ABSTRACT – SPECIAL INTEREST

S-C-1

Demonstration of Resynchronisation of the Left Ventricle after Biventricular Pacing in Patients with Advanced Heart Failure by Tissue Doppler Echocardiography

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Background: Our previous study found that biventricular pacing improved symptoms and cardiac function in patients with severe heart failure. However, the mechanism of benefit is still speculative. This study investigated whether there was evidence of cardiac resynchronisation after biventricular pacing using Tissue Doppler imaging (TDI).

Patients and Methods: 21 patients (64± 13 yrs, 14 male) with class III / IV heart failure and QRS duration >140ms who had biventricular pacemaker implanted (Medtronic. Inc) were followed up by serial echocardiography up to 3 months, and pacing was then withdrawn for 4 weeks. TDI was performed using 3 apical views by a 6 basal, 6 mid segmental model.

Results: The mean ejection fraction was 25± 9%, which increased significantly over 3 months (37± 13%, $p<0.01$). There was progressive decrease in left ventricular end-diastolic (220± 83 vs 174± 62ml, $p<0.05$) and end-systolic volume (185± 78 vs 132 ± 56ml, $p<0.05$) after biventricular pacing. By TDI, there was evidence of delay in the time to peak myocardial sustained systolic contraction (TS_M) at the basal lateral segment when compared to the basal septal segment (224± 48 vs 195± 61ms, $p<0.05$), which was abolished by biventricular pacing (188± 46 vs 178± 34ms, p=NS). The mean TS_M of the 12 segments was insignificantly shortened after biventricular pacing (184± 15 vs 196± 37ms, p=NS). However, when the standard deviation of the mean TS_M of the 12 segments was used as an indicator of the degree of dys-synchrony, it was shortened significantly after biventricular pacing (32± 10 vs 43± 15ms, $p<0.01$), reflecting the improvement of systolic synchrony.

Conclusion: Biventricular pacing reverses left ventricular remodeling and improves cardiac function in heart failure. This is likely secondary to the improvement of left ventricular synchronization during systole and especially abolishment of the lateral wall mechanical delay.

S-C-2

Elevation of Marcophage Migration Inhibitory Factor Level Acute Myocardial Infarction But Not in Acute Myocardial Ischaemia

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Background: Macrophage migration inhibitory factor (MIF) is a pro-inflammatory cytokine. It role in cardiovascular disease is poorly understood. This study investigated whether MIF is involved in acute myocardial infarction (AMI) and other myocardial ischaemic conditions.

Patients and Methods: Serial plasma MIF levels were assayed by ELISA in 37 patients (mean age: 60± 12 years, 89% male) with clinical diagnosis of AMI, 26 patients with unstable angina, 39 patients with stable angina, 26 patients before and after elective uncomplicated percutaneous coronary angioplasty (PTCA) and 31 control subjects who presented with atypical chest pain with normal coronary angiogram.

Results: Plasma MIF levels, like creatine phosphokinase and its MB-isoenzyme levels, was rapidly and highly significantly elevated after an initial episode of AMI (Day 1- AMI: 3114± 789 vs controls: 633± 130 pg/mL; $p < 0.001$ when compared to AMI), accounting for up to 4-7 fold increase than all the other disease groups and controls, indicating that myocyte necrosis may be a major source of MIF. In addition, the MIF level was higher in those who had chest pain at the time of blood taking than those who had not (4269± 922 vs 1848± 303 pg/mL, $p < 0.005$). However, unlike those cardiac enzymes, the plasma MIF level remained high over the first three days, indicating that MIF may contribute to the ongoing local inflammatory response and produced also from cells other than necrotic myocytes. The MIF levels in all the other disease groups were not different from controls, and were not different before and after PTCA or during sequential blood taking for patients with unstable angina.

Conclusion: The pro-inflammatory cytokine, MIF, was rapidly elevated in patients with AMI but not in conditions with transient myocardial ischemia such as angina and PTCA. MIF may be a mediator and play a role in mediating ongoing inflammatory response after AMI.