CASE REPORTS

FAILURE TO WEAN FROM CARDIOPULMONARY BYPASS AFTER MYOCARDIAL REVASCULARIZATION: SUCCESSFUL TREATMENT WITH VERAPAMIL VIA THE AORTIC ROOT

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SUMMARY
Severe ventricular dysfunction in a patient prevented weaning from cardiopulmonary bypass after myocardial revascularization. Calcium chloride and increasing doses of dopamine had no effect. Coronary vasospasm was diagnosed based on ST elevation and myocardial failure. Verapamil 0.5 mg, injected into the aortic root, was followed by a dramatic improvement in cardiac contractility and successful weaning from cardiopulmonary bypass without inotropic support (Br. J. Anaesth. 1993; 71: 589-591)

KEY WORDS

Coronary vasospasm is an important cause of circulatory collapse after successful coronary artery bypass grafting. It may involve internal mammary artery [1-3] and saphenous vein [4, 5] grafts, in addition to native coronary arteries [2, 4, 6-8]. The phenomenon may be observed at any time during and shortly after cardiopulmonary bypass (CPB) [3, 5, 9], in the immediate postoperative period [1-3, 6, 10] and weeks [11] to months [11, 12] after surgery. Detailed reports on coronary vasospasm as a cause of inability to wean a patient from CPB are rare [8, 9]. In one patient [8] spasm persisted despite intracoronary infusion of nitroglycerin 8 mg, but was apparently relieved after nifedipine 30 mg was given via a nasogastric tube. The patient suffered a myocardial infarction. In another report [9], spasm occurred after injection of calcium chloride and was relieved by i.v. nitroglycerin. We are unaware of any report of the use of aortic root injection of verapamil to overcome coronary vasospasm and allow discontinuation of CPB. In the patient reported here, verapamil 0.5 mg relieved the spasm and profound myocardial dysfunction promptly and completely.

CASE REPORT
A 53-yr-old, obese male (101 kg) presented with angina pectoris precipitated by minimal exertion and after heavy meals, unresponsive to nitroglycerin spray, atenolol and aspirin. He had suffered a myocardial infarction 8 months previously. There was no history of vasospastic angina or intake of calcium channel antagonist. Coronary angiography revealed 70-80% stenosis of the right coronary (dominant), left main and left anterior descending arteries. The left ventricle was moderately hypokinetic and there was no valvular disease.

On the morning of the patient's planned coronary bypass surgery (CABG), he received his usual dose of atenolol and was sedated. In the operating room, routine monitors and a pulmonary artery catheter were placed. Anaesthesia was induced with fentanyl 75 μg kg⁻¹ and vecuronium 0.1 mg kg⁻¹ was given to facilitate tracheal intubation. The heart rate was increased with pancuronium 5 mg to 55±3 beat min⁻¹. Pulmonary capillary wedge pressure (PCWP) was stable at about 12 mm Hg. Cardiac index was 3.3 litre min⁻¹ m⁻². Before CPB was commenced, there were three spontaneous and apparently unprovoked episodes of systemic hypotension (systolic pressure 80-85 mm Hg) accompanied by 2-3 mm ST elevation in lead II, an increase in PCWP to 32-35 mm Hg, and the presence of the prominent V waves of mitral regurgitation. All episodes were promptly reversed with small i.v. doses of nitroglycerin, dopamine and phenylephrine.

