MEDIASTINAL EMPHYSEMA AS A COMPLICATION OF ANAESTHESIA—THE SUCCESSFUL MANAGEMENT OF 3 CASES*

M. J. RORKE, B.Sc., M.MED. (ANAES.) (CAPE TOWN) AND C. MOSS, F.F.A.R.C.S. (ENG.), F.F.A.R.C.S. (I), *Department of Anaesthetics, University of Cape Town and Groote Schuur Hospital*

The subject of mediastinal emphysema is not a new one; occasional cases with dramatic symptoms were reported a century or more ago. However, during the past 30 years, experimental investigation and clinical observation have extended our knowledge.

It is true that in most instances mediastinal emphysema is a benign condition, but, even so, it is important that the physician should not confuse it with serious diseases to which it may bear a close resemblance. However, not infrequently grave and threatening symptoms and signs arise, and then a correct appraisal of the situation with prompt action may be life-saving.

During general anaesthesia particularly, mediastinal emphysema can manifest as an acute emergency; it may thus serve a useful purpose to review aspects of this important condition. The 3 case reports illustrate varying aetiological factors during anaesthesia, as well as the immediate resuscitative measures to be adopted.

Mediastinal emphysema is that condition in which gas is present in the mediastinum. This gas may be air, or, during anaesthesia, any of the gases used in the conduct of the anaesthetic.

CLINICAL DESCRIPTION

Aetiology and Pathogenesis

Air may reach the mediastinum by any of 4 routes:

(a) *Along the fascial planes of the neck.*[1,2] During thyroidectomy, tracheotomy or tonsillectomy air might enter the deep fascial planes at a high point and dissect downwards to enter the mediastinum. This train of events might also follow deep wounds of the neck, pharynx and buccal cavity. It is considered more likely, however, that mediastinal emphysema following such operations is often a consequence of anaesthesia and partial respiratory obstruction rather than direct aspiration from the operative site.

(b) *Through a perforation* of the trachea, bronchus or oesophagus into the mediastinum. Disease or trauma occasionally causes rupture of the lower trachea or a main bronchus, and sometimes mediastinal emphysema is seen after lobectomy or pneumonectomy. If air enters from the oesophagus, symptoms of infection may obscure those of air in the mediastinum.

(c) *From the retroperitoneal or intraperitoneal spaces.* Air may travel along the aorta or oesophagus and enter the mediastinum. Retroperitoneally, the injection of air into the perirenal tissues to facilitate outlining the kidneys may be responsible, while the intraperitoneal route may be responsible during the procedure of peritoneoscopy or following perforation of stomach or intestine.

(d) *From the interstitial tissues of the lung.* This is the common route of entry. Certain marginal alveoli are separated from the bronchial and vessel walls of the lung by a space containing tissue fluid and connective tissue. This space is known as the peribronchial or perivascular connective tissue sheath, and if air is introduced into this sheath, the condition of pulmonary interstitial emphysema exists.

The mechanism by which alveolar rupture occurs, and the spread of interstitial emphysema of the lungs, have been described by Macklin from his experiments on cats.[3,4] An increase in the size of marginal alveoli by hyperinflation, with or without a raised intra-alveolar

*Date received: 16 September 1968.

