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EDITORIAL

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Mean systemic pressure: we can now estimate it, but for what?

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There has been much controversy about the reality of the Guyton's model of circulation [1, 2]. According to this model, systemic venous return is proportional to the pressure gradient for venous return divided by the resistance to venous return. The pressure gradient for venous return is the difference between the mean systemic pressure (Pms) and the right atrial pressure (Pra). The Psm, which is a virtual pressure considered to lie at the level of the venules [3], is positively related to stressed blood volume and negatively related to vascular compliance. The stressed blood volume, which represents 30–40 % of the total blood volume, is hemodynamically active and thus participates in venous return through its impact on Pms. The unstressed blood volume, which represents 60–70 % of the total blood volume, is hemodynamically inactive and serves as a blood reservoir that can be mobilized by venoconstriction and can be converted to stressed blood volume under some critical conditions [4]. Since Pms is a marker of effective blood volume, its measurement would be important to obtain at the bedside in order to better characterize a patient's cardiovascular status and its response to therapies. Until recently, the Pms could not be measured routinely in patients. According to the Guyton's theory, the Pms is the vascular pressure existing at zero flow conditions (Fig. 1) and its measurement requires the physician to stop the circulation and wait for equilibrium between the arterial pressure and the venous pressure. Obviously, this is feasible only in experimental conditions in animals [5] or during cardiac arrest in humans [6]. Recently, Maas et al. [3] proposed an elegant method to estimate Pms in intact conditions in mechanically ventilated patients. This method consists of simultaneously measuring central venous pressure (CVP) and cardiac output (CO) during inspiratory-hold maneuvers at four different plateau pressures. A set of four CVP–CO data pairs can thus be obtained. Considering that CVP is a surrogate of Pra and that CO is equal to venous return in apneic steady-state conditions, a set of four Pra-venous return data pairs is thus obtained and can be fitted by linear regression to define the venous return curve. The estimated Pms is defined as the extrapolation of this linear regression to zero flow (Fig. 1). Using this method, Maas and coworkers [3] found that fluid loading increases the estimated Pms and that a 30° head-up position decreases the estimated Pms, all findings consistent with what it is expected from Guyton's theory. Thereafter, a derived method to estimate Pms was proposed by Persichini and coworkers [7]. This method consists of performing two consecutive sets of four ventilatory-hold maneuvers (inspiratory and expiratory holds, both performed at two levels of positive end-expiratory pressure) resulting in eight CVP–CO data pairs [7]. By doing so in 16 septic patients receiving norepinephrine, a very close linear relationship (average $r^2 = 0.71$) between CO and CVP was found in every patient at baseline and after norepinephrine decrease. This strongly argues that Guyton's model of circulation makes sense. The main limitation of the ventilatory-hold methods to

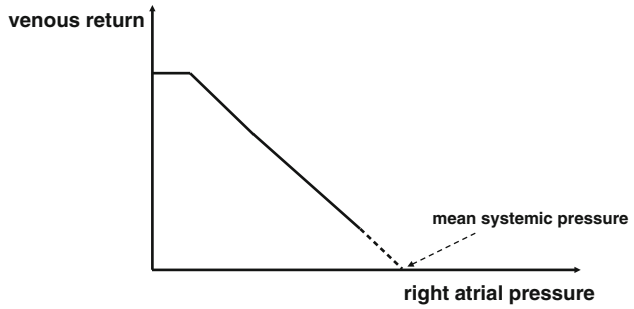

Fig. 1 Relationship between right atrial pressure and venous return according to Guyton's model. Note that at low right atrial pressure, the venous return does not increase further probably because of the collapse of the inferior vena cava at the thorax entry

estimate Pms is that it requires a perfect adaptation of the patient to the ventilator. In addition, because it is cumbersome, this technique cannot be used for routine patient management and thus should be reserved for research purposes. Recently, a noninvasive software algorithm has been developed to estimate a Pms analogue (Pmsa) using the mean arterial pressure (MAP), Pra (or CVP), CO and the patient's anthropometric data. This system automatically collects data from standard bedside and cardiac output monitors. The formula that estimates Pms is proprietary and uses Guyton's model. In a previous study, Maas et al. [8], using the inspiratory-hold method as the reference in postoperative cardiac surgery patients, reported a poor agreement between Pmsa and Pms. However, they found that changes in Pmsa and changes in Pms were directionally concordant in response to head-up tilt and volume loading [8]. In this issue of Intensive Care Medicine, Cecconi et al. [9] also assessed the significance of the Pmsa and the difference between Pmsa and CVP (dVR) during a fluid challenge in post-operative surgical intensive care patients. One-hundred and one fluid challenges were performed in 39 patients. Pmsa increased similarly during a fluid challenge in responders and non-responders (3.1 ± 1.9 vs. 3.1 ± 1.8), whereas the dVR increased in responders but remained unchanged in non-responders [9]. They concluded that the changes in Pmsa and dVR measured during a fluid challenge are consistent with the cardiovascular model described by Guyton. This result is not surprising since Pmsa is calculated from MAP, CVP and CO according to the Guytonian model of circulation. This can thus be viewed as an auto-validation of the proprietary algorithm. Nevertheless, this study does not provide a positive answer to the question of the usefulness of Pmsa to guide fluid management. In this regard, Cecconi et al. [9] clearly show that neither Pmsa nor dVR at baseline predicted fluid responsiveness. This is not really a surprise, since Pmsa and dVR are static variables. It was already demonstrated that static measures of preload such as CVP, pulmonary artery occlusion pressure and left ventricular end-diastolic area are not reliable for predicting the CO response to fluid administration [10]. We have now learned that a static measure of the effective blood volume such as Pms cannot serve as a marker of fluid responsiveness. Nevertheless, knowledge of this easy-to-obtain parameter provides clinicians with additional information that should help to get a more comprehensive picture of the patient's cardiovascular status and its response to therapies.

Conflicts of interest The author declares no conflict of interest.

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