Correspondence

Use of recombinant factor VIIa for major haemorrhage

EDITOR:
Haemorrhage associated with trauma and surgery remains a significant cause of death. The development of coagulopathic bleeding is often difficult to manage. Recombinant-activated factor VII (rFVIIa, NovoSeven®; Novo Nordisk, Bagsvaerd, Denmark), which is structurally similar to the naturally occurring activated coagulation factor VII, is licensed for use in patients with haemophilia A or B and inhibitors to coagulation factors VIII or IX and acquired haemophilia. There is considerable interest in the ‘off label’ adjunctive use of rFVIIa in major haemorrhage. A single randomized study has indicated that rFVIIa reduces blood transfusion following blunt trauma [1]. Other evidence for use of rFVIIa as rescue therapy in major haemorrhage remains largely anecdotal. We present our experience from a UK teaching hospital of the use of rFVIIa as rescue therapy in four cases of major haemorrhage, following surgery and blunt trauma. The response to rFVIIa was not consistent and we discuss possible factors limiting its effectiveness.

Case 1: A 39-yr-old male with a variant of Marfan’s syndrome had been anti-coagulated with warfarin following aortic valve and root replacement. He had also undergone repair of infra-renal and thoraco—abdominal aneurysms and developed a rupture of an aortic patch graft aneurysm. He underwent emergency aneurysm repair, requiring a massive blood transfusion, and shortly afterwards returned to theatre for a laparotomy for abdominal compartment syndrome. rFVIIa 90 µg kg⁻¹ was administered for continued bleeding unresponsive to conventional therapy in the intensive care unit (ICU, see Table 1). There was no immediate response, and considerable volumes of fresh frozen plasma (FFP), cryoprecipitate and platelets and three further doses of rFVIIa were administered over the next 11 h (see Table 1). Bleeding ceased approximately 2 h after his final dose of rFVIIa. Anti-coagulation was recommenced on the fourth ICU day, by which time the prothrombin time (PT) and activated partial thromboplastin time (APTT) had normalized. He was discharged home 3 months after admission.

Case 2: A 53-yr-old male sustained multiple injuries as a result of a 10 m fall onto concrete, including a compound occipital fracture, temporal contusion, subarachnoid and subdural haemorrhage, multiple rib fractures and pulmonary contusions, and a femoral fracture. He received cardiopulmonary resuscitation during 15 min of pulseless electrical activity, which responded to decompression of a tension pneumothorax. He bled considerably from his head wound and chest drain despite FFP and platelet transfusion (see Table 1), and 120 µg kg⁻¹ of rFVIIa was administered prior to laparotomy. Two litres of blood was found in the abdomen and splenectomy was performed. Generalized oozing continued but a bleeding point was not identified. Correction of coagulopathy was advised prior to consideration of cardiothoracic intervention, and a further 120 µg kg⁻¹ rFVIIa was administered following additional transfusion therapy. Bleeding rapidly declined and he stabilized. Unfortunately, the head injuries subsequently found on CT scanning were not amenable to neurosurgical intervention. He developed evidence of brain stem death, and organ harvest was performed approximately 24 h later.

Case 3: A 53-yr-old motorcyclist was involved in a collision with a van, sustaining major pelvic and soft tissue injuries, including disruption of the rectum. Over the first 10 days of treatment, he underwent two laparotomies and application of a pelvic external fixator. He had developed septic shock and acute renal failure by the time of his third visit to theatre for a re-exploration and repacking of his wound. He bled considerably intra- and postoperatively, despite transfusion therapy (see Table 1). A dose of 90 µg kg⁻¹ rFVIIa was administered, but with no immediate response. After further FFP, cryoprecipitate and platelets, a dose of 120 µg kg⁻¹ rFVIIa was given. The bleeding ceased abruptly. He remained in ICU for 51 days, and was discharged home 6 months after his original injuries.

Case 4: A 63-yr-old female underwent elective repair of a supra-renal inflammatory aortic aneurysm,
Correspondence


with re-implantation of the left renal artery. Her operation was difficult and splenectomy also required. She was still bleeding when she returned to ICU, despite transfusion therapy (see Table 1). A dose of 120 µg kg⁻¹ rFVIIa was administered, with some immediate improvement. She improved further with FFP, cryoprecipitate and platelet transfusion. Twelve hours later, laparotomy for abdominal compartment syndrome was required. No bleeding point was isolated, and generalized oozing persisted despite FFP and platelet administration. A dose of 120 µg kg⁻¹ rFVIIa was again administered, and her bleeding stopped. She was discharged from ICU 10 days after her admission.

In these cases, the use of rFVIIa was intended to assist haemostasis when surgical and conventional transfusion therapy had failed. Common to the cases was the consumption and dilution of coagulation factors and platelets, acidosis and, in three of the cases, hypothermia, predisposing towards coagulopathic bleeding. The brain injury of the second case and sepsis and renal failure of the third case may also have contributed to coagulopathy.

rFVIIa is believed to act specifically at sites of injury by forming a complex with exposed vessel wall tissue factor. This complex stimulates factor X-activation and thus thrombin generation on the surface of activated platelets [2]. High-dose administration may compensate for coagulation factor and platelet deficiencies in coagulopathic bleeding. Furthermore, a large ‘thrombin burst’ may enable a more stabilized fibrin plug to develop [2]. rFVIIa reduced blood transfusion after blunt trauma in a recent randomized trial [1]. In retrospective analyses, rFVIIa also reduced blood product requirement when used as rescue therapy for patients with intractable bleeding [3,4], but it did not affect mortality [4].

We used rFVIIa in a variety of clinical situations, with variable timing, dosing, prior blood product administration and variable outcome. We failed to see a consistent immediate response to its administration. The contribution of four doses of 90 µg kg⁻¹ rFVIIa to haemostasis in our first case was not clear. Decline in bleeding was most dramatic in the other cases following a second, 120 µg kg⁻¹ dose. For rFVIIa to be effective, adequate viable platelets and coagulation factors should be circulating. It is possible that, particularly in our first and fourth cases, there had been inadequate platelet replacement and, probably in the third case, insufficient coagulation factor replacement prior to administration of the first dose.

Table 1. Transfusion pre- and post-rFVIIa, coagulation parameters, dosing and response in four cases.

