Case Report

The use of two novel techniques in the coagulopathic trauma patient in intensive care

Andrew Aquilina, Stephen C. Sciberras

Abstract
Severe Trauma is a multisystem disorder with a high mortality in an often young population. It offers a big challenge to the intensivist, and any help that is available might prove decisive in saving the patient's life.

This case report highlights the use of two novel technologies in critically ill trauma patients, namely the use of activated recombinant factor VII (Novoseven®) to correct coagulopathy, and the use of a central venous oxygen saturation monitor (Presep™ with Vigileo™) to optimise perfusion.

Introduction
A 16 year old male, JH, presented to a primary health care centre with severe abdominal pain and abrasions in the right upper quadrant, following a blunt abdominal injury. At the time he was fully conscious with a Glasgow Coma Scale of 15. Haemodynamically, he was maintaining a systolic blood pressure of 160mmHg, but he was tachycardic and pale. Initial treatment was with 500ml of Gelafundin®, and intramuscular pethidine for pain relief. He was then transported urgently to St. Luke’s Hospital A&E department for further management.

A computed tomography (CT scan) of the brain, thorax and abdomen revealed a large liver haematoma with free fluid and a liver laceration involving the right liver lobe. There was no intracranial pathology, and only small pulmonary contusions in the right lower lung.

Keywords
Factor VIIa, continuous central venous oxygen saturation, coagulopathy

The patient was taken to theatre within the hour. Laparotomy confirmed a huge laceration of the right hepatic lobe, with major haemorrhage. Due to the extensive damage, the liver was packed and the abdomen closed. The patient was transferred to intensive care, intubated and ventilated, as the extent of the tear made a rapid recovery highly unlikely. Invasive monitoring included central venous pressure (CVP), continuous invasive radial arterial blood pressure, peripheral oxygen saturation, and end-tidal carbon dioxide (Marquette™ monitor). On admission, transfusion of blood and blood products was started to inhibit fibrinolysis.

In spite of transfusion of large doses of fresh frozen plasma, cryoprecipitate and platelets, it became apparent that the patient was becoming coagulopathic. Abdominal drains had to be emptied hourly (fresh blood). Investigations showed a low platelet count and prolonged INR and APTT ratios confirming a coagulopathy. By now, a state of disseminated intravascular coagulopathy (DIC) was suspected, and tranexamic acid (Cyclokapron®) at a dose of 1g every 8 hours, and aprotinin infusion at a dose of 500,000 units per hour (Trasylol®) (following a loading dose) were started.

Eight hours post admission, JH remained haemodynamically unstable. A norepinephrine (noradrenaline) infusion was started to maintain a systolic blood pressure of around 100mmHg. A non-invasive, continuous cardiac output monitor (Flotrac™ sensor connected to Vigileo™ monitor, Edwards Lifesciences LLC, Irvine, CA) was set up. This demonstrated a hyperdynamic circulatory state, with cardiac output measurements reaching up to 20 L/min. At regular intervals this would drop suddenly to 8L/min (corresponding with a period of severe hypotension) indicating the need for further transfusion.

As the situation was starting to look bleak a decision was taken to start activated recombinant Factor VII (rVFVIIa - Novoseven®, Novo Nordisk A/S, Bagsværd, Denmark). A dose of 90,000iu (1.8mg) every 8 hours was administered initially but this had to be reduced to 60,000iu (1.2mg) after 24 hours due to limited supplies.

Twenty four hours post-admission, JH was still bleeding heavily and he was taken back to theatre. The packs were replaced and no attempt was made to close the abdomen as the bowels were starting to swell and it was feared that the patient might develop an intra-abdominal compartment syndrome. After the second laparotomy JH was even more unstable. His inotropic support now included epinephrine (adrenaline), his
Oxygenation was severely impaired and bleeding continued unabated. At this point JH had received so much blood and blood products and lost so much blood through his open abdomen that assessing his intravascular volume status became difficult. We felt the need to have a better indicator of tissue oxygenation. A decision was taken to insert a Presep™ catheter with Vigileo™ monitor in order to assess the central venous oxygen saturation (ScvO₂) as a surrogate of mixed venous oxygen saturation. The initial value of ScvO₂ was 85% (indicating perfusion which was satisfying tissue oxygen demand). It was noticed that the drop in this value to less than 65% indicated a fresh episode of bleeding. More importantly the drop happened well before the other parameters such as cardiac output and blood pressure changed. Thus we could treat hypoperfusion before it started to harm his tissues. At this time a large right haemothorax containing serosanguineous fluid was drained.

