Gastric Mucosal Lacerations After Cardiac Resuscitation

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Several reports have stressed the complications that may follow cardiac resuscitation (Baringer *et al.*, 1961; Bynum, Connell, and Hawk, 1963; Jude, Kouwenhoven, and Knickerbocker, 1964), but gastric lacerations were not documented until Lundberg *et al.* (1967) reported their occurrence in 5 patients at necropsy. We report another 4 patients with gastric lacerations at necropsy, and one patient surviving cardiac resuscitation in whom there is presumptive evidence that a haematemesis was due to gastric laceration. We support the contention of Lundberg and his associates that gastric mucosal lacerations are a not uncommon complication of cardiac resuscitation.

INCIDENCE

During a period of 16 months there were 140 cardiac arrests in the Central Middlesex Hospital, with 28 (20%) long-term survivors. Of the 112 patients who died, 34 came to necropsy in this hospital, and 4 (12%) of these were found to have gastric lacerations.

CASE REPORTS

Case 1. A previously fit 78-year-old man was admitted with a four-hour history of severe chest pain. Examination revealed signs of cardiac failure and an electrocardiogram showed recent anterior myocardial infarction. He was treated with bed-rest, morphine, oxygen, digitalis, and diuretics. Two days later, as he had clinical signs and chest x-ray changes suggesting pulmonary infarction, anticoagulants were given.

The cardiac failure improved and progress was satisfactory until five weeks after his original admission when he had cardiac arrest. External cardiac massage and mouth-to-mouth ventilation were attempted for a brief period only.

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Necropsy. There was a large, recent, anterior myocardial infarct with early aneurysmal bulging. The anterior descending coronary artery was completely occluded by intramural haemorrhage and fresh red intraluminal thrombus. Other coronary vessels showed severe atheromatous narrowing with loss of more than half of the lumen in several places.

There was a 3 cm. ragged ante-mortem gastric mucosal tear running along the upper part of the lesser curvature. Approximately 50 ml. fresh blood was present in the stomach, which was otherwise empty. There were no other injuries.

Case 2. A 68-year-old woman with no previous medical history was admitted with dyspnoea and chest pain. The only abnormality found on examination was basal lung crepitations, and the electrocardiogram showed recent anterior myocardial infarction. She was treated with digitalis and diuretics only. She made good progress in the ward but cardiac arrest occurred one week after admission. External cardiac massage was carried out but technical problems with intubation prevented adequate ventilation. The attempt to resuscitate her was abandoned after 15 minutes.

Necropsy. An organizing anterior myocardial infarct was found, with severe stenosing atherosclerosis of the anterior descending coronary artery occluding more than half of the lumen.

Multiple fresh mucosal lacerations were present on the lesser curvature of the stomach with zig-zag shapes and ragged edges (Fig. 1). The total area damaged was approximately 8 × 3 cm. extending to within 3 cm. of the cardia. The largest tear was 4 cm. long. About 25 ml. of fresh blood was present in the stomach which was otherwise empty. The fourth part of the sternum and the fourth left costal cartilage had been fractured.

Case 3. A 66-year-old man was admitted with a two-hour history of constricting chest pain. Six months previously he had been admitted to another hospital with myocardial infarction and had been treated

FIG. 1.—The gastric mucosa in Case 2, showing multiple, zig-zag-shaped tears with ragged, gaping edges. (Approx. $\times 2$.)

with anticoagulants since his discharge. The only abnormality found on admission on this occasion was basal lung crepitations. The electrocardiogram showed recent anterior myocardial infarction. He was treated with diuretics and the anticoagulants were continued. Five days later cardiac arrest occurred. External cardiac massage was instituted and artificial ventilation was carried out first by mouth-to-mouth ventilation and later via an endotracheal tube. Complete heart block was present. No peripheral pulse could be produced despite the correction of the acidosis, and the use of isoprenaline and noradrenaline, and the attempt to resuscitate him was therefore abandoned.

