

REVIEW

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Near infrared spectroscopy (NIRS) of the thenar eminence in anesthesia and intensive care

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Abstract

Near infrared spectroscopy of the thenar eminence (NIRS_{th}) is a noninvasive bedside method for assessing tissue oxygenation. The NIRS probe emits light with several wavelengths in the 700- to 850-nm interval and measures the reflected light mainly from a predefined depth. Complex physical models then allow the measurement of the relative concentrations of oxy and deoxyhemoglobin, and thus tissue saturation (StO₂), as well as an approximation of the tissue hemoglobin, given as tissue hemoglobin index.

Here we review of current knowledge of the application of NIRS_{th} in anesthesia and intensive care.

We performed an analytical and descriptive review of the literature using the terms "near-infrared spectroscopy" combined with "anesthesia," "anesthesiology," "intensive care," "critical care," "sepsis," "bleeding," "hemorrhage," "surgery," and "trauma" with particular focus on all NIRS studies involving measurement at the thenar eminence.

We found that NIRS_{th} has been applied as clinical research tool to perform both static and dynamic assessment of StO₂. Specifically, a vascular occlusion test (VOT) with a pressure cuff can be used to provide a dynamic assessment of the tissue oxygenation response to ischemia. StO₂ changes during such induced ischemia-reperfusion yield information on oxygen consumption and microvasculature reactivity. Some evidence suggests that StO₂ during VOT can detect fluid responsiveness during surgery. In hypovolemic shock, StO₂ can help to predict outcome, but not in septic shock. In contrast, NIRS parameters during VOT increase the diagnostic and prognostic accuracy in both hypovolemic and septic shock. Minimal data are available on static or dynamic StO₂ used to guide therapy.

Although the available data are promising, further studies are necessary before NIRS_{th} can become part of routine clinical practice.

Introduction

Oxygen delivery (DO₂) and consumption (VO₂) often are disturbed in critically ill patients [1]. Such disturbance may lead to pathological changes in tissue oxygenation. Thus, monitoring of *tissue oxygenation* appears desirable. Invasive monitoring of systemic DO₂ and VO₂ has been used in intensive care medicine for decades [2]. In contrast, no method for assessing tissue oxygenation has yet gained widespread clinical use. This is unfortunate, because tissue oxygenation may reflect changes in the microcirculation, a similarly important target for therapy during major surgery and in the critically ill. Disturbances in microcirculation are common and well documented in hemorrhage and

critical illness [3,4], and they logically relate to tissue oxygenation. Thus, monitoring of tissue oxygenation may provide useful information not only about the state of tissue oxygenation itself but probably about the state of the microcirculation. A potentially useful method to monitor tissue oxygenation may be offered by near infrared spectroscopy (NIRS)-based technology.

Although the concept of NIRS has already been available during the second half of the 20th century, its main initial application was for chemical analysis [5,6]. Since the end of the 1970s [7], numerous studies have been published about this method [8,9]. NIRS offers real-time noninvasive monitoring of oxy and deoxyhemoglobin in tissues within a few centimeters from the skin (Figure 1). Furthermore, so-called dynamic values, i.e., values registered during short occlusion of the vascular supply of the area under assessment can be measured. These dynamic values might give additional data on local VO₂ and probably the condition of the blood flow of the

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microcirculation [10]. Some NIRS variables also correlate with invasively monitored central circulatory variables [11]. More recently, due to device development, availability, and marketing, clinical research dealing with the utility of NIRS has focused on the specific methodology of NIRS of the thenar eminence (NIRS_{th}).

Clinical application of NIRS_{th} remains a relatively new method in a field where no “gold standards” exist. Thus, clinicians considering the use of NIRS_{th} need to understand the principles and evidence behind it and appreciate the many areas of uncertainty that surround its application. In this review, we assess several of these aspects and suggest studies to address areas of controversy.

The principle of clinical NIRS is to noninvasively measure the attenuation of light by hemoglobin, where the emitted light is in a wavelength range longer than visible light [12,13]. NIRS utilizes a narrower spectrum of wavelengths than pulse oximetry, which penetrate deeper into the tissue [14]. Furthermore, whilst NIRS characterizes the total tissue oxy and deoxyhemoglobin in a quantitative and qualitative fashion [13], generating information on oxygen supply and demand, pulse oximetry monitors only hemoglobin in the arterial (pulsatile) part of the local circulation [15].

The near infrared (NIR) light spectrum ranges from 700 nm to 1000 nm. For clinical applications of NIRS, wavelengths approximately 700 to 850 nm are used. The interval stretches on either side of the isobestic point of hemoglobin, i.e., a given wavelength where absorption of light for oxy and deoxyhemoglobin is identical. This wavelength interval maximizes the difference between oxy and deoxyhemoglobin and minimizes the influence of other chromophores, such as myoglobin, cytochrome oxidase, melanin, and bilirubin, on the measurements [16]. Fortunately, the impact of myoglobin on NIRS for measuring tissue oxygenation is minimal [17,18] and melanin, due to its superficial localization, is not a major

issue in this regard. Increased conjugated bilirubin levels do influence measurements by dampening the signal. However, trends can still be followed even with jaundice [19].

Physical background

NIRS technology is based on sophisticated physical models, which are greatly simplified in the description below. The attenuation of light in a sample or tissue is proportional to the pathlength of the light and the absorption coefficient of the chromophore according to the physical principles referred to in the Lambert-Beer law [20]. The absorption coefficient of a compound, in the former equation, is a product of the concentration of the compound and the specific extinction coefficient of the compound. Thus, if attenuation of NIR spectrum light is measured and if all other components of the equation described above are known, the concentration of the chromophore, e.g., oxyhemoglobin can be measured. Unfortunately, because the pathlength of light varies due to reflection and interference in a complex milieu of different tissues, absolute concentrations are difficult to estimate. However, the pathlength of light is more or less constant and the extinction coefficients of the common chromophores are known physical quantities. Thus, changes in attenuation of light will be directly proportional to relative changes in concentration of the chromophore. Absolute changes in concentration can be approximated by creating mathematical algorithms for light pathlength in a tissue.

Because the absorption coefficient is direct proportional to concentrations of a chromophore in a tissue studied and extinction coefficient of the compound is constant, estimating the absorption coefficient yields approximations of the absolute chromophore concentration. This is possible through advanced modeling of the behavior of NIR light in tissues and the technical possibility of measuring at several NIR wavelengths [21]. Yet, given the large number of assumptions and approximations in the theoretical basis of NIRS, one may consider trends in different NIRS parameters as more robust than discrete values.

