Cuffed and uncuffed tracheal tube in children

Editor—We read with interest the article by Weiss and colleagues regarding the use of cuffed or uncuffed endotracheal tubes. Although this was once a hotly contested debate, we find that more and more centres are switching to the use of cuffed tubes in younger children and infants. Our experience has been the same and the validation of these data in a large prospective randomized trial only confirms our belief that this is indeed safe. One main advantage of the use of the cuffed tube is the absence of the multiple intubation attempts to change the tracheal tube when a significant leak is observed. The micro-cuff may be an additional benefit that we now have over the conventional cuffs. We fully endorse the authors’ view that cuffed tracheal tubes can be used safely in children.

C. M. Buoy*
S. Suresh
Chicago, USA
*E-mail: c-buoy@northwestern.edu

Concomitant levosimendan and esmolol infusion in ischaemic cardiogenic shock

Editor—Beta-adrenergic blockers and positive inotropic therapies are considered alternative strategies for the management of patients with heart failure. Inotropes improve haemodynamics, but have been associated with an increased risk of death and cardiac events with the possible exception of levosimendan.

We recently treated a patient with acute myocardial infarction (AMI) complicated by cardiogenic shock with simultaneous continuous infusion of levosimendan and esmolol.

A 56-yr-old male had a massive anterior AMI at 6 a.m. on a cruise. Onboard thrombolysis reduced the ST segment elevation. At 7:45 a.m., the patient was transported to the nearest hospital where clinical examination showed: arterial pressure (AP) 70/40, heart rate (HR) 145 beats min⁻¹, central venous pressure (CVP) 16 mm Hg, and peripheral oxygen saturation (SpO₂) 88%. Echocardiography showed an ejection fraction (EF) <20%, a restrictive diastolic pattern (the EI/E’ ratio > 13 and the EI/Vp ratio > 3), consistent with high wedge pressure), and mild mitral insufficiency. Non-invasively measured (Flo Trac/Vigileo™, Edwards Lifesciences, Irvine, CA, USA) cardiac index (CI) was 1.8 litre min⁻¹ m⁻². After tracheal intubation and positioning of a fibroptic intra-aortic balloon pump (IABP), levosimendan (0.1 μg kg⁻¹ h⁻¹) and norepinephrine (0.3 μg kg⁻¹ h⁻¹) were started. A percutaneous transluminal coronary angioplasty of the proximal left anterior descending (LAD) was performed with good results. TOE pulsed Doppler sampling of LAD showed an increase in flow velocity of 30% under IABP. Brain natriuretic peptide (BNP) concentration was 1240 pg ml⁻¹.

We recently treated a patient with acute myocardial infarction (AMI) complicated by cardiogenic shock with simultaneous continuous infusion of levosimendan and esmolol.

C. M. Buoy*
S. Suresh
Chicago, USA
*E-mail: c-buoy@northwestern.edu

Concomitant levosimendan and esmolol infusion in ischaemic cardiogenic shock

Editor—Beta-adrenergic blockers and positive inotropic therapies are considered alternative strategies for the management of patients with heart failure. Inotropes improve haemodynamics, but have been associated with an increased risk of death and cardiac events with the possible exception of levosimendan.

We recently treated a patient with acute myocardial infarction (AMI) complicated by cardiogenic shock with simultaneous continuous infusion of levosimendan and esmolol.

A 56-yr-old male had a massive anterior AMI at 6 a.m. on a cruise. Onboard thrombolysis reduced the ST segment elevation. At 7:45 a.m., the patient was transported to the nearest hospital where clinical examination showed: arterial pressure (AP) 70/40, heart rate (HR) 145 beats min⁻¹, central venous pressure (CVP) 16 mm Hg, and peripheral oxygen saturation (SpO₂) 88%. Echocardiography showed an ejection fraction (EF) <20%, a restrictive diastolic pattern (the EI/E’ ratio > 13 and the EI/Vp ratio > 3), consistent with high wedge pressure), and mild mitral insufficiency. Non-invasively measured (Flo Trac/Vigileo™, Edwards Lifesciences, Irvine, CA, USA) cardiac index (CI) was 1.8 litre min⁻¹ m⁻². After tracheal intubation and positioning of a fibroptic intra-aortic balloon pump (IABP), levosimendan (0.1 μg kg⁻¹ h⁻¹) and norepinephrine (0.3 μg kg⁻¹ h⁻¹) were started. A percutaneous transluminal coronary angioplasty of the proximal left anterior descending (LAD) was performed with good results. TOE pulsed Doppler sampling of LAD showed an increase in flow velocity of 30% under IABP. Brain natriuretic peptide (BNP) concentration was 1240 pg ml⁻¹.

