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The Management of the Pulmonary Aspiration Syndrome

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Abstract. The paper describes two cases in which the patients who had suffered from severe pulmonary aspiration received supplemental oxygenation using a membrane oxygenator. One of the patients survived. The management of pulmonary aspiration is reviewed.

Key words: Pulmonary Aspiration, Mortality, Membrane Oxygenation, Antacids, Lung Drainage, Ventilation, Pulmonary and expiratory pressure, Oxygenation, Blood volume correction, Steroids.

Introduction

Much work has been done on the aetiology and factors associated with aspiration pneumonia [1, 3] and the methods whereby it can be prevented [3, 34].

The mortality from aspiration pneumonia remains high. Mucklow and Larard [42] stated that 2 % of all maternal deaths in the USA were the result of inhalation of vomit. Crawford [18] gave a critical appraisal on the report issued by the Department of Health on maternal deaths in England and Wales 1967/69. While the total number of deaths had decreased, those associated with anaesthesia had not, 44 % of these deaths being complicated by regurgitation of gastric content. The estimated maternal mortality rate was 19.2 in 1964/66 and 20.3 in 1967/69 per one million obstetric anaesthetics.

Prevention is paramount, but when faced with an aspiration syndrome it is important to treat the patient appropriately.

Case 1

A 26 year old Phillipino primagravida was admitted at term of induction for labour for pre-eclamptic toxaemia. Forty-eight hours after the commencement of a syntocinon infusion, the cervix became fully dilated. Labour failed to progess. Forceps delivery followed by vacuum extraction were both tried unsuccessfully. It was decided to proceed to Caesarian section.

Anaesthesia was undertaken initially by an inexperienced junior anaesthetist. No food had been taken during the previous twelve hours. A nasogastric tube was not passed;antacids were not given. Following pre-oxygenation, induction, (performed with the patient lying supine with a head down tilt) was commenced using sodium thiopentone and suxamethonium chloride. Although the patient was asleep no relaxation took place. Assuming that the relaxant had entered the tissues, a further dose was given. Sufficient relaxation was achieved for intubation to be attempted but this failed. The patient became cyanosed, vomited and arrested. The heart was restarted within a minute by external cardiac massage. A Consultant Anaesthetist was called who continued the anaesthesia with oxygen, trichlorethylene and either using a face mask, a stomach tube having been passed and a moderate volume of fluid aspirated. The patient vomited several times during the operation. A live baby was delivered. Three hours later she was transferred to an Intensive Therapy Unit, semi-conscious, with a pulse of 180/minute and a blood pressure of 100/60mmHg. Her PaO_2 was 4.13kPa and her $PaCO_2$ 9.33kPa. It was evident that massive aspiration had occurred (the diagnosis being supported by the appearances on chest X-ray (Fig. 1)).

Following intubation and mechanical ventilation a PaO_2 of 9.33kPa (70mmHg) and a $PaCO_2$ 8.0kPa (60mmHg)

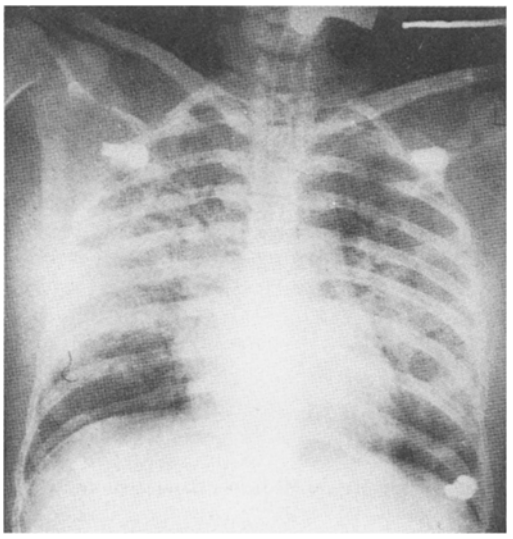


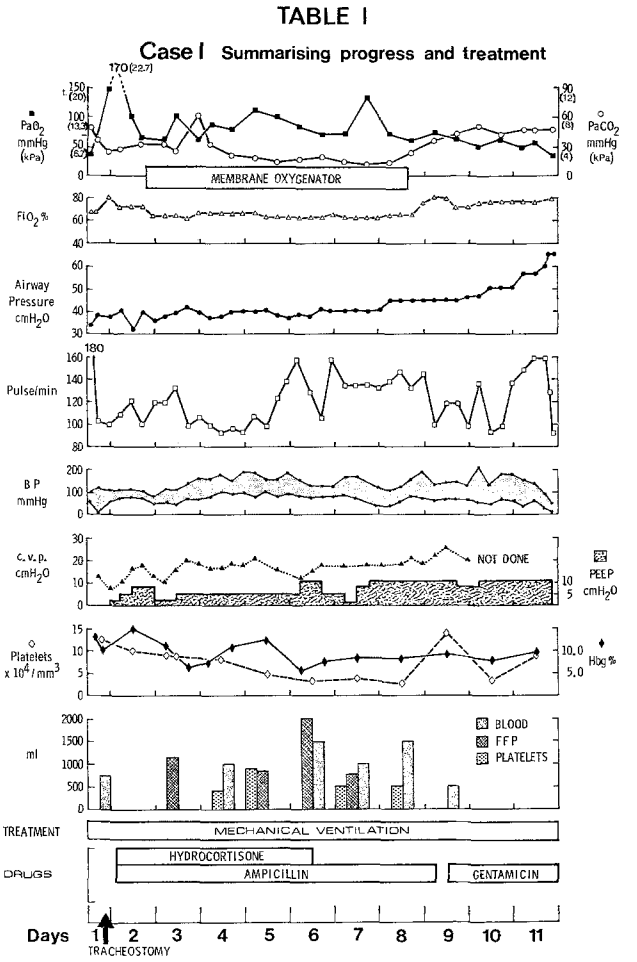
Fig. 1. Case 1. Chest x-ray appearance 4 hours after pulmonary aspiration during induction of anaesthesia for Caesarian section

