Editorial

Microcirculation: A New Focus

Prompt and aggressive fluid resuscitation with precise central venous pressure (CVP) monitoring is a common practice in shock states. The development of multi-organ failure is a direct consequence of tissue hypoperfusion and inadequate oxygen transport. The type and amount of fluid administered has traditionally been guided by hemodynamic variables linked to the systemic circulation. It was believed that optimization of cardiac output would improve the convective transport of oxygen to the tissues. This belief has recently been questioned.

Presence of normal CVP and systolic blood pressure (SBP) does not exclude an abnormality of tissue oxygenation. Several studies have indicated that derangements of microcirculation and its regulation contribute to organ failure. The focus has thus shifted to abnormalities in microcirculation. These are reduced vessel density and altered blood flow due to failure of autoregulation. These alterations in microcirculation are primarily due to endothelial malfunction and glycocalyx rupture. Microthrombi, capillary leakage, leucocyte rolling and rouleaux formation also contributes to the derangement. Functional capillary density (FCD) refers to the density of capillaries with flowing red blood cells carrying oxygen. Excessive fluid therapy could be counterproductive by reducing the FCD and consequently the oxygen diffusing to the cells.[1]

It is now generally accepted that resuscitation has to aim at restoration of a normal microcirculation. Recruitment of microcirculation consists of techniques that not only open occluded vessels but also prevent heterogeneity in flow. Evaluation of the status of microcirculation has become possible recently due to the development of optical techniques such as side stream dark field (SDF). Several strategies to recruit microcirculation have been tried with conflicting results. These were included but are not limited to the use of hydrocortisone, vasopressors like norepinephrine and dobutamine, nitroglycerin, intravenous fluids, red cell transfusion, and drotrecogin.[2]

It is important to realize that hypoperfusion can be worsened by anesthetic agents and techniques such as mechanical ventilation, vasoactive drugs, sedatives and opioids. The last few decades have seen the elucidation of non-anesthetic effects of anesthetic agents particularly on microcirculation. Alterations in microvascular reactivity, nitric oxide pathways and cytokine release are a few possible pathways for such effects.[3] Further research is required in the form of controlled clinical trials to suggest clear protocols for the anesthetist and the intensivist who frequently manage such situations.

REFERENCES

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