On the fourth day, JH stabilised. It became possible to wean off inotrope infusions. Transfusion requirements dropped dramatically and his tests of coagulation started to normalize. He was taken to theatre again for change of packs on the fifth day. Bleeding had stopped so the packs were not replaced. His abdomen was closed on the 8th day and he was taken off ventilation on the 9th day. As expected after such a stormy week (Table 1) his postextubation course was complicated by a confusional state. Generalized seizures made us scan his brain but this proved normal. He remained febrile for several weeks but this too eventually settled. He was transferred to a general surgical ward 27 days post admission and continued to recover.

**Table 1: The daily progress of JH up to the seventh day**

<table>
<thead>
<tr>
<th>Admission</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
</tr>
</thead>
<tbody>
<tr>
<td>(night)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lowest Hb</td>
<td>6.4</td>
<td>6.0</td>
<td>5.8</td>
<td>5.3</td>
<td>5.5</td>
<td>5.9</td>
<td>8.2</td>
</tr>
<tr>
<td>Highest INR</td>
<td>2.6</td>
<td>2.6</td>
<td>1.3</td>
<td>1.5</td>
<td>1.7</td>
<td>2.1</td>
<td>2.1</td>
</tr>
<tr>
<td>Highest APPT</td>
<td>3.0</td>
<td>3.0</td>
<td>3.6</td>
<td>2.3</td>
<td>1.4</td>
<td>1.1</td>
<td>1.1</td>
</tr>
<tr>
<td>Lowest Platelets</td>
<td>72</td>
<td>74</td>
<td>39</td>
<td>55</td>
<td>58</td>
<td>63</td>
<td>64</td>
</tr>
<tr>
<td>Packed Red Blood cells (units)</td>
<td>5</td>
<td>15</td>
<td>14</td>
<td>2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Platelets (units)</td>
<td>6</td>
<td>12</td>
<td>44</td>
<td>6</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cryoprecipitate (units)</td>
<td>6</td>
<td>18</td>
<td>20</td>
<td>12</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fresh Frozen Plasma (units)</td>
<td>7</td>
<td>20</td>
<td>18</td>
<td>8</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>average FiO₂</td>
<td>.40</td>
<td>1.00</td>
<td>.60</td>
<td>.60</td>
<td>.40</td>
<td>.35</td>
<td>.35</td>
</tr>
<tr>
<td>lowest pH</td>
<td>7.195</td>
<td>7.188</td>
<td>7.326</td>
<td>7.393</td>
<td>7.465</td>
<td>7.528</td>
<td>7.428</td>
</tr>
<tr>
<td>ScvO₂</td>
<td>62-80%</td>
<td>62%</td>
<td>62-81%</td>
<td>79-87%</td>
<td>stopped</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Hb: Haemoglobin (g/dL); INR: International Normalized Ratio; APPT: Activated Partial Prothrombin Time; Platelets (x10⁹/L); FiO₂: Fractional Inspired Oxygen concentration; ScvO₂: central venous oxygen saturation

At this point JH had received so much blood and blood products and lost so much blood through his open abdomen that assessing his intravascular volume status became difficult. We felt the need to have a better indicator of tissue oxygenation. A decision was taken to insert a Presep™ catheter with Vigileo™ monitor in order to assess the central venous oxygen saturation (ScvO₂) as a surrogate of mixed venous oxygen saturation. The initial value of ScvO₂ was 85% (indicating perfusion which was satisfying tissue oxygen demand). It was noticed that the drop in this value to less than 65% indicated a fresh episode of bleeding. More importantly the drop happened well before the other parameters such as cardiac output and blood pressure changed. Thus we could treat hypoperfusion before it started to harm his tissues. At this time a large right haemothorax containing serosanguineous fluid was drained.

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**Discussion**

**Activated Recombinant Factor VII**

Factor VII (FVII), a vitamin K–dependent serine protease glycoprotein, is an important part of the coagulation pathway (Figure 1). Since the work by Alexander et al in 1951, it is considered to be the key initiator of coagulation. It is found in plasma at a concentration of 0.5mg/L, with 1% of this being in the activated form (FVIIa). Both forms will bind to Tissue Factor (TF), and this binding facilitates conversion from FVII to FVIIa. The TF-FVIIa complex then enhances the conversion of factor IX to factors IXa and X to factor Xa, and hence initiates both the intrinsic and extrinsic pathways. For a summary of the properties of both FVII and FVIIa (Table 2).

