ABC of Poisoning

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RESPIRATORY COMPLICATIONS

Respiratory complications are the commonest cause of mortality in acute poisoning

Major respiratory complications of acute poisoning

- Obstructed airway
- Respiratory depression
- Aspiration of gastric contents

The manifestations and complications of acute poisoning are largely non-specific, and supportive treatment is the basis of management in most cases. This and the next article deal with the management of the commonest of these complications.

Priority must always be given to assessing and treating any immediate threats to life, particularly those affecting the respiratory or cardiovascular systems. This might seem obvious, but it may be overlooked by a busy doctor anxious to obtain a history from relatives or ambulancemen or concentrating unnecessarily on reaching a precise diagnosis.

Respiratory complications are the commonest cause of death in acute poisoning, and prompt and effective treatment can save life. Death is usually due to an obstructed airway, respiratory depression, or the aspiration of gastric contents. A clear airway should be ensured from the moment the patient is first seen, and if respiration appears inadequate ventilatory support, if necessary by mouth to mouth respiration, should be started at once while help is summoned.

Depression of protective reflexes

Thoracoabdominal paradox

All sedatives and hypnotics and many other drugs and chemicals, taken in sufficient quantity, profoundly depress the central nervous system. As well as affecting control of respiration, this may depress resting muscle tone and the reflexes that maintain and protect the airway. The tongue may fall against the posterior pharyngeal wall and obstruct the airway, particularly if the patient is supine. Partial obstruction will be evident from the snoring nature of respiration and severe obstruction from the sawing motion of thoracoabdominal paradox, suprasternal recession, diminished breath sounds, and, ultimately, cyanosis. Lifting the tongue clear of the pharyngeal wall by drawing the mandible forwards or inserting an oropharyngeal airway will relieve the obstruction. If the protective reflexes are intact the patient will quickly reject the airway.

The unconscious patient without protective reflexes is safest with a cuffed endotracheal tube in place. Until this is done he should be kept head down in the semi-prone coma position since this helps keep the airway patent and makes the aspiration of regurgitated or vomited gastric contents less likely. Vomit or other foreign material in the pharynx must be cleared. Gastric lavage should never be undertaken in such a patient without first passing an endotracheal tube.

If the patient needs intubation a nasogastric tube should be inserted as well. This enables the stomach to be kept empty, prevents gastric dilatation, and allows antacids to be given to prevent gastric erosions.

Respiratory depression

Use of Wright's respirometer.

Patients are more likely to die from too little oxygen than from too much

- Gut motility can be slowed in opioid, sedative, hypnotic, and anticholinergic poisoning
- Drug absorption may continue for many hours
- Continued close observation is vital

Respiratory depression will be clinically obvious if the respiratory rate is very slow or if respiration is irregular with long apnoeic periods. Serious ventilatory depression may, however, occur with a normal or even increased respiratory rate. The expired minute volume can easily be measured using a Wright's respirometer, either with a tight fitting face mask or by connecting it to the endotracheal tube if the patient is intubated. Such respiratory depression results in type II, or ventilatory, respiratory failure. The elimination of carbon dioxide is impaired and the arterial carbon dioxide tension (PaCO_2) rises accordingly. The arterial oxygen tension (PaO_2) falls by a similar amount.

Management of respiratory depression

Ventilatory depression due to opioid poisoning can be reversed with the specific antagonist naloxone; an infusion may be required if an initial bolus produces a satisfactory effect. In other cases intubation (if not already performed to protect the airway) and mechanical ventilation will be necessary. The use of a self inflating bag and mask may be required while things are made ready. This course of action will be obvious and urgent in the patient who is breathing very slowly or irregularly. Otherwise, a carefully measured minute volume of less than 4 l/min suggests that ventilation is likely to be needed, though blood gases provide the best guide. These may be surprisingly normal, even with very low minute volumes, if the metabolic rate has been severely depressed (as, for example, in hypothermia or barbiturate intoxication). Nonetheless, a PaCO_2 of greater than 6.7 kPa (50 mm Hg) in the absence of chronic airways obstruction is likely to require mechanical ventilation.

