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Essay

Restoring microcirculation in anesthesia: Impact, usefulness and controversies☆

Diana Patricia Ñáñez-Varona[a,*], *Adolfo León Tróchez-Zuleta*[b], *William Andrés Vargas-Garzón*[c]

[a] *Anesthesiologist, Hospital Susana López de Valencia, Popayán, Colombia*
[b] *Anesthesiologist, Specialist in Medicine and Critical Care, Intensive care specialist at Clínica La Estancia, Instructor at Universidad del Cauca, Popayán, Colombia*
[c] *Anesthesiologist, Hospital Universitario San José, Popayán, Colombia*

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ABSTRACT

Mentioning microcirculation and endothelial dysfunction to explain the pathophysiology of diseases whose relationship was not well understood is a recent phenomenon. Microcirculation is not only compromised by disease but can be altered by conditions that the anesthesiologist faces (agents and anesthetic techniques). There is significant and increasing evidence that anesthetic agents may alter it. The importance? In a state of hypoperfusion, it can be worsened by other factors (mechanical ventilation, vasoactive medications, sedatives, opioids). In shock's late stage, the support to tissue perfusion given by peripheral circulation is weak and disappears. Therefore, is it beneficial to direct targeted therapies only toward macrovascular goals? Methods for identifying early alteration and direct therapies for restoration are important. The clinical evaluation is rapid and reproducible, and measuring body temperature determines alteration indirectly. There are other methods to determine microcirculation objectively: nowadays, optical evaluation techniques using polarized orthogonal spectral light and sidestream dark-field are the best approach. In hemorrhagic shock the degree of organ dysfunction is determined by microvasculature's alteration. Compensatory mechanisms exist for this purpose, making its measurement and use in perioperative period important. Strategies have been studied to improve tissue perfusion (recruitment of microcirculation). The recentness of the study of microcirculation calls its usefulness into question. It is necessary to determine the clinical impact through controlled clinical trials with protocols on resuscitation strategy, which can complement the current perioperative anesthetic practice.

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* *Corresponding author at*: Departamento de Anestesiología, Hospital Universitario San José, Carrera 6 No. 10N-142, Popayán, Colombia.
E-mail address: diana_pato24@hotmail.com (D.P. Ñáñez-Varona).

Reanimando a la microcirculación en anestesia: impacto, utilidades y controversias

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RESUMEN

Es reciente la mención de la microcirculación y disfunción endotelial para explicar la fisiopatología de padecimientos de los cuales no se entendía su interrelación. La microcirculación no solo está comprometida en enfermedad sino que puede alterarse por condiciones a las que se enfrenta el anestesiólogo (agentes y técnicas anestésicas). Existe evidencia importante y creciente de que agentes anestésicos pueden alterarla; ¿su importancia?: en hipoperfusión puede empeorarse por otros factores (ventilación mecánica, vasoactivos, sedantes, opioides). En fase tardía del choque la participación de la circulación periférica soportando la perfusión tisular es débil hasta desaparecer; entonces: ¿es benéfico encaminar las terapias dirigidas por metas sólo macrovasculares? Se hacen importantes métodos que identifiquen su alteración tempranamente y encaminar terapias para su restauración: la evaluación clínica es rápida y reproducible; la medición de temperatura corporal determina indirectamente su alteración; existen otros métodos de evaluación de la microcirculación más objetivos: hoy en día la evaluación óptica mediante técnica con luz polarizada ortogonal espectral y campo oscuro lateral son la mejor aproximación. En choque hemorrágico es la alteración de la microvasculatura la que determina el grado de disfunción multiorgánica; para ello existen mecanismos compensatorios, lo cual reviste la importancia de su medición perioperatoria y uso en anestesia. Se han estudiado estrategias que mejoran la perfusión tisular (reclutamiento de la microcirculación). Por ser reciente su estudio se controvierte su utilidad; es necesario determinar el impacto clínico mediante ensayos clínicos controlados en protocolos de estrategia de reanimación, lo cual puede complementar la práctica anestésica perioperatoria actual.

Introduction

Recently in medicine, microcirculation and endothelial dysfunction have been mentioned to explain the physiopathology of conditions whose interrelationship was not previously understood. In the complex, integrated response, systems conforming a proinflammatory group and a prothrombotic group interact; the endothelium and microcirculation are the mediators of this interaction. When it is altered, it leads to collapse.[1] Microcirculation is compromised in conditions that anesthesiologists face frequently. There is important and growing evidence that anesthetic agents can alter it.[2] The advance in imaging techniques allows for its evaluation at the patient's bedside. Since these conditions are associated with a poor prognosis, regardless of the overall hemodynamic variables, evaluating microcirculation is important in anesthesiology to understand how different anesthetic techniques impact microcirculation and its usefulness as a possible perioperative monitor in critical patients.[3]

Historical perspective

Knowledge of blood circulation began in ancient Egypt and Greece. Galen of Pergamon (2nd century BCE) proposed that blood was the "vital spirit" that is exhaled by the lungs. Ibn al Nafis (1210–1288) describes pulmonary circulation, stating that between the pulmonary artery and the pulmonary vein "there may be imperceptible passageways". This is perhaps the first mention of the notion of microcirculation. The idea of the heart as a pump came in 1628 with Sir William Harvey.[4]

Current knowledge

Hypoperfusion is the insufficient transport of nutrients and oxygen necessary for normal cell activity. Microcirculation makes up more than 90% of the endothelium, and is composed of small vessels (<100 μm in diameter), arterial capillaries and venules, forming a vascular network that regulates the distribution of blood flow to each organ, exchanging gases, nutrients and products of cell catabolism. It is also involved in the interaction of leukocytes, platelets, and plasmatic components.[1]

Its importance? Hypoperfusion can worsen do to other factors such as mechanical ventilation, vasoactive drugs, sedatives, and opioids used regularly in anesthetic practice and in critical patients. Some tissues do not have the capacity to self-regulate blood flow, being dependent on mechanisms of sympathetic vasoconstriction. In the initial phase of shock, the compensatory mechanism is vasoconstriction through neurohumoral response; in the late phase, the participation of peripheral circulation supporting tissue perfusion is weak to the point of disappearing.[5] From here the question rises, is