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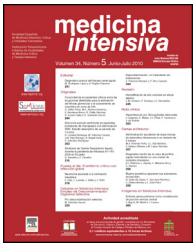

REVIEW

Enteral nutrition in the hemodynamically unstable critically ill patient☆

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Abstract The benefit of enteral nutrition in critically ill patients has been demonstrated by several studies, especially when it is started early, in the first 24–48 h of stay in the Intensive Care Unit, and this practice is currently advised by the main clinical guidelines. The start of enteral nutrition is controversial in patients with hemodynamic failure, since it may trigger intestinal ischemia. However, there are data from experimental studies in animals, as well as from observational studies in humans that allow for hypotheses regarding its beneficial effect and safety. Interventional clinical trials are needed to confirm these findings.

PALABRAS CLAVE
Nutrición enteral;
Shock;
Paciente crítico

Nutrición enteral en el paciente crítico con inestabilidad hemodinámica

Resumen El beneficio de la nutrición enteral en el paciente crítico ha sido demostrado en varios estudios, especialmente si esta es iniciada precozmente, en las primeras 24-48 h de ingreso en la Unidad de Cuidados Intensivos, y en la actualidad esta práctica es recomendada por las principales guías de práctica clínica. El inicio de nutrición enteral en el paciente crítico con inestabilidad hemodinámica es una decisión controvertida, fundamentalmente debido al potencial riesgo de isquemia intestinal asociado a su empleo. Sin embargo, existen datos procedentes de estudios animales y de estudios observacionales en humanos que permiten plantear la hipótesis sobre su efecto beneficioso y seguridad. Son necesarios ensayos clínicos de intervención que establezcan una relación causa-efecto.

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Aims of the review

- To understand the physiopathology of the gastrointestinal tract in the critical patient with hemodynamic instability and the potential benefits of enteral nutrition (EN).
- To review the available evidence on the benefits and risks of EN in patients of this kind.
- To summarize the recommendations of the main clinical practice guides.
- To offer practical ideas and the point of view of a group dedicated to research in this field.

Physiological and physiopathological reminder

The physiology of the splanchnic circulation is complex, but has been known for some years, and a basic understanding of its characteristics is essential in order to correctly interpret the alterations that occur in critical patients with hemodynamic instability. Under resting and normal conditions, 20–25% of cardiac output is located in the splanchnic circulation. In turn, the metabolic activity in this region accounts for 30% of overall body oxygen consumption. When eating, the blood flow in this zone can double thanks to a phenomenon known as postprandial hyperemic response. This response is basically mediated by local factors, giving rise to notorious splanchnic vasodilatation.[1,2] The anatomical configuration of the intestinal microvascularization is complex, with arterial and venous plexuses at mucosal and submucosal level, and in the *muscularis propria*. This system is able to redistribute blood flow in the case of decreased intravascular volume within the systemic circulation, allowing precise regulation of splanchnic blood flow (Fig. 1). In the intestinal villi, the arterial and venous vessels run parallel to each other, though with flow in opposite directions. This anatomical distribution allows the direct passage of low-molecular weight molecules (such as oxygen) from the arterial to the venous circulation, conforming

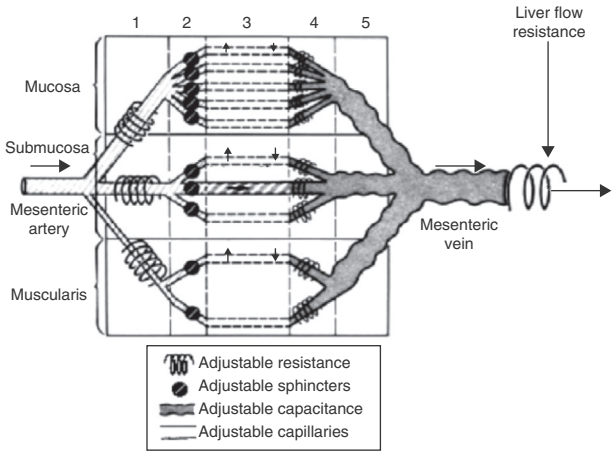

Figure 1 Intestinal microvascularization (I). Schematic representation of the anatomical distribution of the intestinal microvascularization. Regulation can be made at several levels (precapillary, capillary and postcapillary), thus allowing blood flow redistribution.
Figure reproduced from Folkow,[3] with permission.

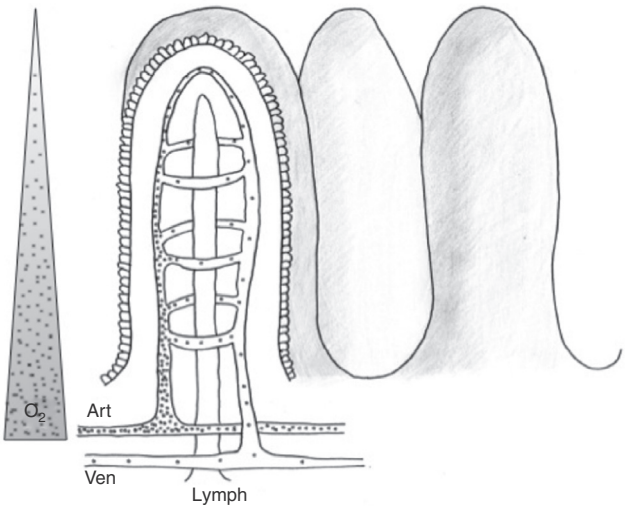

Figure 2 Intestinal microvascularization (II). Schematic representation of the anatomical distribution of the intestinal microvascularization (artery, vein and lymphatic vessel) at villus level. The passage of low-molecular weight molecules (dots) and the countercurrent mechanism are shown. The triangle at left represents the oxygen concentration gradient from villus base to tip.

a ''countercurrent'' exchange mechanism. Although the latter is not relevant under physiological conditions, in the presence of hypoperfusion it generates a decreasing tissue oxygen pressure gradient from the base to the tip of the villus (Fig. 2). As a result, the tip of the intestinal villus is particularly sensitive to tissue hypoxia.

Shock is characterized by blood flow redistribution with vasoconstriction at splanchnic circulatory level and in peripheral tissues, in an attempt to maintain crucial brain and coronary perfusion.[4] This can give rise to an imbalance in the oxygen supply/demand ratio at intestinal level, with resulting ischemia. This situation in turn leads to depletion of the cellular ATP reserves, with rupture of the tight junctions between the epithelial cells. Consequently, the solute concentration gradients between the apical and basolateral compartments are lost, producing intracellular edema, necrosis and apoptosis. The end result of this alteration of the intestinal microcirculation is rupture of the intestinal epithelial barrier, favoring bacterial translocation phenomena. Such persistent alteration of the microcirculation in shock patients[5] can perpetuate the proinflammatory response and favor progression toward multiorgan dysfunction syndrome, which in turn results in increased splanchnic hypoperfusion – thereby completing a vicious circle that can increase the probability of a fatal outcome (Fig. 3). In addition, in this complex scenario, different patient interventions such as vasoactive and inotropic support,[6] mechanical ventilation or the use of hemodynamic support systems, act upon the splanchnic circulation, with consequences that are hard to predict. Considering the above, nutritional support in these patients becomes a genuine challenge. From a conceptual and physiopathological point of view, enteral nutrition (EN) administered to the critical patient with hemodynamic instability would induce an increase in the need for oxygen at intestinal level and in