NARRATIVE REVIEW

BEDSIDE ASSESSMENT AND MANAGEMENT OF MICROCIRCULATION IN SEPTIC SHOCK

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ABSTRACT

Abnormalities of microcirculation in septic shock is one of the highlighted features of the condition. Tissue hypoperfusion and inadequate oxygen delivery at the cellular levels contributes to an oxygen debt and the multi-organ dysfunction seen in this condition. Clinically, the tissue oxygen delivery is manipulated using a combination of fluids, vasopressors and inotropes and this is measured using stroke volume, cardiac output and mean arterial pressure. These parameters indicate the oxygen delivery to the tissues. The downstream parameters including the centra venous oxygen saturation and the mixed venous oxygen saturation indicates the oxygen content of the venous blood. The difference between the two is assumed to be consumed by the tissues. However, in sepsis this may be an oversimplification of the situation. In such situations, bedside evaluation of the microcirculation becomes important. Although some sophisticated evaluation techniques are available which is being used on experimental basis, bedside evaluation of the microcirculation is something that can be done in any intensive care unit. This is a narrative review of some of these simple bedside tests that can be used for assessment of the microcirculation.

Keywords: Shock;Septic shock; Hemodynamics; Resuscitation; Microcirculation; Vasoconstriction

KEY MESSAGE: There is incoherence between macrodynamics and microdynamics in septic shock which can lead to multi-organ dysfunction and mortality. In this review we will highlight various simple bedside monitoring and management of microcirculation.

Introduction

International guidelines suggest that early recognition and management of septic shock is associated with improved outcomes.[1] Early management includes restoration of hemodynamic stability, antibiotics and source control. Hemodynamic stabilization includes fluid therapy and use of vasopressors to

restore mean arterial pressure (MAP), cardiac output (CO), central venous oxygen saturation (ScvO2) or mixed venous oxygen saturation (SVO2). [1] This is basically restoring the macro dynamics back to normal. Unfortunately, there is incoherence between the macrodynamics and microdynamics in septic shock.[2]
During septic shock, the sympathetic response of the body redirects the blood flow to the vital organs including the brain, heart and the kidney at the expense of the skin, muscle and splanchnic circulations, resulting in abnormal microcirculation. This is responsible for the multi-organ dysfunction and high mortality seen in septic shock. [3] Experimental studies using capillaroscopy have shown significant heterogeneity of the microcirculation in shock, highlighting the great disparity between macro- and micro dynamics. This underscores the importance of assessment of regional perfusion and oxygen delivery at the cellular level.[3] While sophisticated equipment's are available for assessment of the microcirculation, it is not for everyday use in all intensive care units (ICU's). Bedside assessment of changes in microcirculation is therefore very important in assessment of patients in shock.

In this review we will highlight various simple bedside monitoring of microcirculation.

Gaps in the existing protocols for management of circulatory shock.

The Surviving Sepsis Campaign suggests that initial resuscitation with fluid therapy, vasopressors and inotropes helps restore the upstream parameters to normal (MAP > 65 mmHg, CO > 2.2 L.min.m2 and maximization of oxygen delivery (DO2)). [1] This does not guarantee normalization of downstream parameters which include the SVO2 (> 65%), ScvO2 (> 70%), lactate (<2 mmol/L) and the arteriovenous CO2 difference (<6-8 mmHg). The combined use of upstream and downstream parameters acts as "bridge to knowledge" on the microcirculation. It is assumed that if the hemoglobin levels and hemoglobin saturations are constant, a reduction in ScvO2 /SVO2 is usually due to a reduction in CO.

Unfortunately, none of these macrodynamic parameters are a window to what happens at the microcirculatory level.[3]

The normal "microcirculation"

The microcirculation and endothelium are the largest organ system in the body, consisting of functional units namely arterioles, capillaries and venules. They are 100-150 m in diameter. Arteriolar length and density vary with the type of organ involved and are responsible for the vascular tone and systemic vascular resistance (SVR). [2,3]

Microcirculatory perfusion is controlled by 3 mechanisms which include myogenic, neuro-humeral and metabolic control. [2] Myogenic control is based on the inherent capacity of the vessel to constrict or dilate in response to intra-luminal pressure and is influenced by the shear stress induced release of nitric oxide (NO) from the endothelium. The metabolic control involves matching the metabolic requirement of the organ with the blood flow. Hypoxia indices release of NO and endothelium derived hyperpolarizing factor from endothelium and red cells (vasodilatation). Factors like adenosine, lactate, H+ and K+ induce a delayed vasodilator response. The neuro-humeral control is dependent on the vessel size and distribution of adrenergic receptors. The first order arterioles are responsible for the blood flow to the entire organ and therefore, for the systemic vascular resistance (SVR) and MAP. Regional blood flow is however, controlled by the third order, small terminal arterioles. [2]