<table>
<thead>
<tr>
<th>Age (yr)</th>
<th>Patient 1</th>
<th>Patient 2</th>
<th>Patient 3</th>
<th>Patient 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>39</td>
<td>53</td>
<td>53</td>
<td>63</td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td>Male</td>
<td>Male</td>
<td>Male</td>
<td>Female</td>
</tr>
<tr>
<td>Pathology</td>
<td>Ruptured patch graft aneurysm</td>
<td>Blunt trauma – head, chest, abdomen</td>
<td>Blunt trauma – abdomen, pelvis</td>
<td>Supra-renal aortic aneurysm repair</td>
</tr>
<tr>
<td>Transfusion prior to first dose rFVIIa</td>
<td>PRBC³</td>
<td>FFP³</td>
<td>Cryo³</td>
<td>Platelets³</td>
</tr>
<tr>
<td>29</td>
<td>12</td>
<td>11</td>
<td>14</td>
<td></td>
</tr>
<tr>
<td>18</td>
<td>6</td>
<td>2</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Coagulation parameters at time of first dose rFVIIa</td>
<td>PT (s)</td>
<td>APTT (s)</td>
<td>Platelets (10⁹ L⁻¹)</td>
<td>Fibrinogen (g L⁻¹)</td>
</tr>
<tr>
<td>18.6</td>
<td>58</td>
<td>55</td>
<td>1.34</td>
<td></td>
</tr>
<tr>
<td>15.6</td>
<td>49.9</td>
<td>241</td>
<td>1.18</td>
<td></td>
</tr>
<tr>
<td>16.9</td>
<td>41.4</td>
<td>115</td>
<td>6.07</td>
<td></td>
</tr>
<tr>
<td>15.7</td>
<td>40.7</td>
<td>48</td>
<td>1.43</td>
<td></td>
</tr>
<tr>
<td>Temperature and pH at time of first dose rFVIIa</td>
<td>Temperature (°C)</td>
<td>pH</td>
<td>Base excess (mmol L⁻¹)</td>
<td></td>
</tr>
<tr>
<td>34</td>
<td>7.28</td>
<td>−10.3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>31.6</td>
<td>7.00</td>
<td>−17</td>
<td></td>
<td></td>
</tr>
<tr>
<td>39.3</td>
<td>7.27</td>
<td>−7.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>34.7</td>
<td>7.20</td>
<td>−2.7</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Doses of rFVIIa</td>
<td>4 × 90 µg kg⁻¹</td>
<td>120 µg kg⁻¹, 120 µg kg⁻¹</td>
<td>90 µg kg⁻¹, 120 µg kg⁻¹</td>
<td></td>
</tr>
<tr>
<td>Transfusion in 48 h after first dose rFVIIa</td>
<td>PRBC³</td>
<td>FFP³</td>
<td>Cryo³</td>
<td>Platelets³</td>
</tr>
<tr>
<td>40</td>
<td>21</td>
<td>8</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>22</td>
<td>20</td>
<td>6</td>
<td>8</td>
<td></td>
</tr>
<tr>
<td>40</td>
<td>20</td>
<td>10</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>4</td>
<td>2</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>Observed effect of rFVIIa on bleeding</td>
<td>Eventual cessation after four doses</td>
<td>Marked reduction after second dose</td>
<td>Marked reduction after second dose</td>
<td>Slight reduction with first dose, marked reduction after second dose</td>
</tr>
<tr>
<td>Outcome</td>
<td>Survived</td>
<td>Died as a result of head injuries</td>
<td>Survived</td>
<td>Survived</td>
</tr>
</tbody>
</table>

Units of a packed red blood cells, b FFP, c cryoprecipitate, d doses of platelets.
of rFVIIa. Furthermore, all four patients were acidaemic and, with the exception of the third patient, hypothermic at the time of initial rFVIIa administration. The least acidaemic patient (Case 4) was the only patient to show any response to the initial dose of rFVIIa. Hypothermia reduces activity of coagulation factors and platelets, and acidosis also impairs coagulation [5]. In vitro, acidosis may produce a marked reduction in the activity of rFVIIa [5]. Adequate platelet and coagulation factor transfusion, timed to coincide with rFVIIa administration, and with avoidance of hypothermia and correction of acidaemia, may have improved the efficacy of rFVIIa in our case series.

The optimal dose of rFVIIa as rescue therapy is unknown. It is possible that the 90 and 120 µg kg⁻¹ doses we used were inadequate. In haemophilic patients, a repeated dose of 90–110 µg kg⁻¹ every 2 h for 24 h provides effective peroperative haemostasis [2]. However, a much higher initial dose of 200 µg kg⁻¹, followed by two doses of 100 µg kg⁻¹, was effective in reducing blood transfusion requirement in blunt haemorrhagic trauma [1].

Three patients survived major haemorrhage with variable responses to rFVIIa. The other patient succumbed to his brain injury, although haemostasis was achieved. In vitro evidence suggests that rFVIIa does not induce or enhance a hypercoagulable state [6], and considering the severity of this patient’s injuries, it is likely that rFVIIa administration was not an important factor in the evolution of his brain injury.

However, the safety of rFVIIa administration in trauma associated with head injury is unproven, and cerebral sinus thrombosis following rFVIIa administration to a young poly-trauma patient has been attributed to increased tissue factor exposure resulting from minor brain injury [7].

Our case series illustrates an unpredictable response to rFVIIa when used for major haemorrhage control in blunt trauma and surgery. Further randomized-controlled trials are required to establish the effectiveness of rFVIIa beyond its current licence, the optimal timing and dose of administration and, at approximately £4000 per 7.2 mg dose, its cost effectiveness. It is evident, however, that adequate management of platelet and clotting factor deficiencies, acidaemia and hypothermia are required to maximize the response to rFVIIa.

Acknowledgements

We would like to thank Ms A. Strong, Clinical Trials Nurse, and Mr S. Davies, Audit Clerk, for their help in collecting this data.

References

Nausea and vomiting after cataract surgery: Does neostigmine have an emetic effect?

EDITOR:
Postoperative nausea and vomiting (PONV) occur commonly after general anaesthesia for ophthalmic surgery, the incidence following cataract surgery being 14% [1]. Conflicting evidence exists regarding the effect of the anticholinesterase neostigmine on PONV [2], but a recent systematic review suggested that omitting routine neostigmine use did not have an antiemetic effect [3]. The results of another study indicate that PONV occurs more frequently when neostigmine is administered [4]. The consistently rapid offset of the neuromuscular blocking effect of mivacurium may make routine omission of neostigmine appropriate [5] if careful neuromuscular monitoring is carried out. It was hypothesized that the omission of neostigmine would result in the decrease in the incidence of PONV. This would have particularly important implications for ambulatory anaesthesia as PONV is a leading cause of delayed discharge or hospital readmission in this setting.

Following hospital Ethics Committee approval and having given written informed consent, 88 patients, ASA I–III, undergoing elective cataract surgery under general anaesthesia were studied. The following exclusion criteria applied: patients with a history of travel sickness, vertigo or vestibular disease, patients who were pregnant, patients suffering from asthma or hepatic, renal or neuromuscular disease. Also excluded were patients currently taking medications known to cause or prevent nausea and vomiting, and those known to interact with neuromuscular blocking drugs. Patients undergoing trabeculectomy surgery were also excluded.

No premedicants were administered. Anaesthesia was induced intravenously (i.v.) using fentanyl 0.75 µg kg⁻¹ and propofol 2–3 mg kg⁻¹ and was maintained with isoflurane (0.8–1.5% end-tidal concentration) and 66% nitrous oxide in oxygen. A laryngeal mask airway was inserted and intermittent positive pressure ventilation was initiated to keep the end-tidal carbon dioxide at 4–4.5 kPa. Peak inspiratory pressure was limited to less than 20 cmH₂O to minimize gastric insufflation. Electrocardiograph, heartrate, non-invasive blood pressure, end-tidal carbon dioxide and concentration of inspired and expired gases were monitored throughout (Datex AS3, Helsinki, Finland). Compound Ringers lactate solution was administered i.v. to correct the fluid deficit due to preoperative fasting.