We recently treated a patient with acute myocardial infarction (AMI) complicated by cardiogenic shock with simultaneous continuous infusion of levosimendan and esmolol.

C. M. Buoy*
S. Suresh
Chicago, USA
*E-mail: c-buoy@northwestern.edu

Concomitant levosimendan and esmolol infusion in ischaemic cardiogenic shock

Editor—Beta-adrenergic blockers and positive inotropic therapies are considered alternative strategies for the management of patients with heart failure. Inotropes improve haemodynamics, but have been associated with an increased risk of death and cardiac events with the possible exception of levosimendan.

We recently treated a patient with acute myocardial infarction (AMI) complicated by cardiogenic shock with simultaneous continuous infusion of levosimendan and esmolol.

A 56-yr-old male had a massive anterior AMI at 6 a.m. on a cruise. Onboard thrombolysis reduced the ST segment elevation. At 7:45 a.m., the patient was transported to the nearest hospital where clinical examination showed: arterial pressure (AP) 70/40, heart rate (HR) 145 beats min⁻¹, central venous pressure (CVP) 16 mm Hg, and peripheral oxygen saturation (SpO₂) 88%. Echocardiography showed an ejection fraction (EF) <20%, a restrictive diastolic pattern (the EI/E’ ratio > 13 and the EI/Vp ratio > 3), consistent with high wedge pressure), and mild mitral insufficiency. Non-invasively measured (Flo Trac/Vigileo™, Edwards Lifesciences, Irvine, CA, USA) cardiac index (CI) was 1.8 litre min⁻¹ m⁻². After tracheal intubation and positioning of a fibroptic intra-aortic balloon pump (IABP), levosimendan (0.1 μg kg⁻¹ h⁻¹) and norepinephrine (0.3 μg kg⁻¹ h⁻¹) were started. A percutaneous transluminal coronary angioplasty of the proximal left anterior descending (LAD) was performed with good results. TOE pulsed Doppler sampling of LAD showed an increase in flow velocity of 30% under IABP. Brain natriuretic peptide (BNP) concentration was 1240 pg ml⁻¹.

We recently treated a patient with acute myocardial infarction (AMI) complicated by cardiogenic shock with simultaneous continuous infusion of levosimendan and esmolol.

C. M. Buoy*
S. Suresh
Chicago, USA
*E-mail: c-buoy@northwestern.edu

Concomitant levosimendan and esmolol infusion in ischaemic cardiogenic shock

Editor—Beta-adrenergic blockers and positive inotropic therapies are considered alternative strategies for the management of patients with heart failure. Inotropes improve haemodynamics, but have been associated with an increased risk of death and cardiac events with the possible exception of levosimendan.

We recently treated a patient with acute myocardial infarction (AMI) complicated by cardiogenic shock with simultaneous continuous infusion of levosimendan and esmolol.

A 56-yr-old male had a massive anterior AMI at 6 a.m. on a cruise. Onboard thrombolysis reduced the ST segment elevation. At 7:45 a.m., the patient was transported to the nearest hospital where clinical examination showed: arterial pressure (AP) 70/40, heart rate (HR) 145 beats min⁻¹, central venous pressure (CVP) 16 mm Hg, and peripheral oxygen saturation (SpO₂) 88%. Echocardiography showed an ejection fraction (EF) <20%, a restrictive diastolic pattern (the EI/E’ ratio > 13 and the EI/Vp ratio > 3), consistent with high wedge pressure), and mild mitral insufficiency. Non-invasively measured (Flo Trac/Vigileo™, Edwards Lifesciences, Irvine, CA, USA) cardiac index (CI) was 1.8 litre min⁻¹ m⁻². After tracheal intubation and positioning of a fibroptic intra-aortic balloon pump (IABP), levosimendan (0.1 μg kg⁻¹ h⁻¹) and norepinephrine (0.3 μg kg⁻¹ h⁻¹) were started. A percutaneous transluminal coronary angioplasty of the proximal left anterior descending (LAD) was performed with good results. TOE pulsed Doppler sampling of LAD showed an increase in flow velocity of 30% under IABP. Brain natriuretic peptide (BNP) concentration was 1240 pg ml⁻¹.