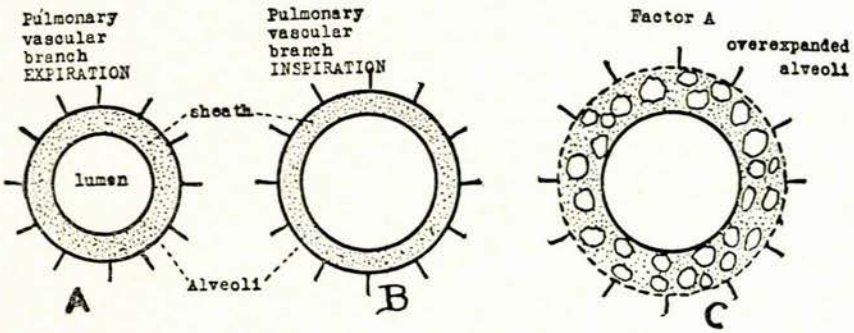

Fig. 1. The outer circle represents the bases of a ring of 12 alveoli around a blood-vessel. The sides of the alveoli radiate like spokes of a wheel. The inner circle represents the endothelial wall of a blood-vessel. The stippled area between the two circles is the sheath of a blood-vessel. A: Conditions at the end of quiet expiration. B: Conditions during inspiration. C: Conditions in overinflation. The inner circle of the vessel wall is dilated as occurs in normal inspiration, but the alveoli are much distended, so that the circumference of their bases is much greater. This puts a tension on the sheath between overexpanded outer circle and normally expanded inner circle. The alveolar bases break, as shown by the dotted lines, and air escapes from the alveoli into the sheath as shown by the large bubbles.

pressure or a decrease in the size of adjacent pulmonary blood-vessels, leads to rupture of alveolar bases (Fig. 1). This causes a progressive passage of air from the alveoli to the pulmonary perivascular connective tissue sheaths. Air continues to pass from the former to the latter, then follows the path of least resistance as it travels along the sheaths in blebs of increasing size towards the hilum of the lung. Continuing leakage of air from the alveoli causes the accumulation of air in the mediastinum (Fig. 2).

The causes of pulmonary interstitial emphysema (and hence mediastinal emphysema) are as follows:[5]

I. *Increase of intrapulmonary pressure.*
   (*a*) Intermittent positive-pressure respiration (IPPR):
      (*i*) During anaesthesia.
      (*ii*) During neonatal resuscitation.
   (*b*) Straining with the glottis closed as in partial or complete laryngospasm.
   (*c*) Occlusion, partial or complete, of the trachea or bronchi, particularly with cough.
      (*i*) Complication of a foreign body in a bronchus.
      (*ii*) Complication of tenacious inflammatory material produced by inflammatory diseases of the lungs such as pertussis, asthma, laryngotracheabronchitis.

II. *Trauma.*
   (*a*) Injury to the chest with or without fracture of the ribs (i.e. closed).
   (*b*) Operations on the chest, particularly the induction of pneumothorax.
   (*c*) Complication of penetrating wounds of the trachea and bronchi.

III. *Spontaneous rupture of the alveoli.* It is suggested that there may be an inherited defect of tissue quality. The precipitating conditions in the presence of the above causes fall into three categories:
   (*a*) There is first atelectasis of some part of the lung followed by hyperinflation in adjoining regions of the same or in the opposite lung.
   (*b*) There is a general overinflation with or without increased intra-alveolar pressure.
   (*c*) There is a decreased blood flow through the pulmonary blood-vessels, usually with increased intra-alveolar pressure or with hyperinflation.

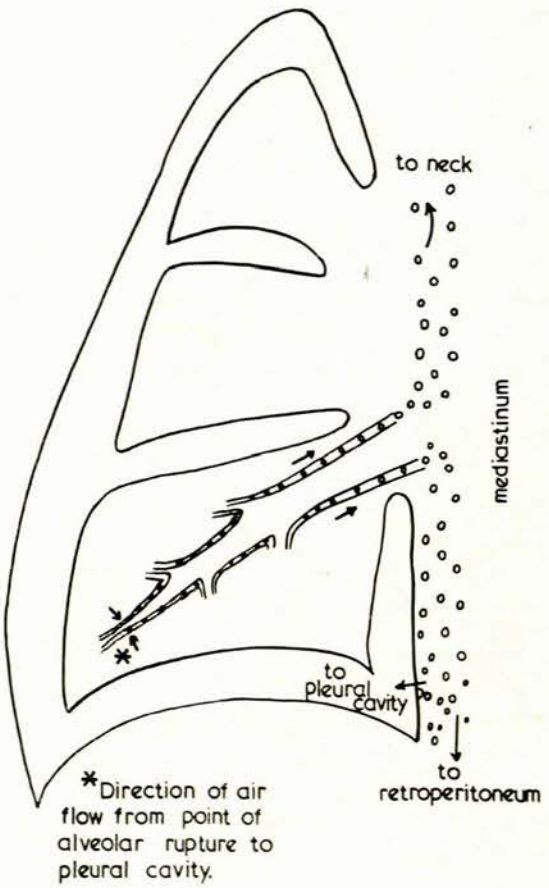

Fig. 2. Diagram showing the pathway of air from the air spaces of the lung to the mediastinum via the perivascular sheaths of the lung. Only the artery is represented. Bubbles of air in the sheaths and connective tissues of the mediastinum are shown as highlighted spherules. One of the many points of invasion of the perivascular sheaths is indicated by the arrows in the lung interior.

