Review Article

Hemodynamic monitoring of ARDS by critical care echocardiography

Mathieu Godement[1], Antoine Vieillard-Baron[1,2]

[1]Medical and Surgical ICU, University hospital Ambroise Paré, Assistance Publique des Hôpitaux de Paris, Boulogne-Billancourt, France; [2]INSERM U-1018, CESP, Team 5 (EpReC, Renal and Cardiovascular Epidemiology), UVSQ, 94807 Villejuif, France

Contributions: (I) Conception and design: None; (II) Administrative support: None; (III) Provision of study materials or patients: None; (IV) Collection and assembly of data: None; (V) Data analysis and interpretation: None; (VI) Manuscript writing: All authors; (VII) Final approval of manuscript: All authors.

Correspondence to: Prof. Antoine Vieillard-Baron. Medical and Surgical ICU, University hospital Ambroise Paré, Assistance Publique des Hôpitaux de Paris, Boulogne-Billancourt, France. Email: antoine.vieillard-baron@aphp.fr.

Abstract: Acute respiratory distress syndrome (ARDS) is a major cause of morbidity and mortality in intensive care units and affects about 10% of critically ill patients and almost 25% of mechanically ventilated patients. It is characterized by life-threatening impairment of pulmonary gas exchange, but in two-thirds of cases is associated with hemodynamic instability. Shock is the primary factor influencing mortality and is driven by sepsis in half of the cases and by a more specific mechanism of ARDS in the other half, which is pulmonary vascular dysfunction, i.e., pulmonary hypertension related to the inflammatory process in the lung, which is very sensitive to a respiratory strategy. ARDS-related right ventricular failure, which is also named acute cor pulmonale (ACP), occurs in 20–25% of patients in the area of lung protective ventilation. In this condition, critical care echocardiography (CCE) plays a central role in adequate hemodynamic assessment and management at the bedside because of its ability to yield information quickly on cardiac dimensions and function, respiratory variations of vena cava dimensions and changes in cardiac output in response to therapy. Added to clinical and laboratory data, with invasive blood pressure monitoring and a central venous catheter, such information can be used to define the cause of circulatory failure, to evaluate the benefit and risk balance of fluid expansion, and to consider a strategy for right ventricle protection. Moreover, in the most severe situations, CCE can also guide the establishment and good functioning of extracorporeal membrane oxygenation (ECMO). In this article, we illustrate and summarize the value of CCE in ARDS and give some physiological pointers to its appropriate use.

Keywords: Acute cor pulmonale (ACP); acute respiratory distress syndrome (ARDS); critical care echocardiography (CCE); extracorporeal membrane oxygenation (ECMO); mechanical ventilation

Received: 23 May 2019; Accepted: 09 July 2019; Published: 27 August 2019.
doi: 10.21037/jeccm.2019.07.04
View this article at: http://dx.doi.org/10.21037/jeccm.2019.07.04

Introduction

Critical care echocardiography (CCE) is more and more frequently used and even more than the pulmonary artery catheter in patients with sepsis (1). In acute respiratory distress syndrome (ARDS) patients, same result was reported in a French database (2). CCE is very suitable for the management of patients with respiratory and circulatory failure (3), as it may directly modify diagnostic and therapeutic procedures. In 2,508 transesophageal echocardiography (TEE) studies, the main indication was hemodynamic instability and findings were left ventricular (LV) dysfunction in 27%, right ventricular (RV) dysfunction in 11% and hypovolemia in 16% of cases. TEE had also a significant on diagnostic and treatment, either surgical or changes in medical therapy (4).

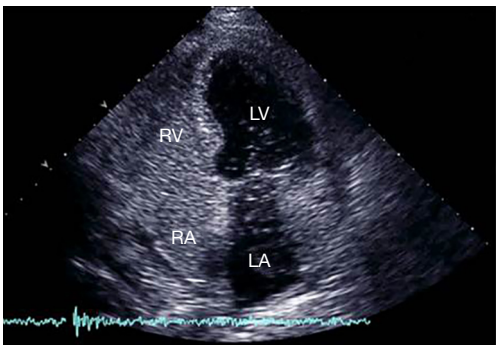

Figure 1 Visualization of an intracardiac shunt through a patent foramen ovale with agitated saline in a patient ventilated for an ARDS with transthoracic echocardiography. ARDS, acute respiratory distress syndrome; LV, left ventricle; LA, left atrium; RV, right ventricle; RA, right atrium.

