ACUTE PULMONARY COLLAPSE—A CASE REPORT

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ACUTE MASSIVE COLLAPSE of the lungs is described as a rare but distinct clinical entity, in which a lobe, whole lung, or both lungs collapse down to a fraction of their former size in a few minutes, without the apparent presence of obstruction. This may occur during or shortly after surgery under all forms of anaesthesia. If the outcome is death, as is often the case, post-mortem reveals little or nothing. The principal feature is wide-spread alveolar collapse, with only a few scattered areas of aerated alveoli. Dissection of the respiratory tract right down to the smallest bronchioles shows no clear-cut mechanical obstruction.

A recent case has provided interest and stimulated a search of the literature for what is known of this condition, and the result is disappointing. For little is known, and apparently little is being learned about this often reported, nearly always fatal clinical entity.

HISTORICAL

Broadly speaking, this condition may be considered a form of acute fulminating atelectasis. In 1835 Jorg¹ originated the term from the Greek *ateles* (incomplete) and *ektasis* (distention). Massive collapse was first described by Pearson-Irvine² in 1876. Pasteur³ described the condition many times in the period 1908 to 1914, the last report being of 16 operative cases. Sporadic reports continued in the literature until Bradford⁴ reported his findings in World War I. He redefined this condition as "an unusual condition, in which the lung, without the presence of any gross lesion such as bronchial obstruction, pleural effusion etc., interfering with the free entry of air, becomes airless to a greater or lesser degree, and therefore useless for respiratory purposes." A most complete review by Pallin and Goldman⁵ summarized knowledge and thinking in 1949, with the conclusion that, "an unbalanced autonomic nervous system contraction of the lung, produced by a hyperactivity of the parasympathetic division can cause a reflex contraction of the lung." They proposed adequate atropine dosage as the prophylaxis.

After a period of sporadic reports in the literature, Churchill-Davidson⁶ reported a case with post-mortem of acute pulmonary collapse. At post-mortem there was no microscopic nor macroscopic evidence of occlusion of air flow to the lungs.

CASE REPORT

Mr. P.H., a white male, aged 22, weight 125 lb., was admitted with a complaint of chest pain of 5 days' duration. Past history showed bronchial asthma of 5 years' duration, and four previous attacks of spontaneous pneumothorax. On examination the only positive findings were limited to the chest, where general inspiratory wheezes were heard over both fields, and X-rays showed a pleuritic reaction in the left mid-lung zone, with some pneumothorax. He was booked for thoracotomy and wedge resection. Preoperative examination revealed BP 110/70, Hg 14.8 gm., haematocrit 46. Pulmonary ventilation

studies were reported as showing "outflow obstruction with little response to bronchodilators, with reduced residual volume."

The patient was premedicated with morphine 8 mg. and scopolamine 0.4 mg. one hour before operation. He was induced with thiopentone 300 mg., intubated after 40 mg. succinylcholine, placed in the right lateral position, and given 100 mg. of gallamine. He was maintained on halothane, using a Jefferson respirator with a 2-litre flow of 50 per cent nitrous oxide and 50 per cent oxygen, at pressures of plus 10 and minus 3 cm. water, and cycled at a rate of 15 per minute. When the chest was opened the left lung was seen to be well expanded, but as the case progressed this lung gradually collapsed within a period of 20 minutes in spite of huge increases in positive pressure.

The endotracheal tube was withdrawn and checked for patency, aminophyllin 10 c.c. was injected, and lung massage tried. After hilar injection with 1 per cent xylocaine, atropine 0.6 mg. was given followed by solucorte 100 mg., but the lung became progressively smaller, shrinking to a small, dark red, rubbery mass the size of a fist.

At this point (1 hour and 45 minutes after induction) anaesthesia was switched from halothane to ether, and another 0.6 mg. of atropine was injected with no result.

The wedge resection was completed, and because the diagnosis of massive pulmonary collapse was made, it was decided to close the chest in spite of the fact that the completely collapsed, rubbery lung could not be inflated to any degree.

During this period ventilation to the other lung appeared normal, and no disturbances of blood pressure or pulse appeared.

At the end of the procedure (2 hours and 40 minutes) the patient was bronchoscoped and the surgeon reported "absolutely no evidence of obstruction or congestion in the bronchi, with apparent patency of all channels." The patient was reintubated and an attempt made at inflation. He was taken to the recovery room, where a portable chest X-ray (Fig. 1) showed complete collapse of the left lung. His general condition was

FIGURE 1

otherwise good. Follow up X rays showed that the lung remained completely collapsed for about 24 hours then rapidly began to re expand and was completely expanded in less than 48 hours (Fig 2). The postoperative course was uneventful.

