CARDIOVASCULAR MEDICINE

Colour tissue velocity imaging can show resynchronisation of longitudinal left ventricular contraction pattern by biventricular pacing in patients with severe heart failure

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Objective: To quantify ventricular resynchronisation by biventricular pacing using colour tissue Doppler velocity imaging (c-TVI).

Design and patients: c-TVI shows regional tissue velocity profiles with a very high time resolution (10 ms). Eighteen patients were studied from an apical four chamber view at baseline and after a one month follow up of biventricular pacing. Regional left ventricular peak tissue velocities and regional time differences during the cardiac cycle were compared in the basal and mid interventricular septal segments of the left ventricle, and in the corresponding segments in the left ventricular free wall.

Results: From baseline to follow up, mean peak tissue velocities changed only during isovolumic contraction in the basal interventricular septum and the left ventricular free wall. At baseline the peak main systolic tissue velocities in the left ventricular free wall were typically delayed by an average of 42 ms in the basal left ventricular site and by 14 ms in the mid left ventricular site compared with the corresponding sites in the interventricular septum. After resynchronisation by biventricular pacing those regional movements were separated by an average of only 7 ms at the basal site, but there was still a 21 ms earlier movement of the left ventricular free wall in the mid left ventricular site. The diastolic movement pattern remained unchanged from baseline to follow up.

Conclusions: c-TVI showed a significant asynchronous regional longitudinal movement of basal left ventricular sites at baseline. A change to a more synchronous longitudinal left ventricular movement pattern during biventricular pacing was demonstrated.

The use of biventricular pacing in patients with heart failure and bundle branch block has increased rapidly in recent years. Beneficial acute haemodynamic effects of biventricular pacing have been shown, consisting of a reduction in pulmonary capillary wedge pressure and an improvement in peak dP/dt without an increase in oxygen consumption^{1–4}, as have long term clinical benefits in terms of improvements in the six minute hall walk test (6MHW), New York Heart Association (NYHA) functional class, maximum oxygen consumption, and quality of life indices.^{5–8}

The mechanisms responsible for the haemodynamic improvement achieved by biventricular pacing are still not entirely clear. However, improved interventricular and intra-ventricular coordination of contraction has been shown using gated blood pool scintigraphy⁹ and echocardiographic phase analysis of the left ventricle in the short axis.¹⁰

In both animal experiments¹¹ and human studies,¹² colour tissue Doppler velocity imaging (c-TVI) reflects myocardial contractility by measuring the movement of cardiac tissue. The peak velocities measured by c-TVI are about 15–20% lower than those measured by pulsed Doppler echocardiography, but the measurements can be used interchangeably for velocity profile recording and for timing of events.¹³

It was recently shown using c-TVI that acute biventricular pacing in patients with severe heart failure resulted in resynchronisation of contraction in four of six basal left ventricular segments.¹⁴ c-TVI recordings can be made at very high frame rates of more than 100 frames per second (fps), thus providing the possibility of comparing the timing of velocity profiles at different left ventricular sites with a time resolution of 10 ms. The regional timing of the four distinct left ventricular tissue velocity peaks represented by the isovolumic contraction

(IVC), peak main systolic velocity (SYS), early diastolic (E), and late diastolic peak velocity (A) can be studied by using c-TVI.

Our aim in this study was to quantify the changes in the longitudinal left ventricular contraction pattern caused by biventricular pacing in patients with severe heart failure and bundle branch block by using c-TVI.

METHODS

Patients

After their informed consent had been obtained, the study group consisted of seven women and 11 men (mean (SD) age 63 (13) years) with ischaemic cardiomyopathy (n = 10), idiopathic dilated cardiomyopathy (n = 5), or valvar heart disease (n = 3). Eleven patients were in sinus rhythm and seven had chronic atrial fibrillation. Seventeen patients had left bundle branch block (caused by a permanent pacemaker in two cases), and one patient had right bundle branch block. All patients had severe heart failure and were optimally treated with diuretics, angiotensin converting enzyme inhibitors, digitalis, and β blockers when tolerated, and were not candidates for cardiac surgery.

