Cardiorespiratory interactions

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Cardiorespiratory interactions were long limited to research, which admittedly was fascinating but apparently had no major clinical applications. In recent years, and in the light of several publications, it appears that these interactions can in fact be used to develop tools for hemodynamic monitoring in ventilated patients, and even to adapt the ventilation parameters in certain ventilated patients, in ARDS for example. These interactions are the result of cardiopulmonary anatomy: the cardiac chambers are subject to intrathoracic pressure, the right ventricle branches upstream of the lung, the two ventricles have a common wall, the interventricular septum.

The cyclic variations of the pulse in mechanical ventilation were described in 1973 by Massumi et al. using the term reversed pulsus paradoxus. They comprise an inspiratory increase in systemic pulse. This increase may be absolute, and certain authors subsequently called it dUp (**Figure 1**). But it may simply be related to the drop in pulse described on expiration, and called dDown (**Figure 2**). It is only possible to make this distinction by disconnecting the patient from the respirator or by making a prolonged end-expiratory pause.

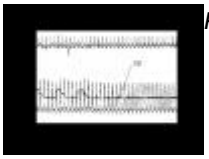

Figure 1: TP: airway pressure. Recording of systemic blood pressure in a patient presenting dUP

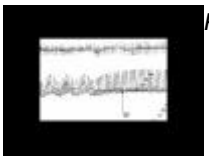

Figure 2 : TP: airway pressure. Recording of systemic blood pressure in a patient presenting dDown

Doppler echocardiography is perfectly suited to the study of the mechanisms underlying these pulse variations. It can be used to investigate cardiac function parameters and to localize each heart beat in the respiratory cycle. For this, it is necessary to display the airway pressure signal on the screen of the echocardiograph. By convention, we have defined heart beat 1 as occurring at the end of expiration, heart beat 2 at the start of insufflation, heart beat 3 at the plateau, and heart beat 4 at the start of expiration (**Figure 3**).

Figure 3 : TEE view of the vessels at the base of the heart. Recording of pulmonary arterial flow by pulsed Doppler in the trunk of the pulmonary artery. Insertion of the airway pressure signal enables localization of the heart beats in the respiratory cycle. By definition, heart beat 1 is end-expiratory, 2 occurs at the start of insufflation, 3 is end-inspiratory, and 4 occurs at the start of expiration

Whereas at steady-state, and throughout the respiratory cycle, the stroke volume (SV) of the left ventricle is on average identical to that of the right ventricle, mechanical ventilation dissociates these two values: the SV of the right ventricle is decreased on insufflation whereas the SV of the left ventricle is increased at the same time (**Film 1** and **Film 2**). Three inspiratory phenomena are responsible for this dissociation: diminished systemic venous return, increased impedance on right ventricular ejection, and improved left ventricular filling.

1- Inspiratory decrease in systemic venous return

The increase in intrathoracic pressure, caused by the pressure increase in the airways on insufflation, leads to a decrease in systemic venous return. This diminution is more marked when the intrathoracic pressure generated is high and the patient's

volemia is low. According to Guyton's concept, this has long been attributed to a decrease in the pressure gradient of the venous return, defined as the difference between the mean systemic pressure and the right atrial pressure.
However, positive-pressure ventilation does not seem to alter this gradient (**Figure 4**), suggesting that the decreased venous return results from a diminution in venous conductance due to the interposition of collapsible veins between the peripheral venous circulation and the right atrium.

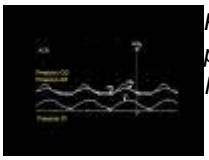

Figure 4: Simultaneous recording of systemic arterial pressure (Pa), right atrial pressure (Pra) and pleural pressure (Ppl) in a mechanically ventilated patient, during cardiac arrest. The increase in pleural pressure, linked to the ventilation, is transmitted both to the systemic pressure and to the right atrial pressure

This hypothesis has been demonstrated in spontaneously breathing patients during serious acute asthma when inspiration can result in the collapse of the inferior vena cava (**Film 3**). Recently, we have confirmed this during mechanical ventilation, but at the superior vena cava (SVC) which is subject to intrathoracic pressure. On clamping of the inferior vena cava for hepatectomy, we first observed the occurrence of inspiratory collapse of the SVC, responsible for marked inspiratory decrease in the right ventricular stroke volume (**Film 4**and **Film 5**). This collapse, defined as an inspiratory reduction of 60% in SVC diameter occurs when the transmural pressure of the vessel (PSVC - Pintrathoracic) falls below its closing pressure. This physiological description has a major clinical impact, as we have since shown that collapse of the SVC is an excellent index of hypovolemia in patients mechanically ventilated for septic shock and can be used to predict the efficacy of volume expansion (**Film 6**and **Film 7**). Lastly, its presence depends not only on the volemia of the patient but also on the ventilation parameters, allowing adjustment of the volemia to a given ventilation, or even the ventilation to a given hemodynamic state (**Film 8**, **Film 9**, and **Film 10**).

