NEUROGENIC RUPTURE OF THE OESOPHAGUS

BY

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It was in 1849 that Rokitanski noticed the relative frequency with which gastric and oesophageal lesions were found in patients who had died of intracranial disease. Cushing, in 1932, described eleven cases of alimentary tract lesions occurring in association with intracranial disease; two of them had rupture of the oesophagus. Fincher and Swanson (1949) described three cases of neurogenic rupture of the oesophagus and concluded that this complication was commoner than had been generally realized. Rupture of the oesophagus was found in fifteen out of 452 consecutive autopsies on patients who had died of intracranial lesions in this hospital between 1951 and 1954. It was only found three times in 1,590 other autopsies performed during the same period, twice in patients with acute lesions of the upper part of the spinal cord and once in a patient who had died in hepatic coma (MacIver et al., 1956). Thirteen more cases have occurred during the last three years.

With two exceptions, in which round perforations were found, the lesion was a longitudinal tear in the lower third of the oesophagus. Its position and shape were similar to those found in spontaneous rupture (Walker, 1914; Collis et al., 1944; Barrett, 1946; Ware and Strieder, 1949; Mackler, 1952; Grigsby et al., 1953) and the left side was involved five times as frequently as the right. Perforation of the pleura had occurred in all except one of the cases and fluid was invariably found in one or other of the pleural cavities, but only seven cases had effusions that were larger than 10 oz. (250 ml). In seven cases the effusions were bilateral. Mediastinal air was found in four cases. In four cases the stomach wall had also perforated.

The intracranial lesions varied widely (table 1) and it was impossible to pin the cause down to direct involvement of the hypothalamus or its connections. It may be the result of an autonomic inco-ordination, with the oesophagus failing to relax when the patient vomits, retches, or even hiccoughs, and the actual rupture is probably just one fatal complication of a serious intracranial syndrome. As the folds of the oesophagus are linear, regurgitation and autodigestion may play a part.

Clinically there were three main types.

(1) The Agonal type, in which the patient vomited a quantity of dark brown fluid, often of coffee grounds appearance, collapsed, and died within 5 minutes.

(2) The Indefinite type, in which deterioration was gradual and in which there was no evidence to suggest when the rupture had occurred.

(3) The Interval type, in which deterioration was sudden or rapid, but which survived long enough for a diagnosis to be made if the condition was suspected. In most the neurological lesion made death inevitable, but in five cases it was just possible that prompt surgical intervention could have resulted in survival. It is this type that is of interest to the anaesthetist.

A typical case of the Interval type was a girl, aged 6 years, with a large extradural haematoma who was unfortunately not admitted to the neurosurgical unit until 26 hours after the original accident. On admission she was deeply comatose,
responding only to strong painful stimuli, and in a state of decerebrate rigidity. Her temperature was 105°F, her pulse rate 164/min, and her respiratory rate 44/min. The basal air entry was good and equal but coarse râles were audible in all areas.

Under orotracheal anaesthesia, and very little was required, the bleeding point was dealt with and a large extradural haematoma evacuated. The brain bulged out when the clot was removed and the bone flap was sacrificed.

Six hours after operation she suddenly collapsed, becoming cyanosed and pulseless, and a quantity of dark brown fluid trickled out of her mouth. On examination 10 minutes later, she was in obvious peripheral circulatory failure with a rapid, feeble, irregular, and almost uncountable pulse. Her respirations were grunting and 48/min. There was dullness on percussion and diminished air entry at the left base, and tinkling, splashing sounds were also audible in this area.

An X-ray of the chest, which was taken with the patient supine, showed a slight increase in density in the midzone on the left side (figure 1) and was not by itself diagnostic. This is, however, the region in which a small pleural effusion will collect when the patient is supine. Unfortunately aspiration was not attempted.

Mephentermine, noradrenaline, and transfusion had no effect on the blood pressure and death occurred 2 hours later. The autopsy showed gross cerebral oedema with cerebellar and uncal pressure cones. There was a linear slit 5 cm long in the left lateral wall of the oesophagus with some surrounding digestion. Perforation had occurred into the left pleural cavity, which contained 10 oz. (250 ml) of dark brown fluid, and there was also a small perforation of the stomach.

Diagnosis is always difficult, because the signs and symptoms found in spontaneous rupture are masked by the neurological lesion, and it depends on the investigation of any sudden or rapid deterioration of the condition of a patient who is a likely candidate for rupture. Cushing (1932) described a typical case in a child with a cerebellar tumour. On the second day after operation there was a sudden deterioration with hyperpyrexia, frequent and copious vomits of dark brown fluid, laboured respiration, cyanosis, and bronchial râles, and the wound was reopened without any intracranial cause for these symptoms being found.

The comatose patient is unable to complain of pain, but two patients in this series did complain of pain in the chest before losing consciousness. In the first, a woman with a high cervical cord lesion, the pain was not severe; in the second, the pain was due to a pulmonary infarction and the oesophageal rupture was on the opposite side. A pyrexia of over 100°F is common, but not invariable; it was recorded in twenty cases. The pulmonary signs are usually equivocal and, like the cardiovascular signs, may be masked or mimicked by an intracranial lesion. It is clinically impossible to detect a small quantity of fluid in the pleural cavity of an unconscious and unco-operative patient with shallow respirations, but if there is a good and equal basal air entry, rupture is unlikely. Death occurs before the classical signs of a large pleural effusion can appear.

When a comatose patient, and particularly a pyrexial one, shows a sudden or rapid deterioration with or without a fall in blood pressure, but
with deepening coma, tachycardia, tachypnoea or grunting respiration, and the presence of generalized moist sounds in the chest, the possibility of a rupture of the oesophagus should be considered.

A straight radiograph of the chest should be taken and an attempt made to demonstrate fluid in the costophrenic angles, but, even with careful posturing in a positive case, the effusion may be too small. The injection of radio-opaque fluid down a Levin’s tube can be done at the same time, but this too is unreliable. A positive aspiration of the pleural cavity would be diagnostic, but a negative aspiration does not prove anything. Unfortunately, not only must a firm diagnosis be made, but it is important to know on which side the rupture has occurred if surgery is to be attempted. The possibility of a cure is remote, but surgical intervention has been successful in at least one case in America (Fincher and Swanson, 1949).

Prevention may offer more hope than cure. It has been noticeable in this unit that, although rupture has occurred in patients who have been given a single dose of chlorpromazine, it has not occurred in any case that has been on regular doses of phenothiazine derivatives and in which any tendency for the temperature to rise to 100°F or higher has immediately been corrected by active cooling. During the last twelve months this treatment has been given to all severe head injuries and postoperatively to many cases with cerebral tumours and aneurysms, and there have only been two cases of rupture of the oesophagus instead of the usual five or six. The first was a woman with a pontine glioma who showed the typical signs of a rapid deterioration with deepening coma, hyperpyrexia, rising pulse and respiratory rates, and increased pulmonary secretions, but did not receive the lytic cocktail or any active cooling. The second was a man with a severe head injury and a compound fracture of the femur who was given a single dose of 50 mg of chlorpromazine 8 hours before he collapsed. It is too early for any statistical proof that this treatment protects the patient against a neurogenic rupture of the oesophagus, but the results so far are encouraging.

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REFERENCES