Technical considerations

The NIRS probes in current use measure reflected light. Thus, the NIR light source is placed beside the light sensor. The distance between the light source and sensor determine the distance from where the main part of the reflected light is measured. The technical limit of the monitored depth is the energy of light that does not damage tissues [21]. The main determinants of signal are small vessels of the microcirculation [22].

The brain [23], kidney [24], lower extremity [25], brachioradialis muscle [26], and thenar eminence [27] are all possible sites for bedside NIRS monitoring. The

advantage of the thenar eminence compared with other sites, in terms of minimizing variability, is the relatively thin fat tissue over the muscle. Additionally, fibrous strands in its subcutaneous tissue limit the extent of edema formation providing the best possible setting for muscle tissue saturation (StO_2) measurement even in obese and critically ill patients [28]. Due to anatomical conditions, both the brachioradial muscle and the muscles of the thenar eminence can be easily subjected to the vascular obstruction test (see below).

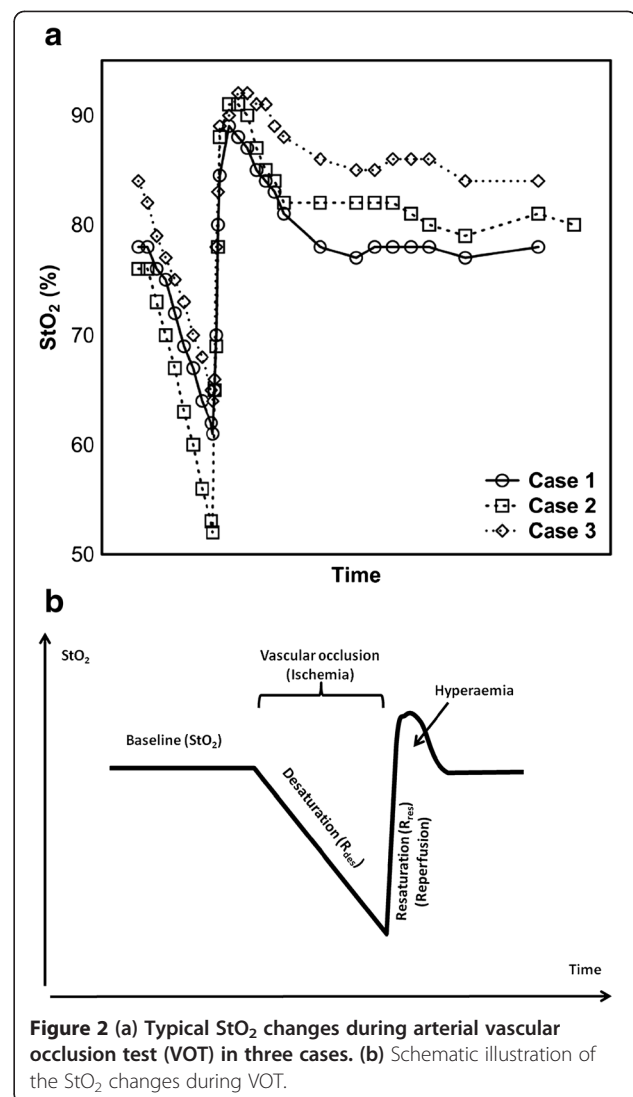
Derived parameters – the vascular occlusion test

Assessing the ratio of oxy and deoxyhemoglobin in the monitored tissue gives continuous StO_2 . Because absolute hemoglobin content also can be estimated, total tissue hemoglobin and its absolute changes are expressed as tissue hemoglobin index (THI), which can be obtained with this method. THI is, however, not total tissue hemoglobin but its approximation, based on the signal strength of hemoglobin in the monitored area. Low StO_2 and THI are common findings in hypovolemic shock states.

By occluding the arterial [16] or the venous [29] blood flow to the thenar eminence, NIRS can assess dynamic changes that reflect VO_2 and postischemic reperfusion and hyperemia. This, vascular occlusion test (VOT), is of special interest in septic shock [30,31] or during anesthesia [32] where the static variables may not be affected despite disturbed circulation.

Arterial and venous vascular occlusion is achieved by a pneumatic cuff on the arm inflated to pressures well above the systolic arterial pressure, aiming to induce ischemia in the thenar muscles and changes in StO_2 (Figures 2a,b). Considerable duration of obstruction is required to obtain a reperfusion response that differentiates healthy volunteers from resuscitated septic shock patients [33]. However, the optimal way of performing VOT is a matter of debate. Both the intensity and/or duration of the VOT are a matter of controversy; some authors advocate a time-targeted VOT [34,35], and others advocate occlusion to a StO_2 -targeted VOT [33,36]. The argument for time-targeted VOT is that maximal ischemic vascular response is reached within a few minutes of VOT [37] and that long vascular occlusion times could lead to inability to complete VOT procedure due to subject discomfort [38]. On the other hand, argument for StO_2 -targeted VOT, i.e., aiming for StO_2 of 40%, is that a standardized level of ischemia is achieved, thus interindividual variations in response to VOT giving varying level of ischemia can be minimized.

The rate of desaturation in the thenar muscles (R_{des} ; $\% \times sec^{-1}$) after vascular obstruction can be used to estimate VO_2 in the thenar muscles. The product of the absolute value of R_{des} and mean THI value quantifies the amount of desaturated hemoglobin [16]. The latter can



be converted to thenar VO_2 using the hemoglobin-oxygen binding constant [39].

In addition, after cuff deflation there is a swift restoration of blood flow that can be described in terms of the derivate of the StO_2 upslope (R_{res} ; $\% \times sec^{-1}$). During this reactive hyperemia, the StO_2 increases over baseline levels, indicating postischemic vasodilatation and capillary recruitment. The integral of the post reoxygenation StO_2 curve over baseline quantifies reactive hyperemia.

The VOT derived variables add to the robustness of NIRS measurements. In a small study, R_{des} , R_{res} , and reactive hyperemia were all lower in septic shock patients than in healthy controls [31]. Moreover, R_{res} had an inverse relationship to sequential organ failure assessment (SOFA) and predicted mortality. Finally, a coefficient of variation of less than 10% has been reported for R_{res} [33].

