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Hemodynamic coherence: a metabolic perspective

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In recent years, the concept of hemodynamic coherence - and its disruption - has gained increasing attention in the critical care community. Hemodynamic coherence refers to the synchronized response between the macrocirculation and the microcirculation, and it assumes that improvements in systemic circulation will lead to proportional enhancements in microcirculatory perfusion [1, 2]. However, it is important to note that under normal, healthy conditions, the macrocirculation and the microcirculation are in fact uncoupled. Meaning that tissue beds regulate their own blood flow according to their metabolic needs through vascular autoregulatory (AR) mechanisms and are, therefore, uncoupled (and protected) from hemodynamic variations. These tissue beds will only become coupled to the macrocirculation in pathological states where AR mechanisms are insufficient to compensate for deep hemodynamic disturbances (such as severe hypotension and/or impaired global blood flow) or in those situations where AR is compromised (in sepsis conditions, for instance), making the tissue bed more vulnerable and dependent on global hemodynamic changes. Therefore, it is important to recognize that hemodynamic coherence reflects a disease state.

In clinical practice, hemodynamic coherence becomes our only window to improve tissue wellness by manipulating macrocirculatory parameters, such as perfusion pressure and cardiac output, through the process known as hemodynamic resuscitation. When hemodynamic coherence holds true, interventions based on systemic targets can be considered effective for improving the microcirculation. At some point, the resuscitation process should be discontinued when hemodynamic coherence is lost again, which often signifies that tissues have reached their maximum oxygen extraction capacity. Importantly, this does not necessarily indicate a return to physiological normality, but rather the exhaustion of the benefit of resuscitative measures. This may result from secondary effects of resuscitation, such as tissue edema or capillary congestion, or to underlying mitochondrial dysfunction. In these situations, further efforts to enhance tissue perfusion, regardless of the intervention, may be harmful and are likely to worsen patient outcomes.

Since macrocirculatory indicators alone may miss ongoing tissue-level oxygen deficits, potentially contributing to silent organ dysfunction, there is growing interest in integrating microvascular monitoring into critical care [3]. Emerging technologies now enable assessment of the microcirculation, either through direct visualization, such as sublingual videomicroscopy, or through measurement of tissue oxygenation status using tools like near-infrared spectroscopy (NIRS).

Although some authors advocate for a framework of resuscitation strategies involving tailored interventions based on the observation of the microcirculation, the clinical utility of these monitoring tools will remain also limited by the lack of effective therapies specifically targeting the microcirculation. Notably, current interventions are likely to be effective only during the transient pathological state of macro–microvascular coherence. Microvascular monitoring, therefore, may be most valuable in identifying these brief windows of coherence more accurately, when interventions can still positively influence the microcirculation. However, once coherence

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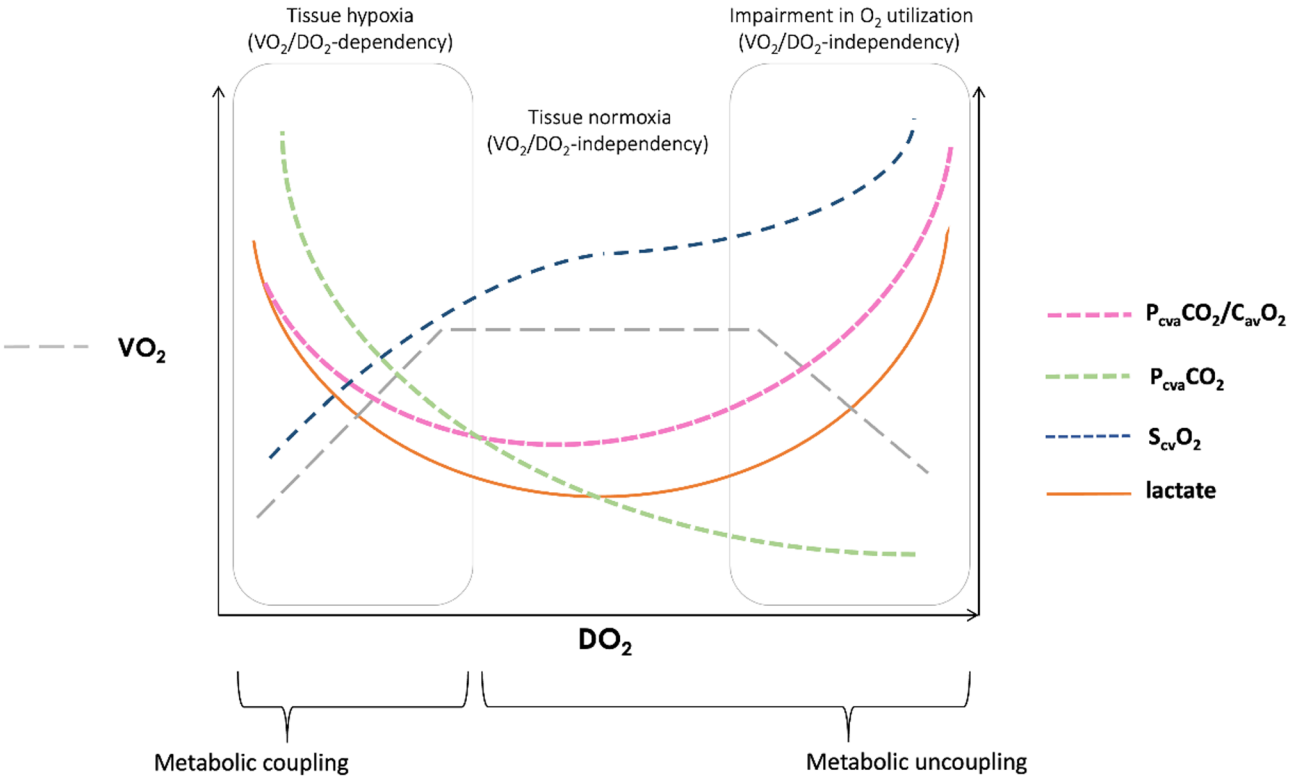

