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EDITORIAL

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The monitoring of acute cor pulmonale is still necessary in "Berlin" ARDS patients

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ARDS in the new Berlin classification has been defined as a heterogeneous syndrome with impairment of oxygenation not fully explained by cardiac failure or fluid overload with bilateral opacities at chest imaging not fully explained by effusions, lobar/lung collapse, or nodules [1]. From an anatomical point of view the lung in ARDS is characterized by the presence of both alveolar and pulmonary capillary injures which increase the pulmonary arterial pressure [2, 3]. The pulmonary vasculature is a low resistance, high capacitance system, which allows a high increase in blood flow without significant changes in pulmonary artery pressure. The mean determinants of pulmonary artery pressure are the cardiac output, the venous pressure, and the vascular lung area [4]. The pulmonary hypertension promotes right ventricular dilatation/dysfunction, an increase in tricuspid regurgitation, liver and kidney congestion, and owing to interventricular dependency, a left ventricular dysfunction with reduction in blood pressure, cardiac output, and multiple organ failure [4].

The first report of the presence of pulmonary hypertension and elevated pulmonary vascular resistance in patients with severe acute respiratory failure was described by Zapol and Snider [5]. In this study, the patients presented a threefold elevation of pulmonary arterial pressure compared to normal subjects for similar cardiac output, suggesting the alteration in the lung vasculature as the main promoting factor. Pulmonary hypertension which can be potentially reversible is usually due to pulmonary vasoconstriction by hypoxemia, hypercapnia, release of endothelin and thromboxane, and to pulmonary microvascular obliteration by diffuse microembolism and activation of complement [2, 3, 6].

In addition the use of high PEEP levels and elevated plateau pressure can reduce the pulmonary blood flow while increasing the pulmonary artery pressure [7].

Subsequently studies reported a high incidence of pulmonary hypertension in ARDS and also found, in non-survivors, a high pulmonary arterial pressure both at the onset of respiratory failure and during the intensive care stay compared to survivors [8–10]. Moreover a multicenter European intensive care study collecting data from 586 patients showed that the pulmonary arterial pressure was an independent risk factor for mortality [10] suggesting that pulmonary hypertension is not only a marker of the severity of ARDS but also it is harmful due to the associated right ventricular dysfunction and hemodynamic impairment [10].

The right ventricular function and pulmonary circulation have been traditionally evaluated by the pulmonary artery catheter, and pulmonary hypertension has been

defined as a mean pulmonary artery pressure greater than 19 or 25 mmHg [11, 12]. However the pulmonary artery catheter is an invasive technique and can be associated with possible complications. As an alternative Jardin et al. [13] proposed to use echocardiography to detect right ventricular dysfunction. Right ventricular dysfunction or the acute cor pulmonale has been defined by a right ventricular enlargement (right/left ventricular area ratio at end diastole higher than 0.6) plus a systolic septal dyskinesia [14]. Patients with higher mean pulmonary pressure presented a significantly higher end diastolic–systolic right ventricular volume [14]. Applying this definition resulted in an incidence of acute cor pulmonale in ARDS of 61 % in 1985 [13] that decreased to 19–25 % in 2001 [15] and it was associated with a significantly longer mechanical ventilation and worse outcome [13, 15].

In this issue of *Intensive Care Medicine*, Boissier et al. [16] in a monocentric study of 226 consecutive ARDS patients report a 22 % prevalence of acute cor pulmonale. ARDS was defined according to the new Berlin classification [1]. Patients were enrolled over a 5-year period (2004–2009) and they were managed with a lung protective strategy with a tidal volume of 6 mL/kg not exceeding a maximal inspiratory plateau pressure of 30 cmH_2O. Transesophageal echocardiography was performed within the first 3 days after the onset of ARDS. Patients with acute cor pulmonale presented higher respiratory rate, driving pressure (tidal distending pressure), arterial carbon dioxide, and lower respiratory system compliance compared to patients without acute cor pulmonale. Higher values of driving pressure were an independent factor associated with acute cor pulmonale. The incidence of shock and 28-day mortality rate were significantly higher in the acute cor pulmonale group; acute cor pulmonale, driving pressure, and lactic acidosis were independent risk factors for mortality.