Muscle relaxation was induced with mivacurium 0.2 mg kg⁻¹ and was monitored by tactile evaluation of response to train-of-four stimulation of the ulnar nerve. Subsequent increments of mivacurium 0.1 mg kg⁻¹ were administered at the reappearance of the third twitch. Neuromuscular blockade was maintained until the end of surgery. According to random allocation, either neostigmine 50 µg kg⁻¹ and glycopyrrolate 10 µg kg⁻¹ (Group N), or saline (Group S) were administered. Standard clinical tests of neuromuscular function were applied prior to tracheal extubation. These were the absence of fade by tactile evaluation of the train-of-four stimulation of the ulnar nerve, and sustained head lift and hand grip, each for 5 s.

Nausea, vomiting and pain were assessed at 30 min, 1, 2, 4 and 24 h after surgery, by an investigator who was unaware of the study group to which the patient belonged. The use of rescue antiemetic medication (prochlorperazine 12.5 mg intramuscularly) and analgesic requirements were recorded. Nausea was graded as follows:

Grade I: No nausea.
Grade II: Mild nausea – no antiemetic medication administered.
Grade III: Moderate nausea – antiemetic medication administered with effect.
Grade IV: Severe nausea – antiemetic medication administered but ineffective.

Emetic events were defined as a single vomit or retch. Pain was assessed using a verbal rating scale (0–10).

Data obtained were analysed using the z-statistic for dichotomous variables. Based on α = 0.05 and β = 0.2 and a one-tail alternative hypothesis to detect an increase in the incidence of postoperative nausea due to administration of neostigmine following cataract surgery under general anaesthesia from 14% [2] to 40%, a minimum sample size of 33 < n < 38 was required. Two-tailed, unpaired t-tests were used for normally distributed data. Contingency tables were analysed using Fisher’s exact test. P < 0.05 was taken to indicate statistical significance.

Correspondence to: Henry P. Frizelle, Division of Anaesthesia, Mater Misericordiae University Hospital, Eccles Street, Dublin 7, Ireland. E-mail: hfrizelle@mater.ie; Tel: +353 1 8302281; Fax: +353 1 8301820
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Eighty-eight patients were recruited to the study of which 18 were excluded due to unadvocated or clinically mandated breach of protocol. Of the 70 patients on whom complete data sets were obtained, 38 were in Group N and 32 in Group S. The groups were similar with regard to age, gender, ASA physical status and duration of surgery (Table 1). No patient gave a history of susceptibility to PONV, to travel sickness or to vertigo. The duration of anaesthesia was similar for both groups. No difference existed between the groups regarding the doses of propofol or fentanyl administered (Table 1). The incidence of postoperative nausea is summarized in Table 2. The neostigmine group had a greater incidence of nausea at 1 and 2 h postoperatively but at no time was the difference statistically significant (P = 0.12). Nausea experienced was all Grade 2; that is, did not require treatment. Only one patient vomited (Group N).

The two groups were similar in terms of postoperative pain measured using the verbal rating scale (Group N 1.2 ± 0.4 vs. Group S 1.4 ± 0.3). The requirement for analgesia was similar for each group, paracetamol being the commonest agent used; Group N 335 ± 543 mg vs. Group S 188 ± 355 mg.

All patients experiencing nausea had a mild form, which resolved without antiemetic medication. The incidence of PONV in this study was less than that quoted in previous studies [2]. Other researchers reported an 18–35% incidence of nausea in patients who had received neostigmine [6,7] with a 33% incidence of vomiting. In our study, only one patient (Group N) vomited. The greater incidences of PONV reported by other groups may be due to the different procedures performed [2,4]. This is reflected in the postoperative management regimens employing both opioids analgesia and early mobilization.

The dose of neostigmine administered may have influenced the observed incidence of nausea and vomiting. Compared to Hovorka’s work, we selected slightly greater doses of neostigmine, calculating the dose on a weight basis, but intraoperative anaesthetic drugs administered were otherwise similar. Nelskyla administered neostigmine 2.0 mg and glycopyrrolate 0.4 mg to all patients in the treatment group, having no impact on the incidence of nausea and vomiting compared with controls [8]. Lovstad studied 90 women undergoing laparoscopic gynaecological surgery [4]. Neostigmine and glycopyrrolate were administered in doses of 50 µg kg⁻¹ and 10 µg kg⁻¹, respectively. Of the treatment group, 30% suffered from nausea in the first 6 h, compared with 11% in the placebo group. Although the doses used are identical to those used in the current study, the incidence of nausea and vomiting are markedly different, again perhaps reflecting the type of surgery performed.

One of the strengths of our study is that the surgical procedure performed is minimally invasive and as such produces little nausea inherent to the procedure. In addition, these patients have lesser analgesia requirements. The low verbal rating scores confirm this. Therefore, the nature of the surgical procedure, opioid administration and pain are unlikely to have acted as confounding factors in this study. However, this contributed to the problem of such a low incidence of the measured variables, nausea and vomiting.

The dose–response relationship of mivacurium with its short duration of action makes it an ideal muscle relaxant for investigation of the effects of neostigmine [5] allowing the omission of antagonism of neuromuscular blockade at the end of the procedure, provided that careful neuromuscular monitoring is performed. Our results are consistent with those of Joshi and colleagues [6] and Hovorka and colleagues [7] and fail to demonstrate a difference in the incidence of PONV between those patients who received neostigmine and those who did not. Whether the muscle relaxant administered plays a role in the incidence of PONV has yet to be elucidated, but it appears that omitting neostigmine may have a clinically relevant antiemetic effect when greater doses, of the order of 2.5 mg or more, are used [3].

We have demonstrated that neostigmine 50 µg kg⁻¹ does not influence the incidence of nausea and vomiting in patients undergoing elective cataract surgery.

### Table 1. Patient characteristics, duration of surgery and doses of drugs administered.

<table>
<thead>
<tr>
<th></th>
<th>Saline (n = 32)</th>
<th>Neostigmine (n = 38)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>64.4 ± 12.0</td>
<td>61.8 ± 17.0</td>
</tr>
<tr>
<td>Gender (M/F)</td>
<td>16/16</td>
<td>16/22</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>71.3 ± 14.0</td>
<td>73.7 ± 12.0</td>
</tr>
<tr>
<td>Duration of surgery (min)</td>
<td>44.5 ± 7.5</td>
<td>39.6 ± 11.0</td>
</tr>
<tr>
<td>Propofol (mg)</td>
<td>131.1 ± 52.0</td>
<td>125.8 ± 42.0</td>
</tr>
<tr>
<td>Fentanyl (µg)</td>
<td>78.4 ± 20.0</td>
<td>81.9 ± 21.0</td>
</tr>
<tr>
<td>Mivacurium (mg)</td>
<td>16.7 ± 9.9</td>
<td>15.1 ± 4.7</td>
</tr>
<tr>
<td>Acetaminophen (mg)</td>
<td>188 ± 355</td>
<td>335 ± 543</td>
</tr>
</tbody>
</table>

Data are mean ± SD or number of patients.

