CLINICAL RESEARCH

Prognosis value of central venous oxygen saturation in acute decompensated heart failure

Valeur pronostique de la saturation veineuse en oxygène dans l’insuffisance cardiaque aiguë

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Received 25 May 2011; received in revised form 2 October 2011; accepted 20 October 2011
Available online 13 January 2012

KEYWORDS
Acute heart failure; Cardiogenic shock; Heart transplant; Central venous oxygen saturation

Summary
Background. — Central venous oxygen saturation (ScvO\textsubscript{2}) provides an estimation of body oxygen consumption/delivery ratio. Its use has been suggested for monitoring treatment of patients admitted for acute decompensated heart failure (ADHF) but the optimal target value has never been clearly reported.

Aims. — We aimed to address the prognostic value of ScvO\textsubscript{2} in ADHF requiring inotrope support.

Methods. — ScvO\textsubscript{2} was prospectively assessed in 60 patients with ADHF requiring inotrope support (mean age 62 ± 16 years; 45 men; left ventricular ejection fraction 25 ± 7%) and was compared with major adverse cardiac events (MACE), defined as heart transplantation, cardiac assistance and death.

Results. — MACE occurred in 22 (35%) patients (14 deaths; eight referred for heart transplantation or cardiac assistance). Admission ScvO\textsubscript{2} (mean 57 ± 13%) did not differ between patients with and without MACE. At 24 hours ScvO\textsubscript{2} (mean 62 ± 7%) increased only in patients without MACE (65 ± 6\% vs. 58 ± 7\%; \textit{p} < 0.0001) and was associated with urine output, vena cava diameter and oxygen consumption reduction. No correlation was observed

Abbreviations: ADHF, acute decompensated heart failure; BP, blood pressure; LV, left ventricular; MACE, major adverse cardiac events; ScvO\textsubscript{2}, central venous oxygen saturation; TAPSE, tricuspid annular plane systolic excursion; VO\textsubscript{2}, organ oxygen consumption.

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doi:10.1016/j.acvd.2011.10.005
between ScvO2 and cardiac output or catecholamine rate. Multivariable analysis showed that ScvO2 at 24 hours remained an independent predictor of MACE. Using the optimal cut-off of 60% derived from receiver operating characteristic curves, MACE were observed in 81% of patients (17/21) with ScvO2 ≤ 60% at 24 hours vs. 13% (5/39) with ScvO2 > 60% at 24 hours.

Conclusion. — In patients admitted for ADHF requiring inotropic support, ScvO2 ≤ 60% despite optimal treatment is a marker of poor outcome and might be an indicator for considering more aggressive therapy.

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Résumé

Contexte. — La saturation veineuse centrale en oxygène (ScvO2) permet une estimation de l’adéquation entre l’apport en oxygène et la consommation tissulaire. Son utilisation pour le monitoring des patients hospitalisés pour insuffisance cardiaque aiguë est préconisée mais sa valeur optimale et l’influence de ses variations sur le pronostic n’ont jamais été étudiées.

Objectifs. — L’objectif est d’étudier la valeur pronostique de la ScvO2 chez les patients hospitalisés pour insuffisance cardiaque aiguë sévère.

Méthodes. — La ScvO2 a été recueillie de façon prospective chez 60 patients (moyenne 62 ± 16 ans; 45 hommes; FEVG 25 ± 7 %) hospitalisés pour une poussée d’insuffisance cardiaque avec nécessité de recours à un traitement inotrope positif. Le critère de jugement incluait la survenue d’événements cardiovasculaires majeurs (décès, assistance ou transplantation cardiaque).

Résultats. — Un événement est survenu chez 22 (35%) patients (14 décès; huit assistances ou transplantation cardiaque). Il n’y avait pas de différence concernant la ScvO2 à l’admission (moyenne 57 ± 13 %) chez les patients avec et sans événements. Après 24 heures de traitement, la ScvO2 (moyenne 62 ± 7 %) n’augmentait que chez les patients sans événement (65 ± 6 % vs 58 ± 7 %; p < 0,0001) et était corrélée à la diurèse, au diamètre de la veine cave inférieure et à la réduction de la consommation en oxygène. La ScvO2 n’était ni corrélée aux doses de catécholamines ni au débit cardiaque. En analyse multivariée, la ScvO2 restait associé au pronostic. La survenue d’un événement était observée chez 81 % (17/21) des patients ayant une ScvO2 ≤ 60 % à 24 heures contre 13 % (5/39) lorsque la ScvO2 était inférieure à 60 % à 24 heures.

