Introduction

Four basic classes of circulatory shock can be clinically defined: hypovolemic, cardiogenic, obstructive, and distributive. Looking at the physiology of cardiac performance, taking a pathophysiologic approach we can distinguish between hypovolemic shock, distributive shock, systolic cardiogenic shock, diastolic cardiogenic shock, or a mix of them. All these types evolve, if not treated early and adequately, towards end-organ failure (dysoxia, microcirculatory failure). Multi-organ dysfunction syndrome (MODS) accounts for most deaths in the intensive care unit (ICU). Disturbances in systemic hemodynamics and organ perfusion resulting in tissue hypoxia appear to play a key role in the onset and maintenance of MODS.

In critically ill patients, as well as those with MODS, hemodynamic monitoring is a cornerstone of care, with these objectives and priorities: (a) rapid assessment of the determinants of the cardiovascular insufficiency (diagnosis of acute circulatory failure); (b) guidance and titration of cardiopulmonary therapies (treatment algorithm); (c) rapid assessment of regional tissue hypoperfusion, even in a compensated shock patient (i.e, with intrinsic acute and/or chronic circulatory failure); and (d) assessment of the optimization of tissue perfusion. New bedside technologies, more or less invasive, are helping caregivers with increasingly sophisticated and evolving monitoring devices. Nevertheless, despite improvements in resuscitation and supportive care, progression of organ dysfunction occurs in a large proportion of patients with acute, life-threatening illness. Early and aggressive resuscitation of critically ill patients may limit or reverse tissue hypoxia, progression to organ failure,
and improve outcome [1]. Sometimes, however, although blood pressure, arterial oxygenation, central venous pressure (CVP), and cardiac output (CO) may be in the “normal range,” the patient may continue to suffer from inadequate microcirculatory perfusion not reflected by “classic” hemodynamic parameters. Consequently, simultaneous monitoring of global and regional tissue oxygenation is needed, because tissue hypoxia plays a crucial role in the pathogenesis of MODS.

Using a functional approach—because no monitoring device, no matter how simple or how complex, invasive or noninvasive, measuring variables directly or indirectly by signal processing, will improve outcome unless coupled with correct diagnosis and a treatment algorithm—we examine hemodynamic data/indices from ‘unstable’ patients for dynamic measures of preload reserve, afterload reserve, cardiac reserve, and perfusion reserve, that could guide and titrate the four major therapeutic options: fluid challenge; vasopressor/vasodilator; inotropic/inodilator; and oxygen/hemoglobin therapy.

**Preload Reserve: Preload and Preload Responsiveness**

Fluid therapy is often the first-line approach to the critically ill patient with circulatory failure. However, only half of such patients have been shown to respond to volume expansion with a significant improvement in hemodynamics, as indicated by an increase in cardiac output, stroke volume, or mean arterial pressure [2]. Nonresponders may suffer deleterious effects from volume expansion such as worsening of gas exchange, longer ventilation time, or cor pulmonale. An inotropic agent and/or vasopressor support should be preferentially used in these patients. Bearing in mind the high risk of volume overload, before giving fluid the clinician should be able to predict, by continuous hemodynamic monitoring, the response of individual patients to volume expansion instead of inotropic/vasoactive agents. Several clinical factors (detected at physical examination) and biological measured variables have been proposed as markers of fluid requirement, although they have limited sensitivity and specificity [3].

Neither cardiac filling pressures, such as CVP and pulmonary artery occlusion pressure (PAOP), nor their changes in response to fluid challenge are sufficiently reliable for predicting response to fluid load [4]. Since the introduction of the pulmonary artery catheter (PAC), many studies have shown that PAOP alone poorly reflects left ventricular (LV) preload, unless LV volume is measured as well [5]. Further, PAOP is a poor predictor of the LV preload responsiveness due to its high dependence on LV compliance. The latter is frequently reduced in critically ill patients, and can change over