FROM THE MORBIDITY CONFERENCE

A CASE OF MYOCARDIAL INFARCT UNDER ANAESTHESIA

BY

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A woman aged 35 was presented for a plastic repair of the right renal pelvis. Both kidneys had been shown on i.v.p. to be hydronephrotic, with impaired function, more severe on the right side.

Pre-operative examination revealed an apprehensive, thin woman of 8 stone 5 lb (53 kg), with a blood pressure of 210/130 mm Hg. Examination of the fundi revealed no abnormality, nor was any cardiac enlargement noted clinically or on radiography of the chest. Her exercise tolerance was excellent. The haemoglobin was 105 per cent and the blood urea 36 per cent.

Seven days previously retrograde pyelography had been carried out without difficulty, using thiopentone 150 mg, nitrous oxide, oxygen and trichloroethylene as anaesthesia.

On this occasion premedication with morphine 10 mg and atropine 0.6 mg was given 1 hour before induction. After the usual test dose a total of 25 mg of d-tubocurarine was given, followed by 125 mg of thiopentone. The lungs were inflated with a 50/50 mixture of nitrous oxide and oxygen for 3 minutes and a tube passed. Anaesthesia was maintained with a 2-to-1 nitrous oxide/oxygen mixture, administered via a circle type absorber, a further 5 mg of tubocurarine being required to maintain control of respiration. The blood pressure at this stage was 200/120 mm Hg.

The operation was begun through a right anterior transverse approach. Bleeding was not severe, but the loss was replaced as it occurred with a slow blood transfusion. During mobilization of the kidney the blood pressure fell suddenly to 100/90 mm Hg and there was a severe bradycardia of 36 beats per minute. Atropine 1.2 mg was given intravenously and the pulse rate rose to 90 per minute and the blood pressure to 180/90 mm Hg. Within 10 minutes the systolic pressure had fallen to 90 mm Hg and the diastolic could not be determined, although the pulse rate remained unchanged. At this point 1 pint (500 ml) of blood had been transfused. Methoxamine 10 mg was given intravenously without effect, nor was there any response to a further 5 mg.

A noradrenaline drip 1/250,000 gave an initial response, the blood pressure rising to 110/70 mm Hg and the pulse to 120 per minute. However, within 5 minutes the blood pressure fell to 40 mm Hg systolic, accompanied by a tachycardia of 180 beats per minute. The latter was an expected response to noradrenaline after atropine administration, but this together with the lack of vasoressor effect led to the abandonment of the noradrenaline drip.

Acute adrenocortical insufficiency was postulated and hydrocortisone 50 mg was given intravenously, and as there was a slight rise in the blood pressure this was followed by a further 50 mg without effect.

By now the patient showed evidence of fairly extensive vasoconstriction with marked "goose flesh". The peripheral pulses were now absent; only a slow (36 beats per minute) carotid pulse could be felt; the pupils had begun to dilate.

A quick reappraisal of the situation suggested that the hypotension must be cardiac in origin, because (a) blood loss had been adequately replaced, (b) there had been no real response to vasoressors and (c) no response to the blood drip. Therefore a 1/250,000 adrenaline infusion was set up. The pulse returned to the wrist with a rate of 140 per minute and a systolic pressure of 60 mm Hg, and these effects could be maintained by the drip.
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With the completion of the operation the curarization was reversed by giving neostigmine 1.25 mg intravenously slowly and without atropine. However, as the pulse rate dropped gradually to 70 per minute from 160 per minute, atropine 0.6 mg was given, followed by a further 1.25 mg of neostigmine. Good reversal was obtained.

Coincident with the onset of spontaneous respiration and awakening, pulmonary oedema occurred and about 1 pint (500 ml) of blood-stained, frothy fluid was removed by endobronchial suction. Controlled respiration with oxygen alone effectively cleared up the pulmonary oedema. The acute left ventricular failure was treated with strophanthin 0.5 mg given intravenously. Other methods of lowering the venous pressure, such as the administration of hexamethonium, were considered but discarded, and aminophylline was thought to be unlikely to be better than strophanthin.

The adrenaline drip was stopped when the blood pressure was stable at 80/40 mm Hg with a pulse rate of 90 per minute; 1 mg of mersalyl was given intravenously to promote a diuresis. An e.c.g. taken at this time showed no abnormality other than sinus tachycardia and low voltage. On clinical grounds alone a left ventricular myocardial infarct was suspected.

Any movement by the patient precipitated attacks of pulmonary oedema which could be controlled by positive pressure respiration and endobronchial suction. To ensure quiescence after such an attack, a 50/50 N₂O/O₂ mixture was administered via the endobronchial tube for 10–15 minutes.

The attacks of pulmonary oedema became less frequent and 19 hours after induction of anaesthesia the patient was extubated, the patient being alert and co-operative.

Convalescence from then on was uneventful, urinary output being excellent and serum chemistry remaining unchanged. The electrocardiogram the next day showed deep negative T-waves in all leads and 1 week later the negative T-waves in V6 had a slight S.T. depression, which 7 days later (i.e. 2 weeks after operation) had become more marked.

The diagnosis of a large left ventricular myocardial infarct occurring under anaesthesia was made.

CORRESPONDENCE

HYPOTHERMIA FOR COARCTATION?

Sir,—On page 95 of the March 1959 issue of your Journal I read “Hypothermia seems likely to remain the method of choice for patients requiring operation for coarctation of the aorta”, whereas on page 125 of the same issue I read “Hypothermia is unnecessary in the correction of coarctation of the aorta in view of the extensive collateral circulation”. Am I correct in continuing to assume the latter to be the generally accepted view?

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