The escape of air from the mediastinum.[6] When sufficient pressure develops, air always escapes from the mediastinum. Sometimes, however, it escapes when there is little increase in pressure, and at other times it is retarded when there is evidence of a great increase in pressure. The avenues of escape are:

(*i*) Into the root of the neck with the production of subcutaneous emphysema, or, as it is inaccurately known, surgical emphysema.
(*ii*) Into the retroperitoneum along the aorta or oesophagus.
(*iii*) Rupture through the mediastinal pleura into the ipsilateral and/or contralateral pleural cavity to produce possibly a bilateral pneumothorax.
(*iv*) Laterally into the interstitial tissue of the opposite lung or unbloated parts of the same lung, splinting them.

Symptoms

Minor degrees of mediastinal emphysema might increase progressively in the postoperative period. The progression of benign mediastinal emphysema to a rapidly fatal malignant stage may be aborted if the symptoms are appreciated and therapy is started. The nature and severity of the symptoms depend in general on the degree of distension of the lung and mediastinum, and the accessibility of escape channels.

Dyspnoea. Respiration may be seriously interfered with by the splinting action of the air in the connective tissue of the lung.

Cyanosis and venous stasis. The circulation may be hampered by means of collapse of the pulmonary vessels, a decreased venous return, cardiac tamponade and impairment of coronary blood flow.

Chest pain. Because of its varied characteristics, this most frequently encountered symptom has often been mistaken for angina pectoris or myocardial infarction.

Signs

Diminished cardiac dullness or hyper-resonance over the sternum and precordium is best elicited with the patient recumbent. In the upright position and particularly when the patient leans forward, this sign may dis-

appear. In many cases the manoeuvre of alternate percussion in the recumbent and forward position may be of decided help diagnostically.

Mediastinal crepitations. The presence of air in and about the mediastinal tissues produces sounds that have been variously described as crackling, clicking, bubbling, crunching, grinding, rasping or snapping. Apart from these subjective characterizations, the sound produced by the bubbles or air has certain objective features. It is generally most easily detected in the left lateral position when recumbent, and during the expiratory phase of respiration. It is less audible when the patient is upright. The sound accompanies systole and diastole, but it may be louder during systole. When the presence of mediastinal emphysema is suspected the heart should be listened to carefully as the patient is moved from one position to another.

Evidence of increased mediastinal pressure. Engorged veins, a rapid pulse, fall in blood pressure, pulmonary oedema and circulatory failure all result because of several factors acting alone or together. The venous congestion on the systemic side is brought about by the limited respiratory movements and by the compression of the pulmonary arteries and veins, preventing blood from getting into the left side of the heart. The venous return from the systemic circuit is also impaired.

The heart is unable to fill owing to the distension of the surrounding lungs compressing it, and to the pressure upon it of bubbles of air in the mediastinum. There is a rise of pressure in the mediastinum.

Pneumothorax. Of interest regarding the presence of pneumothorax is the fact that it is most often on the left side. No adequate explanation for this has ever been offered. In many cases the pneumothorax may be small and escape detection. There is an intimate relationship between mediastinal emphysema and pneumothorax—the latter being secondary to the air in the mediastinum. Very often they occur together. The important clinical point is that when pneumothorax exists, the presence of mediastinal emphysema may be overlooked.

Air reaches the pleura by way of a tear in the delicate wall of the mediastinum, often demonstrable in the posterior mediastinal wall at varying levels, most commonly just above or below the hilar region. There is also a region in the posterior mediastinum between oesophagus and aorta in which two layers of mediastinal pleura are in apposition, and rupture at this point may put the two pleural cavities into communication, producing bilateral pneumothorax. When pneumothorax occurs following a blow on the chest, without fracture of the ribs or an open wound, and at the same time subcutaneous emphysema in the neck is evident, there can be little doubt that the sequence of events has been rupture of the alveoli, pulmonary interstitial emphysema, mediastinal emphysema with escape of air into the neck, and perforation of the mediastinal pleura with the production of pneumothorax. When the pneumothorax is contralateral, this conclusion is inescapable.

