Assessment of right ventricular function
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Purpose of review
To emphasize the importance and clinical implications of right ventricular function assessment in many situations encountered by intensivists and to explain in practical terms how to perform such an assessment at the bedside.

Recent findings
Assessment of right ventricular function requires a full understanding of the physiology of the right ventricle. The right ventricle, unlike the left ventricle, is sensitive to acute increases in its afterload. The right ventricle is usually dilated when its failure is the cause of shock. Three possible causes of right ventricular failure must be evaluated: decrease in contractility, pressure overload and volume overload. Many devices are available to assess right ventricular performance, but they are not equal in terms of accuracy and invasiveness. Although noncontinuous, echocardiography is probably the best device and this technique can also be employed by nonexpert intensivists. Two main signs must be sought: right ventricular dilatation and paradoxical septal motion.

Summary
Assessment of right ventricular function in the ICU is crucial in many situations because right ventricular failure may be responsible for, or participate in, shock in pulmonary embolism, acute respiratory distress syndrome and septic shock. The best method is echocardiography, which is noninvasive and accurate.

Keywords
echocardiography, hemodynamics, right ventricular function

Introduction
Right ventricular function [1**] is crucial for hemodynamics in many clinical situations encountered by intensivists for many reasons. The first reason relates to the physiology of the right heart. In normal conditions, the right ventricle pumps blood to the pulmonary circulation at a low pressure. The normal right ventricular pressure/volume loop differs from that of the left ventricle: the isovolumetric contraction pressure is very low, and the right ventricle continues to eject blood into the circulation long after the beginning of its relaxation [2]. This illustrates the fact that right ventricular systolic function, unlike left ventricular systolic function, is sensitive to any acute increase in afterload. Because the wall stress of the right ventricle is low in normal conditions, coronary perfusion occurs during both diastole and systole [3]. Coronary perfusion is related in part to the pressure gradient between aortic pressure and right atrial pressure, and so perfusion of a dilated and overdistended right ventricle may be very sensitive to changes in blood pressure. Normalization of blood pressure, by norepinephrine for instance, may help the failed right ventricle by partly correcting ‘functional’ ischemia [4].

The second reason is that the right and the left ventricles are located together in a closed space within the pericardium. Thus, acute right ventricular dilatation may restrict the left ventricle and thus impair its diastolic function [5,6]. This phenomenon is called right ventricular/left ventricular interdependence and explains why in some situations the right ventricle may be preload-independent, whereas the left ventricle is strongly preload-dependent.

Finally, the third reason is that the systemic venous return is directly proportional to the capacity of the right ventricle to pump blood and thus to reduce right atrial pressure (RAP) [7]. Right ventricular function impairment can induce an increase in RAP and then a decrease in systemic venous return. In this setting, the main factor limiting the efficacy of a blood volume expansion may be right ventricular rather than left ventricular function [8].
Incidence and clinical consequences of right ventricular dysfunction

Right ventricular dysfunction may be related to decreased contractility, right ventricular pressure overload or right ventricular volume overload [9], and thus right ventricular function assessment should take into account contractility, preload and afterload. Four clinical situations often encountered in the ICU are especially associated with right ventricular dysfunction: pulmonary embolism, severe acute respiratory distress syndrome (ARDS), septic shock and right ventricular infarction.

Right ventricular infarction is a special case because, in this setting, right ventricular failure is the cause and not the consequence of the disease. We will not deal with right ventricular infarction in this review.

In 161 patients hospitalized for an anatomically massive pulmonary embolism, we have reported a 61% incidence of acute cor pulmonale [10]. Acute cor pulmonale reflects acute right ventricular dysfunction related to an abrupt increase in right ventricular afterload and is usually detected by echocardiography (see below). As recently reemphasized by European experts, detection of acute cor pulmonale allows stratification of patients according to their risk of mortality [11]. The value of right ventricular function assessment in deciding on the utility of fibrinolysis in acute pulmonary embolism, however, is still debated [11*].

Recently, in 75 patients ventilated for ARDS and treated with protective mechanical ventilation, we reported a 25% incidence of acute cor pulmonale [12]. Whereas the obstruction of the pulmonary circulation is proximal in massive pulmonary embolism, it is more distal during ARDS and relates both to injury to the pulmonary circulation and to injury induced by mechanical ventilation [13,14]. In the past, when ventilation was more aggressive, injury of the pulmonary circulation resulted from destruction of pulmonary capillaries and tended to be irreversible. Now, when a protective ventilation strategy is applied, injury consists of remodeling, (i.e. musculization of normally nonmuscularized arteries, mediated by hypoxia and hypercapnia) and may be reversible [15]. Acute cor pulmonale has many consequences for these patients. Most importantly, right ventricular dysfunction is independently associated with mortality [16,17*]. This suggests that adaptation of ventilatory settings to right ventricular function may be beneficial [18*]. Adapting ventilatory settings mainly consists in strictly limiting plateau pressure to below 30 cmH\textsubscript{2}O, (ideally below 27 cmH\textsubscript{2}O) when the right ventricle fails [19*]. Second, if prone positioning improves oxygenation, this may confer a benefit in terms of right ventricular function [20*].