### Table 2. Episodes of PONV.

<table>
<thead>
<tr>
<th></th>
<th>0.5 h</th>
<th>1 h</th>
<th>2 h</th>
<th>4 h</th>
<th>24 h</th>
<th>Cumulative total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Saline (n = 32)</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>2</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>Neostigmine (n = 38)</td>
<td>1</td>
<td>3</td>
<td>5</td>
<td>2</td>
<td>1</td>
<td>12*</td>
</tr>
</tbody>
</table>

*P = 0.12 compared to saline group.
Unexpected aortic dissection detected by transoesophageal echocardiography in the operating theatre at the beginning of cardiac surgery

EDITOR:
The expanding use of transoesophageal echocardiography (TOE) in adult cardiac surgery has provided a large amount of information not previously available. We report an unusual case of ascending aortic dissection detected in the operating theatre by TOE immediately after anaesthetic induction in a patient scheduled for combined mitral and aortic valve replacement. The patient had undergone diagnostic coronary angiography 1 week earlier.

A 46-yr-old male was admitted to our emergency department for progressive dyspnoea and supraventricular tachycardia (170 bpm). He had a history of rheumatic mitral and aortic valve disease, epilepsy and endogenous depression. The blood pressure (BP) was 170/105. Cardiovascular risk factors included hypercholesterolaemia and active smoking. His regular medication included valproic acid, fluoxetine and acetylsalicylic acid. He was diagnosed with acute pulmonary oedema and transferred to the intensive care unit (ICU) to initiate medical therapy.

A chest X-ray showed cardiomegaly. Transthoracic echocardiography demonstrated moderate mitral regurgitation with severe stenosis and severe aortic stenosis. The left ventricular ejection fraction was 25%. The patient improved under medical treatment and he was referred for combined mitral and aortic valve replacements after 7 days.

Routine preoperative coronary angiography, with 5-F diagnostic catheters, had been performed with great technical difficulty. Selective catheterization of the right coronary ostium failed because of unfavourable anatomy. The left coronary angiogram demonstrated a dominant left coronary artery system without significant disease. The distal right coronary artery (RCx) was perfused via collaterals from the left coronary artery. A left ventriculogram showed a dilated ventricle with an ejection fraction of 25–30% and confirmed the echocardiographic valvular findings.

The patient was premedicated with diazepam, scopolamine and morphine. He was anaesthetized with a combined high-dose opioid/volatile anaesthetic technique: fentanyl (30 µg kg$^{-1}$), etomidate (0.2 mg kg$^{-1}$) and sevoflurane (0.8–1.5 minimal alveolar concentration (MAC)). Rocuronium was used for muscle relaxation. Monitoring consisted of radial arterial pressure, central venous and pulmonary arterial

It remains to be elucidated if this is true following surgery associated with a greater incidence of PONV.

References


H. P. Frizelle, E. Curran, C. Twomey, J. MacAdoo, G. Shorten
Department of Anaesthesia and Intensive Care Medicine
Cork University Hospital
Wilton, Cork, Ireland
pressures. A multiplane TOE probe was inserted (Acuson Computed Sonography™ 128 XP/10 C; Soma Technology Inc., Cheshire, Connecticut, USA). The pre-cardiopulmonary bypass (pre-CPB) examination revealed a double-lumen image with an intimal flap in the ascending aorta. The entry port was located near the right coronary sinus. The aortic arch was preserved. There was no pericardial effusion.

The surgical approach was modified. In addition to mitral and aortic replacements, the dissected ascending aorta was replaced with a Dacron graft. CPB was established by cannulation of the femoral artery, and the superior and inferior caval veins. The patient was cooled to 20°C. The dissection involved 75% of the aortic circumference, with a total length of 4 cm. The intimal tear was 2 cm above the right coronary ostium. Neither inflammatory nor cystic degeneration was found at pathological examination. CPB lasted 125 min, with 13 min of circulatory arrest. Aortic cross-clamping was 96 min. The patient remained haemodynamically stable during the procedure. The patient was discharged from the ICU on postoperative day 4 and from hospital 20 days after surgery without neurological sequelae.

According to population-based studies, including prehospital mortality, the incidence of spontaneous aortic dissections is \( \geq 3/100,000 \text{ yr}^{-1} \) [4]. Aortic dissections can also be a consequence of cardiac catheterization, cardiac surgery, or other invasive vascular procedures [1]. Many cases of intraoperative iatrogenic aortic dissections have been published in recent years [2,3], all of them as a direct traumatic consequence of cardiac surgery. Anaesthesiologists using intraoperative TOE can be involved in the early recognition of iatrogenic aortic dissections or spontaneous ones not detected preoperatively in patients undergoing cardiac surgery.

Catheter-induced ascending aortic dissection is a rare but probably underestimated complication of cardiac catheterization. Several mechanisms may contribute to aortic dissections during interventional procedures, such as retrograde dissection of the coronary artery following contrast injection or balloon angioplasty, catheter-induced aortic wall lacerations during manipulation and/or forceful contrast injection through an end-hole catheter can be responsible [5]. Patients predisposed to aortic injury are those with severe atherosclerosis, thin or dilated ascending aorta, cystic medial necrosis, collagen vascular disease, history of aortic coarctation or long-standing hypertension [2,6].

The clinical diagnosis of iatrogenic aortic dissection is difficult due to its usually atypical presentation. Chest or back pain is less prevalent in patients with iatrogenic than in those with spontaneous dissections. However, the former are much more likely to develop cardiac complications such as myocardial ischaemia or infarction [1]. Imaging studies are less likely to demonstrate pathological findings, such as widening of the mediastinum or an abnormal aortic contour on chest X-ray in cases of iatrogenic aortic dissection [1]. The sensitivity of transthoracic echocardiography has been reported to be as low as 59%, but it is close to 100% with TOE [7].

In cardiac surgery, unexpected aortic dissection is associated with an increased mortality, between 20% and 50% depending on the time of diagnosis [6]. Inappropriate perfusion of the false lumen during CPB may have catastrophic consequences including ischaemia of the brain, spinal cord and kidneys, and/or rupture of the aortic wall to the pleura or peritoneum.

In summary, we have presented a case of unexpected ascending aortic dissection, probably caused by a recent routine coronary angiogram, in a cardiac surgical patient. The routine use of intraoperative TOE proved invaluable in this case.

J. A. Varela, F. J. Hortal, M. Zaballos
Department of Anaesthesiology and Intensive Care
Gregorio Marañon General Hospital
Madrid, Spain
M. J. Riego
Department of Anaesthesiology and Intensive Care
Gregorio Marañon General Hospital
Professor of Anaesthesiology
Compleutense University of Madrid
Madrid, Spain

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Mediastinal haemorrhage mimicking tamponade during en-bloc oesophagectomy

EDITOR:
Concealed mediastinal haemorrhage is rare during oesophagectomy procedures. It may occur in patients with major chest trauma following aortic injury. Occasionally patients undergoing aortic arch surgery may present with postoperative mediastinal bleeding. We report a case in which concealed mediastinal haemorrhage complicated an en-bloc oesophagectomy procedure in the early stages of the operation. The diagnosis was not clear because the bleeding was not obvious, as it involved the posterior mediastinum. The mediastinal haemorrhage mimicked cardiac tamponade and was unresponsive to fluid replacement.