2- Increased impedance at right ventricular ejection

Blood flow in the pulmonary circulation depends on a pressure gradient between the upstream pressure, i.e. the pulmonary arterial pressure, and the downstream pressure, i.e. the pulmonary venous pressure. Insufflation, or the application of a positive end-expiratory pressure (PEEP), generates an increase in transpulmonary pressure (lung distension pressure defined as Palveolar - Ppleural). This increase in transpulmonary pressure leads to an extension of West zone II (Pulmonary arterial pressure > Alveolar pressure > Pulmonary venous pressure) at the expense of West zone III (Pulmonary arterial pressure > Alveolar pressure > Pulmonary venous pressure), which then hinders blood flow in the alveolar vessels. The resulting increase in right ventricular afterload may account for dilatation of the right ventricle. This is particularly true in ARDS where the transpulmonary pressure generated is large because of the drop in pulmonary compliance (**Film 11** and **Film 12**). From the pulmonary arterial flow obtained by pulsed Doppler in the trunk of the pulmonary artery, we have developed several indices to evaluate right ventricular afterload during ventilation. These indices can be used to assess tolerance of ventilation, and notably of the PEEP applied.
Thus, the mean acceleration of the flow in the pulmonary artery is an index inversely proportional to right ventricular afterload and is directly proportional to the systolic function of the right ventricle. It is calculated as the ratio of the maximum flow velocity over the acceleration time of this flow (time between the start of ejection and peak ejection) (**Figure 5**).

Figure 5 : TEE - View of vessels at the base of the heart. Pulsed Doppler recording of flow in the trunk of the pulmonary artery. Vmax: maximum velocity, Tacc: acceleration time. Vmax (m/s or cm/s) is measured at peak flow; Tacc (ms) is measured between the start and the peak of ejection. The mean acceleration of the right ventricle (m/s-2) is calculated as ratio between Vmax and Tacc

On insufflation, we have demonstrated a drop in mean acceleration at heart beat 3 (**Film 13**, **Film 14**), reflecting an increase in impedance on right ventricular ejection. From the Doppler flow in the pulmonary artery, we have also used the isovolumetric contraction time of the right ventricle (from the start of the QRS to the start of ejection). It is directly proportional to right ventricular afterload and corresponds to the pressure of isovolumetric contraction measured beforehand using a pulmonary arterial catheter.
Insufflation (**Figure 6**), as well as a high-frequency ventilatory strategy (**Figure 7**), can lengthen this Doppler time.

Figure 6: TEE - View of vessels at the base of the heart. Pulsed Doppler recording of flow in the trunk of the

pulmonary artery. IVCT: isovolumetric contraction time (ms). IVCT is measured between the start of the QRS and the start of right ventricular ejection. On the left, measurement of IVCT at the end of expiration. On the right, its measurement in the same patient, at the end of insufflation, reveals a lengthening which results in an increase in right ventricular afterload

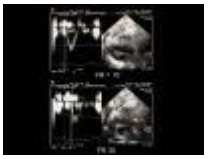

Figure 7 : TTE - View of vessels at the base of the heart by the subcostal route. Recording of pulmonary arterial flow by pulsed Doppler at the pulmonary annulus. IVCT: isovolumetric contraction time (ms). Measurement of IVCT, in end-expiratory phase, in the same patient at a respiratory frequency (RR) of 15 (above) and 30/min (below). Ventilation at high respiratory frequency lengthens IVCT

3- Improvement of left ventricular filling

The left ventricle is directly filled by the pulmonary venous return. The pulmonary circulation thus represents the preload reserve of the left ventricle. On insufflation, the blood is expelled from the pulmonary capillaries to the left atrium and left ventricle. This phenomenon is responsible for the dUp which can be observed on a blood pressure curve. It is more marked when the pulmonary circulation is correctly filled by prior right ventricular ejection and when allowed by the elastance of the atrium and left ventricle. In echocardiography this outflow effect is visualized by a significant increase in the size of the left atrium on insufflation (**Film 15**), as well as by an increase in Doppler flow in the pulmonary veins (**Film 16**). By improving left ventricular filling, it is responsible for an increase in ventricular ejection (**Film 2**).

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