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HEMODYNAMIC MONITORING IN EMERGENCY. THE PHYSIOTHERAPY PERSPECTIVE: PHYSIOLOGY AND EFFECTS OF MECHANICAL VENTILATION

MONITORIZAÇÃO HEMODINÂMICA EM EMERGÊNCIA. VISÃO DO FISIOTERAPEUTA: FISIOLOGIA AOS EFEITOS DA VENTILAÇÃO MECÂNICA

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ABSTRACT

In various cardiac emergency situations, rapid bedside decision making should be well-founded, to ensure better therapeutic efficacy that is based on a physiological and pathophysiological knowledge of cardiac dysfunction and adequate hemodynamic monitoring of the patient, enabling correct indication of invasive/non-invasive mechanical ventilation. This opinion article therefore reiterates some hemodynamic aspects to be remembered and applied in the physiotherapist daily routine.

Keywords: Emergency; Hemodynamics Monitoring; Mechanical Ventilation.

RESUMO

Em diversas situações de emergência cardiológica, a tomada de decisão rápida a beira leito deve estar bem fundamentada para melhor eficácia terapêutica e baseada no conhecimento fisiológico e fisiopatológico da disfunção cardíaca que associada com a adequada monitorização hemodinâmica do paciente possibilitam indicar ou contraindicar o uso da ventilação mecânica invasiva e não-invasiva. Portanto, o presente artigo de opinião reitera alguns aspectos hemodinâmicos a serem lembrados e aplicados no dia a dia do fisioterapeuta.

Descritores: *Emergência; Monitorização Hemodinâmica; Ventilação Mecânica.*

INTRODUCTION

From the execution of simple activities, such as a physical exercise session, to the management of critically ill patients, hemodynamic monitoring is imperative, especially in cardiac emergencies.[1] Cases of acute ST-segment elevation myocardial infarction (STEMI) that develop acute lung edema and cardiogenic shock should be efficiently identified early and distinguished from cases of right ventricular myocardial infarction to assist in decision-making regarding the use of invasive or non-invasive mechanical ventilation and adequate adjustment of the ventilatory parameters according to the underlying hemodynamic repercussions.[2]

The understanding of these hemodynamic repercussions is currently possible because of the contributions of a German physician, Adolf Eugen Fick, who first measured the cardiac output (CO) in 1870 and formulated the Fick's Law. Briefly, this law describes the close relationship between the tissues and the cardiovascular and respiratory systems and defines CO as the ratio between oxygen consumption and the arteriovenous oxygen difference of the tissues based on several pathophysiological changes in numerous clinical situations that are still studied and investigated today.[1,2]

In 1955, while investigating blood flow, Guyton observed that CO and central venous pressure (CVP) are influenced by the following factors: "when hemodynamic changes of the circulatory system occur, it is not possible to predict what will happen with the CO, unless both the effect of the change in the heart's ability to pump blood (cardiac function) and the blood's tendency to return to the blood vessels (venous return [VR]) are considered."[3,4] Therefore, it is possible to graphically plot the factors that determine VR (blood volume, venous compliance, venous resistance, and right atrial pressure [RAP]) and cardiac function (preload, contractility, and heart rate).

This close relationship is depicted in Figure 1, which shows both VR and CO curves as a function of RAP.[1,4,5] As RAP decreases, more blood returns to the heart, thereby increasing ventricular performance and improving CO via the Frank-Starling mechanism (points abcd); after fluid resuscitation (displacement to point d), an additional increase in CO occurs.

The application of some physiological concepts to the management of the patient with cardiac emergency—for instance when a drop in blood pressure (BP) occurs without changes in CO—shows that the triggering factor was a reduction in the systemic vascular resistance (SVR); in such cases, medications for vasoconstriction, such as noradrenaline, can

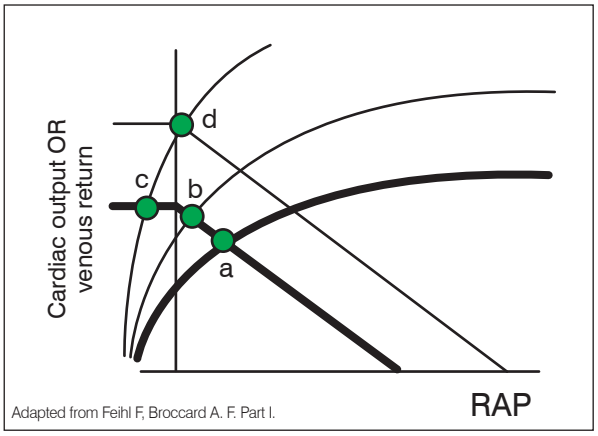

Figure 1. Guyton's graphical analysis of cardiac output/venous return regulation and correlation with right atrial pressure (RAP).

