Central venous oxygen saturation: analysis, clinical use and effects on mortality

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ABSTRACT

Aims and Objectives: The aim of this literature review was to provide a clear definition of central venous oxygen saturation (ScvO2), highlight the differences between ScvO2 and mixed venous oxygen saturation (SvO2), show how it can be used clinically and the effect central venous oxygen saturation has on mortality.

Background: Many articles concentrate on the individual aspects of ScvO2, such as its use in early goal-directed therapy, but few provide a full overview of what it means, how to interpret results and how it can be used clinically.

Search strategies: Keywords were searched for including central venous oxygen saturation ScvO2 mixed venous oxygen saturations ScvO2 early goal-directed therapy sepsis and mortality. Where possible only publications within the last 10 years were used but key publications were not excluded if they were out with this time frame.

Conclusions: Central venous oxygen saturation (ScvO2) is a very important measurement which can be easily taken in a critical care environment by both medical and nursing staff. It provides an understanding of the patient’s oxygen delivery, oxygen consumption and cardiac output. It has a key role within early goal-directed therapy and has been shown to decrease mortality when taken and analysed appropriately.

Relevance to clinical practice: This literature review will highlight to nursing staff within the critical care environment the importance of central venous oxygen saturation measurement and interpretation. By raising awareness of the importance of this measurement it is hoped nursing staff will be proactive in both taking this test and analysing the results, therefore facilitating better care for the septic, critically ill patient and improving outcomes for these patients.

Key words: Advanced patient assessment • Clinical guidelines • Haemodynamic monitoring • Intensive care nursing • Resuscitation • Sepsis and shock

The aim of this literature review was to provide a clear definition of central venous oxygen saturation (ScvO2), highlight the differences between ScvO2 and mixed venous oxygen saturation (SvO2), show how it can be used clinically and the effect central venous oxygen saturation has on mortality. Many articles concentrate on the individual aspects of ScvO2, such as its use in early goal directed therapy, but few provide a full overview of what it means, how to interpret results and how it can be used clinically. To investigate this further, a literature search was carried out of existing articles using databases including the NHS Knowledge Network, OVID, CINAHL, the Cochrane Library and Medline. Articles were found by searching for keywords including central venous oxygen saturation, mixed venous oxygen saturation, sepsis, ScvO2, SvO2. Articles which were under 10 years old were included, however, articles which were pertinent to the topic were not excluded if out with this time frame.

INTRODUCTION

Central venous oxygen saturation (ScvO2) reflects the transport and metabolism of oxygen (Davidson et al., 2010; Pope et al., 2010; Haase and Perner, 2011; Textoris et al., 2011) and is a surrogate marker of mixed venous oxygen saturation but is simpler to measure (El Masry et al., 2009; Haase and Perner, 2011; Walley, 2011). It gives an indication of how much oxygen is extracted by the organs before the blood returns to the right side of the heart, showing the balance between oxygen delivery and consumption. This provides an indication of cardiac output and shows if the patient’s oxygenation needs are being met (Maddirala and Khan, 2010). ScvO2 can be used to measure cardiac output,
guide clinical practice when using early goal-directed therapy treatment protocols, understand and treat arterial hypoxaemia, rapidly estimate shunt fraction and help identify patients at risk of weaning failure (Teixeira et al., 2010; Haase and Perner, 2011; Walley, 2011). Tissue hypoxia suggested by ScvO2 can be an early marker of sepsis (Maddirala and Khan, 2010) as when oxygen delivery has been compromised or oxygen consumption has exceeded its supply, subsequent oxygen venous return is diminished (Davidson et al., 2010).

ScvO2 can be measured from a blood sample taken from the tip of a central venous catheter placed close to or within the right atrium (Maddirala and Khan, 2010; Walley, 2011).