Conclusion. — Chez les patients hospitalisés pour insuffisance cardiaque aiguë avec indication à un traitement inotrope positif, une ScvO2 ≤ 60 % malgré un traitement médical maximal est associé à un mauvais plus agressif. Cette valeur pourrait faire considérer des options thérapeutiques

Background

Despite improvements in medical treatment, acute decompen-sated heart failure (ADHF) with cardiogenic shock remains associated with high mortality (50–80%) [1]. In clinical practice, conventional treatment includes catecholamine and intravenous diuretic support, monitored by echocardiography and clinical data. European Society of Cardiology guidelines [2] recommend the use of central venous oxygen saturation (ScvO2) for monitoring these patients. ScvO2 (i.e. the oxygen saturation from a blood sample taken from the superior vena cava) provides an estimation of the body oxygen consumption/delivery ratio. According to Fick’s equation, ScvO2 is determined by organ oxygen consumption (VO2; mL/minute) and supply, which depends on arterial oxygen saturation (SaO2; %), haemoglobin concentration (Hb; g/L) and cardiac output (CO): ScvO2 = SaO2 – (VO2/1.34*Hb*CO).

In patients admitted for a severe sepsis or an acute myocardial infarction, ScvO2 correlates with CO; low ScvO2 is associated with poor prognosis. The use of ScvO2 appears superior to clinical markers for monitoring patients with haemodynamic instability and treatment adjusted to ScvO2 seems to provide a better outcome. However, the impact of ScvO2 in patients admitted for ADHF remains unclear. The purpose of this study was to address the optimal target of ScvO2 for monitoring patients admitted for ADHF requiring inotropic support.

Methods

Population study

We prospectively included 60 consecutive patients (mean age 62 ± 16 years; 45 men) admitted from November 2008 to March 2010 to the Intensive Care Unit of Henri Mondor University Hospital for ADHF requiring inotropic support. Inotropic support was added when low CO (cardiac index less than 2.2 L/minute per meter square) or impaired left ventricular (LV) ejection fraction (< 40%) was associated with low blood pressure (BP) (systolic BP < 90 mmHg or a drop in mean BP of more than 30 mmHg) or when persistent signs of low organ perfusion or oligoanuria (< 0.5 mL/kg per hour)
were reported. Patients were excluded when a palliative care decision was taken, when superior vena cava catheterization failed or when associated hypovolaemic or septic shock was suspected. Patients admitted for acute myocardial infarction were excluded.

Of the 60 patients included, six had no heart failure history or known LV dysfunction and 27 (45%) had ischaemic cardiomyopathy. Admission LV ejection fraction and cardiac index averaged 25 ± 7% and 1.9 ± 0.7 L/minute per meter square (confidence interval 1.1–2.7), respectively. Current medications before hospitalization included beta-blocker therapy (70%), angiotensin-converting enzyme inhibitors (79%) and oral loop diuretics (84%). The study was approved by our local (Henry-Mondor Hospital) ethics committee and all patients gave informed consent to participate.

Catecholamine and diuretic infusion

Patients admitted for ADHF received standard care according to our local protocol, which follows current guidelines [2]. All drugs were systematically delivered through a central venous catheter positioned via the jugular or subclavian vein. Optimal positioning of the central venous catheter was confirmed using chest radiography. The catheter had to be at the junction between the superior vena cava and the right atrium. The radiological landmark used to determine the appropriate position was the carina. Intravenous loop diuretics and vasodilator drugs were first recommended before inotropic support when the mean BP was more than 65 mmHg. In patients with low BP and clinical signs of low organ perfusion or with persistent oligoanuria, dobutamine infusion was recommended at a starting rate of 5 µg/kg per minute. Dobutamine was increased by 2.5 µg/kg per minute to a maximum of 20 µg/kg per minute, according to the physician’s clinical judgement. In patients with severe haemodynamic instability or under beta-blocker therapy at admission, dobutamine was started at the rate of 20 µg/kg per minute. Norepinephrine was given if the mean BP remained less than 65 mmHg. Intravenous loop diuretics were systematically delivered with a starting dose adjusted to the severity of volume overload, renal function and previous diuretic oral dose used. The diuretic rate was titrated to obtain euvolaemia assessed using clinical (right ventricular congestion reduction) and echocardiography data (vena cava dilatation and LV pressure reduction). Dobutamine infusion was decreased progressively (0.1 µg/kg per minute per hour) when euvolaemia and haemodynamic stability were reached. No patient needed mechanical ventilation support and, when required, oxygen was delivered through an oxygen mask to obtain an arterial oxygen saturation greater or equal to 95%.

