ABSENT TACHYCARDIA IN HYPOVOLAEMIC SHOCK AFTER CHOLECYSTECTOMY

SINGAPORE MED J 1996; Vol 37: 328

Dear Sir,

Tachycardia is often considered amongst the earliest cardiovascular responses to hypovolaemia, and usually occurs before the detectable fall in blood pressure. We report here a case of hypovolaemic shock with no accompanying tachycardia.

In preparation for a routine cholecystectomy, a 49-year-old female was given 75μg of fentanyl and 6mg of pancuronium intravenously. After 3 minutes of oxygenation, anaesthesia was induced with 100 mg diprivan and sustained with N_2O and isoflurane. The patient's blood pressure and pulse remained within 130-140/70-80 mmHg and 70-80 beats per minute respectively, throughout the procedure. However, one and a half hours post-operatively, the patient's systolic blood pressure fell to about 110 mmHg. This was followed by episodes of hypotension where the systolic blood pressure varied between 80 and 110 mmHg, the rises coinciding with fluid infusions. The systolic pressure at one instance dropped to as low as 60 mmHg. The diastolic pressure varied between 45 and 70 mmHg. Interestingly, despite the widely fluctuating arterial blood pressure, the patient's heart rate remained fairly constant, varying only between 70 and 85 beats per minute. The patient was conscious, cold, clammy with noticeable pallor. Her chest X-ray, ECG and cardiac enzymes were normal. She was taken back to the operation theatre and approximately 1.5L of blood and clots were removed from the peritoneal cavity. The bleeding vessel in the liver bed was ligated. The patient's blood pressure remained stable thereafter. She made an uneventful recovery and was discharged well.

The precise reason for the absent rebound tachycardia in the presence of hypotension in this patient is uncertain. Perioperatively, there are three main causes of hypotension: (i) hypovolaemia, (ii) anaesthetic overdose or adverse reaction, and (iii) pump failure. An acute loss of circulating blood leads to a reduction in baroreceptor input to the brain stem and, as a result, a corresponding increase in the activity of the sympathetic nervous system. Increased sympathetic discharge causes three general types of cardiovascular responses: (a) the heart rate and level of myocardial activity increase, (b) generalised arteriolar constriction in the skin, viscera and muscles, and (c) constriction of the capacitance vessels, particularly those in the viscera squeezing blood, normally stored in these depot areas, into the active circulation. These help maintain cardiac output. The compensatory mechanisms produce most of the signs and symptoms observed following severe haemorrhage. Increased sympathetic activity, as evidenced by these changes, was observed in the patient except for the increase in heart rate. The precise reason for the absence of the compensatory increase in heart rate is obscure. Isoflurane is known to be both a myocardial depressant[1,2] and a vasodilator[3,4] and could explain the hypotensive crisis in some instances. But because the hypotension in this patient occurred one and a half hours post-operatively and tachycardia secondary to isoflurane-induced hypotension is well documented[5], it is unlikely that the fall in blood pressure was secondary to the anaesthetic agents. Besides, the finding of about 1.5L of blood and clots in the peritoneal cavity and the administration of intravenous fluids raising the blood pressure, albeit temporarily, suggest that the hypotension in this patient was primarily due to decreased circulating volume. The reason for the absent tachycardic response to hypovolaemia in this patient remains obscure. It nevertheless indicates the possible existence of a defective baroreceptor reflex as a cause of uncompensated shock in some individuals.

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