Necropsy. The pericardial sac contained 250 ml. of fresh blood. This was due to rupture of a large, recent, full-thickness, anterior myocardial infarct. The anterior descending coronary artery showed recent occlusion by thrombosis. The other major coronary arteries showed severe stenosing atherosclerosis and there was an old posterior infarct.

The stomach contained a recent meal. A 2 cm. long fresh bruised mucosal tear was present on the lesser curvature, 3 cm. below the cardia (Fig. 2). Fracture of the sternum with extensive rib fractures had also occurred.

Case 4. A 65-year-old woman was admitted with a three-day history of chest pain. She had signs of complete heart block, confirmed by the electrocardiogram which also showed recent posterior myocardial infarction. A bipolar pacing catheter was passed transvenously to the apex of the right ventricle and she was placed on demand pacing. Sinus rhythm returned two days later. Five days after admission she developed a

sudden pyrexia ($39.4^{\circ}\text{C}.$) for which no cause could be found, but as the pacing catheter was a potential source of infection it was removed. The pyrexia settled with antibiotics and she remained well and in sinus rhythm

FIG. 2.—Case 3. Close-up view of 2 cm. long bruised, gaping mucosal tear on the lesser curve. (Approx. $\times 3$.)

until 11 days after her admission when cardiac arrest occurred. External cardiac massage and mouth-to-mouth ventilation were instituted, and, after a few minutes, endotracheal intubation was carried out. External direct current countershock was given for ventricular fibrillation, but alternating asystole and ventricular fibrillation eventually proved refractory to treatment, so the attempt to resuscitate was abandoned after 30 minutes.

Necropsy. There was a recent infarct of the myocardium involving the posterior wall of the left ventricle and the interventricular septum. The coronary arteries were moderately atheromatous, and the right coronary artery was occluded by fresh thrombus 4 cm. from its origin. The pericardium contained loculated cysts of turbid yellow fluid. 1 cm. below the lower end of the oesophagus there was a vertical gastric mucosal laceration 3 cm. in length (see Fig. 3). There was some bruising on the anterior wall of the stomach overlying the tear.

FIG. 3.—Case 4. 3 cm. long ragged mucosal tear 1 cm. below the oesophago-gastric junction. (Approx. $\times 2$.)

Case 5. A 62-year-old man, previously fit, was admitted after two attacks of atypical chest pain. The electrocardiogram showed minor non-specific abnor-

malities, but 24 hours after admission an epileptiform fit was followed by ventricular fibrillation. External cardiac massage and mouth-to-mouth ventilation were instituted and direct current countershock was given unsuccessfully. After endotracheal intubation and intravenous sodium bicarbonate, direct current countershock restored sinus rhythm. Large doses of lignocaine and propranolol failed to prevent ventricular fibrillation occurring on five subsequent occasions in the next 18 hours. Countershock restored sinus rhythm effectively on each occasion. The electrocardiogram then showed changes of recent anterior myocardial infarction. Ten hours after his first cardiac arrest he vomited blood-stained fluid. There had been no previous vomiting. A nasogastric tube was passed and about 500 ml. of heavily blood-stained vomit was aspirated. He gradually regained consciousness 48 hours after the initial episode and, though confused and disoriented for many days, has apparently made a full recovery. There was no further bleeding and a barium meal 18 days after his admission was normal.

HISTOLOGY

The lacerations showed similar appearances in each patient. Tearing extended through the mucosa, muscularis mucosae and submucosa, involving blood vessels, so that a variable amount of fresh haemorrhage was present in the ragged edges. Punctate intramucosal haemorrhages were seen nearby. The muscle coat was intact in each instance. The gastric mucosa elsewhere in the stomach showed superficial chronic gastritis in one patient and was normal in the other three patients.

DISCUSSION

The incidence of gastric mucosal lacerations found at necropsy in our series (12%) is similar to that of Lundberg *et al.* (1967) who found it in 10 per cent of patients on whom cardiac resuscitation had been attempted.

