MASSIVE PULMONARY COLLAPSE DURING THORACOTOMY

BY

D. D. IMRIE,* R. M. A. MCCLELLAND† AND W. B. SHARDLOW‡

Department of Anaesthetics, The Welsh National School of Medicine, Cardiff

SUMMARY

A case is described of an incident of acute massive collapse of one lung which was seen to occur in a 28-year-old woman at operation for mitral valvotomy. The endotracheal tube was satisfactorily placed and no abnormality was found on bronchoscopy. The lung slowly expanded during manual inflation with very high pressures assisted by surgical massage of gas from the re-expanded to the collapsed lobules. Recovery was uneventful.

Massive pulmonary collapse was first recorded as a complication of diphtheria by Pearson-Irvine (1876). Brockman, Elliot and Dingley, and Pasteur all reported the condition as a postoperative complication in 1914, and since then there have been over eighty other reports in the literature. Waters (1940) found the incidence to be 0.142 per cent after 29,648 operations, and Pallin and Goldman (1949) claimed that it accounted for only 0.2 per cent of all postoperative pulmonary complications.

On a few occasions acute massive collapse has been described during anaesthesia (Bergmani and Shepard, 1927; Cassels and Rapoport, 1944; Chadourne et al., 1950; Foregger, Retig and Conroy, 1949; Leclercq, 1952; Pallin and Goldman, 1949; Peterson and Pallin, 1942; Santee, 1927; Schotz, 1943; Tesoriere, 1954), but only twice (Grigor, 1954; Kyle, 1963) has the collapsed lung been seen during thoracotomy.

Thus, because the opportunity of actually observing the lung rarely presents itself, we feel it is worth while to record a single case and to discuss the possible aetiology of this rare complication of anaesthesia.

CASE REPORT

A 28-year-old housewife was admitted to hospital in congestive cardiac failure but treatment with digitalis resulted in rapid improvement. She had a past history of attacks of winter bronchitis and rheumatic fever at 12 years of age. Clinical examination suggested that the underlying lesion was stenosis of the mitral valve, and one month later the patient was referred to a thoracic surgeon for mitral valvotomy.

Pre-operative investigation indicated a moderately severe degree of stenosis with enlargement of the left atrium, right ventricular hypertrophy and auricular fibrillation. There were no signs of cardiac failure, blood pressure was 120/80 mm Hg, pulse rate 67/min, haemoglobin 13.9 g per cent, and she weighed 63 kg. There was no clinical or radiological evidence of respiratory disease.

Premedication consisted of papaveretum 10 mg and hyoscine 0.4 mg administered subcutaneously 1 hour before operation.

After 3 minutes pre-oxygenation anaesthesia was induced with sodium thiopentone 350 mg, followed by tubocurarine 45 mg; endotracheal intubation was carried out with a 9.5 mm Magill cuffed oral endotracheal tube. Anaesthesia was maintained with 3 l./min of a 50 per cent mixture of nitrous oxide and oxygen using a Blease intermittent positive pressure ventilator and a to-and-fro Waters canister carbon dioxide absorption system. The lungs were easily inflated with pressures of 25-30 cm H₂O recorded on the pressure manometer of the ventilator.

The chest was opened after about 20 minutes and the left lung, which was expanding normally, was retracted by an assistant while the surgeon opened the pericardium and examined the heart. Fifteen minutes later the surgeon requested that the lung be re-inflated before the valvotomy. When the lung retractor and protecting wet pack were removed the lung was completely collapsed around the hilum. It appeared to be quite airless, of rubbery consistency, and resembled a lobe of the liver. Vigorous manual ventilation with a high fresh gas flow rate, which caused the ventilator manometer pressure to exceed the maximum on the scale (40 cm H₂O), failed to re-expand the lung. The trachea and bronchi were palpated and the endotracheal tube was found to be correctly placed. Despite this the endotracheal tube was replaced by a short 9 mm Magill cuffed endotracheal tube and again an

Present addresses:
* Nottingham General Hospital, Nottingham.
† King's College Hospital, London (after January 1, 1967).
‡ The Mount Sinai Hospital, New York.
attempt was made to re-inflate the lung without success. During this time the other lung seemed to be inflating normally.

Bronchoscopy was performed and, apart from finding the calibre of the left main bronchus to be slightly smaller than normal, no abnormality was found. The endotracheal tube was replaced and aminophylline 0.25 g administered intravenously.

Eventually after 10 minutes manual inflation with the oxygen by-pass control fully open, thus causing pressures far in excess of the maximum recordable by the ventilator manometer, the lung began to re-expand in discrete segments. By surgical massage of gas from the re-expanded to the collapsed lobules the whole lung was finally re-inflated, and was then found to be easily maintained in this state with manometer pressures of 25 cm H₂O.

The operation was completed without further incident and when the chest cavity was closed with two underwater drainage tubes the lung inflated easily. The tubocurarine was reversed by neostigmine 2.5 mg preceded by atropine 1.2 mg, and anaesthesia was discontinued. Manual inflation with 100 per cent oxygen was followed within 2-3 minutes by spontaneous respiration and recovery of consciousness. No respiratory difficulty was observed nor were any abnormal sounds heard on auscultation; chest X-ray 1 hour later showed no signs of atelectasis. Apart from slight fever and an elevated erythrocyte sedimentation rate on the first postoperative day, recovery was uneventful.

