

Central Venous Oximetry – A Simple Soothsayer for the Complicated Acute Coronary Syndrome?

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Acute coronary syndrome (ACS) complicated with acute pulmonary edema or cardiogenic shock remains to be a great clinical challenge to cardiologists, even though the inception of many newly introduced treatment modalities has significantly improved the clinical outcome.^{1,2} A reliable and simple monitoring system is important for being able to closely follow the clinical progression of the disease, determining how intensively we should treat the patients, and thus, potentially improving the clinical result. Although many scoring systems have been introduced to predict the clinical outcomes of ACS,^{3,4} a simple and easily-available way for the close monitor of clinically-variable courses of the complicated ACS is yet to be established.

The saturation of central venous oxygen (ScVO₂) has been used as a surrogate of the saturation of mixed venous or pulmonary arterial oxygen (SmVO₂),⁵ which in general, mirrors the condition of overall tissue perfusion when arterial oxygen content and oxygen consumption remain constant.⁶ As early as in late 1960s, the drop of ScVO₂ had been proposed as a good indicator for the development of congestive heart failure (CHF) and cardiogenic shock after acute myocardial infarction;^{5,7,8} on the contrary, a return of the ScVO₂ level denotes a good treatment response.^{5,8} In some cases, interestingly, the change of ScVO₂ levels is even far ahead of the clinical deterioration or improvement.⁸ Given that ScVO₂ is a more easily available clinical data than SmVO₂ or cardiac index in terms of technical demands, serial ScVO₂ determinations could be a good index for monitoring the situation of tissue perfusion in ACS patients, especially for those with a Killip class of III or IV.

Nevertheless, there has been no direct evidence heretofore showing that ScVO₂-guided ACS treatment achieves a better clinical outcome. Surprisingly, ScVO₂ has been set as an early goal in the treatment of the critically-ill patients with sepsis.⁹⁻¹¹ Rivers, et al.¹² clearly

demonstrate that the strategy with an additional early goal to achieve ScVO₂ > 70% results in a significantly better outcome in patients with severe sepsis, as compared with the traditional strategy alone (i.e., to keep a central venous pressure between 8 and 12 mmHg, a urine output more than 0.5 ml/kg/min and a mean arterial pressure higher than 65 mmHg). An evidence-based consensus even shows that the routine augmentation of cardiac index, a commonly used outcome-predicted parameter, to a predefined elevated level in septic patients is not helpful and thus not recommended,¹⁰ implying that ScVO₂ might be more outcome-informative than the cardiac index and therefore a better guide in the treatment of patients with sepsis.

In this issue of *Acta Cardiologica Sinica*, Hsin, et al.¹³ for the first time demonstrate that, in patients with complicated ACS, the event-survivors have a significantly higher event-ScVO₂ level than that in the event-non-survivors, and that there is a trend of increased ScVO₂ levels from the subsequent serial check-ups in the event-survivors, in contrast to a trend of the decreased levels in the event-non-survivors, implying that the event-ScVO₂ levels and the subsequent change of ScVO₂ (Δ ScVO₂) might divulge the survival information for patients with complicated ACS, and thus, are potentially ideal treatment goals. However, owing to the extremely small case number of this pilot study, it is impossible to analyze whether an event- ScVO₂ level provides more outcome information than the traditional APACHE II or TISS score, not to mention the possibility of showing the impacts of different treatment modalities on the changes of ScVO₂ levels and their clinical outcomes. Furthermore, there are still some unsolved limitations concerning ScVO₂ *per se*. Firstly, ScVO₂ has been reported to be a poor surrogate of SmVO₂, especially in shock patients, whose blood supply is significantly re-distributed to maintain cerebral perfusion. As a result,

the saturation of superior vena cava is significantly higher than the $SmVO_2$.¹⁴ In this regard, saturation of right atrium is more likely to reflect $SmVO_2$ and thus may be a more ideal parameter.¹⁴ Secondly, the baseline values of $ScVO_2$ differ individually due to distinct levels of individual O_2 consumption.⁷ The $\Delta ScVO_2$ has therefore been reported to be a more reliable indicator than $ScVO_2$ in the monitoring of critically-ill patients.¹⁴