The fifth patient who survived cardiac resuscitation, but had a haematemesis 10 hours later, probably had a gastric mucosal tear. A normal barium meal and the absence of a past history of indigestion makes a chronic peptic ulcer unlikely. The remaining possibility is stress ulceration. We therefore reviewed the incidence of stress ulceration in the 34 necropsies in this series. Three patients had stress ulcers. All had been extremely ill for several days with, respectively, status asthmaticus (8 days), hypertensive renal failure and myocardial infarction (8 days), and fulminating ulcerative colitis (14 days). Evidence from patients with burns (Sevitt, 1967) also shows that stress ulcers are uncommon in the first 24 hours. It seems most probable, therefore, that this patient survived cardiac resuscitation complicated by gastric mucosal lacerations.

Lundberg *et al.* (1967) reported 5 patients who, having had external cardiac massage and mouth-to-mouth ventilation, were found to have gastric

lacerations at necropsy. Valtonen and Hakola (1964) also reported a woman with Adams-Stokes attacks who, after receiving external cardiac massage and mouth-to-mouth ventilation, had a haematemesis on the way to the hospital where she was found to have a ruptured stomach. Rupture of the stomach on the lesser curve was probably due to external cardiac massage and mouth-to-mouth ventilation in the patient documented by Demos and Poticha (1964).

In reviewing the total of 12 case reports (including the 5 patients described in this paper), it appears that the only features common to all are mouth-to-mouth ventilation and external cardiac massage. In 8 patients an endotracheal tube was passed and 3 received external countershock. Drugs were given to several patients, but no single drug was given to all. The tears of the gastric mucosa, therefore, must be related to external cardiac massage, mouth-to-mouth ventilation or, most probably, a combination of the two.

Distension of the stomach with air or oxygen may cause rupture of the stomach in the absence of blunt trauma (Cole and Burcher, 1961). It has been shown that gastric distension can occur with only a few breaths of mouth-to-mouth ventilation, and Safar (1958) has recorded volumes of air in the stomach as high as 1900 ml. This is particularly liable to happen with obese patients if the neck is not fully extended. A rise in intragastric pressure, particularly if reflux into the duodenum and oesophagus is prevented, will result in tearing first the gastric mucosa and, later, rupture of the stomach wall (Moritz, 1954). Distension of fresh cadaver stomachs with water results in similar tears when viewed by a cystoscope, the mucosa tearing before the more distensible muscle wall (Lion-Cachet, 1963).

The lacerations found in these patients resemble those of the Mallory-Weiss syndrome (Mallory and Weiss, 1929) in appearance though sited rather lower down the lesser curve. The mechanism by which they are formed may, however, be similar. Atkinson *et al.* (1961) suggested that the Mallory-Weiss syndrome occurs in patients with a hiatus hernia when there is a high pressure gradient across the wall of the stomach. The intragastric pressure and intra-abdominal pressure are high during vomiting but the intrathoracic pressure is low so that there will be a high pressure gradient across the gastro-oesophageal junction. In patients having external cardiac massage the intragastric pressure may be high from the introduction of air and will increase as the sternum is depressed. At this time the oesophagus is also compressed so that air leakage will be prevented and the intrathoracic

pressure will rise. In this situation the pressure gradient will exist between the high intragastric pressure and the low intra-abdominal pressure so that mucosal lacerations would be expected at a lower site on the lesser curve.

SUMMARY

Four patients who had external cardiac massage and mouth-to-mouth ventilation were found to have gastric mucosal lacerations at necropsy. Evidence is presented to suggest that a fifth patient surviving cardiac resuscitation had a haematemesis due to such lacerations. In patients on whom cardiac resuscitation has been attempted, the incidence of gastric mucosal lacerations appears to be about 10 per cent. The mechanisms thought to produce gastric mucosal lacerations are discussed.

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