were achieved. Airway pressures were elevated to 36 cm H_2O. A bolus dose of 500 mg methyl prednisolone hemisuccinate was given intravenously, followed by hydrocortisone sodium hemisuccinate 100 mg intravenously six hourly, Ampicillin 1.0 g I.V. six hourly was prescribed. 4 hours later it was noted that the urine was uniformally blood stained. The haemoglobin level had fallen from 12.6 g/dl on admission to 10.5 g/dl in spite of transfusion of 800 mls of blood in addition to other I.V. fluids Clotting factors were normal and there was no evidence of a haemoglobinopathy or G.6.P.D. deficiency. It was thought that the haemoglobinuria could be related to the one dose of cephalothin given on admission, or to blood incompatibility. Further patient progress can be seen in Table 1.

The patient's lung condition remained critical, worsening by the evening of the second day. In addition to other signs indicating this, the PaO_2 with an FiO_2 of 0.8 had fallen from 13.33kPa (100mmHg) to 9.2kPa (70mmHg). It was considered that supplementary oxygenation using a membrane oxygenator was necessary. Later in the evening the patient was taken to theatre where the Cardiac By-Pass Team from the Birmingham Children's Hospital performed the necessary cannulations for the use of the oxygenator.

With membrane oxygenation it was possible to lower the FiO_2 to 0.6 and the PEEP from 8 to 5 cms H_2O. Airway pressures, however, remained constant at 40 cms H_2O. Membrane oxygenation was continued for 6 days after which it had to be discontinued because of heavy bleeding, despite platelet infusions and replacement of the labile factors. During the succeeding 5 days the patient's conditon gradually deteriorated and, despite the use of increasingly higher inspiratory oxygen percentages and PEEP, the PaO_2 never rose above 8.66kPa (64.5mmHg). On the ninth day following admission, the $PaCO_2$ started to rise and on the day of her death, eleven days after admission, there was overt deterioration. Cuff deflation

Table 1. Case 1. Summarising progress and treatment



could no longer be tolerated, thick strawcoloured and mucus was being aspirated from the bronchi and the blood gas values deteriorated. The blood pressure and the urine output both fell and the pulse began to rise. The patient suffered an a-systolic arrest at 21.30 hrs.

Discussion

This patient demonstrates the problems in management of a patient who aspirates during induction of anaesthesia, especially when intubation proves difficult. It would appear that severe irreversible lung damage had occurred following the initial insult. Membrane oxygenation undoubtedly prolonged life, but had to be withdrawn because it proved impossible to maintain platelet count and coagulation factors at a satisfactory level by replacement therapy. Clinical and biochemical features all suggested a gradual deterioration in lung function from the start. It was not possible at any time to assess whether hypoxic cerebral damage had occurred because it was necessary to sedate the patient to maintain adequate mechanical ventilation. The lungs at post mortem showed early absecess formation in the left lower lobe. Sputum culture two days prior to death grew pseudomonas aeroginosa sensitive to

gentamicin which had been given in a dosage sufficient to maintain a therapeutic blood level. It seems unlikely in this case that lung infection was a major contributory cause of death. Blood cultures were consistently negative, despite the insertion of many cannulae.

Case 2

A 35 year old white multigravida with three previous uneventful pregnancies was admitted to the ward at mid-day with severe lower abdominal pain, where she was observed until 18.00 hrs., when a diagnosis of ruptured ectopic pregnancy was made. She had taken nothing by mouth from mid-day until the time of operation at 20.30 hrs. Two units of blood had been infused preoperatively. Neither had a Ryles tube been passed nor had she received any pre-operative antacid. A pre-medication had not been given and the patient was fully concious on arrival in the theatre. There was no suggestion of aspiration during induction and anaesthesia proved uneventful. During the operation 500 mls of blood and 100 mls of dextrose saline were given. The operative procedure was entirely uneventful, a right salpingectomy being performed for a tubal rupture in the ampullary region. Some 1000 mls of blood was found in the peritoneal cavity.

The patient was fully conscious and talking before being returned to the ward in a lateral slightly head down position at 21.00 hrs. It was noted on return to the ward that the previously clean pillow was slightly bile stained but the patient remained pink, fully conscious and in no respiratory distress. At 22.30 hrs. the patient complained of pain and was given Papaveretum 20 mg intra-muscularly as instructed. At 00.30 hrs. the nursing staff noted that the respiratory rate had increased to 40 per minute and the pulse to 120 a minute; the blood pressure was 140/70 mmHg. By 02.40 hrs. the blood pressure had fallen to 90/60mmHg and the House Surgeon was called. The patient was seen to be breathless, the pulse rate 106 and the blood pressure confirmed at 90/60mmHg. There was no evidence of post-operative bleeding but breath sounds were noted to be diminished and crepitations were heard on the left side of the chest posteriorly. It was considered that the patient had partially collapsed her left lower lobe. A chest X-ray was taken but not seen until later on in the day. Intravenous infusion was maintained with 500 mls of dextrose saline to be infused over the subsequent four hours. The blood pressure having risen to 100mmHg systolic, a second dose of Papaveretum 20 mg was given intra-muscularly at approximately 02.45 hrs. At 04.30 hrs. the nursing staff thought the patient was slightly cyanosed and 35 % oxygen was given via ventimask. By this time the pulse had increased to 150 per minute, the respiratory rate to 30 a minute and the blood pressure was 100/70mmHg. By 09.30 hrs. the patient's condition had markedly deteriorated. She was conscious but had a tachypnoea and was cyanosed, the pulse rate had risen to 160 a minute and the blood pressure had dropped to 70mmHg systolic.