We recently treated a patient with acute myocardial infarction (AMI) complicated by cardiogenic shock with simultaneous continuous infusion of levosimendan and esmolol.
Laryngeal mask airway Supreme™ for asleep–awake–asleep craniotomy

Editor—The asleep–awake–asleep technique with airway protection using laryngeal mask airway (LMA) has been proved safe for the anaesthetic management of awake craniotomies. However, re-insertion of LMA after awake test in a fixed neck position may sometimes be difficult. LMA Supreme™ (SLMA; Laryngeal Mask Company, Singapore) is a new disposable LMA with gastric access block that combines the desirable features of the LMA Unique™, LMA Proceal™ (PLMA), and intubating LMA Fastrach™ (ILMA). We report a successful use of SLMA for ‘asleep–awake–asleep’ craniotomy.

A 65-yr-old man (168 cm, 65 kg) was undergoing awake craniotomy for the removal of a frontotemporal glioma. When the patient arrived in the operating theatre, he was positioned with the neck slightly to the right in preparation for craniotomy. Anaesthesia was induced with target-controlled infusion of propofol and continuous infusion of remifentanil. A fully deflated and lubricated size-4 SLMA was inserted at the initial attempt without any excessive insertion force using a single-handed rotational technique like the ILMA2 by an anaesthetist standing at the patient’s right side with downward jaw traction and jaw thrust by another anaesthetist. This procedure was set to simulate re-insertion of SLMA after an awake test. Oropharyngeal leak pressure >30 cm H2O was achieved when the cuff was inflated with 25 ml of air. The vocal cords were visible through an endoscope from the distal end of the SLMA. A well-lubricated 14 Fr size gastric tube was inserted.

1 Task Force for Diagnosis and Treatment of Acute and Chronic Heart Failure 2008 of European Society of Cardiology. ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure 2008. Eur Heart J 2008; 19: 2388–442

doi:10.1093/bja/aqe013

Table 1 Clinical course from day 0 to day 4

<table>
<thead>
<tr>
<th></th>
<th>Day 0</th>
<th>Day 1</th>
<th>Day 2</th>
<th>Day 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR (beats min⁻¹)</td>
<td>140</td>
<td>63</td>
<td>65</td>
<td>60</td>
</tr>
<tr>
<td>AP (mm Hg)</td>
<td>70/40</td>
<td>110/68</td>
<td>120/65</td>
<td>125/60</td>
</tr>
<tr>
<td>CVP (mm Hg)</td>
<td>16</td>
<td>11</td>
<td>12</td>
<td>10</td>
</tr>
<tr>
<td>CI (litre min⁻¹ m⁻²)</td>
<td>1.8</td>
<td>2.4</td>
<td>2.7</td>
<td>2.8</td>
</tr>
<tr>
<td>BNP (pg ml⁻¹)</td>
<td>1240</td>
<td>750</td>
<td>480</td>
<td>320</td>
</tr>
<tr>
<td>EF (%)</td>
<td>&lt;20</td>
<td>36</td>
<td>40</td>
<td>42</td>
</tr>
<tr>
<td>Levosimendan i.v. (µg kg⁻¹ h⁻¹)</td>
<td>0.1</td>
<td>End</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Norepinephrine (µg kg⁻¹ h⁻¹)</td>
<td>0.3</td>
<td>End</td>
<td></td>
<td></td>
</tr>
<tr>
<td>E/E'</td>
<td>13</td>
<td>9</td>
<td>8</td>
<td>8</td>
</tr>
<tr>
<td>Bisoprolol oral (mg)</td>
<td>Bolus+c.i.</td>
<td>Continue</td>
<td>End</td>
<td></td>
</tr>
<tr>
<td></td>
<td>6.25 b.i.d.</td>
<td>Continue</td>
<td>End</td>
<td></td>
</tr>
</tbody>
</table>

F. Guarracino*
G. Landoni
R. Baldassarri
L. Nobile
M. Stefani
Pisa, Italy
*E-mail: f.guarracino@ao-pisa.toscana.it

reduction of HR and prolongation of diastole. On the basis of these findings, a continuous infusion of esmolol was added to the ongoing levosimendan infusion.

