Different Methods for Assessment of Fluid Responsiveness in Hypovolemic Patients

Essay

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Abstract

Background: Hypovolemia is a decrease of the volume of circulating blood, it may be due to external fluid losses caused by bleeding or losses from the gastrointestinal or urinary tracts, skin surface, or internal losses due to extravasation of blood or exudation or transudation of body fluids.

Fluid responsiveness is a measure of 'preload dependence' or 'preload reserve' but not all 'fluid responders' necessarily need volume loading. The initial assessment of volume status is most often based on clinical signs and symptoms in the prediction of fluid responsiveness, like skin turgor, urine color or production, fluid balance and the presence of peripheral edema.

Aims: The aim of this essay is to investigate fluid responsiveness in hypovolemic patients by different methods of assessment.

Conclusion: The heart-lung interaction is the fundamental mechanism of functional hemodynamic assessment. Intrathoracic pressure variations affect venous return and concomitantly diastolic cardiac filling as well as systolic cardiac performance. The Frank-Starling mechanism describes the relationship between diastolic myocardial distension, that is, preload, and systolic cardiac function.

Echocardiography is used in ICU for morphologic heart evaluation informing aspects of chambers and valves in addition to systolic and diastolic functions. There is a growing interest in this method for volume dynamic and volume responsiveness assessments. It is an essential tool for guiding resuscitation in critically ill patients. Resuscitation often requires the infusion of intravenous fluid in an effort to reverse organ dysfunction.

Keywords: Hypovolemic Shock, Fluid Responsiveness, Critically Ill, Echocardiography,

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Contents

List of Abbreviations

AA : Aortic area

ABG : Arterial blood gas analysis

ACES : Abdominal and cardiac evaluation with sonography in shock

ACLS : Advanced cardiac life support

AFV : Aortic flow velocity

ARDS : Acute respiratory distress syndrome

CABG : Coronary artery bypass grafting

CO : Cardiac output

CVP : Central venous pressure

DO_2 : Oxygen transport

ECG : Electrocardiogram

FOCUS : Focused cardiac ultrasound

FR : Fluid responsiveness

FT : Flow time

GEDV : Global end diastolic volume

ICU : Intensive care units

IgE : Immunoglobulin E

IJV : Internal jugular vein

IPPV : Intermittent positive pressure ventilation

LAP : Left atrial pressure

LiDCO : Lithium dilution continues cardiac output

LVEDV : Left ventricular end diastolic volume

LVOT : Left ventricular outflow tract

MAP : Mean arterial pressure

MOF : Multiorgan failure

PACs : Pulmonary artery catheters

PAOP : Pulmonary artery occlusion pressure

Pap : Pulmonary arterial pressure

PE : Pulmonary embolism

PEEP : Positive end expiratory pressure

PH : Pulmonary hypertension

PICCO : Pulse index continues cardiac output

PLAX : Parasternal long axis

PLR : Passive leg raising

POC : Point-of-care

PPV : Pulse pressure variation

PVI : Plethysmographic variability index

RUSH : Rapid ultrasound in shock

RV : Right ventricular

SIRS : Systemic inflammatory response syndrome

SOFA : Sepsis related Organ Failure Assessment

SpO_2 : Mixed oxymetric monitoring

SPV : Systolic pressure variation

$SvcO_2$: Central venous oxygen saturation

SVV : Systolic volume variation

TDI : Tissue Doppler imaging

TED : Transesophageal Doppler

TEE : Transesophageal echocardiogram

TTE : Transsthoracic echocardiogram

VBG : Venous blood gas

VTI : Aortic velocity-time integral

List of Figures

Introduction

Hypovolemia is a decrease of the volume of circulating blood, it may be due to external fluid losses caused by bleeding or losses from the gastrointestinal or urinary tracts, skin surface, or internal losses due to extravasation of blood or exudation or transudation of body fluids.

