Commentary: Tricuspid valve ring characteristics: Physiologically important, clinically relevant, or too little too late?

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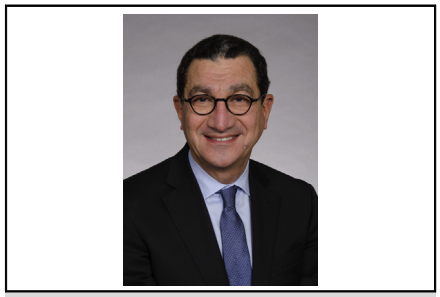

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Central Message

Functional tricuspid regurgitation is currently diagnosed and treated late, frequently beyond the point of meaningful physiological remodeling. Future research aims to evolve diagnosis and treatment.

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Functional (secondary) tricuspid valve regurgitation (FTR) is the most common (>85%) clinical presentation of significant tricuspid regurgitation (TR) but is a poorly characterized and understood set of overlapping conditions. Precise quantification and correct anatomic and physiologic diagnoses leading to more systematic, durable, and effective approaches remain elusive.[1-3] FTR is a covariant with many known comorbid conditions (such as older age, lower ejection fraction, right ventricular [RV] dysfunction and dilatation, pulmonary hypertension, mitral regurgitation, left ventricular dysfunction, liver and renal dysfunction, and atrial enlargement and fibrillation) but is independently associated with a nearly 2-fold increase in mortality.[4,5] TR severity has to be assessed in conjunction with tricuspid valve (TV) annular size, TV morphology (leaflet tethering, chordal abnormalities, etc), RV size, function,and pulmonary vascular resistance.[2] Treating concomitant TV dilatation and TR at the time of mitral valve surgery has been shown to be effective, is associated with minimal added morbidity and mortality,[6-8] and currently meets class I and IIa indications for therapy in both American and European guidelines. Current therapies rely on TV annuloplasty devices with varied characteristics such as size, shape, form (planar and non-planar), and rigidity. There is also an increased recognition that in addition to annular therapies, severe leaflet tethering, when present, may also need to be addressed with additional interventions (such as leaflet augmentation, neochords, or subannular papillary muscle therapies). Because of historically high mortality for isolated FTR interventions, there remains significant uncertainties as to appropriate patient selection, timing, scope of repair, with yet-to-be-determined effectiveness, and durability of such approaches. This condition, understandably, remains undertreated.[9]

This recent publication by Malinowski and colleagues[10] represents another facet of their ongoing efforts to better assess and define TV anatomy, function, and acute pathology.[11] In this specific publication, the impact of different annuloplasty ring characteristics on TV function was assessed with a stated goal of better device design in the hope of promoting TV repair durability. Using a known model of ovine, acute, normal (non-dilated) hearts and sonomicrometry and pressure transducers, the authors were able to demonstrate that all 3 annuloplasty rings reduce the diastolic TV annular area (by nearly one-half). In comparison with flexible Duran and Tri-Ad rings, the Contour rigid non-planar ring was better at maintaining the 3-dimensional annular (ellipsoid) saddle-shape, reducing the magnitude of cardiac strain RV wall stress and remodeling, and increasing leaflet coaptation but doing so at the expense of reducing annular ''physiological'' contraction. Maintaining annular shape and height may be associated with decreased leaflet strain and stress and enhanced repair durability but at the potential risk of a greater incidence of ring dehiscence. The authors fully acknowledge that clinical (chronic) TR significantly impacts TV annular contraction, dilation, flattening, and strain. Chronic pathologic changes limit extrapolation of these findings to common and varied clinical scenarios. It is difficult to draw conclusions of

whether these varied ring characteristics will differentially impact RV stress or longer-term outcomes in different temporal phases, severity, and anatomic presentations of clinically relevant TR.

It is evident that our understandings of TV physiology and pathology are currently still limited. Most significantly, FTR is currently diagnosed late, frequently beyond the point of meaningful physiological remodeling of associated profound accompanying RV or right atrial pathology. It is unclear whether current therapies (both open surgical and emerging catheter-based) are sufficiently tailored and complete, or are simply too little too late, to effectively impact long-term survival. We hope that future research will lead to earlier, more nuanced pathophysiologic staging and diagnoses and also lead to more timely, targeted, and effective therapies.

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