Active Aorto-iliac Bypass for Thoraco-abdominal Aortic Aneurysm Repair

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Introduction

Surgical repair of thoraco-abdominal aortic aneurysms (TAAA) requires interruption of flow to vital structures including the spinal cord, kidneys and viscera. Ischaemic damage will occur if the clamp time is prolonged,1,2 and in the absence of distal organ perfusion, postoperative renal failure is common.3,4 In addition, the incidence of paraplegia is markedly reduced by distal perfusion techniques.¹

Application of an aortic cross clamp increases the left ventricular afterload, particularly when the clamp is placed on the thoracic aorta,⁵ as up to 45% of the cardiac output is normally directed to the renal and splanchnic circulation. The arterial blood pressure above the clamp may become unsustainably high, resulting in left ventricular failure (LVF), myocardial infarction (MI), cerebral haemorrhage and raised intracranial pressure (ICP).⁵,⁶ Pharmacological approaches to reducing the blood pressure frequently result in further elevation of the ICP.⁷

The technique of partial left heart bypass with shunting of blood from the left atrium to a femoral or iliac artery via a roller or centrifugal pump is widely used to overcome these problems.⁸,⁹ Blood is drained from the left atrium resulting in reduction in left ventricular preload, the left ventricular stroke volume is therefore reduced decreasing the cardiac output, and producing a fall in blood pressure proximal to the aortic clamp. The distal aorta is retrogradely perfused while the proximal anastomosis is completed. However this technique can only usually be used where facilities for cardiopulmonary bypass are available.

Another technique described to maintain distal perfusion is the use of a temporary axillo-femoral bypass graft.¹⁰ This has the disadvantage of requiring an additional operative procedure and also relies on left ventricular pump function. This paper describes a modification of this technique, using an extra-corporeal proximal-to-distal aortic bypass, driven by an active centrifugal pump.

Technique

A 63-year-old man presented with a 7 cm thoraco-abdominal aortic aneurysm. This arose distal to the left subclavian artery, involved the visceral and renal arteries and extended to the aortic bifurcation (Crawford Type III). Anaesthetic technique included the use of a Robertshaw double lumen endobronchial tube for one lung ventilation and intraoperative analgesia was provided by a remifentanil infusion. A thoracic epidural was sited but not used until required for post operative analgesia. Right radial and femoral arterial catheters were inserted in order to directly monitor both proximal and distal blood pressure and a pulmonary artery flotation catheter inserted via the right internal jugular vein. A spinal drain placed in the L 3/4 interspace was primed to drain if cerebrospinal fluid (CSF) pressure rose above 10 mmHg. The urinary bladder was catheterised.

One million units of aprotinin¹¹ were given over an hour, followed by an infusion of 500 000 units per hour. Blood suctioned from the surgical wound was saved for autologous transfusion and processed using a Haemonetics Cell Saver. All intravenous fluids were warmed. A Haemonetics Rapid Infusion System was utilised and a forced air warming blanket was applied to the patient.
Active Aorto-iliac Bypass for TAAA Repair

Cardiac output and systemic vascular resistance (Table 1). The clamp was moved down the graft and the anastomosis tested. The renal and visceral anastomoses were then performed, with a total ischaemic time of 15 min. Prior to the reperfusion of the viscera 170 ml of mannitol 20% was given. Arterial blood gases taken after visceral reperfusion showed a metabolic acidosis with a pH 7.29 and Base Excess $-7.7$, which had returned to normal without the use of sodium bicarbonate solution by the end of the procedure (Table 2). Throughout these procedures lower limb perfusion was maintained by the bypass system.

The final step was completion of the distal anastomosis. This required a period of limb ischaemia with discontinuation of the active bypass for a short period of 2 min to facilitate surgical access. Bypass was discontinued and the aorta and iliac arteries de-cannulated. Total active bypass time was 70 min.

Postoperatively the patient was recovered on the Intensive Therapy Unit (ITU). An epidural infusion was commenced. He was extubated within 12 h and remained in the ITU for two days prior to transfer to a High Dependency Unit. The patient made a good recovery with no renal impairment and no paraplegia. There were no adverse cardiac sequelae.

The same technique was utilised to repair a 6.6 cm Crawford type III TAAA in a 65-year-old man. He had undergone repair of an infrarenal AAA three years previously and had suffered a myocardial infarction ten months earlier, requiring coronary artery bypass grafting. He had a history of essential hypertension and was taking Diltiazem and low dose aspirin. His TAAA was repaired as above using an active aorto-iliac bypass commenced via a Carmino-bonded $\frac{3}{8}$ inch tubing driven by a Biomedicus centrifugal pump (Fig. 1). Prior to initiation of bypass 5000 units of heparin was given and frusemide ($1 \mu g/kg^{-1}/min^{-1}$) and dopamine infusions ($3 \mu g/kg^{-1}/min^{-1}$) were commenced. A phenylephrine infusion was used to maintain proximal mean blood pressure above 65 mmHg. ST segment analysis of the ECG revealed 2 mm depression in lead V5, which was treated with an infusion of glyceryl trinitrate, $1 \mu g/kg^{-1}/min^{-1}$, which was continued into the postoperative period.