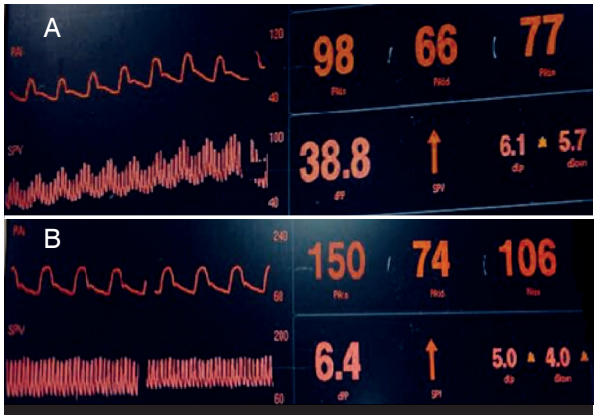

Figure 2. Delta PP (DPP) monitoring. A Patient initially presented with a DPP of 38.8 with arterial hypotension (systolic pressure, 98 mmHg). B After fluid resuscitation, DPP decreased to 6.4 with arterial hypertension (systolic pressure, 150 mmHg). Values below 12 indicate lower or no responsiveness to fluid resuscitation.

be used. However, if the decrease in BP is accompanied by a reduction in CO, CVP should be measured to determine whether heart failure or a decreased VR has occurred.[6] The decrease of both CO and CVP indicates a decreased VR, a clinical condition that can be reversed with adequate fluid resuscitation; otherwise, if the underlying triggering factor is the heart and inotropic drugs should be used.[7]

Basic physiological concepts can also be used at the bedside to analyze the respiratory repercussions in the cardiovascular system. Inspiration exerts a direct effect on the superior and inferior vena cava; the lower the filling volume (that is, lower the VR), the greater the influence of negative or positive respiratory pressures on its content, which leads to great variability in its diameter during the respiratory cycle.[8] This variability shows the patient's situation in relation to the Frank-Starling curve (optimal filling of the ventricles promotes contraction capacity and blood pumping); this information can be used to determine if the patient is in the in the ascending portion of the curve, where an induced increase in preload results in increased systolic volume and CO.

These repercussions from the respiratory system are exemplified in the physiotherapist's clinical practice; for example, in patients on mechanical ventilation, with high intrathoracic positive pressures caused by tidal volume and/or positive end-expiratory pressure (PEEP) that results in decreased filling and right ventricular (RV) ejection, especially in patients with insufficiency.[8,9] Consequently, reduction of the CO in the RV during inspiration reduces preload, systolic volume, and the CO of the left ventricle (LV) during expiration, causing oscillations in the arterial pressure curve (invasive BP) and variations in the pulse pressure (ΔPP) (Figure 2). This analysis is based on a non-invasive observational approach and can predict which patients will have the greatest negative repercussions of mechanical ventilation, a tool that should be further explored by the physiotherapists at the bedside. However, to use this approach, some conditions must be met: 1) patient should be on invasive mechanical ventilation; 2) patient should be sedated and paralyzed; 3) tidal volume should be 8 mL/kg; 4) arrhythmias and valve disease should be absent; and 5) arterial pulse traces and mechanical or respiratory ventilation should be monitored on the same screen.[10]

Considering this hemodynamic review and its practical implications as well as bedside approaches to cardiovascular assessments in emergency situations, how can clinicians apply mechanical ventilation in patients with respiratory failure related to acute ventricular dysfunction?

Cases of respiratory failure related to LV failure with consequent cardiogenic pulmonary edema are caused by an increased final diastolic volume since the impaired ventricular cardiac pump ejects less volume into the systemic circulation, thus increasing the residual blood content in the left chamber, generating a cumulative retrograde effect and affecting the pulmonary circulation with alveolar extravasation.[11]

The application of positive pressure in these cases is beneficial since it decreases afterload by reducing the transmural pressure (pressure gradient generated during systole minus the pressure around the cavity, that is, a decreased pressure difference between the internal and external areas of the heart), increases the ejection fraction and the CO, facilitating anterograde flow and thus decreasing pulmonary edema. These repercussions on the ventricle depend on the functional status, intrathoracic pressure, transmural pressures, and preload. There is robust evidence that the mechanical effect of positive pressure on CO can control acute respiratory failure in addition to improving LV function.[12,13]

On the other hand, the effects of positive pressure and PEEP on RV function result from the combination of decreased VR and increased pulmonary vascular resistance. Therefore, in cases of respiratory failure associated with RV failure/infarction, much attention should be given to the volume and the applied pressure levels. Mechanical ventilation at high pressures increases RV work by increasing the afterload caused by the mechanically driven increase in lung volume that causes direct vascular compression. Thus, in cases of RV perfusion impairment, the administration of PEEP leads to dilation and increased end-systolic and diastolic volumes, which aggravate cardiac dysfunction. This also affects LV diastolic function owing to deviation of the interventricular septum in the direction of the LV in addition to the direct reduction in LV preload and, therefore, CO.[14,15]

Therefore, in emergency situations known to be associated with RV failure, the repercussions are more evident; thus, the application of low intrathoracic pressures becomes necessary until adequate volume support is achieved. For this, a careful

hemodynamic assessment is essential owing to increases in heart rate, decreases in blood pressure reduction, and signs of RV heart failure such as jugular swelling and worsening of the ventilation-perfusion relationship and CO.[4,15]

In agreement with the above, comparative hemodynamic studies with several modes of mechanical ventilation show that, regardless of the specific mode of ventilation, mean airway pressure is the main factor responsible for the cardiovascular effects associated with mechanical ventilation. Increased inspiratory time, decreased expiratory time (especially when associated with inversion of the I:E ratio), long inspiratory pauses, use of high tidal volumes, use of decreasing inspiratory flows, and PEEP are approaches that tend to increase mean airway pressures, which may compromise the hemodynamic situation, especially in cardiac patients.[15]

CONCLUSION

The complex effects of ventilatory adjustments on hemodynamics should be understood and recognized by the physiotherapist to ensure the most appropriate and customized values for each patient according to case evolution, which can start with STEMI, and differentiating between RV and LV in the presence of acute pulmonary edema associated with cardiogenic shock.

CONFLICTS OF INTEREST

The authors declare that they have no conflicts of interest in conducting this study.

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