LUNG MASSAGE FOR TOTAL BRONCHOSPASM: A CASE REPORT

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This report concerns a fit although over-weight middle-aged man, who gave no history of asthma, admitting only a chronic "smoker's" cough, and who, after more than an hour of halothane anaesthesia, deve'oped a total bronchospasm which responded dramatically to direct lung compression or 'massage' as recently described by Smolnikoff.[1,2]

A European male aged 38 came to operation for tantalum insertion into a carcinoma of the bladder. He was obese and noticeably anxious, and exhibited the smoker's respiratory syndrome[3] and a classical 'bull-neck'; otherwise he was unremarkable. Premedication consisted of 3 gr. of seconal the night before, and 50 mg. of promethazine with 100 mg. of pethidine intramuscularly 75 minutes before the operation. For induction of anaesthesia 400 mg. of thiopentone was injected, and anaesthesia was maintained with a mixture of 1,000 ml. of oxygen and 2,000 ml. of nitrous oxide per minute, containing 2% halothane vaporized outside a circle absorption system. A face mask was strapped over an oropharyngeal airway. After 10 minutes of uneventful spontaneous respiration the surgeon reported inadequate abdominal relaxation, and partial laryngospasm manifested itself. Endotracheal intubation was performed with a no. 10 cuffed Magill tube and facilitated by 25 mg. of suxamethonium; and, for 2-3 minutes only, controlled respiration yielded adequate operating conditions, with the breathing sounds quite normal. Twenty minutes later relaxation was again inadequate, and then 80 mg. of flaxedil was administered, and controlled respiration was persistently used. After 70 minutes of anaesthesia 1·5 ml. of 4% lignocaine was injected down the endotracheal tube because some resistance to inflation of the lungs, as well as abdominal rigidity, had appeared during the preceding minute or two. Total bronchospasm nevertheless supervened within 3-4 minutes, in spite of the continued use of 2·5% halothane and intermittent positive pressure vigorously applied to the airway. Wheezing had previously indicated bronchiolar constriction, but at no time was there any evidence of liquid in the bronchial tree. While 0·65 mg. of atropine, 0·5 ml. of 1 in 1,000 adrenaline, and 4 ml. of 10% calcium chloride, were being given intravenously, all pulses became impalpable. A thoracotomy was immediately performed while an intravenous infusion of 1,000 ml. of normal saline containing 8 mg. of noradrenaline was started.

The exposed left lung was so distended that it ballooned out of the pleural incision, rendering the thoracotomy virtually impossible without some laceration of the lung — notwithstanding the fact that the endotracheal tube was disconnected entirely from the anaesthetic machine to demonstrate that there was no positive pressure being applied at all. (Gas did actually escape audibly from the endotracheal tube on disconnection for a period that seemed to be at least 30 seconds.) Earlier the cuff was deflated and the tube replaced with another in spite of the difficulty the patient's bull-neck presented to laryngoscopy. Another desperate attempt to move oxygen into the already overdistended lungs again failed completely. Thereupon, bearing in mind Smolnikoff's experience,[1,2] the surgeon was asked to empty the lung as much as possible between his two hands. This promptly and impressively resulted in an uneventfully expanding lung on inflation, and a normally collapsing lung during the intervening phases of zero airway pressure.

The surgeon thought that the heart was beating, but when he opened the pericardium the tip of the left ventricle was seen to contract rhythmically while the rest of the heart remained in standstill. Cyanosis and widely dilated pupils as well as absent carotid impulses, even with the cardiac massage, persisted for 35 minutes altogether before a normal heart beat appeared shortly after the intracardiac injection of 8 ml. of 1 in 10,000 adrenaline and 2 ml. of 10% calcium chloride. A tracheotomy was performed and hypothermia induced postoperatively, but the patient died after 54 hours.

CONCLUSION AND COMMENTS

The case just described confirmed the value of lung 'massage' for complete bronchospasm occurring during anaesthesia. Although the manoeuvre did not save this patient's life, it may well be life-saving in these very rare instances when absolutely fruitless attempts at lung inflation are 'just like pushing against a stone wall'. Judging from an earlier case of my own in 1954, in which a fatal total bronchospasm occurred in an insufficiently relaxed but fit Bantu man during laparotomy when the surgeon unexpectedly lifted the peritoneum, and from those reported by Smolnikoff,[1,2] Gast[4] and Kucher;[5] and the case now described, cardiac arrest always follows exceedingly rapidly once total bronchospasm has become manifest. When this extremely rare contingency has to be faced it therefore appears unwise to rely on the intravenous admini-

stration of drugs, because the venous return must be severely curtailed or, more likely, altogether absent in view of the fact that the thoracic veins are actually collapsed rather than merely depleted. It is well known that excessive inflation of the lungs interferes with venous return even in the open chest by an increase of resistance in the pulmonary bed as well as by tamponade of the heart and great vessels.[6] In any case, no certainly efficacious bronchodilator drug exists.

Quite apart from anaesthesia, death during an attack of asthma is known to occur but not usually, if ever, from total bronchospasm. As Smolnikoff[1,2] points out, there is an essential difference between the bronchospasm of ordinary asthma and the rare total variety seen only in anaesthesia, general or local; the variety associated with anaesthesia is in fact not particularly feared in asthmatics, occurring classically in obese but otherwise healthy middle-aged men.[7] Like 'true' instances of cardiac arrest during anaesthesia there are usually little, if any, premonitory signs. It represents a grave accident inherent in the practice of surgery with anaesthesia, but is fortunately so rare that few anaesthetists encounter it more than once or twice in a lifetime. Heavy smoking, with its associated syndrome[3,8] is a predisposing factor.

Finally, while muscle relaxant drugs for associated skeletal muscular rigidity and bronchodilators like adrenaline, aminophylline and atropine are all valuable in the prevention of bronchial constriction, attention must be given to effective circulation[9,10] with exactly the same urgency as in cases of cardiac arrest from other causes. Once manual compression of the lung has been instituted it will invariably be necessary for the assistant to massage the heart, also using the palms of both hands, if necessary, to produce the absolutely vital *palpable* carotid pulsations. Although all this activity through the chest incision may lead to some inconvenience to the two 'masseurs', and enlargement of the incision, it is entirely feasible. It must always be remembered that interruption of the cerebral circulation is followed within seconds by a loss of consciousness, and within minutes by irreversible changes in the brain.

The most important lesson, however, to be learnt from this case is the need for adequate suppression of the patient's reflex response to stimuli, and the fact that, in spite of numerous claims to the contrary, phenothiazine derivatives do not always provide this; neither does halothane or ether guarantee absence of reflex irritability even during the deep stages of anaesthesia. Thus Edwards *et al*[11] found no case of the undoubtedly genuine idiopathic variety among 16 cases in which the anaesthetist recorded that bronchospasm occurred during anaesthesia and played an important role in the subsequent death of the patient.

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ADDENDUM

Extracts from the detailed but largely non-contributory autopsy report are as follows: 'Section of the specimen of lung shows the presence of an acute fibrinous pleurisy, marked congestion and oedema, hyaline membrane formation, numerous pigmented and unpigmented alveolar phagocytes, a focus of acute inflammation and some metaplasia of the epithelium of the respiratory bronchioles and ducts . . . marked curling of the epiglottis . . . small foci of pyelonephritis with a granular cast . . . moderate fatty changes in the liver . . . bruised myocardium . . . fibrinous pericarditis . . . no metastases . . .'

In the brain nothing was found, except, on histological examination, 'some extravasation of blood into the perivascular spaces.'