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CASE REPORT

Termination of cardiopulmonary bypass facilitated by insulin

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Summary

A 61-year old woman with severe coronary artery disease underwent three-vessel coronary bypass surgery. Difficulty was encountered in terminating cardiopulmonary bypass, but there was dramatic improvement following the administration of intravenous insulin. The use of insulin to facilitate termination of cardiopulmonary bypass is discussed.

Key words

Anaesthesia; cardiac surgery, cardiopulmonary bypass.
Treatment; insulin.

For 50 years it has been known that insulin improves myocardial contractility.[1,2] More recently the beneficial effect of insulin on myocardial function after periods of ischaemia has been reported.[3] Despite various methods of myocardial preservation during cardiac surgery with cardioplegic solutions and/or profound local hypothermia, decreased myocardial performance at the termination of cardiopulmonary bypass (CPB) is still a common problem. A case is presented in which the administration of insulin resulted in successful termination of CPB in a diabetic patient, as indicated by several indices of myocardial performance.

Case history

A 61-yearold, 60 kg woman was admitted to the hospital for coronary artery bypass surgery. Three months before admission right and left heart catheterisation and selective coronary arteriography had been performed. The latter revealed severe coronary artery disease with 70% obstruction of the left main coronary artery and 80% and 70% obstruction of the left anterior descending and circumflex branches respectively. The right coronary artery was 60% obstructed. Overall left ventricular contraction, however, was good and the cardiac output as determined by thermodilution was 4·1 litres/minute. Pressures within the left and right heart were within normal limits as were the pulmonary artery and pulmonary capillary wedge pressures.

The patient's regular medication at the time of surgery consisted of nitroglycerin and propranolol for control of angina. Diabetes was controlled with insulin, 16 units of lente and 8 units of soluble in the morning and 6 units of lente in the

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evening. Pre-anaesthetic medication consisted of morphine 8 mg and hyoscine 0·4 mg intramuscularly and 12 units of soluble insulin subcutaneously, and was given one hour before arrival in the operating room. Before induction of anaesthesia a 20 gauge teflon catheter was inserted into her left radial artery and a 7Fr-thermodilution Swan-Ganz catheter was placed in the pulmonary artery via the right internal jugular vein.

Anaesthesia was induced with diazepam 0·6 mg/kg and lignocaine 6 mg/kg, and muscle relaxation was achieved with pancuronium 0·12 mg/kg. Anaesthesia was maintained with 50% N_2O in O_2 and lignocaine by infusion, a total of 15 mg/kg being administered before institution of CPB. This anaesthetic combination has been shown to provide stable haemodynamics intraoperatively without causing post-bypass myocardial depression.[4] Asystole was achieved by the infusion of potassium-containing cardioplegia solution.

Three vessel anastomoses were performed during CPB at an oesophageal temperature of 28·5°C. After 107 minutes of CPB, the patient was rewarmed and the heart successfully defibrillated. At this time the heart was in sinus rhythm, systolic arterial pressure was below 60 mmHg, cardiac output less than 2·0 litres/minute and left atrial pressure (LAP) 15 mmHg. However, it was not possible to terminate CPB, despite the use of various inotropic agents, including dopamine, calcium, isoprenaline and a mixture of noradrenaline and phentolamine. Despite the institution of intra-aortic balloon counterpulsation, the systolic arterial blood pressure remained below 80 mmHg and cardiac output was less than 2·5 litres/minute. At this time the blood glucose was 17·0 mmol/litre, the acid-base state was normal, and the plasma sodium, potassium and ionised calcium were within normal limits and the LAP was 16 mmHg. Soluble insulin, 15 units, was administered intravenously and within 5 minutes a 30% increase in cardiac output and a concomitant increase in arterial systolic pressure to 140 mmHg occurred. Stroke volume index (SVI) increased from 9·60 to 13·46 ml/beat/square metre and left ventricular stroke work index (LVSWI)[5] increased from 7·09 to 15·56 g m/square metre/beat. Fifteen minutes later cardiac output decreased to 1·5 litres/minute and systolic arterial pressure decreased to 100 mmHg. Ten units of soluble insulin were given, and again cardiac output and systolic arterial blood pressure increased to 2·7 litres/minute and 160 mmHg, respectively. SVI increased from 9·4 to 14·67 ml/beat/square metre and LVSWI increased from 8·46 to 18·75 g m/square metre/beat. The LAP was kept at 14–16 mmHg throughout this period. On a third occasion the haemodynamic values again deteriorated with immediate and successful reversal following intravenous insulin. These events are summarised in Table 1. Following the third dose of insulin it was possible to discontinue intra-aortic balloon counterpulsa-

