Review Article

Bronchospasm during general anaesthesia-an alarming threat

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Abstract

Bronchospasm and wheezing are not synonymous terms. Bronchospasm is only one cause of wheezing and before the diagnosis can be made, the other causes of wheezing must be excluded. Cyanosis may occasionally be the first sign of severe bronchospasm. An anaesthetist must be alert to diagnose such a case promptly and immediate treatment should be started. Confirmation of diagnosis is not simply a matter of detecting wheezing on auscultation of the chest. It is equally possible to hear no wheezes in the presence of severe bronchospasm, as a result of minimal air movement. Urgent management of such a case includes, Oxygenation, Steroids, Bronchodilators, Lignocaine, Inhalational Anaesthetic agents and other supportive drugs. Close monitoring must be ensured.

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Introduction

Bronchospasm is such a condition which always puzzles an anaesthetist. It is often awesome as well as distressing situation for anaesthetist and also for a surgeon. It requires quick diagnosis and urgent management. Degrees of bronchospasm exist, from mild to severe and even life-threatening. Bronchospasm causes obstruction to gas flow, and it is the consequences of respiratory obstruction that usually attract the attention of the anaesthetist.[1,2] Intubation makes bronchospasm worse but may become necessary. Irritability is greatest at the carina. Acute infection increases sensitivity. A tendency to asthma strongly predisposes to bronchial spasm and wheezing.

Diagnosis

Cyanosis may be the first sign of severe bronchospasm. If the patient is breathing spontaneously, it may be noticed that the movements of the reservoir bag are reduced, or that the patient`s chest and abdomen are moving i.e. `see-saw` or paradoxical respiration as if he or she were obstructed. The expiratory phase may be prolonged, and accompanied by with active expiration using the abdominal and neck musculature.[3] If the patient is being ventilated, it is the high inflation pressure which commonly alerts the anaesthetist to the problem. This may be accompanied by prolonged expiration as observed, or measured by respirometer. It is uncommon for audible expiratory wheeze to be the first sign of bronchospasm, but as anaesthetic tubing can conduct and amlify such noises, this is a possibility, especially if the anaesthetist listens at the distal end of the expiratory tubing.[4,5] Confirmation the diagnosis is not simply a matter of detecting wheezing on auscultation of the chest. It is equally possible to hear no wheezes in the presence of severe bronchospasm, as a result of minimal air movement, and to hear pronounced expiratory wheeze in its absence, turbulent air flow in the tubing or apparatus.[5] It is mainly by exclusion that confirmation is achieved.

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Other causes of respiratory obstruction with resultant turbulent airflow producing a wheezing noise or an increase in inflation pressure include the following.

Patient factors

Airways obstruction due to chronic disease or inspissated secretions, foreign bodies, blood clots, etc. Obstruction to free movement of chest or abdomen by physical or surgical objects. Increase in muscular tone, i.e. coughing, straining, inadequate relaxation. Pneumothorax, particulary a tension pneumothorax, pulmonary oedema.[5-8]

Equipment factors

Incorrect placement of tracheal tube: oesophageal, endobroncheal. Tracheal tube bevel against tracheal wall. Obstructed airway, i.e. poorly maintained airway, blocked Guedel or kinked nasopharygeal tube or tracheal tube. Blockage, kinking of tubes or malfunction of valves in breathing/ventilating circuit.[7]

Physiological Significance

Bronchospasm causes an increase in resistance to gas flow. The electrical equation I = V/R, and by extension to a physiological context: Gas flow = inflation pressure/airways resistance[7] to maintain the same gas flow, and hence alveolar ventilation, as the resistance increases, so must the inflation pressure. When bronchospasm is mild, this can be achieved by the spontaneously breathing patient at the expense of an increased work of breathing and raised oxygen demand or during intermittent positive pressure ventilation by increasing inflation pressure.[8]

There comes a point, however, where inflation pressure is limited by the patient`s ability to achieved the Necessary negative intrathoracic pressure, or where the intermittent positive pressure ventilation system, by virtue of the compliance of the components of the circuit, notably the reservoir bag, or by pressure-limiting valves within the system, can no longer generate the inflation pressure necessary to maintain gas flow at a level compatible with normal alveolar ventilation. At this stage, hypoventilation, and hence hypercapnia, will occur. When the degree of bronchospasm is so severe that as flow falls below that required to allow the amount of oxygen equal to the metabolic requirement to pass to the alveoli, hypoxia will result.[8,9] The above simple treatment ignores the role of gas flow limitation during expiration, which is a hallmark of bronchospasm. This limits the rate of gas flow in expiration and hence alveolar minute ventilation. The natural response to the consequent hypoventilation is to attempt to maintain or increase inspired minute volume, causing an increase in end-expiratory lung volume, i.e. gas tapping. This further decreases the compliance of the lungs and chest wall.

The consequences of bronchospasm are therefore

01. Increase inflation pressure, which at high pressure may cause barotraumas, including interstitial emphysema and pneumothorax.

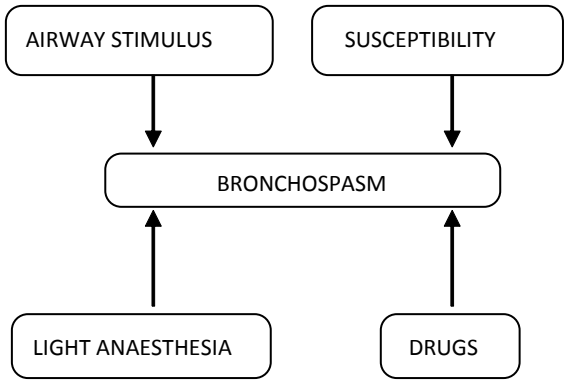
02. Decreased alveolar ventilation, which may cause hypercapnia leading to tachycardia, hypertension and dysrhythmias, and eventually hypoxia and death.

