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Tricuspid Regurgitation, Isn't It Time to Look Around the Valve Rather Than the Valve Itself?

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It was not until a couple of decades ago that the tricuspid valve has received attention to clinicians. Based on anecdotal experience in patients with Fontan physiology[1] and also in a case of a patient without tricuspid valve because of endocarditis,[2] clinicians have had a misconception that the tricuspid valve may be a 'rudimentary' or even an 'unnecessary' valve in the heart.

Nowadays these misconceptions have been disputed by firm clinical observations that the severity of the tricuspid regurgitation (TR) is an independent predictor of mortality.[3] Although the event rates are still high with surgical correction of severe TR,[4][5] it is equally well-known that TR is a correctable disease and provided that patients are carefully selected,[5] surgical correction may be a life-saving procedure. Furthermore, the rate of significant early morbidity and mortality after surgical correction has dropped to as less as 2% in certain experienced centers.[6] Therefore, the next legitimate question regarding TR is what factors are associated with the development of TR and consequently, which patients need intensive surveillance.

It has been shown in previous papers that the morphology and certain changes in the valve itself leads to significant TR.[7] Although previous papers have looked into the clinical significance of TR,[8] only a few articles have touched on the impact of the surrounding structures/chambers on the development of TR.[9] This is important because a significant majority of TR is functional TR. Furthermore, the majority of papers have only focused on TR secondary to significant concomitant left-sided valve disease.[10][11] The interesting article by Park et al.[12] have brought to us an interesting suggestion to this important and yet, unresolved question. Although it is well known that atrial fibrillation is an important contributor to the development of significant TR,[10][11] geometric or functional parameters that begets TR has not been investigated enough in previous papers, especially in isolated atrial fibrillation patients such as that in the paper in this issue of the Journal.

What the authors have found in their 89 atrial fibrillation patients with a variety of TR degree is that persistent atrial fibrillation, tricuspid annular diameter and the tenting height of the tricuspid valve are the significant determinants of significant TR. Perhaps a more interesting finding was that structures that surround the tricuspid valve, that is the right atrium (RA) and the right ventricle (RV), are significantly associated with the severity of TR.

Of course, the dilatation of the RA and RV may be a consequence of the TR itself. Many physicians have conceptually thought that the enlargement of the tricuspid annular diameter would be the most important factor of TR development. Likewise, it is not difficult to imagine that functional TR may be easier to develop than functional MR because in contrast to the fibrous skeleton that wraps around the mitral annulus, there is no equivalent structure that is located around the tricuspid annulus. However, the authors have nicely shown that the conformation change of the RV, as represented by the RV spherical index, is also associated with the development of significant TR. Therefore, the authors provide a good argument that TR may beget TR by affecting the overall conformation/shape of the right-sided chamber and not just the annulus itself, a finding that has been suggested in another previous paper.[9] The concept that the authors suggest are also partially supported by a previous *in vitro* study that demonstrates papillary displacement alone may also cause functional TR.[13] Therefore, the right-sided atrioventricular valve may be more susceptible by the changes in the chambers that are directly connected to it.

Then what is the remaining step that is left to investigate? Certainly, it remains to be proved whether there is a causality relationship between the conformational change of the right

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side chambers and significant TR. As the authors have already acknowledged, a prospective and consecutive study would be justified to accurately describe and confirm the above findings. Likewise, whether certain measures to reduce the 'contributors' would lead to the amelioration of TR should also be saught in further studies. Clinicians would also greatly benefit from development of a reliable parameter of RV function considering the indeterminate shape of the RV and RA. It is time to look 'around' the dysfunctional valve rather than to look 'at' the valve!

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