A consensus of experts in the field of hemodynamic monitoring recognized CCE as a true hemodynamic monitoring device, albeit discontinuous (5). An echo study has to be done as soon as possible in the case of hemodynamic instability, if the patient is not obviously fluid-responsive with low central venous pressure (5). TEE is probably less operator-dependent than transthoracic echocardiography (TTE), the reason why TEE is better than TTE for hemodynamic monitoring when performed in intubated patients; it then allows reproducible and sequential hemodynamic assessments (6). For an accurate hemodynamic evaluation, it has been reported that intensivists may use TEE in a qualitative approach based on 4 main parameters: respiratory variation of the superior vena cava (SVC), LV systolic function, RV size, and paradoxical septal motion (7).

CCE is therefore key in patients developing ARDS and this review illustrates and discusses the main reasons why, which are defining the cause of circulatory failure, optimizing hemodynamic and respiratory management by looking at the effect of mechanical ventilation on RV function which requires good knowledge of heart-lung interactions, and managing veno-venous extracorporeal membrane oxygenation (VV ECMO), if required.

ARDS and circulatory failure

Fifty years after its first description by Ashbaugh *et al.* (8), the management of ARDS remains a challenge for intensivists. Despite advances in supportive care during the last ten years, ARDS is still associated with high mortality and morbidity. In a recent large multicenter prospective cohort study conducted in 50 countries in different continents, the prevalence of ARDS was 10.4% among admissions to the ICU, with an in-hospital mortality of 40% (9). ARDS is frequently associated with hemodynamic instability. In one study more than 60% of patients experience shock (10) and in other studies around 65% require infusion of catecholamines (11,12). Circulatory failure appears as the main factor associated with mortality (13). The definition of ARDS recently revisited by an expert panel (14) is still debated because neither the stratification by severity nor the PaO_2/FiO_2 ratio was independently associated with mortality (15). In the case of shock, the PaO_2/FiO_2 ratio as a marker of severity remains questionable because some hemodynamic effects can interact. For example, the low PvO_2 effect or the reopening of a patent foramen ovale (*Figure 1*) tends to overestimate the severity of ARDS. Conversely, a low cardiac index may decrease lung shunt, leading to underestimation of severity. In this way, refractory hypoxemia is rarely the cause of death and most patients die from shock and multiorgan failure (9).

In ARDS, shock is schematically driven by two main mechanisms. On the one hand, there is an obstructive mechanism due to the effect of pulmonary hypertension on RV function, resulting from alteration of the pulmonary capillary circulation (16) and the deleterious effects of mechanical ventilation (MV) (17). On the other hand, sepsis-related circulatory failure occurs in half of patients (18). In this case, it may be associated with relative or absolute hypovolemia, vasoplegia, and myocardial depression (19), and the role of CCE is in part discussed by De Backer and colleagues in another paper of this issue dedicated to critical care ultrasonography. Briefly, the complexity and heterogeneity of patients with septic shock implies individualized approaches to hemodynamic management and CCE is very suitable as it may detect combined mechanisms of circulatory failure. A recent study using a clustering approach based on clinical and CCE parameters in a large population of septic shock patients characterized different cardiovascular phenotypes. Five different clusters were defined: well resuscitated, LV systolic dysfunction, LV hyperkinesia, hypovolemia, and right ventricular failure. This new approach to the characterization of shock is especially interesting as it could help physicians to optimize hemodynamic support quickly at the bedside (20). The only specificity in ARDS is the application of a low tidal volume which may limit the accuracy of the parameters of fluid responsiveness, while the largest study

in the field included 22% of ARDS and most patients were ventilated with a tidal volume below 8 mL/kg (21). This study reported respiratory variations of the SVC as the most specific parameter of fluid responsiveness (21) (*Figure 2*). ARDS-related RV failure is the main mechanism of shock for the remaining unstable ARDS patients. It is related to an abrupt increase in pulmonary artery pressure. Most studies report the incidence of acute cor pulmonale (ACP), echocardiographically defined by RV dilatation with paradoxical septal motion. However, based on the physiology of the right ventricle, experts have rather defined RV failure as a "significant" RV dilatation leading to systemic venous congestion (22,23), which may be highly suspected by CCE (*Figure 3A*). Paradoxical septal motion, when present, reflects the fact that this RV failure is mediated by systolic overload (*Figure 3B*). The prevalence of ACP during ARDS reaches 20–25% in studies where patients are ventilated with a lung protective approach; its occurrence has been reported to be associated with higher mortality (24,25). Nevertheless, both mechanisms of circulatory failure in ARDS, sepsis and pulmonary hypertension, are often interlinked since pneumonia is the first cause of ARDS and has also been identified as an independent risk factor for ACP (10).