Table 1 Patient characteristics at admission.

<table>
<thead>
<tr>
<th></th>
<th>Event free (n = 38)</th>
<th>MACE (n = 22)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>ScvO2 (%)</td>
<td>57 ± 13</td>
<td>57 ± 14</td>
<td>0.9</td>
</tr>
<tr>
<td>SaO2 (%)</td>
<td>96.8 ± 1.9</td>
<td>96.2 ± 3.8</td>
<td>0.4</td>
</tr>
<tr>
<td>Lactates (mmol/L)</td>
<td>2.2 ± 2.0</td>
<td>3.1 ± 3.4</td>
<td>0.2</td>
</tr>
<tr>
<td>pH</td>
<td>7.43 ± 0.1</td>
<td>7.41 ± 0.1</td>
<td>0.4</td>
</tr>
<tr>
<td>BUN (mmol/L)</td>
<td>15 ± 9</td>
<td>19 ± 8</td>
<td>0.1</td>
</tr>
<tr>
<td>Creatinine (µmol/L)</td>
<td>172 ± 98</td>
<td>194 ± 95</td>
<td>0.4</td>
</tr>
<tr>
<td>Bilirubin (µmol/L)</td>
<td>20 ± 11</td>
<td>29 ± 20</td>
<td>0.02</td>
</tr>
<tr>
<td>BNP (pg/L)</td>
<td>3663 ± 3431</td>
<td>6841 ± 7856</td>
<td>0.03</td>
</tr>
<tr>
<td>Sodium (mmol/L)</td>
<td>131 ± 4</td>
<td>129 ± 6</td>
<td>0.2</td>
</tr>
<tr>
<td>Potassium (mmol/L)</td>
<td>4.1 ± 0.8</td>
<td>4.2 ± 0.8</td>
<td>0.5</td>
</tr>
<tr>
<td>Haemoglobin (mg/dL)</td>
<td>12.0 ± 2.0</td>
<td>11.6 ± 2.3</td>
<td>0.4</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>97 ± 16</td>
<td>98 ± 19</td>
<td>0.8</td>
</tr>
<tr>
<td>Mean BP (mmHg)</td>
<td>72 ± 11</td>
<td>69 ± 13</td>
<td>0.3</td>
</tr>
<tr>
<td>Heart rate (beats/minute)</td>
<td>84 ± 17</td>
<td>94 ± 20</td>
<td>0.1</td>
</tr>
<tr>
<td>Cardiac index (L/minute per square meter)</td>
<td>1.8 ± 0.6</td>
<td>2.0 ± 0.9</td>
<td>0.4</td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>26 ± 6</td>
<td>23 ± 9</td>
<td>0.3</td>
</tr>
<tr>
<td>EDV (mL)</td>
<td>208 ± 84</td>
<td>229 ± 95</td>
<td>0.5</td>
</tr>
<tr>
<td>ESV (mL)</td>
<td>156 ± 68</td>
<td>174 ± 81</td>
<td>0.5</td>
</tr>
<tr>
<td>E/A</td>
<td>2.7 ± 1.2</td>
<td>2.3 ± 1.1</td>
<td>0.3</td>
</tr>
<tr>
<td>SPABP (mmHg)</td>
<td>45 ± 12</td>
<td>41 ± 8</td>
<td>0.4</td>
</tr>
<tr>
<td>TAPSE (mm)</td>
<td>13 ± 5</td>
<td>15 ± 7</td>
<td>0.3</td>
</tr>
<tr>
<td>IVC (mm)</td>
<td>20.3 ± 3.9</td>
<td>21.1 ± 5.4</td>
<td>0.7</td>
</tr>
<tr>
<td>Dobutamine (µg/kg per minute)</td>
<td>8.7 ± 4.6</td>
<td>8.6 ± 3.7</td>
<td>NS</td>
</tr>
<tr>
<td>Furosemide (mg/24 hours)</td>
<td>499 ± 408</td>
<td>510 ± 355</td>
<td>NS</td>
</tr>
<tr>
<td>Noradrenaline</td>
<td>10 (26)</td>
<td>9 (41)</td>
<td></td>
</tr>
<tr>
<td>Rate (mg/hour)</td>
<td>0.9 ± 0.25</td>
<td>0.9 ± 0.7</td>
<td></td>
</tr>
</tbody>
</table>