Poisoning with a drug likely to produce dangerous arrhythmias (such as tricyclic antidepressants) strengthens the need for artificial ventilation, as hypoxia and hypercapnia increase the likelihood of rhythm disturbances. Supplementary oxygen may relieve the hypoxaemia but will not correct the hypercapnia. Occasionally the comatose patient may be relying in part on a hypoxic respiratory drive. This should not discourage the administration of supplementary oxygen, but rather favours its use in a controlled manner and under close monitoring (with frequent blood gas estimations); preparations should also be made for mechanical ventilation. Patients are more likely to die through lack of oxygen than because of it.

Continued observation of the patient

A patient who on admission maintains his own airway and has an adequate respiratory rate and minute volume may deteriorate, particularly if drug absorption is continuing. Continued, close observation is therefore vital until it is clear that the patient is making a sustained recovery. Even this remark has to be qualified since glutethimide poisoning may lead to late apnoea due to accumulation of a toxic metabolite.

Aspiration of gastric contents

To prevent aspiration

- Attention to airway
- Place in coma position
- Take care with gastric lavage
- Intubate early, rather than too late

The possibility that gastric contents have been aspirated should be considered in any unconscious patient without protective airway reflexes. The clinician must be particularly alert if witnesses describe the patient vomiting or if he was found in a pool of vomit or with vomit staining of the mouth or clothes. Fluid resembling gastric contents (or giving an acid reaction to litmus) obtained from the endotracheal tube is clear evidence that aspiration has occurred, but is seen in only a few cases. Coarse crackles are likely to be heard on auscultation and areas of fluffy shadowing may be seen on the chest radiograph. Radiographs of the chest should be taken in all unconscious patients as they may provide the only indication that aspiration has occurred.

A radiograph of the chest should be taken in all unconscious patients.

Aspiration of gastric contents can produce a bacterial pneumonia or a chemical pneumonitis; the latter may remain localised or become widespread and result in the adult respiratory distress syndrome (ARDS). These effects of aspiration may all result in type I, or hypoxaemic, respiratory failure and require the same basic supportive measures. The aim is the maintenance of adequate oxygenation. In its pure form type I respiratory failure produces arterial hypoxaemia because areas of lung have very low ventilation:perfusion ratios. The PaCO_2 may, initially, be normal or even low, with hypercapnia developing only if ventilation becomes depressed through exhaustion or drug induced central respiratory depression. The latter is particularly likely in patients poisoned with depressant drugs and so a mixed picture, with elements of both ventilatory and hypoxaemic failure, is often seen.

Management of inhalation of gastric contents

Once any debris has been removed from the airways the main goal in treatment is to maintain adequate arterial oxygenation and tissue oxygen delivery (PaO_2 8-10 kPa, 60-75 mm Hg). In less severe cases adequate oxygenation may be achieved simply by increasing the inspired oxygen fraction (F_iO_2) using a mask or nasal cannulae. When supplementary oxygen is given to the intubated patient the inspired gas mixture must be adequately humidified. If these measures fail to relieve the hypoxaemia, if exhaustion supervenes, or if the picture is complicated by central respiratory depression, then intermittent positive pressure ventilation will be required.

In assessing the need for mechanical ventilation the clinician needs to consider further factors. Because of the vital role of the circulation in tissue oxygen delivery, any circulatory inadequacy such as hypotension should be corrected. When this is not possible the indications for ventilation are strengthened. The patient's respiratory workload should be taken into account: a patient with normal blood gases who is working to the point of exhaustion to maintain them needs help far more urgently than one who has slightly abnormal gases but is keeping them stable with minimal distress. Progressively increasing respiratory and heart rates, the use of accessory muscles, sweating, a dyskinetic respiratory pattern, and hypercapnia together suggest impending exhaustion. Poisoning may have major effects on the patient's metabolic rate and therefore his oxygen requirements. These will be depressed in massive barbiturate overdose or hypothermia but greatly increased with hyperthermia or grossly increased muscle activity (as in poisoning with phencyclidine or monoamine oxidase inhibitors). Finally, the importance of any blood gas abnormalities should be assessed against the background of the likely normal values for that patient.

