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UPDATE IN INTENSIVE CARE: HEMODYNAMIC MONITORING IN CRITICALLY ILL PATIENTS

Assessment of cardiovascular preload and response to volume expansion☆

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Abstract Volume expansion is used in patients with hemodynamic insufficiency in an attempt to improve cardiac output. Finding criteria to predict fluid responsiveness would be helpful to guide resuscitation and to avoid excessive volume effects.

Static and dynamic indicators have been described to predict fluid responsiveness under certain conditions.

In this review we define preload and preload-responsiveness concepts.

A description is made of the characteristics of each indicator in patients subjected to mechanical ventilation or with spontaneous breathing.

PALABRAS CLAVE
Monitorización hemodinámica;
Respuesta a volumen;
Precarga-dependencia

Valoración de la precarga y la respuesta cardiovascular al aporte de volumen

Resumen El aporte de volumen es de especial interés en los pacientes ingresados en cuidados intensivos con inestabilidad hemodinámica en los que buscamos optimizar el gasto cardiaco. La predicción de la respuesta a esta expansión de volumen, evaluando el grado de precarga-dependencia nos permitiría realizar una reanimación guiada evitando los efectos deletéreos del volumen.

Actualmente, disponemos de parámetros tanto estáticos como dinámicos que identifican esta precarga-dependencia en diferentes escenarios.

En este capítulo definiremos los conceptos de precarga y precarga-dependencia para luego describir cada uno de los parámetros hemodinámicos conocidos para poder predecir la respuesta a volumen, tanto en pacientes con ventilación mecánica como en respiración espontánea.

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Introduction

Volume expansion constitutes first line treatment in situations of hemodynamic instability, though only 50% of all patients respond to fluid administration with an increase in systolic volume. Moreover, volume expansion can cause pulmonary adverse effects secondary to the increase in extravascular water; emphasis is therefore placed on the importance of using reliable parameters capable of identifying those patients who are likely to respond adequately to volume expansion. Specifically, the hemodynamic parameters proposed for deciding volume administration should be able to identify those patients that will derive benefit from the added volume, increasing their systolic volume (SV) (responders) and, at the same time, should be able to avoid useless (non-responders) and potentially harmful treatment.

In addition to the classical static parameters, in recent years new technologies and new dynamic parameters have been developed that reportedly offer better performance as predictors of patient response to volume expansion.

This chapter describes the physiological concepts related to preload and preload dependency, as well as the available predictive parameters in both mechanical ventilation (MV) and in spontaneous breathing.

Physiological review: concept of preload and preload dependency

Preload

Preload is one of the main determinants of cardiac output (CO). Classically, preload has been defined as the maximum degree of myocardial fiber stretch or tension before the start of ventricular contraction, and is determined by the mean sarcomere length at the end of diastole.[1] Clinically, this definition is not practical and is hard to apply; as a result, it is often replaced by more accessible ventricular filling measures such as the intracavitary pressures or end-diastolic volumes. Although none of these parameters precisely reflect cardiac muscle fiber elongation, they offer a more or less valid approximation to the true preload value. Accordingly, ventricular end-diastolic volume is accepted by consensus as a synonym of preload, and in turn under normal conditions the intracardiac pressures are taken to be a substitute for the intracardiac volumes.

Preload dependency

According to the Frank–Starling law, there is a positive relationship between preload and systolic volume; accordingly, the greater the ventricular preload (and therefore the degree of cardiac muscle stretch), the greater the systolic volume. However, this relationship, in the same way as in most physiological phenomena in the body, is not linear but rather traces a curve. Accordingly, once a concrete preload value has been reached, further increments do not give rise to significant additional systolic volume elevations.

The graphic representation or plot of this behavior is called the Frank–Starling curve or ventricular function curve

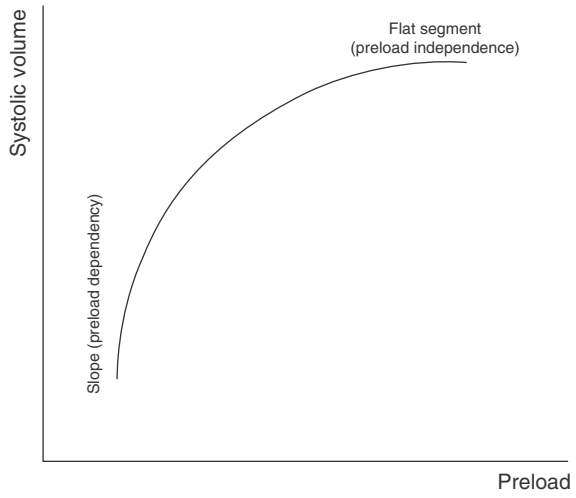

Figure 1 Ventricle function or Frank–Starling curve.

(Fig. 1), in which two zones can be distinguished: (a) a slope where minimum preload changes give rise to a marked increase in systolic volume (preload dependency zone); and (b) a flat or level segment where the ejection volume hardly varies with changes in preload (preload independence zone). This curve shows that in order to produce an increase in left systolic volume, both ventricles must be operating in the region of the slope or preload dependency zone of the Frank–Starling curve. If this condition is not met, any treatment measure aiming to increase preload (such as the administration of fluids) will only induce a rise in intracardiac pressures, with no hemodynamic benefits of any kind.

Therefore, the relationship between the changes in preload and systolic volume depends on the morphology and degree of slope or gradient of the Frank–Starling curve, which are determined by the contractile capacity of the heart and the ventricular postload. Thus, for one same increase in preload there will be a variable increase in systolic volume depending on the morphology and the zone in which the two ventricles are operating along the Frank–Starling curve (Fig. 2).

Lastly, it is important to underscore that each individual patient can present a series of ventricle function curves, dependent on the changes in postload or cardiac contractility. Accordingly, a patient in the flat or level segment of the Frank–Starling curve and without a positive response to the administration of fluids, may shift to the sloped portion of the curve with the administration of inotropic drugs prescribed to improve cardiac contractility–thereby improving the response to fluid expansion measures.

Thus, preload dependency is the capacity of the heart to modify systolic volume in response to changes in preload, and depends on the basal preload value and the zone of the Frank–Starling curve in which both ventricles are operating. Accordingly, in order to define a patient as being preload dependent, we must study not only the absolute preload value but also the zone of the ventricle function curve in which the patient's heart is operating.