

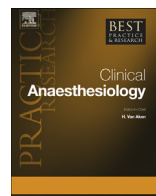


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# Monitoring the microcirculation in critically ill patients



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Alterations in microvascular perfusion have been identified in critically ill patients, especially in sepsis but also in cardiogenic shock, after cardiac arrest, and in high-risk surgery patients. These alterations seem to be implicated in the development of organ dysfunction and are associated with outcome. Even though microvascular perfusion can sometimes be homogeneously decreased as in acute hemorrhage or in non-resuscitated cardiogenic shock, heterogeneity of perfusion is observed in sepsis and in resuscitated hemorrhagic/cardiogenic shock. Heterogeneity of perfusion has major implications for monitoring, as many techniques cannot detect microcirculatory alterations when heterogeneity of flow is present in significant amount. Indeed, devices such as laser Doppler or O<sub>2</sub> electrodes and near-infrared spectroscopy have a relatively large sampling volume and measurements are affected by the highest values in the field. Using these techniques during a vascular occlusion test may help to characterize microvascular reactivity; however, microvascular reactivity sometimes fails to represent actual microvascular perfusion. Videomicroscopic techniques can nowadays be applied at bedside but are still restricted to some selected patients (quiet or sedated patients). Tissue PCO<sub>2</sub> is an elegant alternative but is not yet broadly used. In this manuscript, we discuss the main advantages and limitations of the techniques available for bedside evaluation of the microcirculation in critically ill patients.

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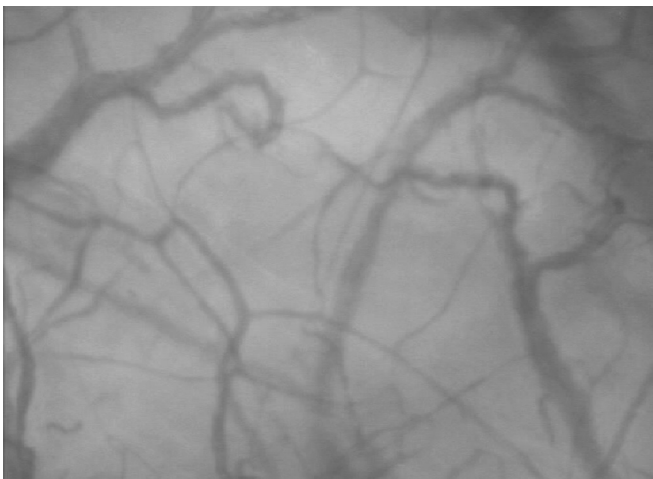
## Introduction

Shock is characterized by an impairment in tissue perfusion, of various causes, leading to impaired metabolism and organ dysfunction, and associated poor outcome [1]. Even though there is no doubt that initial therapy should aim at achieving some minimal values of arterial pressure and cardiac output, further increasing blood pressure and/or cardiac output, once these initial goals are reached, often fails to improve tissue oxygenation and outcome [2]. There are basically three levels in the journey of oxygen from the heart to the cells. The first level is the systemic circulation, constituted by cardiac output, oxygen content, and blood pressure. The second level is at the regional level, mostly constituted by the microcirculation, which is responsible for the distribution of flow inside each organ. The third level is at the mitochondrial level, which constitutes cytochrome chain and responsible for oxygen utilization. Recent trials have shown that goal-directed therapy aiming at optimizing systemic oxygen transport failed to improve survival [3,4]. This suggests that alterations either in microvascular perfusion or in oxygen utilization were not improved by optimization of systemic hemodynamics. Alterations in microvascular perfusion frequently occur in critically ill patients, and especially in patients with sepsis [5]. As these often persist after the correction of systemic hemodynamic abnormalities [5,6], and as their severity is associated with a poor outcome [5–9], it is important to raise awareness on the characteristics of the diseased microcirculation and on the tools available to evaluate the microcirculation “at the bedside”.

Different techniques can be used to evaluate microcirculation at bedside. The choice of the device or technique should be guided by the expected type of alteration, which mostly depends on the underlying disease (i.e., sepsis, trauma). In this manuscript, we discuss the techniques that are the most currently used at the bedside to assess the microcirculation in critically ill patients.

## Microcirculation alterations in critically ill patients

Normal microcirculation is characterized by a dense network of perfused capillaries (Fig. 1). In normal conditions, there is minimal heterogeneity, most of the visualized capillaries being perfused even though flow in the various capillaries varies according to metabolic needs of the surrounding tissues. Adaptation to metabolic needs occurs by opening and closing capillaries, and adapting the velocity of circulating cells within these. Modulation of precapillary sphincters is partly under the influence of systemic factors, with sympathetic stimulation and circulating substances but most of



**Fig. 1.** Sublingual microcirculation of a healthy individual. Visualization of sublingual microcirculation of a healthy individual. Note the rich density of capillaries, all of which are perfused.

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