The speedy recognition and treatment of the obstructed airway and of the pneumoperitoneum undoubtedly saved the life of this moribund patient.

Case II

A boy aged 5½ was admitted to hospital for bronchography under general anaesthesia. Acute mediastinal emphysema and a bilateral shallow pneumothorax occurred as a result of a sudden delivery of oxygen into an intratracheal catheter, the accident being due to a faulty check-valve. Acute emphysema quickly developed, beginning with the eyelids and rapidly involving the face, neck, and upper chest. Needles were inserted into the neck, and the superior mediastinum was entered; no air could be aspirated. The left chest seemed hyperresonant, but no air escaped from a needle inserted into it. Oxygen was given by the B.L.B. mask, and the child made a complete and uneventful recovery. He was discharged five days after bronchography, the radiograph taken on discharge showing that both lungs had completely re-expanded.

Conclusions

Prevention.—While it is not always possible to prevent this condition, as in Case I, there are certain important rules to be observed: (a) maintenance of a free airway; (b) avoidance of high intra-alveolar pressures—e.g., due to sudden rush of oxygen or anaesthetic gases or to the patient straining; (c) care in manipulating tubes so as to avoid damaging the respiratory tract mucosa.

Treatment.—Attention to the following points in treatment, with the possible exception of (c), has been an important factor in saving the lives of patients: (a) Aspiration of the ballooned tissues of the neck should be attempted, as described by Barrett and Dillwyn Thomas (1944). (b) Obstruction of the airway should be relieved; pure oxygen should be administered under low pressure. (c) Signs of pneumothorax should be sought and, if present, pleural puncture should be carried out. (d) The abdomen should be examined and needled if gross tympanitic distension is present.

I am indebted to Mr. K. W. Mackenzie and Prof. H. A. Harris for their help and criticism.

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A NOTE ON FAT EMBOLISM

BY

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Fat embolism enjoys a curiously honoured place in surgical literature, for no very obvious reason. Everyone knows of it because the most elementary textbook intended for the beginner invariably devotes valuable space to it; but how many surgeons have consciously seen a case and recognized it? The great similarity of all the accounts in the textbooks, large and small, suggests that the writers themselves have had no practical experience of the subject, and that, like their readers, they have gained their information from books. What makes this more extraordinary is that the British surgeon has written so little about it. When Watson (1937) wrote a full account of a case he appended a copious bibliography of some 50 items, in which the only paper from a British source was one dated 1882. Since then Robb-Smith (1941) has dealt fully with the condition from a pathological standpoint, Rowlands and Wakeley (1941) have recorded three cases, and Wilson and Salisbury (1944) eight cases in 1,000 battle casualties.

Put briefly, it may be said that fat embolism of the lungs is probably much more common than is recognized, though embolism in the systemic circulation is very rare. Robb-Smith thought that, of a series of 125 cases of death from accident, in 29 fat embolism probably played a major part in producing a fatal result; but both the symptoms and the post-mortem picture so closely resemble those found in bronchopneumonia that unless special staining technique is employed, such as he describes, the true nature of the condition will be missed.

The difficulty in systemic embolism is to explain how the fat passes through the lung capillaries in a globule large enough to cause trouble elsewhere. Possibly minute globules which

can pass through the lungs coalesce into larger ones on reaching the heart and being subjected to the churning movements of that organ. This would appear to be more probable than the ingenious, if fantastic, suggestion of one writer that this condition can occur only in a patient with a patent foramen ovale.

The case now under consideration is a little unusual in that there were no pulmonary signs at all. The usual course of events is that after a fracture there is a period of 24 hours when the patient is comfortable, and then, somewhat suddenly, he becomes dyspnoeic and cyanosed and sweats profusely. Sputum is scanty and frothy, and may, but only rarely, have fat in it. Later he may show delirium, strongly resembling delirium tremens. By this time the surgeon has probably most unfairly blamed the anaesthetist on the one hand and the patient's habits on the other. The anaesthetist, if he knows the literature, will retaliate with an accusation of rough manipulation—again perhaps unjustly, as the case described below had neither manipulation nor anaesthetic.

Case History

A man aged 59 sustained a fracture of the left tibia and fibula, with some displacement, on March 21, at 12.30 p.m. He was transferred from one hospital to another owing to shortage of beds and was admitted, quite comfortable, in box splints. At 6.30 that evening, before he could be put up in plaster, he suddenly collapsed, with faintness, cold and clammy skin, and a pulse rate of 56. There was no dyspnoea or cyanosis. During the night he was wandering and strange in manner, and the next morning he was comatose. The limbs were a little spastic, the knee- and elbow-jerks were exaggerated, and the plantar reflexes extensor; abdominal reflexes were absent. Pupils were equal but very small. Petechial haemorrhages were present in the skin. Lumbar puncture produced a clear fluid under normal pressure: cells, 2 per c.mm., all small lymphocytes; protein, 0.04%; no globulin. His condition rapidly deteriorated, the temperature rising steadily to 104°, and death occurring 72 hours after admission.

At the necropsy only the brain was examined, and that showed the typical picture of multiple pin-point haemorrhages scattered through the white matter only. The excellent coloured photograph in Watson's article might have been taken from this case. I am indebted to Dr. H. H. Gleave for the report on the pathological specimen:

"Brain.—Macroscopic:—The cerebrum shows numerous tiny points of haemorrhage scattered in the white matter. The grey matter is not affected. **Microscopic:**—The small vessels of the white matter show perivascular haemorrhage and necrosis. These changes occur without any evident relation to a vessel. No change is seen in the cortex. Frozen sections stained for fat show globules and masses of fat in some of the vessels. No fat can be demonstrated in the majority of the vessels, but where it is present the appearance is characteristic. There is no inflammatory reaction around the necrosed area."

This follows closely the descriptions given in the other articles already quoted. The interesting point is that, although quite definite fat emboli can be seen in the vessels, they are neither so numerous nor so striking as the areas of necrosis and haemorrhage.

Comment

The symptoms in this case appear to be quite typical. Briefly, the important points to look for, as shown by this and other cases, are:

1. A latent period of three hours to three days.
2. A sudden collapse associated with profuse sweating and cyanosis, but no fall of blood pressure. As one American writer puts it pithily: "Shock—three hours. Fat embolism—three days. Pulmonary embolism—three weeks." This collapse and the very profuse sweating which has been referred to by all observers should help to distinguish this condition from pulmonary oedema or bronchopneumonia.
3. Pin-point pupils, exaggerated deep reflexes with absent abdominal reflexes but no localizing signs. Cerebrospinal fluid normal.
4. Hyperpyrexia in the later stages. Petechial haemorrhages make the diagnosis certain. Fat may occasionally be found in the sputum or urine, but not often enough to be of much help.

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