Influence of conduction time intervals on symptoms and exercise tolerance in patients with heart failure with left ventricular systolic dysfunction

A Champagne de Labriolle, C Leclercq, J C Daubert

RESULTS

In univariate analysis (analysis of variance or \( \chi^2 \) tests), symptom grade (NYHA class) increased and exercise tolerance decreased as conduction times increased. PR interval was 166 (31) ms, 185 (36) ms, 209 (45) ms, and 228 (56) ms in patients in NYHA classes I, II, III, and IV, respectively (p = 0.0003). Exercise time (\( r = -0.34, p = 0.0007 \)), maximum heart rate at peak exercise (\( r = -0.29, p = 0.004 \)), and peak \( V_O2 \) (\( r = -0.29, p = 0.004 \)) decreased as the PR interval increased. Likewise, QRS duration was 117 (35) ms, 135 (36) ms, 145 (38) ms, and 166 (28) ms in patients in NYHA classes I, II, III, and IV, respectively (p = 0.01). QRS widening was also associated with decreased exercise time (p = 0.02). A non-significant trend was observed towards a lower peak \( V_O2 \) (\( r = -0.19, p = 0.07 \)), LVEF was 30 (10)% in NYHA classes I, II, III, and IV, and left ventricular systolic dysfunction was of non-ischaemic origin in 87%. The mean (SD) LVEF was 26 (9)% in NYHA classes I, II, III, and IV, respectively (p = 0.0001). Decreased LVEF was also associated with shorter exercise time (\( r = 0.33, p = 0.0009 \)) and lower peak \( V_O2 \) (\( r = 0.29, p = 0.004 \)).

DISCUSSION

Although the correlations were relatively weak, the study results suggest that PR interval prolongation, but not QRS widening, contribute to increased symptoms and exercise intolerance regardless of baseline LVSD severity in patients with chronic heart failure. Not only does PR interval measured at rest reflect advanced LVSD but its prolongation also constitutes a major functional parameter that, after optimisation, potentially contributes to improving symptoms and exercise tolerance.

In conclusion, symptoms and exercise tolerance were independently affected by both the baseline severity of LVSD and the intracardiac conduction status. Although the correlations were relatively weak, the study results suggest that PR interval prolongation, but not QRS widening, contribute to increased symptoms and exercise intolerance regardless of baseline LVSD severity in patients with chronic heart failure. Not only does PR interval measured at rest reflect advanced LVSD but its prolongation also constitutes a major functional parameter that, after optimisation, potentially contributes to improving symptoms and exercise tolerance. Thus was the rationale of the first attempts at pacing for heart failure in the early 1990s, but controlled trials did not show any sustained benefit with short atrioventricular delay DDD pacing. Failure may be related to increased ventricular asynchrony resulting from right ventricular apical pacing that counterbalances the relative benefit of correcting atrioventricular asynchrony in the left heart.

In contrast, in this study QRS widening did not appear to be related to LVEF and left ventricular dimensions as an independent factor in symptom deterioration and exercise capacity impairment. Lastly, LVEF was confirmed as an independent factor in symptom deterioration and decreased exercise time.
present study essentially addressed the effect of spontaneous electrical asynchrony on exercise tolerance, some conclusions regarding cardiac resynchronisation can be drawn from our results. Firstly, the pathophysiological bases of electrical treatment for heart failure are valid. Secondly, atrioventricular asynchrony as a result of a prolonged PR interval may be sufficient to propose pacing for treating heart failure. Lastly, QRS duration should not be regarded as the only criterion for selecting patients for this new treatment.

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REFERENCES

IMAGES IN CARDIOLOGY

Transient pericardial effusion after cardiac surgery: often unrecognised

A 52 year old man with ischaemic heart failure was evaluated with cardiovascular magnetic resonance imaging (CMR) before coronary artery bypass surgery (CABG), demonstrating a dilated left ventricle with depressed ejection fraction (panel A). Surgery was performed without complications and the patient recovered well. CMR was repeated three months after the procedure for routine evaluation of cardiac function. Besides recovery of left ventricular function, approximately 350 ml of pericardial effusion was observed located posterior to the left and right ventricle (panel B). There were, however, no signs of tamponade from a clinical point of view and the patient was asymptomatic. Follow up CMR six months after CABG revealed notable resolution of the pericardial effusion (panel C).

The presence of pericardial effusion shortly after cardiac surgery is mainly detected clinically when a patient develops right and/or left sided heart failure, usually accompanied by chest discomfort, a pericardial rub, fever, and leucocytosis. This condition is often referred to as post-pericardiotomy syndrome. The vast majority of patients recover from cardiac surgery without these symptoms, and therefore no diagnostic effort is being made to detect pericardial effusion. However, postoperative pericardial effusion is considerably more common than clinically apparent, and occurs in as many as 85% of patients. Although anti-inflammatory agents may be useful to facilitate resolution, postoperative pericardial effusion is usually transient and the clinical course benign.

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True fast imaging with steady state precession (FISP) cine end diastolic short axis images. F, fat; LV, left ventricle; PE, pericardial effusion; RV, right ventricle.
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