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EDITORIAL

Do we really need novel echocardiographic modalities to confirm the superiority of the intact His-Purkinje conduction system over pacing modes?

KEYWORDS
Echocardiography;
cardiac resynchronization therapy;
Doppler;
Speckle tracking echocardiography

Assessment of left ventricular function in patients with ischemic cardiomyopathy has been mainly based on the assessment of left ventricle ejection fraction (LVEF).[1] However, over the past few decades, it has become apparent that LVEF cannot reliably determine the prognosis or even the course and clinical diagnosis of ischemic cardiomyopathy in many cases.[2,3] Moreover, intra and inter ventricular mechanical dyssynchrony may further deteriorate left ventricle performance and clinical status. At this point, cardiac resynchronization therapy (CRT) has been proposed to improve patients' quality of life and prognosis.[4] Nevertheless, questions remain regarding cardiac CRT, including a) who may benefit from such an approach, b) where is and how can we determine the best site for left ventricle pacing, c) how can we measure response to CRT and d) what methods can optimize response to CRT?

Over the years, ultrasound systems and advanced software have allowed for the introduction of improved and reliable techniques for the assessment of left ventricle function. Evolving from m-mode assessment of limited parts of left ventricle motion to real time 2-dimensional images and, more recently, using real time 3-dimension images, which can be reliable and semi-automatically estimate stroke volume and ejection fraction.[5–7] Furthermore, myocardial performance can now be assessed by additional modalities such as tissue Doppler imaging (TDI) and speckle recognition, which can be used to measure myocardial deformation strain and strain rate to provide additional information on myocardial performance, which cannot be assessed by simply measuring changes in cavity volumes.[8–11] In addition, these modalities have less inter- and intra-observer variability, which may be advantageous in the recognition of patients with mechanical dyssynchrony and the serial assessment of response to treatment (see Figure 1).

Similar development was also observed in the assessment of left ventricle dyssynchrony.[12] M-mode can assess the delay between anteroseptal and posterior walls but with limited clinical value.[13] Tissue Doppler imaging allows for the identification of delays in multiple segments of the left ventricle as summarized in the dyssynchrony index, with the major limitation of angle dependency.[14] Currently, speckle tracking echocardiography and 3-dimensional imaging could provide a full data set from the entire ventricle. It may be used to provide time-volume curves for each of the 16 or 17 segments of the left ventricle, from which we can easily estimate the time-to-peak segmental contraction from each segment, allowing reproducible identification of the sequential conduction in each patient.[15] Despite the ability of recent echocardiographic modalities to assess even insignificant dyssynchrony, a series of large randomized studies failed to confirm the superiority of echocardiography over the robust electrocardiographic assessment in predicting response to CRT.[16,17] However, the role of echocardiography in the optimization of CRT continues to

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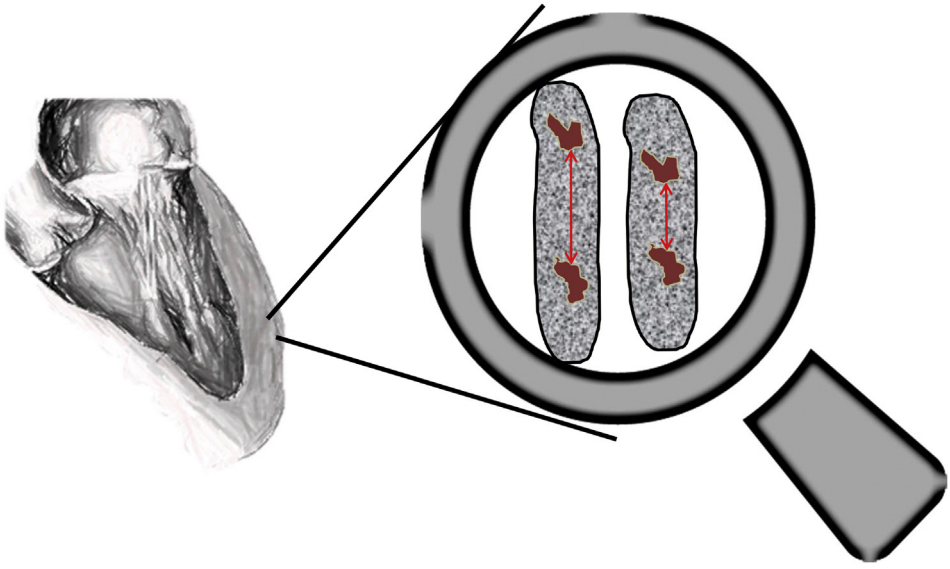

Figure 1 Principles of speckle tracking echocardiography

be valuable; studies have shown that left ventricle lead placement in the site of the latest activation may improve response or even outcome of heart failure patients especially when this site is not in scar tissue in patients with ischemic cardiomyopathy.[18,19]

Toumanidis et al. examines this exact issue in an experimental model of ischemic heart failure in the territory supply by the left anterior descending artery.[20] They confirmed that the natural conduction system is the best way to improve cardiac performance and left ventricle pacing at the apex outside the ischemic area may produce better results.

Currently, the optimum site of left ventricle pacing cannot be determined based on simple tests but must incorporate echocardiographic measurements, clinical data, anatomy and the physiologic knowledge of the natural conduction system.

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