REPAIR OF TRAUMATIC TRANSECTION OF THE THORACIC AORTA: ESMOLOL FOR INTRAOPERATIVE CONTROL OF ARTERIAL PRESSURE

S. G. FENNER, A. MAHONEY AND J. N. CASHMAN

SUMMARY

We report the intraoperative use of esmolol for control of arterial pressure during repair of a traumatic transection of the descending thoracic aorta. A mean infusion rate of esmolol 50.5 μg kg⁻¹ min⁻¹ resulted in a decrease in mean arterial pressure to 63 mm Hg and heart rate to 99 beat min⁻¹ and was associated with excellent surgical conditions. The infusion rate of esmolol was titrated easily against mean arterial pressure, which increased rapidly on discontinuing its infusion. Control of arterial pressure with esmolol was comparable to that achieved with sodium nitroprusside, but without the reflex tachycardia or decrease in PaO₂, associated with the latter agent.

KEY WORDS


CASE REPORT

A previously healthy 20-yr-old man was admitted to hospital following a road traffic accident. Clinical and radiological examinations at the referring hospital had revealed multiple fractures, central abdominal peritonism, but no evidence of significant head or chest injury. After resuscitation with i.v. fluids, laparotomy was performed and damaged small bowel and mesentery were resected, his fractured right elbow was stabilized in plaster of Paris and a right distal tibial traction pin was inserted. On the first day after operation the patient complained of central chest pain and another chest x-ray revealed widening of the superior mediastinum. A provisional diagnosis of intrathoracic aortic transection was made. The patient was transferred to this Regional Cardiothoracic Unit for investigation and management of suspected intrathoracic aortic injury. On his arrival, assessment revealed a conscious patient with an arterial pressure of 110/70 mm Hg, heart rate 100 beat min⁻¹, normal peripheral pulses and good urine output. A subsequent arch aortogram revealed a leak of contrast just distal to the left subclavian artery, consistent with transection of the descending thoracic aorta. The patient was transferred immediately to the operating theatre for repair of this defect via a left thoracotomy.

In the anaesthetic room, the right radial and posterior tibial arteries were cannulated for measurement of proximal and distal arterial pressures during aortic cross-clamping. A 7-French gauge thermodilution pulmonary artery catheter (American Edwards Laboratories) was placed via the right internal jugular vein. In the operating theatre full invasive haemodynamic monitoring was commenced during preoxygenation. Anaesthesia was induced with diamorphine 200 μg kg⁻¹ i.v. followed by thiopentone 2 mg kg⁻¹ i.v. Suxamethonium 1.5 mg kg⁻¹ i.v. was administered to facilitate intubation with a 37-French gauge left-sided double-lumen endobronchial tube. Further increments of diamorphine were given to a total dose of 400 μg kg⁻¹.

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i.v. Anaesthesia during the 5-h procedure was maintained by intermittent positive pressure ventilation with 25–50 % nitrous oxide and enflurane in oxygen with intermittent boluses of fentanyl (total 15 μg kg⁻¹ i.v.) and pancuronium (total 250 μg kg⁻¹ i.v.). It was decided to use an i.v. infusion of esmolol (Brevibloc-Dupont Pharmaceuticals) to control systemic arterial pressure during aortic dissection, before aortic cross-clamping. The authors were interested also to compare the cardiovascular conditions provided by esmolol with those provided by sodium nitroprusside (SNP) (currently our preferred agent for the intraoperative control of arterial pressure). Therefore, after 1 h the infusion of esmolol was changed to an i.v. infusion of SNP.

During the course of surgery cardiac output, pulmonary artery pressures, pulmonary capillary wedge pressures (PCWP) and arterial blood-gas measurements were recorded at intervals in addition to the usual variables recorded during an intrathoracic vascular procedure. After induction of anaesthesia, arterial pressure stabilized at 80/55 mm Hg with a heart rate of 110–120 beat min⁻¹. However, within 15 min of the commencement of surgery, arterial pressure and heart rate had increased to 130/60 mm Hg and 140 beat min⁻¹, respectively. At this point an infusion of esmolol 150 μg kg⁻¹ min⁻¹ was commenced without a bolus loading dose. This produced a rapid decrease in heart rate and arterial pressure to 95 beat min⁻¹ and 80/45 mm Hg, respectively (figs 1, 2). During the next 60 min of one lung-ventilation, before aortic cross-clamping, the infusion of esmolol was adjusted to maintain mean arterial pressure (MAP) at an average of 63 mm Hg, with an average heart rate of 99 beat min⁻¹ (fig. 1).

