CARDIOVASCULAR MEDICINE

Effect of multisite pacing on ventricular coordination

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Objective: To determine the effect of multisite pacing on left ventricular function.
Design: Prospective observational study.
Patients: 18 patients with heart failure with a dilated poorly functioning left ventricle (LV) and left bundle branch block.
Interventions: Pacing for 5 minutes in random order at the right ventricle (RV) apex, RV outflow tract, mid posterolateral LV, RV apex and LV simultaneously, and RV outflow tract and LV simultaneously. The best achieved measurements with RV, LV, and biventricular pacing were compared.
Main outcome measures: LV dimension, filling characteristics, and long axis indices were measured on echocardiography simultaneously with LV pressure. Cycle efficiency (%)—that is, the ratio of the area of the acquired pressure dimension loop to that of the ideal loop for that segment—quantified coordination.
Results: The pacing site that gave the best achieved cycle efficiency differed between patients (biventricular in five, LV in two, RV in seven, and no site in four). In patients with baseline incoordination (cycle efficiency $\leqslant$ 72%, n = 12) cycle efficiency improved significantly with RV pacing (cycle efficiency 76%, p = 0.01) but not with LV (65%) or biventricular (67%) pacing. LV based pacing induced premature short axis contraction in a subset of patients (n = 4), which was associated with a prolonged time from the Q wave on the ECG to the onset of inward movement of the long axis (from apex to mitral ring): biventricular 145 ms, LV 105 ms, RV 85 ms (biventricular *v* RV, p < 0.05). Excluding patients with baseline incoordination in whom premature activation occurred, pacing at all sites led to a similar increase in cycle efficiency (RV 78%, LV 72%, biventricular 73%).
Conclusions: Ventricular coordination can be improved with pacing in patients with baseline incoordination. Short and long axis fibres may be asynchronised in a subset of patients with LV or biventricular pacing, which may worsen coordination. The clinical significance of these findings remains to be defined.

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In the past decade pacing has been increasingly proposed as a potential treatment of advanced heart failure. Originally dual chamber pacing was used with variable results,[1–3] and more recently multisite[4 5] and left ventricle (LV) based pacing[6] have been advocated. Several studies have assessed cardiac haemodynamics with multisite pacing but few have assessed the mechanical consequences of multisite pacing on ventricular function.

Improved ventricular coordination or "cardiac resynchronisation" is often cited as the mechanism of improvement with pacing, although LV coordination has never been directly measured in paced patients. Pressure–dimension relations, unlike those between pressure and volume, allow the timing of the pressure generated by the ventricle to be compared with contraction and relaxation in a localised area represented by the dimension. If there is a change in one dimension during an isovolumic period it implies a compensatory change in dimension somewhere else in the LV, and incoordination is manifest by a shape change during the isovolumic periods. Incoordination is represented by loss of the optimal relation between pressure and dimension (fig 1).[7]

Normal ventricular function requires longitudinal as well as circumferential fibres, and the relation between long and short axis motion in healthy people is characteristic. Bundle branch block delays the onset of long axis shortening, and these changes vary with the activation pattern in intermittent bundle branch block. Therefore, analysis of ventricular long axis motion provides a means of investigating the consequences of abnormalities of activation (fig 2).[8 9]

We aimed at determining the effects of multisite pacing on ventricular coordination, ventricular long axis function, and ventricular filling characteristics in patients with advanced heart failure and left bundle branch block (LBBB).

PATIENTS AND METHODS

Study group

After giving informed consent, 23 patients (19 men, four women) in sinus rhythm with LBBB, a dilated poorly functioning LV (end diastolic diameter > 60 mm, shortening fraction < 25%), and stable heart failure (New York Heart Association (NYHA) functional class IIb or III) were enrolled. Patients with severe mitral regurgitation on echocardiography or with atrial fibrillation were excluded. The local ethics committee approved the protocol.

Catheterisation protocol

Patients fasted on the day of evaluation and were treated with their usual medication, excluding the morning dose of diuretic. Patients were lightly sedated (diazepam 2.5 mg intravenously) at the start of the procedure. The right femoral artery was cannulated and a 6 French gauge micromanometer tipped catheter (SPC 464D, Millar, Houston, Texas, USA) was positioned in the LV cavity and calibrated using a TC 510 control box (Millar) in the standard manner. Heparin was given to maintain an activated clotting time of 250 seconds. One 6 French electrode (Josephson, Bard, Billerica) was positioned

Abbreviations: LBBB, left bundle branch block; LV, left ventricle; NYHA, New York Heart Association; RV, right ventricle; RVOT, right ventricular outflow tract