Venous occlusion is performed by inflating a pneumatic cuff above venous pressure on the arm [29]. In this setting, NIRS shows increased THI due to vascular congestion and eventually decreasing StO_2 . The venous occlusion method also can be used to estimate local VO_2 . Some have reported varying reproducibility of VO_2 measurements with venous occlusion [40].

NIRS during the perioperative period

Although there is an extensive literature on NIRS used before [41], during [42-45] or after surgery [46,47], relatively little has been published on $NIRS_{th}$ during the perioperative period. However, in a recent publication R_{res} , measured in patients undergoing major abdominal surgery patients, was *decreased* in fluid responsive patients [32]. In the same study, fluid responsiveness was detected by invasive methods, such as pulse pressure variation.

Data are conflicting on the ability of $NIRS_{th}$ to detect blood loss, an area of central interest in anesthesia. In awake volunteers, a 500-ml blood loss at blood donation did not lead to changes in NIRS variables [27]. On the other hand, hemodynamically significant hypovolemia, in awake volunteers, did decrease StO_2 and THI [48]. A possible explanation could be that tissue hemoglobin and oxygenation at the thenar eminence are not affected by blood loss within the capacity of the compensatory mechanisms of hypovolemia.

In patients after cardiac surgery, StO_2 and THI did not correlate with global circulatory parameters, but changes in body-finger temperature correlated with changes in StO_2 [49]. However, StO_2 during the perioperative period in cardiac surgery is lower in patients who develop certain postoperative complications [50] than those who do not [51]. StO_2 at the thenar eminence does not predict mortality in cardiac surgery [51] or surgical-site infections in colon surgery [52].

Intensive care applications

In a general intensive care population with most patients in resuscitated shock, StO_2 and R_{res} appear related to capillary refill time and central to peripheral temperature gradient, but not to the etiology of shock [53]. In a mixed group of patients with increased blood lactate levels observed during 8 hours of resuscitation [54], half had low StO_2 (<70%) on admission. There was no difference in systemic circulatory variables between patients with low or normal StO_2 , but SOFA scores and acute physiology and chronic health evaluation II scores were higher in patients with decreased StO_2 . However, blood transfusion substantially increasing blood hemoglobin did not increase StO_2 in a mixed group of stable patients [55] and the correlation between blood hemoglobin and tissue hemoglobin index

may be limited [31,55]. In patients with hypovolemic or septic patients, more homogenous observations have been described.

NIRS in hypovolemic shock

The rationale for monitoring peripheral tissue as the thenar eminence in hypovolemic shock is centralization of circulation to vital organs leading to decreased blood flow in muscles [56]. In acute hemorrhage, activation of the sympathetic nervous system [57] should decrease thenar muscles blood flow, with increased oxygen extraction and decreased tissue hemoglobin content. In this setting, $NIRS_{th}$ may thus act as a sensor of the vascular response to hypovolemia. In trauma patients with severe shock, StO_2 is lower in than in milder grades of shock or in normal individuals [58], although patients with shock can present with StO_2 values as in controls [38].

In a study of severe postpartum hemorrhage, the StO_2 range overlapped during and after hemorrhage, but StO_2 increased after control of bleeding [59]. The lowest StO_2 in the trauma bay has been shown to be as good as the lowest systolic blood pressure at identifying severe shock as defined by experienced clinicians [58]. Furthermore, StO_2 within 1 hour of admission is lower in trauma patients who develop multiorgan dysfunction (MODS) or die, and a strongest predictor of MODS or death than other diagnostic modalities [60,61]. Low StO_2 within 1 hour of admission was as sensitive as a high base deficit in identifying patients who developed MODS or died, although specificity for both was low [62]. Finally, low StO_2 within 1 hour of admission identifies trauma patients who will require blood transfusion within the next 24 hours [63].

The discriminatory power of *dynamic* NIRS parameters, however, is of greater interest. R_{res} was lower in trauma patients compared with controls with little overlap [38]. Moreover, low R_{res} predicted increased troponin I levels in postpartum bleeding [59]. Thus, in hypovolemia, low static StO_2 predict adverse outcome but dynamic NIRS parameters seem to be more promising.

NIRS in sepsis

In septic shock, although hypovolemia can be a finding [64], there is a substantial microcirculatory disturbance with closed capillaries, arteriovenous shunting, and decreased flow [65]. As a result of these phenomena, oxygen content in the vessels of the microcirculation could be normal, making clinical interpretation of NIRS data complex. These pathophysiologic changes may be not necessarily mirrored by low StO_2 , but rather by low R_{res} and impaired posts ischemic hyperemic response.

Several studies report a difference in StO_2 between healthy subjects and patients with severe sepsis or septic

shock [16,30,34,66,67], whereas others do not [31,68,69]. Although variation in population characteristics could partly explain these results, in all studies StO_2 values overlap between septic shock patients and healthy volunteers. This is not surprising because, in sepsis, StO_2 in sepsis can be at the higher end of the normal spectrum or markedly low [65]. Dynamic NIRS parameters, however, improve the power of the method to distinguish pathologic tissue oxygenation from normal. The R_{des} of the thenar muscles is slower in septic shock patients, indicating a lower rate of tissue VO_2 [16,31,68,69]. Furthermore, R_{des} varies with the severity of systemic infections [69]. Similarly, R_{res} appears lower in septic patients compared with healthy controls [16,30,31,34,70] and decreases with increasing disease severity [30,31]. R_{res} ranges overlap minimally [30,68,70] or not at all [34] when comparing healthy controls and septic patients, and R_{res} improves as septic shock resolves [34]. Finally postischemic hyperemia is decreased in septic patients compared with healthy controls [30,31]. Thus, $NIRS_{th}$ could be a method for bedside assessment of the microcirculation [71].

Monitoring global hemodynamics with $NIRS_{th}$ also has attracted interest. In sepsis, treatment based on venous saturation in the superior vena cava ($ScvO_2$) [64] or the pulmonary artery (SvO_2) [72] is used, because these are markers of global DO_2 and VO_2 balance [73]. Noninvasively obtained surrogates of $ScvO_2$ or SvO_2 would be valuable. In this regard, StO_2 correlates to $ScvO_2$ [74] and SvO_2 [67] in patients with severe sepsis or septic shock; however, correlation coefficients are relatively low. The accuracy of estimating SvO_2 could be substantially improved by calculating the “NIRS-derived SvO_2 ” [67]. In severe sepsis and severe heart failure, StO_2 , however, did not estimate SvO_2 [75]. Still, data suggest that patients with severe sepsis or septic shock and low StO_2 also have low $ScvO_2$, suggesting hypodynamic circulation [74].

