The Right Ventricle in Critical Illness

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Abstract: Right ventricular (RV) dysfunction frequently occurs in critically ill patients. Common conditions associated with RV dysfunction encountered by the intensivist include acute cor pulmonale, sepsis, severe left ventricular (LV) failure, and RV infarction. Echocardiography is an invaluable tool that allows quick and accurate assessment of RV function at the bedside. Serial echocardiographic evaluation also permits dynamic monitoring of the RV function to assess for disease progression or response to therapy.

This review describes simple methods of two-dimensional (2D) echocardiographic evaluation of RV function as relevant to the most commonly encountered clinical scenarios in the intensive care unit (ICU). To perform this evaluation, the clinician requires only basic ultrasound skills. Advanced echocardiographic assessment, such as application of Doppler analysis, is beyond the scope of this article.

Keywords: Shock, Fluid responsiveness, Pulmonary embolus, Left ventricular dysfunction, Cardiac tamponade, Focused ultrasound examination.

NORMAL RV ANATOMY AND PHYSIOLOGY

The RV is comprised by two distinct parts: the body and outflow tract. The superficial muscle fibers in the body of the RV are continuous with those of the LV, whereas deeper muscular layers are in continuity with the interventricular septum (IVS). The RV contraction occurs in a peristalsis-like motion, starting at the base with the outflow tract contracting 25-50 msec later and remains contracted into early diastole.

There are some striking differences between the structure and function of the RV compared to the LV. The muscle mass of the RV is approximately one sixth of that of the LV. The end-diastolic thickness of the RV wall is ≤ 3mm, compared to ≤11mm for the LV. Coronary perfusion of the RV occurs continuously, while the LV, because of the high pressure that it mounts within its walls during contraction, is perfused almost exclusively during diastole [1, 2]. The RV pumps the same volume of blood as the LV, but it does so against the lower pressure of the pulmonary vasculature, which requires only one-sixth of the work. In cross section, unlike the circular structure of the LV, the RV has a semi-circular, serpentine shape. This structure is thought to account for its dramatic volume-accommodating properties while maintaining normal cardiac output. On the other hand, such a structure is suboptimal in mounting high pressure. Indeed, large changes in RV preload do not generate substantial Frank-Starling forces, up until the point when the RV becomes more circular in shape [1, 2].

Both the RV and the LV make contributions to the RV output [3]. Under normal circumstances those contribution are relatively independent, and it has been shown that even if the pressure inside the LV cavity approaches zero, the RV is still able to maintain normal pressures [4]. However, under conditions of RV and/or LV failure the functions of the two ventricles become intimately interdependent, and an increase in RV volume may cause a reduction in LV volume *via* compression of the IVS.

Unlike the LV, when faced with an acutely elevated after-load the RV increases its stroke work *via* increase in contractility (so-called *Anrep effect*), with only minimal contributions from the Frank-Starling mechanism or adrenergic stimulation [5, 6]. In this situation, because elevated tissue pressure during systole may restrict RV coronary flow to diastole only, a strained RV has the potential to become ischemic. As compensatory mechanisms are exhausted, the LV, which normally contributes about 25% to the RV work *via* IVS contraction, may further enhance RV stroke volume by increasing the IVS contribution to as much as 35% [7]. However, at some point the RV may be so pressure and volume overloaded, that its further dilation results in LV compression reduction in LV cavity size and a drop in cardiac output. This clinical picture is reflected by the echocardiographic D-sign (see below), and may herald impending cardiovascular collapse.

ECHOCARDIOGRAPHIC DETECTION OF RV PATHOLOGY

In the ICU setting, the bedside RV echocardiography begins with an assessment of RV size. The apical four-chamber view can be used to first appreciate the shape of the RV. In this view the RV normally has a triangular shape, which becomes more oval as its pathologic dilation occurs. There are many quantitative parameters that can help the intensivist evaluate the RV size. In practice however, the easiest way to do so is to compare the RV to the LV. The RV inner surface area at end-diastole should normally be no more than 0.6 that of the LV. Moderate RV dilation would elevate this ratio to 0.7-0.9, and severe dilation to > 1 [8].