With this treatment, from day 0 to day 3 (Table 1), we observed an improvement in EF, the diastolic phase, haemodynamics, and BNP concentration. The patient’s trachea was extubated on day 1 and he was weaned from IABP on day 3. An oral beta-blocker was started to wean the patient from esmolol on day 2.

Administration of beta-adrenergic agents results in increased MVO2 and can worsen myocardial ischaemia. They also impair diastolic relaxation and increase the HR, further exacerbating ischaemia. In contrast, levosimendan does not increase MVO2 nor impair the diastolic phase, and appears safe and effective in left ventricular failure due to AMI.

The concurrent systolic and diastolic impairment precluded the administration of beta-agonists so as to avoid further worsening the diastolic phase and myocardial injury in our patient. The calcium sensitizer-dependent effect of levosimendan allowed us to use this drug in conjunction with a beta-blocking agent: after 2 h of continuous levosimendan, we started esmolol in order to improve the diastolic function. Its favourable pharmacokinetics allowed us to target HR gently and precisely. As a result, the diastolic time prolonged, both LV relaxation and compliance improved, and coronary flow augmentation by IABP increased. A key element in this case was the prompt and repeated use of echocardiography. It allowed us to make the correct diagnosis, target the ventricular abnormalities, and check the effectiveness of therapy.

In this case of cardiogenic shock complicating AMI, echo-guided targeting of systolic and diastolic dysfunction by combined continuous administration of levosimendan and esmolol led to improved cardiac output, increased coronary flow, and shock recovery.

F. Guarracino*
G. Landoni
R. Baldassarri
L. Nobile
M. Stefani
Pisa, Italy
*E-mail: f.guarracino@ao-pisa.toscana.it

1 Task Force for Diagnosis and Treatment of Acute and Chronic Heart Failure 2008 of European Society of Cardiology. ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure 2008. Eur Heart J 2008; 19: 2388–442

doi:10.1093/bja/aqe013

Table 1 Clinical course from day 0 to day 4

<table>
<thead>
<tr>
<th></th>
<th>Day 0</th>
<th>Day 1</th>
<th>Day 2</th>
<th>Day 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR (beats min⁻¹)</td>
<td>140</td>
<td>63</td>
<td>65</td>
<td>60</td>
</tr>
<tr>
<td>AP (mm Hg)</td>
<td>70/40</td>
<td>110/68</td>
<td>120/65</td>
<td>125/60</td>
</tr>
<tr>
<td>CVP (mm Hg)</td>
<td>16</td>
<td>11</td>
<td>12</td>
<td>10</td>
</tr>
<tr>
<td>CI (litre min⁻¹ m⁻²)</td>
<td>1.8</td>
<td>2.4</td>
<td>2.7</td>
<td>2.8</td>
</tr>
<tr>
<td>BNP (pg ml⁻¹)</td>
<td>1240</td>
<td>750</td>
<td>480</td>
<td>320</td>
</tr>
<tr>
<td>EF (%)</td>
<td>&lt;20</td>
<td>36</td>
<td>40</td>
<td>42</td>
</tr>
<tr>
<td>Levosimendan i.v. (µg kg⁻¹ h⁻¹)</td>
<td>0.1</td>
<td>End</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Norepinephrine (µg kg⁻¹ h⁻¹)</td>
<td>0.3</td>
<td>End</td>
<td></td>
<td></td>
</tr>
<tr>
<td>E/E'</td>
<td>13</td>
<td>9</td>
<td>8</td>
<td>8</td>
</tr>
<tr>
<td>Bisoprolol oral (mg)</td>
<td>Bolus+c.i.</td>
<td>Continue</td>
<td>End</td>
<td></td>
</tr>
<tr>
<td></td>
<td>6.25 b.i.d.</td>
<td>Continue</td>
<td>End</td>
<td></td>
</tr>
</tbody>
</table>