A 60-yr-old male, ASA III, with oesophageal cancer, was scheduled to undergo en-bloc oesophagectomy and colon interposition. Past medical history included hypertension, chronic obstructive pulmonary disease, reflux disease and smoking. The surgical plan included initial exploration of the right chest for mobilization of the oesophagus, followed by abdominal and left-sided neck explorations for completion of en-bloc oesophagectomy. The transverse colon would then be mobilized and interposed in place of the oesophagus. Preoperative preparation included blood gas analysis and pulmonary function tests, which were within normal limits. A dobutamine stress echo was negative for cardiac disease.

Following induction of anaesthesia, a 39-G double lumen tube was placed uneventfully. Proper placement was confirmed with auscultation and fibreoptic bronchoscopy. Anaesthesia was maintained with isoflurane and intermittent boluses of fentanyl. Inhalation induction was confirmed with auscultation and fibreoptic bronchoscopy. Anaesthesia was maintained with isoflurane and intermittent boluses of fentanyl. Inhalation induction was confirmed with auscultation and fibreoptic bronchoscopy. Anaesthesia was maintained with isoflurane and intermittent boluses of fentanyl. Inhalation induction was confirmed with auscultation and fibreoptic bronchoscopy. Anaesthesia was maintained with isoflurane and intermittent boluses of fentanyl. Inhalation induction was confirmed with auscultation and fibreoptic bronchoscopy. Anaesthesia was maintained with isoflurane and intermittent boluses of fentanyl. Inhalation induction was confirmed with auscultation and fibreoptic bronchoscopy. 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no abnormality with the upper lobe bronchus. A repeat haemoglobin revealed a drop from 12 to 9.5. During this time, the surgeons were exploring the left side of the neck for completion of the oesophagectomy. During the exploration of the neck, it was found that the mediastinum was distended and contained a large haematoma which was causing compression of the mediastinal structures and the upper lobe of the left lung. Approximately 2 L of blood and blood clot were evacuated. Haemodynamics and oxygenation improved shortly after the decompression allowing the dopamine to be discontinued. The site of bleeding was identified as one of the small upper branches of the aorta supplying the oesophagus. The remaining anastomoses were completed.

Discussion

During surgical procedures, minimal mediastinal bleeding usually does not present with haemodynamic or pulmonary problems. Excessive bleeding may present with haemodynamic compromise. Severe hypotension can be the result of compression of the left atrium, pulmonary veins and superior mediastinal structures, such as the superior vena cava (SVC). The hypoxaemia can possibly be explained by the compression of the left lung by the expanding mediastinum from expanding haemorrhage. There was a reduction in venous return due to compression of the pulmonary veins and left atrium. Reduction in pulmonary perfusion may have compounded the hypoxaemia.

Mediastinal haemorrhage may not present with the typical signs of cardiac tamponade. In cardiac tamponade, there is direct compression of the atrium and ventricle from the bleed. In mediastinal haemorrhage, there is compression of the SVC, left atrium, pulmonary vein and left ventricle. Transoesophageal echocardiography can be helpful in the diagnosis but was not feasible in this instance. In the above patient, the mediastinal haematoma exceeded 2 L, which was significant enough to cause mechanical effects on the vessels and result in the above symptoms.

Oesophagectomy is becoming a standard procedure for cancer of the oesophagus, achalasia and multiple oesophageal strictures. Transhiatal oesophagectomies are more commonly performed than en-bloc oesophagectomies. In transhiatal oesophagectomies, the mediastinal dissection is performed blindly. During dissection the superior mediastinum may not be visualized completely and it is possible to miss a transected arterial branch which supplies the oesophagus.

During the last 10 yr, we have performed approximately 1500 oesophagectomies and have seen two cases of mediastinal haemorrhage which became significant in the postoperative period. In addition to haemodynamic instability, these patients have presented with increased output from the posterior mediastinal drains. This case was unusual in that it occurred during the early part of the dissection and was not readily diagnosed.

D. Thangathurai, P. Roffey, M. Mogos
M. Riad, A. Boborquez
Department of Anesthesiology
University of Southern California
Los Angeles, CA, USA

Airway management in a patient with a vascular injury and rapidly expanding neck haematoma

EDITOR:
Management of the difficult airway is one of the most challenging problems for the anaesthetist in daily practice [1]. This is even more important when a rapidly expanding neck haematoma dictates immediate airway control. In a peripheral hospital setting, lack of a management protocol, imaging modalities which are not readily available, having no qualified vascular or otorhinolaryngological surgeon on site and previous experience of staff with the above condition can further complicate the situation. In such a clinical scenario, familiarity with difficult airway algorithm, specialized airway equipment, forethought and decisiveness of the anaesthetist can avert crisis and lead to a successful outcome. I would like to report a case of penetrating neck injury, torn common carotid artery and rapidly expanding neck haematoma, which was managed initially in a small peripheral hospital in Ireland.

Correspondence to: Strahil Kotsev, Department of Anaesthesia, Beaumont Hospital, 81 Kilmore Avenue, Artane, Dublin 5, Ireland. E-mail: strahilkotsev@hotmail.com; Tel: +353 851439229; Fax: +353 18479291
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A 49-yr-old male was brought to the Emergency Department in a peripheral hospital after an industrial accident. A metal object rebounded after a punch and impacted into the lateral side of his neck. The paramedics reported external bleeding initially from the neck wound, which was stopped after applying manual pressure. Later on, a neck haematoma developed. The medical history of the patient was unremarkable, apart from recent ingestion of food. There was no loss of consciousness during or after the accident. On admission physical examination revealed a conscious patient, Glasgow Coma Score 15, able to move all the extremities, pupils equal and reactive to light. There was no dysphonia, dysphagia, paraesthesia, weakness, paralysis, dysphonia or hoarseness. Respiratory rate was 30 breaths min\(^{-1}\), chest auscultation demonstrated good air entry bilaterally, peripheral oxygen saturation was 100% breathing 40% oxygen by facemask. The blood pressure (BP) was 205/115 mmHg, heart rate 120 beats min\(^{-1}\), peripheral pulses present, capillary refill was normal and the electrocardiogram showed a sinus tachycardia.

Inspection of the neck revealed a large haematoma mainly on the right side but involving the anterior part as well. There was a small wound at the entry side of the metal projectile which was oozing blood. A bruit and a thrill were present over the right side of the neck. Full blood count, electrolytes and arterial blood gases were normal. Blood was obtained urgently for cross match and transfusion cervical spine and chest X-rays were done performed which revealed no signs of cervical spine, pharyngeal, oesophageal or tracheal injury, but marked deviation of the trachea.