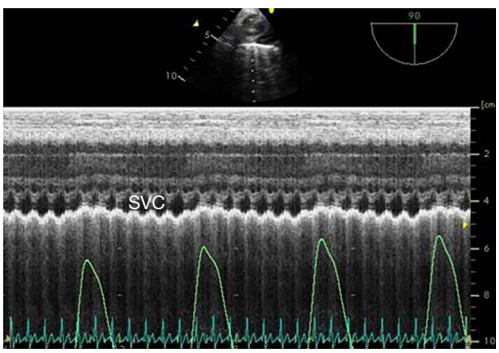

Figure 2 Longitudinal upper esophageal view of the superior vena cava (SVC), combining 2-D and M modes, showing cyclic collapse of the superior vena cava in a patient in shock, still hypovolemic with ARDS-related septic shock. ARDS, acute respiratory distress syndrome.

Hemodynamic monitoring in ARDS by CCE

CCE has a growing role in the management of ARDS patients, mainly because of the huge interactions between the lung (and then ventilation) and the right ventricle. The choice of the modality of echocardiography (transthoracic, TTE versus TEE) strictly depends on the clinical condition of individual patients, on the skills of the operator, as well as on the clinical inquiry. If possible, TEE should be preferred since it overcomes technical problems associated with acoustic views and measurements can usually be performed easily (26). Since patients with severe ARDS often require prolonged periods of prone positioning, TEE has also be proven to be performed safely and efficiently in this situation (27). Finally, it has been reported that TEE is more accurate than TTE in reporting ACP (24) and that factors limiting the feasibility of TTE are high PEEP or significant weight gain (28), both of which are frequently observed in ARDS.

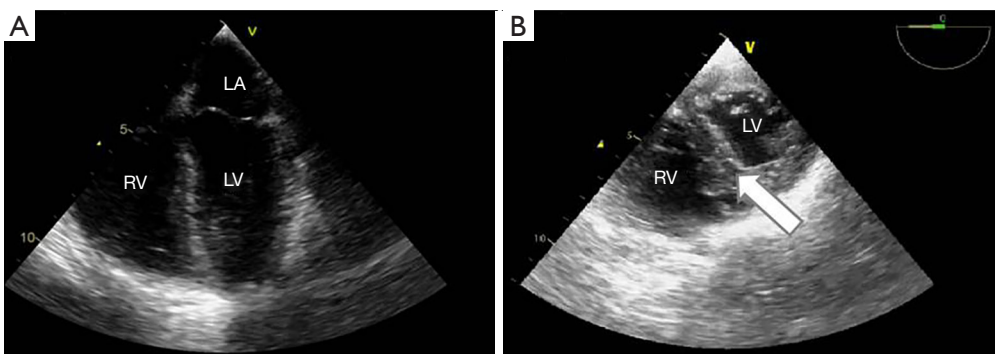

Figure 3 Acute cor pulmonale in a patient ventilated with a lung protective approach. (A) Mid-esophageal view demonstrating a severe dilatation of the right ventricle, a marker of RV failure. (B) Short axis view by a transgastric approach visualizing a paradoxical septal motion (arrow) with the "D-shape" of the left ventricle, reflecting that RV failure is related to systolic overload (abrupt increase in RV afterload). LA, left atrium; RV, right ventricle; LV, left ventricle.