Variables related to DO_2 may correlate with NIRS parameters. R_{res} correlates with cardiac output and to a lesser extent with blood lactate levels in septic patients [34], whereas StO_2 does not correlate with lactate or base deficit [67]. A low StO_2 predicts a very low DO_2 in early sepsis with high sensitivity and specificity [76]; however, moderately low DO_2 does not correlate with StO_2 . Neither was change in R_{res} correlated to change in cardiac output in septic patients. Finally, StO_2 did not correlate with the severity of illness [67], but a $StO_2 < 78\%$ in resuscitated patients predicted mortality [77]. Low R_{res} correlates with organ failure [31] and R_{res} is lower in nonsurvivors than survivors [34].

Recently, it has been reported that increasing blood pressure with noradrenalin infusion from 65 mmHg to 85 mmHg in resuscitated sepsis patients normalized R_{res} [78]. These patients, although seemingly resuscitated

according to the Surviving Sepsis Campaign guidelines, could improve the thenar perfusion by achieving higher mean arterial pressure [79]. These data could suggest that NIRS can identify patients who benefit from treatment beyond the traditional goals, thus the usefulness of NIRS as a bedside tool to optimize tissue oxygenation [71]. Thus, in resuscitated, septic patients, dynamic NIRS of the thenar eminence provides information on microcirculation and trends could be used to guide treatment.

NIRS in miscellaneous conditions

In patients with chronic heart failure, thenar StO_2 , R_{des} , and R_{res} are low [80]. $NIRS_{th}$ parameters in these patients improved after 6 hours of dobutamine or levosimendan infusion [80], or 3 months of regular exercise training [81]. Patients with cirrhosis demonstrated a supranormal hyperemic response after vascular occlusion test [82], which increases with increasing severity of liver disease [82].

NIRS-derived and central hemodynamic parameters

The performance of $NIRS_{th}$ in estimating global circulatory parameters is highly dependent on the coupling between the circulation of the hand and the central circulation. Static NIRS parameters, such as StO_2 , should, in theory, be related to centrally measured circulatory parameters. However, this relationship has not been demonstrated in general in critically ill patients [54] or after cardiac surgery [49]. Although, global venous saturations have been described to correlate to StO_2 in sepsis, this relationship is weak [67,74,83]. In sepsis, correlation between StO_2 and SvO_2 can be improved with correction equations [67]. Only substantial deviations from normal DO_2 levels are detected reliably by StO_2 in sepsis [76]. In these patients, R_{res} correlates with cardiac index and blood lactate levels [34].

Low StO_2 also predicts MODS in a mixed population of critically ill patients [54], in trauma patients [60-62], and in postcardiac surgery patients [51]. In sepsis, dynamic NIRS variables, such as R_{des} [69] and R_{res} [31,34], have been associated with organ failure.

Limitations

$NIRS_{th}$ monitors peripheral muscle as a marker of perfusion elsewhere. The theoretical concern is whether the small volume of distal muscle can be a good indicator of the state of the tissue oxygenation in the rest of the body and the vital organs. For example, local factors, such as obstruction to flow by atherosclerosis, an arterial catheter, or thrombosis after previous arterial catheterization, could affect measurement. Although a brief report suggests that catheterization of the radial artery in adult, elective, surgical patients does not affect StO_2 [84], this

may not be the case in critically ill patients with circulatory failure.

StO₂ is not the same as tissue blood flow in the tissue or even oxygen supply. StO₂ is affected by local VO₂, which could be affected by states that alter muscle metabolism, such as muscle relaxants. The balance between the metabolic state of the muscle and other vital organs may vary between individuals and intraindividually during the course of disease, affecting the global relevance of some of the NIRS variables. Also tissues overlaying the thenar muscles can influence measurements [85]. Very dark skin with high melanin content or thick, edematous, or injured connective tissues and low hemoglobin levels also could pose problems with NIRS measurements [86].

There are many studies on NIRS in the perioperative period and critical illness where many different sites are monitored. Moreover, the methodology of NIRS is not standardized [87,88]. Several probes are available on the market, different occlusion protocols are used, and different parts of downslope and the upslope StO₂ curves at vascular occlusion are used in calculations. Furthermore, studies are conducted on patients in different phases of disease, which may represent different pathophysiologic situations. Hence, comparing results can be difficult (Table 1).

Static NIRS variables are influenced by the temperature of the hands compared with core temperature [53] and could be a reason for the overlap between patients with normal and abnormal peripheral circulation. In anesthesia with volatile anaesthetics, vasodilatation is a common feature [89] and monitoring StO₂ would be expected to be less affected by peripheral vascular tone.

The question of whether organ failure in sepsis is mostly dependent on disturbed mitochondrial oxygen metabolism [90-92], or on limited DO₂ [93,94], is a matter of debate. NIRS does not assess mitochondrial oxygen metabolism in sepsis, although decreased tissue VO₂, R_{des}, could imply mitochondrial dysfunction. However, decreased tissue VO₂ also could depend on shunting of delivered oxygen [65]. This phenomenon is not measured by NIRS either. However, with all of its limitations, NIRS_{th} is a noninvasive method that, given that supporting data will be available, could become part of anesthetic and intensive care monitoring in a manner similar to pulse oximetry.

Conclusions NIRS_{th} eminence estimates StO₂ in peripheral muscles. In hypovolemia, such StO₂ is decreased and relates to severity of disease and outcome. Hence, StO₂ measurements could aid with the clinical management of these patients. In unresuscitated or inadequately

Table 1 Summary of tissue saturation (StO₂), desaturation rate (R_{des}), and resaturation (R_{res}) values in different shock states in the literature