Recombinant FVIIa (rFVIIa) – eptacog alpha (Novoseven®) - is produced by transfecting baby hamster cells with the F7 gene. No human plasma-derived components are used in the formulation of rFVIIa, so there is no risk of human viral transmission. It is available as a dry powder for reconstitution, in vials with 1.2mg, 2.4mg, or 4.8mg of rFVIIa. After reconstitution each vial contains approximately 0.6mg/ml rFVIIa. Each ml would then contain 30,000 units. The cost for this treatment (basic NHS Price) would be £664.72 for the 1.2mg preparation, £1,329.44 for the 2.4mg preparation, and £2,658.88 for the 4.8mg preparation.
Mode of action

Its mode of action is similar to natural FVIIa, but it is not just a means of augmenting FVII levels. At the pharmacological doses given, rFVIIa attains concentrations up to 10 times higher than that found in blood. At these levels, rFVIIa does not need TF to initiate conversion of Factor X to Xa on activated platelet surfaces. This means that rFVIIa bypasses both the intrinsic and extrinsic pathways (Figure 1). Nevertheless its action is still localised to the area of endothelial damage where platelets are activated. It has a half-life of 2.7 hours, though this might be less in heavily bleeding patients.6

rFVIIa in trauma

rFVIIa is licensed by the US Food and Drug Administration for:

1. treatment of bleeding in individuals with haemophilia A and B with acquired inhibitors to FVII, FIX (licensed in 1999).
2. persons with congenital factor VII deficiency (licensed in 1999).
3. prevention of bleeding in surgical procedures for both inhibitor patients and FVII-deficient patients (licensed in 2005).7

In the European Union, rFVIIa is also licensed for patients with Glanzmann’s thrombasthenia with antibodies to GPIIb IIIa and/or HLA, and showing poor response to platelet transusions.4

Other unlicensed indications for rFVIIa include use in patients with liver disease, thrombocytopenia, or qualitative platelet dysfunction and in patients with no coagulation disorders who are bleeding as a result of extensive surgery or major trauma.

Most of the evidence to support the use of rFVIIa in trauma derives from case reports, with few clinical studies being performed. Use of rFVIIa for trauma patients started as early as 1999, when Kenet et al8 published the first successful use of the drug in a soldier who had sustained a high velocity gunshot to the inferior vena cava, complicated with a traumatic coagulopathy. In liver trauma, Martinowitz et al9 showed that rFVIIa is effective in reducing blood loss in experimental Grade V liver trauma in swines with coagulopathy, but only if surgical control of the bleeding was obtained by packing. In another case series involving seven patients, Martinowitz and colleagues9 showed that the use of rFVIIa stopped the haemorrhage, decreased the number of blood products transfused, and improved laboratory markers of coagulopathy. The authors suggested that rFVIIa should be considered as an adjunctive haemostatic agent in trauma patients.

There are very few randomised controlled trials on the use of rFVIIa in trauma scenarios. One is that by Boffard and colleagues11, who reported the use of rFVIIa in 247 patients with penetrating or blunt injuries. The patients in the investigation arm were given an initial dose of 200μg/kg, then another two doses of 100μg/kg at one and three hours after admission. Controls received placebo injections. Statistically significant reductions in blood transfusions were observed for patients with blunt trauma, with trends in a reduction in mortality and critical complications, including ARDS. Similar trends were observed for patients with penetrating injuries. Sub-group analysis12 showed that coagulopathic trauma patients appear to derive particular benefit from early adjunctive rFVIIa therapy.

Dosage and safety

The recommended dose by the manufacturer for haemophiliacs is 30μg/kg in patients without inhibitors to Factor VII or VIII, and 90μg/kg in those with inhibitors. Since rFVIIa is not licensed for haemorrhagic situations except for haemophilic patients, the exact dose to use in trauma is currently unknown. The dosages mentioned in the literature vary from low dose, such as 10μg/kg, to higher doses, up to 212μg/kg.13

Once administered, bleeding usually stops in about 15 minutes, but in a number of cases, repeated doses have had to be given. The dosage interval varies from 2 to 12 hours.

Studies have been performed in order to demonstrate any factors which would help to select patients who would benefit from rFVIIa and those who would not. The major factor seems to be the pH, with acidosis causing a drastic loss of efficacy of the drug in vitro (up to 90% loss of efficacy at a pH of 7.10).14 Clinical studies have confirmed this, together with adding the Revised Trauma Score and pre-administration PT as markers for futility for rFVIIa usage.15 It seems that early use of rFVIIa, before acidosis and haemorrhage become severe, might improve morbidity and mortality. This was confirmed in a study by Perkins and colleagues16, with patients receiving rFVIIa early requiring about 20% less blood products, but the reduction in complications and mortality was not statistically significant.