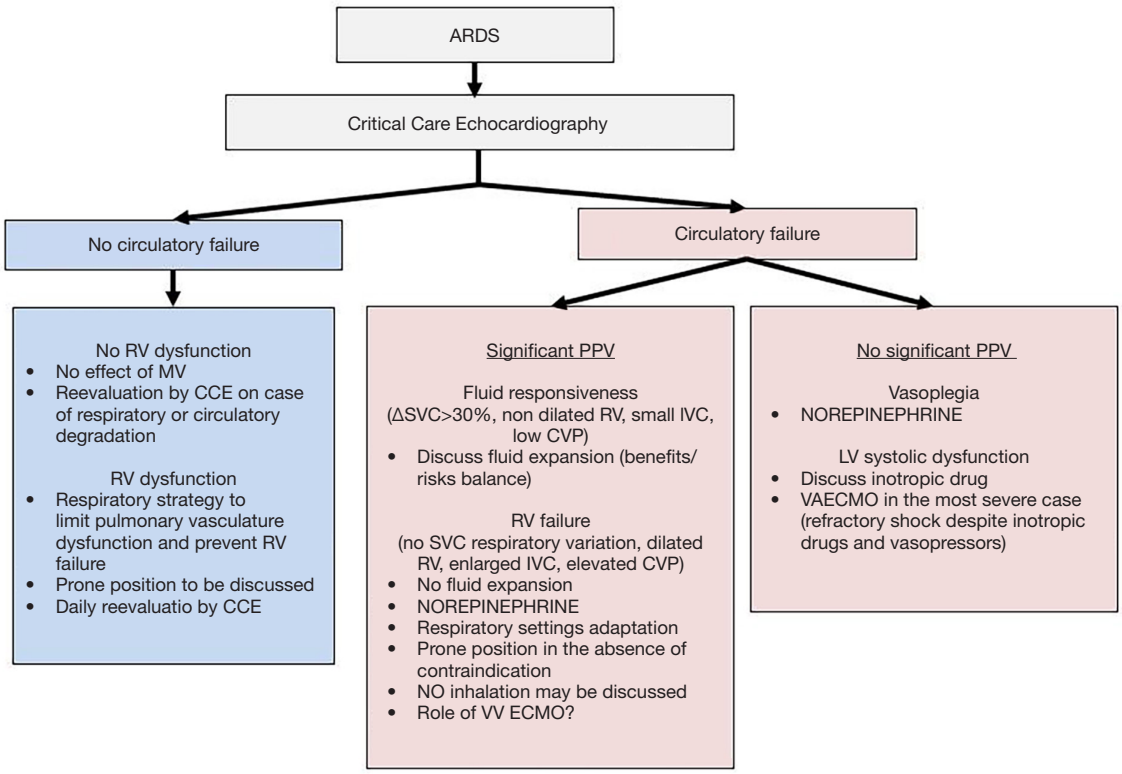

Figure 4 Algorithm of hemodynamic management based on the CCE for ARDS patient. ARDS, acute respiratory distress syndrome.

During ARDS, assessment of RV size is a key objective. It may be easily reached by comparing the RV end-diastolic area (RVEDA) with the LV end-diastolic area (LVEDA) (26). An RVEDA/LVEDA ratio between 0.6 and 1 indicates moderate RV dilatation, while an RVEDA/LVEDA ratio >1 indicates severe RV dilatation. ACP has been defined as the combination of an RVEDA/LVEDA ratio >0.6 and the presence of paradoxical septal motion during end-systole (24) (*Figure 3*). LV ejection fraction should also be assessed and clinically interpreted taking into account the dosage of inotropic and vasoactive drugs, if administered. Valvular diseases (especially mitral and aortic regurgitation) should also be investigated as they could influence the course and severity of the lung disease and influence treatments, as for fluid requirement. Since a patent foramen ovale may worsen hypoxemia in ARDS patients, echocardiographic assessment should establish whether or not it is present, and, if so, its severity (*Figure 1*). Patent foramen ovale has recently been reported to concern between 16 and 19% of ARDS patients (29).

Because of positive pressure ventilation on the one hand, and the dramatic decrease in lung compliance on the other hand, ARDS patients are especially subject to wide variations of airway pressure, especially alveolar and transpulmonary pressures. Some experts have recently emphasized the pivotal role of evaluating such interactions for hemodynamic assessment of patients suffering from ARDS (30). They proposed combining continuous invasive blood pressure monitoring with CCE and the use of pulse pressure variation (PPV) to dichotomize ARDS patients (30). As CCE only allows discontinuous hemodynamic monitoring, it has to be associated with a more continuous modality to have real-time monitoring of blood pressure which can be used as a "warning" signal in the case of significant PPV. From a practical point of view, the absence of any PPV indicates that the circulatory system (both LV and RV) is not altered by the effect of mechanical ventilation. Conversely, significant PPV mainly illustrates the effects of tidal inflation on the right ventricle and forces physicians to explore the underlying mechanisms further by performing CCE. Thereafter, two different situations well characterized by CCE may explain PPV: a decrease in RV preload or an increase in RV afterload. In *Figure 4*, we propose an algorithm of hemodynamic management based