	Study	Controls	Severe sepsis/septic shock	Hemorrhagic shock
StO₂ (%)	Skarda et al. [16]	75 ± 15	87 ± 6	
	Creteur et al. [30]	80 ± 7	72 ± 11	
	Doerschug et al. [31]	84 ± 10	82 ± 13	
	Mayeur et al. [33]	82 ± 4	80 ± 10	
	Nanas et al. [68]	82 ± 6	76 ± 17	
	Pareznik et al. [69]	83 (79-93)	89 (65-92)	
	Georger et al. [70]	82 ± 4	75 ± 9	
	Podbregar et al. [75]	84 ± 4	90 ± 7	
	Crookes et al. [58]	87 ± 6.4		45 ± 27
	Gómez et al. [38]	88 ± 5		86 ± 9
R_{des} (%/s)	Mayeur et al. [33]	0.18 ± 0.05	0.16 ± 0.06	
	Nanas et al. [68]	0.6 ± 0.18	0.25 ± 0.1	
	Pareznik et al. [69]	0.62 (0.46-0.94)	0.12 (0.06-0.18)	
	Georger et al. [70]	0.22 ± 0.05	0.16 ± 0.07	
	Gómez et al. [38]		0.17 ± 0.06	0.15 ± 0.09
R_{res} (%/s)	Skarda et al. [16]	3.3 ± 0.7	2.3 ± 1	
	Creteur et al. [30]	4.8 ± 1.6	2.3 ± 1.3	
	Doerschug et al. [31]	4.7 ± 1.1	2.3 ± 1.5	
	Mayeur et al. [33]	5.4 ± 1.1	2.3 ± 1.4	
	Nanas et al. [68]	12 ± 3.7	2.4 ± 1.7	
	Georger et al. [70]	2.3 ± 0.5	1 ± 0.6	
	Gómez et al. [38]	5.4 ± 1.3		2.5 ± 1.3

Data are given as mean ± SD or median (range).

resuscitated septic shock, R_{des} and R_{res} are decreased, indicating disturbed tissue oxygen metabolism and microvascular reserve. The pathologic findings in these dynamic NIRS parameters could be of value in resuscitated sepsis where macrocirculatory failure has been corrected and during anesthesia to monitor adequacy of peripheral perfusion and fluid status. Despite its limitations, $NIRS_{th}$ takes monitoring from global to local level. The existing literature on $NIRS_{th}$ is mainly focused on validation of this technique. Future studies that implement $NIRS_{th}$ into treatment algorithms in anesthesia and intensive care would be valuable to define the place for this monitoring modality in daily management of critically ill patients.

Competing interests

The authors declare that they have no competing interests.

Authors contributions

All authors conceived the manuscript. Both ML and NCZW reviewed the literature. ML wrote the first draft. RB reviewed and refined the first draft. Further drafts were completed by ML, RB and NCZW. All authors read and approved the final manuscript.

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References

1. Seymour CW, Kahn JM, Cooke CR, Watkins TR, Heckbert SR, Rea TD: Prediction of critical illness during out-of-hospital emergency care. *JAMA* 2010, **304**:747–754.
2. Forrester JS, Ganz W, Diamond G, McHugh T, Chonette DW, Swan HJ: Thermodilution cardiac output determination with a single flow-directed catheter. *Am Heart J* 1972, **83**:306–311.
3. Hungerer S, Nolte D, Botzlar A, Messmer K: Effects of Diaspirin Crosslinked Hemoglobin (DCLHb) on microcirculation and local tissue pO_2 of striated skin muscle following resuscitation from hemorrhagic shock. *Artif Cell Blood Substit Biotechnol* 2006, **34**:455–471.
4. Trzeciak S, Dellinger RP, Parrillo JE, Guglielmi M, Bajaj J, Abate NL, Arnold RC, Colilla S, Zanotti S, Hollenberg SM, et al: Early microcirculatory perfusion derangements in patients with severe sepsis and septic shock: relationship to hemodynamics, oxygen transport, and survival. *Ann Emerg Med* 2007, **49**:88–98. 98 e81–82.
5. Borchman D, Yappert MC, Herrell P: Structural characterization of human lens membrane lipid by infrared spectroscopy. *Invest Ophthalmol Vis Sci* 1991, **32**:2404–2416.
6. Metsala M, Schmidt FM, Skytta M, Vaittinen O, Halonen L: Acetylene in breath: background levels and real-time elimination kinetics after smoking. *J Breath Res* 2010, **4**:046003.
7. Jobsis FF: Noninvasive, infrared monitoring of cerebral and myocardial oxygen sufficiency and circulatory parameters. *Science* 1977, **198**:1264–1267.
8. Taillefer MC, Denault AY: Cerebral near-infrared spectroscopy in adult heart surgery: systematic review of its clinical efficacy. *Can J Anaesth* 2005, **52**:79–87.
9. Santora RJ, Moore FA: Monitoring trauma and intensive care unit resuscitation with tissue hemoglobin oxygen saturation. *Crit Care* 2009, **13**(Suppl 5):S10.
10. Bartels SA, Bezemer R, Milstein DM, Radder M, Lima A, Cherpanath TG, Heger M, Karemaker JM, Ince C: The microcirculatory response to compensated hypovolemia in a lower body negative pressure model. *Microvasc Res* 2011, **82**:374–380.
11. Mozina H, Podbregar M: Near-infrared spectroscopy during stagnant ischemia estimates central venous oxygen saturation and mixed venous oxygen saturation discrepancy in patients with severe left heart failure and additional sepsis/septic shock. *Crit Care* 2010, **14**:R42.
12. Warior R, Gaffke JN, Haller RG, Bertocci LA: A modular NIRS system for clinical measurement of impaired skeletal muscle oxygenation. *J Appl Physiol* 2000, **88**:315–325.
13. Wahr JA, Tremper KK, Samra S, Delpy DT: Near-infrared spectroscopy: theory and applications. *J Cardiothorac Vasc Anesth* 1996, **10**:406–418.
14. McMorrow RC, Mythen MG: Pulse oximetry. *Curr Opin Crit Care* 2006, **12**:269–271.
15. Sinex JE: Pulse oximetry: principles and limitations. *Am J Emerg Med* 1999, **17**:59–67.
16. Skarda DE, Mulier KE, Myers DE, Taylor JH, Beilman GJ: Dynamic near-infrared spectroscopy measurements in patients with severe sepsis. *Shock* 2007, **27**:348–353.
17. Seiyama A, Hazeki O, Tamura M: Noninvasive quantitative analysis of blood oxygenation in rat skeletal muscle. *J Biochem* 1988, **103**:419–424.
18. Mancini DM, Bolinger L, Li H, Kendrick K, Chance B, Wilson JR: Validation of near-infrared spectroscopy in humans. *J Appl Physiol* 1994, **77**:2740–2747.
19. Madsen PL, Skak C, Rasmussen A, Secher NH: Interference of cerebral near-infrared oximetry in patients with icterus. *Anesth Analg* 2000, **90**:489–493.
20. Uludag K, Kohl M, Steinbrink J, Obrig H, Villringer A: Cross talk in the Lambert-Beer calculation for near-infrared wavelengths estimated by Monte Carlo simulations. *J Biomed Opt* 2002, **7**:51–59.
21. Murkin JM, Arango M: Near-infrared spectroscopy as an index of brain and tissue oxygenation. *Br J Anaesth* 2009, **103**(Suppl 1):i3–i13.
22. Creteur J: Muscle StO₂ in critically ill patients. *Curr Opin Crit Care* 2008, **14**:361–366.
23. Wolf M, Ferrari M, Quaresima V: Progress of near-infrared spectroscopy and topography for brain and muscle clinical applications. *J Biomed Opt* 2007, **12**:062104.
24. Owens GE, King K, Gurney JG, Charpie JR: Low renal oximetry correlates with acute kidney injury after infant cardiac surgery. *Pediatr Cardiol* 2011, **32**:183–188.
25. Shuler MS, Reisman WM, Kinsey TL, Whitesides TE Jr, Hammerberg EM, Davila MG, Moore TJ: Correlation between muscle oxygenation and compartment pressures in acute compartment syndrome of the leg. *J Bone Joint Surg Am* 2010, **92**:863–870.
26. Rodriguez A, Lisboa T, Martin-Loeches I, Diaz E, Trefler S, Restrepo MI, Rello J: Mortality and Regional Oxygen Saturation Index in Septic Shock Patients: A Pilot Study. *J Trauma* 2011, **70**:1145–1152.
27. Jeger V, Jakob SM, Fontana S, Wolf M, Zimmermann H, Exadaktylos AK: 500 ml of blood loss does not decrease non-invasive tissue oxygen saturation (StO₂) as measured by near infrared spectroscopy - A hypothesis generating pilot study in healthy adult women. *J Trauma Manag Outcomes* 2010, **4**:5.
28. Poeze M: Tissue-oxygenation assessment using near-infrared spectroscopy during severe sepsis: confounding effects of tissue edema on StO₂ values. *Intensive Care Med* 2006, **32**:788–789.
29. Girardis M, Rinaldi L, Busani S, Flore I, Mauro S, Pasetto A: Muscle perfusion and oxygen consumption by near-infrared spectroscopy in septic-shock and non-septic-shock patients. *Intensive Care Med* 2003, **29**:1173–1176.
30. Creteur J, Carollo T, Soldati G, Buchele G, De Backer D, Vincent JL: The prognostic value of muscle StO₂ in septic patients. *Intensive Care Med* 2007, **33**:1549–1556.
31. Doerschug KC, Delsing AS, Schmidt GA, Haynes WG: Impairments in microvascular reactivity are related to organ failure in human sepsis. *Am J Physiol Heart Circ Physiol* 2007, **293**:H1065–H1071.
32. Futier E, Christophe S, Robin E, Petit A, Pereira B, Desbordes J, Bazin JE, Vallet B: Use of near-infrared spectroscopy during a vascular occlusion test to assess the microcirculatory response during fluid challenge. *Crit Care* 2011, **15**:R214.
33. Mayeur C, Campard S, Richard C, Teboul JL: Comparison of four different vascular occlusion tests for assessing reactive hyperemia using near-infrared spectroscopy. *Crit Care Med* 2011, **39**:695–701.

