four of our patients treated with 9 α -fluorocortisol in whom a gain in weight resulted, the secretion rates fell to between 20 and 70 μ g./day, with a mean for the four of 45 μ g. In neither of the severely magnesium-depleted subjects was the aldosterone secretion rate low enough to indicate any significant degree of inhibition.

It must be concluded, therefore, that even severe magnesium depletion results in no significant change in the aldosterone secretion rate, and that fluctuations in magnesium metabolism have a negligible influence on aldosterone secretion.

We are grateful to Dr. C. C. Booth and to Professor Russell Fraser for allowing us access to their patients, and to Dr. I. MacIntyre for the plasma-magnesium estimations.

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Medical Memoranda

An Unusual Case of Cardiac Arrest

Cardiac arrest may be due to a variety of causes, many of which are made clear only in retrospect. A large number of cases have now been treated successfully by thoracotomy with direct cardiac massage and, more recently, by the method of external cardiac massage as described by Kouwenoven *et al.* (1960).

CASE HISTORY

The patient, a 48-year-old single woman, had a long history of duodenal ulcer. In 1959 the ulcer perforated, requiring surgical closure. This was followed a few weeks later by a hysterectomy for fibroids. Also in the same year she required a blood transfusion for severe haematemesis. In 1962 her symptoms returned and a barium-meal examination showed duodenal deformity, causing pyloric stenosis. On admission for vagotomy and pyloroplasty her cardiovascular and respiratory systems were clinically normal.

Anaesthesia was induced at 8.25 a.m. with pethidine 20 mg., thiopentone 300 mg. (2½% solution), and suxethonium bromide 100 mg. The patient was inflated for 8–10 breaths with a face-mask, using the Boyle circle absorber. A cuffed orotracheal tube was passed and both lungs were seen to inflate.

Spontaneous respiration began about one minute later and 20 mg. of tubocurarine chloride was given. An intravenous infusion of normal saline was then set up. At 8.37 a.m. she was noticed to be cyanosed, with engorged jugular veins. No radial or carotid pulse could be felt and there were no heart sounds. The pupils were beginning to dilate and inflation with oxygen produced only minimal chest movement.

Immediate thoracotomy was performed, and as the pleura was opened there was an audible hiss due to the escape of gas under tension. The lung was collapsed and the heart flabby and motionless. Direct manual massage of the heart was immediately begun, producing a good carotid pulse. Within one minute a response was obtained and after five minutes the heart was contracting strongly in normal rhythm. The pupils started to constrict and the systolic blood-pressure at 8.50 a.m.

was 80 mm. An infusion of 200 ml. of 2½% bicarbonate solution was started (Brooks and Feldman, 1962).

A more detailed inspection of the lung showed it to be covered with numerous thin-walled bullae, mainly over the lower lobe. There were a few fibrous adhesions between this part of the lung and the diaphragm. A little saline was then instilled into the pleural cavity, and bubbles were seen to be emerging from a small rupture in one of the bullae. This was sutured with catgut and the chest closed with an underwater drainage tube.

At 9.10 a.m. the systolic blood-pressure was 100 mm. and a transfusion of 1 pint (570 ml.) of blood was started. When spontaneous respiration returned at 9.25 a.m. the right side of the chest was seen to have less respiratory excursion than the left and was hyperresonant to percussion. X-ray examination in the recovery room confirmed a tension pneumothorax on the right side.

At this time the patient opened her eyes and objected to the orotracheal tube, which was removed. She then complained of dyspnoea and right-sided chest pain. A dramatic improvement in her symptoms followed aspirating the pneumothorax with a trocar and cannula, which was afterwards connected to an underwater drain. A further x-ray examination showed lung expansion, and at 10.35 a.m. she was well awake and fully orientated, with no evidence of mental impairment.

Daily chest x-ray examination post-operatively showed gradual lung expansion. On the fourth day the needle was removed from the right side of the chest. The next day the patient's breathing became difficult, and a further tension pneumothorax on the right side was aspirated, the needle this time being connected to a low-pressure pump. The tube was removed from the left side of the chest on the sixth day, and the needle from the right side four days later. An E.C.G. showed no abnormality.

The patient was discharged to convalescence three weeks after the cardiac arrest. Throughout a six-months follow-up she has been well, with no gastric symptoms.

COMMENT

Cardiovascular collapse during intermittent positive-pressure respiration (I.P.P.R.) was described by Hunter (1955) in patients with artificially induced pneumothorax. The production of pneumothorax due to rupture of the oesophagus has been reported by Temple (1949) and Dundee (1955).

This case of cardiac arrest was due to bilateral tension pneumothorax. There was no pre-operative evidence of the underlying lung pathology, nor was I.P.P.R. carried out at a pressure greater than that usually employed. It is evident that had external cardiac massage been attempted valuable time would have been wasted. Thoracotomy revealed the cause of the arrest and direct cardiac massage produced a successful outcome. We believe that this method should always be employed where suitable facilities are available. It affords the optimum conditions for observing the heart and returning it to normal rhythm and may reveal intrathoracic causes of the cardiac arrest.

We should like to thank Dr. F. F. Cartwright and Dr. C. D. T. James for their advice, and Mr. L. T. Cotton for permission to publish this case.

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