Only 20-30% of the capillaries function under normal resting conditions. However, during hypoxemia, there is a rapid recruitment of capillaries due to relaxation of precapillary sphincters that results in a dynamic environment at the level of the capillary network. The functional capillary (FCD) density is the number of capillaries that is traversed by red cells at a time. Changes in FCD is linked to the metabolic requirements of the tissue. Once the terminal arterioles are fully dilated, more proximal arterioles (first generation arterioles) dilate and results in increase bulk flow to tissues. Reduction in

FCD reduce the capillary surface area for gas exchange, increase diffusion distance and increase arterio-venous shunting. (2)
The venules play an important role in immune mechanism. Due to their high capacitance and distensibility, they act as a reservoir for large amounts of blood. Finally, blood rheology is not only important for the pattern of blood flow in the capillaries but also for the FCD. The later can influence the amount of oxygen and nutrients being transferred to the mitochondria of cells. (2)

Microcirculation in pathological conditions

During shock, there is significant dissociation between the macrocirculation and microcirculation. However, the type of changes at the microcirculatory level is dependent on the type of shock. In cardiogenic shock, FCD and arteriole diameter are reduced.

The microcirculatory changes are early in hemorrhagic shock. The immediate response in traumatic shock is a reduction in tissue perfusion (CI) and reduced oxygen delivery (DO2) except for the vital organs. When the critical levels of DO2 is reached, the lactate levels start increasing. Thus, in these patients there is reduced CO, DO2, ScvO2 and SVO2 with elevated lactates.

In septic shock, heterogeneity of tissue perfusion is the characteristic feature. FCD decreases resulting in some of the capillaries closing. Hence, although the CO and DO2 is increased, the reduced FCD results in cells near the functional capillaries being able to meet the oxygen demands but the cells further away may not be able to receive adequate O2. This results in generation of an oxygen debt in the tissues. Although oxygen extraction may be increased, it will not meet the metabolic requirements of the entire tissue. Hence, tissue hypoxemia can co-exist in the presence of a normal or elevated CO, DO2 and SVO2. (2).

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Basis of bedside monitoring of microcirculation

During shock, blood flow is re-directed from the skin, muscle and splanchnic region to maintain the perfusion of vital organs. Unlike the vital organs, which have an autoregulatory mechanism the skin and muscle is endowed only with sympathetic fibers and activation of the sympathetic system is usually associated with vasoconstriction. The hepatosplanchnic circulation is highly sensitive to the adrenergic response during shock which is mediated through sympathetic stimulation, activation of the renin-angiotensin mechanism and release of vasopressin. This leads to splanchnic vasoconstriction, which, if prolonged, can lead to massive translocation of inflammatory mediators & intestinal

bacteria to the systemic circulation. However, unlike the skin, this territory offers some degree of flow autoregulation that offers certain degree of protection during circulatory dysfunction. (4)

In. study of 30 patients in septic shock, Brunauer et al compared bedside indices of microcirculation namely skin temperature, capillary refill time (CRT) and mottling score with ultra-sound based resistive index (RI) from the kidney, spleen and intestine up to 72 hours after intensive care unit admission. RI is a Doppler based evaluation of organ perfusion and values > 0.7 indicates increased vasoconstriction. The first assessments were made 10 hours after initial resuscitation. There was excellent correlation between the bedside indices of microcirculation with each other and with RI, lactate levels, urine output, base deficit and Sequential Organ Failure Assessment (SOFA) score. (5) The study concluded that peripheral indices are good indicators of visceral perfusion.

Bedside Indices of Microcirculation

Skin temperature as an indicator of microcirculation

a) Subjective assessment of skin temperatures:
Cold clammy extremities are often seen in patients who are in shock and correlates well with serum lactate levels and SVO2. (6) In septic patients, persistence of cold extremities after 48 hours of resuscitation is associated with higher organ dysfunction but not death. (7) Successful resuscitation of shock states is also associated with improvement in skin temperatures. The dorsal surface of the clinician's hand or fingers should be used for assessment of skin temperature as this area is more sensitive. Skin temperature is normally classified as cold, slightly reduced, normal and warm. However, it is observer dependent and influenced by ambient temperature.

b) Temperature gradients:
Core-to-peripheral (dTc-p), peripheral-to-ambient (dTp-a) and forearm-to-fingertip (Tskin-diff) temperature gradients provide an objective assessment of outcome. The dTp-a gradient decreases and dTc-p gradient increases when peripheral vasoconstriction (shock) increases provided ambient temperature remains constant. The higher the dTp-a, the greater is the CO, the chances of survival and better peripheral perfusion. Survival is associated with a dTp-a gradient of at least 20C.The peripheral temperature is measured on the ventral surface of the big toe as it is away from other monitors and has negligible local heat production.

