Crisis management during anaesthesia: pulmonary oedema

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Pulmonary oedema is the abnormal accumulation of fluid in the interstitial or alveolar spaces of the lung. It occurs for a number of reasons which can be explained on the basis of a disturbance in the normal Starling equation. It involves changes in hydrostatic or oncotic pressure across the alveolar membrane or in the permeability of the alveolar membrane such that fluid moves across from the capillaries into the alveolar space. Traditional teaching is that pulmonary oedema occurring in patients outside hospital tends to be cardiogenic in aetiology, with usually left heart diastolic dysfunction causing back pressure across the pulmonary system, resulting in extravasation of fluid into the alveoli. However, recent evidence suggests that increased afterload as in neurogenic pulmonary oedema may also be important in cardiogenic causes.

In patients undergoing anaesthesia, causes of pulmonary oedema other than cardiogenic are encountered. Decreased plasma oncotic pressure (due to hypoproteinaemia), fluid overload, alterations in the permeability of the alveolar-capillary membrane (such as typically seen in acute respiratory distress syndrome (ARDS)), and post-obstructive and neurogenic pulmonary oedema may all occur. In many cases the aetiology is multifactorial with no clear identifiable precipitant. Because recognition may be delayed and management variable, it was decided to examine the place of a structured approach to the diagnosis and management of pulmonary oedema occurring in association with general anaesthesia.

In 1993 a “core” crisis management algorithm, represented by the mnemonic COVER ABCD–A SWIFT CHECK (the AB precedes COVER for the non-intubated patient) was proposed as the basis for a systematic approach to any crisis during anaesthesia where it is not immediately obvious what should be done or where actions taken have failed to remedy the situation. This was validated against the first 2000 incidents reported to the original Australian Incident Monitoring Study (AIMS). AIMS is an ongoing study which involves the voluntary anonymous reporting of any unintended incident which reduced or could have reduced the safety margin for a patient.

It was concluded that, if this algorithm had been correctly applied, a functional diagnosis would have been reached within 40–60 seconds in 99% of applicable incidents, and that the learned sequence of actions recommended by the COVER portion would have led to appropriate steps being taken to handle the 60% of problems relevant to this portion of the algorithm. However, this study also showed that the 40% of problems represented by the remainder of the algorithm ABCD–A SWIFT CHECK were not always promptly diagnosed or appropriately managed. It was decided that it would be useful, for these problems, to develop a set of sub-algorithms in an easy-to-use crisis management manual. This study reports on the potential place of the COVER ABCD–A SWIFT CHECK algorithm in the diagnosis and initial management of pulmonary oedema, provides an outline of a specific crisis management algorithm for this problem, and provides an indication of the potential value of using this structured approach.

METHODS

Of the first 4000 incidents reported to AIMS, those that made reference to pulmonary oedema were extracted and analysed for relevance, presenting features, type of surgery, cause, management and outcome. The COVER ABCD–A SWIFT CHECK algorithm, as presented elsewhere in this series of papers, was applied to each relevant report to determine the stages at which the problem might have been diagnosed and to confirm that activating the COVER portion would have led to appropriate initial steps being taken. As pulmonary oedema is not dealt with by this algorithm, a specific sub-algorithm was developed (see fig 1) and its putative effectiveness was tested against the reports. How this was done is described elsewhere in this series of articles. The potential value of this structured approach—that is, the application of COVER ABCD–A SWIFT CHECK to the diagnosis and initial management of this problem and the
PULMONARY OEDEMA/ARDS

SIGNs (1)*
Respiratory distress/tachypnoea
Desaturation
Increased inspiratory pressure
Pink frothy sputum—ETT ILMA (diagnostic)
Creptations or branchiasthmatic

PRECIPITATING FACTORS
Fluid overload (2)
Non-cardiogenic:
- Post airway obstruction (3)
- Anaphylaxis
- Neurogenic
- Sepsis
- Pulmonary aspiration
- Multiple organ failure
Cardiogenic (4)

EMERGENCY MANAGEMENT
Titrated inspired oxygen concentration against SpO2
Head up 15°/sit up
If self ventilating/apply CPAP (5)
Intubate if necessary
IPPV and PEEP if intubated
Consider drug therapy: morphine/GTN/frusenide (6)

The sub-algorithm forms a facing page of the Crisis Management Manual12. * Numbers in brackets refer to Notes in the right hand panel.

