

## Emergency bedside ultrasound diagnosis of sub-massive acute pulmonary embolism: a case of the McConnell sign

Michael M. Liao · Jonathan Theoret ·  
Elisa M. Dannemiller · Catherine Erickson ·  
Geoffrey E. Sanz · John L. Kendall

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### Abstract

**Introduction** This is a case of a healthy 61-year-old man with acute onset of dyspnea and atrial flutter where bedside emergency ultrasound was used to identify a classic echocardiographic finding called the “McConnell sign”. The clinical presentation and this echocardiographic finding led to the presumptive diagnosis of acute pulmonary embolism.

**Materials and methods** This is a case report and brief review of the literature.

**Conclusion** Bedside echocardiography has important diagnostic value in the evaluation of suspected acute pulmonary embolism. Findings, such as the McConnell sign are relatively quick and easy to identify at the bedside and could provide valuable information to rapidly guide management decisions when further research defines its role in emergent bedside ultrasound.

**Keywords** Emergency ultrasound · McConnell sign · Bedside · Pulmonary embolism · Diagnosis · Echocardiography

### Case report

A 61-year-old athletic man presented to the emergency department (ED) by ambulance after becoming suddenly dyspneic. The patient was resting after completing a training session when he suddenly developed trouble breathing. His room air oxygen saturation was 98%, his heart rate 150, respiratory rate 55, blood pressure 155/68 and he was afebrile. The cardiac monitor showed new atrial flutter confirmed by electrocardiogram (ECG). Further history revealed that he had no risk factors for venous thromboembolic disease. A bedside echocardiogram (Supplementary Video 1; Fig. 1) was performed by the ED team and revealed a dilated right ventricle with right ventricular (RV) mid-free wall akinesis with normal RV apical contractility, a classic echocardiographic finding called “McConnell sign [1]”. Based on the clinical presentation and the bedside ultrasound findings, the decision was made to empirically anticoagulate for presumed acute pulmonary embolism. The patient was admitted to the medical intensive care unit. Secondary to renal insufficiency, a CT pulmonary angiogram (Fig. 2) was performed 2 days later showing large bilateral left main and right main pulmonary artery emboli, nearly occlusive and extending into almost all segmental arteries. A temporary inferior vena cava filter was placed in hospital, chronic warfarin was initiated, and his atrial flutter was rhythm and rate controlled with amiodarone and diltiazem, respectively. He was discharged home 9 days later without further events.

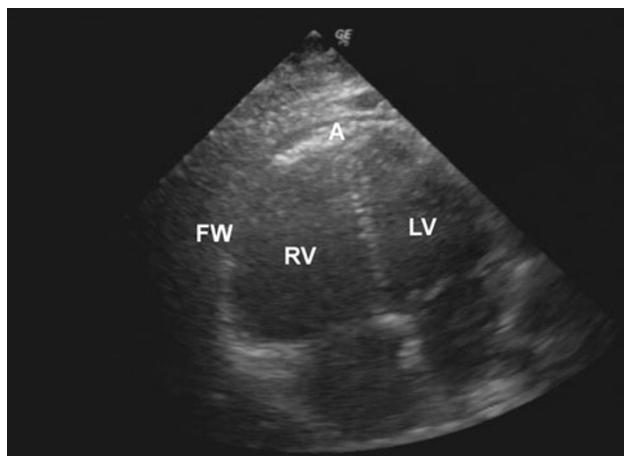
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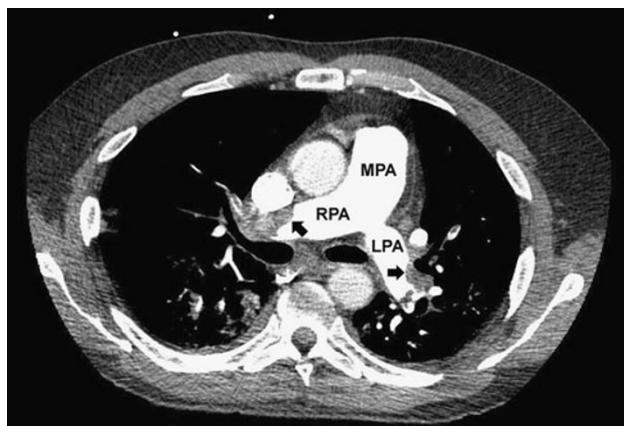
M. M. Liao · J. Theoret · E. M. Dannemiller · C. Erickson ·  
G. E. Sanz · J. L. Kendall (✉)  
Department of Emergency Medicine, Denver Health Medical  
Center, 777 Bannock Street, Mail Code 0108,  
Denver, CO 80204, USA  
e-mail: john.kendall@dhha.org