Relationship between SvO2 and ScvO2

Mixed venous oxygen saturation (SvO2) is the amount of delivered oxygen left over after the tissues have consumed oxygen (Walley, 2011), obtained from blood samples from the pulmonary artery where venous blood is mixed after it has circulated through the superior vena cava, inferior vena cava, coronary sinuses and the right side of the heart. (Jesurum, 2004).

It is obtained from the distal port of a pulmonary artery catheter, with a normal SvO2 ranging from 65% to 75% (Davidson et al., 2010). ScvO2 has largely replaced SvO2 now as it is time consuming and challenging to insert a pulmonary artery catheter.

However, ScvO2 is only an approximation of SvO2 and this should be remembered when interpreting measurements (Chawla et al., 2004; Reinhart et al., 2004; Dueck et al., 2005; Walley, 2011).

Measuring ScvO2 has the potential for cost and time savings, and has fewer risks than pulmonary artery catheterization, which offsets the exact matching of O2 saturation values according to Reinhart et al. (2004).

Disadvantages to ScvO2

The ability of ScvO2 to reflect systemic oxygen delivery and consumption is dependent on many factors such as sedation, recent intubation (Hernandez et al., 2009) and the position of the catheter tip in the patient’s body (Vesely, 2003) therefore these factors may affect the interpretation (Haase and Ferner, 2011).

ScvO2 is only an approximation of SvO2 and this should be remembered when interpreting measurements (Dueck et al., 2005; Walley, 2011). This is supported by Chawla et al. (2004), who found that SvO2 was consistently lower than ScvO2 ($p = 0.0001$) with a mean bias of $-5.2 \pm 5.1\%$. Similarly, Rivers et al. (2001b) found that in patients in shock superior vena cava ScvO2 consistently overestimated the actual SvO2 value.

Reinhart et al. (2004) took continuous parallel measurements of both SvO2 and ScvO2 in 26 patients and found that in critically ill patients who have circulatory failure that ScvO2 is generally higher than SvO2. The findings of their study showed that ScvO2 values closely paralleled SvO2 but averaged about 7% ± 4% higher.

Kopterides et al. (2009) investigated the impact of placement of the tip of the central venous catheter on ScvO2. They found that when the tip was 15 cm away from the inlet of the right atrium, ScvO2 overestimated SvO2 by 8%. However, when the tip was advanced to the right atrium, ScvO2 became more accurate and only overestimated SvO2 by 1%.

Using a prospective observational multi-centre study Hernandez et al. (2009) showed that ScvO2 increases significantly following an emergency intubation in the majority of patients. Their data was collected over three university affiliated hospitals between December 2006 and March 2008, with a total of 103 emergency intubations analysed. Central venous samples were taken immediately before intubation and 15 minutes later and showed that although the ScvO2 may appear to rise it may not represent a true improvement in global dysoxia.

When should SvO2 be used instead of ScvO2?

The reliability of ScvO2 measurements depends on multiple factors including catheter placement, anatomy and the physiological state of the patient, therefore if an accurate mixed venous oxygen saturation is needed then there is no alternative than to insert a pulmonary artery catheter (Walley, 2011).

Varpula et al. (2006) carried out a prospective study of 16 patients with septic shock and found that mean SvO2 was always lower than mean ScvO2. They discovered significant variances between samples of SvO2 and ScvO2 which were taken at the same time and so conclude that ScvO2 should not be used as an estimate of SvO2 when treating a septic patient in Intensive Care following resuscitation.

Lorentzen et al. (2008) found through their prospective observational study of 20 patients going for elective cardiac surgery that ScvO2 could not replace SvO2, particularly in patients who were having aortic valve surgery. They found that the overall bias between ScvO2 and SvO2 was 1.9, the bias during coronary artery bypass surgery was 0.6 and during aortic valve replacement was 6.4. Therefore in the instance of
cardiac surgery is could be said SvO₂ should be used rather than ScvO₂.