Relative hypovolemia follows increases in venous capacitance due to release of inflammatory mediators as in sepsis or as a side effect of drugs. In these distributive forms of circulatory failure, the intravascular volume may be normal, but the increases in the capacity of the vascular bed preclude adequate venous return. In each instance, volume repletion may be essential to restore critical levels of cardiac output and arterial pressure, resulting in more normal perfusion of vital organs and tissues **(Cecconi et al., 2014).**

The likelihood that there will be a favorable response to fluid administration is initially estimated on the basis of conventional clinical examination. Nevertheless, the history, physical signs, and routine laboratory tests. Signs of dehydration (eg, diminished skin turgor, thirst, dry mouth, hypernatremia, hyperproteinemia, elevated hemoglobin/ hematocrit) are especially misleading

.Extravascular volume deficits do not become clinically apparent until they exceed10% of body weight **(Cherpanath et al., 2014).**

Arterial hypotension is a nonspecific sign, which may be due to heart failure, vascular obstruction, as in the instance of a massive pulmonary embolism, or vasodilation quite independent of intravascular volume. Volume deficits are typically compensated for by increases in heart rate, which maintain cardiac output when stroke volumes are reduced. This response is inconsistent, especially in patients with intrinsic heart disease and during treatment with commonly used antiarrhythmic drugs. Stress, pain, fever, anemia, or drugs produce endogenous adrenergic stimulation with compensating increases in heart rate and vasoconstriction **(Cherpanath et al., 2014).**

In the last decade, with improved knowledge and practical application of physiology and heart-lung interaction, along with critical patient monitoring techniques, new volume responsiveness assessment methods were described, called dynamic methods. Described as such are pulse pressure variation (PPV), systolic pressure variation (SPV), systolic volume variation (SVV), in addition to techniques using echocardiography to evaluate superior and inferior vena cava collapsibility **(Cherpanath et al., 2013).**

The dynamic evaluation methods have good accuracy to predict fluid responsiveness, with much higher predictive values than static measurements. However, an important limitation of these methods is that indices and measurements were validated for specific groups of patients under sedation and volume controlled mechanical ventilation, with no respiratory effort and no arrhythmias. Other studies that tried to reproduce these results in different settings, did not reach the same results. In spontaneous breathing patients, or in those under mechanical ventilation with respiratory effort, fluid responsiveness assessment still requires additional studies **(Teboul and Monnet, 2008).**

Fluids must be considered as other drugs with beneficial but also adverse effects especially in patients with a limited cardiac reserve. For this reason, it is helpful to know, if the patient will respond to fluids. Several studies have shown that hemodynamic parameters classically use to evaluate vascular volumes such as central venous pressure (CVP) and pulmonary artery occlusion pressure (PAOP), are not able to predict the response to fluids administration. Volumetric parameters such as global end diastolic volume (GEDV) and left ventricular end diastolic volume (LVEDV), are better related to volume status but are not able to accurately predict fluid responsiveness. Assessing dynamic volume responsiveness

in spontaneous breathing patients, evidences related to CVP variation (ΔCVP), Pulse pressure variation (PPV) and methods using transthoracic echocardiogram and esophageal Doppler **(Donati et al., 2015).**

Transthoracic echocardiography is becoming a powerful noninvasive tool in the daily care of the critically ill. Assuming there is equipment and local expertise TTE is a repeatable and reliable method of predicting volume responsiveness in the critically ill. Importantly, TTE techniques appear useful in patients with spontaneous respiratory effort and those with arrhythmias: this is in contrast to many of the techniques that involve invasive monitoring which have been shown to be inaccurate in these situations **(Michard, 2011).**

Trans-aortic stroke volume variation with the respiratory cycle, stroke volume difference following passive leg raising, and IVC diameter changes with respiration all provide good prediction of the likelihood of a response to a fluid bolus. The techniques can be used individually to address the needs of different patients and in combination **(Mandeville and Colebourn, 2012).**

Aim of the Essay

The aim of this essay is to investigate fluid responsiveness in hypovolemic patients by different methods of assessment.