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Moreover, instead of measuring the surface area ratio directly, with some experience the intensivist will simply compare the RV-to-LV size and subjectively classify the RV as normal (Fig. **1**), moderately enlarged (Fig. **2**), or severely enlarged (Figs. **3**, **4**). The accuracy of this simplified technique was demonstrated by Viellard-Baron *et al*. In their study, among the ICU echocardiographers there was a high correlation between the subjective RV to LV size comparison and the actual quantitative measurement of the RV/LV ratio, with good interobserver variability [9].

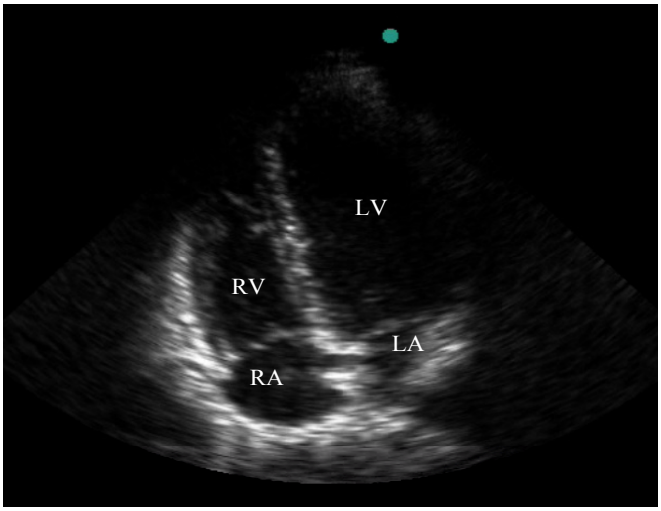

Fig. (1). Normal right ventricle in the apical four-chamber view. Note the following: the size of the RV cavity is appreciably smaller than that of the LV; the RV has a normal triangular shape in this view. Note also how the heart apex is normally comprised by the LV.

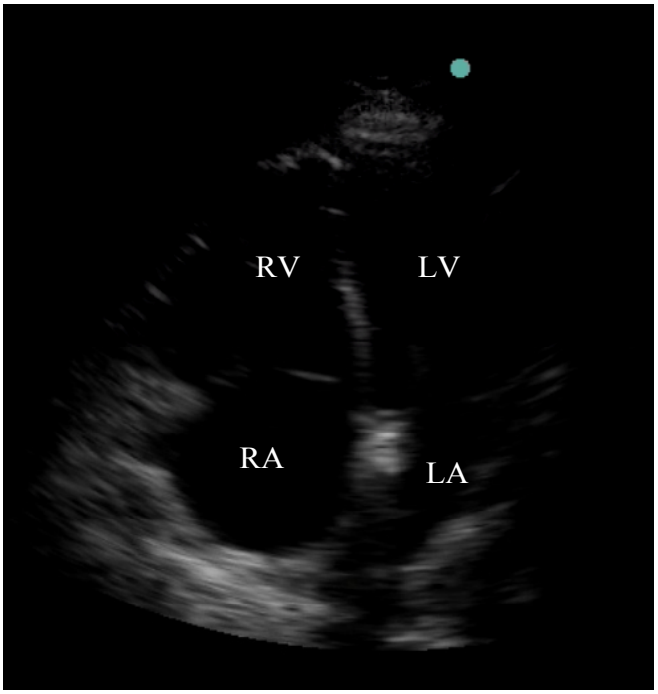

Fig. (2). Moderate RV dilation. Note how the RV and LV sizes are almost similar; the cardiac apex is made up by both the RV and LV; the RA is enlarged; with only minimal experience, the echocardiographer should easily recognize this RV as hypokinetic.