The left internal mammary artery and saphenous vein were mobilized and wrapped in papaverine-soaked gauze. The vein was also filled with heparinized blood containing papaverine. Subsequently, CPB was initiated smoothly and asystole was achieved throughout the CPB period with cardioplegia solution (Na 140, K 20, Mg 3, Ca 2, Cl 115; hydrocortisone 100 mg litre⁻¹ and hypothermia (28°C)). Saphenous vein grafts were anastomosed to the distal posterolateral circumflex artery and the right coronary artery, and the internal mammary artery was grafted to the left anterior descending artery. Total aortic cross-clamp time was 74 min. Phenylephrine 100 μg was given 20 min from the initial aortic cross-clamp for arterial pressure regulation. A small dose of nitroglycerin was given during the re-warming phase. Normal sinus rhythm and normothermia were achieved and the relatively empty heart was allowed to beat for 30 min. Cardiac contractions were very poor.
An attempt was made to discontinue CPB by occlusion of the venous cannula, transfusion and the administration of calcium chloride 1 g i.v. (given routinely at this stage in our institution, unless the bypass time was very short and cardiac contractility obviously excellent). This resulted in overdistension of the ventricles, 4-mm ST-segment elevation in lead II, markedly increased pulmonary arterial pressures, markedly reduced systemic pulse pressure and poor pumping action by the ventricles. The severe hypokinesis in marked contrast to the pre-CPB state, showed no improvement with the addition of dopamine 5 μg·kg⁻¹·min⁻¹. CPB was promptly re-instituted.

Another attempt at weaning from CPB 30 min later was again unsuccessful. No calcium chloride was given at this and subsequent attempts. An intra-aortic balloon pump (IABP) and a left atrial catheter were inserted. After another 1 h of CPB, a third attempt at weaning resulted in failure. CPB was re-instituted for the fourth time.

In view of the widespread ST changes consistent with coronary vasospasm, verapamil 0.5 mg in saline 1 ml was then injected into the aortic root. One minute later, another attempt at weaning was successful, without the use of any inotropic support. Ventricular contraction was vigorous and the systolic pressure > 100 mm Hg, while the left- and right-sided filling pressures remained decreased (13–17 mm Hg). The ST changes largely resolved and an infusion of verapamil 1 mg h⁻¹ was commenced. Nitroglycerin was used after injection of protamine to allow transfusion of pump and other fluids into the patient. The final cardiac index was 3.4 litre min⁻¹ m⁻². The remainder of the surgery was uneventful.

In the Intensive Care Unit, the patient was given verapamil 1–2 mg h⁻¹ and hypertension was treated with nitroglycerin as required. Cardiac performance was deemed satisfactory even when the IABP was not used. There were no further episodes of coronary vasospasm and the IABP was discontinued 2 days later. Cardiac enzymes were not measured after operation. Except for pneumonia and confusion in the Intensive Care Unit, the remainder of the postoperative course was uneventful. The patient was discharged 10 days after surgery and remained angina-free and had continued to take atenolol and aspirin, but no calcium antagonist, during 14 months of follow-up.

**DISCUSSION**

The mechanisms of severe deterioration of myocardial function when weaning from CPB include [13] poor myocardial protection, excessive oxygen demand and decreased oxygen supply. Myocardial protection in this patient was performed with a standard technique used in thousands of successful cases in this institution, and probably elsewhere. We have no reason to believe the technique to have been inadequate. Conditions that cause excessive oxygen demand (hypertension and tachycardia) were absent. A primary decrease in oxygen supply was thus probably responsible. Laboratory measurements throughout the case excluded hypoxaemia and anaemia. Coronary insufficiency as a result of inadequate revascularization or diffuse stenosis was not the cause. Coronary air embolism may occur soon after unclamping of the aorta with the resumption of cardiac contractions, but it usually disperses quickly with adequate coronary perfusion pressure and rarely recurs [13]. The persistent and unrelenting nature of the ischaemia in this patient does not support air embolism as the major culprit, but it is difficult to exclude embolism or thrombosis as a contributory factor, although the relatively small PCV and impaired haemostatic properties of the blood after CPB do not promote thrombus formation. Our patient, a Caucasian, was unlikely to have had sickle cell disease. The most convincing evidence suggesting that coronary vasospasm was the dominant cause of this patient's profound ventricular failure was the rapid resolution of myocardial dysfunction and ischaemic changes immediately after the injection of verapamil.