The wound was initially cleaned and dressed. Tetanus prophylaxis and antibiotics were given. Three 14-G intravenous cannulae, two in the contralateral arm and one in the foot were inserted and the dorsalis pedis artery cannulated by the anaesthetist. BP was controlled by small increments of morphine and esmolol. In view of the rapidly expanding neck haematoma and imminent airway compromise intubation of the trachea was undertaken. Considering the patient’s condition, severely distorted airway anatomy, recent ingestion of food, the available equipment and the experience of the anaesthetic team, awake fibreoptic intubation was selected. The patient was transferred to the operating theatre. Pledgets, soaked in lidocaine and epinephrine were inserted to block the anterior ethmoidal nerve and the sphenopalatine ganglion and nerves. Topical anaesthesia of the larynx and trachea was achieved with local anaesthetic and epinephrine injected through the suction port of the bronchoscope. Midazolam 2 mg and esmolol 20 mg increments were given intermittently to provide sedation and to blunt the haemodynamic response. Successful endotracheal intubation was achieved at the first attempt using a 6.5 mm reinforced tube and the correct position verified using a capnograph. A propofol targeted-controlled infusion, neuromuscular blocking agents and opioids were administered and the patient was transferred to a tertiary centre. Computerized tomography (CT) scan revealed a tear in the common carotid artery which was repaired by a vascular surgeon.

Managing a patient with penetrating neck injury and rapidly expanding neck haematoma, can prove challenging even for an experienced anaesthetist. Lack of hospital protocols, imaging modalities and specialist surgical expertise can make the situation even worse.

Intubation attempts should be restricted to when prolonged transport time is anticipated, the patient appears moribund, haemodynamically unstable or respiratory arrest is imminent. In the above case, the rapidly expanding neck haematoma alerted the anaesthetists to the potential of a carotid injury and precipitous loss of the airway. Although a well-established technique [2], and a quick and safe procedure in experienced hands, percutaneous tracheostomy, retrograde intubation or surgical cricothyrotomy were not considered as viable options because of the grossly distorted airway anatomy.

We considered rapid sequence induction in this case as unwise because airway assessment had indicated likely difficulties in obtaining airway control. Awake blind intubation was not selected, because of the inability to provide adequate topical analgesia, especially below the vocal cords and the real risk of provoking clot dislodgement and precipitating massive bleeding. Awake fiberoptic intubation was chosen, based on the availability of appropriate equipment and expertise of one of the members of the anaesthetic team. We planned to restrict the attempts to one only, avoiding injudicious multiple trials. As a second option we planned to proceed with an inhalational induction, being aware of the fact that the risk of aspiration does not necessarily preclude its use [3].

The involvement of anaesthetists from peripheral hospitals in difficult airway and trauma management training programmes on a regular basis, gives them the confidence to take a decision and the ability to execute it should it become necessary [4]. Training in fiberoptic intubation should be part of every trainee’s programme ensuring that the technique is no longer the domain of a few interested enthusiasts [5].

The case demonstrates the validity of the concept that no single treatment modality is universally applicable in the management of the difficult airway and the anaesthetist should be capable of instituting a variety of techniques [6]. It emphasizes the need for clear policies as to which hospitals should receive...
Anaesthetic management of a patient with epidermolysis bullosa undergoing percutaneous nephrolithotomy

EDITOR:
Epidermolysis bullosa (EB) is a rare family of inherited blistering skin and mucous disorders characterized by blister formation in response to mechanical trauma or frictional force [1,2]. Prevalence is estimated to 1:300,000 [3]. EB patients’ anaesthetic management necessitates great care to avoid skin trauma and further bulla formation [4]. We report a case of a successful combined spinal–epidural anaesthesia in a patient with recessive dystrophic EB (type Hallopeau-Siemens) who underwent percutaneous nephrolithotomy in the prone position. Combined spinal–epidural anaesthesia obviated the need for difficult airway management [5].

A 48-yr-old female with a history of Caesarean section under regional anaesthesia and correction of an oesophageal stricture (under general anaesthesia and postoperative pain due to airway complications) due to recessive dystrophic EB (type Hallopeau-Siemens) was admitted in our unit. She was scheduled to undergo percutaneous nephrostolithotomy because of renal stones. She was malnourished (body mass index = 16.9 kg m$^{-2}$, albumen 34 g L$^{-1}$, haemoglobin 10.5 g dL$^{-1}$). Mouth opening was limited (2.5 cm) because of oropharyngeal scars. Mallampatti score was III. Examination revealed multiple lesions in the oropharynx (Fig. 1) and oesophagus and lesions at the dorsal part of the hand and inguinal region. The main risk was the aggravation of these lesions such that all types of trauma to skin or mucous membranes needed to be avoided. After discussing the case between the surgeons and the anaesthesiologists, the technique decided upon was combined spinal–epidural anaesthesia.

The patient was not premedicated. All contact and friction points were protected with fat pads. All sticking plasters were avoided. Monitoring was

Reference


Figure 1.
Blister (B) on the tongue, moderate poor dentition and limited mouth opening (around 2.5 cm).
secured by an oximeter attached to the ear, an automatic non-invasive blood pressure (BP) monitor well padded such that it did not touch the skin and electrodes just fixed to the skin using their jelly alone. The patient was positioned in the left lateral decubitus position. Aseptic preparation was obtained using a soft swab immersed in chlorohexidine. The block was inserted at the L2–L3 level without infiltrating the skin. Bupivacaine 0.5% 7.5 mg with 50 µg of clonidine were slowly injected intrathecally. The epidural catheter was fixed using a very small plaster. After verifying the level of anaesthesia to higher than T6 using simple tactile stimulation with a small cotton wool ball and by observing the haemodynamic stability, the patient was turned into the prone position with padding at all points of support. The duration of the procedure was 2 h. The patient was haemodynamically stable. At the end of the intervention, the patient complained of pain at the level of T8–T10. Lidocaine 2% 6 mL (without epinephrine) was injected into the epidural catheter. This was sufficient to obtain a satisfactory level of anaesthesia until the end of the surgery. The catheter remained in situ for 24 h after the operation and a continuous infusion (7 mL h\(^{-1}\)) of 2 mg mL\(^{-1}\) ropivacaine was infused into the catheter. The postoperative period passed without complications and without any mucous or cutaneous lesions appearing. Feeding was restarted on the same day. The patient left the hospital on the third day. Contacting the patient after 1 week confirmed the absence of any complication. She was satisfied with the management.

Anaesthetic management of patients with EB needs to take care to prevent any trauma to skin or mucous membrane [1]. A team-based approach is important involving the anaesthesiologist, the dermatologist and the surgeon. An extensive preoperative evaluation of any lesion must be undertaken to exclude newly appearing traumatic lesions [5]. Management of these patients in the operating room depends on the following principles: avoidance of all adhesive material, padding all pressure points, monitoring using minimally invasive interventions, protecting the arms on which the BP cuff is used by cotton bands, fixing the intravenous lines with bandages instead of plasters, ocular protection with eye-wash liquid in case of general anaesthesia to prevent any corneal abrasion. The absence of a sedative premedication permits the patient to adjust their position on the operating table themselves and thus avoid any rubbing, a source of cutaneous lesion. The formation of a bulla is usually more directly related to a rubbing effect than just simple contact. In this case report, the patient did not develop any major cutaneous lesions or associated severe pathology [6]. Our precautions appear therefore to have been adequate [4,7–9].