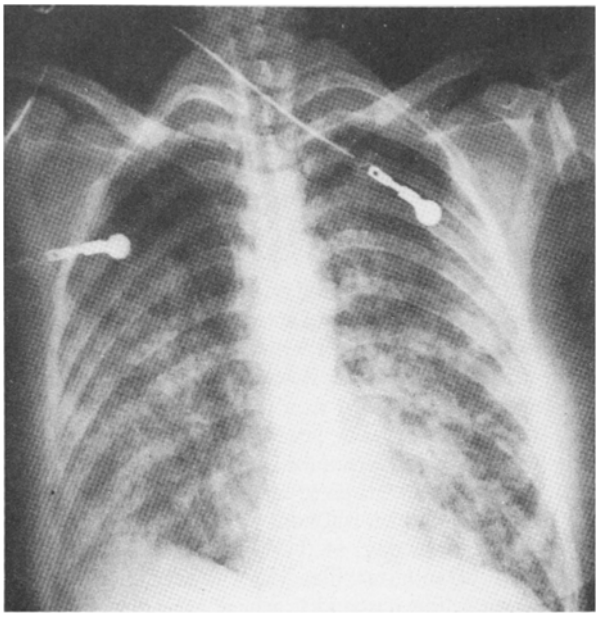
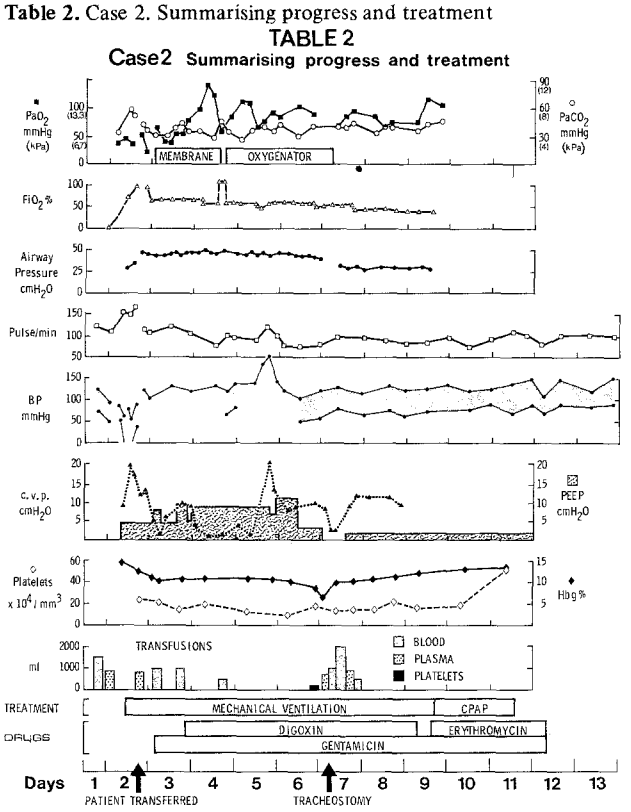


Fig. 2. Case 2. Chest x-ray appearance 12 hours following a 'symptomless' postoperative pulmonary aspiration

The chest X-ray taken at 0.500 hrs. showed extensive pulmonary aspiration. Tha patient was transferred to the Intensive Therapy Unit. Her condition had deteriorated rapidly. She had become semi-conscious with marked cyanosis, a respiratory rate of 40 per minute, a pulse rate of 168 a minute; her blood pressure was unrecordable. Prednisolone hemisuccinate 1.0 g was given intravenously. A nasogastric tube was passed, 200 mls of fluid aspirated and 15 mls of magnesium trisilicate mixture was given down the tube. The central venous pressure read 9 cm H_2O. A repeat chest X-ray showed extensive shadowing throughout both lung fields (Fig. 2) and Astrup Analysis with an FiO_2 of 0.4 via a face mask showed pH 7.13, PaO_2 5.86kPa (44mmHg) and a $PaCO_2$ of 4.8kPa (36mmHg) with a base deficit of 16 m.mol/l.

The patient was intubated, copious blood stained secretions being obtained via the endotracheal tube. Continuous positive pressure ventilation was started with a minute volume of 12.5 l/minute, a respiratory rate of 15/minute, FiO_2 of 0.7, with PEEP of 5 cms H_2O. On this regime, in spite of infusion of 800 mls plasma protein fraction and 200 mls Dextran 70 in saline, the blood pressure remained unrecordable. PEEP was therefore stopped. The respiratory rate was increased to 16, and the minute volume reduced to 10.5 - 11.0 l/minute. The patient's subsequent progress and treatment is shown in Table 2. In spite of a good urine output, a falling pulse rate and a steadily rising blood pressure, the patient's lung condition steadily deteriorated. The central venous pressure rose from 9 - 20 cm H_2O, the airway pressure to 30 cm H_2O in the absence of PEEP. The blood gases at 18.00 hours with an FiO_2 of 0.8 were PaO_X 5.73kPa (43mmHg) and $PaCO_2$ 6.4kPa (48mmHg) and the base deficit which had lessened considerably began to increase because of deteriorating peri-

Table 2. Case 2. Summarising progress and treatment



pheral perfusion. In view of the steady deterioration, it was decided that the patient could not survive without supplemental oxygenation. At 19.00 hours the patient was transferred by helicopter, hand ventilated on oxygen, to the Intensive Therapy Unit in the Birmingham Children's Hospital.