The patients received biventricular pacing according to accepted criteria of an ejection fraction < 35%, QRS width > 120 ms, and severe heart failure in NYHA functional classes

Abbreviations: A, late diastolic peak velocity; c-TVI, colour tissue velocity imaging; E, early diastolic peak velocity; fps, frames per second; IVC, isovolumic contraction; MRI, magnetic resonance imaging; SYS, peak main systolic velocity; 6MHW, six minute hall walk test

Table 1 Patient characteristics at baseline and follow up

	Baseline	Follow up	p Value
NYHA functional class	III–IV (mean 3.4)	II–III (mean 2.6)	<0.05
Ejection fraction (%)	21.6 (5.8)	30.7 (6.4)	<0.05
LVEDd (cm)	7.3 (1.1)	7.1 (1.2)	NS
LVEDs (cm)	6.4 (1.0)	6.1 (1.3)	0.073
LAD (cm)	5.1 (1.1)	5.3 (1.2)	NS
Body weight (kg)	82 (14)	77 (11)	<0.05
QRS duration (ms)	173 (23)	166 (21)	<0.05
6MHWt distance (m)	351 (141)	460 (159)	<0.05
HR during examination (beats/min)	75 (17)	82 (15)	NS

Values are mean (SD).

HR, heart rate; LAD, left atrial diameter; LVEDd, left ventricular end diastolic diameter; LVEDs, left ventricular end systolic diameter; NYHA, New York Heart Association; 6MHWt, 6 minute hall walk test.

III and IV. They were studied at baseline and after biventricular pacing for at least one month of follow up, with clinical evaluation, body weight, ECG, 6MHWt, and echocardiography.

Pacemaker system

The pacemakers used were the InSync 8040 ($n = 10$) and the InSync 8042 ($n = 8$) (Medtronic Inc, Minneapolis, Minnesota, USA). The InSync 8040 is a multisite stimulator for cardiac resynchronisation, with two parallel connected ventricular channels providing similar stimulation of the right and left ventricle. The InSync 8042 pacemaker has two separate ventricular channels for right ventricle and left ventricle, respectively, and with the possibility of programming different interventricular stimulation delays from 4 ms to 80 ms. Guided by fluoroscopy, a long guide catheter (Attain LDS model 6216, Medtronic Inc) was placed in the coronary sinus. With the aid of an inflatable balloon catheter, venograms of the coronary veins were made in the frontal and left lateral oblique view. Guided by the venograms, special coronary vein leads (lead model 2187 ($n = 3$), 4191 ($n = 5$), or 4189 ($n = 10$); Medtronic) were advanced to a stable position in a lateral coronary vein. Acceptable thresholds below 3 V for left ventricular pacing were obtained. Left ventricular stimulation at 10 V was done to exclude phrenic nerve stimulation. The right ventricular lead was placed in the apex of the right ventricle, and a third lead with passive fixation was placed in the right auricle in the patients who were in sinus rhythm.

Echocardiographic methods

The examination was undertaken with the patients in the left lateral recumbent position using the System FiVe digital ultrasound machine (GE Vingmed Ultrasound, Horten, Norway), with a combined phased array transducer providing colour tissue Doppler, cross sectional, M mode, spectral Doppler, and colour Doppler echocardiography. Calculation of the ejection fraction was done in accordance with the recommendations of the committee of the American Society of Echocardiography (ASEC), using Simpson's rule.¹⁵ By using M mode echocardiography the left ventricular end diastolic, left ventricular end systolic, and left atrial diameters were measured from the parasternal long axis view following the ASEC recommendations. During the study, heart rate was measured from a continuously recorded single channel ECG.

Colour tissue velocity imaging

Colour tissue Doppler data were obtained in the apical four chamber view at frame rates close to 100 fps, allowing exact evaluation of the left ventricular longitudinal movement pattern with a time resolution of 10 ms. When postprocessing these colour Doppler data, a user defined region of interest was selected, including subendocardial, mid-wall, and subepicardial zones. This was to prevent the region of interest from moving out of range because of lateral movement of the

respective left ventricular areas during the cardiac cycle. The simultaneous velocity curves of four selected left ventricular segments were compared for a mean of two consecutive beats to minimise the variability between measurements. A larger number of beats could not be studied because the data would have exceeded the storage capacity of the computer. The left ventricular tissue velocities and regional time differences of peak tissue velocities were measured during IVC, SYS, E, and A. The four left ventricular segments selected for longitudinal velocity sampling were the basal left ventricular and the mid left ventricular segments of the interventricular septum and the corresponding segments in the left ventricular free wall.

Statistics

The data are expressed as mean (SD). The statistical methods used were Student's *t* test for normally distributed paired and unpaired data and the Mann–Whitney/Kruskal–Wallis test for non-parametric data, as appropriate, using commercial software (SPSS Inc, release 10.1). A probability value of $p < 0.05$ was considered significant.

