LETTERS TO THE EDITOR

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Functional mitral regurgitation in non-ischaemic dilated cardiomyopathy patients: importance of papillary muscle dyssynchrony

We read with great interest the article by Tigen *et al.*[1] recently published in this journal.

The authors report systolic papillary dyssynchrony assessed by 2-D longitudinal speckle tracking as the only independent predictor of significant functional mitral regurgitation in a population of non-ischaemic dilated cardiomyopathy patients, including analysis of mitral valve geometric parameters, global longitudinal strain, and NYHA functional classification.

Although no geometric parameters of left ventricular geometry, proven to be predictors of tenting area and subsequently of regurgitant orifice area,[2] have been directly taken into account, the role of the dyssynchrony of papillary muscle has long been postulated as a major contributor to mitral regurgitation.

Previously published data have shown the correlation between mitral regurgitation and the dyssynchrony of segments containing papillary muscles insertion.[3] Acute improvement in mitral regurgitation after cardiac resynchronization therapy (CRT) by reduction of papillary dyssynchrony has also been described,[4] but direct data on papillary muscle dyssynchrony correlating to mitral regurgitation have been lacking to date.

As previously described, the presence of significant mitral regurgitation confers poor response to CRT,[5] and its persistence worsens the prognosis of these patients.[6] Shedding light on the underlying mechanisms of mitral regurgitation in dilated cardiomyopathy raises new challenges and the clinical role of these findings on patient selection and patient evolution after CRT must be elucidated.

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Papillary muscle dyssynchrony: what is the optimal cut-off value?

We appreciate the useful comments by Cabrera-Bueno *et al.* underscoring the importance of mitral regurgitation (MR) in patients with dilated cardiomyopathy (DCM). Cardiac resynchronization therapy (CRT) improves functional MR during the acute and chronic phase of heart failure by improving the coordination of the left ventricular segments and papillary muscle contractions.[1,2]. However, MR volumes of the patients with minimal papillary muscle dyssynchrony did not improve following CRT.[2] The best echocardiographic cut-off value to predict significant papillary muscle dyssynchrony, which in turn, would help patient selection for CRT is still unknown. The papillary muscles usually get closer to each other in the dilated and spheroidized left ventricles of patients with DCM; therefore, the opposing wall delay value of 65 ms might not be applicable in these patients. Most CRT studies on papillary muscle dyssynchrony have investigated the possible papillary muscle insertion regions on the left ventricle in their strain analysis. In patients with heart failure, usually lateral, posterior, and inferior myocardial segments are the most delayed segments independent of QRS duration.[3,4] The papillary muscles are usually localized to the lateral, inferior, and posterior segments, and these segments already activate later than other myocardial regions. The interpretation of delay between already most delayed segments might not be accurate. Most of the studies on this topic reported different cut-off values and the optimal delay time to choose CRT candidates is still unclear. Therefore, appropriate echocardiographic cut-off level for the prediction of papillary muscle dyssynchrony is needed. This information can be useful and indicate the severity of MR.

Cabrera-Bueno *et al.* demonstrated that baseline presence of important MR which was defined as a regurgitant orifice area $>0.2\ cm^2$ was associated with a lack of response in reverse remodelling in patients with non-ischaemic DCM although there was significant improvement in MR and intraventricular dyssynchrony.[5] In another study, the differences in pre-CRT infero-anterior time-to-peak two-dimensional radial strain of >110 ms and presence of non-severe MR were the significant predictors of MR reduction after CRT.[6] It is speculated that the maximum benefit of CRT on MR and the associated left ventricular remodelling seems to be present in patients with at least mild MR, but without severe MR.[7] In our