Subcutaneous emphysema. The detection of this sign needs little comment. If subcutaneous emphysema appears in the neck under circumstances in which there is no other cause for it, then we are assured that mediastinal emphysema is present. It is conclusive evidence of mediastinal emphysema and sometimes the only evidence.

Electrocardiographic changes. In the majority of cases ECG changes are absent. Approximately 25% of records show positive findings, but no characteristic pattern is presented.

Radiological examination of the chest is valuable in establishing the diagnosis. It should be emphasized that several studies may be necessary and the diagnosis cannot be ruled out on the basis of a single film taken in the postero-anterior projection. Lateral views of the chest are essential to demonstrate air trapped behind or in front of the heart. Air in the mediastinum is often visible as streaks of increased radiance running along the borders of the heart. Extensive mediastinal emphysema may be present although a lateral film does not always show a substernal collection of air.

CASE REPORTS

The following 3 case reports illustrate aetiological factors, methods of presentation, and the immediate resuscitative measures mandatory when faced with this complication during anaesthesia.

Case 1

A healthy 2½-year-old child, weighing 32 lb., required suturing of lacerations about the eye. Anaesthesia was induced with 500 mg. of 5% thiopentone administered rectally, and was deepened with the patient breathing nitrous oxide, oxygen and ether. Endotracheal intubation using a 5·5-mm. portex orotracheal tube was easily effected without trauma and under direct vision. Following confirmation of correct positioning and patency of the airway, a T-piece circuit consisting of a Cobb's connection was attached, with a total fresh gas flow setting of 8 litres/min. Almost immediately the child was noticed to be holding his breath (wrongly ascribed to too light a level of anaesthesia), and within seconds subcutaneous emphysema of his neck and face became evident. With the minimum of delay, the tube was jerked out, with the subsequent explosive escape of a large volume of gas from between the lips, and the simultaneous deflation of the chest. Adequate spontaneous respiration started shortly afterwards and 100% oxygen was given by mask.

Examination revealed subcutaneous emphysema of the anterior chest wall, neck and face, extending up to the temples. The colour, pulse, and blood pressure of 100 mm.Hg were satisfactory; the heart sounds were distant, as were the breath sounds, and air entry was less on the right than on the left side.

A needle connected to an underwater drain was inserted into the 3rd right intercostal space in the midclavicular line. Bubbles escaped through the underwater seal, with marked improvement of respiratory excursions.

X-ray of the chest at this stage showed bilateral pneumothoraces (Fig. 3) and extensive subcutaneous emphysema of the chest wall. When there was no further escape of gas the needle was withdrawn, the surgery completed and the patient returned to the ward where he was given large doses of antibiotic. Recovery was rapid and complete and X-ray of the chest 36 hours later was completely normal.

Subsequent examination of the anaesthetic apparatus revealed a cork stopper embedded deeply inside the Cobb's connector. This effectively prevented the escape of gas from the open limb of the T-piece, thereby subjecting the patient's lungs to an uninterrupted flow of fresh gases.

This case illustrates the aetiological factor of sustained positive intrapulmonary pressure with consequent over-inflation due to apparatus failure. It also demonstrates the spread of the anaesthetic mixture via the mediastinum to the head and neck, retroperitoneally, and to the chest wall with the production of bilateral pneumothoraces. It further highlights the successful immediate resuscitative measures instituted.