03. Increased end-expiratory lung volume which causes increased inflation pressure.

04. Absolute respiratory obstruction which will give rise to hypoxia.[8,9]

Aetiology

Although bronchospasm may cause in the absence of any of the aetiological factors shown below, one or more of these factors is commonly operative in situations where bronchospasm occurs.[5,9,10]



Aetiological factors commonly operative when bronchopasm occurs

Susceptibility

Patients with a history of smoking, upper respiratory tract infection, bronchospastic disease or atopic disposition are more likely to develop bronchosasm[5,6]. The anaesthetist mat not be aware of such susceptibility.

Airway Stimulus

The ability of the airways to respond to unwelcome invasion is limited to attempts at expulsion/coughing/ and preventing the intruder from gaining access to the small airways/bronchospasm/. The degree of response, if it occurs, is proportional to the intensity of stimulus. This may range in severity from the blowing of cold dry irritant gases in to the respiratory tract, through the insertion of an airway or endotracheal tube, to the placement of an endobroncheal tube.[5-9]

Light anaesthesia

The ability of the airway to react to invasive insults is reduced at deep levels of anaesthesia, and heightened at lighter planes of anaesthesia. Attempts deepen light anaesthesia rapidly using irritant inhalational agents are a common cause of bronchospasm and laryngospasm.[10,11]

Drugs

Certain drugs are associated with an increased incidence of bronchospam, notably beta-adrenoreceptor blocking agents and drugs which are associated with the release of histamine. Severe bronchospasm is a facet of anaphylaxis which may be initiated by anaesthetic agents.[9,10]

Management

01. Confirm the diagnosis. Exclude non-bronchospastic causes of respiratory obstruction, high inflation pressure or wheeze. Establish or continue close monitoring of the patient. If a particular stimulus was associated with the onset of bronchospasm, try to eliminate or reduce the stimulus if this can be done safely. Establish an unobstructed airway, even though on occasions this may seem illogical i.e. intubating a patient who developed bronchospasm during insertion of a Guedel airway. Careful spraying of the vocal cords and trachea with local with local anaesthetic reduces the irritation that would otherwise result from this procedure.[4,7,12]

02. Give oxygen. In severe bronchospasm it may be necessary to give oxygen to prevent hypoxia. Be mindful that light anaesthesia in part resulting from withdrawal of nitrous oxide may worsen bronchospasm-maintain adequate anaesthesia, using intravenous agents if necessary.[3,8]

03. Inhalational can be effective bronchodilators if the bronchospasm is not due to irritation of the airways and if minute ventilation is sufficient to allow their passage in significant concentrations to the small airways. Ether is the most effective, but is now rarely available. Halothane, enflurane and isoflurane is also effective.[3-9]

04. Phosphodiesteres inhibitors can relieve bronchospasm, but should be used cautiously. Aminophylline is usually available in anaesthetic settings. Give up to 5mg/ kg by slow intravenous injection. Rapid injection can cause dangerous dysrythmias, particularly in presence of hypercarbia and hypoxia.[1,2,8]

05. Sympathomimetic agents: it is often not possible to administer these directly to the respiratory tract by aerosol in the anaethetic setting, and so these are usually given by injection. Sulbutamol 3ug / kg or terbutaline 5ug / kg are effective beta2- Adrenoreceptor stimulus. If bronchospasm is severe and unrelenting, it may be necessary to use adrenalin 5-10 ug i.v. or 0.5-1mg s.c./ i.m and repeat as necessary. All sympathomimetics may cause dangerous dysrythmias and especially in the presence of of halothane, hypoxia and hypercarbia and the ECG should be monitored closely.[7]

06. Other drugs: lignocaine, given intravenously in a dose of up to 1.5 mg/kg, may reduce airway reactivity and help to decrease the incidence of dysrythmias. Ketamine has bronchodilator activity and is a non-inhalational method of maintaining anaesthesia; however, hypertion, tachycardia and emergence phenomena limit its use. Steroids and antihistamine agents have been advocated in this context; however, they are unlikely to be effective during the time course of the acute problem. Consideration should be given to the mode of ventilation. A spontaneously breathing patient is unlikely to be able to maintain adequate gas exchange in the face of moderate to severe bronchospasm. It may be necessary to convert to intermittent positive pressure ventilation.[5,7] Allowing a long expiratory time during intermittent positive pressure ventilation will allow better emptying of the lungs. A longer inflation time will limit inflation pressure. However, there comes a point when prolonged inflation and expiration times will limit minute ventilation, and compromises will have to be made. The ventilation system may not be able to deal with the high inflation pressures necessary, and a different system may have to be used.[4-11]

Conclusion

Severe bronchospasm is a frightening occurrence. Although not always predictable, if it is detected and treated early the vicious circle of increased oxygen demand by the body, producing rapid early hypoxia and hypercarbia which aggravates the bronchospasm can be broken. It should be always borne in mind that the one drug that is life saving in these patients is oxygen and efforts must be made to ensure that as much oxygen as possible is delivered to the patient's alveoli. Special attention should be paid to the patient and close monitoring is absolutely essential.

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