Table 1. Summary of haemodynamic values before and after administration of insulin

		1st insulin dose (15 units)		2nd insulin dose (10 units)		3rd insulin dose (10 units)	
	Pre-surgery	Before	After 5 min	Before*	After 5 min	Before*	After 5 min
SAP (mmHg)	150	75	140	100	160	125	150
Cardiac output (litres/minute)	4·1	1·7	2·8	1·5	2·7	1·8	3·2
LAP (mmHg)	14†	16	16	15	15	15	14
SVI (ml/beat/metre^2)	33·97	9·60	13·46	9·40	14·67	10·20	17·40
LVSWI (g m/square metre/beat)	38·38	7·09	15·56	8·46	18·75	11·51	24·37

SAP = systolic arterial pressure; LAP = left atrial pressure; SVI = stroke volume index; LVSWI = left ventricular stroke work index.

* 15–20 minutes following preceding insulin dose.

† Left ventricular end diastolic pressure.

tion and all inotropic support. The immediate postoperative blood glucose was 14 mmol/litre and there were no further episodes of hypotension in the postoperative period.

Discussion

Despite improvement in myocardial preservation techniques, low cardiac output following myocardial revascularisation remains a problem. On most occasions the clinical condition of the patient improves with the administration of one of the more commonly used inotropic agents. These include dopamine, dobutamine, isoprenaline, noradrenaline, and glucagon. Mixtures of vasodilator and inotropic drugs have also been successfully employed, namely phentolamine plus noradrenaline[6] and nitroprusside plus dopamine[7] or adrenaline.[8] Intra-aortic balloon counterpulsation may be used in those patients whose cardiac output remains low despite the use of these agents. This mechanical assistance may increase cardiac index by 30%.[9] In the case presented here none of these measures enabled CPB to be successfully terminated.

Insulin is employed in a number of situations in which myocardial function is impaired. These include patients with congestive cardiac failure,[10] patients with coronary artery disease,[11] and patients who have suffered an acute myocardial infarction.[12] Insulin also reverses the myocardial depression produced by halothane.[13] The mechanism of the positive inotropic effect of insulin is unclear; however, subendocardial hypoperfusion and ischaemia are commonly associated with the low output syndrome that occurs after CPB.[14] In dogs, glucose-insulin-potassium solution improves subendocardial perfusion after anoxic arrest and CPB.[15]

In another study, perfusion of the heart during CPB with a glucose-insulin-potassium solution had a beneficial effect on myocardial function.[16] Insulin was also clearly responsible for a marked improvement in the recovery of ventricular function after a 30-minute period of severe ischaemia in a study on isolated rat hearts.[17]

At the cellular level insulin increases glucose transport across the cell membrane, but most evidence suggests that the positive inotropic effect of insulin is not related to glycolysis.[18] The rise in plasma osmolarity and reduction in plasma free fatty acids which occurs following insulin administration are probably more important in improving cardiac function.[3,11]

Insulin secretion decreases during CPB[19] and may lead to intra-operative hyperglycaemia. In the case presented however, the blood glucose was carefully monitored and at the first attempt to terminate CPB was 17 mmol/litre. Potassium-containing cardioplegia solution may lead to increased plasma potassium levels, but none had been infused in the hour before the first unsuccessful attempt to terminate CPB. The plasma potassium value at this time was 3·99 mmol/litre.

Since this case the authors have successfully used insulin in a number of patients to increase myocardial function at the termination of CPB, and it is concluded that administration of insulin with careful monitoring of plasma glucose and potassium levels may facilitate termination of CPB. This may be especially helpful in the diabetic patient.

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