## HIT initiative: discussion with the seniors

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Topic of discussion: Assessment of functional tricuspid regurgitation with echocardiography

(1) Which are the main limitations of the conventional approach for evaluation of functional tricuspid regurgitation (FTR) based on 2D and Doppler echocardiography?

So far, we have approached the patient with functional tricuspid regurgitation by transferring the concepts and echocardiographic parameters developed for patients with mitral regurgitation. Even the cut-off values of the effective regurgitant orifice area (EROA) for severe tricuspid regurgitation are the same (40 mm<sup>2</sup>) without accounting for the different anatomy of the two valve apparati and the peculiar haemodynamic environment in which the tricuspid valve is working. The tricuspid valve is trileaflet, and the orifice of the tricuspid regurgitant valve is neither circular nor flat. Therefore, the geometric assumptions needed to use the PISA formula to calculate the EROA or to measure the diameter of the vena contracta are not met in FTR. For the same reasons, and as it has been demonstrated by three-dimensional echocardiography, the shape of the proximal isovelocity surface is not a hemisphere. Accordingly, a single radius cannot account for the size and the complex shape of the PISA. In addition, the right ventricle is a volume pump, and the regurgitant volume in FTR varies greatly during the systolic period (larger at proto- and end-systole and smaller at meso-systole), with loading conditions (e.g. diuresis or volume overload, or even patient position can dramatically change the severity of the regurgitation) and with respiratory cycle. By simply measuring parameters, which do not a solid anatomic and functional ground, taken at one point during cardiac systole, we are oversimplifying a complex pathophysiological problem.

(2) Which are the new pathophysiological concepts that we need to integrate in our paradigm to try to better understand the pathophysiology of FTR and assess its severity?

First, we need to rethink the pathophysiology of FTR. Tricuspid annulus dilation is only one of the mechanisms that creates FTR. Papillary muscle displacement and leaflet tethering are also involved. The different mechanisms have a different role in FTR associated with different pathophysiological conditions (e.g. FTR in pulmonary hypertension vs. FTR in chronic atrial fibrillation). Second, the role of the right atrial volume as a determinant of tricuspid annulus size has been ignored so far. Last, but not the least, all measurements we perform should be related to the haemodynamics (right ventricular and right atrial volumes, pulmonary pressure) of the patient at the time of the echocardiographic study, integrated over time throughout systole and with respiratory phase.

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(3) However, independent on the severity of tricuspid regurgitation, we just need to measure the tricuspid annulus diameter to select patients for concomitant tricuspid annulus valvuloplasty at the time of left side valve surgery!

This is another paradigm that needs to be shifted. The cut-off value of 40 mm (or 21 mm/m<sup>2</sup>) to indicate the need of concomitant tricuspid annuloplasty in patients undergoing left side cardiac surgery comes from a single, uncontrolled study (i.e. how the echocardiographic measurement of 40 mm could correspond to the intraoperative 70 mm has never been clarified!), there is no agreement on which view you have to use (four-chamber of right ventricular focused four-chamber view), and, despite the mitral annulus size can change up to 25-30% during cardiac cycle, there is no precise timing at which to take the measurement (current guidelines just indicate 'diastolic diameter'). In addition, there is no anatomical landmark that guides the echocardiographer to measure the right diameter, and we know that, due to the crescentic shape of the right ventricle and the oval shape of the tricusEACVI-HIT Communication 945

pid annulus, slight rotations of the probe produce dramatic changes in tricuspid annulus diameter measurements by 2D echocardiography.

(4) What is the role of 3D echocardiography in the assessment of patients with FTR?

The role played by 3D echocardiography in the anatomical and functional assessment of patients with tricuspid regurgitation will be more and more important. This is particularly true because, due to the anatomical position of the right heart within the chest, the tricuspid valve is better visualized by transthoracic than by transoesophageal approach in the majority of patients. Preliminary studies have shown the ability of three-dimensional transthoracic echocardiography to assess the morphology, size, and functional changes of the various components of the tricuspid apparatus (tricuspid valve, tricuspid annulus, right ventricle, and right atrium) without geometrical assumptions about their shape. Similarly, single-beat full-volume 3D colour Doppler acquisition will hold

the promise to provide an integrated, quantitative assessment of tricuspid regurgitant volume independent on the shape of the regurgitant orifice.

(5) In your opinion, what are the future directions in the echocardiographic assessment of tricuspid valve?

In addition to the measurement of the severity of the regurgitation in relation to the haemodynamic load, the accurate assessment of the size and function of the components of the tricuspid valve apparatus will allow a more objective selection of patients to address to surgical tricuspid annuloplasty, to tailor the surgical procedure to the specific pathophysiology of that patient, and to select the right patient for the different transcatheter devices that are going to be developed for treating inoperable or high-risk patients. Moreover, a robust quantitative assessment of FTR severity and of the remodelling of the cardiac structures involved in FTR will allow an accurate monitoring of the transcatheter procedures as well the assessment of their efficacy.