34. Payen D, Luengo C, Heyer L, Resche-Rigon M, Kerever S, Damoisel C, Lossner MR: Is thenar tissue hemoglobin oxygen saturation in septic shock related to macrohemodynamic variables and outcome?. *Crit Care* 2009, **13**(Suppl 5):S6.
35. Damoisel C, Payen D: Vascular occlusion tests: Do we need another definition? *Crit Care Med* 2011, **39**:2587–2588.
36. Mayeur C, Teboul JL: Vascular occlusion tests: Do we need another definition? *Crit Care Med* 2011, **39**:2588–2589.
37. Farnebo S, Thorfinn J, Henricson J, Tesselaaar E: Hyperaemic changes in forearm skin perfusion and RBC concentration after increasing occlusion times. *Microvasc Res* 2010, **80**:412–416.
38. Gomez H, Torres A, Polanco P, Kim HK, Zenker S, Puyana JC, Pinsky MR: Use of non-invasive NIRS during a vascular occlusion test to assess dynamic tissue O₂ saturation response. *Intensive Care Med* 2008, **34**:1600–1607.
39. McGee WT, Mailloux P, Jodka P, Thomas J: The pulmonary artery catheter in critical care. *Semin Dial* 2006, **19**:480–491.
40. Van Beekvelt MC, Colier WN, Wevers RA, Van Engelen BG: Performance of near-infrared spectroscopy in measuring local O₂ consumption and blood flow in skeletal muscle. *J Appl Physiol* 2001, **90**:511–519.
41. Bundgaard-Nielsen M, Ruhnau B, Secher NH, Kehlet H: Flow-related techniques for preoperative goal-directed fluid optimization. *Br J Anaesth* 2007, **98**:38–44.
42. Sako H, Hadama T, Miyamoto S, Anai H, Wada T, Iwata E, Hamamoto H, Tanaka H, Urushino K, Shuto T: Effect of prostaglandin E1 on ischemia-reperfusion injury during abdominal aortic aneurysm surgery. *Surg Today* 2006, **36**:140–146.
43. Schoen J, Husemann L, Tiemeyer C, Lueloh A, Sedemund-Adib B, Berger KU, Hueppe M, Heringlake M: Cognitive function after sevoflurane- vs propofol-based anaesthesia for on-pump cardiac surgery: a randomized controlled trial. *Br J Anaesth* 2011, **106**:840–850.
44. Kwak HJ, Lee D, Lee YW, Yu GY, Shinn HK, Kim JY: The intermittent compression device on the lower extremities attenuates the decrease in regional cerebral oxygen saturation during sitting position under sevoflurane anesthesia. *J Neurosurg Anesthesiol* 2011, **23**:1–5.
45. Hirano Y, Omura K, Tatsuzawa Y, Shimizu J, Kawaura Y, Watanabe G: Tissue oxygen saturation during colorectal surgery measured by near-infrared spectroscopy: pilot study to predict anastomotic complications. *World J Surg* 2006, **30**:457–461.
46. Mutoh T, Ishikawa T, Suzuki A, Yasui N: Continuous cardiac output and near-infrared spectroscopy monitoring to assist in management of symptomatic cerebral vasospasm after subarachnoid hemorrhage. *Neurocrit Care* 2010, **13**:331–338.
47. Kahraman S, Kayali H, Atabey C, Acar F, Gocmen S: The accuracy of near-infrared spectroscopy in detection of subdural and epidural hematomas. *J Trauma* 2006, **61**:1480–1483.
48. Bartels SA, Bezemer R, de Vries FJ, Milstein DM, Lima A, Cherpanath TG, van den Meiracker AH, van Bommel J, Heger M, Karemaker JM, Ince C: Multi-site and multi-depth near-infrared spectroscopy in a model of simulated (central) hypovolemia: lower body negative pressure. *Intensive Care Med* 2011, **37**:671–677.
49. Uilkema RJ, Groeneveld AB: Correlates of thenar near-infrared spectroscopy-derived tissue O₂ saturation after cardiac surgery. *Interact Cardiovasc Thorac Surg* 2007, **6**:265–269.
50. Bennett-Guerrero E, Welsby I, Dunn TJ, Young LR, Wahl TA, Diers TL, Phillips-Bute BG, Newman MF, Mythen MG: The use of a postoperative morbidity survey to evaluate patients with prolonged hospitalization after routine, moderate-risk, elective surgery. *Anesth Analg* 1999, **89**:514–519.
51. Sanders J, Toor IS, Yurik TM, Keogh BE, Mythen M, Montgomery HE: Tissue oxygen saturation and outcome after cardiac surgery. *Am J Crit Care* 2011, **20**:138–145.
52. Govinda R, Kasuya Y, Bala E, Mahboobi R, Devarajan J, Sessler DI, Akca O: Early postoperative subcutaneous tissue oxygen predicts surgical site infection. *Anesth Analg* 2010, **111**:946–952.
53. Lima A, van Bommel J, Sikorska K, van Genderen M, Klijn E, Lesaffre E, Ince C, Bakker J: The relation of near-infrared spectroscopy with changes in peripheral circulation in critically ill patients. *Crit Care Med* 2011, **39**:1649–1654.
54. Lima A, van Bommel J, Jansen TC, Ince C, Bakker J: Low tissue oxygen saturation at the end of early goal-directed therapy is associated with worse outcome in critically ill patients. *Crit Care* 2009, **13**(Suppl 5):S13.