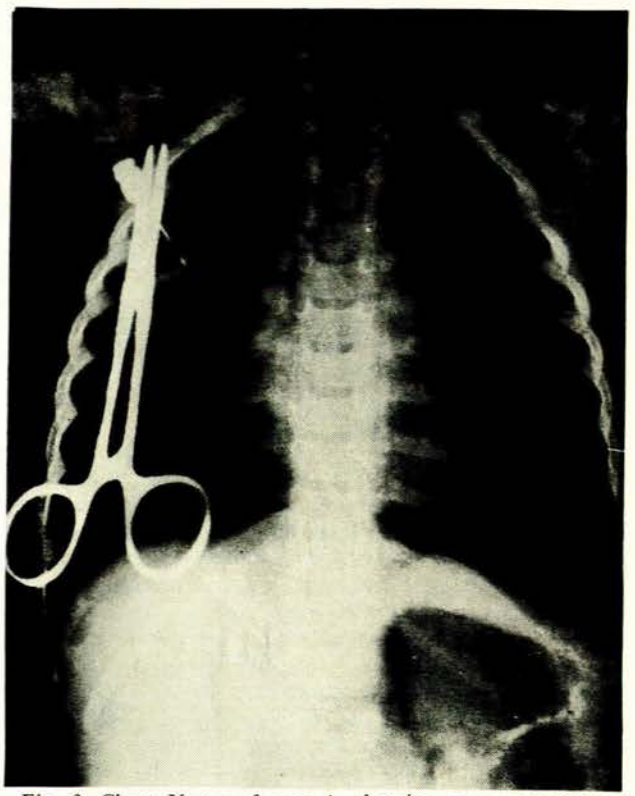

Fig. 3. Chest X-ray of case 1, showing severe pneumothorax.

Case 2

A female aged 25 years was to have an elective caesarean section at 38 weeks' gestation. The patient was small in stature (4 ft 10 in.) and weighed 115 lb. at term; a previous caesarean section had been necessary for cephalopelvic disproportion.

After intravenous premedication with atropine, 0·6 mg., anaesthesia was induced with thiopentone, 250 mg. Succinylcholine, 50 mg. intravenously, permitted endotracheal intubation easily and correct positioning of the tube was confirmed by auscultation.

It was planned to passively hyperventilate using an Engstrom volume-guaranteed ventilator. A fresh gas flow of 17 litres of premixed nitrous oxide and oxygen (50% nitrous oxide, 50% oxygen) was delivered at a frequency of 20 excursions/min. and a maximum inspired pressure of 45 cm. of water. Each inspired tidal volume was thus 850 ml. To the inspired gases was added 0·1% methoxyfluorane via a Pentec (Cyprane) vaporizer. d-Tubocurarine, 25 mg., was injected intravenously to provide adequate muscle relaxation. The radial artery was cannulated with a No. 18 Rochester disposable needle from which samples were taken for blood gas studies to coincide with certain selected events during surgery. These investigations were undertaken as part of a planned research programme investigation—the effects of hyperventilation during caesarean section. The intra-arterial blood pressure was continuously measured using a Statham—P23B pressure transducer. Surgery proceeded uneventfully until the delivery of a healthy 4 lb. 8 oz. female (Apgar 4 at 1 min.; 7 at 3 min.; 9 at 5 min.). An arterial blood sample was taken to coincide with delivery and ergometrine, 0·5 mg., was given intravenously. Twenty-five seconds after this the following occurred simultaneously:

1. The surgeon noted the progressive rapid forward protrusion of the uterus and adnexa and suggested that the patient was 'pushing and inadequately relaxed'.

2. The anaesthetist observed the alarming spectacle of the patient's face and neck swelling rapidly, accompanied by proptosis of the eyes and projection of the tongue.

3. The blood pressure trace showed a rapid fall of systolic and diastolic peak pressure and a reduction of pulse pressure until the pressure trace became virtually a straight line at ±30 mm.Hg.

The ventilator was immediately disconnected from the endotracheal tube, with an audible release of gas under pressure. Gentle manual intermittent positive pressure with oxygen via a closed circuit Boyle's machine was started after an interval of 20 seconds.

Auscultation revealed diminished air entry of both lungs with adventitious sounds suggestive of bronchospasm. Gross subcutaneous emphysema was present in the head, neck and face. A diagnosis of mediastinal emphysema with extension to the head, neck and abdomen, and with the production of bilateral pneumothoraces was made (Fig. 4). Following a chest X-ray, bilateral intercostal drainage in the second interspace in the midclavicular line allowed the trapped gas to escape, with immediate increase in lung compliance. Subsequent X-rays showed relief of the bilateral tension pneumothoraces, but the subcutaneous emphysema of the head and neck persisted. The patient awoke promptly, following reversal of the muscle relaxant and discontinuance of the anaesthesia. Broad-spectrum antibiotics were prescribed and the intercostal underwater drains were removed 4 hours after insertion. Further convalescence was uninterrupted.

