Update in Anaesthesia

Management of bronchospasm during general anaesthesia

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Summary

Bronchospasm is a relatively common event during general anaesthesia. Management begins with switching to 100% oxygen and calling for help early. Stop all potential precipitants and deepen anaesthesia. Exclude mechanical obstruction or occlusion of the breathing circuit. Aim to prevent/correct hypoxaemia and reverse bronchoconstriction. Consider a wide range of differential diagnoses including anaphylaxis, aspiration or acute pulmonary oedema.

INTRODUCTION

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. Ongoing management should address the underlying cause.

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 is no history of reactive airways disease.

RECOGNITION OF BRONCHOSPASM

Bronchospasm during anaesthesia usually manifests as 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 (Boxes 1 and 2). With capnography, narrowed airways and prolonged expiration result in a delayed rise in end-tidal carbon dioxide, producing a characteristic 'shark-fin' appearance (Figure 1). 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.[2]

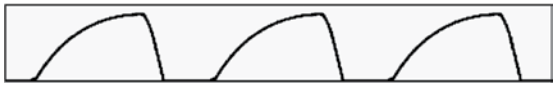

Figure 1. *The characteristic 'shark-fin' capnograph suggestive of airway obstruction*

Box 1. *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
Tension pneumothorax
Foreign body in the tracheobronchial tree

Box 2. *Causes of increased peak airway pressure during IPPV*

Anaesthetic equipment
- Excessive tidal volume
- High inspiratory flow rates

Airway device
- Small diameter tracheal tube
- Endobronchial intubation
- Tube kinked or blocked

Patient
- Obesity
- Head down position
- Pneumoperitoneum
- Tension pneumothorax
- Bronchospasm

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DIFFERENTIAL DIAGNOSIS

Bronchospasm occurs most commonly and approximately equally during the induction and maintenance stages of anaesthesia and is less often encountered in the emergence and recovery stages.[3] 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. Other features of allergic or anaphylactic reaction include cutaneous signs (rash, urticaria, angioedema) and cardiovascular signs (tachy/bradycardia, hypotension, circulatory collapse).

When assessing bronchospasm there are other important differential diagnoses and contributing factors to consider:

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. A recent death in the UK (initially treated as severe bronchospasm) was found to be due to blockage of the breathing circuit with the protective cap from an IV giving set. The subsequent Department of Health report 'Protecting the Breathing Circuit in Anaesthesia' (2004) reiterated the importance of checking the breathing circuit prior to each patient and ensuring the availability of another means of ventilation (i.e. a self-inflating bag).[4]

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).

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.

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.

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.

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 encouraged 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.[5]

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.[6]

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.[7]

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.[8] Regional techniques where appropriate can also avoid the need for general anaesthesia and intubation.

Figure 2. *Algorithm to guide management of intraoperative bronchospasm. Red numbers refer to notes over leaf.*

Management of patient with suspected bronchospasm during general anaesthesia

A

On suspecting bronchospasm

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

Difficulty with ventilation/falling SpO_2 → **CALL FOR HELP**

B

Immediate management; prevent hypoxia & reverse bronchoconstriction

- Deepen anaesthesia **1**
- If ventilation through ETT difficult/impossible, check tube position and exclude blocked/misplaced tube **2**
- If necessary eliminate breathing circuit occlusion by using self-inflating bag
- In non-intubated patients exclude laryngospasm and consider aspiration
- DRUG THERAPY; see Box D **3**

Consider transfer to HDU / ICU

C

Secondary management, provide ongoing therapy and address underlying cause

- Optimise mechanical ventilation
- Reconsider allergy/anaphylaxis - expose and examine the patient, review medications
- If no improvement consider pulmonary oedema/pneumothorax/pulmonary embolus/foreign body
- Consider abandoning / aborting surgery
- Request & review chest X-ray
- Consider transfer to a critical care area for ongoing investigations and therapy

D

1st Line Drug Therapy	2nd 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^{-1}	**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 EXTREMIS: Epinephrine (Adrenaline)** **Nebulised**: 5mls 1:1000 **Intravenous**: 10mcg (0.1ml 1:10,000) to 100mcg (1ml 1:10,000) tirtrated to response

Follow up. If a serious allergic/anaphylactic reaction was suspected or identified the anaesthetist must ensure the patient is referred to a specialist allergy/immunology centre for further investigation. The patient, surgeon and general practitioner should also be informed.