If intermittent positive pressure ventilation is required the use of large tidal volumes (10-15 ml/kg) will minimise any tendency to atelectasis. Though it seems illogical in the poisoned patient, sedation or paralysis may even be necessary to ensure effective mechanical ventilation. It may also be necessary to add a positive end expiratory pressure to the ventilatory pattern. This produces an increase in lung volume and a resulting increase in PaO_2 . The addition of positive end expiratory pressure should be considered if the PaO_2 is less than 10 kPa (75 mm Hg) with an F_iO_2 of 0.5. Some consider that the prophylactic use of positive end expiratory pressure may prevent development of the adult respiratory distress syndrome, but this remains unproved. Although positive end expiratory pressure is almost always associated with an improvement in PaO_2 , this benefit may be outweighed by its adverse effects on cardiac output (and thus on tissue oxygen delivery). Therefore the direct measurement of cardiac output using a balloon tipped pulmonary artery catheter and a thermodilution technique should be seriously considered. This also allows the sampling of pulmonary arterial blood and the measurement of mixed venous oxygen tension, which, with certain reservations, may provide a useful guide to overall tissue oxygenation.

Aspiration pneumonia - main objectives

- Maintain tissue oxygen delivery
- Methylprednisolone (given early)
- Bronchodilators if indicated
- Careful control of fluid balance
- Maintain haemoglobin ($>10\text{ g/dl}$)
- Antibiotics only when indicated
- General care (nursing, physiotherapy, nutrition)

- Positive end expiratory pressure increases PaO_2
but
decreases cardiac output
- Overall effect on tissue oxygen delivery is uncertain

Correct nursing care and regular physiotherapy are essential in prevention and management of chest complications

Any adverse circulatory effects of intermittent positive pressure ventilation, with or without positive end expiratory pressure, usually respond to a fluid challenge, preferably with a colloid solution. Close attention to fluid balance is important, however, and this may best be done while monitoring the pulmonary capillary wedge pressure. Inotropic support may occasionally be required to maintain an adequate circulation during mechanical ventilation with positive end expiratory pressure.

The use of steroids after aspiration is controversial. Despite many benefits suggested by studies in vitro, substantial evidence of clinical benefit is lacking. None the less, used early, in adequate doses and for a short period, some benefit is possible. Three 8 hourly doses of methylprednisolone (30 mg/kg intravenously) should be given beginning as soon as possible in patients who are seen early. Bronchodilator drugs are indicated if bronchospasm is present.

Fluid balance—Close attention should be paid to fluid balance. Any undue increase in pulmonary capillary wedge pressure will tend to worsen pulmonary oedema, especially if there is abnormal pulmonary capillary permeability. Maintaining plasma oncotic pressure, and thus the use of colloid rather than crystalloid solutions where possible, would seem logical. In practice, the benefits appear limited, presumably because the increased membrane permeability reduces the osmotic gradient between interstitial and intravascular spaces. Such a policy may, however, still be helpful in any remaining areas of normal pulmonary capillary integrity.

Further measures—Haemoglobin concentration should be kept above 10 g/dl, by transfusion if necessary, to maximise the oxygen carrying capacity of the blood. Appropriate chest physiotherapy is essential. Nutritional support will be needed if the course is prolonged, and it may have to be given intravenously if enteral nutrition is impossible.