Table I shows the results of blood gas analysis.

TABLE I. BLOOD GAS ANALYSIS

Entity	*Pre-operative*	*At delivery*	*Before intercostal drainage*	*After intercostal drainage*
pH	7·361	7·460	7·270	7·406
PCO_2	29·1	19·7	32·0	20·4
BE	−7·9	−8·7	−11·3	−10·4
Std.HCO_3	17·7	17·2	15·3	16·0
BB	37·7	36·2	33·6	35·4
PO_2	106·5	197·9	78·5	206·0
PCV	36·5	32·5	42·0	41·2

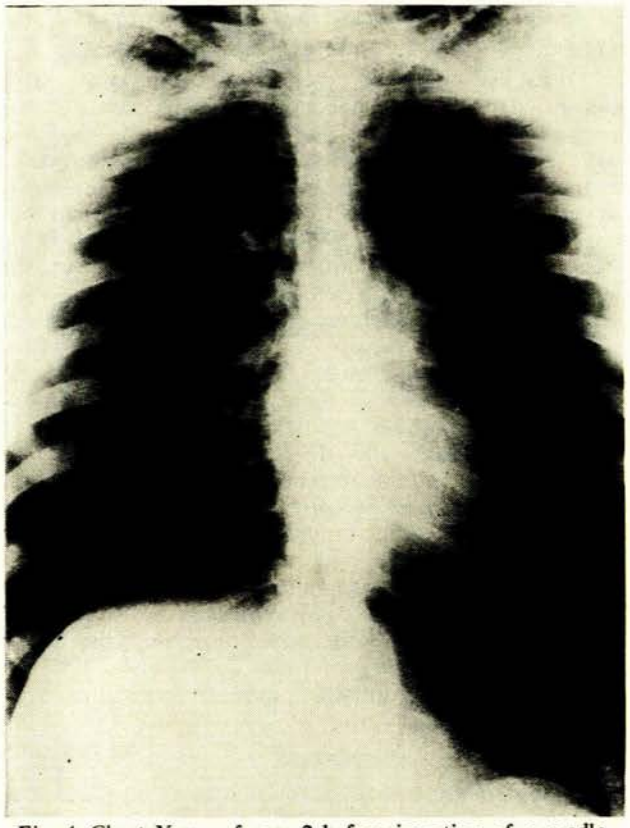

Fig. 4. Chest X-ray of case 2 before insertion of a needle.

This case serves once again to illustrate the underlying aetiological factor—increased intrapulmonary pressure with overinflation of alveoli and rupture leading to mediastinal emphysema. The extensions of the air from the mediastinum were clearly illustrated, the neck presenting as subcutaneous emphysema, the intraperitoneal extension lifting up and pushing forward the uterus and adnexa and the chest extension presenting as bilateral tension pneumothoraces. A factor in the rapidity of resolution is possibly the solubility of the gases involved. This case further illustrates the dilemma that faces the anaesthetist once this complication has occurred—the curarized patient needing IPPR which, in itself, may potentiate the extension of the mediastinal emphysema. However, this dilemma is fortunately resolved by the insertion of a needle to relieve the tension pneumothorax.