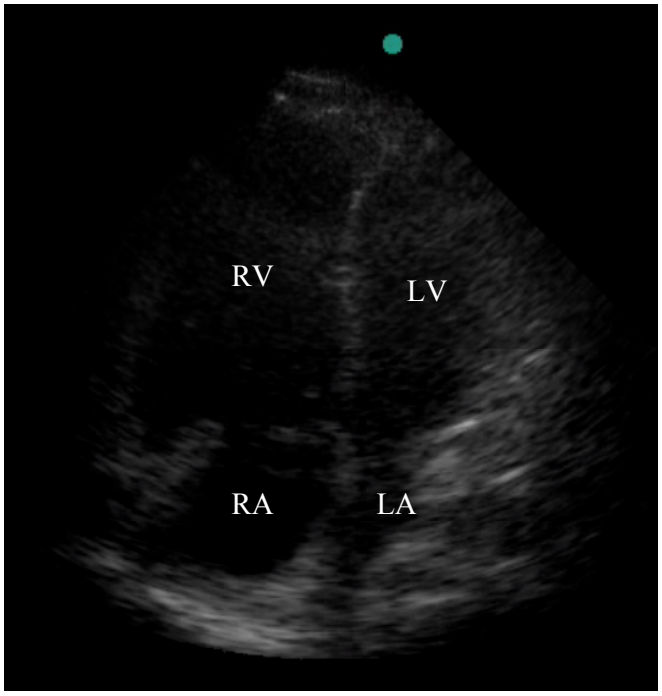

Fig. (3). Severe RV enlargement. The RV makes up the cardiac apex, and its cavity is greater than that of the LV; the RV is grossly hypokinetic; the RA is markedly enlarged as well.

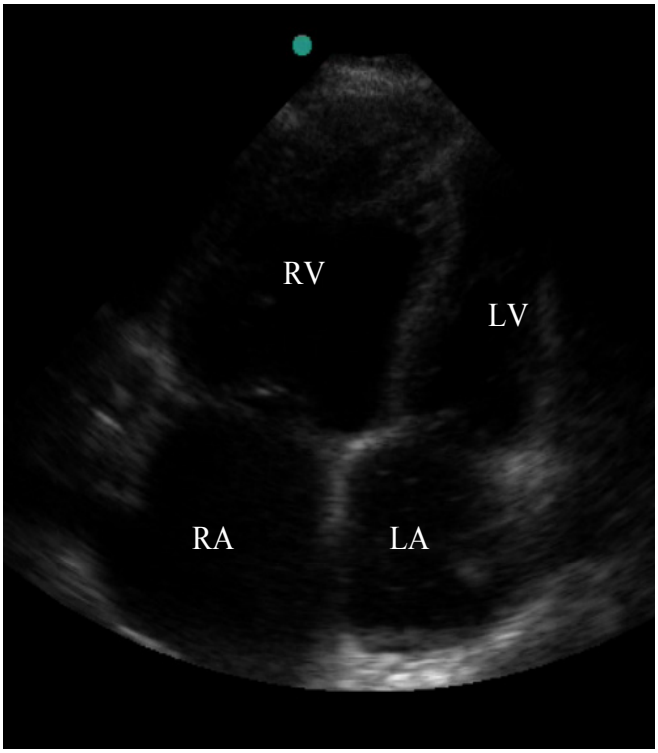

Fig. (4). Extreme RV dilatation. Note severe RV enlargement and hypokinesis; again, the RA is also severely dilated.

An acute rise in the pulmonary pressure may frequently occur in critically ill patients. The causes of a sudden elevation in RV after-load, which defines acute cor pulmonale, include pulmonary embolism (PE), acute respiratory distress syndrome (ARDS), hypoxia in general, positive-pressure ventilation, and acute left heart failure (which could be LV-related or valvular).

In such conditions, during systole the RV faces an increased resistance in the pulmonary vasculature. As a result, the RV contraction time becomes prolonged, and continues past the beginning of the LV diastole. The LV relaxes, and the pressure inside it falls, while the RV pressure remains high. This difference transiently pushes the IVS to the left, into the LV. Such paradoxical septal wall motion can be readily appreciated on echocardiography, and it represents a distinct sign of RV systolic failure. The parasternal short view on transthoracic echocardiography (TTE) is usually utilized for this purpose, where the appearance of the compressed LV at the beginning of its diastole resembles the letter "D" (Fig. **5**, video **1**). Caveat: a common mistake among beginner echocardio-graphers is to obtain a short-axis view at such an angle that the LV appears D-shaped; when in reality it may be normal when scanned appropriately. Therefore, before concluding that the LV is D-shaped, the probe should be manipulated to ensure that it is not merely the scanning angle that is responsible for the apparently abnormal LV shape.