MANAGEMENT OF BRONCHOSPASM - Figure 2

Notes on the algorithm

1. Increasing the inspired concentration of all volatile anaesthetic agents will produce bronchodilatation (the exception is desflurane, which at higher alveolar concentrations has been shown to increase airway resistance[9]). If bronchospasm is severe, the effective delivery of volatile anaesthetic agents will be difficult. An intravenous agent may be necessary and propofol is desirable as it obtunds airway reflexes to a greater degree than thiopentone. If propofol is not available, ketamine is widely available and produces bronchodilatation.

2. Exclude oesophageal/endobronchial intubation. Consider a kinked tube or obstruction caused by secretions, mucous, cuff herniation, or the tube abutting the carina. A suction catheter may be passed down the tracheal tube to assess patency and clear secretions.

3. Box D covers in more detail the main agents used to treat acute bronchospasm. In the first instance treatment is with an inhaled beta agonist such as salbutamol. This can be repeated several times or given 'back-to-back'. Administration must be downstream of the heat and moisture exchange filter (HMEF) and can be with an in-line adaptor (Figure 3), nebuliser, or if these are not available, the metered dose inhaler (MDI) can be placed in the barrel of a 60ml syringe, the plunger replaced and a 15cm length of IV tubing attached to the end by Luer lock (Figure 4). This tubing is then fed down the ETT and reduces the deposition of aerosol on the tracheal tube. As an emergency, the MDI can be discharged directly down the ETT although much of the aerosol will not reach the patient's airways.

Salbutamol can also be given intravenously. Anticholinergic drugs such as inhaled ipratropium bromide block parasympathetic constriction of bronchial smooth muscle. In unresponsive bronchospasm, consider the use of epinephrine (adrenaline), magnesium sulphate, aminophylline, or ketamine.

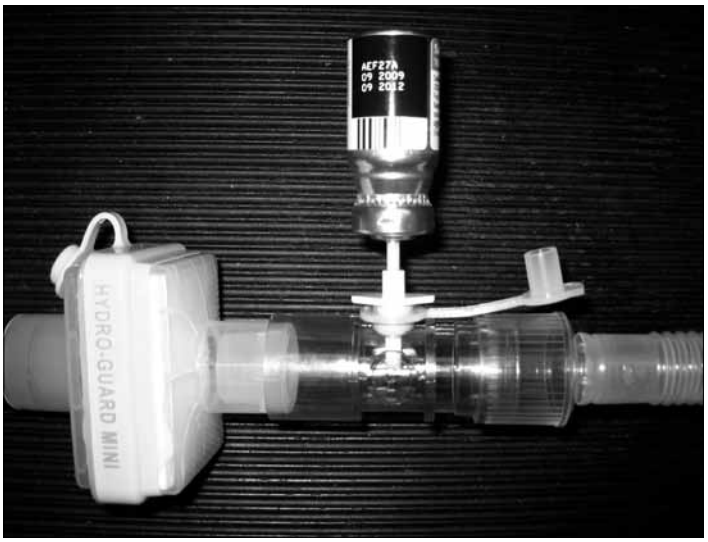

Figure 3. *A metered dose inhaler (MDI) adaptor fitted in the breathing circuit, on the patient side of the heat and moisture exchanger. Depress the canister by hand during inspiration to administer the drug*

