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Diagnosing acute respiratory distress syndrome with the Berlin definition: Which technical investigations should be the best to confirm it?

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The distinction between hydrostatic pulmonary edema (HPE) and acute respiratory distress syndrome (ARDS) could be challenging in the management of acutely ill patients. The Berlin definition of acute respiratory distress syndrome (ARDS) stipulate that respiratory failure must not be explained by cardiac failure of fluid overload. The situation needs an objective assessment to exclude hydrostatic edema, if no risk factor is present.[1] The differential diagnosis is based on the evaluation of the left atrial pressure, which is considered as normal in ARDS.[1–3] Nevertheless, it has been shown that in ARDS, 30% of them do have a component of hydrostatic lung edema that is shown by an increase of the wedge pressure measured by pulmonary artery catheter above 15 mmHg.[4] Besides this, the differential diagnosis between ARDS and hydrostatic lung edema remains in some cases very challenging. The transpulmonary thermodilution (TPTD) known as pulse contour cardiac output (PICCO) system or the calibrated pulse wave analysis method (VolumeView™/EV1000™, Edwards Lifesciences, Irvine, CA, USA) is now widely used in the intensive care unit. TPTD allows measurement of cardiac output, intra thoracic blood volume (ITBV), pulmonary blood volume (PBV), global end diastolic volume (GEDV) and extravascular lung water (EVLW).[5] EVLW is the amount of water that is contained in the lung outside the pulmonary vessels including interstitial, alveolar, intracellular and lymphatics fluids.[6] Jozwiak *et al.* demonstrated that EVLW is an independent prognostic factor in patients with ARDS. EVLW and mean cumulative fluid balance are independently associated. EVLW is a marker of lung injury and not simply the result of a liberal fluid strategy leading to fluid overload.[6] In numerous cases, EVLW could be normal in ARDS. EVLW is underestimated if indexed to actual body weight and must be indexed to predict body weight.[6,7] EVLW is underestimated in the presence of a non-homogenous form of ARDS.[8] Pulmonary microemboli or vascular damage due to ARDS could also lead to an inhomogeneous distribution of the cold marker.[9] An increase in peep and tidal volume during mechanical ventilation induces a decrease in PBV and changes in ventricular dimension. The ratio between heart and PBV could change in those conditions.[10] The pulmonary vascular permeability index (PVPI) is the ratio between EVLW and PBV or the ratio between EVLW and ITBV.[11,12] This index reflects the pulmonary microvascular permeability.[12] Increased pulmonary vascular permeability is an important pathological feature of ARDS.[2] PVPI > 3 allows the diagnosis of ARDS with a sensitivity of 85% and a specificity of 100%, while a PVPI Under 1.7 excludes the diagnosis of ARDS.[11] The PVPI shares the same limitations with EVLW.[10] GEDV is the volume of all four cardiac chambers at the end of the diastole.[10] GEDV includes the cardiac volume and is also a part of the superior vena cava volume and the volume

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of the aorta that are between the bolus injection site and the thermistor.[13] A lot of studies argue that volume markers of cardiac preload measured by TPTD are better than pressure markers. GEDV is better correlated than pulmonary artery occlusion pressure (PAOP) with stroke volume.[10] We must keep in mind that if the ventricular compliance is low, small changes in volume will induce large changes in pressure. So, changes in volume will underestimate the change in cardiac preload.[10] Secondary, the risk of hydrostatic edema depends directly upon the pressure gradient between the pulmonary capillary and the interstitium. This difference of pressure is better assessed by PAOP than by any volume index.[10] A last limitation is that GEDV doesn't distinguish between left and right cavities. A right dilatation could be misinterpreted as an elevated left ventricular preload.[10] In case of valvulopathy, GEDVI et ELWI could also be misinterpreted. GEDV could be normal in mitral stenosis or elevated in insufficiency due to the duration of the curve.[14]

TPTD is an interesting tool in the assessment of the pulmonary edema but the interpretation must be cautious. Some authors have proposed to include thermodilution in the Berlin definition.[9,15] This proposal was refuted on the basis of the cost, the invasiveness of the method and the low availability of the technique around the world. On top of this, thermodilution has methodological limitations.[16] Another argument was that thermodilution did not allow the distinction between hydrostatic and inflammatory edema.[9] The study by Kushimoto refuted this last argument by introducing the PVPI.[11] Before the Berlin criteria were available, the definition of ARDS did include the PAOP value measured by pulmonary arterial catheters (Swan-Ganz catheter). This was even more invasive, expensive and methodologically limited than transpulmonary thermodilution.[9] Pulmonary edema now includes cardiogenic pulmonary edema, ARDS (at risk, moderate or severe), and a combined pulmonary edema Cardiac ultrasound is essential to detect the mix situation with combined pulmonary edema,[15] especially when we are in front of the 30% of ARDS with left heart failure. Likewise, the use of cardiac ultrasound is also essential for adequate diagnosis in case of valvulopathy or in situation of heart failure without elevated volume of the cardiac cavities. A complete pulmonary edema assessment requires thermodilution and cardiac ultrasound together. At the end, pressures are as important as volumes to identify both components (ARDS and hydrostatic lung edema) in order to guide therapy. Finally, with the Berlin definition of ARDS and in order to adequately diagnose ARDS, we do need two technical investigations: thermodilution and cardiac ultrasound together as thermodilution may miss the diagnosis in 30% of the cases.

Conflict of Interests

The authors declare to have no competing interests

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