Data are mean ± standard deviation or number (%). BNP: brain natriuretic peptide; BP: blood pressure; BUN: blood urea nitrogen; E/A: early/late diastolic filling flow; EDF: end-diastolic volume; ESF: end-systolic volume; IVC: inferior vena cava; LVEF: left ventricular ejection fraction; MACE: major cardiac events; SaO2: arterial oxygen saturation; ScvO2: central venous oxygen saturation; SBP: systolic blood pressure; SPABP: systolic pulmonary artery blood pressure; TAPSE: tricuspid annular plane systolic excursion.
### Transthoracic echocardiography

Transthoracic echocardiography was systematically performed at admission and after 24 hours. CO was assessed using the conventional pulsed Doppler method positioned at the LV outflow tract. LV volumes and ejection fraction were quantified using Simpson’s biplane method. Early and late diastolic filling flows were quantified by the amplitude of the tricuspid annular plane systolic excursion (TAPSE). The inferior vena cava diameter was assessed from the subcostal view by M mode during the expiration period [3,4].

### Haemodynamic monitoring

Invasive BP monitoring was performed using a radial arterial catheter. BP and urine output were monitored every hour during the first 12 hours and every 3 hours thereafter. ScvO2 assessment was performed by a standard gas analyser using a blood sample taken from the central venous catheter. ScvO2 was assessed before and every 12 hours after inotropic support was initiated.

### Clinical outcomes

The primary endpoint was defined by the occurrence of major adverse cardiac events (MACE) that included death, heart transplantation or cardiac assistance during the hospitalization period. Heart transplantation and cardiac assistance were indicated in eligible patients (age less than 65 years without severe comorbidity) with a maximal catecholamine dose (dobutamine 20 µg/kg per minute) despite an optimal volume load or a maximal diuretic dose (furosemide 1 g/24 hours); the decision was made by an expert committee that included cardiac surgeons and cardiologists not involved in the study. The French Transplantation Agency (Agence de la Biomédecine) guidelines do not include ScvO2 as a criterion for patient referral for heart transplantation or cardiac assistance.

### Statistical analysis

Normally distributed continuous variables were expressed as mean ± standard deviation and nominal variables as percentages. Comparisons between patients with and without MACE were done using Student’s t test or variance analysis for continuous variables and the chi-square test for categorical variables.
dichotomous values. Paired analysis was performed for repeated values. Multivariable analysis by stepwise regression was used to identify the independent predictor of outcome. For the first step, all variables with \( p < 0.1 \) were included in the model. Statistical difference was considered as significant when \( p < 0.05 \).

**Results**

Admission dobutamine and furosemide rates averaged \( 8.7 \pm 4.3 \) \( \mu \)g/kg per minute (range \( 3–20 \)) and \( 529 \pm 366 \) \( \mu \)g/kg per 24 hours, respectively. Norepinephrine was added in 23 (38\%) patients with persistent low BP at the rate of \( 0.22 \pm 0.1 \) \( \mu \)g/kg per minute. Overall, \( CO \) (3.2 \pm 1.3 \( \text{L/minute} \) to 3.6 \pm 1.2 \( \text{L/minute} \); \( p = 0.06 \)), systolic BP and renal function improved under treatment (Tables 1 and 2).