Similarly, Lequeux et al. (2010) found that the difference between SvO₂ and ScvO₂ differed significantly during cardiac surgery which used cardiopulmonary bypass. Therefore they also suggest that ScvO₂ should not be used as a surrogate for SvO₂ in these patients.

FEMORAL SCVO₂
Davidson et al. (2010) conducted a study of 39 patients who had ScvO₂ and SfvO₂ taken simultaneously to assess if femoral-based central venous oxygen saturation was a reliable substitute for internal jugular or subclavian ScvO₂. They found ScvO₂ and SfvO₂ did not have a consistent correlation as 50% of measurements of ScvO₂ and SfvO₂ diverged by 5–15%. The use of SfvO₂ could therefore lead to unnecessary treatment such as the use of inotropes or blood transfusions.

The unreliability of femoral-based central venous oxygen saturation is supported by Walley (2011) who states that femoral venous blood is not mixed, is not downstream of any vital organs and so therefore cannot be used for anything other than to interpret oxygenation of the distal leg.

LEVELS OF SCVO₂
Low ScvO₂ may indicate a decrease in oxygen delivery, an increase in oxygen extraction or both (Pope et al., 2010). It is a valid therapeutic target in early septic shock and can be a diagnostic marker for low cardiac output with an excessive extraction of oxygen, low haemoglobin concentration or a low level of arterial oxygen pressure, cardiogenic shock and hypovolaemia (Maddirala and Khan, 2010; Textoris et al., 2011; Haase and Perner, 2011).

High levels of ScvO₂ can mean a very high oxygen delivery in excess of tissue requirements and/or decreased cellular consumption of oxygen and/or a large arterio-venous shunt (Textoris et al., 2011). This can be caused by sepsis, post cardiac arrest, distributive shock, high cardiac output, hypothermia or cellular poisons (Maddirala and Khan, 2010).

It has been suggested by Perz et al. (2011) than a low ScvO₂ is represented as less than 60-8% and high ScvO₂ as greater than 77-4%.

CLINICAL USE
ScvO₂ monitoring can have diagnostic and therapeutic uses in understanding the efficacy of interventions in treating critically ill, haemodynamically unstable patients (Maddirala and Khan, 2010) and has been shown to be a better indicator of tissue oxygenation and utilization of oxygen than routine observations (Rivers et al., 2001b).

The Surviving Sepsis Campaign (2008), organized by the European Society of Intensive Care Medicine, International Sepsis Forum and the Society of Critical Care Medicine, developed international guidelines for the management of severe sepsis and septic shock (Society of Critical Care Medicine, 2012). Within this ScvO₂ is seen to play a key role in the management of the critically ill, septic patient.

To maintain ScvO₂ it is recommended two strategies are used (Dellinger et al., 2008; Society of Critical Care Medicine, 2012).

The first strategy involves raising the central venous pressure to 8mmHg or above with fluid boluses. If the patient is still hypovolaemic and their haematocrit is less than 30%, these patients should be transfused with packed red cells. Dellinger et al. (2008) state the rationale for transfusion is that ScvO₂ may increase due to increased oxygen delivery to ischaemic tissue beds and will keep the central venous pressure greater than 8 mmHg for longer than fluids alone.

The second strategy is the use of inotropes. If the patient is well filled with a central venous pressure greater than 8 mmHg then cardiac output might not be enough to meet the metabolic demands of tissues despite an adequate circulating volume. Therefore a dobutamine infusion should be used to increase oxygen delivery to the peripheries and prevent ischaemia. If the dobutamine infusion causes hypotension then noradrenaline should be added in to counteract this (Society of Critical Care Medicine, 2012).

However, if the patient is mechanically ventilated, a higher target of a central venous pressure greater than 12 mmHg should be targeted. This is due to the PEEP the patient is receiving and the subsequent increase in intrathoracic pressure. Once this target has been achieved, inotropes should then be considered. This also applies to patients who have raised intra-abdominal pressures (Dellinger et al., 2008; Society of Critical Care Medicine, 2012).

