigger bronchospasm. IV agents including beta-blockers, prostaglandin inhibitors (NSAIDs) and cholinesterase inhibitors (neostigmine) are implicated. Histamine release (thiopentone, atracurium, mivacurium, morphine, d-tubocurarine) can also precipitate bronchospasm; care should be taken with these drugs in higher risk patients.

6. Airway soiling

Unexplained bronchospasm, especially in patients without increased risk of airway hyperreactivity, should prompt consideration of airway soiling due to secretions, regurgitation or aspiration. This is particularly true with the use of the laryngeal mask airway (LMA) but may also occur with an uncuffed endotracheal tube (ETT) or an inadequately inflated/punctured cuff. A history of gastro-oesophageal reflux or sudden coughing in a patient breathing spontaneously with an LMA should increase the suspicion of airway soiling.

PREVENTION OF BRONCHOSPASM

Patients with asthma and COPD should be thoroughly assessed and care taken to ensure they are optimised for surgery. Wheezing, cough, increased sputum production, shortness of breath and diurnal variability in peak expiratory flow rate (PEFR) indicate poor control. Recent or frequent exacerbations or admission to hospital may be an indication to postpone non-essential surgery. Patients should be advised to continue their medication until the time of surgery. Preoperative bronchodilators, inhaled or oral corticosteroids, chest physiotherapy and referral to a respiratory physician may all be appropriate.

A careful medication history should be taken with particular reference to drug sensitivities. NSAID-induced bronchospasm in adult asthmatics may be as high as 15% and so a thorough history is vital.

All patients should be counselled and encouraged to stop smoking preoperatively. Six to eight weeks of abstinence before surgery significantly reduces the risk of respiratory complications including bronchospasm.

URTI in children increases the risk of bronchospasm and so it may be necessary to postpone surgery. The complete resolution of symptoms (approximately 2 weeks) correlates well with a decreased incidence of airway hyperreactivity.

Pretreatment with an inhaled/nebulised beta agonist, 30 minutes prior to surgery, induction of anaesthesia with propofol and adequate depth of anaesthesia before airway instrumentation reduces the risk of bronchospasm.

The use of an LMA (in suitable patients) has been shown to reduce the incidence of bronchospasm compared to tracheal intubation. Regional techniques where appropriate can also avoid the need for general anaesthesia and intubation.

MANAGEMENT OF PERIOPERATIVE BRONCHOSPASM

On suspecting bronchospasm

- Switch to 100% oxygen
- Ventilate by hand
- Stop stimulation /surgery
- Consider allergy / anaphylaxis; stop administration of suspected drugs/ colloid /blood products

Immediate management; - prevent hypoxia & reverse bronchoconstriction

- Deepen anaesthesia
- If ventilation through ETT difficult/impossible, check tube position and exclude blocked/misplaced tube
- If necessary eliminate breathing circuit occlusion by using self-inflating bag
- In non-intubated patients exclude laryngospasm and consider aspiration
- DRUG THERAPY SHOULD BE STARTED IMMEDIATELY

1ST LINE DRUG THERAPY:

SALBUTAMOL:

- Metered Dose Inhaler:
 - 6-8 puffs repeated as necessary (using in-line adaptor/barrel of 60ml syringe with tubing or down ETT directly)
- Nebulised: 5mg (1ml 0.5%) repeated as necessary
- Intravenous: 250mcg slow IV then 5mcg.min⁻¹ up to 20mcg.min

2ND LINE DRUG THERAPY

Ipratropium bromide: 0.5mg nebulised 6 hourly

Magnesium sulphate: 50mg.kg⁻¹ IV over 20min (max 2g)

Hydrocortisone: 200mg IV 6 hourly

Ketamine: Bolus 10-20mg. Infusion 1-3mg.kg⁻¹.h⁻¹

IN LIFE THREATENING SITUATIONS:

Epinephrine (Adrenaline)

Nebulised: 5mls 1:1000

Intravenous: 10mcg (0.1ml 1:10,000) to 100mcg (1ml 1:10,000) titrated to response

ANTIHISTAMINE: inj.chlorpheniramine 10 mg slow IV can be given

SECONDARY MANAGEMENT

The secondary management of acute bronchospasm should provide ongoing therapy and address the underlying cause. Corticosteroids and antihistamines have a role in the secondary treatment of bronchospasm and should be given early if the problem is not settling with initial measures.

Further consideration should be given to allergy/anaphylaxis and a thorough examination made for cutaneous and cardiovascular signs.

Review the medication history and consider all drugs given in the perioperative period. Examine the patient and reconsider alternative diagnoses such as acute pulmonary oedema, tension pneumothorax, pulmonary embolism or foreign body.

If the indication for surgery is not life-threatening, consider abandoning surgery, especially if there is ongoing difficulty with ventilation, falling oxygen saturations or haemodynamic compromise. In a non-intubated patient with severe bronchospasm, it may be necessary to intubate the trachea and mechanically ventilate the lungs while therapy is initiated. Avoid of histamine releasing is important muscle relaxants

If the bronchospasm has resolved or improved with initial management, so that there is no ongoing compromise of the respiratory or cardiovascular systems, it may be appropriate to wake the patient and provide any subsequent therapy on the recovery ward.

POSTOPERATIVE CARE

With ongoing symptoms a chest radiograph should be requested and reviewed to exclude pulmonary oedema and pneumothorax. If appropriate, regular therapy (bronchodilators, corticosteroids, chest physiotherapy) should be arranged. With ongoing bronchospasm, arrangements should be made for the patient to go to a high dependency or intensive care unit.

In the event that a serious allergic or anaphylactic reaction was identified or suspected, remember to take samples for mast cell tryptase. It is the responsibility of the anaesthetist to ensure the patient is completely safe and can maintain tissue oxygenation without compromise