The dTc-p is based on the concept that conduction of heat from the core to the surface is dependent on the degree of vasoconstriction. When vasoconstriction is present, heat conduction is minimal and the core to surface temperature gradient increases and converse happens in vasodilatation. The normal dTc-p is 3-70C. [3] However, cold ambient temperature and vasodilatory shock can interfere with this assessment. (8)

T skin-diff overcomes the problems of ambient temperature. Here fingertip and the forearm are exposed to the same ambient temperature. The fingertip probe is attached to the ventral surface of the finger and the forearm probe, to the radial aspect of the forearm. When vasoconstriction decreases fingertip blood flow, the fingertip temperature decreases and the T skin-diff increases. Studies have shown a Tskin-diff of 00 at the beginning of vasoconstriction and 40 for severe vasoconstriction. (9)

Capillary Refill Time (CRT).

CRT is defined as the time taken for the skin color (distal right or left index finger) to return to normal after firm pressure has been applied for a defined period. A delayed CRT indicates impaired microcirculation. The normal cut off values are < 2 secs in infants and healthy young adults, 2.9 secs for adult female and up to 4.5 secs for elderly (7). In order to reduce inter-rater variability, standardization of applied pressure and time is required. The applied pressure, sustained for 15 seconds, should remove blood at the fingertip of the physician's nail, characterized by the appearance of a thin white

distal crescent under the nail. (7).

In a study of 549 septic shock patients, Ait-Oufella showed significant inter-rater concordance calculated at 80% for index CRT and 95% for knee CRT. CRT measured at both sites significantly corelated with lactate levels, SOFA scores and mortality at 14 days (10) The CRT at 6 hours of resuscitation was highly predictive of 14-day mortality with an AUC of 84% for CRT index finger and 90% for knee CRT. A threshold CRT of 2.4 secs predicted 14-days mortality with a sensitivity and specificity of 84% and 73%. A knee CRT cut-off of 4.9 secs predicted 14-day mortality with a sensitivity of 82% and specificity of 84%.

In the ANDROMEDA-SHOCK trial (11) 416 patients with septic shock were resuscitated targeting either lactate levels or CRT. Normalization of CRT versus reduction of lactate by 20% every 2 hours were the goals in the two groups. The primary objective was mortality at 28 days and secondary objectives included 90-day mortality and organ failure scores. Although there was no statistically significant difference in mortality, organ failure scores were significantly lower in the CRT group. Lower lactate levels with less resuscitation fluid requirements were seen in the CRT group.

Skin Mottling

In critically ill patients, mottling of the skin is described as a patch discoloration of the skin around the knee. In the absence of diffuse intravascular coagulation, this is due to heterogenic constriction of microvasculature. Ait-Oufella et al developed a scoring system which ranged from 0-5 with scores of 5 indicating extension of mottling beyond the inguinal ligament. Mottling score had very high inter-observer agreement and was predictive of mortality even when systemic vasodilatation is a feature. Six hours after initial resuscitation, mottling score could predict 14-day mortality in patients with septic shock with an odds ratio (OR) of 16 for a score of 2-3 and OR of 74 for scores of 4-5. (12) Death occurred within 12-24 hours for scores of 4-5 and within 24-72 hours for scores of 2-3.

Mottling scores did not correlate with MAP or CO, highlighting the disparity between macro and micro-circulation.

This was confirmed in another South African study which showed that mortality was 100% with mottling score 4 or more, mortality of 77% for scores of 2-3 and 45% for scores of 1. [13]

In 42 cirrhotic patients with shock, mottling scores were the strongest predictors of death at 6 hours after resuscitation. [14] However, their sensitivity was lower due to delayed appearance of mottling probably related to the pre-existing vasodilatory state. Persistence of mottling greater than 2, beyond six hours of resuscitation is a predictor of mortality.

Peripheral perfusion index (PPI)

The PPI is derived from the photoelectric plethysmographic signal of pulse oximetry. It is the ratio between pulsatile and the non-pulsatile component of the pulse oximetry and is independent of the patient's oxygen saturation. PPI reflects the relation between sympathetic and parasympathetic system. An increase in sympathetic stimulation can increase vascular tone and decrease the ratio between the two. Although septic shock may be associated with a state of vasodilatation, this does not apply to the peripheral microcirculation and therefore a lower ratio is indicative of vasoconstriction. A PPI < 1.4 was indicative of poor tissue perfusion. (15)

In patients with septic shock, a PPI < 0.3 is an indication for vasopressor therapy and a value < 0.2 is predictive of mortality. (16) In patients who have been resuscitated after out-of-hospital cardiac arrest, the PPI at 30 minutes after ROSC correlates with patient outcome. PPI has also been used as an indicator of fluid responsiveness and also for assessment of effectiveness of peripheral nerve blocks.