During anaesthetic management, oropharyngeal and oesophageal lesions may result from intubation which is described as being more difficult in EB [7]. Twenty-five per cent of difficult intubations are attributed to difficult mouth opening, dentition and bleeding tendency from the oropharyngeal mucosa [8,10–12]. Regional anaesthesia has distinct advantages. The upper airway can be avoided and the awake patient can position herself avoiding potential traumas during manipulations [13,14]. Infiltration anaesthesia is contraindicated. Spinal anaesthesia may be utilized for surgery to the lower extremities [15]. The site of puncture must be located on an area without a bulla. In our case report, because of the oropharyngeal problems, the need for a prone position, the unknown duration of surgery and the level of sensory block required combined spinal–epidural anaesthesia was chosen. Opioids were avoided because of the potential for pruritus and so 0.5% bupivacaine with clonidine was used. After diminution of the sensory level, a satisfactory return to an adequate sensory level was quickly provided with 2% lidocaine without epinephrine injection through the epidural catheter, allowing the remainder of the surgical procedure to proceed without intervention on the airway.

In summary, we report successful anaesthetic management in a patient with EB complicated by oropharyngeal involvement using combined spinal–epidural anaesthesia. Management of patients with EB necessitates great care in anaesthesia to prevent any aggravation of mucocutaneous lesions. Generally, as in our report, regional anaesthesia can resolve many problems associated with general anaesthesia concerning intubation, position and postoperative analgesia.

L. Nguyen, V. Minville, B. Riu
F. Atallah, O. Fourcade
Department of Anesthesia and Intensive Care
University Hospital of Toulouse
University of Paul Sabatier
Toulouse, France

References
2.9 mmol L\(^{-1}\) an osmolality of 316 mosm kg\(^{-1}\), heart rate (HR) 110 beats min\(^{-1}\) and hyperventilation. She became unresponsive in the room she had visual loss, deteriorating mental state and was admitted to our hospital. In the emergency centre and had been discharged home. The following day her symptoms worsened, and she was admitted to the emergency department with symptoms of fatigue, pain and lethargy to another hospital. We present a 52-year-old woman who had serious

**EDITOR:**

We present a 52-year-old woman who had serious symptoms of methanol intoxication. She was initially admitted to the emergency department with symptoms of fatigue, pain and lethargy to another centre. She had normal cranial tomography (CT) findings in that centre and had been discharged home. The following day her symptoms worsened, and she was admitted to our hospital. In the emergency room she had visual loss, deteriorating mental state and hyperventilation. She became unresponsive with a Glasgow Coma Scale score of 3. Other observations were as follows: blood pressure (BP) 90/60 mmHg, heart rate (HR) 110 beats min\(^{-1}\) and body temperature 38°C. The arterial blood gases were: pH 6.94, \(PCO_2\) 13.3 mmHg, \(PO_2\) 189.9 mmHg (breathing oxygen via a face mask at 6 L min\(^{-1}\)) and \(HCO_3\) of 2.9 mmol L\(^{-1}\). The anion gap was 32 mmol L\(^{-1}\) with an osmolality of 316 mosm kg\(^{-1}\). She was intubated orally in the emergency room and admitted to the intensive care unit (ICU). It was subsequently learned from her relatives that the patient had suffered with chronic shoulder, back, knee and elbow pain and she had used cologne and spirit to massage her legs and arms. In the last 3 days she had more frequent massage because of aggravated pain. There was no known history of drinking such substances. The patient was mechanically ventilated in the ICU. After correction of acidosis with intravenous (i.v.) \(NaHCO_3\) (150 mmol \(NaHCO_3\) bolus, 150 mmol \(NaHCO_3\) infusion for 24 h) her blood gases were: pH 7.37, \(PCO_2\) 34.6 mmHg, \(PO_2\) 61 mmHg (FiO\(_2\) 0.3), \(HCO_3\) 20.4 mmol L\(^{-1}\). Mechanical ventilation was terminated after 4 h in the ICU, and the patient was extubated after 8 h. Her vital signs were: BP 110/70 mmHg, HR 110 beats min\(^{-1}\) and body temperature 37°C. The patient’s neurological status quickly improved. The blood chemistry was: Na 147 mmol L\(^{-1}\), K 3.2 mmol L\(^{-1}\), Cl 112 mmol L\(^{-1}\), urea 54 mg dL\(^{-1}\), creatinine 1.9 mg dL\(^{-1}\), glucose 214 mg dL\(^{-1}\). Her APACHE II score at 24 h was 23. Therapy included \(NaHCO_3\) infusion for correction of metabolic acidosis, haemodialysis (two periods, each lasted 4 h) for elimination of toxins, ethyl alcohol (1 g kg\(^{-1}\) bolus and 0.1 g kg\(^{-1}\) h\(^{-1}\) infusion via a nasogastric tube) and folate (1 mg kg\(^{-1}\), six times per day). After 3 days she was discharged from the ICU to a ward then to home on the 5th day. By the time of discharge, she had 3/5 right monoparesis, left hemihypeaesthesia and bilateral optical disc atrophy. CT findings were frontal subcortical necrosis and bilateral lentiform nucleus hypodensity.

Methanol intoxication usually occurs when taken orally but inhalational and dermal exposure to methanol can cause clinical intoxication. Although methanol is not toxic itself, more than 95% is metabolized to formaldehyde by alcohol dehydrogenase and then to formic acid by aldehyde dehydrogenase. The remainder, which is not metabolized, is excreted via the kidneys and lungs. Acidosis is mainly caused by formic acid. Methanol has poor affinity for alcohol dehydrogenase which can delay clinical toxicity by up to 24 h after ingestion. Ocular damage and
methanol intoxication is reported to be rare and amount of methanol administration. Dermal skin to hospital, she had increased the frequency spirit and cologne topically. In the days before admission but her history included extensive use of methanol orally. Our patient has no history of oral all of the patients who need ICU treatment have taken osmolality and the patients clinical condition. Almost cannot be measured so the diagnosis was based on the clinical history, anion gap, metabolic acidosis, high osmolality and the patients clinical condition. Almost all of the patients who need ICU treatment have taken methanol orally. Our patient has no history of oral ingestion but her history included extensive use of spirit and cologne topically. In the days before admission to hospital, she had increased the frequency and amount of methanol administration. Dermal methanol intoxication is reported to be rare [4]. Our patient suffered more serious intoxication than has been previously reported. Cutaneous absorption of methanol may increase blood levels and methanol can enter the systemic circulation following 4 h of exposure [5]. Methanol intoxication may cause liver damage. It has been demonstrated experimentally that N-acetyl cysteine may prevent liver damage in such conditions [6]. We prescribe N-acetyl cysteine for our methanol intoxicated patients in the ICU (300 mg three times per day i.v.). The patient had normal liver function tests during her follow up in hospital.