Dellinger et al. (2008) see ScvO₂ as an important diagnostic test within early goal-directed therapy in septic patients. They state that ScvO₂ should be maintained above 70% within the first 6 hours of treatment once hypotension has been shown to be reactive to a fluid challenge. This recommendation was graded as 1C using the GRADE system which is a structured grading system for rating quality of evidence and grading strength of recommendation in practice (Lo-Biondo-Wood and Haber, 2010).
EFFECTS ON MORTALITY

The Surviving Sepsis Campaign (2008) was heavily influenced by Rivers et al. (2001a) who evaluated the effectiveness of early goal-directed therapy before admission to the intensive care unit using a prospective, randomized study. The study lasted 3 years (March 1997–March 2000) with a good sample size of 236, hence the results of this study can be seen to have external validity (Parahoo, 2006).

From the 236 patients 119 were treated with standard therapy and 117 received early goal-directed therapy. To avoid bias, the clinicians were not told of which group the patients had been assigned to, and so could not influence observations, recording of data or results (Parahoo, 2006).

Baseline recordings show that those assigned to the standard therapy group had a higher initial ScvO2, 49.2 ± 13.3% compared with the early goal directed therapy group of 48.6 ± 11.2%.

During the initial 6 hours after the start of treatment, patients receiving standard therapy had a significantly lower ScvO2 than those patients receiving early goal-directed therapy, 65.3 ± 11.4% compared with 70.4 ± 10.7%. Similarly, between the 7 and 72 hour period following the beginning of therapy, patients receiving standard therapy continued to have a significantly lower ScvO2 than those receiving early goal-directed therapy.

Overall, those patients receiving standard therapy had a higher in hospital mortality rate, 28 day mortality rate, 60 day mortality rate and increased length of hospital stay than those receiving early goal-directed therapy. This suggests that using ScvO2 has an important role to play in the treatment of a septic patient and along with other factors such as mean arterial pressure and central venous pressure can provide significant outcome benefit for patients with severe sepsis and septic shock over standard therapy (Rivers et al., 2001a).

Pope et al. (2010) conducted a study which built upon the findings of Rivers et al. (2001a) and aimed to show that abnormal ScvO2 led to increased mortality. Although this study had a sample size of 619 patients, which was more than double that of Rivers et al. (2001a), it could be said that Pope et al. (2010) had a weaker study. They used secondary analysis, hence did not play a part in collecting the data, therefore there may be deficiencies in the data used (Polit and Beck, 2010).

Two measurements were recorded; the initial ScvO2 taken immediately after central line insertion and the maximum ScvO2 (ScvO2max) described as the highest measurement recorded for each individual during the first 6 hours of early goal-directed therapy. Parameters for ScvO2 levels were set as, 0–70% = hypoxia, 70–89% = normoxia and 90–100% = hyperoxia.

The mean initial ScvO2 of all 619 patients was 73% with an initial mortality rate for normoxia calculated as 23%, hypoxia being 25% and hyperoxia was 31% mortality. Those who stayed normoxic throughout the 6 hours had a mortality rate of 22%, those who stayed hypoxic had a mortality rate of 40% and those who stayed hyperoxic had a mortality rate of 31%. Hypoxia which normalized had a mortality rate of 19% and those who became hyperoxic had a mortality rate of 33%.

With early goal-directed therapy the ScvO2 increased from 58% to 73% in the hypoxic group, stayed the same (93%) in the hyperoxic group and rose from 79% to 82% in the normoxic group. From these results, it can be said that by increasing ScvO2 in the hypoxic group to a normal level, mortality was more than halved.

The findings of Pope et al. (2010) are supported by Textoris et al. (2011). They found that mortality rates were 30% when ScvO2max was less than 80% and over 80% the mortality rate was 48% in patients treated using early goal-directed therapy.