After esmolol was discontinued, MAP was controlled for the remaining 20 min before application of the aortic cross-clamps with an infusion of SNP which was commenced at a rate of 1.5 μg kg⁻¹ min⁻¹ (fig. 1). This produced a rapid decrease in arterial pressure to 72/44 mm Hg (fig. 2), but was associated with an increase in heart rate to 125 beat min⁻¹ (fig. 1). There was an increase in cardiac index during infusion of SNP compared with values obtained both before induction and during the infusion of esmolol. Arterial blood-gas measurements revealed a decrease in PaO₂ from 10 kPa before infusion of SNP to 7.7 kPa during infusion of SNP, despite unchanged ventilation and inspired oxygen concentration.
Table I. Proximal mean arterial pressures (MAP), pulmonary capillary wedge pressures (PCWP), cardiac indices and systemic arterial oxygen partial pressures (PaO₂) in relation to intraoperative events and drug infusions during repair of transected thoracic aorta. OLV = One lung ventilation; x-clamp = aortic cross-clamps applied; n/a = not available

<table>
<thead>
<tr>
<th>Time (min)</th>
<th>MAP (mm Hg)</th>
<th>PCWP (mm Hg)</th>
<th>Cardiac index (litre min⁻¹ m⁻²)</th>
<th>Arterial PaO₂ (kPa)</th>
<th>Drug infusions</th>
<th>Events</th>
</tr>
</thead>
<tbody>
<tr>
<td>-5</td>
<td>77</td>
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<td>4.50</td>
<td>n/a</td>
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</tr>
<tr>
<td>75</td>
<td>54</td>
<td>11</td>
<td>3.21</td>
<td>10.00</td>
<td>Esmolol 60 μg kg⁻¹ min⁻¹</td>
<td>OLV</td>
</tr>
<tr>
<td>135</td>
<td>70</td>
<td>12</td>
<td>5.90</td>
<td>12.40</td>
<td>Nil</td>
<td>OLV + x-clamp</td>
</tr>
<tr>
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<td>12</td>
<td>4.94</td>
<td>n/a</td>
<td>Nil</td>
<td>OLV + x-clamp</td>
</tr>
<tr>
<td>285</td>
<td>56</td>
<td>6</td>
<td>5.12</td>
<td>7.70</td>
<td>SNP</td>
<td>OLV</td>
</tr>
</tbody>
</table>

Cardiac index measurements, arterial PaO₂, MAP and PCWP obtained before induction, during the infusion of esmolol, during aortic cross-clamping and during the infusion of SNP are shown in Table I.

The aortic cross-clamps were applied for 75 min, during which time the 95% transection of the descending aorta was repaired with a 16-mm Gelseal (Vascutek) graft whilst distal perfusion pressure (measured at the right posterior tibial artery) was maintained with a 7-mm Gott shunt (Argyle). At the surgeon’s request, SNP was discontinued after aortic cross-clamping in order to maintain distal arterial pressure, which had decreased rapidly after the application of the cross-clamps. Distal MAP stabilized at 50 mm Hg after stopping SNP; proximal MAP was 117 mm Hg. The infusion of SNP was recommenced just before the aortic cross-clamps were released and was adjusted to achieve a proximal...
MAP of 60–70 mm Hg. Unfortunately, this was associated with an increase in heart rate from an average of 123 beat min⁻¹ during aortic cross-clamping to 132 beat min⁻¹ after the cross-clamps had been released. A reduction in this heart rate was achieved before the end of surgery by administration of three increments of labetalol 10 mg i.v.

