Commentary

Central venous oxygen saturation and emergency intubation - another piece in the puzzle?

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Abstract

A recent multicentre observational study examined the effect of emergency intubation on central venous oxygen saturation ($Scvo_2$) in critically ill patients. The main finding was that $Scvo_2$ significantly increases 15 minutes after emergency intubation and institution of mechanical ventilation with 100% oxygen, especially in those patients with pre-intubation $Scvo_2$ values <70%, regardless of whether these patients suffered from severe sepsis. However, in only one-quarter of this subgroup was the $Scvo_2$ normalized to $\geq 70\%$ solely by this intervention. In contrast, in patients with pre-intubation $Scvo_2 \geq 70\%$, the $Scvo_2$ failed to increase after intubation. A rise in $Scvo_2$ can be expected when whole body oxygen extraction remains unchanged after intubation and ventilation with pure oxygen.

The value of central venous oxygen saturation (Scvo₂) remains a matter of debate, and Hernandez and coworkers [1] have contributed another piece of the puzzle, with their study on the effects of emergency intubation on Scvo₂ in critically ill patients.

Since the work presented a few years ago by Rivers and colleagues [2], 'early goal-directed therapy' (EGDT), which they found to be associated with significantly improved outcome in sepsis, has been considered as a fundamental strategy in the initial management of sepsis and septic shock. In principle, the aim of this strategy is to achieve predefined targets in early resuscitation (during the first 6 hours) for mean arterial pressure, central venous pressure and Scvo₂. The measured parameters serve as surrogates in goaldirected therapy, which aims to balance tissue oxygen supply and demand. The presence of a low Scvo2 level in patients with early sepsis portends increased morbidity and mortality, and Rivers and colleagues found that correcting this value in accordance with the EGDT algorithm improved morbidity and mortality. These findings refuelled interest in the measurement of Scvo₂ in critically ill patients.

In the previous issue of *Critical Care*, Hernandez and coworkers [1] reported beneficial effects of emergency intubation on Scvo₂ in a multicentre study including 108 critically ill patients suffering from different types of septic and nonseptic conditions. About 50% of the patients presented with sepsis, which is similar to the proportions in the study conducted by van Beest and colleagues [3] and our own observations [4,5].

The study employed a simple design, made necessary by the emergency situation under which it was conducted. Basically, patients presenting with critical conditions were intubated and mechanically ventilated with pure oxygen. Scvo, was measured twice: immediately before intubation and after 15 minutes of mechanical ventilation. In the subgroup of patients with initial Scvo₂ <70%, a significant improvement in this parameter was observed. Interestingly, this is the second investigation outside Europe to report low initial Scvo values, which is in contrast to the reports by van Beest and colleagues [3] and Bracht and coworkers [4,5]. In fact, these two studies reported substantially higher initial Scvo2 values as compared with the study by Hernandez and coworkers [1] and the EGDT study conducted by Rivers and colleagues [2]. which led to growing concerns about the relentless pursuit of normalizing impaired Scvo2 during sepsis, because patients with low initial Scvo₂ are fairly scarce [6].

Nevertheless, the data reported by Hernandez and coworkers [1] are important and encouraging. In fact, the original concept proposed by Rivers and colleagues [2] was mainly based on stabilization of haemodynamics; in contrast, the primary goal of Hernandez and coworkers was to improve arterial oxygenation by adequate respiratory support, which naturally resulted in improved peripheral oxygenation. Thus, some important questions arise from these data. First, the

effects of mechanical ventilation are rather unpredictable, especially in severely compromised haemodynamic states. Indeed, the lack of precise data on haemodynamics may be considered a major weakness of the study. However, the intention was to investigate the immediate effects of early intubation in an emergency situation, and under these conditions haemodynamic data that require invasive cardiovascular monitoring are not immediately available. However, although the authors report rather low peripheral oxygen saturations and a high respiratory rate before intubation, neither blood carbon dioxide values nor the exact criteria for intubation or procedures to avoid intubation are reported. Within this context, it is conceivable that in the given respiratory situation most of the patients did profit from intubation in terms of Scvo2, but - strikingly - in patients with low Scvo₂ before intubation this was true only in 25%.

So what could be the reasons for this interesting finding? From a physiological point of view, it is likely that the increase in Scvo, after intubation is caused by a combination of two mechanisms. First, systemic oxygen delivery increased before intubation because of pre-oxygenation with 100% oxygen, which results in a large amount of physically dissolved oxygen [7]. Secondly, sedation and paralysis required for intubation most likely lower systemic oxygen demand and thus increase Scvo₂ via a net decrease in oxygen extraction ratio. These two mechanisms should outweigh the potential disadvantages of mechanical ventilation in terms of cardiovascular homeostasis [8]. Moreover, changes in acid-base status may affect the Scvo₂ response [9]: initiating mechanical ventilation may cause a fall pH due to the relief afforded by hyperventilation, and consequently the haemoglobin dissociation curve might be shifted to the right, which would result in a less pronounced increase in Scvo2. Finally, because the overall status of the patients is not entirely clear from the data presented, it is still possible that some patients had an underlying oxygen supply/demand dependency, so that any increase in oxygen delivery was accompanied by an increase in consumption, and consequently unchanged Scvo₂. Unfortunately, most of these physiological variables were not presented, so the reasons for the above-mentioned striking findings remain a matter of speculation. Not all patients staying in the intensive care unit (ICU) or after unplanned ICU admission had a central venous catheter in place, a fact that certainly might have excluded a number of patients with acute respiratory failure admitted to the ICU.

In conclusion, the authors elegantly demonstrate that emergency intubation may allow Scvo_2 to be improved in septic and nonseptic patients within 15 minutes. The data also clearly show that a resuscitation bundle - as postulated by EGDT - is necessary to achieve the target $\text{Scvo}_2 \geq 70\%$. Still, as confirmed by several studies, the incidence of low Scvo_2 will stay a matter of debate.

Competing interests

The authors declare that they have no competing interests.

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