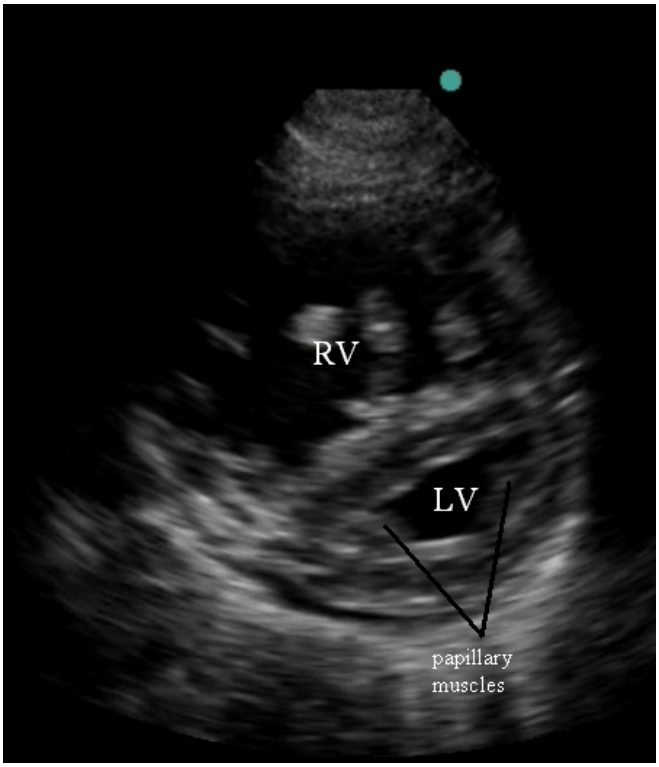

Fig. (5). Echocardiographic sign of RV strain in the parasternal short view. Note the characteristic D-shaped appearance of the LV in cross-section.

In addition to this visual assessment, RV systolic strain can be measured by multiple quantitative methods. One simple method involves measuring LV eccentricity index (EI) (Fig. **9**). First, a short axis view is obtained at the level of LV papillary muscles. Next, two LV diameter measurements are taken at end-systole: diameter one (D1) – perpendicular to the IVS and bisecting it; and diameter two (D2) – the diameter perpendicular to D1. The EI is the ratio of D2/D1, and if it is >1, then RV is considered overloaded in systole [10]. One caveat is that an atrial septal defect could elevate EI. While there are other quantitative methods, these are either impractical for the purpose of quick bedside assessment, or require advanced ultrasound skills.

The pulmonary arterial pressure (PAP) can be measured using the Bernouilli's equation. This requires visualization of tricuspid regurgitation (TR) using Doppler and is considered part of advanced echocardiography:PAP = 4 x [TR jet velocity]^2 + right atrial pressure (RAP). For an inferior vena cava (IVC) size < 2 cm, the RAP may be assumed to be ≤ 10 mmHg.

PRACTICAL IMPLICATIONS

Echocardiographic RV evaluation provides important information in numerous clinical situations encountered by the intensivist on a daily basis. Below are common examples.