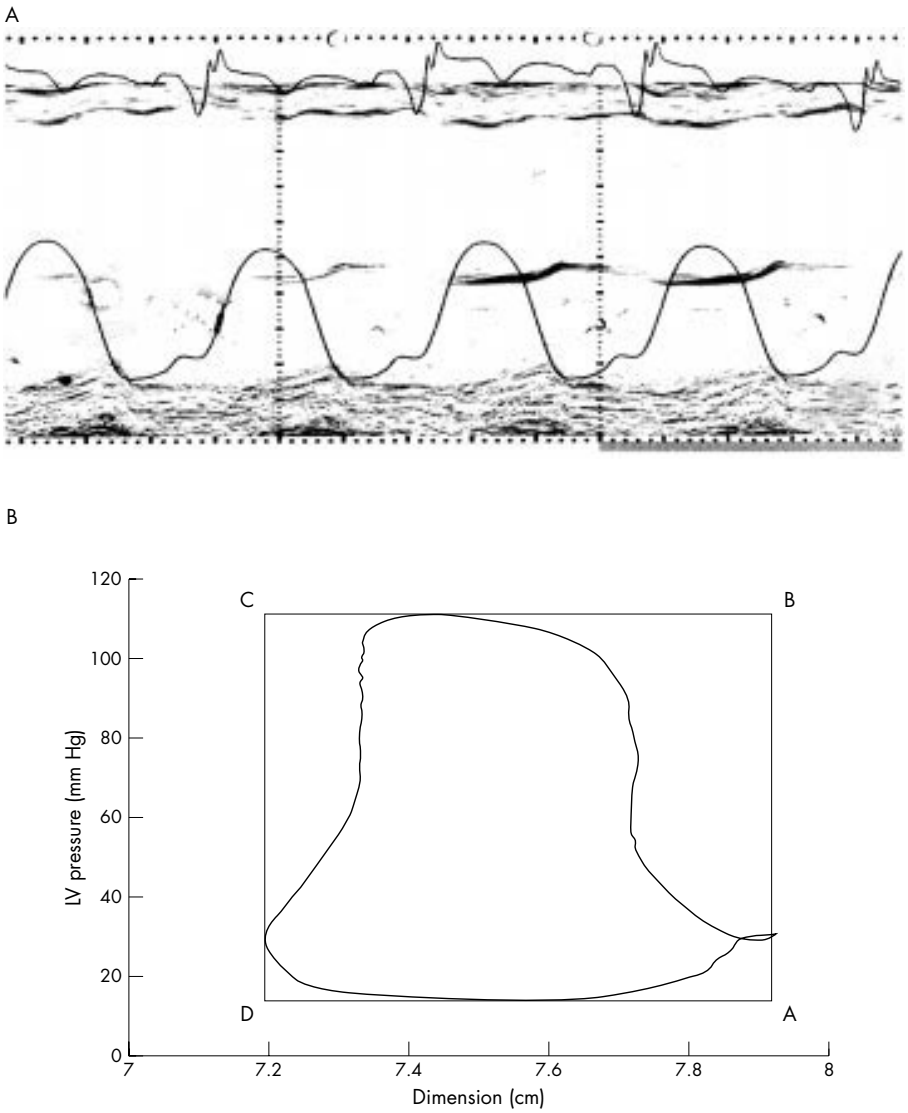

Figure 1 (A) M mode echocardiographic trace and simultaneously acquired left ventricular pressure trace. The pressure waveform and endocardial borders are then digitised to generate a pressure dimension loop. There is no active contraction in this segment during maximal pressure generation and contraction occurs during pressure reduction, indicating incoordination. (B) The inner line is the actual loop generated for that segment. Rectangle ABCD represents the ideal pressure dimension loop for the segment studied. AB = isovolumic contraction, BC = period of active contraction, CD = isovolumic relaxation, and DA = period of filling. Area of actual loop to ideal loop is the cycle efficiency. Cycle efficiency in this example was 64%.

in the right atrial appendage and another in the right ventricle (RV). The pacing lead was positioned at either the RV apex or the RV outflow tract (RVOT), depending on the site that was to be paced. A 7 French AL2 catheter (Bard, Billerica) was used to engage the coronary sinus os and an angiogram was then taken (hand injection, approximately 20 ml of Visipaque contrast agent) to help direct the placement of the LV electrode. A 0.010 inch guidewire (ACS, California, USA) was then advanced to the lateral marginal vein and a 3.2 French octapolar catheter (Cardima Tracer, Freemont, California, USA) was advanced over the wire for LV epicardial pacing between the base and apex.