Taken together, although $ScVO_2$ has the merit of its easy accessibility and does show its potential in the prediction of clinical outcomes for patients with complicated ACS, it is still too early for now to declare its clinical usefulness. A large-scaled, prospective, randomized clinical trial to test whether $ScVO_2$ -directed treatment would render a better clinical outcome than the traditional treatment strategy for patients with complicated ACS is of great interests and may provide invaluable information for the development of a better therapeutic guideline.

REFERENCES

1. Cleland JG, Coletta AP, Clark AL. Clinical trials update from the American College of Cardiology 2007: ALPHA, EVEREST, FUSION II, VALIDD, PARR-2, REMODEL, SPICE, COURAGE, COACH, REMADHE, pro-BNP for the evaluation of dyspnoea and THIS-diet. *Eur J Heart Fail* 2007;9:740-5.
2. Cleland JG, Coletta AP, Abdallah AT, et al. Clinical trials update from the American Heart Association 2007: CORONA, RethinQ, MASCOT, AF-CHF, HART, MASTER, POISE and stem cell therapy. *Eur J Heart Fail* 2008;10:102-8.
3. Morrow DA, Antman EM, Charlesworth A, et al. TIMI Risk Score for ST-Elevation Myocardial Infarction: a convenient, bedside, clinical score for risk assessment at presentation: an intravenous nPA for treatment of infarcting myocardium early II trial substudy. *Circulation* 2000;102:2031-7.
4. Antman EM, Cohen M, Bernink PJLM, et al. The TIMI Risk Score for Unstable Angina/Non-ST Elevation MI: A Method for Prognostication and Therapeutic Decision Making. *JAMA* 2000; 284:835-42.
5. Goldman RH, Braniff B, Harrison DC, Spivack AP. The use of central venous oxygen saturation measurements in a coronary care unit. *Ann Intern Med*. 1968;68:1280-7.
6. Valentine PA, Fluck DC, Mounsey JP, et al. Blood-gas changes after acute myocardial infarction. *Lancet* 1966;2:837-41.
7. Goldman RH, Klughaupt M, Metcalf T, et al. Measurement of central venous oxygen saturation in patients with myocardial infarction. *Circulation*. 1968;38:941-6.
8. Hutter AM, Jr., Moss AJ. Central venous oxygen saturations. Value of serial determinations in patients with acute myocardial infarction. *JAMA*. 1970;212:299-303.
9. Rivers EP, Ander DS, Powell D. Central venous oxygen saturation monitoring in the critically ill patient. *Curr Opin Crit Care*. 2001;7:204-11.
10. Rhodes A, Bennett ED. Early goal-directed therapy: an evidence-based review. *Crit Care Med*. 2004;32:S448-50.
11. Gunn SR, Fink MP, Wallace B. Equipment review: the success of early goal-directed therapy for septic shock prompts evaluation of current approaches for monitoring the adequacy of resuscitation. *Crit Care*. 2005;9:349-59.
12. Rivers E, Nguyen B, Havstad S, et al. Early Goal-Directed Therapy in the Treatment of Severe Sepsis and Septic Shock. *N Engl J Med* 2001;345:1368-77.
13. Hsin HT, Li AH, Yeih DF, et al. The application of oxygen saturation of central venous blood ($ScVO_2$) in complicated acute coronary syndrome as a probable disease monitor - a pilot study. *Acta Cardiol Sin* 2008;24:126-33.
14. Scheinman MM, Brown MA, Rapaport E. Critical assessment of use of central venous oxygen saturation as a mirror of mixed venous oxygen in severely ill cardiac patients. *Circulation*. 1969;40: 165-72.