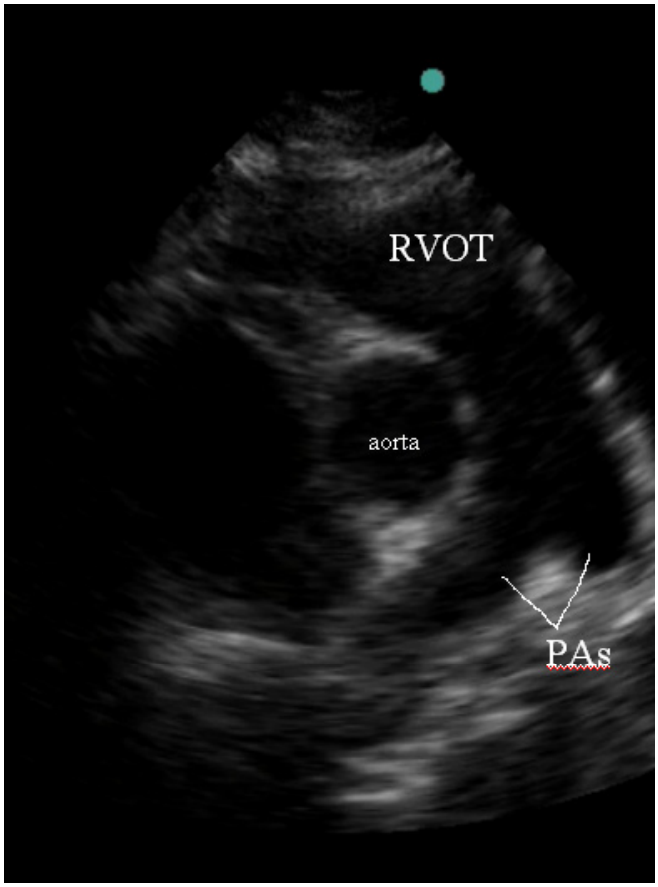

Fig. (6). Modified parasternal short view allowing the visualization of proximal pulmonary arteries; RVOT= RV outflow tract.

ACUTE SEVERE DYSPNEA: NARROWING THE DIFFERENTIAL

We routinely perform limited sonographic examination of the RV, LV, chest, and lower extremities on patients admitted to our ICU with acute respiratory failure. Studies have demonstrated the high accuracy of ultrasonography-aided differentiation between the common causes of acute respiratory failure [13, 14]. When a patient is admitted to ICU for acute dyspnea, the clinician should look for sonographic signs of cardiac tamponade (early diastolic RV collapse), CHF (LV dysfunction, B-lines on lung ultrasound, pleural effusions), pneumothorax (absence of lung sliding; lung point) or pneumonia (consolidation pattern). In the absence of these signs, the presence of a PE becomes a

distinct possibility. A lower extremity ultrasound compression study should then be performed to look for DVT. Occasionally, a saddle embolus may be visualized by TTE at the proximal pulmonary arteries (PAs). To visualize the PAs, the probe is first placed in the parasternal short axis position and then angled up (Fig. **6**). A large hyperechoic substance at the bifurcation of the PAs suggests a saddle embolus (Fig. **7**, video 4). Rarely, a highly mobile thrombus may be visualized in the right heart itself – a rather ominous clinical situation (Fig. **8**, video 5).

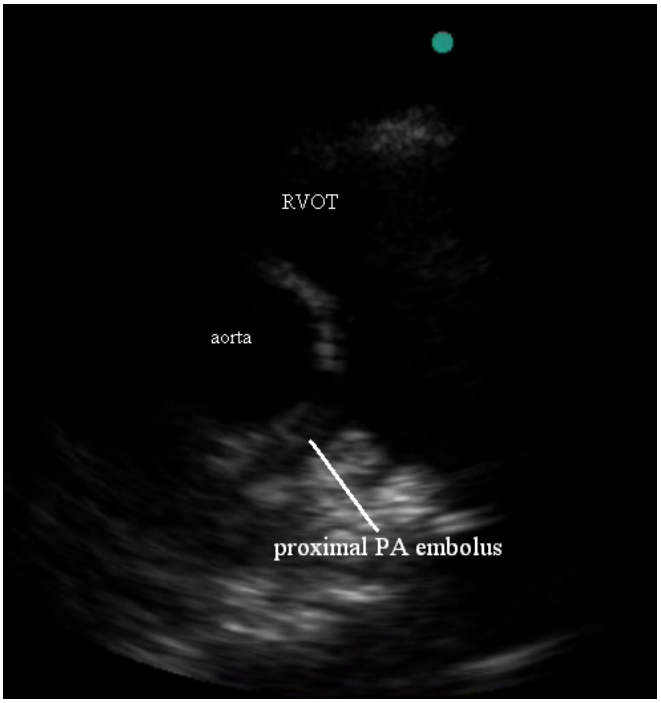

Fig. (7). Modified parasternal short view showing a hyperechoic substance in the region of the proximal PA, consistent with a saddle embolus; RVOT= RV outflow tract.

ACUTE RESPIRATORY DISTRESS SYNDROME: DANGER OF RV VOLUME OVERLOAD

Patients intubated for ARDS have a high propensity to develop cor pulmonale, especially if they are on escalating PEEP. Viellard-Baron *et al*. reported a 25% incidence of cor pulmonale when patients were ventilated with plateau pressure of <30 cm water and a mean PEEP of 6-7 mmHg [15]. If patterns of cor pulmonale are detected on TTE, conservative volume management is indicated, as further fluid loading may worsen the RV strain and ischemia.