Table 2: Summary of properties of FVII and FVIIa

<table>
<thead>
<tr>
<th></th>
<th>FVII</th>
<th>FVIIa</th>
</tr>
</thead>
<tbody>
<tr>
<td>Synthesis</td>
<td>liver, coded by F7 (13934)</td>
<td>cleaved FVII</td>
</tr>
<tr>
<td>Structure</td>
<td>single chain, 48kDa</td>
<td>two chains - light: 20kDa - heavy 30kDa</td>
</tr>
<tr>
<td>Concentration</td>
<td>0.5mg/L</td>
<td>0.0036 mg/L</td>
</tr>
<tr>
<td>Half-time</td>
<td>3 – 6 hours</td>
<td>2.5 hours</td>
</tr>
<tr>
<td>- age, female gender, pregnancy, hypertriglycerideremia</td>
<td>Xa, thrombin, IXa, XIa, XIIa</td>
<td></td>
</tr>
<tr>
<td>- decreased with</td>
<td>Warfarin, Vit K deficiency, liver failure, mutations</td>
<td></td>
</tr>
</tbody>
</table>
Monitoring the use of rFVIIa is difficult. Firstly, rFVIIa is usually given to patients who are not responding to traditional management with fresh frozen plasma, cryoprecipitate and platelets. This means that any change in laboratory values would be difficult to attribute to rFVIIa alone. Most studies show that there is a decrease in prothrombin time and the activated thromboplastin time with the use of rFVIIa. Thromboelastogram studies demonstrate faster and better clot formation. There is no ideal way of assessing its effect: how then do you decide whether to continue administration or whether to increase or decrease the dose? Furthermore, the use of concomitant platelet transfusion is debatable, but rFVIIa does need an adequate platelet count in order to work.

There have been concerns regarding the effect of rFVIIa on thromboembolic rates. Although sporadic cases of deep venous thrombosis and pulmonary embolism, cerebrovascular events, and even right atrial thrombosis have been described in case reports, the complication rate is not high. For instance, during the period 1996–2000, more than 140,000 doses of rFVIIa were administered for haemophilia, with a complication rate of less than 0.02%. The complication rate may be higher if the doses used in trauma are administered.

rFVIIa is a recombinant protein, and hence one would expect common adverse reactions such as fever, injection site reaction, arthralgia, headache, nausea, vomiting, pain, oedema and rash. Patients who show allergies to mouse, hamster, or bovine protein should not be exposed to the drug (Table 3).

**rFVIIa in our case**

In our case, rFVIIa was started shortly after admission. JH was already in established DIC, and like most other patients described in the literature, he had been given conventional haemostatic agents, with little effect. We used a dose of 90,000 units (1.8mg) initially, then 60,000 units (1.2mg), which would correspond to a dose of 30μg/kg and 20μg/kg respectively. This would probably be considered as a low-dose, although lower doses have been mentioned in the admittedly scanty trauma literature.

The use of rFVIIa in our patient did not result in an abrupt termination of bleeding. In fact, treatment had to be continued for two days, for 8 doses, before bleeding was controlled. This could be due to a number of factors. The ongoing DIC in our patient would have resulted in a massive consumption of rFVIIa, with a loss of effect at the target site. Furthermore, the liver packs themselves used to control the bleeding might have propagated the DIC. Finally, the rFVIIa might have been ineffectuous in this case due to inadequate dose. Our patient did have an acidosis before the second laparotomy, but this was controlled by the time rFVIIa was started. It might also be that the levels in the blood increased gradually with each dose until an adequate concentration allowed clotting to occur.

It must be noted that trauma management is a complicated issue, and the outcome, that is survival, is not solely dependent on the amount of bleeding. Organ failure and sepsis are complicating factors that have dramatic effects on mortality. Hence, the reduction in transfusions, and better control of bleeding with rFVIIa may not necessarily translate into better survival. As an example, in a study by Payne, 68% of patients with bleeding, due to either haematological (30% of patients) or due to surgical causes (53%), who were given rFVIIa responded to the drug. However, only 40% survived, with the highest mortality being registered in the patients with bleeding of

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**Table 3: Side effect profile of rFVIIa**

**Adverse reactions**

Most common: pyrexia, hemorrhage, injection site reaction, arthralgia, headache, hypertension, hypotension, nausea, vomiting, pain, edema and rash

Rare (0.1% - 0.01%): lack of effect

Very rare (<0.01%): Coagulopathic disorders such as increased D-dimers and consumptive coagulopathy; myocardial infarction; nausea; fever; pain (especially at injection site); increase in alanine transaminase, alkaline phosphatase, lactate dehydrogenase and prothrombin levels; cerebrovascular disorders including cerebral infarction and cerebral ischaemia; venous thrombotic events; haemorrhage.