Permanent visual loss may be seen in methanol intoxicated patients. Optic atrophy and blindness may occur according to the severity of intoxication. This patient also had some neurological deficit. CT and magnetic resonance imaging findings may include bilateral haemorrhagic necrosis in the putamen and caudate nuclei, diffuse subcortical white and grey matter necrosis, cerebellar and optic nerve necrosis, cerebral and intraventricular haemorrhage and diffuse cerebral oedema [7]. Our patient had developed a frontal subcortical infarct and bilateral lentiform nuclei hypodensity.

It is important for the clinician to consider unusual routes of administration when investigating poisonings.

T. Adanir, M. Y. Ozkalkanli, M. Aksun
Izmir Ataturk Training and Research Hospital
Anesthesiology and Reanimation Clinic
Intensive Care Department
Izmir, Turkey

References


An anaesthesia face mask with no lumen

EDITOR:

Complications related to anaesthetic devices include design, manufacture, use, maintenance and service mistakes. The incidence of these complications is 10–14% at units that have regular maintenance and quality control. Complications related to anaesthesia breathing circuit devices are seen in 12–18% and serious complications occur in 4.3% [1]. Disposable devices are becoming more commonly used in anaesthesia practice. Serious complications may occur due to production defects of these devices at their first use.
At every stage of anaesthesia, safety must be the first concern and continual vigilance is necessary, if possible complications are to be detected.

Case report

A routine dose of thiopental and vecuronium was given to a 42-yr-old female, American Society of Anesthesiologists (ASA) Grade I, scheduled for ophthalmic surgery. The number 4 Rüsch brand anaesthesia face mask which had already been checked was found not to be an adequate fit to the patient’s face. A new face mask, number 5 of the same brand [Anatomical face mask, black 5F245 REF 154600 size 5 Quant. 1 LOT 99/48/A T488, CE 0473, Rüsch Manufacturing (UK) Ltd, Portadown Road, Lurgan BT66 8RD County, Armagh, Northern Ireland], was taken out from its package by an assistant senior doctor and ventilation attempted. However, ventilation proved to be impossible and a high pressure was obtained at the inflating bag. The anaesthesia breathing circuit was examined and it was seen that there was a rubber diaphragm between the mask connecting ring and its body which completely prevented the passage of anaesthetic gases. Ventilation was performed using another face mask and the anaesthesia continued without problem. The vital signs of the patient remained stable. The manufacturer was informed about the problem of the anaesthesia face mask.

Safety and equipment defects have been discussed at a symposium in our country [1]. Defects were reported with catheters, endotracheal tubes and a number of respiratory devices. It is recommended that these devices should be checked before use [3–6]. Although single use breathing devices have advantages, some manufacturing defects may be seen [2]. Our case of a completely closed lumen in a face mask by a rubber diaphragm is a manufacturing error and we can find no similar case in the literature (Fig. 1). It is clear therefore that the face mask must also be included in the pre-anaesthesia check before every case.

M. A. Yasar, Ö. L. Erban, A. Bestas L. Avci, M. Ezici
Department of Anaesthesia and Reanimation
Firat University, Faculty of Medicine
Elazig, Turkey

References


Figure 1.
The anaesthesia face mask totally sealed by a rubber layer.

Anaesthetic breathing circuit obstruction by a massive blood clot

EDITOR:
A 60-yr-old female weighing 90 kg was admitted to hospital with severe oral bleeding. She was a known case of carcinoma of the left pyriform sinus and had undergone chemotherapy and radiotherapy over the previous 12 months. An oropharyngeal pack was required to control the bleeding while an elective tracheostomy was undertaken. The bleeding was initially controlled but she again experienced massive bouts of bleeding and approximately 2 L of blood was lost. Clinical examination revealed bilateral cervical lymph node enlargement with the node on the left side probably eroding into the left external
carotid artery. Due to her poor general condition, an angiography was not performed. She was scheduled for an emergency ligation of the left external carotid artery.

Pre-anaesthetic examination revealed a conscious, spontaneously breathing, elderly woman with no other apparent co-morbidity. Auscultation of the chest revealed bilateral equal air entry with scattered wheezes. She had received 5 units of blood transfusion and was maintained on oxygen supplementation through a T piece.

In the emergency operating theatre, she was connected to the anaesthetic circuit (Ohmeda Exel 210 SE, Ohmeda Madison, USA) and 100% oxygen was administered. Her tracheostomy tube was suctioned which showed a little blood stained fluid. The initial blood pressure reading was 92/40 mmHg and the arterial oxygen saturation was 95%. Her right femoral vein was cannulated. Soon after, the patient started bleeding through the oral packs and had bouts of vigorous coughing. Anaesthesia was induced with ketamine 100 mg intravenously and vecuronium 4 mg was given for muscle relaxation. On attempting manual ventilation, resistance was noted. Bilateral air entry was absent and peak airway pressures reached 60 cmH₂O. The patient was immediately disconnected from the breathing circuit and tracheal suctioning revealed a 3 cm clot with a few millilitres of blood stained fluid. The breathing circuit was reconnected and ventilation attempted, but this again proved unsuccessful. The patient began to develop a bradycardia with hypotension and decreasing arterial oxygen saturation. The breathing circuit was immediately disconnected and manual ventilation begun with a self-inflating (airway mask breathing unit, AMBU) bag. Ventilation was easy and the patient quickly stabilized. Examination of the anaesthetic breathing circuit revealed a 35 cm long blood clot weighing 300 gm in the inspiratory limb (Fig. 1). The remainder of the anaesthetic proceeded uneventfully.

This case illustrates the possibility of the anaesthetic breathing circuit becoming obstructed due to a massive blood clot. Our patient had a tracheostomy and the cuff was kept inflated. The formation of a huge clot in the lower respiratory tract could have been due to seepage of blood around the tracheostomy tube cuff because of tight oropharyngeal packing. Though a small clot was aspirated on tracheal suction, we never anticipated such a massive clot in her lower respiratory tract, because auscultation of her chest preoperatively did not reveal significant findings. In a previous report, Arney and colleagues have emphasized that a large clot in the lower airway may cause only a minimal impact on the respiratory function [1].

Runciman and colleagues [2] suggested that in cases of unexpected obstruction to ventilation during anaesthesia, eliminating the anaesthesia breathing circuit, as the cause of obstruction should be an immediate priority. In their crisis management algorithm, the authors have outlined the steps involved in the process of eliminating the whole circuit.

We had carried out a complete pre-anaesthetic check of the anaesthesia machine. The tracheostomy tube was in a satisfactory place and the patient had been breathing spontaneously. Our ability to ventilate the patient with a self-inflating (AMBU) bag ruled out patient airway obstruction and implicated the anaesthesia circuit as the cause of failure to ventilate.

In conclusion, we present a case of obstruction of an anaesthetic breathing circuit by a massive blood clot. Apart from complete anaesthesia machine check, the availability of an auxiliary ventilating device should always be confirmed before induction of anaesthesia.

Case reported from: Department of Anaesthesia, Post Graduate Institute of Medical Education and Research (PGIMER), Chandigarh, India.

N. Gopinath
Department of Anaesthesia
Bradford Royal Infirmary
West Yorkshire, England, United Kingdom

I. Bala
Department of Anaesthesia
PGIMER
Chandigarh, India

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