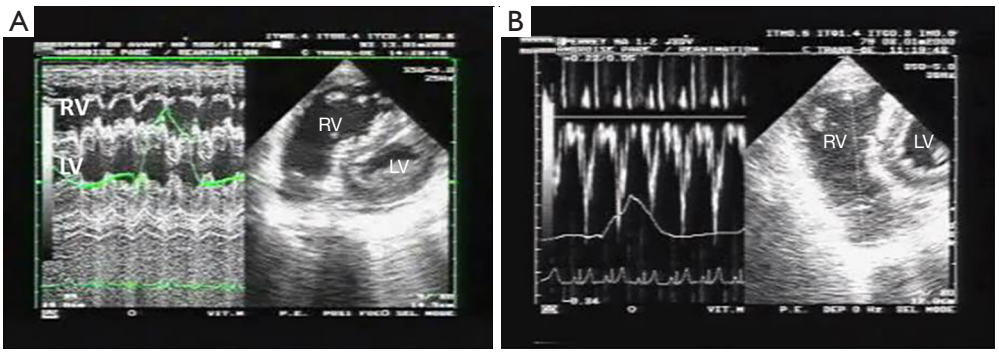

Figure 5 This ventilated patient exhibited cyclic RV dilatation at each tidal inflation (A), associated with a significant decrease in RV stroke volume (B). Superior vena cava did not exhibit any respiratory variation. RV, right ventricle; LV, left ventricle.

on the association of blood pressure monitoring and CCE.

Decrease in RV preload during tidal ventilation is the consequence of the decrease in systemic venous return because of the increase in intrathoracic pressure, suggesting that the patient is fluid-responsive. Management of fluids is still the subject of intense debate in the intensive care community. Besides the fact that hypovolemia when not corrected may promote shock and organ hypoperfusion, and is a confounder for severity evaluation as previously discussed, the main issue is identification of those fluid-responsive patients who would really benefit from more fluid, bearing in mind that positive fluid balance is associated with a worse outcome in ARDS (31,32). Although it does not decrease mortality, fluid restriction improves oxygenation and lung injury score (33,34). For such tricky management, respiratory variation of the SVC (ΔSVC) evaluated by TEE is especially interesting as it could reflect the conflict between the amount of blood in the thorax and the respiratory settings. Furthermore, ΔSVC, when adequately used, is much more specific than sensitive (21) (*Figure 2*). In cases of a non-dilated right ventricle, significant ΔSVC, and low central venous pressure (or small inferior vena cava by a subcostal view), PPV can help physicians to gauge the benefits and risks of fluid expansion (30).

PPV can also illustrate the increase in RV afterload driven by high transpulmonary pressure. Decreased lung compliance leads to a significant increase in transpulmonary pressure, i.e., plateau pressure minus intrathoracic pressure (35,36). Increase in transpulmonary pressure and RV afterload are well correlated (37,38) and ARDS patients are then exposed to the risk of RV failure (10). In this situation, PPV, when observed, is classically described by CCE as the association of a decrease in RV stroke volume with RV dilatation (either cyclic during tidal ventilation or constant along the respiratory cycle), dilated inferior vena cava, high central venous pressure (CVP), and no or minimal ΔSVC (30) (*Figure 5*). In these cases, PPV, when significant, becomes a false positive for fluid responsiveness (39) and must lead to adaptation of the respiratory settings, the control of hypercapnia, or discussion of prone positioning of the patient, while more fluid expansion is not indicated or even contraindicated as it can worsen RV failure (40).

Furthermore, CCE is not performed blindly but rather because of abnormal vital signs suggesting poor organ perfusion (skin mottling, elevated lactate, oliguria, and so on). Moreover, CVP, when combined with CCE, may be informative, low CVP suggesting a potentially underfilled right ventricle and high CVP an overloaded right ventricle. CCE allows a paradigm shift from invasive, quantitative, and continuous hemodynamic monitoring to less invasive, qualitative, discontinuous and functional monitoring (41,42). This is why a single measurement of cardiac output is probably less informative than serial evaluations, before/after fluids, before/after dobutamine, before/after application of a high PEEP, or before/after prone positioning. For this latter indication, CCE visualizes the improvement of RV function following prone positioning (43). In a systematic review, Wetterslev *et al.* reported that cardiac output measurements using either echocardiography or thermodilution were not interchangeable, while trends were (42). According to our experience, we suggest that CCE be performed on ICU admission in ARDS patients to provide valuable information, not only to evaluate the clinical status of that moment but also to highlight clinical elements indicating previous diseases (valvular disease, pre-existing chronic heart disease…). Thereafter, CCE should be periodically repeated according to the clinical course, but certainly should be done at least once a day. Besides, patients should