On admission to this Unit she was extremely cyanosed with generalised oedema. There was considerable peripheral vasoconstriction (foot temperature 28°C, core temperature 37°C). She was nursed on a weigh-bed. The cuffed orotracheal tube was changed to a cuffed nasotracheal tube. The left radial artery was cannulated for arterial pressure recording and gas tension measurements, and the innominate vein cannulated. A Blease ventilator was used with FiO_2 of 0.8, PEEP 4 cms H_2O and an airway pressure of 50 cms H_2O. Her systolic blood pressure was 120mmHg, CVP 8mmHg and PaO_2 7.2kPa. (54mmHg). The bladder was catheterised. In view of the pulmonary oedema, she was given 800 ml of double strength plasma, frusemide 40 mg and thymoxamine 60 mg in an attempt to improve peripheral perfusion. Despite these measures, two hours after admission the PaO_2 had fallen to 5.06kPa. (38mmHg). Chest x-ray at this time showed extensive diffuse bilateral shadowing with hyperinflation of the left lower zone (Fig. 3).

At 00.45 hours on the third day of her illness she was taken to the theatre. She was heparinized (1 mg/kg body weight (80 mg)). The right femoral vein was cannulated proximally and distally, the two cannulae being linked by a Y-tube, and the right internal jugular vein was cannulated towards the right atrium. The wounds were closed with

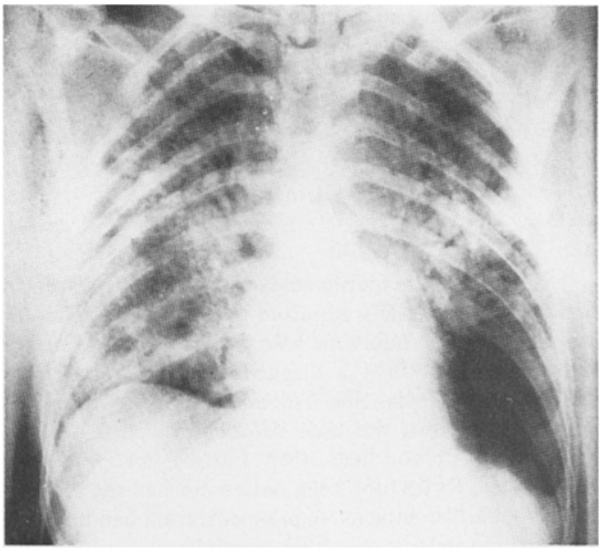


Fig. 3. Case 2. Chest x-ray appearance of the same patient on the third day following pulmonary aspiration. At this time the patient was being ventilated with a pulmonary inspiratory oxygen percente of 80 and PEEP of 4 cms H_2O. The PaO2 in spite of these measures was only 5.07kPa

meticulous haemostasis. The PaO_2 with an FiO_2 of 0.6 was 4.0kPa (30mmHg). On return to the Intensive Therapy Unit at 02.30 hours the cannulae were connected to a primed heparinized membrane oxygenator ("Modulung" 3.0 M^2 membrane oxygenator, Travenol Labs. Inc.). Pancuronium bromide 8 mg 1 - 2 hourly, phenoperidine 2 mg 4 hourly and diazepam 5 mg 4 hourly were ordered. Intravenous fluids were prescribed as 2000 ml/24 hours each 500 ml unit containing 15,000 units of heparin, given at such a rate as to keep the clotting time to 30 minutes. By 08.00 hours the heparin was increased to 20,000 units per 500 ml. Gentamicin was prescribed 80 mg 8 hourly, and digoxin 0.5 mg initially, to be followed by 0.25 mg 12 hourly.

By 14.00 hours on the third day there was frank frothy pulmonary oedema fluid in the nasotracheal tube and suction was stopped; the PaO_2 was 5.6kPa. (42mmHg). At 16.00 hours PEEP of 10 cms H_2O was applied when the PaO_2 was 7.06kPa (53mmHg) and by 22.40 hours the PaO_2 had risen to 9.86kPa (74mmHg).

Over the subsequent two days the patient continued to make satisfactory progress (Table 2). The gentamicin dosage was increased to 120 mg 8 hourly.

On the sixth day, bowel sounds were audible and a milk drip was commenced via a nasogastric tube. At 15.00 hours she was wide awake, her PaO_2 14.0kPa (105mmHg) on an FiO_2 0.5. Pancuronium bromide was discontinued and 6 packs of platelets given intravenously together with 540 ml purified protein fraction.

On the seventh day, at 04.00, the clotting time had increased to 60 minutes, bleeding was occurring from the nose alongside the nasoendotracheal tube and by 07.00 hours the haemoglobin had dropped to 6 gm/dl. It was decided to discontinue membrane oxygenation. The pa-

tient was taken to the operating theatre. A blood transfusion was commenced and under anaesthesia the internal jugular vein was decannulated, the vein ligated and a portion of vein was removed. The femoral vein cannulae were removed and the vein reconstructed, using the portion of internal jugular vein previously excised. In view of the bleeding from the nasal septum, a tracheostomy was done. A feeding gastrostomy was performed. On return to the Intensive Therapy Unit the PaO_2 was 8.8kPa (66mmHg) with FiO_2 0.4 and by 14.30 hours the PaO_2 was 10.66kPa. (79.5mmHg).

At 22.00 hours good spontaneous respiration was established.

On the eighth day gastrostomy feeds were commenced. The patient started to breath spontaneously. During the day when the patient tired IPPV was used for periods up to 90 minutes; she was ventilated overnight.