Case 3

A 20-year-old White female required an examination under anaesthesia, dilatation and curettage and peritoneoscopy. Pre-operative examination revealed nothing abnormal. Premedication was instituted with Valium, 10 mg., and atropine, 0·6 mg., intramuscularly 1 hour preoperatively. Anaesthesia was induced with thiopentone, 300 mg., and Scoline, 50 mg., allowed an atraumatic intubation using a No. 9 cuffed endotracheal tube. Correct positioning of the tube was established and spontaneous respiration returned normally. For the dilatation and curettage and insertion of a peritoneoscope, spontaneous respiration through the circle absorber of the Boyle's machine (with spill), using an 8-litre flow was maintained. A nitrous oxide oxygen mixture (70%/30%) with 0·5% halothane was administered. Monitoring of pulse rate, blood pressure and tidal volume was carried out. After the peritoneoscope had been inserted intra-abdominally, the patient was tilted steeply head down, and respiration was then controlled using the previous mixture. Air was pumped into the abdominal cavity by the surgeon with a sigmoidoscope bulb connected to the peritoneoscope. While auscultating the chest during this procedure the anaesthetist noticed decreased heart sounds and absent breath sounds on the left. At the same time it became very difficult to ventilate the patient—the clinical impression being one of partial respiratory obstruction. No improvement was achieved by deflating the cuff or partially withdrawing the endotracheal tube or by levelling the table. The patient remained pink and the pulse satisfactory. The surgeon was informed and decided to abandon the procedure, the bulb was disconnected from the end of the peritoneoscope and air rushed out with a gush and the peritoneoscope was then withdrawn. It then became easier to ventilate the patient and although air entry was present on the left side, it was much reduced. Subcutaneous emphysema of the neck, extending to the left cheek, was noticed. As the pulse rate, blood pressure and colour were all satisfactory, the patient was extubated. Consciousness returned rapidly. X-ray examination at this stage revealed air in the soft tissues of the neck, chest and abdomen and the presence of a left pneumothorax (Fig. 5). The patient appeared comfortable

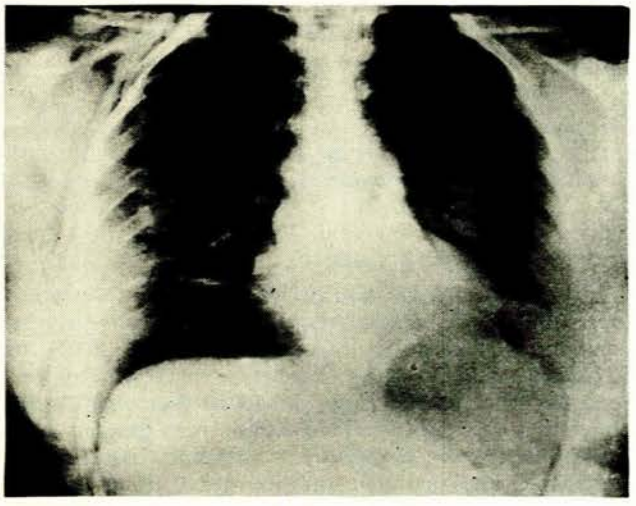

Fig. 5. Chest X-ray of case 3.

with a normal colour but showed some tachypnoea. An underwater drain was inserted into the second left interspace in the midclavicular line. An X-ray 4 hours later disclosed almost complete re-expansion of the left lung. The drain was removed within 48 hours, a check X-ray having shown complete absorption of the pneumothorax.

This case illustrates the introduction of air intraperitoneally, the extension to the mediastinum, and the production of a left-sided pneumothorax.

DISCUSSION

The basis of reducing the incidence of mediastinal emphysema and its serious complications is to recognize and avoid the predisposing causes. Scrupulous attention must be paid at all times to the avoidance of respiratory obstruction so as to eliminate high pressures in the respiratory tree. The anaesthetic apparatus in use must be blameless in this regard, from the use of the widest bore non-kinkable endotracheal tube possible to the elimination of expiratory resistance by valves or any other part of the apparatus. The often-heard plea for early tracheostomies when laryngeal obstruction is progressing is re-emphasized; during the procedure of elective or emergency tracheostomy it is essential to provide a perfectly clear airway. Meticulous care must be exercised in the use of controlled respiration using intermittent positive pressure. General overinflation with or without increased inter-alveolar pressure must be carefully guarded against.

Bargh,[7] presenting two cases of pneumothorax in the neonate, drew attention to the work of Day and his colleagues. Day *et al.*[8] stated that injury to a lung arises from distension and not from pressure, and that distension is proportional not only to pressure but also to the duration of pressure. A given pressure applied for a longer time will produce greater expansion of the lungs than the same pressure for a shorter time. It thus behoves anaesthetists employing IPPR to be conscious of the inflatory pressure used as well as the duration of inflation. Trauma to the respiratory passages naturally should be avoided in the use of airways and laryngoscopes during intubation or tracheobronchial toilet. If the mucosa is abraded it is advisable to exert extra care during inflation of the lungs.

