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Monitoring microcirculation

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The clinical relevance of microcirculation and its bedside observation started gaining importance in the 1990s since the introduction of hand-held video microscopes. From then, this technology has been continuously developed, and its clinical relevance has been established in more than 400 studies. In this paper, we review the different types of video microscopes, their application techniques, the microcirculation of different organ systems, the analysis methods, and the software and scoring systems. The main focus of this review will be on the state-of-art technique, CytoCam-incident dark-field imaging, and the most recent technological and technical updates concerning microcirculation monitoring.

Introduction

Microcirculation has been a source of interest since its description by Malpighi and van Leeuwenhoek in the 17th century. Clinical relevance and bedside observation, however, have gained importance since the 1990s following the introduction of hand-held vital microscopes (HVMs). Form then, the technology has been continuously developed, and its clinical relevance has been established in more than 400 studies [1–3]. Microcirculation consists of microvessels that are less than 100 μm in diameter, namely capillaries, arterioles, and venules. Capillaries are less then 10–20 μm in diameter where predominantly oxygen is supplied to the cells to meet the metabolic needs of cells and remove waste products. Arterioles are surrounded by a smooth muscle layer that regulates the blood flow

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under metabolic, myogenic, and humoral control. Venules are downstream collecting vessels that are larger than 20 μm in diameter where the blood flow is the slowest in microcirculation and leukocyte rolling, sticking, and extravasation can be best observed.

Microcirculation is highly adaptive to its cellular environment and continuously interacts with the systemic circulation. Under physiological conditions, vascular regulations ensure a constant blood flow under constant metabolic demand that is independent of changes in the blood pressure, a process called autoregulation. Thus, the O_2 supply from microcirculation is matched to the oxygen needs of the parenchymal cells as they perform functional activity. Resuscitation procedures use these adaptive processes to ensure that therapies are aimed at correcting systemic hemodynamic variables. Ince recently defined the adaptive changes required by the successful resuscitation aimed at systemic circulation as hemodynamic coherence (HC). However, in some conditions such as sepsis, shock reperfusion, and iatrogenic injury, a loss of HC may occur, in which resuscitation based on correction of macrohemodynamic parameters may not result in simultaneous improvement in the tissue perfusion and even cause further harm during conditions such as hemodilution, edema, and tamponade due to high venous pressures or vasoconstriction of the arterioles. On the basis of these ideas, the following four types of HC losses are described (Fig. 1): (i) heterogeneity (i.e., sepsis), (ii) hemodilution (i.e., iatrogenic excessive fluid administration), (iii) vasoconstriction/tamponade (i.e., iatrogenic vasopressors or tamponade of the microcirculation due to targeting high venous pressures), and (iv) tissue edema [4].

The technology underlying imaging with hand-held microscopy has advanced considerably in recent years [5,6]. In addition, other microcirculatory techniques have been introduced to identify the

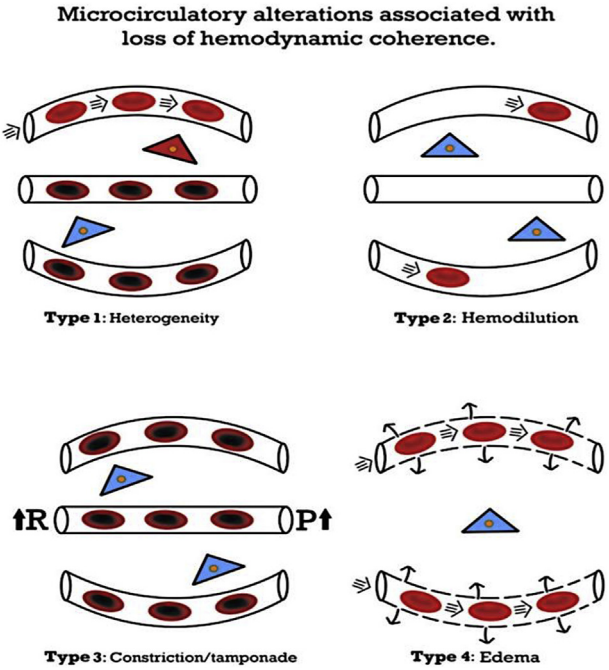

Fig. 1. Conditions in microcirculation where there is a loss of HC between the systemic circulation and microcirculation. Here, targeting the macrocirculation during resuscitation procedures does not result in parallel resuscitation of the microcirculation. Type 1 loss of HC occurs during states of inflammation, such as sepsis, where there is endothelial and red blood cell dysfunction, resulting in the plugging of vessels and a heterogeneous microcirculatory flow that causes functional shunting and a deficit in oxygen extraction. Type 2 loss of HC occurs during excessive fluid administration, which causes a reduction in the blood viscosity and dilution of blood. As a result, microvessels are not filled with oxygen-carrying red blood cells. Type 3 loss of HC occurs during excessive vasopressor therapy, resulting in vasoconstriction or venous tamponade caused by high venous pressures. In type 4 loss of HC, extravasation of fluid increases the diffusion distance.