On the ninth day a CPAP circuit was set up with FiO_2 0.35, when the PaO_2 was 14kPa (105mmHg) and $PaCO_2$ - 5.6kPa (42mmHg). The following day the arterial line and urinary catheter were removed, together with the nasal pack which had been inserted previously to control the haemorrhage. The patient was fully conscious, rational and sitting up.

On the tenth day CPAP was discontinued.

On the eleventh day the tracheostomy tube was removed and food was taken by mouth and on the twelfth day the patient was walking with assistance.

The following day she was transferred back to the Intensive Therapy Unit at Whipps Cross Hospital. She was mentally clear and had not suffered any hypoxic cerebral damage. She was a good colour, her respiratory rate normal and her chest was clinically clear. Chest x-ray showed residual basal shadowing (Fig. 4). Blood gases were not taken. The gastrostomy tube was removed three days after transfer. The patient made an uneventful recovery and was discharged from the hospital 22 days after the initial admission. Chest x-ray taken (Fig. 5) 3 days before discharge showed no abnormality.

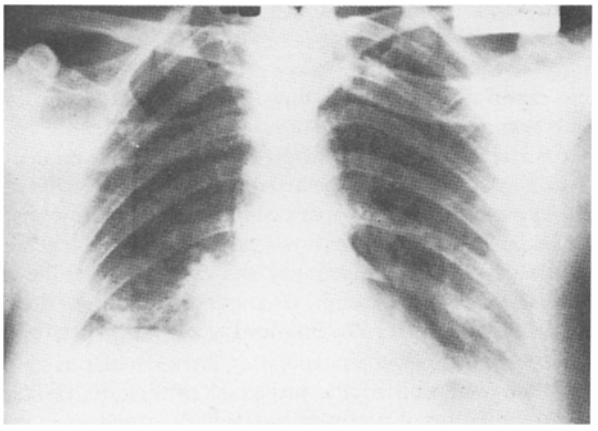


Fig. 4. Case 2. Chest x-ray appearance of the same patient 14 days following pulmonary aspiration. The patient had been breathing spontaneously without C.P.A.P. for 3 days

Respiratory function studies performed 4 weeks after aspiration were within normal limits apart from the diffusing capacity (D.L.C.O.) at rest. This was at the lower limit of normal being 20.7 ml/min/mmHg (predicted value of 26.1).

Comments

This case illustrates that pulmonary aspiration can occur in a conscious patient whose cough reflex has been suppressed. Assuming that aspiration occurred whilst she was on the way back to the ward it also illustrates that a patient may appear well at the time of aspiration only for the condition to develop insidiously during the ensuing hours. Deterioration occurred during the night which may have made recognition of cyanosis difficult. The second dose of papaveretum may well have minimised symptoms – suppressing the hypoxic drive and reducing the classical physical sign of tachypnoea. There is evidence, having discussed the case with the attending nursing staff, that the respiratory rate fell after the papaveretum and tachypnoea only became evident the following morning. It was fortunate that the patient did not sustain irreversible hypoxic cerebral damage.

Early diagnosis and appropriate management from the onset could well have prevented the necessity for supplemental oxygenation. It illustrates the hazard of anaesthesia in any emergency operative procedure. The preoperative use of a single dose antacid as 15 ml of suitably flavoured 0.3 molar sodium citrate as suggested by Lahiri, Thomas and Hodgson [34] is to be recommended in patients undergoing emergency anaesthesia. The severity of the pulmonary lesion following aspiration appears to be closely related to the pH of the gastric contents; a pH of three should be regarded as the critical level below which aspira-

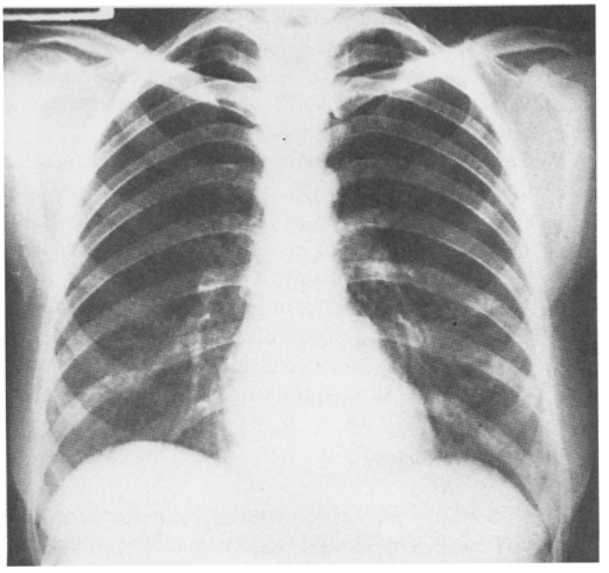


Fig. 5. Case 2. Chest x-ray appearance of the same patient 10 days following severe pulmonary aspiration. At the height of the illness a membrane oxygenator was required in order to maintain an adequate PaO_2

tion symptoms become severe [17]. Taylor [53] however presented a case report of severe pulmonary aspiration in a patient whose gastric contents had a pH of 3.5. A single dose of 15 ml of magnesium trisilicate mixture is known to be insufficient to raise a preceding gastric pH of less than 3 to above this level [34].

Initial ventilation of this patient at the Childrens' Hospital, Birmingham, with an FiO_2 of 0.8 and PEEP of 4 cms H_2O, produced a PaO_2 of 7.2kPa (54mmHg). In spite of the use of double strength plasma, thymoxamine and diuretics, the PaO_2 fell over the subsequent hour to 4.53kPa (34mmHg). Clearly this patient would have suffered irreversible cerebral damage had the PaO_2 remained at that level. Increasing the FiO_2 would have exposed the lungs to the danger of oxygen toxicity and increasing PEEP would have further decreased cardiac output with consequent fall in peripheral, pulmonary and renal perfusion. All the clinical and biochemical findings confirm that this patient would have probably died without the timely use of the membrane oxygenator.

