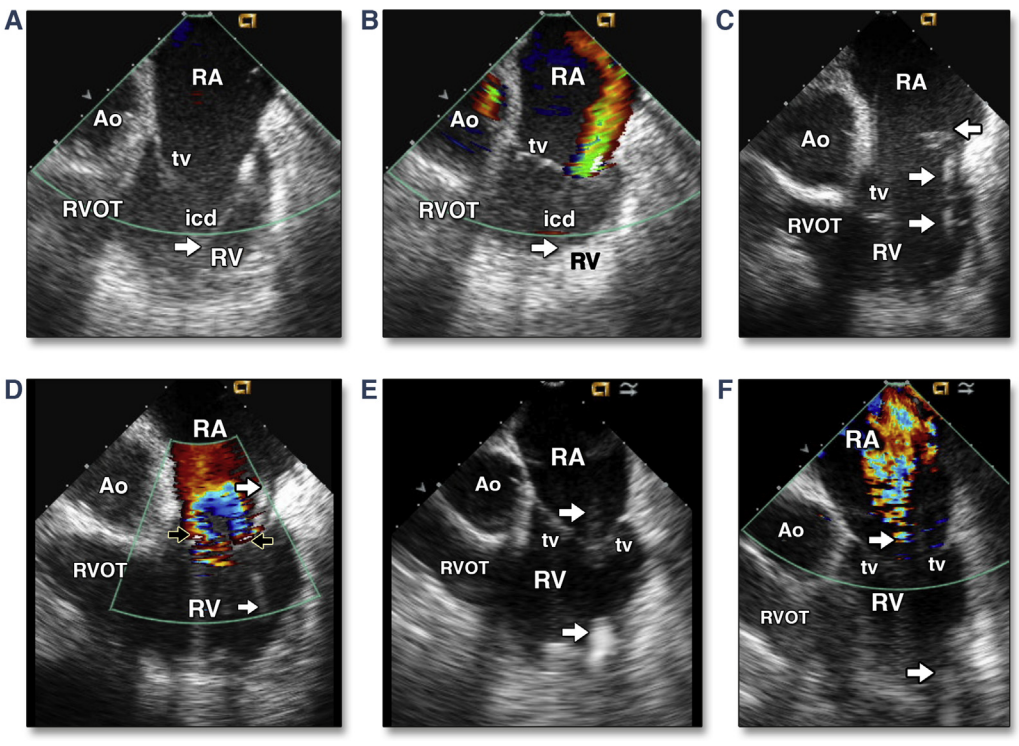


**FIGURE 1 Intracardiac Echocardiographic Images of Intracardiac Device Lead and Tricuspid Regurgitant Severity With Different Etiologies**

Intracardiac echocardiographic imaging with the transducer placed in the right atrium in 3 patients. The first patient had severe left ventricular (LV) dysfunction (left ventricular ejection fraction [LVEF] = 20%), normal right ventricular (RV) size, and pulmonary artery systolic pressure (PASP) estimated at 55 mm Hg, an implantable cardioverter defibrillator (ICD) lead **(arrow)** placed at the RV anterolateral wall with impingement of the posterior leaflet **(A)**, and moderately eccentric tricuspid regurgitation (TR) with color Doppler imaging (CDI) **(B)**. The second patient had moderate LV dysfunction (LVEF = 35%); enlarged RV and tricuspid annulus; PASP of 51 mm Hg; an ICD lead **(rightward arrows)** placed at the RV apex; multiple small thrombi **(leftward arrow)** attached at the lead **(C)**; and severely centralized TR with CDI, which shows that the lead **(arrows)** is slightly impinging the posterior leaflet **(D)**. The third patient had arrhythmogenic RV cardiomyopathy, enlarged RV and tricuspid annulus, PASP of 33 mm Hg, LVEF = 30%, a centralized location of ICD lead **(arrows)** placed at the RV apex **(E)**, and moderately to severely centralized TR with CDI **(F)**. Ao = aortic root; RA = right atrium; RVOT = RV outflow tract; tv = tricuspid valve.

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## REPLY: Tricuspid Regurgitation Severity Associated With Positioning of RV Lead or Other Etiology Assessed by Intracardiac Echocardiography

We would like to thank Dr. Ren and colleagues for their interest and feedback and appreciate the opportunity to reply. The main limitation of our study (1) is that it is retrospective; therefore, assumptions of cause and effect regarding tricuspid regurgitation (TR) are

impossible to make. As a "proof of concept," we used transthoracic echocardiography to evaluate the feasibility of visualizing tricuspid valve leaflet motion and device location in 3 dimensions (3D) as well as the degree of TR. In our single-center study, 3D evaluation of lead location was feasible in 90% of studies and an association did exist between lead position and degree of TR (1).

As opposed to mitral regurgitation, TR lacks standardized recommendations for severity and data for etiology (2). For TR severity assessment, a comprehensive "semiquantitative" approach is recommended due to the lack of standardized reference values (2). Three-dimensional effective regurgitant orifice area and regurgitant fraction may provide more precise volumes; however, they are more difficult to reliably obtain and lack reference values.

Tricuspid valve regurgitation, regardless of the etiology, is associated with high morbidity and mortality. In a study by Nath et al. (3), the investigators showed that significant TR was associated with worse outcomes. In their study (n = 5,223), the severity of TR correlated with worse outcomes, independent of right ventricular size, left ventricular function, or pulmonary artery systolic pressures (3). Furthermore, Lin et al. (4) described device-associated TR requiring cardiac surgery as well as several mechanisms leading to valve malfunction, including adherence, impingement, perforation, or entanglement (4).

Ren et al. have experience with intracardiac echocardiography and should be applauded for their efforts using this imaging modality in clinical practice. This imaging modality may be useful to guide lead placement in the future; however, routine intracardiac echocardiography to guide device placement is not the standard of care and should be considered investigational.

We proposed 3D echocardiographic guidance as a possible way to limit device lead-associated TR; however, several questions need to be answered prior to considering a prospective clinical trial to investigate this issue:

1. Do device leads remain in the same location after insertion?
2. Is guidance of lead location possible? If so, what imaging modality would best guide lead placement (3D transthoracic echocardiography, 2D/3D transesophageal echocardiography, or intracardiac echocardiography)?

Once these questions are answered with randomized, prospective studies, the clinical utility of lead placement under imaging guidance could be determined.

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## Imaging Findings in Aortic Intramural Hematoma

I greatly enjoyed reading the comprehensive iReviews article from Baliga et al. (1). The authors have provided a detailed summary of the topic with compelling illustrations and multidetector computed tomography images. However, I feel it is necessary to point out an error in the description of Figure 8 that could be misleading to readers. The images in question are described as depicting an intramural hematoma; however, it is rather showing an example of an aortic mural thrombus. Intramural hematoma can be recognized by its location within the aortic media, deep to the intima, well-illustrated by Figure 2 (1) of the review. As noted in the text, hematoma can be localized to this area by identification intimal calcifications that are displaced by the thickened wall. In the provided Figure 8 (1), there is no displaced intimal calcification, but rather, the area labeled as hematoma is located within the vessel calcifications, clearly depicted on the noncontrast image. This confirms the diagnosis of mural thrombus rather than intramural hematoma. Furthermore, acute intramural hematoma will be highly attenuated on noncontrast computed tomography (2), which is not the case in Figure 8 (1) of