**Serious adverse reactions**

Arterial thrombotic events (such as myocardial infarction or ischaemia, cerebrovascular disorders and bowel infarction); venous thrombotic events (such as thrombophlebitis, deep vein thrombosis and pulmonary embolism). In the vast majority of cases, patients were predisposed to such events.

**Potential adverse reactions**

Patients with disseminated intravascular coagulation (DIC), advanced atherosclerotic disease, crush injury, septicemia, or concomitant treatment with activated or nonactivated prothrombin complex concentrates (aPCCs/PCCs) may have a potential risk of developing thrombotic events in association with NovoSeven treatment. Disseminated intravascular coagulation (DIC) was reported in a few patients who received NovoSeven during treatment with aPCCs/PCCs.

**Anaphylaxis**

No spontaneous reports of anaphylactic reactions, but patients with a history of allergic reaction should be carefully monitored. Contraindicated in patients with known hypersensitivity to NovoSeven or mouse, hamster, or bovine protein.

One case of angioneurotic oedema reported in patient with Glanzmann’s thrombasthenia after administration of NovoSeven.
haematological cause. This was confirmed in a recent study by Rizoli, who showed that there was a strong reduction in early mortality (within 24 hours), but the trend for better overall mortality was not statistically significant.

Although it is difficult to prove that the use of rFVIIa proved life-saving, we feel that the treatment was one determining factor in our patient’s survival.

Central venous oxygen saturation monitoring

The oxygen saturation of blood returning to the heart is a measure of the balance between oxygen supply and oxygen demand. In turn, this reflects cardiac output, haemoglobin levels, and arterial oxygen saturation (oxygen supply), and the tissue metabolism (oxygen demand), as shown in Figure 2.

Anything which upsets either the supply or the demand will result in a deviation from the normal values of 60 – 80%. The venous saturation will only truly reflect the global tissue perfusion if it is sampled in the pulmonary arteries (mixed venous oxygen or SvO₂). Accessing the pulmonary arteries requires the insertion of a pulmonary artery catheter. The insertion of a pulmonary artery catheter carries a risk of complications which some people deem unjustifiable. A less risky alternative would be useful. An alternative site is venous blood sampled from a central venous catheter i.e. a central venous oxygen saturation: ScvO₂. This will sample blood from the superior vena cava (head and upper body).

The evidence

Reinhart has shown that in anaesthetised dogs, the correlation coefficient between SvO₂ and ScvO₂ over a broad range of cardiorespiratory conditions, including hypoxia, haemorrhage, and resuscitation was 0.97. However, the values were not identical, and not interchangeable for the purpose of calculating shunt fractions. The study by Ladakis was one of a number of studies which showed that such a relationship existed even in critically ill patients, but others have proven otherwise. However, all studies in the literature note that ScvO₂ is always higher than SvO₂, by about 5 – 18%. Hence, it is necessary to use a higher threshold for action, with the literature quoting a figure of around 75% at which complications are avoided. Also, all studies have shown that the trend in ScvO₂ follows that of SvO₂.

In haemorrhage, ScvO₂ has been found to be a reliable marker of the extent of blood loss. Scalea et al. have found that only ScvO₂ and the cardiac index (in itself a strong determinant of ScvO₂) showed a linear relationship to blood loss in anaesthetized dogs. Tissue extraction started to increase when blood loss was only 3-6% of circulating blood volume. In humans, a blood loss of 100ml would reduce SvO₂ by about 1%. Furthermore, even in patients who appear stable when monitored by heart rate, blood pressure, pulse pressure, urine output, central venous pressure, a ScvO₂ of 65% is related to more serious injuries, and blood transfusions. Hence ScvO₂ may be considered to be an early marker of decompensation.

In the majority of cases a decrease in SvO₂ and ScvO₂ represents an alert that the global oxygenation of tissues is inadequate, regardless of the source of this decrease. Examples are shown in Figure 2.