**Outcome**

During the hospitalization period (11 \pm 12 \text{days}), MACE occurred in 22 (37\%) patients. No MACE occurred in the first 24 hours. Deaths from refractory heart failure occurred after 14 \pm 15 days in 14 patients not eligible for cardiac assistance because of advanced age or comorbidity (72 \pm 14 years). Cardiac assistance and heart transplantation were required in eight patients (46 \pm 10 years, range 35–63) after 15 \pm 8 days under catecholamine. In patients without MACE (\( n = 38 \)), dobutamine infusion was maintained for 5 \pm 4 days after admission and successfully weaned in 3 \pm 3 days. At admission, patients who experienced MACE had a higher plasma concentrations of bilirubin (29 \pm 20 \mu\text{mol/L} vs. 20 \pm 11 \mu\text{mol/L}; \( p = 0.02 \)) and brain natriuretic peptide (6841 \pm 7856 \mu\text{pg/mL} vs. 3663 \pm 3431 \mu\text{pg/mL}; \( p = 0.03 \)). At 24 hours, hyponatraemia and plasma concentrations of bilirubin and lactate were greater in the MACE group (Table 2). Urine output was greater (4.2 \pm 1.9 \text{L/24 hours} vs. 2.9 \pm 1.6 \text{L/24 hours}; \( p = 0.02 \)) and the vena cava diameter was smaller (18 \pm 6 \text{mm} vs. 22 \pm 5 \text{mm}; \( p = 0.04 \)) in patients who did not experience MACE. In contrast, diuretic and inotrope starting rates and cardiac index at admission (2.0 \pm 0.9 \text{L/minute/m}^2 vs. 1.8 \pm 0.5 \text{L/minute/m}^2) and at 24 hours (2.1 \pm 0.7 \text{L/minute/m}^2 vs. 2.0 \pm 0.6 \text{L/minute/m}^2) did not differ according to outcome.

**Admission ScvO\textsubscript{2} and outcome**

Admission ScvO\textsubscript{2} before dobutamine infusion was available in 49 (82\%) patients and averaged 57 \pm 13\% (range 28–77; Fig. 1). Admission \text{ScvO}_2 \leq 60\% was observed in 57\% (28/49) of patients, without difference between those with and without MACE (61\% vs. 55\%). Admission \text{ScvO}_2 correlated with sodium \( r = 0.33; \ p = 0.02 \) and the plasma concentration of brain natriuretic peptide \( r = 0.31; \ p = 0.02 \) but not with \text{CO}, haemoglobin concentration and arterial oxygen saturation.

**Changes in ScvO\textsubscript{2} and outcome**

At 24 hours, \text{ScvO}_2 increased in patients without MACE but remained unchanged in those who experienced MACE (Fig. 1). Percentages of MACE according to \text{ScvO}_2 quartiles are presented in Fig. 2. Using receiver operating characteristic curves, the optimal cut-off value for predicting MACE was 60\% (Fig. 3; Table 3). In patients with \text{ScvO}_2 > 60\% at 24 hours (\( n = 39 \)), dobutamine was safely withdrawn in 87\% (34/39) within 3 \pm 3 days. After dobutamine withdrawal, \text{ScvO}_2 slightly decreased (68 \pm 6\% vs. 65 \pm 8\%; \( p = 0.03 \); Fig. 1).

In contrast, when \text{ScvO}_2 was less or equal to 60\% at 24 hours (\( n = 21 \)), MACE occurred in 81\% (17/21) of patients. In addition, the last \text{ScvO}_2 was less or equal to 60\% in 90\% (19/21) of patients who experienced MACE. No difference was observed between death and cardiac assistance or heart transplantation. By multivariable analysis (Table 4), \text{ScvO}_2, urine output and bilirubin concentration were independent predictors of MACE. Area under receiver operating characteristic curves for these variables are shown in Fig. 3.

![Figure 1](image1.png) "Changes in central venous oxygen saturation (ScvO\textsubscript{2}) mean \pm standard error) according to outcome. MACE: major cardiac events (cardiac assistance, heart transplantation or death). * \( p < 0.05 \) versus admission; † \( p < 0.05 \) versus 24 hours; ‡ \( p < 0.05 \) versus before catecholamine weaning."