RESULTS

Pacemaker programming

The 11 patients in sinus rhythm received atrio-biventricular pacing, and the seven patients with atrial fibrillation received biventricular pacing. Interventricular pacing delays ($n = 8$) were optimised to those that provided the highest cardiac output measured by pulsed Doppler echocardiography¹⁶ at rest by testing various programmed interventricular pacing delays from 4–80 ms. The optimal interventricular pacing delay was on average 37 (25) ms, with the left ventricle paced before the right in all eight cases. For the whole group with atrio-biventricular pacing, the left ventricle was stimulated on average 16 (25) ms before the right. All patients had acceptable thresholds for right ventricular and left ventricular pacing, and 100% biventricular pacing was achieved during follow up.

Clinical improvement

From baseline to follow up there was a significant mean improvement of I in NYHA functional class and 87 (59) metres in mean 6MHWt distance; body weight was reduced by an average of 5 kg (table 1). All patients improved at least one half in NYHA functional class. In all patients the ejection fraction improved by ≥ 15 relative percentage points, and in 15 patients it improved by $> 25\%$. Only one patient showed a 7% reduction in 6MHWt distance despite an increase in ejection fraction from 20% to 37%.

Comparing the patients with atrial fibrillation and those in sinus rhythm from baseline to follow up showed that the patients with atrial fibrillation improved in NYHA functional class (3.6 to 2.9), ejection fraction (20% to 31%), and 6MHWt distance (270 m to 403 m). The improvements were of a similar order to those in the patients in sinus rhythm (NYHA

Figure 1 Upper panel: peak velocities (in cm/s) in systole (IVC) in basal and mid parts of interventricular septum (IS) and lateral free wall (LFW). Lower panel: time differences (in ms) in systole (IVC). Time differences between basal LFW and IS (A), mid and basal IS (B), mid-LFW and IS (C), and mid and basal LFW (D) are shown. Significant differences ($p < 0.05$) between baseline and follow up are marked with an asterisk. IVC, isovolumic contraction.

functional class improved from class III (mean 3.2) to class II (mean 2.1); ejection fraction from 23% to 30%; and 6MHW distance from 390 m to 480 m).

Left ventricular function and dimension

The left ventricular ejection fraction was improved by an average of 27 (18) relative percentage points ($p < 0.001$) from baseline to follow up (table 1). From baseline to follow up there was no significant difference in left ventricular end diastolic diameter and left atrial diameter. However, the reduction in left ventricular end systolic diameter almost reached significance ($p = 0.073$).

Colour tissue Doppler imaging

During colour tissue Doppler imaging, the frame rate was 95.7 (20.6) fps (median 89) at baseline and 100.5 (20.8) fps (median 96) at follow up. The mean peak tissue velocity values from baseline to follow up changed only in a slight but significant reduction in the basal interventricular septum, an increase in the left ventricular free wall during the IVC period (fig 1), and a reduction in mid left ventricular free wall from -1.4 (1.2) to -0.8 (0.7) cm/s during A.

Reproducibility

At baseline, the correlation coefficient between beats 1 and 2 for IVC peak tissue velocity ranged from 0.880 to 0.924 ($p < 0.001$), for SYS from 0.552 to 0.965 ($p < 0.05$), for E from 0.745 to 0.962 ($p < 0.001$), and for A from 0.636 (mid lateral, $p = 0.124$) to 0.892–0.946 ($p < 0.005$) at the four selected left

ventricular segments. At follow up the correlation coefficient for IVC peak tissue velocity ranged from 0.858 to 0.973 ($p = 0.001$), for SYS from 0.718 to 0.965 ($p < 0.005$), for E from 0.893 to 0.952 ($p < 0.001$), and for A from 0.783 (basolateral, $p = 0.66$) to 0.824–0.988 ($p < 0.05$) at the four sites selected.

Mean peak tissue velocity

From baseline to follow up only three of the four regional left ventricular mean peak tissue velocities showed a significant change. Those were IVC at the basal interventricular septum (from 2.6 (1.7) cm/s at baseline to 1.1 (1.9) cm/s at follow up) and at the basal left ventricular free wall (from 1.3 (1.5) cm/s at baseline to 2.9 (1.6) cm/s at follow up); and A at the mid left ventricular free wall (from -1.4 (1.2) cm/s at baseline to -0.8 (0.7) cm/s at follow up).

Peak tissue velocity time difference

A positive value of the time difference of peak tissue velocity in basal interventricular septum and mid interventricular septum versus the corresponding sites in the left ventricular free wall represents an earlier longitudinal movement of the interventricular septal sites, and a negative time difference between the same sites represents an earlier left ventricular free wall longitudinal movement.