Discussion

These two cases illustrate how the serverity of pulmonary aspiration cannot necessarily be assessed by the condition of the patient at the time of aspiration. Constant viligance and blood gas monitoring where aspiration is suspected is the only method whereby early diagnosis and appropriate management can be instituted.

Cameron, Mitchell and Zuidema [12] emphasised that aspiration can occur in a wide variety of clinical situations but the acutely ill comatose patients with underlying neurologic or gastrointestinal diseases with a nasogastric tube in place, are particularly at risk.

Obstetric patients are considered particularly prone to vomiting and regurgitation while under general anaesthesia, but it is not known to what extent they are at greater risk than surgical patients. It is difficult to assess from the literature whether the mortilaty from massive aspiration is higher in the obstetric patient than in others.

The immediate management of pulmonary aspiration has been summarised by [38]. This involves clearance of the airway, the possible use of bronchial lavage, artificial ventilation and the correction of blood volume and acid base balance. Steroids and antibiotics are generally used; various other drugs have been recommended by different authors. Supplemental oxygenation using a membrane oxygenator may have to be considered in patients with severe hypoxia refractory to the usual treatment.

Clearance of the Airway

Prompt removal of aspirated material from the bronchial tree is imperative. This should be performed in conjunction with appropriate posturing and physiotherapy to ensure adequate drainage of the lung segments involved. Efficient suction via an endotracheal tube decreases the amount of gastric juice in the bronchial tree and therefore allows a more efficient buffering action of bronchial secretions; it also removes small particulate and epithelial matter [3]. Aspiration should be continued until the airways are clear but over vigorous suction may lead to bronchial mucosal trauma, subsequent bleeding and a predisposition to infection. Prolonged suction may increase the degree of hypoxia which therefore has to be performed efficiently and for short periods only, the patient being handventilated on oxygen between the periods of suction.

Bronchial Lavage

Lavage has been recommended by various authors [3, 51]. It has been found to be of no benefit experimentally and such treatment may lead to additional areas of involvement. Pulmonary surfactant is known to play an important role in preventing alveolar collapse, maintaining a near-zero surface tension at the air interface, thereby preventing intra-alveolar oedema [49]. Surfactant may be dispersed into the alveoli during bronchial lavage, and may be lifted off its normal position over the epithelial cells during the formation of massive pulmonary oedema [49]. Experimental work suggests that in mild pulmonary oedema, the serum proteins that leak into the alveoli may act in conjunction with surfactant and lower the surface tension of the air fluid interface [27].

Bronchial lavage would seem to be contraindicated in the management of these patients and may be dangerous when used in patients who are hypoxic. Bronchoscopy is not indicated in the management of a patient with aspiration of liquid gastric contents.

Artificial Ventilation

The value of mechanical ventilation in the treatment of the aspiration syndrome has been reported by numerous investigators, both experimentally and clinically [6, 10, 26]. Cameron and his associates [10] used an aspirate with pH 1 and changed a universally fatal model to one with 100 per cent survival after 6 hours intermittent positive pressure ventilation. The PaO_2 in the nonsurvival, nonventilated group of animals ranged between 8 - 9.3kPa (60 - 70 mmHg) in the first eight hours after aspiration. As a result of their experimental studies, they suggested that a clinical trail is warranted in the use of routine positive pressure ventilation after aspiration. The use of a positive end expiratory pressure in order to raise the arterial oxygen tension of severely hypoxic patients on artificial ventilation has now been well accepted. It has been shown to be of value in treating patients with alveolar instability and closure but to be of little use when hypoxaemia is caused by obstructed airways or pneumonitis [39]. Pulmonary aspiration produces congestion of the alveolar capillaries, intra-alveolar oedema and desquammation of the bronchial epithelium, leading to nonventilation of perfused alveoli with attendant hypoxaemia. PEEP in experimentally induced pulmonary oedema does not facilitate mobilisation of interstitial fluid nor improve lung mechanics, but produces an improvement

in gas exchane through inflation of previously collapsed areas [7].

Chapman et al. [16] ventilated dogs with continuous positive pressure ventilation immediately after aspiration of 2 ml/kg body weight of hydrochloric acid and showed that the lung appearance was normal except for small haemorrhages at the posterior bases, in contrast to those that breathed spontaneously where the lungs were haemorrhagic and congested. Experimental evidence is sufficiently convincing to justify the use of C.P.P.V. in all patients requiring ventilation for the aspiration syndrome. PEEP may have an adverse haemodynamic effect and therefore the pressure used should be, if at all possible, 10 cms H_2O or less. Experimentally, cardiac output starts to fall at an end-expiratory pressure of 10 cms H_2O or more – this is probably the effect of increased intrathoracic pressure impeding venous return [35]. PEEP should be used during weaning since it appears to minimise the increase in intrapulmonary right to left shunt which normally occurs during weaning from controlled ventilation [24]. Patients with the aspiration syndrome are frequently hypovolaemic. Attention should be paid to the restoration of a normal circulating blood volume since PEEP will only accentuate the decrease in cardiac output, increase the intrapulmonary shunt and contribute to hypoxaemia. It is possible that the incidence of pneumothorax is increased when using PEEP, but it appears to be a late rather than an early complication when pressures of 10 cms H_2O or less are used and is generally associated with pulmonary sepsis [54].

