The Value of Bedside Echocardiogram in the Setting of Acute and Chronic Pulmonary Embolism

David W. Lee, MD[a], Kavitha Gopalratnam, MBBS[b], Hubert James Ford III, MD[c], Lisa J. Rose-Jones, MD[a,*]

KEYWORDS

• Echocardiography • Acute pulmonary embolism • Chronic pulmonary embolism • Right ventricle • Right heart failure • Pulmonary hypertension

KEY POINTS

- Echocardiography can be valuable in the evaluation and risk stratification of patients with acute and chronic pulmonary embolism (PE).
- Patients with acute PE who have echocardiographic evidence of right ventricular dilatation and/or right ventricular dysfunction have an increased risk of mortality.
- A minority of patients with acute PE can develop chronic thromboembolic pulmonary hypertension.
- Patients with chronic thromboembolic pulmonary hypertension often have echocardiographic evidence of elevated pulmonary arterial pressures, right ventricular hypertrophy, right ventricular dysfunction, and/or left ventricular impaired relaxation.

INTRODUCTION

Acute pulmonary embolism (PE) is associated with an estimated mortality rate of up to 11% at 2 weeks and 17% at 3 months.[1] In a large international registry of patients with acute PE, those with hemodynamic instability had an estimated 3-month mortality rate of 58%.[1] In a smaller prospective cohort, patients with normal systemic blood pressure and echocardiographic evidence of right ventricular (RV) dysfunction had an estimated 5% in-hospital mortality rate.[2] Because of the high stakes, patients with suspected acute PE require timely diagnosis and risk stratification to guide treatment. Although computed tomography pulmonary angiography remains the preferred choice of imaging for the diagnosis of PE (see Farbod N. Rahaghi and colleagues' article, "Diagnosis of DVTs and PE's – New Imaging Tools & Modalities," in this issue), bedside echocardiography (echo) guides risk stratification of patients with acute PE by providing valuable prognostic information regarding right heart structure, function, and hemodynamics. Although echo has not been associated with reduced mortality in acute hemodynamically stable PE,[3] the application of echocardiographic data to risk stratify patients with acute and chronic PE can significantly aid the clinician in determining the best therapeutic approach.

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[a] Division of Cardiology, University of North Carolina, 160 Dental Circle, CB #7075, Chapel Hill, NC 27599, USA; [b] Division of Pulmonology, Yale New Haven Health, Bridgeport Hospital, 267 Grant Street, Bridgeport, CT 06610, USA; [c] Division of Pulmonary and Critical Care Medicine, University of North Carolina, 130 Mason Farm Road, CB #7020, Chapel Hill, NC 27599, USA
* Corresponding author.
E-mail address: Lisa_Rose-jones@med.unc.edu

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THE RIGHT VENTRICLE AND HEMODYNAMICS IN ACUTE PULMONARY EMBOLISM

There are key anatomic differences between the left ventricle (LV) and the RV.[4,5] Compared with the LV, the RV is composed of a thinner wall. The RV muscle fibers are arranged in series, creating a crescent-shaped cavity. By contrast, the LV muscle fibers are arranged in parallel, creating a concentric shaped cavity. This anatomic difference allows for the RV to have better compliance without significantly increasing pressures. Finally, the RV wall is composed of superficial circumferential muscle fibers and inner longitudinal muscle fibers. Although the circumferential fibers are responsible for inward contraction, the longitudinal fibers provide significant base-to-apex excursion. Given this unique orchestration of muscle fibers, the echocardiographic assessment of RV function can be challenging.

From a hemodynamic standpoint, both the RV and the pulmonary arterial circulation comprise a high-capacitance, low-resistance system. In acute PE, thromboembolic occlusion of the pulmonary artery (PA) leads to a sudden increase in vascular resistance, and thus RV afterload, for which the RV cannot readily compensate. When a certain threshold is reached with increased PA occlusion due to clot burden, there is decreased LV preload, which then leads to a drop in LV stroke volume and subsequent systemic hypotension. In addition, the now dilated, high-pressure RV has interventricular septal shift that encroaches on and compresses the LV. This reverse Bernheim effect further decreases LV filling and causes a further drop in LV stroke volume[6] (**Fig. 1**).