Infection—Antibiotics should *not* be given routinely, but if infection is suggested by purulent sputum, pyrexia, and leucocytosis treatment is required. Although infection should ideally be treated on the basis of culture and sensitivity testing, it is often necessary to start treatment before the results are available. In this case a broad spectrum antibiotic should be used in combination with metronidazole because of the prevalence of anaerobes among the oral flora. Prophylactic antibiotics given to patients after aspiration do not improve survival. Furthermore, unselective early use of antibiotics may produce adverse drug reactions and infection with resistant organisms. None the less, infection needs to be detected when it does occur, and frequent regular bacteriological examinations of sputum and white cell counts should be performed.

Complete obstruction—Another, less common, complication after aspiration is the obstruction of a major airway by a large piece of solid regurgitated food or other foreign body (for example a dental plate). This can lead to complete asphyxia or partial or total collapse of the lung distal to the obstruction. If this seems likely on clinical and radiological grounds urgent removal of the obstruction is indicated, by bronchoscopy if necessary.

Pulmonary oedema

Poisoning with myocardial depressant drugs, such as β blockers, or drugs such as disopyramide or mexiletine may rarely result in cardiogenic pulmonary oedema. Although they may need artificial ventilation, these patients will also require the use of inotropic agents and measures to reduce pulmonary capillary wedge pressure.

Pulmonary oedema may also result from increased permeability of the alveolar-capillary membrane. This may occur after exposure to inhaled toxins (such as irritant gases and metal fumes) or ingested poisons (including salicylates and opioids). This non-cardiogenic pulmonary oedema can rapidly progress to produce the adult respiratory distress syndrome.

Adult respiratory distress syndrome

Causes of ARDS in poisoning

- Aspiration of irritant liquids
eg gastric contents
petroleum distillates
- Infection
- Circulatory failure of any cause
- Inhaled toxins
eg heavy metal fumes
phosgene
- Poisoning with certain drugs
eg heroin
methadone
barbiturates
aspirin
paraquat

The adult respiratory distress syndrome seems to represent the lung's final common pattern of response to a wide variety of insults and may complicate poisoning. The pathogenetic mechanisms remain unclear, but the syndrome is characterised by increased permeability of the alveolar-capillary membrane, which allows fluid to leak from the pulmonary capillaries into the interstitial and alveolar spaces of the lung. This occurs in the absence of left ventricular failure or hypoproteinaemia and has been described as non-cardiogenic or "permeability" oedema.

This process results in arterial hypoxaemia, reduced lung compliance, increased respiratory work, and a fall in functional residual capacity. Clinically there is progressive respiratory distress and hypoxaemia. Auscultation may reveal surprisingly little, but fine crackles are often heard and the chest radiograph shows characteristic widespread, fluffy infiltrates, which may progress to a generalised "white out." A normal pulmonary capillary wedge pressure will eliminate cardiogenic causes, and if plasma protein concentrations are normal, the oedema is unlikely to be due to reduced plasma oncotic pressure.

The principles of treatment are as outlined and consist primarily of cardiorespiratory support to maintain adequate tissue oxygen delivery at the lowest possible F_iO_2 , minimising the risks of pulmonary oxygen toxicity, while keeping the pulmonary capillary wedge pressure as low as possible.

Other conditions

- Neuromuscular blockade may cause ventilatory failure
- Monitoring of vital capacity is essential

Secondary bacterial pneumonia
often complicates prolonged coma

Neuromuscular blockade—Another cause of ventilatory failure in acute poisoning is seen with agents that interfere with neuromuscular conduction (such as the long-acting anticholinesterase insecticides, hemlock, and botulinus toxin).

Again, principles of management in terms of ventilatory support (and antidotes when appropriate) are as described. Serial measurements of vital capacity (measured with a face mask and Wright's respirometer) may be useful in monitoring progress and assessing the need for mechanical ventilation. A patient who is unable to manage 12-15 ml/kg is likely to need help.

Secondary bacterial pneumonia may occur in any patient who has suffered prolonged coma and is particularly common in barbiturate poisoning. Contributory factors include respiratory depression, diminished cough, absence of sighing, and ciliary immobility. These lead to basal atelectasis and retention of secretions with secondary infection. Antibiotics and regular physiotherapy are the mainstay of treatment.

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