Table 1. *Drug doses for use in bronchospasm*

Drug	Adult dose	Paediatric dose
Salbutamol	*MDI* (metered dose inhaler) 6-8 puffs *Nebulised* - 1ml 0.5% (5mg) *IV* - 250mcg slow IV then 5mcg.min $^{-1}$ up to 20mcg.min $^{-1}$	*MDI* 6-8 puffs *Nebulised* <5yrs 2.5mg, >5yrs 2.5-5mg *IV* – 4mcg.kg^{-1} slow IV then 0.1-1mcg.kg^{-1}.min^{-1}
Epinephrine (Adrenaline)	*IV* - 10mcg-100mcg (0.1-1.0 ml 1:10,000) titrated to response *IM* – 0.5-1.0mg if no IV access	*IV* – 0.1-1.0mcg.kg^{-1} (0.01- 0.1ml.kg^{-1} of 1:100,000) *IM* - <6 months 50mcg, 6 mths-6yrs 120mcg, 6-12 yrs 250mcg, >12yrs 500mcg
	Nebulised 5ml 1:1000	*Nebulised* 0.5ml.kg^{-1} 1:1000 (max 5mls)
Ipratropium bromide	*Nebulised* 0.5mg 6 hourly	*Nebulised* (2-12yrs) 0.25mg 6 hourly
Magnesium sulphate	2g IV over 20min (unlicenced)	50mg.kg^{-1} IV over 20min (max 2g, unlicensed)
Ketamine	Infusion: 1-3mg.kg^{-1}.h^{-1} Bolus dose: 10-20mg	Infusion: 1-3mg.kg^{-1}.h^{-1}
Aminophylline	5mg.kg^{-1} IV over 20min then 0.5mg.kg^{-1}.h^{-1} infusion Omit loading dose if taking theophylline	5mg.kg^{-1} IV over 20min then 1mg.kg^{-1}.h^{-1} (<9yrs), 0.8mg.kg^{-1}.h^{-1} (9-16yrs) infusion Omit loading dose if taking theophylline
Hydrocortisone	200mg IV 6 hourly	<1yr 25mg, 1-5yrs 50mg, 6-12yrs 100mg 6 hourly
Chlorphenamine	10mg slow IV	<6 months 250mcg.kg^{-1} IV, 6 months-6yrs 2.5mg IV, 6yrs-12yrs 5mg IV

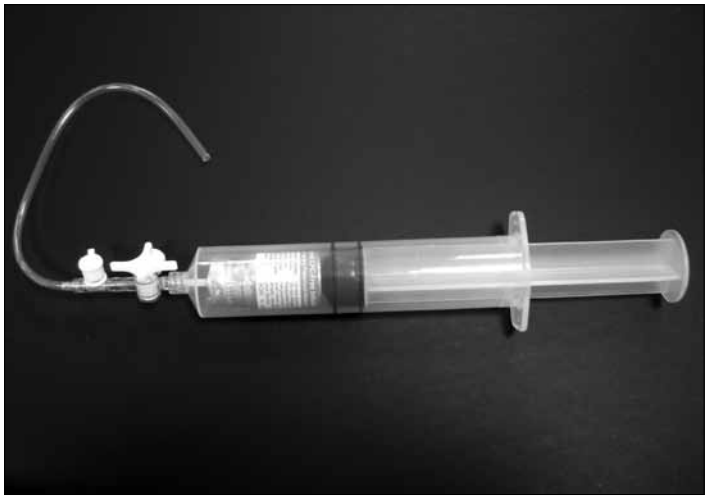

Figure 4. *An MDI canister can be placed in the barrel of a 60ml syringe and a 15cm length of IV tubing attached via the Luer lock. Feed the tubing down the ETT and press the plunger to administer the drug, then reconnect the breathing circuit and ventilate*

SECONDARY MANAGEMENT

The secondary management of acute bronchospasm should provide ongoing therapy and address the underlying cause. Corticosteroids and antihistamines (Box D) 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. If this is the case then avoidance of histamine release is important and an appropriate muscle relaxant should be used (e.g. rocuronium or vecuronium if available).

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.

Mechanical ventilation

The primary aim of mechanical ventilation in acute bronchospasm is to prevent or correct hypoxaemia. Tidal volumes may need to be reduced to avoid high peak airway pressures and barotrauma.

Hypercapnia is tolerated if oxygenation is adequate, as long as severe acidosis does not develop (pH<7.15).

Ventilation should incorporate a long expiratory time to allow complete exhalation and reduce 'breath-stacking' and intrinsic PEEP. Intrinsic PEEP can increase intra-thoracic pressure, decrease venous return and cause hypotension. Minimising intrinsic PEEP is best achieved with a slow respiratory rate, an inspiratory:expiratory ratio of at least 1:2. If bronchospasm is severe, only 3-4 breaths per minute may be possible if you allow full expiration - it is useful to either auscultate or listen at the end of the disconnected endotracheal tube to confirm that expiration has finished, before commencing the next breath. Rarely, to facilitate this, it is necessary to apply manual external pressure to the chest. There is no consensus on application of (external) PEEP, but many advocate trying to match the applied PEEP to the estimated iPEEP.

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 referred to a specialist allergy/immunology centre for further investigation. The patient, surgeon and general practitioner should also be informed.

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