Changes in the pattern of left ventricular movement from baseline to follow up

At baseline during IVC, there was an average of 18 (23) ms earlier longitudinal movement of the interventricular septum

Figure 2 Upper panel: peak velocities (in cm/s) in systole (SYS) in basal and mid parts of interventricular septum (IS) and lateral free wall (LFW). Lower panel: time differences (in ms) in systole (SYS). Significant differences ($p < 0.05$) between baseline and follow up are marked with an asterisk. SYS, peak main systolic velocity.

compared with the free wall in the left ventricular basal site, and 21 (20) ms earlier movement in the mid left ventricular site. At baseline during SYS, there was an average of 42 (40) ms earlier longitudinal movement of the interventricular septum compared with the free wall in the left ventricular basal site, and 14 (53) ms earlier movement in the mid left ventricular site. At baseline during early diastole in E, the corresponding left ventricular tissue velocity time differences between the interventricular septum and the left ventricular free wall were -9 (49) ms in the basal region and -17 (60) ms in the mid left ventricular region, respectively.

At follow up during biventricular pacing the corresponding peak tissue velocity time differences in the basal and mid left ventricular sites during systolic IVC and during SYS had changed significantly compared with baseline, as shown in figs 1 and 2. The majority of the other tissue velocity time differences in both the interventricular septum and the left ventricular free wall, and between the other left ventricular sites, remained unchanged in systole and diastole. However, there was a 42 (61) ms delay in the basal left ventricular free wall compared with the mid interventricular septum during SYS at baseline, changing to an 8 (65) ms delay at follow up, and a 39 (57) ms delay in the mid left ventricular free wall compared with the basal interventricular septum during A at baseline, changing to a 22 (35) ms delay at follow up.

Compared with baseline, biventricular pacing changed the regional peak left ventricular tissue velocity time differences of interventricular septal and left ventricular free wall movement significantly. Thus longitudinal movement

changed from asynchronous to almost simultaneous in the basal left ventricular site (average difference 5 (15) ms) and the mid left ventricular site (average difference -2 (29) ms) during the IVC period of systole. The SYS peak tissue velocity time difference between the interventricular septum and the left ventricular free wall at the basal left ventricular site also changed significantly from baseline to biventricular pacing—from delayed contraction of the left ventricular free wall (by 42 (40) ms) to almost simultaneous longitudinal movement of the interventricular septum and the left ventricular free wall (average difference 7 (47) ms). The SYS peak tissue velocity time difference between the interventricular septum and the left ventricular free wall at the mid left ventricular site changed from a 14 (53) ms delay in the free wall at baseline to a 21 (53) ms delay in the interventricular septum at follow up.

Figure 3, recorded from one of the patients, shows a 57 ms delayed left ventricular left ventricular free wall at baseline and an almost synchronous SYS peak velocity in interventricular septum and in left ventricular free wall (6 ms) at follow up.

Comparison of patients with atrial fibrillation and sinus rhythm

Comparing the patients with atrial fibrillation and those in sinus rhythm showed that the mean peak tissue velocities in IVC, SYS, and E did not differ at any of the four examined left ventricular sites, either at baseline or at follow up. Examining the tissue velocity time differences in basal and mid left ventricular segments at baseline and follow up showed significant

Figure 3 Colour tissue velocity imaging (c-TVI) recorded in the apical four chamber view (upper left panel colour imaging, lower left panel grey scale). Postprocessing c-TVI curves are shown before and after implantation in the same patient. Before implantation the basal part of the left ventricular free wall (LFW) was 57 ms delayed compared with the interventricular septum (IS). After implantation an almost synchronous SYS peak velocity between IS and LFW is seen (6 ms).

differences only at baseline during IVC at the mid left ventricular sites between the patients with atrial fibrillation (37 (12) ms) and those in sinus rhythm (6 (14) ms) ($p < 0.05$).

DISCUSSION

Many patients with heart failure suffer from intra- and inter-ventricular asynchronous contraction and relaxation, indicated on the surface ECG^{17,18} and demonstrable by conventional echocardiographic methods. The patients studied here—all of whom had a conventional indication for implantation of biventricular pacing pacemakers—improved their ejection fraction, 6MHW distance (except one patient), and NYHA functional class by a similar degree to other published studies, and can thus be regarded as representative of this cohort of patients.^{6–8} The fact that all patients showed an improvement in ejection fraction, 6MHW distance, or NYHA functional class means that our c-TVI results are applicable to responders to biventricular pacing. Patients with atrial fibrillation and those in sinus rhythm did not differ in their clinical outcome or c-TVI findings and were therefore examined as one group.