55. Creteur J, Neves AP, Vincent JL: Near-infrared spectroscopy technique to evaluate the effects of red blood cell transfusion on tissue oxygenation. *Crit Care* 2009, **13**(Suppl 5):S11.
56. McKinley BA, Parmley CL, Butler BD: Skeletal muscle PO₂, PCO₂, and pH in hemorrhage, shock, and resuscitation in dogs. *J Trauma* 1998, **44**:119–127.
57. Bonanno FG: Physiopathology of shock. *J Emerg Trauma Shock* 2011, **4**:222–232.
58. Crookes BA, Cohn SM, Bloch S, Amortegui J, Manning R, Li P, Proctor MS, Hallal A, Blackburne LH, Benjamin R, et al: Can near-infrared spectroscopy identify the severity of shock in trauma patients? *J Trauma* 2005, **58**:806–813. discussion 813–806.
59. Heyer L, Mebazaa A, Gayat E, Resche-Rigon M, Rabuel C, Rezan E, Lukasiewicz AC, Madadaki C, Pirracchio R, Schurando P, et al: Cardiac troponin and skeletal muscle oxygenation in severe post-partum haemorrhage. *Crit Care* 2009, **13**(Suppl 5):S8.
60. Beilman GJ, Blondet JJ, Nelson TR, Nathens AB, Moore FA, Rhee P, Puyana JC, Moore EE, Cohn SM: Early hypothermia in severely injured trauma patients is a significant risk factor for multiple organ dysfunction syndrome but not mortality. *Ann Surg* 2009, **249**:845–850.
61. Moore FA, Nelson T, McKinley BA, Moore EE, Nathens AB, Rhee P, Puyana JC, Beilman GJ, Cohn SM: Massive transfusion in trauma patients: tissue hemoglobin oxygen saturation predicts poor outcome. *J Trauma* 2008, **64**:1010–1023.
62. Cohn SM, Nathens AB, Moore FA, Rhee P, Puyana JC, Moore EE, Beilman GJ: Tissue oxygen saturation predicts the development of organ dysfunction during traumatic shock resuscitation. *J Trauma* 2007, **62**:44–54. discussion 54–45.
63. Smith J, Bricker S, Putnam B: Tissue oxygen saturation predicts the need for early blood transfusion in trauma patients. *Am Surg* 2008, **74**:1006–1011.
64. Rivers E, Nguyen B, Havstad S, Ressler J, Muzzin A, Knoblich B, Peterson E, Tomlanovich M: Early goal-directed therapy in the treatment of severe sepsis and septic shock. *N Engl J Med* 2001, **345**:1368–1377.
65. De Backer D, Ospina-Tascon G, Salgado D, Favory R, Creteur J, Vincent JL: Monitoring the microcirculation in the critically ill patient: current methods and future approaches. *Intensive Care Med* 2010, **36**:1813–1825.
66. De Backer D, Ospina-Tascon G, Salgado D, Favory R, Creteur J, Vincent JL: Monitoring the microcirculation in the critically ill patient: current methods and future approaches. *Intensive Care Med* 2010, **36**:1813–1825.
67. Mulier KE, Skarda DE, Taylor JH, Myers DE, McGraw MK, Gallea BL, Beilman GJ: Near-infrared spectroscopy in patients with severe sepsis: correlation with invasive hemodynamic measurements. *Surg Infect (Larchmt)* 2008, **9**:515–519.
68. Nanas S, Gerovasili V, Renieris P, Angelopoulos E, Poriazi M, Kritikos K, Sifaka A, Baraboutis I, Zervakis D, Markaki V, et al: Non-invasive assessment of the microcirculation in critically ill patients. *Anaesth Intensive Care* 2009, **37**:733–739.
69. Pareznik R, Knezevic R, Voga G, Podbregar M: Changes in muscle tissue oxygenation during stagnant ischemia in septic patients. *Intensive Care Med* 2006, **32**:87–92.
70. Georger JF, Hamzaoui O, Chaari A, Maizel J, Richard C, Teboul JL: Restoring arterial pressure with norepinephrine improves muscle tissue oxygenation assessed by near-infrared spectroscopy in severely hypotensive septic patients. *Intensive Care Med* 2010, **36**:1882–1889.
71. Silva S, Teboul JL: Defining the adequate arterial pressure target during septic shock: not a 'micro' issue but the microcirculation can help. *Crit Care* 2011, **15**:1004.
72. Velissaris D, Pierrakos C, Scolletta S, De Backer D, Vincent JL: High mixed venous oxygen saturation levels do not exclude fluid responsiveness in critically ill septic patients. *Crit Care* 2011, **15**:R177.
73. Baele PL, McMichan JC, Marsh HM, Sill JC, Southorn PA: Continuous monitoring of mixed venous oxygen saturation in critically ill patients. *Anesth Analg* 1982, **61**:513–517.
74. Mesquida J, Masip J, Gili G, Artigas A, Baigorri F: Thenar oxygen saturation measured by near infrared spectroscopy as a noninvasive predictor of low central venous oxygen saturation in septic patients. *Intensive Care Med* 2009, **35**:1106–1109.
75. Podbregar M, Mozina H: Skeletal muscle oxygen saturation does not estimate mixed venous oxygen saturation in patients with severe left heart failure and additional severe sepsis or septic shock. *Crit Care* 2007, **11**:R6.