FURTHER CARE
Consider and investigate likely cause
Review perioperative fluid balance/renal function
Non-cardiogenic: consider post airway obstruction
- Allergy/anaphylaxis → page 48
- Aspiration → page 16
- Sepsis → page 64
- Multiple organ failure, eg major trauma, pancreatitis
- Renal—renal function tests
Cardiogenic:
- ECG
- Cardiac enzymes
- Echocardiogram
- Chest X-ray

Consider admission to high dependency area/CU

The COVER ABCD—A SWIFT CHECK algorithm was successful in the management of the initial physiological upset, however it was considered that the use of this specific sub-algorithm would be required once the initial diagnosis of pulmonary oedema was made.

(1) Hypoxia – 46%; pink frothy sputum – 23%; increased airway pressures – 14%; respiratory distress – 14%; creptations or wheeze – 9%.

(2) Fluid overload was judged to be the cause in 46% of incidents. 81% of these had pre-existing conditions making them more susceptible to overhydration: age >70, cardiovascular disease or hypertension, renal failure and chronic airflow limitation.

(3) 23% of incidents were judged to be post-upper airway obstruction.

(4) 14% were judged to be cardiogenic in origin, eg, valvular heart disease, ischaemia/infarction, cardiac failure, arrhythmia.

(5) CPAP is important specific therapy for pulmonary oedema (in addition to treatment for hypoxia).

(6) Preload reduction:
- Morphine 1 mg IV doses
- GTN infusion 50 mg in 500 ml
- Commence at 0.1 ml/kg/hr
- Fluid reduction:
- Frusenide 0.5 mg/kg IV if fluid overload (place urinary catheter)
- If hypertensive:
- Adrenaline infusion: start with 0.00015 mg/kg/min
- Adrenaline: for easy adult dosing, see page 71
- Titrated against heart rate and blood pressure


Figure 1 Pulmonary oedema/ARDS.

application of the sub-algorithm for pulmonary oedema was assessed in the light of the AIMS reports by comparing its potential effectiveness for each incident with that of the actual management as recorded in each report.

RESULTS
Pulmonary oedema was mentioned in 37 reports (<1%) but, in two of these, pulmonary oedema was not one of the active problems at the time of the incident. This left 35 reports which constitute the database for this study.

The aetiology of the pulmonary oedema was varied. It was associated with fluid overload in 16 patients (46%), most of whom (81%) had a clinical condition that may have made them more susceptible to fluid overload. These conditions included age >70 years, a past history of cardiac disease or hypertension, renal failure, and chronic obstructive airways disease. It occurred following upper airway obstruction in eight patients (23%), appeared to be cardiogenic in five (14%), and to be due to neurogenic causes and anaphylaxis in one case each. Three (9%) had no obvious cause for the pulmonary oedema from examination of the report.

Seventeen (49%) of the incidents occurred outside the operating or procedure room. The COVER algorithm is not relevant to these incidents as it is designed for patients breathing gas from an anaesthetic machine. Twenty-three of the patients (66%) were admitted to the intensive care unit and two (6%) died. Neither death could be directly attributed to the pulmonary oedema.