J. Theoret  
Department of Emergency Medicine,  
University of Alberta, Edmonton, AB, Canada

G. E. Sanz  
Department of Emergency Medicine, Queen's University,  
Kingston, ON, Canada



**Fig. 1** Apical 4-chamber view of the heart. *RV* right ventricle (dilated), *LV* left ventricle, *A* apex (normal contraction), *FW* free wall of right ventricle (hypokinetic)



**Fig. 2** Contrast-enhanced CT pulmonary angiogram. *MPA* main pulmonary artery, *RPA* right main pulmonary artery, *LPA* left main pulmonary artery, arrows pulmonary emboli

## Discussion

Acute pulmonary embolism (APE) is a common and devastating disorder in the ED population, with a substantially increased risk of morbidity and mortality if missed or untreated in the ED [2]. Bedside echocardiography plays many important roles in the workup and management of APE, including diagnosis, prognosis, and guidance of aggressive therapy. Echocardiographic findings of APE are often indirect, unless a rare intracardiac thrombus is identified. The commonly identified findings include RV dysfunction, RV dilation, tricuspid regurgitation, and leftward septal displacement [3].

The diagnostic utility of echocardiography in an ED patient depends on whether the suspected APE is massive (i.e., associated with hypotension or shock) or sub-massive.

In the situation of suspected massive APE, echocardiography has a vital role. The European Society of Cardiology (ESC) Task Force for APE published guidelines in 2008 stating that “the absence of echocardiographic signs of RV overload or dysfunction practically excludes PE as a cause of hemodynamic compromise [4]”. Conversely, the presence of RV dysfunction on echocardiography could be used to presume massive APE and justify aggressive reperfusion therapy [4]. In addition, echocardiography could evaluate for other etiologies of hypotension, including cardiac tamponade, cardiogenic shock, or hypovolemia [5].

For the suspected sub-massive APE, as was the case in our patient, the diagnostic utility of echocardiography remains unclear. The ESC Task Force suggests that for patients with moderate or high pre-test probability of APE, a positive echocardiogram for RV dysfunction could be considered diagnostic [4]. However, a recent meta-analysis that pooled results from two prospective observational studies came to the conclusion that only patients with a high pre-test probability of APE could be accurately diagnosed with echocardiography [6]. Unfortunately evidence remains limited because of small sample sizes and heterogeneity of echocardiogram criteria for APE.

The McConnell sign was first described in 1996 as a distinct regional pattern of RV dysfunction (i.e., regional akinesis of the RV mid-free wall, but normal wall motion at the RV apex) that was more specific than simply RV dysfunction for APE [1]. Echocardiographic evidence of RV dysfunction is relatively common in APE, found in 40–70% of cases [7]. However, it is a relatively nonspecific finding, found also in cardiomyopathy, pulmonary hypertension, valvular heart disease, and RV infarction. McConnell and colleagues found this pattern to have a sensitivity of 77% (95% CI 46–94%) and a specificity of 94% (95% CI 86–98%). However, their study was small, consisting of 85 hospitalized patients with known RV dysfunction from any cause, of which 13 had acute pulmonary embolism [1].

Two subsequent studies have looked at the validity of McConnell sign for diagnosis of APE. In 2002, Kurzyna et al. prospectively evaluated 100 consecutive patients with suspected APE and found that the McConnell sign had a sensitivity of 19% (95% CI 11–31%) and a specificity of 100% (95% CI 87–100%) [8]. It should be noted that enrollment was at a tertiary hospital for referred pulmonary disease and only eight patients were enrolled directly from the ED. In 2005, Casazza et al. noted that the McConnell sign was found in 67% of patients retrospectively identified to have RV infarction, thus warning that McConnell sign lacks specificity in these patients [9]. However, this would suggest that patients with confirmed McConnell sign should also have an ECG with right sided leads to differentiate RV infarct from APE.

The McConnell sign to our knowledge has not been reported in the ED setting. Interestingly, Bova et al. suggest that using echocardiography alone to assess for RV dilation and/or Doppler evidence of pulmonary hypertension could reduce additional imaging workup by 12–28%, however will incorrectly treat 4–14% [10]. Perhaps, adding the McConnell sign to this suggested pathway could further reduce additional imaging or reduce false positives.

In summary, we present a case where bedside echocardiographic evidence of the McConnell sign led to a presumptive diagnosis of APE which was later confirmed on CT pulmonary angiogram. The diagnostic utility of echocardiography in patients suspected to have APE depends largely on the clinical presentation. In the hypotensive and unstable patient, it has tremendous diagnostic value. In the normotensive patient with suspected APE, its diagnostic value is less well established, but is likely dependent on the pre-test probability and useful mainly with a positive echocardiographic finding. Specific echocardiographic findings such as the McConnell sign are relatively quick and easy to identify at the bedside and thus could provide valuable information to rapidly guide management decisions if further research defines its role in emergent bedside ultrasound.

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**Conflict of interest** None.

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