SEPSIS: COMMON CAUSE OF RV DYSFUNCTION IN ICU

Sepsis causes global myocardial suppression in both the LV and the RV. The RV dysfunction seems well-tolerated with adequate fluid loading, but once the patient is intubated and positive-pressure ventilation is applied, the increased RV after-load may precipitate cor pulmonale. There is evidence that if central venous pressures rise >10-14 mm Hg, further volume loading may further exacerbate RV dysfunction, leading to a net decrease in cardiac output [16, 17]. In this setting, conservative fluid management is indicated, and agents such as dobutamine [17], which may be superior to norepinephrine in this setting [18], may be of benefit; Levosimendan may be used to enhance RV contractility [19].

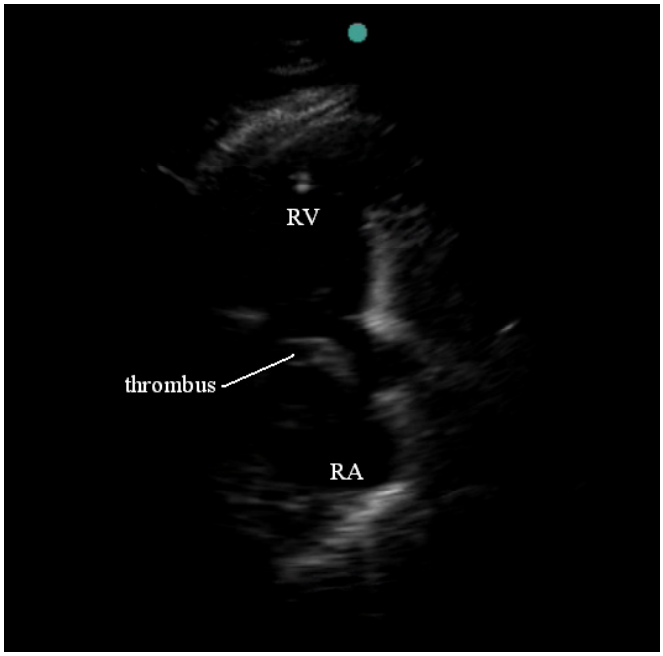

Fig. (8). Apical four-chamber view where a large, highly mobile thrombus is seen going through the RA and RV. Note the dilated RA and hypokinetic RV (LV view is foreshortened here to better visualize the RA and RV).

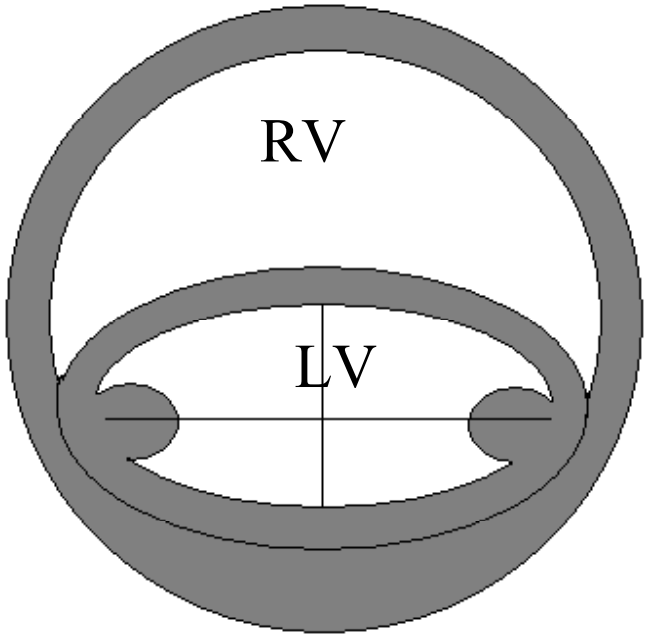

Fig. (9). Simple method involves measuring LV eccentricity index (EI).

SUPPLEMENTARY MATERIAL

This article contain 5 video files and it can be viewed at www.bentham.org/open/toccmj

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