76. Mesquida J, Gruartmoner G, Martinez ML, Masip J, Sabatier C, Espinal C, Artigas A, Baigorri F: **Thenar oxygen saturation and invasive oxygen delivery measurements in critically ill patients in early septic shock.** *Shock* 2011, **35**:456–459.
77. Leone M, Bliidi S, Antonini F, Meyssignac B, Bordon S, Garcin F, Charvet A, Blasco V, Albanese J, Martin C: **Oxygen tissue saturation is lower in nonsurvivors than in survivors after early resuscitation of septic shock.** *Anesthesiology* 2009, **111**:366–371.
78. Thoof A, Favory R, Salgado DR, Taccone FS, Donadello K, De Backer D, Creteur J, Vincent JL: **Effects of changes in arterial pressure on organ perfusion during septic shock.** *Crit Care* 2011, **15**:R222.
79. Dellinger RP, Levy MM, Carlet JM, Bion J, Parker MM, Jaeschke R, Reinhart K, Angus DC, Brun-Buisson C, Beale R, et al: **Surviving Sepsis Campaign: international guidelines for management of severe sepsis and septic shock: 2008.** *Crit Care Med* 2008, **36**:296–327.
80. Nanas S, Gerovasili V, Dimopoulos S, Pierrakos C, Kourtidou S, Kaldara E, Sarafoglou S, Venetsanakis J, Roussos C, Nanas J, Anastasiou-Nana M: **Inotropic agents improve the peripheral microcirculation of patients with end-stage chronic heart failure.** *J Card Fail* 2008, **14**:400–406.
81. Gerovasili V, Drakos S, Kravari M, Malliaras K, Karatzanos E, Dimopoulos S, Tasoulis A, Anastasiou-Nana M, Roussos C, Nanas S: **Physical exercise improves the peripheral microcirculation of patients with chronic heart failure.** *J Cardiopulm Rehabil Prev* 2009, **29**:385–391.
82. Thomson SJ, Cowan ML, Forton DM, Clark SJ, Musa S, Grounds M, Rahman TM: **A study of muscle tissue oxygenation and peripheral microcirculatory dysfunction in cirrhosis using near infrared spectroscopy.** *Liver Int* 2010, **30**:463–471.
83. Napoli AM, Machan JT, Forcada A, Corl K, Gardiner F: **Tissue oxygenation does not predict central venous oxygenation in emergency department patients with severe sepsis and septic shock.** *Acad Emerg Med* 2010, **17**:349–352.
84. Lopez PP, Jonas RB, Muir MT, Cohn SM: **The impact of radial artery catheterization on thenar muscle tissue oxygen monitoring.** *J Trauma* 2009, **67**:895.
85. van Beekvelt MC, Borghuis MS, van Engelen BG, Wevers RA, Colier WN: **Adipose tissue thickness affects in vivo quantitative near-IR spectroscopy in human skeletal muscle.** *Clin Sci (Lond)* 2001, **101**:21–28.
86. Kurth CD, Thayer WS: **A multiwavelength frequency-domain near-infrared cerebral oximeter.** *Phys Med Biol* 1999, **44**:727–740.
87. Bezemer R, Lima A, Myers D, Klijn E, Heger M, Goedhart PT, Bakker J, Ince C: **Assessment of tissue oxygen saturation during a vascular occlusion test using near-infrared spectroscopy: the role of probe spacing and measurement site studied in healthy volunteers.** *Crit Care* 2009, **13**(Suppl 5):S4.
88. Gomez H, Mesquida J, Simon P, Kim HK, Puyana JC, Ince C, Pinsky MR: **Characterization of tissue oxygen saturation and the vascular occlusion test: influence of measurement sites, probe sizes and deflation thresholds.** *Crit Care* 2009, **13**(Suppl 5):S3.
89. Dahlgren G, Ohqvist G, Brodin LA, Fischerstrom A, Settergren G: **Isoflurane and intravenous anesthesia used for induction before coronary artery bypass grafting.** *Acta Anaesthesiol Scand* 1989, **33**:99–104.
90. Harrois A, Huet O, Duranteau J: **Alterations of mitochondrial function in sepsis and critical illness.** *Curr Opin Anaesthesiol* 2009, **22**:143–149.
91. Fink MP: **Cytopathic hypoxia. Is oxygen use impaired in sepsis as a result of an acquired intrinsic derangement in cellular respiration?** *Crit Care Clin* 2002, **18**:165–175.
92. Brealey D, Singer M: **Mitochondrial dysfunction in sepsis.** *Curr Infect Dis Rep* 2003, **5**:365–371.
93. Ince C: **The microcirculation is the motor of sepsis.** *Crit Care* 2005, **9**(Suppl 4):S13–S19.
94. Trzeciak S, Dellinger RP, Parrillo JE, Guglielmi M, Bajaj J, Abate NL, Arnold RC, Colilla S, Zanotti S, Hollenberg SM: **Early microcirculatory perfusion derangements in patients with severe sepsis and septic shock: relationship to hemodynamics, oxygen transport, and survival.** *Ann Emerg Med* 2007, **49**:88–98. 98 e81–82.

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