Fig. 1 Oxygen delivery (DO_2), oxygen consumption (VO_2) and metabolic markers during resuscitation. The nature of the VO_2/DO_2 relationship determines whether the metabolic response to increased perfusion is coupled, or uncoupled, which may reflect either a restored VO_2/DO_2 balance or impaired tissue oxygen utilization. Importantly, relying solely on lactate levels may prompt unnecessary or even harmful efforts to further increase DO_2. A combined assessment of multiple metabolic markers provides a more accurate understanding of the patient's baseline metabolic profile at the bedside. *VO_2*, Oxygen consumption; *DO_2*, Oxygen delivery; *$S_{cv}O_2$*, central venous oxygen saturation; *$PcvaCO_2$*, central venous-to-arterial carbon dioxide difference; *$PcvaCO_2/CavO_2$*, Central venous-to-arterial carbon dioxide difference combined with arterial-to-venous oxygen content difference.

is lost, these tools do not restore our ability to intervene effectively, other than by helping us detect when further resuscitation is likely futile or harmful.

Can we evaluate hemodynamic coherence at the bedside?.

Recognizing the limits of hemodynamic coherence fundamentally challenges our current approach to hemodynamic resuscitation. In the absence of robust evidence and broader integration of microcirculatory monitoring into clinical practice, clinicians must still rely on indirect surrogates of global tissue perfusion and oxygenation to assess the impact of hemodynamic interventions at the tissue level. Traditional global indicators such as oxygen delivery (DO_2) often fail to accurately reflect the actual oxygen reaching the tissues. Moreover, regardless of our capacity to transport oxygen to the tissues, the ultimate goal is to restore the balance between oxygen delivery and the metabolic demand. Therefore, parameters reflecting the relationship between DO_2 and oxygen consumption (VO_2) may offer a more accurate assessment of the adequacy of tissue perfusion. Given the challenges of bedside measurement, DO_2 and VO_2 are not routinely monitored, and various metabolic surrogates have been proposed to evaluate the VO_2/DO_2 relationship.

From a metabolic perspective, hemodynamic coherence during resuscitation would be described by the presence of VO_2 dependency on DO_2, a state in which increases in oxygen delivery after hemodynamic interventions are accompanied by corresponding increases in oxygen consumption (Fig. 1). Considering the lack of a reliable single metabolic marker for detecting VO_2/DO_2 dependency, a multimodal, integrative strategy, leveraging multiple complementary parameters, holds greater promise for the accurate assessment of tissue hypoxia [4]. This is not a minor issue, as recent recommendations have omitted physiologically relevant metabolic parameters, such as venous oxygen saturations, and many prospective resuscitation trials rely on control groups in which therapeutic goals are guided exclusively by lactate kinetics as the sole metabolic endpoint [5, 6]. Conversely, the integration of venous oxygenation and carbon dioxide-derived parameters has demonstrated stronger performance in detecting VO_2/DO_2 dependency [4, 7, 8].

Finally, several studies have emphasized the link between microcirculatory and metabolic parameters,

extending beyond traditional macrocirculatory metrics. Ospina-Tascón et al. found that the central venous-to-arterial CO_2 difference ($P_{cva}CO_2$) closely correlates with microvascular perfusion, more so than cardiac output [9]. $P_{cva}CO_2$ also aligns with tissue oxygen saturation (StO_2) measured by NIRS [10]. Furthermore, combining $P_{cva}CO_2$ with the arterial-to-venous oxygen content difference ($C_{av}O_2$) has been associated with NIRS-based markers of local metabolism and may help identify ongoing anaerobic metabolism [10].

While the conceptual transition from a macro-micro to a macro-metabolic framework for understanding hemodynamic coherence is not without its limitations, a thorough metabolic assessment at the bedside may still represent our most practical means of detecting when hemodynamic interventions fail to translate into meaningful improvements at the tissue level.

In summary, hemodynamic coherence represents a pathological state during which clinicians may still be able to improve microcirculatory perfusion through hemodynamic interventions. Microcirculatory monitoring technologies may prove useful for a more precise recognition of this coupling between macro and microvascular dynamics. Ultimately, the loss of hemodynamic coherence indicates the exhaustion of our available strategies to rescue the microcirculation, marking a point at which the priority shifts toward avoiding further harm to the patient. While more advanced monitoring tools are gradually becoming available, the use of combined metabolic parameters may currently assist clinicians in recognizing the loss of hemodynamic coherence.

Abbreviations

$C_{av}O_2$	Arterial-to-venous oxygen content difference
DO_2	Global oxygen delivery
NIRS	Near-infrared spectroscopy
$PcvaCO_2$	Central venous-to-arterial carbon dioxide difference
$PcvaCO_2/CavO_2$	Central venous-to-arterial carbon dioxide difference combined with arterial-to-venous oxygen content difference
$ScvO_2$	Central venous oxygen saturation
StO_2	Tissue oxygen saturation
VO_2	Global oxygen consumption

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