The normal PA systolic pressure (PASP) ranges between 15 and 30 mm Hg. In acute PE, depending on the severity, the PASP is typically elevated. If there is no significant gradient across the RV outflow tract, pulmonary valve, or proximal pulmonary arteries, then the PASP is equivalent to the RV systolic pressure (RVSP). The simplified Bernoulli equation using the tricuspid regurgitation (TR) jet velocity on echo can be used to estimate the RVSP.[7]

$$\text{RVSP} = 4 \times (\text{peak TR jet velocity})^2 + \text{mean RA pressure}$$

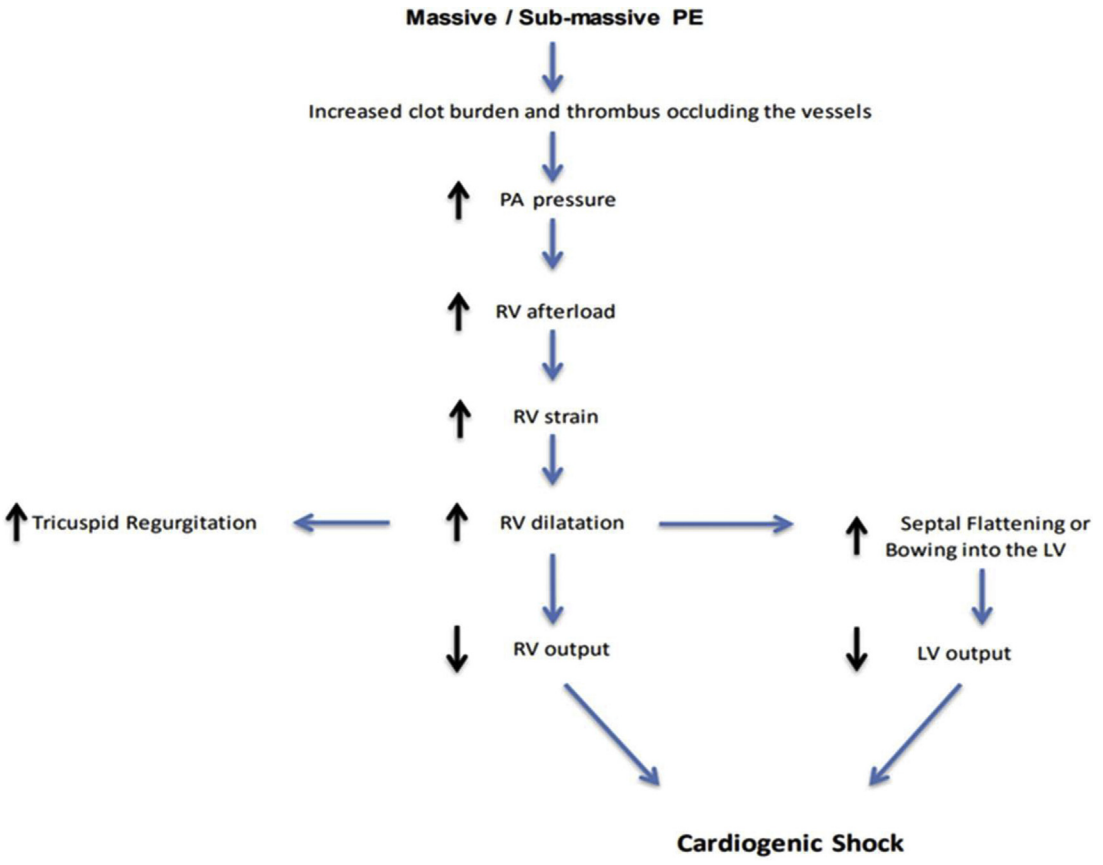

Fig. 1. Pathogenesis of RV failure leading to shock secondary to massive/submassive pulmonary embolism.