Heart-lung interactions, a long story with many pioneers

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“Learning is either a continuing thing or it is nothing”

Respiration and circulation are two convective systems that meet together in the thorax. Both act synergistically to ensure the blood oxygenation, the elimination of CO\textsubscript{2}, and the blood transport from the periphery to the heart and from the heart to the periphery. Three centuries ago, the British physiologist Sir Stephen Hales observed that the level of the blood column in a glass tube inserted into the carotid artery of a horse varied cyclically with respiration (1). The major progresses came from the last 80 years when physiological measurements could be done with efficient technologies and experimental models (2). In addition to the physiological progresses, the understanding of heart-lung interactions became essential when it was translated to clinical situations involving respiratory and circulatory changes. Various life-threatening situations involve the basic concepts of heart lung interactions such as: the Kussmaul dyspnea during severe keto-acidotic diabetes; positive pressure breathing (PPB) using artificial ventilation for respiratory supportive therapy; optimization of cardio-pulmonary resuscitation (CPR) based on the concept of “thoracic pump”; acute left ventricular failure with pulmonary edema, etc.... The play with these concepts have frequently been included in the International Societies recommendations to better treat patients avoiding negative impacts and promoting positive effects for left and right ventricular dysfunctions, volume mobilization for organ perfusion, limitation of congestion, improve loading conditions for both right and left ventricles (LV).

Several key concepts are commonly used, some of them being developed in critical care medicine by authors of this issue of the journal dedicated to heart-lung interactions, to limit the risk or to use the benefit of such interaction to improve the circulatory system in severe conditions (3-8). Among them the transmural pressure and the concept of Starling resistance applied to lung circulation is essential as mentioned in this issue. Application of a positive pressure increases pleural pressure, constraining the chamber volumes. Such external constrain depends largely on the structures compliance (9). Keeping in mind this concept to interpret the measured intravascular pressure is essential for the clinician, despite the usual lack of quantification of the surrounding pressure. Driving pressures to move blood from different structures remains the useful pressure gradient. This gradient from the peripheral veins to the right atrium has to be sufficient to mobilize blood towards the right ventricle (RV) against venous resistances. The sudden increase in intravascular pressure of intrathoracic vascular structures related to PPB may then dramatically reduce this gradient and the venous return. Such effect is however largely dependent on the venous compliance (10). The venous return determines the RV preload, a key factor for this heart pump functioning as a volume pump. The pulmonary pressure generated by the RV pushes blood towards the LV to provide an adequate LV preload. Because of the RV characteristics, increase in RV afterload has a major impact on RV function, which can be summarized under the term acute “cor pulmonale” (11). Such frequent acute afterload increase may then result from: (I) compression related to alveolar pressure and ventilation support. The pulmonary vessels being collapsible vessels, they can at maximum collapse when external pressure is over the intra-vascular pressure with a stop flow (West Zone 1) (5); (II) acute vasoconstriction as observed in regional pulmonary hypoxia (pulmonary hypoxic vasoconstriction) (12,13) and/or acute systemic or lung inflammation, or vasopressors infusion; (III) acute vascular obstruction related to micro-clotting in acute inflammation or during regional pulmonary embolism (14). The reduction in flow generated by RV leads to a reduced LV preload, which is followed...
by a reduced stroke volume ejected by LV, with a potential arterial hypotension mainly for systolic blood pressure. Looking in details to the modifications observed during a PPB cycle, insufflation increases RV afterload, reducing the RV stroke volume. The squeezing of the lung vascular structure pushes blood out of the lung vascular bed to the LV increasing the LV preload, proportionally with the pulmonary blood volume (10). Consequently, the LV stroke volume increases with a higher systolic blood pressure.Expiration induced a better ejection from RV, filling the pulmonary circulation with a reduction in LV preload. As a consequence, LV stroke volume is reduced with a decrease in systolic blood pressure. The amplitude of inspiration/expiration variations in clinic have been first described as demonstration of a central hypovolemia by Perel et al. and later by Michard et al. (15,16). They hypothesized that such PPB induced variations are amplified by a reduced central blood volume, a consequence of a global hypovolemia. This concept has been generalized to assess hypovolemia with important limitations linked to the measurement context (17). As mentioned above, the compliance of the structures has a major impact on the generated transmural pressures. The interactions between heart and lung have been shown to be attenuated when the lung has a reduced compliance as observed in acute lung injury (18).

PPB has also been shown to have positive effect when LV function is altered (19). Even the relative importance of underlying mechanism(s) remains debated, the effectiveness of noninvasive PPB was shown efficient to treat acute pulmonary edema related to acute LV dysfunction, as observed in acute coronary syndrome (20). Early application of a moderate CPAP was more efficient than the classic medical treatment, being efficient more rapidly, with a lower rate of intubation and inotrope use (21). Being confirmed by other studies, this heart lung interaction-based therapy was added in the American Heart Association recommendations.
The last major application mentioned in this Editorial about the usefulness of heart-lung interactions for acute situation, concerned CPR. In presence of cardiac arrest, the only mean to mobilize blood in and out the thorax is to generate adequate pressure gradient to increase venous return and then to compress the loaded intrathoracic blood volume out of the thorax. The concept of thoracic pump developed by Max Harry Weil was further amplified on the physiological basis (22). The efficiency of thoracic pump amplified by an active decompression technique has been demonstrated (23). This conceptual technique was further reinforced (more negative intra-thoracic pressure) by the addition of an impedance valve during active decompression to generate more negative intra-thoracic pressure (24). Then the compression is pushing more blood out of the thorax generating more flow for the tissue perfusion.

The outstanding team of authors writing papers in this issue is giving all the details and rationale for using heart lung interaction explaining the physiology and then the application to the life-threatening situations. This essential understanding will never be old-fashioned for the clinician, since physiology is solid and patho-physiological treatment will be always better than evidence based-medicine for individual treatment.

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Footnote

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