Finally, many studies have reported the presence of right ventricular dysfunction in patients with septic shock. Kimchi et al. [21] have reported an incidence as high as 50% and we have reported an incidence higher than 30% [22]. This dysfunction may be related to sepsis per se, but in some patients may also result from acute lung injury requiring positive pressure ventilation. It has several consequences: a ‘beneficial’ one because it may protect the pulmonary circulation, which explains why left ventricular systolic dysfunction in sepsis is very uncommonly associated with high filling pressures [23]; but it limits the efficacy of blood volume expansion, even if the left ventricle is preload-sensitive. Interestingly, we have demonstrated in septic shock patients that significant pulse pressure variations are not necessarily associated with fluid responsiveness, but can be related to right ventricular failure (Fig. 1) [24].

Figure 1 Significant pulse pressure variations

![Figure 1](https://example.com/figure1.png)

Significant pulse pressure variations (a) in a patient hospitalized for septic shock and mechanically ventilated. Variations were due to the presence of acute cor pulmonale related to acute respiratory distress syndrome (ARDS; b). Low blood pressure and pulse pressure variations were not corrected by fluid infusion. FC, heart rate; LA, left atrium; LV, left ventricle; PA, arterial pressure; PPV, pulse pressure variation; RA, right atrium; RV, right ventricle.
Figure 2 Graphic representation of different hemodynamic tools, according to their invasiveness and their clinical effectiveness at the bedside in evaluating right ventricular function

Echocardiography is the best tool, whereas transpulmonary thermodilution, esophageal Doppler and radionuclide ventriculography are not suitable.

CO, cardiac output; EDA, end-diastolic area; EF, ejection fraction; PAC, pulmonary artery catheter; PAOP, pulmonary artery occlusion pressure; PAPd, diastolic pulmonary artery pressure; RAP, right atrial pressure; RV, right ventricle; SI, stroke index; TEE, transesophageal echocardiography; TPT, transpulmonary thermodilution; TTE, transthoracic echocardiography.

Figure 3 Long axis of the left ventricle by a transesophageal approach

The patient in panel (a) had a normal right ventricle, with a triangular shaped filling chamber. The patient in panel (b) had a severely dilated right ventricle: the right ventricle had lost its normal shape and the right ventricular/left ventricular end-diastolic area ratio was more than 1. LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.
How to assess right ventricular function at the bedside

Intensivists have to keep in mind that right ventricular function may change very quickly depending on the time of evaluation: before or after fibrinolysis? With a high plateau pressure or after adjusting ventilatory settings? Before or after volume expansion? With or without noradrenaline? Thus, right ventricular function evaluation must be repeated throughout the patient’s clinical course and after treatment changes. This does not necessarily require continuous monitoring, but rather evaluations at different time points.

Many tools have been proposed over the past 30 years, from more sophisticated and invasive ones to simpler, noninvasive ones. These techniques are not equal in terms of efficacy and accuracy (Fig. 2). In 1984, Kimchi et al. [21] were the first to evaluate right ventricular ejection fraction using radionuclide ventriculography in 25 septic shock patients. They defined right ventricular dysfunction as a right ventricular ejection fraction below 38% [21]. A few years later, Parker et al. [25] also studied right ventricular function in septic patients using a bedside nuclear camera and demonstrated that a decrease in right ventricular ejection fraction was associated, in 82% of cases, with a decrease in left ventricular ejection fraction. However, this technique has pitfalls and is difficult to use in clinical practice.

Some physicians have proposed calculating right ventricular ejection fraction with a modified pulmonary artery catheter equipped with a fast response thermistor. In 1989, Vincent et al. [26] reported in septic shock patients a mean right ventricular ejection fraction of 24% compared with 32% in ‘controls’. Using the same technique, Jardin et al. [27] studied right ventricular function in ARDS. However, whatever the known limitations of thermodilution in mechanically ventilated patients, ejection fraction is not well suited physiologically for accurate evaluation of right ventricular function. Its ‘normal’ value is unknown because its values in healthy volunteers vary widely, from 30 to 60%. In normal conditions, the right ventricle can be comparable to a passive conduit, and...
thus in patients, clinical status does not depend on right ventricular contractility per se, but on the relation between contractility and afterload. For instance, a right ventricle with an ejection fraction of 30% and a low afterload is able to eject blood normally into the pulmonary circulation, whereas a right ventricle with an ejection fraction of 40% and a significantly increased afterload may be responsible for a critical clinical situation.