After operation the lungs were ventilated mechanically for 48 h and the patient sedated with an i.v. infusion of papaveretum and intermittent i.v. bolus doses of diazepam. Arterial pressure was controlled with i.v. infusions of labetalol and SNP. During this period the patient underwent a 12-h orthopaedic operation for internal fixation of his fractured long bones. The patient was discharged eventually back to the referring hospital 4 days after the aortic repair.

**DISCUSSION**

Esmolol is an ultra-short acting β₁-adrenoceptor antagonist. In normal subjects after i.v. administration, it has distribution and elimination half-lives of only 2 min and 9 min [1], respectively. Esmolol has a rapid onset of action: an effect on heart rate is observed within 3 min [2]. It is metabolized rapidly, predominantly by erythrocyte esterases, to an acid metabolite (a 1500-fold less potent adrenoceptor antagonist than esmolol) and methanol. Plasma cholinesterases are not involved in the metabolism and there is thus no significant effect on the inactivation of suxamethonium [3, 4]. Less than 1% of an administered dose of esmolol is recovered unchanged in the urine; more than 70% of the acid metabolite is recovered from the urine [5].

Esmolol has been shown to be effective in attenuating the hypertension and tachycardia resulting from laryngoscopy and tracheal intubation [2, 6, 7], for the control of intraoperative hypertension and tachycardia [7, 8] and has been used effectively as part of the management of arterial pressure during resection of phaeochromocytoma [9]. In addition, esmolol has been used successfully to control atrial tachyarrhythmia [10] and in the management of angina pectoris [11].

The authors are unaware of any previous report of the use of esmolol to control arterial pressure during surgery to repair transection of the thoracic aorta. In such cases, control of arterial pressure is important before application of aortic cross-clamps in order to prevent further aortic disruption and haemorrhage [12]. Furthermore, it is important after application of the cross-clamps in order to prevent acute left ventricular failure [12]. Even at relatively low infusion rates, esmolol proved to be extremely effective in controlling this patient's arterial pressure before aortic cross-clamping. The effect of esmolol on arterial pressure was adjusted easily because of its short elimination half-life. The negative chronotropic effect of esmolol in this patient was observed clearly when compared with SNP, which produced tachycardia. This effect of esmolol may be of considerable advantage in patients with ischaemic heart disease, in whom the tachycardia and reduced coronary artery perfusion pressure produced by SNP may impair the myocardial oxygen supply and demand relationship, which may in turn be associated with myocardial ischaemia [8, 13]. The negative chronotropic effect of esmolol may also be useful in younger patients who usually exhibit marked compensatory tachycardia when controlled hypotension is induced with an arteriolar dilating agent such as SNP. Furthermore, SNP causes a much greater decrease in diastolic arterial pressure than esmolol [14]. This may have important implications for maintenance of coronary and renal artery perfusion pressures. A further undesirable action of SNP is its effect on hypoxic pulmonary vasoconstriction (HPV). Esmolol has no discernible effect upon HPV, demonstrated by minimal changes in PCWP and PaO₂, as opposed to SNP which causes decreases in both [14]. We were interested to observe these differences between esmolol and SNP in our patient (table I).

Esmolol is relatively contraindicated in patients with left ventricular dysfunction, as it may reduce cardiac output further. Sodium nitroprusside usually causes an increase in cardiac output (as seen here) and therefore may be the preferred agent in this situation [14]. Some of the disadvantages of using SNP may be attenuated by concomitant use of a β-adrenoceptor antagonist such as propranolol or the mixed α- and β-adrenoceptor antagonist, labetalol [12, 15]. Both of these agents have elimination half-lives which are substantially longer than those of esmolol or SNP, making fine control of arterial pressure and heart rate during anaesthesia unpredictable and difficult to achieve. Any deleterious effects of β-adrenoceptor antagonism, caused by esmolol, should resolve quickly and spontaneously as a result of its rapid metabolic breakdown. A more
logical approach to control of arterial pressure during anaesthesia and surgery for cases such as this might be to use infusions of a short-acting vasodilating agent such as SNP in combination with an ultra-short acting β-adrenoceptor antagonist such as esmolol.

REFERENCES