Changes in regional left ventricular contraction timing from baseline to biventricular pacing

During IVC, longitudinal left ventricular movement showed a significant change from baseline to biventricular pacing, from asynchronous to almost simultaneous movement of the interventricular septum and the left ventricular free wall in both the basal segment (18.3 v 4.9 ms) and the mid left ventricular segment (21.2 v –1.7 ms). During SYS, longitudinal movement in the basal interventricular septum and left ventricular free wall also changed significantly, from a 43 ms delay in the left ventricular free wall at baseline to an almost synchronous movement of the interventricular septum and free wall (7 ms) during biventricular pacing. During biventricular pacing, the mid left ventricular interventricular septum was delayed (–21 ms) compared with mid left ventricular free wall; this was completely different from the baseline value, when the mid left ventricular free wall was delayed by 14 ms compared with the septum. This latter observation can be explained by the fact that the mid left ventricular free wall was stimulated by the lead tip located in the lateral coronary vein, close to the left ventricular free wall, on average 16 (25) ms earlier than was the interventricular septum by the right ventricular apical lead.

The diastolic longitudinal left ventricular movement pattern that represented the complex relaxation of the left ventricle in patients with severe heart failure was not influenced by the earlier stimulation of the left ventricle during biventricular pacing.

Regional left ventricular mean peak velocities

The mean systolic and diastolic peak tissue velocities at baseline and during resynchronisation therapy remained unchanged in all left ventricular segments except during IVC, showing reduced mean peak tissue velocity in the basal interventricular septum and increased peak velocity in the basal left ventricular free wall. This could be explained by the fact that earlier stimulation of the left ventricular free wall during pacing leads to an immediate longitudinal movement of the free wall before stimulation of the interventricular septum, with no tethering effect of other contracting left ventricular segments. The unchanged mean regional peak tissue velocities during SYS and E from baseline to the better performing left ventricle during resynchronisation shows the importance for global left ventricular performance of obtaining more synchronous regional longitudinal contraction. The observed lack of change in regional contractility from baseline to biventricular pacing may explain a previous observation that there was no increase in myocardial oxygen consumption during biventricular pacing.⁸

The improved systolic performance of the left ventricle in patients with severe heart failure effected by biventricular pacing may have been a result of the documented resynchronisation of the regional contraction pattern, with no increase in regional peak tissue velocities.

Methods for demonstrating asynchronous left ventricular contraction

The use of three dimensional tagged magnetic resonance imaging (MRI) to show asynchronous contraction of left ventricle¹⁹ is time consuming and has poor time resolution owing to a lower frame rate of 30 fps compared with echocardiographic methods such as c-TVI. Furthermore, three dimensional tagged MRI cannot be employed in patients with implanted pacemakers. Gated blood pool scintigraphy has been used to demonstrate asynchronous contraction of the left ventricle and to show the effect of biventricular pacing, but this method also has a relatively poor time resolution of 30 fps.⁹

Long axis ventricular contraction plays an important role in left ventricular systolic function,²⁰ and in recording c-TVI, angle correction of tissue velocity for the four selected left ventricular segments assumes less importance, as the angle between the ultrasound beam and the direction of the longitudinally moving left ventricular tissue in the four chamber long axis view is very small. The four selected left ventricular sites may thus be regarded as representative for detecting regional time differences in longitudinal left ventricular contraction/movement patterns during the cardiac cycle.

The time resolution of 90–100 fps obtained in this study allowed us to evaluate the timing of left ventricular tissue movement within a range of 10 ms, thus providing a unique opportunity to measure changes in the left ventricular regional contraction pattern produced by biventricular pacing.

Limitations

Our short term follow up of one month's biventricular pacing showed resynchronisation of longitudinal left ventricular contraction, but to demonstrate reverse remodelling of the left ventricle with biventricular pacing requires a longer term follow up. The possible changes in radial or rotational movement of the left ventricle effected by biventricular pacing were not addressed in this study. The storage limitation of the computer made it impossible to study more than two consecutive cardiac cycles to minimise respiratory influence and beat to beat variation.

Conclusions

The new echocardiographic method of colour tissue velocity imaging can contribute to a better understanding of the mechanisms whereby left ventricular global systolic function is improved by biventricular pacing. To the best of our knowledge this study is the first to quantify the resynchronising effect of biventricular pacing on the left ventricular regional contraction pattern. This method will probably also be of great value in selecting patients with severe heart failure, who will be haemodynamic responders to biventricular pacing. A still unanswered question is whether patients with heart failure and without bundle branch block may also have asynchronous left ventricular contraction and thus be potential candidates for biventricular pacing.

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