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_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 intra-thoracic 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 reduce 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

Conflicts of Interest: The author has no conflicts of interest to declare.

References

1. Sette P, Dorizzi RM, Azzini AM. Vascular access: an historical perspective from Sir William Harvey to the 1956 Nobel prize to André F. Cournand, Werner Forssmann, and Dickinson W. Richards. J Vasc Access 2012;13:137-44.
2. Cournand A, Motley HL, Werko L. Mechanism underlying cardiac output change during intermittent positive pressure breathing (IPP). Fed Proc 1947;6:92.
3. Fessler HE, Brower RG, Wise RA, et al. Effects of positive end-expiratory pressure on the gradient for venous return. Am Rev Respir Dis 1991;143:19-24.
4. Fessler HE, Brower RG, Wise RA, et al. Effects of positive end-expiratory pressure on the canine venous return curve. Am Rev Respir Dis 1992;146:4-10.
5. Permutt S, Riley RL. Hemodynamics of collapsible vessels with tone: the vascular waterfall. J Appl Physiol 1963;18:924-32.
6. Pinsky MR, Summer WR, Wise RA, et al. Augmentation of cardiac function by elevation of intrathoracic pressure. J Appl Physiol Respir Environ Exerc Physiol 1983;54:950-5.
7. Robotham JL, Rabson J, Permutt S, et al. Left ventricular hemodynamics during respiration. J Appl Physiol Respir Environ Exerc Physiol 1979;47:1295-303.
8. Marini JJ, Culver BH, Butler J. Effect of positive end-

expiratory pressure on canine ventricular function curves. J Appl Physiol Respir Environ Exerc Physiol 1981;51:1367-74.
9. Magder S. Volume and its relationship to cardiac output and venous return. Crit Care 2016;20:271.
10. Payen DM, Brun-Buisson CJ, Carli PA, et al. Hemodynamic, gas exchange, and hormonal consequences of LBPP during PEEP ventilation. J Appl Physiol (1985) 1987;62:61-70.
11. Vieillard-Baron A, Naeije R, Haddad F, et al. Diagnostic workup, etiologies and management of acute right ventricle failure : A state-of-the-art paper. Intensive Care Med 2018. [Epub ahead of print].
12. Pinsky MR. Determinants of pulmonary arterial flow variation during respiration. J Appl Physiol Respir Environ Exerc Physiol 1984;56:1237-45.
13. Marshall BE, Marshall C, Frasch F, et al. Role of hypoxic pulmonary vasoconstriction in pulmonary gas exchange and blood flow distribution. 1. Physiologic concepts. Intensive Care Med 1994;20:291-7.
14. Naeije R, Manes A. The right ventricle in pulmonary arterial hypertension. Eur Respir Rev 2014;23:476-87.
15. Michard F, Chemla D, Richard C, et al. Clinical use of respiratory changes in arterial pulse pressure to monitor the hemodynamic effects of PEEP. Am J Respir Crit Care Med 1999;159:935-9.
16. Perel A. Intrathoracic blood volume and global end-diastolic volume should be included among indexes used in intensive care for assessment of fluid responsiveness in spontaneously breathing patients. Crit Care Med 2006;34:2266-7; author reply 2267.
17. Benes J, Kirov M, Kuzkov V, et al. Fluid Therapy: Double-Edged Sword during Critical Care? Biomed Res Int 2015;2015:729075.
18. Teboul JL, Monnet X. Pulse pressure variation and ARDS. Minerva Anestesiol 2013;79:398-407.
19. Buda AJ, Pinsky MR, Ingels NB Jr, et al. Effect of intrathoracic pressure on left ventricular performance. N Engl J Med 1979;301:453-9.
20. Plaisance P, Pirracchio R, Berton C, et al. A randomized study of out-of-hospital continuous positive airway pressure for acute cardiogenic pulmonary oedema: physiological and clinical effects. Eur Heart J 2007;28:2895-901.
21. Ducros L, Logeart D, Vicaut E, et al. CPAP for acute cardiogenic pulmonary oedema from out-of-hospital to cardiac intensive care unit: a randomised multicentre study. Intensive Care Med 2011;37:1501-9.
22. Weil MH, Noc M. Cardiopulmonary resuscitation: state of the art. J Cardiothorac Vasc Anesth 1992;6:499-503.
23. Plaisance P, Lurie KG, Vicaut E, et al. A comparison of standard cardiopulmonary resuscitation and active compression-decompression resuscitation for out-of-hospital cardiac arrest. French Active Compression-Decompression Cardiopulmonary Resuscitation Study Group. N Engl J Med 1999;341:569-75.
24. Plaisance P, Lurie KG, Payen D. Inspiratory impedance during active compression-decompression cardiopulmonary resuscitation: a randomized evaluation in patients in cardiac arrest. Circulation 2000;101:989-94.

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