Echocardiography allows for direct and noninvasive visualization of the right ventricle and is the best current tool for evaluating right ventricular function at the bedside. A simple evaluation can be done by nonexpert physicians or a more sophisticated evaluation by experts [28**]. Physicians who are not experts in echocardiography have to keep in mind that a severely failed right ventricle is dilated, and it is very unlikely that the right ventricle is the cause of shock if right ventricular size is normal. Thus, physicians need to be able to recognize dilatation. Most of the time, this is easy because the dilated right ventricle loses its typical triangular shape (Fig. 3). The best way to assess right ventricular size is to calculate the right ventricular/left ventricular end-diastolic area ratio in the four-chamber view [29]. A normal ratio is below 0.6 [30]. We have especially defined right ventricular dilatation as severe when the right ventricle is bigger than the left ventricle [29]. In this situation, experts have recommended avoidance of fluid infusion [31]. The second echocardiographic sign that nonexperts in echocardiography have to detect is paradoxical septal motion in systole (Fig. 4), which reflects the presence of right ventricular systolic overload. Acute cor pulmonale, which occurs in different situations and has clinical consequences, as noted above, is associated with a combination of dilatation and septal dyskinesia in systole [32]. Finally, in expert hands, right ventricular function assessment by echocardiography can be completed using the Doppler method. In particular, recording of the right ventricular ejection flow is very informative [32]. We favor measuring acceleration time of the flow because its shortening (<100 ms) usually reflects pulmonary hypertension. We note the pattern of right ventricular ejection flow: biphasic flow is very suggestive of acute proximal or distal pulmonary obstruction and its normalization always reflects right ventricular function improvement (Fig. 5).

Figure 6 Significant pressure gradient between pulmonary capillary wedge pressure and diastolic pulmonary artery pressure

Significant pressure gradient between pulmonary capillary wedge pressure and diastolic pulmonary artery pressure in a patient with a massive pulmonary embolism (a) and a patient with severe acute respiratory distress syndrome (ARDS; b). In the patient with massive pulmonary embolism, note that right atrial pressure was higher than pulmonary capillary wedge pressure. In the ARDS patient, application of a positive end-expiratory pressure of 20 cmH₂O induced a marked increase in the pressure gradient, reflecting poor right ventricular tolerance. PAP, pulmonary artery pressure; PCWP, pulmonary capillary wedge pressure; RAP, right atrial pressure.
In patients with a pulmonary artery catheter in place, few hemodynamic patterns have proven to be good surrogates of acute cor pulmonale. Monchi et al. [16] and Osman et al. [17] have considered ARDS patients to have acute cor pulmonale when central venous pressure is higher than pulmonary artery occlusion pressure. Others have calculated the pressure gradient between diastolic pulmonary artery pressure and pulmonary capillary wedge pressure (Fig. 6) [33]. But these definitions have not been adequately validated and need to be used with caution. Although pulmonary artery pressures do not speak directly to right ventricular size and function, their response to therapy, taken along with the response of stroke volume, may be useful in some settings.

Finally, new approaches, such as transpulmonary thermodilution, have recently been proposed for hemodynamic monitoring. Physicians need to be aware that they are totally inaccurate for detecting right ventricular dysfunction [34]. They can only detect a low-flow state, without indicating its origin. The same is true for esophageal Doppler.

Conclusion
Assessment of right ventricular function in the ICU is crucial in many situations because right ventricular failure may be responsible for, or participate in, shock in pulmonary embolism, ARDS and septic shock. The best method is echocardiography, which is noninvasive and accurate.

References and recommended reading
Papers of particular interest, published within the annual period of review, have been highlighted as:
• of special interest
•• of outstanding interest

Additional references related to this topic can also be found in the Current World Literature section in this issue (pp. 274–275).


18. Vieillard-Baron A. Is right ventricular function the one that matters in ARDS?•• of special interest Definitively yes. Intensive Care Med 2009; 35:4–6; doi: 10.1007/ s00134-008-1307-1 1; 11. This editorial explains why and how right ventricular dysfunction can be independently associated with poor prognosis in ARDS. This reemphasizes the fact that right ventricular function assessment must be taken into account in management of ventilatory settings.


This study reports in a very large cohort of ARDS patients the influence of plateau pressure on mortality and on incidence of acute cor pulmonale.


This study demonstrates that, in severe ARDS, prone positioning improves not only oxygenation, but also right ventricular function in patients with acute cor pulmonale.


Cardiopulmonary monitoring