Oxygen has to be used during artificial ventilation in order to maintain adequate tissue oxygenation. The amount of oxygen available to the tissues is the product of cardiac output and arterial oxygen content.

Available oxygen =
Cardiac output x arterial oxygen x Haemoglobin x 1.34
saturation concentration

It is difficult to define the lower limit of available oxygen which is compatible with survival, but 400 ml S.T.P.D./minute has been suggested by [43] as a level which could be tolerated for a short period during anaesthesia. The arterial oxygen pressure and hence oxygen saturation is invariably reduced in pulmonary aspiration. Kontos et al. [33] have demonstrated in man that arterial hypoxia results in a compensatory increase in cardiac output; this compensation may be compromised if the dynamic blood volume is low. Thus, maintenance of cardiac output by appropriate blood volume replacement plays an important role in attaining adequate tissue oxygenation in the aspiration syndrome. Since there are so many variable factors associated with the aspiration syndrome, it is probably wise to maintain a PaO_2 on C.P.P.V. of 8 - 13kPa (60 - 100mmHg). The FiO_2 should be kept at 0.6 or less during the first 24 hours of C.P.P.V., although there is experimental evidence to suggest that the traumatised lung if exposed to high oxygen percentages within 24 hours of trauma, has a protective mechanism against oxygen toxicity [55]. After 24 hours the FiO_2 should be lowered to 0.5 or less when long term therapy is anticipated [48].

Correction of Blood Volume and Acid Base Imbalance

It is well recognised both experimentally [16] and clinically [40] that severe pulmonary aspiration is associated with a fall in cardiac output, probably reflecting a hypovolaemic state secondary to pulmonary oedema [2]. In spite of a low dynamic blood volume, the central venous pressure may be raised, thereby giving the impression of normovolaemia; this is probably related to pulmonary venospasm [6] and not to bronchospasm (which may be absent) as suggested by [40]. A low cardiac output may increase the already existing intrapulmonary shunt, and in the presence of a raised central venous pressure (i.e. greater than 10 cms H_2O at the midaxillary level measured in the horizontal plane) a fluid challenge of 200 mls of an appropriate volume expander should be given (Sykes 1963). PAWP (if the catheter is correctly placed) generally accurately reflects left atrial pressure but as PEEP is increased may be totally misleading. Lozman and coworkers [36] showed that at 5 cms H_2O PEEP or less, there was a statistically significant correlation between left atrial pressure and PAWP measured with a Swan Ganz catheter; this was not so at PEEP levels of 10 cms H_2O or above.

The fluid used for volume replacement should be carefully considered. Although no documented evidence could be found, protein loss into the alveoli will inevitably lead to a fall in serum proteins and a decrease in plasma osmotic pressure. This, in conjunction with destruction of lung surfactant [11] will increase the tendency towards pulmonary oedema. It is known that volume replacement with cryralloid solutions leads to a large increase in the interstitial space and that this increase is rapid once the serum albumin falls below 25 g/l [14]. Additionally the resultant haemodilation may increase the tendency towards the 'wet lung syndrome' [14] producing a further deterioration. Volume replacement should therefore be with dried plasma, plasma protein fraction or salt free albumin. An haematocrit of around 30 per cent should be aimed for, since oxygen transport capacity is known to be highest at this value [31]. Whole blood should be given if the haematocrit is below this level.

A metabolic acidosis is frequently present in massive pulmonary aspiration being related to poor peripheral perfusion secondary to hypovolaemia; the arterial oxygen level has little effect on the serum lactate until it has reached 4.8kPa (36mmHg) or less [13]. Brown, Kim and Shoemaker [8] showed that the onset of increase in pulmonary vascular resistance in hypovolaemic shock was pH dependent and that two-thirds of the rise could be abolished by maintaining venous pH in the normal or slightly alkalotic range during the hypotensive period. Timely and appropriate dosage of intravenous sodium bicarbonate is therefore important in the management of the aspiration syndrome.

Steroids

Parenteral steroids have been recommended as anti-inflammatory agents since 1955 [20] and have consequently been universally used in the management of patients with pulmonary aspiration. All case reports lack a control. The series of 40 patients with recognised aspiration of liquid gastric contents reported by [41] did not receive steroids and all survived. Downs et al. [21] and [16] have made an excellent evaluation of steroid therapy in experimental aspiration pneumonitis. It appears that once a maximal tissue response has occurred, steroid therapy is not beneficial. Downs et al. [21] found that if steroids play a beneficial role in treatment, it seems to be limited to animals that aspirate material in the narrow pH range of 1.5 - 2.1. Cameron and coworkers [12] treated 47 patients suffering from aspiration pneumonia with steroids; there was no apparent effect on the overall mortality. The dosage and type of corticosteroid to be used appears equally controversial. It is probably wise to use a steroid with minimal saltretaining properties such as methyl prednisolone sodium succinate and to use large doses (30 mg/kg body weight intravenously 8-hourly) for a limited period of 48 - 72 hours. Large doses of prednisolone may lead to an increase in urine output and hypokalaemia, so care must be taken to maintain an adequate circulating blood volume. Potassium supplements are invariably required.

Antibioticas

Workers have stated that steroids alter the tissue response to infection and that its use in aspiration should always be accompanied by antibiotic therapy [19]. Bacterial infection appears to have little part to play in the initial illness [4, 9] and it is probably wise to withhold antibiotics until anaerbic and aerobic cultures of the tracheal aspirate are available. Bartlett, Gorbach and Finegold [5] performed a prospective study on 54 cases of pulmonary infection following aspiration. Specimens were either transtracheal aspirates, empyema fluid or blood. Anaerobic bacteria were recovered in 93 per cent of patients and were the only pathogens in 42 per cent, the predominant species being Bacteroides, indicating that anaerobes play a key role in most cases of infection following aspiration.