FIGURE 2

DISCUSSION

Clearly this is not the same condition as postoperative pulmonary collapse which it is agreed is the most common form of atelectasis and is caused by mucous plugs in the bronchial tree followed by a collapse of peripheral lung tissue.

The mechanism of acute pulmonary collapse of one or both lungs is not known. Simple absorption of entrapped alveolar gases could not account for such a speedy result. Churchill Davidson suggests that pain stimulation in the presence of inadequate reflex suppression leads to bronchiolar smooth muscle spasm. Oedema of bronchial and bronchiolar mucous membrane caused by venous compression during spasm may lead to obstruction and very rapid atelectasis. There is apparently no evidence that asthmatic patients are more likely to develop this condition. The same author reports a case in which at post mortem there was no evidence of occlusion to air flow to the lungs and he negates the

idea of mechanical interference. He suggests that it is possible that the smaller bronchioles were plugged with thin watery mucus which was no longer visible in the post-mortem specimen. He concludes by raising the question as to whether or not the condition could have been caused by reflex bronchiolar spasm. Consideration of lung anatomy makes this unlikely, but views are changing. Nowhere in the complete review by Widdicombe⁸ is there a specific reference to the lung's inherent ability to collapse actively. But interest in this area is mounting, as evidenced in the recent scientific display entitled "The Lung—Elastic Sponge or Muscular Organ?"⁹

In 1960 Raffensberger *et al.*¹⁰ reviewed the literature for previous reports of experimental work that has been done to determine the aetiology of massive atelectasis. The combination of respiratory depression, rapid absorption of gas from the alveoli, and bronchospasm secondary to vagus nerve reflexes was responsible, in their opinion, for the production of massive pulmonary collapse. To prevent this complication, repeated doses of atropine during surgery, hyperventilation with room air at the end of anaesthesia, and mechanical respiratory assistance in selected cases were suggested. The usefulness of hilar blocking is also obvious, but was not suggested.

A new approach was made by Smolnifoff,¹¹ when he discussed two fatal cases of complete bronchospasm under anaesthesia, and suggested pulmonary massage as the sensible answer to the management of the cases with spasticity of bronchiolar musculature and resultant hypercarbia and hypoxia. Although there may be a place for pulmonary massage for patients in status asthmaticus who have proved refractory to other treatments, in the above-reported case it appeared futile.

Churchill-Davidson makes the only straightforward effort to explain the phenomenon in the light of known anatomical and physiological knowledge. He summarizes:

Three main arterioles supply the external arteriolar plexus, which in turn sends branches through the muscle layer to supply the capillary plexus of the lung. This plexus, which lies beneath the mucosa, then drains back through the muscle layer into the outer venous plexus. Spasm of the bronchiolar musculature would then not only reduce the size of the lumen, but also might be sufficient to cause further obstruction by impeding the venous return while still allowing arterial blood to be pumped into the capillary plexus. This would result in edema of the bronchiolar mucous membrane, making rapid collapse possible.⁶

This is the only logical attempt so far to explain this phenomenon, which deserves and needs further study.

SUMMARY

A case of unilateral acute massive pulmonary collapse under anaesthesia is reported. A survey of the literature discloses that while the entity has been known for a long time, no real progress has been made towards explaining the causative mechanism of this often fatal condition.

RÉSUMÉ

On connaît depuis 1876, le syndrome clinique du collapsus pulmonaire massif et soudain; toutefois, malgré des rapports fréquents dans la littérature, l'on connaît peu de chose du mécanisme de production de cette complication redoutable. On a suggéré différentes causes, y compris des réflexes vagues provoquant une constriction du poumon, une obstruction par du mucus suivie d'un collapsus distal, enfin, un arrêt réflexe du courant sanguin pulmonaire entraînant de l'oedème et une diminution de la ventilation.

Entre autres moyens de lutter contre ce syndrome, on suggère de donner des doses répétées d'atropine au cours de la chirurgie, de contrôler la respiration, de faire du massage pulmonaire et un blocage du hile.

Nous avons présenté un cas de collapsus pulmonaire massif unilatéral au cours d'une anesthésie où, de toute évidence, il ne s'agissait pas d'obstruction mécanique. Tout ce que nous avons suggéré a été fait sans succès; le poumon que nous n'avons pas réussi à réexpandre au cours de la chirurgie s'est réexpandu spontanément 48 heures après l'opération.

Quand ce phénomène est bilatéral, il est toujours fatal et il mérite de plus amples recherches.

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