Echo in PE

Key Points:
- Acute PE cannot explain a PASP > 50 mm Hg
- Most patients with an acute PE will have some echocardiographic abnormality of the right ventricle
- Positive troponin in acute PE seems to be correlated with RV dysfunction and increased mortality

1. **What degree of pulmonary hypertension can be explained by an acute PE?**
   - Obstruction of > 50% of the pulmonary vascular bed is required to see significant elevation of the PA pressures
   - The normal RV is unable to generate PASP > 50 mm (mean 40) acutely

2. **Aside from pulmonary HTN, what is the data about the utility of echocardiograms in acute PE?**
   - More than 80% of patients with a documented PE have some type of right heart abnormality on echo, most commonly one of the following:
     - Direct visualization of thrombus (rare)
     - RV dilatation
     - RV hypokinesis with apical sparing (a.k.a. McConnell’s sign)
     - Abnormal interventricular septal motion (a.k.a. “septal bowing”)
     - TR
     - PA dilatation
     - Lack of inspiratory collapse of IVC
   - The presence of any 2 of the 3 following criteria has a 56% sensitivity and 90% specificity, in one study, for acute PE (confirmed by P-Agram):
     - RV hypokinesis
     - RVEDD > 27 mm (without RV hypertrophy)
     - TR velocity of > 2.7 m/sec
   - In the same study, using pre-test probabilities of PE of 10%, 50%, and 90%, the post-test probabilities with a positive echo were 38%, 85%, and 98%. With a negative echo, post-test probabilities were 5%, 33%, and 81%.

3. **Please tell us about the latest data on troponin in PE!**
   - A prospective study of 106 patients with acute PE found that troponinI is elevated in 41% and was correlated with echocardiographically detected RV dysfunction, “complicated in-hospital course,” and overall mortality. The higher the troponin, the higher the mortality.
   - Negative predictive value of troponins for a major clinical event was 93%

References