The reported incidence of recognized coronary vasospasm (about 0.8–2.5% [7, 13]) is in keeping with our experience. Spasm is usually recognized because of its catastrophic presentation. It was responsible for six of 19 cases (32%) of sudden circulatory collapse after myocardial revascularization in one study [7], while Holter monitoring for at least 12 h after operation revealed an 8% incidence of ST elevation consistent with coronary vasospasm in another [14]. Less severe spasm may be missed and unreported [15]. We propose that the following factors may play a preventive role; routine continuation of nitrates and calcium antagonists before operation on the day of surgery; the use of high-dose opioid anaesthesia; routine wrapping of vessels in papaverine-soaked gauze and the use of papaverine-containing blood to fill the vessels before grafting; the presence of magnesium in the cardioplegic solution; the perfusion of the coronary arteries with warm cardioplegia solution towards the end of CPB; the administration of nitroglycerin towards the end of CPB; and the treatment of perioperative hyper-tension with nitrates.

The cause of coronary vasospasm associated with CPB is unknown. Many factors have been proposed [7–9, 13]: excessive alpha adrenergic stimulation, beta adrenergic block, alkalosis and reduced $P_{aCO_2}$, cold, local trauma, release of vasoconstrictor substances from the endothelium, ischaemia, release of vasoconstrictor substances by platelets, increased concentration of potassium, reduced extracellular magnesium concentration, sudden increase in extracellular calcium, increase in plasma vasopressin concentration, vasodilator and calcium antagonist withdrawal, administration of protamine or transfusion-induced histamine liberation causing an H₁ receptor mediated coronary vasoconstriction, and an increased thrombin concentration upon heparin antagonism with protamine. The relative importance of these factors is unknown. Our patient had three probable episodes of spasm before CPB, suggesting the presence of triggering factors and susceptibility. The recurrence of spasm in a much more severe fashion after coronary artery bypass suggests that surgical manipulation, hypothermic CPB, or both,
had contributed to the problem. It is possible that the phenylephrine used in the early phase of CPB in this patient contributed. The ventricles were functioning poorly before calcium chloride was given, but the drug did not help and could possibly have exacerbated the spasm [9].

Coronary vasospasm should be part of the differential diagnosis of ischaemia after CPB, irrespective of the presence or absence of preoperative Prinzmetal's angina and the type of vessel used for grafting. Recognition is by ST segment elevation or, less frequently, ST depression [4, 5, 16] accompanied by decreased contractility, arrhythmias, or both. When spasm occurs in isolated epicardial vessels, the surgeon should locate it by carefully examining the coronary vessels when and where possible, and should also palpate or inspect for suspected areas of akinesis or dyskinesia that had not been present before.

The treatment of coronary vasospasm is with vasodilators and avoidance of exacerbating factors whenever possible. I.v. nifedipine and nitroglycerin [8, 9, 13] are successful only occasionally, and may worsen hypotension. When circulatory collapse occurs with spasm after the chest is closed, or in the intensive care unit, cardiac massage may necessitate re-opening of the sternotomy site. When bedside resuscitation is unsatisfactory, return to the operating room for re-establishment of anticoagulation and CPB has been life saving. The intracoronary infusion of verapamil [6], nitroglycerin [6], or papaverine [2] by a cardiologist in the catheterization laboratory has been successful also. When spasm occurs while the patient is still on the operating table with the chest open, treatment will include one or more of the following: re-institution of CPB; administration of a calcium channel antagonist [10]; coronary perfusion with papaverine [3], nitroglycerin [5] or a calcium antagonist by aortic root or direct coronary injection; i.v. nitroglycerin [8]; the replacement of spastic coronary vessels with new grafts; the direct application of papaverine to the spastic vessel [1]; and avoidance of conditions that exacerbate coronary vasospasm.

Many inotropes and vasopressors (calcium chloride, dopamine, adrenaline, phenylephrine) may exacerbate coronary vasospasm. In the face of myocardial depression, the natural tendency is to increase the doses of these agents; this seems unlikely to improve outcome of coronary vasospasm, as recurrence is well documented [7, 8].

Verapamil prolongs A-V node conduction and thus may add to the effect of hyperkalaemia. It is probably prudent to give an infusion of verapamil after the initial relief of coronary vasospasm, as recurrence is well documented [7, 8].

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REFERENCES