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Letter to the Editor

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Time to change approach – from morphology to function and pathophysiology: The lesson of postoperative tricuspid regurgitation

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Worsening of tricuspid regurgitation after mitral valve surgery represents a big issue in cardiac rehabilitation.

The indications for surgical repair of the tricuspid valve (TV) present, to date, some aspects of uncertainty. This is especially true when making a decision on tricuspid annuloplasty at the time of mitral valve surgery. It is well known that patients with severe functional (secondary) tricuspid valve regurgitation (TVR) treated surgically at the time of mitral valve repair may have a better long-term prognosis compared to untreated subjects. Even in the absence of significant TVR, annular dilation (>40 mm or 21 mm/m^2) is another indication for tricuspid annuloplasty, although less strong in terms of scientific evidence.[1] To date, however, satisfactory answers are lacking on at least three fundamental issues: (1) what are the effective prognostic determinants (both clinical and instrumental, e.g. annular size) associated with worsening TVR in patients undergoing surgery of the mitral valve; (2) is the progression of TVR similar in subjects with primary and secondary forms (i.e. degenerative vs. ischaemic) of mitral insufficiency? (3) does the surgical choice (mitral repair vs. replacement) influence the progression of TVR?

A recent article by Sordelli et al.[2] offers a contribution to our knowledge on this topic, assessing prospectively the predictive role of TV annulus size on TVR progression in 706 subjects operated for degenerative mitral regurgitation. The measurements were conducted by a three-dimensional technique, enabling measurement of the TV annular antero-posterior diameter, wider and most closely related to the surgical measurement, compared to the septal-lateral diameter.[3] This work shows some surprising results. First, it does not show any prognostic correlation between the TV annulus size and worsening of TVR, which rather seems related to the recurrence of mitral regurgitation and worsening of pulmonary hypertension. This link was also reported by Gatti et al.,[4] who showed a higher (although not significant) freedom from recurrent TVR after mitral valve replacement than after repair. This additional finding may represent an answer to our third question, stressing the need for durable mitral valve repairs. Functional TVR, long a neglected entity, is a progressive disease, which may worsen over time, and surgery should not be delayed to avoid irreversible pulmonary hypertension and right ventricular (RV) dysfunction. We believe that the results reported by Sordelli et al. could possibly contribute to a critical review of the current recommendation for tricuspid surgery. Their observations provide some clues on the first of the three questions we have just proposed, although the full spectrum of this anatomo-functional dilemma remains inconclusive. Thus there is a need for further research on the prognostic determinants of the progression of functional parameters, beyond the morphological data.

Tricuspid annular dilation is actually related not just to RV enlargement, but right atrial dilation/remodelling and dysfunction (i.e. exhausted reservoir and booster) may also play a big role. In this context permanent atrial fibrillation has been shown to be an independent and powerful risk factor for TVR progression. This underscores the non-benign nature of this arrhythmia, responsible through a vicious circle for right-sided heart failure, becoming addictive to ventriculo-arterial miscoupling in impairing the global efficiency of right heart chambers.

Actually, the simple preload lowering in the right heart can lead to a significant reduction in secondary TVR, underlying the dynamic nature of right atrioventricular interaction. And the right ventricle is no longer an innocent bystander in left heart disease; the left and right ventricles are highly interdependent,[5] and 30–50% of RV contractile force is generated by left ventricular contraction.[6] Moreover, although less

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documented, it is known that inter and intra-ventricular dyssynchrony, so common in post-surgical patients, may contribute to the development and progression of functional TVR. It is also recognised that implantable cardioverter defibrillator and cardiac resynchronisation therapy wires, common in these patients, may increase TVR by several mechanisms: perforation of valve leaflets, interference with papillary muscle function, lead entrapment or their adhesion to the valve tip due to fibrous tissue. All these mechanisms primarily affect leaflet coaptation, resulting in a more complex and challenging surgical valve repair (e.g. useless ring annuloplasty).

When dealing with RV function, the importance of contractile reserve has recently been disclosed, but it may become apparent only during exercise echocardiography.[7]

Actually, the limitations of tricuspid annular plane systolic excursion (TAPSE) are well recognised: preload-dependent measure, poor accuracy after cardiac surgery. Furthermore, indirect measurements of PASP through tricuspid regurgitation jet velocity may be unreliable in patients with severe TVR. However, the ratio between TAPSE (longitudinal RV fibre shortening) and pulmonary artery systolic pressure (PASP) (developed pressure), used as an in-vivo expression of the length/force relationship, may better disclose prognosis compared with either variable in isolation, during the follow-up of postoperative TVR. Prospective studies are needed to clarify its real utility in this subset of patients.

This comprehensive approach to the right ventricle–pulmonary circulation unit as a whole[8] underscores the importance of an integrated use of different techniques, combining imaging and function, such as with stress echocardiography and the cardiopulmonary exercise test, opening a new window in the study of several conditions.[9] The integration of the length to force relationship (e.g. TAPSE/PASP ratio) to cardiopulmonary exercise test data (e.g. minute ventilation/carbon dioxide production slope) reveals different pathophysiological phenotypes, and testifies to the opportunity of a complete heart–lung assessment in these patients.[10]

We would like to point out the need for a more extensive reflection on the dynamic characteristics of TVR physiology. In this regard, an integration of multiple parameters in future prospective studies could possibly elucidate this complex matter.

Author contribution

GS and SM contributed to the conception of the work, both drafted the manuscript and critically revised it. GS and SM gave final approval and agree to be accountable for all aspects of work ensuring integrity and accuracy.

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