**DISCUSSION**

The most obvious cause of collapse of an entire lung is obstruction of the main bronchus, either within the lumen of the bronchus or by compression of the bronchial wall from the outside. In the patient we have described the successive endotracheal tubes were correctly positioned in the trachea and did not enter the right main bronchus; it is also unlikely that the cuffs of both the endotracheal tubes could have herniated in such a way as to block the entrance to the left main bronchus on two consecutive occasions. The left main bronchus might have been kinked by surgical retraction initially, but the lung should have re-expanded in the usual manner when the retractors and pack were removed with "normal" pressures recorded by the manometer of the ventilator. Bronchoscopy confirmed that the lumen of the left main bronchus was patent as far as the basal segmental bronchi and so blockage by excessive secretions or a foreign body could not have caused the collapse of the whole of the left lung.

The site of obstruction could have been widespread at a lower level in the bronchial tree and bronchospasm has been suggested as a possible mechanism of massive collapse of the lung. Churchill-Davidson (1953) considered that at a light level of anaesthesia painful stimuli may produce a reflex bronchiolar smooth muscle pain. This causes venous congestion which results in oedema of the mucous membrane of the small air passage with occlusion of the lumen of the bronchioles. The trapped air in the alveoli is absorbed and atelectasis follows. Raffensperger, Diffenbauch and Strohl (1960) also suggested the idea of a reflex bronchospasm associated with respiratory depression to account for the rapid absorption of alveolar gas. Additional evidence in support of this mechanism is that postmortem examination of fatal cases of massive collapse of the lung frequently fails to find any physical obstruction to the bronchi or bronchioles (Wylie and Churchill-Davidson, 1960).

With any form of airway obstruction and subsequent absorption collapse of the alveoli, a certain finite amount of time is necessary for complete absorption of alveolar air. Nitrous oxide is absorbed relatively rapidly—Corylos and Birnbaum (1932) give a range of 17 to 35 minutes—and Mapleson (personal communication, 1966) believes, on theoretical grounds in the circumstances described, that absorption would almost certainly be practically complete towards the lower limit of this time range. The longest possible duration of obstruction in our patient was 15 minutes, that is from the time the chest was opened and lung was seen to be inflating normally until the retractor and pack were removed, and this was hardly long enough to allow such an extensive degree of collapse distal to an obstruction.

Some atelectasis almost always occurs during thoracotomy, due to direct compression of lung tissue by the surgical retraction and pack. There is usually no difficulty, however, in inflating the collapsed areas at the conclusion of the operation when the peak inflating pressure is increased. We had to use an excessive pressure for several minutes even to achieve partial inflation, and then it was necessary to actively massage gas into the remaining atelectatic parts of the lung. In any case, collapse by direct compression rarely affects the whole lung to such a marked degree.

There may, therefore, be some unexplained mechanism responsible for acute massive collapse
of the lung. The stability of the expanded lung is believed to be due to the presence of a lipoprotein substance in the alveoli (Pattle, 1963). This substance, “surfactant”, has a variable surface tension activity which reduces the surface tension at the alveolar air/fluid interface as expiration nears completion and the alveoli decrease in size (Clements, 1962). In this way total collapse of the alveoli is prevented (Bondurant and Giammona, 1965).

A decrease in the concentration of surfactant offers a simple explanation of the mechanism of acute massive collapse, both with regard to the lack of postmortem and clinical findings of obstruction and the physical appearance of the completely collapsed lung. With collapse due to airway obstruction some air must be trapped initially in the alveoli and is absorbed later, whereas if alveolar collapse is the prime factor then no air will remain—a situation fully compatible with an airless lung of rubbery consistency which resembled a “lobe of liver”. Loss of surfactant would also explain the great difficulty in re-inflating the lung, and then the rapid return of stability once the whole lung has been eventually expanded.

The possible causes of a decreased concentration of surfactant are obscure. Gruenwald and associates (1962) have suggested that the production of this substance is dependent on the adequacy of the pulmonary capillary blood flow. Pulmonary capillary blood flow is reduced in cardiac failure and is altered in distribution in mitral stenosis (Dollery and West, 1960). Our patient may consequently have had a lower than normal concentration of surfactant at the alveolar air/fluid interface before surgery. Thoracotomy and surgical manipulation may have further lessened pulmonary capillary blood flow and so further decreased the concentration of surfactant, thus accounting for sudden massive collapse of the lung.

Kyle (1963) concluded that “no real progress has been made towards explaining the causative mechanism . . .” and that it “deserves and needs further study”. While presenting another possible explanation that accords better with the clinical findings and observation of the collapsed lung, we are obliged to agree with him.

ACKNOWLEDGEMENT

We wish to thank Professor W. W. Mushin for help and criticism in the presentation of this article.

REFERENCES


COLLAPSUS PULMONAIRE MASSIF AU COURS D'UNE THORACOTOMIE

SOMMAIRE
On décrit le cas du collapsus massif aigu d'un poumon survenu chez une femme de 28 ans au cours d'une commissurotomie mitrale. La sonde endotrachéale était correctement placée et on n'a pas trouvé d'anomalie à la bronchoscopie. Le poumon s'est lentement dilaté par le massage manuel avec de très fortes pressions associé au massage chirurgical de gaz des lobules rétablis aux lobules collabés. Guérison sans incidents.

NOTICE

REFRESHER COURSE IN ANAESTHESIA for General Practitioners and part-time Anaesthetists

MAY 17 to MAY 20, 1967

Numbers limited to 30. Particulars from Dr. R. A. FISHER, Postgraduate Medical Centre, Royal Victoria Hospital, Bournemouth, Hampshire.