There were a number of symptoms or signs alerting the anaesthetist to the presence of pulmonary oedema as listed in fig 1. Hypoxia presenting as desaturation or cyanosis was the commonest sign (46%). Pink frothy sputum was noted in 23% and is diagnostic of pulmonary oedema. Increased airway pressure was observed in 14%, respiratory distress in
14%, and pulmonary crepitations or wheeze on auscultation of the chest in 9%. In none of these cases was the COVER algorithm adequate alone to deal with the incident as the initial problem progressed to a diagnosis of pulmonary oedema. When the sub-algorithm for pulmonary oedema (fig 1) was tested against these incidents, once the diagnosis was made, it was thought that it would have been successful in all but one of the cases. This was one death which appears to have been unavoidable due to uncontrollable bleeding in a patient with pre-existing cardiac failure.

DISCUSSION

Pulmonary oedema is a potential cause of hypoxia in the perioperative patient. The accumulation of excessive alveolar fluid results in hypoxia due to interference with diffusion across the alveolar capillary membrane. Frothy (sometimes bloodstained) sputum may be expectorated or observed in the endotracheal tube. This is a pathognomonic sign of pulmonary oedema which, as indicated above, may have various aetiologies. The diagnosis of pulmonary oedema may be supported by finding crepitations and sometimes wheezes on auscultation of the lung. However, these may also be heard with aspiration pneumonitis or infection. A chest radiograph may help to confirm the diagnosis but is not specific. Generalised opacity is characteristic with a typical peripheral “bat’s wing” appearance. However, the opacity may be unilateral or asymmetrical, particularly in the acute period (<4 hours). Alveolar opacities on the chest radiograph may also be due to inhalation of enteric contents, infection, or alveolar haemorrhage. The diagnosis of pulmonary oedema is made on assessment of the combination of history, symptoms, clinical signs, and investigations and on excluding alternative diagnoses such as aspiration pneumonitis and pneumonia.

Increased permeability (or non-cardiogenic) pulmonary oedema (ARDS) may be differentiated from normal permeability pulmonary oedema by assessing cardiac filling pressures. This can be done by examination of the jugular venous pressure or invasively with a central venous or pulmonary artery catheter. A pulmonary artery occlusion pressure of 18 mm Hg is regarded by convention as a threshold pressure differentiating the two. However, this is not specific as normal filling pressures may be seen in cardiogenic pulmonary oedema, particularly in patients on mechanical ventilation. Similarly, cardiac compromise may coexist in a patient with ARDS.

When pulmonary oedema is suspected a cause should always be sought. The causes listed in fig 1 are those commonly found in perioperative patients. Post-obstructive pulmonary oedema is a rare entity outside the operating suite and intensive care unit. It may be over reported here due to its association with the reportable incident of airway obstruction and its dramatic appearance in patients who are often previously fit and well.2,11 The pathophysiological basis for this entity is not well understood.10,11

Pulmonary oedema will usually present with hypoxia. If it occurs during anaesthesia, the presenting signs will be apparent using COVER in 95% of cases, with a further 5% during consideration of the A in ABCD. Definitive diagnosis relies on recognising a pattern of signs and symptoms, but may not be possible in some cases until intravascular pressures have been measured and other investigations have been undertaken. Once the diagnosis of pulmonary oedema is made, this sub-algorithm should then be followed. However, the physiological abnormalities sometimes have been reversed by COVER before this sub-algorithm is reached.

Finally, it is important that a full explanation of what happened be given to the patient, that the event and the results of any tests should be documented in the anaesthetic record, and that the patient be given a letter to warn future anaesthetists. If a particular precipitating event was significant or a particular action was useful in resolving the crisis, this should be clearly explained and documented.

CONCLUSION

Pulmonary oedema occurred in less than 1% of the 4000 perioperative incidents reported to AIMS. Initial management as suggested by the COVER ABCD algorithm and the pulmonary oedema sub-algorithm will sometimes be successful in treating the physiological abnormalities before a diagnosis is made. A sub-algorithm aimed at the management of perioperative pulmonary oedema was developed. This has been tested against the 35 incidents reported and appears to be appropriate.

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