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Can transcutaneous oxygen saturation be used to detect tissue hypoperfusion?

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Sir: Although considered to be extremely important in the management of critically ill patients, only limited techniques are clinically available for the assessment of tissue oxygenation. Parameters of global oxygen delivery (i.e., cardiac index), systemic oxygen balance (i.e., central venous oxygen saturation, ScvO₂) [1], and anaerobic metabolism (i.e., serum lactate) [2] are widely used; however, more detailed information on regional perfusion/function would be desirable for guiding treatment. In particular, monitoring of the hepatosplanchnic tract can be obtained by the indocyanine green plasma disappearance rate (ICG-PDR) [3]. More

recently, transcutaneous assessment of muscle oxygen saturation (StO₂) has become available and may be a reliable predictor of MODS in trauma patients [4]; however, StO₂ was described not to differ in septic patients (even in non-survivors) from healthy volunteers, even though vasoreactivity may be different [5]. In this case, we present a patient in whom we measured global and non-invasively obtainable regional parameters during two typical clinical interventions, i.e., fluid resuscitation, in order to optimize cardiac output by Frank–Starling mechanism as well as inotropic support.

A 51-year-old woman with small bowel perforation and peritonitis developed septic shock. Echocardiography found mitral valve insufficiency caused by endocarditis. Due to requirement of norepinephrine (0.05 µg/kg min⁻¹), hemodynamic monitoring was extended by the transpulmonary thermodilution technique (PiCCO, Pulsion Medical Systems, Munich). Using this system, cardiac index, cardiac filling, and extravascular lung water were measured. In addition, we used a transcutaneous finger clip system for measurement of ICG-PDR (LiMON, Pulsion Medical Systems,

Munich, Germany) and a NIRS sensor placed on the right thenar (Inspectra, Hutchinson Technology, Arnhem, The Netherlands) for assessment of StO₂. Furthermore, we measured ScvO₂ and serum lactate at each time point. When intravascular fluid depletion was initially confirmed, first 500 ml of Ringer's solution was infused before a control measurement was obtained 30 min later. Then dobutamine (4 µg/kg min⁻¹) was added and measurements were repeated again 30 min after beginning this treatment. Ventilator settings were unchanged during the time period (BiPAP, FiO₂ 0.45, PEEP 10 mbar, P_{insp} 30 mbar, I:E 1:2, respiratory rate 20/min, minute volume 12.6 l/min). Data are summarized in Table 1. Hemoglobin content was unchanged as no blood was transfused.

In this patient, we found that initially abnormal StO₂ allowed non-invasive detection of a (severely) disturbed hemodynamic state. Fluid challenge markedly improved regional (StO₂, ICG-PDR) variables without significant impact on global hemodynamics (i.e., cardiac index, mean arterial pressure); however, ScvO₂ also increased, and this finding may be interpreted as some local improvement in muscle oxygenation

Table 1 Global and regional parameters. HR, heart rate; MAP, mean arterial pressure; CVP, central venous pressure; PaO₂, arterial oxygen pressure; PaCO₂, arterial carbon dioxide pressure; pHa, arterial pH; EVLWI, extravascular lung water index; ScvO₂, central venous oxygen saturation; StO₂, transcutaneous muscle oxygen saturation; ICG-PDR, indocyanine green plasma disappearance rate

	Baseline ^a	Fluid challenge ^b	Dobutamine ^c
HR (1/min)	65	65	80
MAP (mmHg)	63	59	66
CVP (mmHg)	5	8	7
PaO ₂ (mmHg)	159	168	119
PaCO ₂ (mmHg)	37	38	41
pHa	7.43	7.42	7.43
Cardiac index (l/min m ⁻²)	1.22	1.33	1.76
Stroke index (ml/m ²)	19	20	22
EVLWI (ml/kg)	15	16	16
Lactate (mmol/l)	1.3	1.2	1.4
ScvO ₂ (%)	48	55	56
StO ₂ (%)	51	90	98
Tissue hemoglobin index (%)	10.4	12.2	11.2
ICG-PDR (%/min)	3.9	4.5	6.0

^a Baseline = before starting fluid and dobutamine administration

^b Fluid challenge = after administration of Ringer's solution

^c Dobutamine = after starting administration of dobutamine

rather than an increase in total blood flow. The StO_2 rapidly normalized, however, even though the other hemodynamic variables (cardiac index, ScvO_2 , and ICG-PDR) were still profoundly abnormal. These data suggest that a normal StO_2 may not be reassuring, and this is a major limitation for using this variable for guiding resuscitation. We then added dobutamine, by which heart rate and cardiac index, but not stroke volume, further increased. Interestingly, ICG-PDR increased even more than before, whereas StO_2 and ScvO_2 only slightly further increased.

In conclusion, we found parallel changes in ScvO_2 , ICG-PDR, and StO_2 in a patient with septic shock during fluid resuscitation and inotropic therapy; however, normalization of StO_2 in the presence of still-altered other global and regional variables questions whether or not StO_2 is a sensitive indicator of hemodynamic alteration (even regional) perfusion. Larger studies are needed to study the mechanisms involved and to emphasize the usefulness of StO_2 in critically ill patients.

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