Other Drugs

Digoxin should be used if there is evidence of cardiac failure and should be particularly considered in patients known to have preceding myocardial damage. Bronchospasm may be a prominent initial feature of pulmonary aspiration and antispasmodics have been widely recommended in treatment [37]; their use, however, has to be carefully considered in relation to their pharmacological activity.

Most bronchodilators increase minute volume and alveolar ventilation, but hypoxaemia may become worse as a result of an increase in cardiac output, resultant increase in pulmonary blood flow and an intensification of the ventilation/perfusion deficit [44]. This situation has been reported following aminophylline [46], adrenaline [47] and isoprenaline [32].

These bronchodilators may also produce an increase in heart rate or cardiac dysrhythmias and are dangerous when used in an hypoxic patient. Salbutamol, however, has been found to increase the FEV to a greater degree than isoprenaline with little increase in heart rate [45] and an insignificant increase in oxygen uptake in doses up to 1 uG/kg body weight infused over 5 minutes [25]. Cardiac output has been shown to increase with salbutamol and is probably related to an increase in blood flow through the peripheral circulation [25]. Salbutamol, when carefully titrated intravenously, may be of value in the management of bronchospasm in the aspiration syndrome but should only be used once the dynamic blood volume has been restored. Because of its action on the peripheral vasculature, a further 'top up' of blood volume may be required during its use.

The Membrane Oxygenator

Extracorporeal oxygenation of the blood in acute respiratory failure following trauma ("shock lung") was first successfully employed by [29] and since then the same unit has reported a further three long term survivors (two "shock lung", one fat embolism syndrome) in a total series of twenty eight patients [28]. Chang et al. [15] described the successful support given to a young woman with post-operative pulmonary embolism.

To date the best results have been achieved in patients with "shock lung" or pulmonary fat embolism. The long-term survival figures, for all types of cases, reported from centres in the U.S.A. are not high (15 out of 130 patients, [39]). A prerequisite for the use of extracorporeal oxygenation is the presence of a condition which is theoretically reversible and the absence of a potentially irreversible complicating disease. If the presently accepted treatments of acute respiratory failure are not producing clinical, haemodynamic and biochemical improvement of a patient, extracorporeal oxygenation should be considered. Criteria, which may be too rigid and erring on the side of conservatism, have been stated in an Editorial [22] and are:

1. An FiO_2 of 1.0 for longer than 48 hours.
2. A positive end-expiratory pressure in excess of 10 cms H_2O, at a high FiO_2, for longer than 48 hours.
3. A PaO_2 of less than 6.6kPa (49.5mmHg) for more than 24 hours. The technique should be employed before the clinical condition of the patient has become desperate. It is preferable that it should be used only in centres familiar with extracorporeal circulatory techniques [23].

Conclusion

In the light of our experience and preceding discussion, the following conclusions can be made.

1. Early diagnosis and appropriate management is essential in order to decrease the mortality from pulmonary aspiration.

2. The ultimate degree of pulmonary destruction cannot be gauged at the time of aspiration. Experimentally the prognosis does not necessarily correlate with blood gas changes occurring during the first 6 hours following aspiration.

3. Continuous positive pressure ventilation appears to be one factor that radically alters the prognosis, and experimental data is sufficiently convincing to justify its use in all patients who have aspirated. FiO_2 should be maximally 0.6 for the first 24 hours and then reduced to 0.5 or less. The level of PEEP used should be as low as possible in order to achieve a PaO_2 between 8.0 - 13.33kPa (60 - 100mmHg). When weaning the patient off PEEP, the level should be reduced slowly; abrupt stoppage may lead to a recrudescence of pulmonary oedema [54] (in Discussion).

When breathing spontaneously the patient should be allowed to breath against a PEEP of 2 - 5 cms H_2O.

4. Hypovolaemia is a well recognised feature of pulmonary aspiration. Central venous (CVP) and pulmonary artery wedge pressure (PAWP) monitoring may not be reliable indications of dynamic blood volume. In the presence of a raised CVP volume depletion can be assessed by response to a fluid challenge. PAWP appears to correlate fairly closely with left atrial pressure until PEEP reaches a level of 10 cms H_2O or above. Correct placement may be difficult. An incorrectly placed catheter in the pulmonary artery will reflect pulmonary vascular resistance and not left atrial pressure. Crystalloid solutions should not be used for volume replacement. A metabolic acidosis should be corrected with intravenous sodium bicarbonate.

5. Steroids have no proven value in the aspiration syndrome but until there is further evidence, should be used in large doses (prednisolone sodium succinate 30mg/kg body weight intravenously 8-hourly) for a period of 24 hours.

6. Antibiotics should not be used until anaerobic and aerobic cultures of the tracheal aspirate are available. The antibiotic course should be limited to 5 days.

7. Antispasmodics should not be used unless there is overt bronchospasm at the time of aspiration. Salbutamol titrated intravenously in an adult dosage of 1 - 5 uG/minute so that the pulse rate does not rise 10/minute above the basal value, may be considered. It should be used with caution in patients with pulse rates greater than 130/minute and should not be used until hypovolaemia has been corrected.

8. Membrane oxygenator. Membrane oxygenation is indicated if, in spite of the above measures, the PaO_2 fails to be maintained above 6.6kPa (49.5mmHg). It may also be indicated in patients where, because of PEEP and high ventilatory airway pressures, the PaO_2 cannot be maintained above 6.6kPa (49.5mmHg) without a fall in cardiac output and resultant inadequate tissue perfusion.

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