be re-evaluated with CCE whenever higher ventilatory support is required or any new undifferentiated state of shock develops or a significant PPV "warning" signal is present. Although not yet validated in a randomized controlled study, an RV protective approach in part based on the evaluation of RV function by CCE has recently been formalized, putting the pulmonary circulation and the right ventricle at the center of the decision-making process, by correcting the risk factors for developing RV failure, a PaO_2/FiO_2 ratio <150 mmHg, a driving pressure ≥ 18 cmH_2O, and a $PaCO_2$ ≥48 mmHg (10).

Potential role of CCE in managing extracorporeal membrane oxygenation

ECMO has been known since the 1970's, but its use has exploded in the last 10 years. Although its value in ARDS is still questionable, some experts suggest that VV ECMO could represent an effective bridge to recovery in patients with very severe ARDS and to standard medical management including prone positioning in whom either hypoxemia is refractory or lung protective ventilation is no longer feasible (44). VV ECMO could then facilitate the use of more protective ventilation by removing carbon dioxide from the blood and increasing blood oxygenation (45). CCE may be used for different objectives: (I) to evaluate RV function before ECMO, (II) to evaluate RV function during ECMO so as to see whether the previously failing right ventricle is fully unloaded, (III) to check that the cannula is properly positioned, (IV) to monitor cardiac output as during ECMO most devices based on thermodilution cannot be accurately used.

To date, there is a paucity of data on cardiac function assessed by CCE in patients with ARDS unresponsive to conventional therapy and then submitted to VV ECMO. In a recent prospective study of echocardiographic evaluation before ECMO initiation, 34% of the patients showed normal findings, 43% exhibited isolated pulmonary hypertension, and 23% showed RV dilation and pulmonary hypertension (46). VV ECMO has been proven to reduce RV afterload (47). In 13 consecutive patients with severe respiratory failure requiring VV ECMO, Miranda *et al.* reported that pulmonary artery pressure dropped significantly immediately after starting VV ECMO before ventilator settings were altered, followed by a slight drop in CVP and an increase in cardiac index, with stable doses of vasopressors (48). Both oxygenation and decarboxylation play a role here in unloading the right ventricle. Therefore, the presence of pulmonary hypertension in patients with severe ARDS may not be a contraindication for VV ECMO, as this may even alleviate RV failure related to the pulmonary hypertension. Those results, combined with the negative experience of venoarterial (VA) ECMO for ARDS during the 1980's, explain the fact that most refractory ARDS patients are now initiated on VV ECMO, when required. This is illustrated by a study based on the ELSO registry, in which only 18% of ARDS patients with shock (defined by the need for one or more inotropes/vasopressors) were started on VA ECMO, and this percentage seemed to drop over the years (49). The need for a conversion from VV to VA ECMO in this registry is reported as only 4.1% (49). The same results were observed in the recently published Eolia trial, in which only 6% of the patients receiving VV ECMO were switched to VA or conversely (44). However, VA ECMO could certainly be considered when ARDS is associated with severe RV failure leading to "obstructive" shock (30) (or when combined with severe septic cardiomyopathy leading to very low cardiac output and severely reduced LV ejection fraction despite adequate use of inotropic support and/or norepinephrine (50).

Conclusions

CCE is an incredible technique for hemodynamic monitoring in ARDS. It may allow the independent determination of the need for more fluid, for norepinephrine or dobutamine infusion, but also for adjustment of the mechanical ventilation settings and respiratory strategy. In the most severe cases, CCE affords support to the setting up and tailoring of ECMO, if used. Since CCE is never performed blindly, intensivists should remember that it helps improve patient management and is not a goal in itself. It means that intensivists should not treat an "abnormal" echo picture, but rather interpret echo studies in light of the clinical situation. CCE should always be associated with clinical and laboratory data, continuous invasive monitoring of blood pressure, and a central venous catheter in severe cases.

Acknowledgments

None.

Footnote

Conflicts of Interest: The authors have no conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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doi: 10.21037/jeccm.2019.07.04
Cite this article as: Godement M, Vieillard-Baron A. Hemodynamic monitoring of ARDS by critical care echocardiography. J Emerg Crit Care Med 2019;3:36.

