Mechanical synchrony and survival in heart failure

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Patients with heart failure and left ventricular (LV) dysfunction continue to have an adverse prognosis. A number of clinical tools are helpful in identifying patients at particularly increased risk, and echocardiography is a remarkably powerful predictor of outcome. In addition to the established role of ejection fraction and LV volumes, shape and filling characteristics, left atrial size, mitral regurgitation and haemodynamics are predictors of recurrent heart failure and death. The extent of scar, viable myocardium and ischaemia are important determinants that can be added from stress and contrast echocardiography. Recent technical advances including the measurement of global longitudinal strain have added incremental information, perhaps of most value in patients with mild LV impairment, where the ejection fraction is a less reliable predictor of outcome.

In addition to its adverse functional correlates, left bundle branch block has long been a recognised marker of increased risk in heart failure. The initial paper linking mechanical dyssynchrony with outcome was published in 2004. That study of 104 patients with ejection fraction <45% and no previous myocardial infarction showed intraventricular but not interventricular dyssynchrony to be linked with outcome, with annual heart failure hospitalisation in patients with and without LV dyssynchrony to be almost 100% and only 50%, respectively. These findings have been confirmed in less severe heart failure and in the presence of a narrow QRS. In a study of 55 patients with complete left bundle branch block, functional class II (before hospital admission) and an ejection fraction of 40% (9), there was a lower (41%) incidence of cardiac events over approximately 2 years of follow-up, but intraventricular delay was also associated with events, with an optimal cut-off of 100 ms between the RV free wall and the most delayed LV segment.

In an earlier study of 106 patients with a narrow QRS and heart failure, Cho et al showed that the maximum temporal difference between eight segments was an independent predictor of events and mortality, independent of age, therapy, ejection fraction and QRS duration. These findings are supported by observations of the role of mechanical dyssynchrony as a predictor of adverse outcome in ischaemic heart disease. In an interesting analysis of 215 patients with ischaemic cardiomyopathy undergoing coronary bypass surgery, the presence of mechanical delay >119 ms between basal segments was predictive of in-hospital mortality, and dyssynchrony after revascularization was an independent predictor of clinical events. In 318 patients undergoing dobutamine stress echocardiography for known or suspected coronary artery disease, the magnitude of intraventricular dyssynchrony was an independent predictor of survival only in patients with asymptomatic LV dysfunction. The lesson of these studies has been that although the relative importance of intraventricular dyssynchrony depends on the clinical setting, this is an important and independent predictor of outcome.

Cho et al provide further evidence that LV mechanical synchrony is a predictor of recurrent cardiac events in patients with systolic heart failure in this edition of Heart (see page 1029). In this prospective follow-up study of 167 patients who were hospitalised with heart failure (ejection fraction <35%), 42% had cardiac events over nearly 3 years of follow-up, including a cardiac mortality of 25%. Patients with either a wide QRS or mechanical evidence of dyssynchrony were approximately twice as likely to have events, and those with a combination of both of these were four times as likely to have events in follow-up. Although this is a categorical analysis based on QRS duration of 120 ms and septal-to-lateral delay at 65 ms, this binary approach to allocating risk is one that fits well with clinical practice and is justified by the results of previous trials. These observations identify mechanical dyssynchrony as a predictor of adverse outcome in patients both with and without ischaemic heart disease. The importance of this signal in its own right, independent of markers of electrical synchrony, warrants careful consideration with respect to potential treatment implications. Of course, the problem with prognostic studies is that they do not distinguish risk from treatable risk. The remaining unresolved question in the area of mechanical synchrony is the extent to which selection for cardiac resynchronization therapy can improve outcome in patients who demonstrate this phenomenon. Despite a host of single-centre papers documenting a number of functional and even outcome benefits from cardiac resynchronization therapy in patients with mechanical synchrony, these markers remain technically difficult to measure, with the low concordance between observers and disappointing recent results in multi-centre studies. These findings on the prognostic implications of mechanical dyssynchrony should encourage ongoing work to define simple markers of dyssynchrony that are suitable robust for everyday use.

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