Textoris et al. (2011) recorded maximum and minimum ScvO2 levels over a 72 hour period, however, continuous sampling was not carried out and inclusion into the study only required two ScvO2 samples to be taken over this period. Therefore, important fluctuations in ScvO2 may have been missed and the minimum and maximum recordings may not be entirely accurate. Textoris et al. (2011) excluded patients from the study who were in intensive care for less than 24 hours, therefore creating a selection bias. These patients would either be the healthiest patients who would be discharged to a lower level of care, or the sickest patients who did not survive the first 24 hours of their admission. In either case, these results could have had a significant impact on the study and their findings.

Unlike other studies, Textoris et al. (2011) state that the position of the central venous catheter was confirmed to be in the superior vena cava by chest X ray, therefore recordings can be said to be uniform as they were taken from the same site in all patients.

From this study, Textoris et al. (2011) suggest that ScvO2 greater than 80% in the first 72 hours of resuscitating septic shock patients can increase mortality. However, to make these results more robust, a prospective study should be carried out, as the retrospective design of this study relied upon data already collected, which was not collected for research purposes, hence lacks rigour (Polit and Beck, 2010). Furthermore, ScvO2 should be measured continuously, which would improve external validity (Lo-Biondo-Wood and Haber, 2010), and allow the findings to be extrapolated.
In contrast to Textoris et al. (2011), it was found by Bracht et al. (2007) that a ScvO₂ of less than 60% during an unplanned admission to intensive care was associated with high mortality. Interestingly, standard Intensive Care treatment increased but did not normalize the ScvO₂ in those patients who presented initially with a low ScvO₂. This was shown by analysing 98 consecutive admissions, requiring a central venous catheter, into the intensive care unit over a 3 month period. Patients who did not require a central venous catheter or who would refuse blood products were excluded, therefore a large quantity of study participants and recordings were missed \( (n = 248) \). Within 2 hours of insertion of this line, a ScvO₂ was taken and was repeated every 6 hours. This study did not use early goal-directed therapy and therefore those providing treatment were blind to the results. The same analyser was used for all samples, ensuring accuracy of results.

Bracht et al. (2007) found that patients with an ScvO₂ of 60% or lower on admission had a higher 28 day mortality rate than those who had a ScvO₂ of greater than 60% on admission. This was analysed using Fishers exact test, which was an appropriate choice as it works best when the sample size is smaller (Polit and Beck 2010). The average ScvO₂ was 70 ± 12% on admission, and increased to 71 ± 10% 6 hours later however there was a significant increase in those patients with a baseline ScvO₂ less than 60% with their values rising from 52% ± 5% to 63% ± 9%.

**CONCLUSION**

In conclusion, the author found that ScvO₂ is a very important measurement, which can be easily taken by nursing and medical staff. It will help to provide an understanding of the patient’s oxygen delivery, oxygen consumption and cardiac output. When used within early goal-directed therapy, it has been shown to decrease mortality, however, the author noted that care must be taken to understand the impact of decreased and increased ScvO₂, as both can have impact on mortality.

**WHAT IS KNOWN ABOUT THIS TOPIC**

- It is known that central venous oxygen saturation is a significant measurement in the treatment of septic patients which can guide treatment.
- It is known that both a high and low central venous oxygen saturation can lead to poor outcomes.
- Central venous oxygen saturations are taken commonly within Intensive Care Units and can be used to influence treatment decisions.

**WHAT THIS PAPER ADDS**

- By presenting a literature review, this article provides a comprehensive guide to central venous oxygen saturation in a single article highlighting both the advantages and disadvantages of monitoring central venous oxygen saturation.
- It explains the basic principles of central venous oxygen saturation in a simple, easy to understand format, which should improve nurses’ knowledge and practice.
- This article emphasizes why central venous oxygen saturations are taken from patients within Intensive Care Units specifically and how this influences the treatment for these patients.
- This article highlights the distinct differences between ScvO₂ and SvO₂.

**REFERENCES**


