

## BRONCHOSPASM: A CASE REPORT

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BRONCHIAL SPASM, when it occurs during the course of a general anaesthetic, is often an error in technique, the cause of which should be immediately detected and corrected. Difficult ventilation results from spasm of the bronchi accompanied by a fixation of the chest wall. When muscular fixation of the chest predominates, ventilation can be improved by deeper anaesthesia or muscle relaxants. The bronchial component can be caused by insufficient anaesthesia, respiratory tract disease and certain drugs. Thiopentone, cyclopropane, and d-tubocurare may cause bronchospasm by their histamine-like action. Pethidine, Aminophyllin, Isoproterenol, ether, atropine and epinephrine may be used to overcome bronchospasm.

### HISTORY

Mr. M. P. was a 67-year-old Russian with a 3-month history of persistent cough and copious blood-tinged sputum. He smoked very little but drank heavily. There was no history of asthma, bronchitis or pneumonia, but examination of his chest showed a moderate degree of emphysema with some basal rales. A chest X-ray disclosed parenchymal infiltration with cavitation involving the superior segment of the lower left lobe. In view of the diagnosis of lung carcinoma with abscess, surgery recommended a thoracotomy and possible pneumonectomy.

### PREMEDICATION

Atropine 0.4 mg. and Pethidine 75 mg. were given one hour preoperatively. On arrival in the operating room, he was alert, breathing comfortably with a systolic blood pressure 140 mm. Hg and a pulse rate of 84.

### ANAESTHETIC COURSE

Following oxygenation, induction was accomplished by thiopentone 450 mg. and succinylcholine 60 mg. After spraying the throat with 4 per cent lidocaine, a No. 10 cuffed endotracheal tube was passed easily and inflated. Auscultation indicated satisfactory aeration of both lungs. One litre of N<sub>2</sub>O and one litre of O<sub>2</sub> were used in the circle of a Boyle's machine. D-tubocurare 15 mg. was injected into the intravenous tubing. Respiration was maintained by a Jefferson respirator at a rate of 20 per minute with a positive pressure of 18 cm. H<sub>2</sub>O and a negative pressure of 8 cm. H<sub>2</sub>O.

When the patient was postured on his right side, breath sounds became coarse and rhonchi were heard throughout the chest. Suction yielded very little sputum. As he had a swallowing reflex, he was given Pethidine 20 mg. followed shortly by thiopentone 100 mg., d-tubocurare 6 mg. and finally by Aminophyllin 50 mg. Respiration, though noisy, seemed adequate. Pulse, blood pressure and the E.C.G. tracing were normal and the operation proceeded.

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Cyclopropane, 200 cc per minute, was added to the gas mixture when the surgeon finished with the cautery. The negative phase of the respirator was turned off when the pleura was opened. Aminophyllin, thiopentone and Pethidine were injected at intervals. The rate of the respirator was reduced to 14 per minute to adapt to the long expiratory phase of the patient's respiration. The addition of ether made ventilation easier but the breath sounds continued to be noisy.

During the first two hours, blood pressure, pulse and E C G. remained normal. A few ventricular extrasystoles appeared with incision of the pericardium and responded to 1 per cent Procaine topically. The surgeon complained of over-distension of the lung, so the positive pressure was reduced. This decreased oxygenation so markedly that the pressure was increased again. At this time it was decided to withdraw the endotracheal tube slightly in case it might be causing reflex stimulation at the carina. The airway was checked afterwards and found to be patent. At this time the E C G showed a multifocal arrhythmia and the surgeon reported the onset of asystole. Simultaneously a bronchospasm developed which was so severe that no air returned from the lungs and they became grossly distended. Use of the negative phase on the respirator seemed to decrease the inflation but was not continued. Asystole occurred several times and responded to massage each time. A recordable blood pressure was not obtained during the period of acute pulmonary distension. The interval of severe hypoxia lasted from ten to fifteen minutes. Finally after giving Aminophyllin 150 mg. intravenously and injecting the hilar area with procaine 1 per cent, it became possible to ventilate the lungs. Tracheal suction produced asystole again. Responding at once to massage, the heart began to fibrillate but this stopped with further massage. The systolic blood pressure rose sharply to 140 mm Hg owing to the entrance of trapped Methoxamine into the circulation.

The surgeon proceeded with the pneumonectomy and the chest was closed one and a half hours later. During this time the patient received O<sub>2</sub> by manually controlled intermittent positive pressure with the addition of N<sub>2</sub>O and cyclopropane for chest closure. The blood pressure fell briefly on two occasions because of interference with the position of the heart by the assistants. Normal cardiac rhythm returned before the end of the operation.

#### POSTOPERATIVE COURSE

Neither spontaneous respiration nor consciousness returned at the end of the operation. Tracheal suction produced a slight response. Neostigmine and nikethamide were ineffective. Finally the use of carbon dioxide (7 per cent) produced respiration with no pause between phases.

In the recovery room the patient required a continuous drip with Phenylephrine to maintain his blood pressure for the the first two hours. The E.C G tracing was normal. When stimulated, he moved both arms and his right leg. He had received a total of 2,500 cc. of blood to control his pressure with an estimated blood loss of 1,200 cc.

Two hours later, breathing became more mechanical. Examination of the optic fundi revealed engorged veins and blurred discs. The injection of 50 cc. of dextrose (50 per cent) produced quieter respiration and his response to stimulation increased. Chest X-ray showed considerable mediastinal shift and 50 cc. of air were added to the left pneumothorax. Pulmonary oedema developed during the next hour and cleared with the use of Aminophyllin 200 mg., Digoxin 0.5 mg. and a phlebotomy of 250 cc. of blood. The E.C.G. tracing, which had recorded ventricular extrasystoles, returned to normal.

During the evening and night he improved to the extent of developing a lash reflex. Next day an E.E.G. showed practically no alpha activity and no response to reflexes. The findings suggested brain-stem damage. He died 48 hours after the operation and unfortunately no autopsy report was obtained.

#### SUMMARY

A case report of bronchospasm during a pneumonectomy has been presented. The difficulty in ventilation was due to spasm of the bronchial tree, and fixation of the chest wall was not a factor. The gross inflation of the lungs did not respond to treatment before there was severe cerebral anoxia. It is believed that this was the cause of death.

#### RÉSUMÉ

Au cours d'une pneumonectomie pour carcinôme chez un vieillard de 67 ans, il est apparu un spasme bronchique. Les manipulations dans le thorax ont entraîné une arythmie ventriculaire qui a dégénéré en asystolie. A ce moment-là, le spasme bronchique est devenu tellement prononcé que l'air ne sortait plus des poumons qui sont devenus distendus à l'excès. Le cœur a répondu rapidement au massage et la distension pulmonaire a persisté s'accompagnant d'une hypoxie aigue qui a duré de 10 à 15 minutes. On n'a réussi à ventiler le malade qu'après avoir pratiqué une injection intraveineuse d'aminophylline et une injection autour du hile des poumons de procaine à 1%. Une heure et demie plus tard, la pneumonectomie était terminée. La respiration spontanée est réapparue avec l'emploi de gaz carbonique à 7%, mais le malade n'a pas repris conscience. Un électroencéphalogramme, pratiqué le lendemain, a montré des lésions cérébrales et le malade est mort 48 heures après l'opération.