An increasing number of studies are showing that using the ScvO₂ in a clinical protocol does improve the outcome in a critical care setting. Rivers and coworkers used continuous ScvO₂ monitoring in an early goal-directed therapy algorithm in severe sepsis and septic shock patients. With a threshold ScvO₂ of 70%, the mortality rate in the treatment arm was decreased by 16.7% in comparison with the control group. This study forms the basis for the recommendation for early goal-directed therapy by the Surviving Sepsis Group (Figure 3).

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**Figure 2: Oxygen balance between oxygen delivery and oxygen demand**

<table>
<thead>
<tr>
<th>Low SvO₂</th>
<th>Low oxygen delivery</th>
<th>Hypoperfusion</th>
<th>Cardiogenic shock</th>
<th>Septic shock</th>
<th>Anemia</th>
<th>Hypoventilation</th>
<th>Acute respiratory failure</th>
</tr>
</thead>
<tbody>
<tr>
<td>High SvO₂</td>
<td>High oxygen delivery</td>
<td>Anemia</td>
<td>Severe anaerobic</td>
<td>Shock</td>
<td>High output cardiac failure</td>
<td>Low oxygen demand</td>
<td>Low oxygen delivery</td>
</tr>
</tbody>
</table>

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**Table 4: Using the Presep™ Catheter with the Vigileo™ monitor**

Prepare patient for central venous access

Calibrate system using in vitro method

- With Presep™ still unflushed and with calibration cup still on, connect optical module to monitor.
- Enter haemoglobin level.
- Calibrate.
- Insert catheter into patient with aseptic technique.
- Use ScvO₂ as per ITU protocols.

Use Presep™ as normal central line

Every 12-24 hours, or prn, perform in vivo calibration

- Set up monitor for in vivo calibration.
- Press DRAW BLOOD on menu, then slowly draw 2ml of venous blood over 30 seconds, from distal port of Presep™ catheter. This will allow monitor to time the sample.
- Send sample for co-oximetry, if necessary also for haemoglobin estimation.
- Use values in order to continue the calibration process.
Measuring ScvO₂

ScvO₂ can be measured in two ways: by analysing sampled blood with a co-oximeter (venous blood gas), or by continuous reflection spectrophotometry. Although the former is considered the gold standard, it is cumbersome, requires nursing staff to repeatedly sample blood and can only be done intermittently. Spectrophotometry is a relatively new technique that has been found to have good clinical correlation with co-oximetry. 27 Current central venous catheters with such saturation probes at the distal end, such as the Presep™, need to be calibrated against a co-oximeter, both before insertion and during its use (Table 4), since the absorption spectrum of red blood cells depends on the relative concentrations of oxyhaemoglobin and haemoglobin. Hence, calibration should be performed more frequently in unstable patients, or in those with frequent changes of haemoglobin (company quotes changes of 6% or more35).

ScvO₂ in practice

The use of ScvO₂ is being promoted as a means of quickly identifying sudden deteriorations in the clinical condition of the patient before decompensation occurs. It is also being integrated in clinical protocols as an end-target parameter, the most notable of such protocols32 being incorporated in the Surviving Sepsis Campaign 2004 guidelines (Figure 3). In trauma, it has already been shown that a decrease in ScvO₂ is one of the earliest signs of bleeding, since an increase in oxygen extraction is one of the earliest compensatory mechanisms, even before any changes in heart rate, systolic, mean and diastolic blood pressure, and stroke index.37

The Presep™ was used late in the case of JH, on the third day after his admission. Hence its use in this case cannot be described as an “early goal-directed therapy.” Still, the Presep™ did allow us to pick up new episodes of bleeding, since each time this happened, the value dipped to 60%. In fact, not even the use of a non-invasive, pulse-contour cardiac output monitor proved to be adequate in the case of JH, since it did not give us early warning of bleeding. Pulmonary artery catheters with spectrophotometry are available locally, but the use of the pulmonary artery catheter in our ITU has become rare, so we felt that the use of the Presep™ offered a better risk-to-benefit ratio.

To obtain the best use of central venous saturation monitoring, it is important to realise that the catheter alone does not improve prognosis. Rather, it is the use of the information to direct therapy which alters outcome. Ideally it should be used in the context of previously established guidelines. We shall be introducing these guidelines in the near future.

Conclusion

The use of two new techniques allowed us to optimise the management of a critically ill victim of blunt abdominal trauma with massive liver injury and severe coagulopathic bleeding. We recommend the establishment of local guidelines to guide the use of these techniques.

References