![Figure 2](image2.png) "Percentage of major cardiac events according to central venous oxygen saturation (ScvO\textsubscript{2}) quartiles."
Figure 3. Receiver operating characteristic curves for predicting major cardiac events for central venous oxygen saturation (ScvO₂), urine output, lactate and bilirubin at 24 hours. AUC: area under the curve.

Table 3  Sensitivity, specificity, predictive positive values and negative predictive values according to central venous oxygen saturation cut-off values.

<table>
<thead>
<tr>
<th>ScvO₂ (%)</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
<th>PPV (%)</th>
<th>NPV (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>57</td>
<td>64</td>
<td>95</td>
<td>88</td>
<td>82</td>
</tr>
<tr>
<td>60</td>
<td>77</td>
<td>89</td>
<td>81</td>
<td>87</td>
</tr>
<tr>
<td>63</td>
<td>86</td>
<td>58</td>
<td>54</td>
<td>88</td>
</tr>
</tbody>
</table>

NPV: negative predictive value; PPV: predictive positive value; ScvO₂: central venous oxygen saturation.

Relationship between ScvO₂ and other haemodynamic variables

Patients with ScvO₂ > 60% at 24 hours had a greater urine output (4.0 ± 0.3 L vs. 3.0 ± 0.3 L; *p* = 0.04) and vena cava diameter reduction (−18% [−4 ± 6 mm] vs. +15% [2 ± 4 mm]; *p* = 0.007) than patients with ScvO₂ ≤ 60%, while Fick’s equation-derived VO₂ was lower (102 ± 37 mL/minute per square meter vs. 133 ± 48 mL/minute per square meter; *p* = 0.02). In contrast, no difference was observed between ScvO₂ and cardiac index (2.0 ± 0.6 L/minute per square meter vs. 1.9 ± 0.7 L/minute per square meter; *p* = 0.1 for 24-hour ScvO₂ > 60% and ≤ 60%, respectively) or catecholamine rate (dobutamine 9.1 ± 3.9 µg/kg/minute vs. 8.7 ± 4.3 µg/kg per minute; *p* = 0.3 for 24-hour ScvO₂ > 60%
and ≤ 60%, respectively). In addition, we did not observe a significant association between ScvO2 and the TAPSE value at admission and 24 hours after treatment.

**Discussion**

Prognosis of ADHF with cardiogenic shock remains poor despite improvement in medical treatments. Management of patients with uncompensated heart failure and cardiogenic shock is conventionally based on the diagnosis and treatment of underlying causes and on symptomatic treatment with diuretics and inotrope support. The monitoring of these drugs relies on clinical and echocardiography data while ScvO2 is poorly used because its assessment requires invasive catheter positioning and no study has clearly addressed its optimal target in ADHF. The present study demonstrates that an early increase in ScvO2 > 60% at 24 hours is strongly correlated with a favourable outcome. MACE occurred mostly (81%) in patients with ScvO2 that remained ≤ 60%, while event-free survival with successful dobutamine weaning was observed in 88% of patients with ScvO2 > 60% at 24 hours. Importantly, changes in ScvO2 appear to be more associated with a greater reduction in vena cava diameter and urine output (which are indirect markers of response to diuretic treatment and venous congestion) than with the increase in CO, as commonly believed.

The usefulness of ScvO2 for monitoring shock has been addressed in septic shock, with an impressive decrease in mortality with ScvO2-adjusted treatment [5]. The prognostic value of ScvO2 has also been addressed in different critical haemodynamic situations [5–9]. In acute coronary syndromes, venous blood oxygen saturation has been used to identify patients at risk of heart failure or cardiogenic shock [9,10]. Few studies have clearly addressed the prognostic value of ScvO2 in ADHF, despite current guidelines recommending its use for monitoring drug therapy and studies suggesting its superior sensitivity over clinical markers [11,12]. ScvO2 reflects the balance between organ oxygen consumption and supply. A decrease in ScvO2 may be observed when oxygen consumption increases or when oxygen supply is inadequate (anaemia, hypoxaemia or low CO). In cardiogenic shock, the decrease in ScvO2 is commonly attributed to an inadequate oxygen delivery related to low CO [9]. ScvO2 has been suggested for monitoring CO to guide catecholamine titration. However, this approach is under debate and several studies have reported a lack of correlation between ScvO2 and CO in advanced heart failure patients [13–15]. Our results are consistent with these studies and we believe that the correlation between changes in ScvO2 during ADHF and CO may be hidden by the oxygen consumption reduction related to a decrease in organ congestion. This hypothesis is supported by the correlation observed between changes in ScvO2 and urine output, vena cava diameter and VO2 reduction. However, we cannot exclude that our data may be biased by the limitation of echocardiography in the assessment of CO.