The major limitation with PPI is that it may be unreliable in patients with cold extremities where the signals may be affected. It cannot be used in patients on extra corporeal membrane oxygenation.

The factors affecting PPI are the CO and the balance between the sympathetic and parasympathetic activity, either of these has to remain constant for a relevant value.

Tissue oxygen saturation (StO2) and Vascular Occlusion test (VOT)

NIRS electrodes, when used in assessment of microcirculation, is applied directly on the thenar muscles and reflects the saturation from the entire microcirculation including arterioles, venules and capillaries. The basal StO2 values which represents the local balance between the oxygen delivery and consumption are taken after 3-5 minutes of stabilization.

Vascular occlusion test (VOT) is dynamic and specific and can be done in two ways. In the time targeted approach, the cuff pressure is elevated to 30 mmHg above systolic pressure for 3 minutes followed by its release. Maximum ischemic response is achieved in a few minutes and patient discomfort arising from prolonged ischemia of the arm is avoided. In the StO2 targeted approach the cuff pressure is elevated above systolic pressure till the StO2 value drops to <40%. The advantage is that a standardized level of ischemia is achieved in all patients thereby reducing inter-individual variations.

The baseline StO2 reflects the level of saturation existing at rest. The rate of desaturation in the thenar muscles (Rdes: StO2%/sec) after vascular occlusion can be used to assess the oxygen consumption (VO2) in the thenar during the brief period of arterial occlusion during which vasodilatation occurs in the arterioles, meta-arterioles and precapillary spncters. The ascending limb (Rres: StO2%/sec) signifies the increase arterial inflow following the period of ischemia. However, the Rres overshoots the baseline during the recovery phase. This reactive hyperemia is related to the microvascular reserve in additionally recruited capillaries. The area under reactive hyperemia denotes the oxygen extraction capacity, reflecting the extent of hyperemic response when the capillaries are all dilated. (17) Baseline StO2 values < 70% are usually associated with more sick patients requiring ICU admission. (17).

In a study of 170 patients with shock, sepsis or a control group, Shapiro et al showed that the recovery slopes in septic shock patients were significantly lower with impaired oxygen recovery. (18) In addition, the Rdes was slower (less steep) signifying reduced oxygen consumption by the tissues during the vaso-occlusive phase of the dynamic test. These findings indicate reduced microcirculation in shock with reduced ability to exchange and deliver oxygen. In this study, the initial StO2 was not significantly different between groups.

Ultra sound-based evaluation at bedside.

Ultrasound based techniques can evaluate the splanchnic circulation. Using Doppler techniques, the resistive index (RI) can be calculated which is the resistance offered by the microvasculature to the blood flow.

The renal Doppler resistive index (RDRI) is an indicator of renal perfusion. It is useful in predicting acute kidney injury (AKI) in patients with sepsis. A curved probe with low frequency is used and a pulsed wave Doppler is used to interrogate the blood flow. The higher the RI, the greater the degree of vasoconstriction and chance of organ failure. In normal situations, the RI is < 0.7. In patients who are hypo-perfused, the diastolic flow will decrease, and the RI will increase whereas in patients who are under-perfused (severe hypovolemia), only the systolic flow will be present with no diastolic flow resulting in very high RI.

CONCLUSION

Resuscitation in any patients who is in shock is aimed at restoring the circulating blood volume and attaining an adequate CO, CI and MAP (65 mmHg). Once target variables are attained, the SVO2 and ScvO2 along with lactate and P(v-a) CO2 measures

give an idea whether the increased DO2 is met with an increase in tissue oxygen uptake. If the ScvO2 or P(a-v) CO2 remains low, it is an indication that DO2 is inadequate. If ScvO2 / SVO2 is too high and the P(a-v) CO2 difference is high, it is an indication that there is peripheral shunting of oxygenated blood and the microcirculatory oxygen delivery is inappropriate. Indices of tissue perfusion including the CRT, mottling score, VOT, PPI and Doppler based PI are good bedside indices of the microcirculation and organ perfusion. There are a few drugs that can be used to improve organ perfusion although more studies are required to establish a basis for their regular use. Management of shock using bedside indicators of microcirculation should be a must rather than an option in critically ill patients.

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