Effective bypass was established, the aorta cross-clamped and the proximal anastomosis performed. There were minimal changes in arterial blood pressure, cardiac output and systemic vascular resistance (Table 1). The clamp was moved down the graft and the anastomosis tested. The renal and visceral anastomoses were then performed, with a total ischaemic time of 15 min. Prior to the reperfusion of the viscera 170 ml of mannitol 20% was given. Arterial blood gases taken after visceral reperfusion showed a metabolic acidosis with a pH 7.29 and Base Excess $-7.7$, which had returned to normal without the use of sodium bicarbonate solution by the end of the procedure (Table 2). Throughout these procedures lower limb perfusion was maintained by the bypass system.

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He was recovered on the Intensive Therapy Unit, where an epidural infusion was commenced and he was ventilated for 12 h. He showed no renal impairment and no paraplegia and after two days was discharged to the High Dependency Unit.

**Discussion**

The effect of clamping the proximal aorta on the blood pressure, cardiac output and ventricular function has been previously described. When a passive shunt is used cardiac output and left ventricular stroke work
Table 1. Effect of the proximal aortic cross clamp on haemodynamic parameters

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<thead>
<tr>
<th></th>
<th>Case 1</th>
<th></th>
<th>Case 2</th>
<th></th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Pre-clamp</td>
<td>Post-clamp</td>
<td>Pre-clamp</td>
<td>Post-clamp</td>
</tr>
<tr>
<td>Radial mean arterial pressure mmHg</td>
<td>85</td>
<td>95</td>
<td>75</td>
<td>73</td>
</tr>
<tr>
<td>Cardiac output l/min</td>
<td>5.4</td>
<td>5.1</td>
<td>4.8</td>
<td>4.3</td>
</tr>
<tr>
<td>Systemic vascular resistance Dyne/s/cm$^{-5}$</td>
<td>1110</td>
<td>1300</td>
<td>1025</td>
<td>1045</td>
</tr>
</tbody>
</table>

Table 2. Effect of bypass on arterial acid–base balance

<table>
<thead>
<tr>
<th></th>
<th>Case 1</th>
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<th>Case 2</th>
<th></th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Pre-bypass</td>
<td>Post-bypass</td>
<td>Pre-bypass</td>
<td>Post-bypass</td>
</tr>
<tr>
<td>pH</td>
<td>7.42</td>
<td>7.42</td>
<td>7.4</td>
<td>7.33</td>
</tr>
<tr>
<td>Base excess mmol/l$^{-1}$</td>
<td>−1.1</td>
<td>−2.7</td>
<td>−2.6</td>
<td>−5.1</td>
</tr>
<tr>
<td>Bicarbonate mmol/l$^{-1}$</td>
<td>22.7</td>
<td>17.9</td>
<td>22.3</td>
<td>18.7</td>
</tr>
</tbody>
</table>

remain depressed suggesting impaired left ventricular function.

Clamping of the proximal aorta also renders organs whose arterial supply is distal to the cross clamp ischaemic. This results in renal failure in approximately 30% of patients after TAAA repair, and spinal cord ischaemia resulting in paraplegia or paraparesis in up to 20% of patients. Liver and gut ischaemia may be implicated in the genesis of postoperative multi-organ failure.

The pathogenesis of paraplegia is multifactorial. In addition to the disconnection of arteries supplying the cord, the CSF pressure is increased by the hypertension produced by proximal aortic clamping. Spinal artery pressure is reduced further by vasodilators used to treat the proximal hypertension, and sodium nitroprusside may increase CSF pressure directly.

Distal perfusion reduces proximal hypertension, distal organ ischaemia, and the requirement for vasodilators. Various methods of distal aortic grafting have been described. Axillo-femoral bypass grafts can be used and this technique is available to vascular surgical units without access to extra-corporeal bypass equipment. Passive shunts have been described, but flow may be insufficient. Partial left heart bypass is now the most widely used procedure. This method may incorporate a venous reservoir, or avoid this using a centrifugal pump. In the former case, full heparinisation is required which may be reduced if heparin-bonded tubing is used. Cardiopulmonary bypass is also used in some centres, usually from the femoral route.

Our decision to use an actively pumped aortic shunt, derives from experience of veno-venous bypass gained during liver transplantation. For type III TAAA repair, proximal and distal arterial pressures can be kept the same by control of pump speed. The technique is simple, and results in no major changes in blood pressure, cardiac output or filling pressures. The renal and splanchnic ischaemic time can be kept to a minimum and CSF pressure does not change significantly.

The technique described differs significantly from the more commonly used atrio-femoral bypass in two respects. Firstly, there is no need to cannulate the left atrium, which in itself may cause problems outside a cardiac surgical unit. Secondly, the physiology of the bypass technique is different. Aortic clamping causes proximal hypertension due to an increase in afterload.

In our technique, this problem is avoided by avoiding the increase in afterload, rather than reducing proximal blood pressure by reducing preload. The lack of requirement for the use of vasodilators or other agents to control blood pressure is testament to the haemodynamic stability produced by this technique.

Nevertheless it must be stressed that this technique is only applicable to patients with type III TAAAs who have adequate non-diseased artery between the left subclavian artery and the commencement of the aneurysmal dilatation. If this proximal portion of the descending aorta is calcified, severely diseased or aneurysmal (Type II TAAA), left heart bypass techniques should be employed. In addition, this technique does not involve cooling of the patient and total renal and visceral ischaemic times must be kept to a minimum by precise and efficient anastomotic technique.

References


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