Multisite stimulation for correction of cardiac asynchrony

Cardiac electrical stimulation as primary or adjunctive treatment of congestive heart failure is entering its second decade of existence. Initial trials of conventional DDD pacing1–5 were followed by bifocal right ventricular stimulation.6 The concept of multisite stimulation for haemodynamic support was introduced in 1994.7 Various studies within the cardiac cycle.

During the cardiac cycle, the E wave location has changes with respect to the A wave, and the filling time with respect to the complete cardiac cycle, and, on occasion, by early passive ventricular filling flow superimposed on atrial systole dependent flow. Dual chamber pacing, by linking ventricular to atrial activation, normalises flow patterns of ventricular filling, provided atrial contraction is mechanically effective (fig 1). This principle guides the optimisation of the atrioventricular delay under Doppler echocardiographic guidance in dual chamber pacing. The end point consists of shortening the atrioventricular delay to a minimum without interrupting the end of the A wave by premature mitral valve closure (fig 2).

The concept of interventricular asynchrony

Atrioventricular resynchronisation alone has not been predictably effective in improving haemodynamics except in some patients with prolonged PR interval.2 Candidates for the implantation of a cardiac stimulator often present with intraventricular conduction disorders expressed as a widened QRS, an anomaly which may also be caused by the pacemaker itself. In the hope of minimising atrioventricular asynchrony, a delay is unfortunately imposed between right and left ventricular activation by stimulating the ventricle from the peripheral Purkinje network instead of preserving the normal sequence via the His bundle. This activation/contraction delay causes a delay in subsequent chamber relaxation, disturbing its filling (fig 3). As a result, the right-to-left electromechanical delay caused by typical pacing from the right ventricular apex may disrupt the synchrony of right and left ventricular systole. This spontaneous or iatrogenic asynchrony can be easily quantified by measuring the delay between the onset of ventricular electrical activation and the onsets of aortic and pulmonic ejection, respectively. The difference between these two intervals represents the interventricular mechanical delay (fig 4), and a shortening in both the pre-ejection delays and intervals represents the interventricular mechanical delay (fig 5).

The concept of intraventricular systole-diastolic asynchrony

Finally, heterogeneity of intraventricular myocardial contraction may be so pronounced as to result, within the same ventricle, in the coincidence of territories in systolic phase,
while others are already relaxing. A marker of such heterogeneity is the QRS width and, sometimes, its polyphasic morphology. Intraventricular asynchrony is unlikely in presence of spontaneous QRS complexes < 120 ms, and paced QRS complexes < 180 ms in duration, and may be greatly accentuated by stimulation confined to the right heart chambers. It is often present in ischaemic cardiomyopathy, where the propagation of ventricular activation wavefronts may be disrupted by the encounter of scarred or ischemic tissue. This abnormal process is characterised by the presence of myocardial segments contracting as ventricular filling has already begun. This, on time-movement echocardiographic examination (fig 6), appears as segmental wall thickening occurring after the onset of the next early diastolic filling phase. Simultaneous stimulation of left and right cardiac chambers is, currently, not capable of predictably correcting this abnormality. However, its successful elimination often decreases the severity of mitral regurgitation, an important contribution to clinical improvement in some patients.

Patient selection
Multisite stimulation for congestive heart failure is a recent therapeutic method, which will, hopefully, be validated in the year 2000. It should be reserved for patients whose abnormal ventricular function may be corrected or improved by palliating conduction disorders, in the hope of changing the sequence of mechanical events, enhancing contractile efficiency, and, in some cases, lessening the severity of ventriculo-atrial regurgitation by changing systolic left ventricular geometry. Other mechanisms have been proposed, which remain speculative. Although the cardiac mechanics related to multisite stimulation are only partially understood, echocardiography has emerged as a non-invasive and easily reproducible test of choice in the selection of candidates for this new treatment. The absence of preimplant visible asynchrony probably predicts little improvement by multisite stimulation, but this point needs confirmation. Conversely determination of the nature of the asynchrony should facilitate the choice of an optimal synchroniser configuration. Modification of the electromechanical intervals in the postoperative course should also provide useful information for assessing the success or the failure of cardiac resynchronisation.
Three dimensional reconstruction of femoral pseudoaneurysm using contrast enhanced axial CT angiography

A 45 year old man was admitted for an electro-physiological procedure using a retrograde aortic approach via an 8 French right femoral artery sheath. The patient received low molecule weight heparin after the procedure. A large pulsatile mass with a diffuse haematoma and bruit developed in the right groin two days after the procedure. Duplex ultrasound scanning (ATL HDI 3000, 4–7 MHz probe) demonstrated a large cavity (10 × 18 cm) with arterial turbulent flow (left). However, the origin of the false aneurysm from the artery could not be clearly identified. Contrast enhanced axial computed tomographic (CT) angiography with three dimensional reconstruction (Lightspeed CT scanner and Advantage Windows, Volume Rendering, General Electric, USA) was performed and confirmed a multiple saccular false aneurysm arising from the common femoral artery (below, left panel). The neck of the false aneurysm was localised by the CT angiogram and manual compression was applied at that site. After 20 minutes of compression, the pulsatility and the bruit from the false aneurysm ceased. A repeat CT angiogram confirmed the obliteration of the false aneurysm (below, right panel). The patient was discharged from the hospital the following day and only a small resolving haematoma was detected during follow up.

As this case illustrates, the presence of a multiple saccular false aneurysm may lead to difficulties in identifying its origin by duplex ultrasound scanning. CT angiogram is useful in such cases for diagnosing and guiding treatment of the iatrogenic pseudoaneurysm after femoral artery catheterisation.
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Heart 2000 84: 579-581
doi: 10.1136/heart.84.6.579

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