What does this study add to the ARDS field? First, using the new Berlin definition for ARDS it confirms the presence of acute cor pulmonale to a similar extent as previous studies in which the patients were managed with a low tidal volume strategy [15, 17]. Second, the size of tidal volume was a risk factor for acute cor pulmonale suggesting the importance to limit end inspiratory stress/strain for reducing the harm not only on the lung parenchyma but also on the right ventricular performance. Third, in addition to the driving pressure, the presence of the acute cor pulmonale significantly increased the risk of mortality. Jardin and Vieillard-Baron nicely showed that for an airway plateau pressure below 26 cmH_2O the mortality rate was no different in patients with and without acute cor pulmonale; conversely for higher plateau pressure the mortality was significantly higher in patients with acute cor pulmonale [18]. Thus in the presence of right ventricular dysfunction a protective ventilation with low plateau pressure is especially recommended.

The right ventricle function in ARDS patients mainly depends on the amount on intrathoracic pressure applied (size of tidal volume and level of PEEP), the gas exchange, and the balance between lung recruitment and overdistension. Consequently, for proper ARDS management (Fig. 1), it is recommended to assess the right ventricular function with at least a pulmonary artery catheter or by transthoracic, transesophageal echocardiography which in addition to the estimation of pulmonary

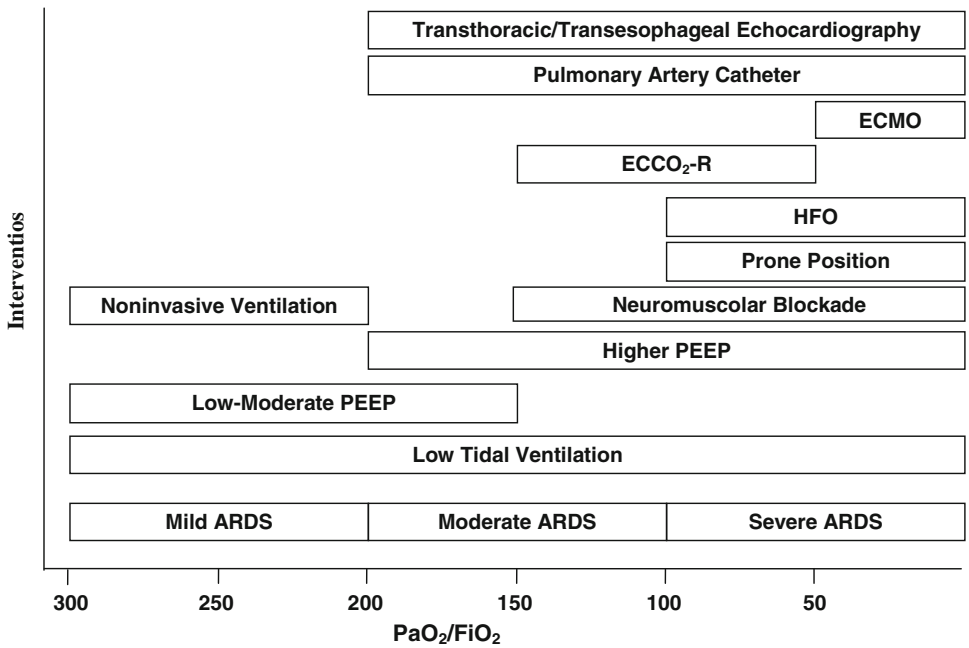

Fig. 1 Suggested potential interventions according to the degree of hypoxemia. Modified from Ferguson et al. [1]

artery pressure can evaluate the presence of a patent foramen ovale [19] and acute cor pulmonale [13]. To eliminate the discontinuous nature of the hemodynamic monitoring of echocardiography a miniaturized transesophageal echocardiography probe which can be inserted in the patients for several days has been developed and it has been shown to provide relevant hemodynamic information [20].

Unfortunately at the present time, no guidelines or consensus statements are available for the management of right ventricular dysfunction in ARDS patients and consequently it is only based on expert opinion.

Conflicts of interest On behalf of all authors, the corresponding author states that there is no conflict of interest.

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