Golding *et al.*[9] presented a case of mediastinal emphysema with extension believed to be due to laceration of the pharynx during attempted passage of a nasogastric tube. The subsequent use of nasal oxygen in the immediate postoperative period, with coughing, may have aggravated or even initiated this condition.

As with any complication occurring during anaesthesia, successful management is frequently related to early recognition and energetic treatment. It should firstly be established that there is no obstruction of the airway, and the flow of anaesthetic gases should be momentarily disconnected to ascertain that no unduly high gaseous pressure is entering the respiratory passages. If respiration is spontaneous and rhythmic, the occurrence of mediastinal emphysema may present insidiously as subcutaneous emphysema or as a pneumothorax. When subcutaneous emphysema makes its appearance during general anaesthesia, the anaesthetist should assume the presence of mediastinal emphysema and the possible development of a pneumothorax or a tension pneumothorax. Signs of respiratory difficulty with concomitant poor air entry and respiratory exchange will alert the anaesthetist to the presence of a pneumothorax. If the patient is not too distressed and signs of cardiac embarrassment are minimal, time is available for X-raying the chest and reassessment of the situation and consultation with thoracic surgical colleagues. If the presenting symptom, however, is a tension pneumothorax, then the management is the same as outlined below during IPPR.

If IPPR is employed, mediastinal emphysema may again be signalled by subcutaneous emphysema or the signs of pneumothorax, or cardiac embarrassment. Gentle manual positive pressure with oxygen should be commenced immediately. If the cardiac embarrassment is minimal, there is still time to obtain X-rays and confirm the diagnosis. Constant observation and monitoring should alert the anaesthetist to the progression of the pneumothorax and the development of tension. If, however, the mediastinal emphysema presents as a tension pneumothorax with severe cardiac embarrassment as evidenced by pulse and blood pressure, the widest needle available must be inserted through the 2nd or 3rd intercostal space in the midclavicular line to allow the air in the chest to escape and the lung to re-expand. This manoeuvre is an acute emergency and may be life-saving. A No. 2 Braunula may be conveniently used; if it is more elective a trocar and cannula may be available. The needle should be connected via tubing to underwater drainage. A check X-ray should now be available. If there is still either a large pneumothorax or a continued air escape through the underwater tube, then the needle and tubing should be left connected for 24 hours after the air leak has ceased. Antibiotics must be prescribed.

SUMMARY

The subject of mediastinal emphysema as a complication of anaesthesia is presented. Following alveolar rupture due to increased intra-alveolar pressure or hyperinflation, air tracks to the mediastinum may spread to the neck, abdomen, lungs or pleural cavity. The symptoms and signs are discussed. Three case reports detailing methods of presentation and management are described. The development of a tension pneumothorax may cause severe cardiac embarrassment; emergency treatment is best carried out by inserting a wide-bore needle into the midclavicular line in the 2nd or 3rd interspace, thus initiating re-expansion of the lung. Anaesthetists must be aware of this complication of mediastinal emphysema during anaesthesia, since rapid diagnosis and energetic treatment may avert tragedy.

We wish to thank our colleagues in the Departments of Anaesthetics, Radiology and Thoracic Surgery for their willing co-operation; Prof. A. B. Bull, Head of the Department of Anaesthetics, for his encouragement and advice; and Dr J. G. Burger, Medical Superintendent of Groote Schuur Hospital, for permission to publish these findings.

REFERENCES

1. Forbes, G. B., Salmon, G. and Herweg, J. T. (1947): J. Pediat., **31**, 172.
2. Works, W. P. (1943): Arch. Otolaryng., **37**, 26.
3. Macklin, C. C. (1939): Arch. Intern. Med., **64**, 912.
4. Macklin, C. C. and Macklin, M. T. (1944): Medicine (Baltimore), **23**, 281.
5. Hammon, L. (1945): J. Amer. Med. Assoc., **128**, 1.
6. Nicholas, J. N. (1958): Brit. J. Anaesth., **30**, 63.
7. Bargh, W. (1964): *Ibid.*, **36**, 456.
8. Day, R., Goodseller, A. M., Apgar, V. and Beck, G. J. (1952): Pediatrics, **10**, 593.
9. Golding, M. R., Urban, B. J. and Steen, S. N. (1966): Brit. J. Anaesth., **38**, 482.