Bilirubin, which reflects the severity of venous congestion in ADHF, has been found to be strongly associated with a worse outcome in our study and by others [16]. This underlines the deleterious impact of overload, as reported by Mullens et al. [17], who showed that cardiorenal syndrome in ADHF is not related to CO reduction, as commonly believed, but mainly to the severity of venous congestion. Consistently, indirect overload reduction markers (urine output and vena cava diameter reduction) correlated with a favourable outcome and improvement in ScvO2, probably through oxygen consumption reduction. This hypothesis is supported by Lautt et al., who showed that venous congestion induced by vena cava occlusion (cat model) reduces hepatic blood flow but is compensated by an increase in oxygen extraction [18]. The superiority of venous congestion reduction over CO restoration has been mainly reported in ADHF patients with preserved BP and might explain the lack of beneficial effect of dobutamine in these patients [19,20]. In cardiogenic shock, the use of inotropic drugs cannot be avoided because of the low BP and persistent anuria despite adequate diuretic support. However, the rate and target of catecholamine infusion remain unclear. Our study demonstrates that venous congestion reduction may be more important than CO restoration and suggests that ScvO2 should be used to monitor both catecholamine and diuretic rates in order to ensure a favourable response to diuretic and inotropic support.

**Study limitations**

Haemodynamic monitoring of cardiogenic shock is routinely performed by echocardiography and not by a Swan-Ganz catheter [21]. This may limit the interpretation of LV load

**Table 4** Predictor outcome in acute decompensated heart failure with cardiogenic shock.

<table>
<thead>
<tr>
<th></th>
<th>Univariate analysis</th>
<th>Multivariable analysis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No MACE (mean ± SD)</td>
<td>MACE (mean ± SD)</td>
</tr>
<tr>
<td>ScvO2 at 24 hours (per %)</td>
<td>65 ± 6</td>
<td>58 ± 7</td>
</tr>
<tr>
<td>Urine output (per L)</td>
<td>4.2 ± 1.9</td>
<td>2.9 ± 1.6</td>
</tr>
<tr>
<td>Bilirubin at 24 hours (per 10 μmol/L)</td>
<td>19 ± 10</td>
<td>29 ± 19</td>
</tr>
<tr>
<td>Lactates at 24 hours (per mmol/L)</td>
<td>1.6 ± 0.5</td>
<td>2.0 ± 0.6</td>
</tr>
<tr>
<td>Sodium at 24 hours (per mmol/L)</td>
<td>132 ± 4</td>
<td>129 ± 6</td>
</tr>
<tr>
<td>IVC at 24 hours (per mm)</td>
<td>18 ± 6</td>
<td>22 ± 5</td>
</tr>
</tbody>
</table>

CI: confidence interval; IVC: inferior vena cava; MACE: major cardiac events; OR: odds ratio; ScvO2: central venous oxygen saturation; SD: standard deviation.
pressure given the limited accuracy of echocardiography markers in this population [22]. However, the use of the vena cava diameter provides a fair assessment of venous congestion and right atrial pressure. In addition, despite an existing correlation, venous blood oxygen saturation from the superior vena cava and pulmonary artery may differ; these results should therefore be validated for venous oxygen saturation from pulmonary artery. Finally this study was done on a small sample of patients and its results — especially the 60% cut-off value — need to be validated in larger populations.

Conclusion

In ADHF requiring inotrope support, improvement in ScvO₂ to more than 60% at 24 hours appears to correlate with event free outcome. ScvO₂ improvement seems to be more related to vena cava diameter reduction and urine output than to CO improvement. In patients admitted for ADHF with cardiogenic shock, ScvO₂ ≤ 60% at 24 hours despite maximal doses of catecholamine and diuretic may be an indicator for the consideration of more aggressive therapeutic options.

Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

References