Pacing protocol

Pacing in VDD mode (atrial sensing, ventricular pacing) was undertaken at the five sites in random order. The atrioventricular delay was programmed to 100 ms (always less than the patient's PR interval). Capture without fusion was ensured by further reducing the atrioventricular delay and looking for any change in ECG morphology in all subjects. Data were recorded after five minutes of pacing. Pacing was then suspended for five minutes followed by pacing in another site randomly chosen by random number generation. This process was repeated until all sites were paced. Reliable capture was verified by QRS duration and morphology change. The sites paced were RV apex, RVOT, mid-posterolateral LV through the coronary sinus, LV and RVOT simultaneously, and LV and RV apex simultaneously. The RVOT position was verified with a 12 lead ECG and fluoroscopy. All single site pacing was bipolar and biventricular pacing was bipolar tip to tip (LV cathode, RV anode). Atrial and ventricular pacing thresholds were then measured and pacing was performed at twice the diastolic pacing threshold. The best achieved measurement obtained from either the RV apex or the RVOT (RV) was then compared with the best achieved parameter with biventricular pacing (either LV and RVOT or LV and RV apex) and with LV pacing alone.

Data acquisition

A Hewlett-Packard 750 ultrasound imaging system (Hewlett-Packard, Palo Alto, California, USA) was used to image the LV.

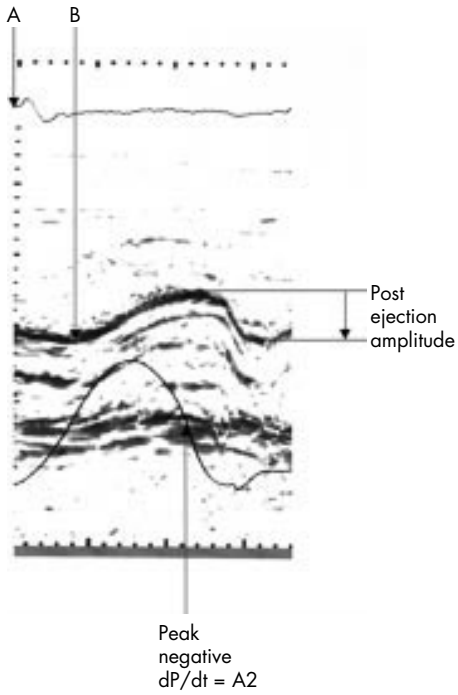

Figure 2 Long axis movement of the left ventricle during contraction is inward apical movement towards the mitral valve. A = q wave, B = start of inward motion of long axis. After closure of the aortic valve (A2) the long axis moves outwards from the mitral valve and the excursion is the postejection long axis amplitude.

Two operators determined a consistent plane independently at the start of each study. The pressure trace from the Millar LV catheter was displayed on the same channel as the M mode echocardiogram. All traces were recorded on paper at a speed of 100 mm/s with a superimposed ECG. Two dimensional guided M mode recordings of LV minor axis using leading edge methodology were obtained from the standard lateral parasternal window with the patient in the prone position. LV pressure was recorded simultaneously (fig 1A).

LV long axis M mode was recorded from the apical four chamber view with the cursor positioned at the left and septal angles of the mitral ring.[9] Postejection amplitude was measured as the amplitude of movement towards the ventricular apex occurring after peak –dP/dt (a surrogate for the aortic component of the second heart sound (A2); fig 2). Time to onset of inward motion was measured from the Q wave on the ECG to the start of inward motion. Transmitral filling velocities were obtained using the transducer in the pulsed wave mode from the apical four chamber view with the sample volume at the tips of the mitral leaflets.

Generation of pressure dimension loops and assessment of cycle efficiency

A paper printout of the M mode echocardiogram showing LV dimension and the simultaneous LV pressure trace was scanned by a Hewlett-Packard ScanJet 3c scanner. Continuous lines representing the contours of the LV posterior wall, the LV septal endocardium, and the LV pressure trace, plus three time points and two reference points, were manually drawn in Paint Shop Pro 5 software version 5.01 (Jasc Software, Inc, Eden Prairie, Minnesota, USA). Each bitmap file was then processed using software written in house. The speed of the paper recording and calibration of the LV pressure and dimension were given as input parameters to calibrate these in real physical units.[10]

Statistical analysis

The null hypothesis stated that there was no difference in the effect of biventricular pacing, LV pacing, and RV pacing on ventricular coordination, long axis function, and ventricular filling time. To determine the number of subjects needed, power calculations were based on studies already published at the inception of the project.[4 5] From estimates of mean and SD, 18 patients would be needed to determine the power of the difference between variables.

The best achieved measurement obtained from RV pacing was then compared with the best achieved measurement with biventricular pacing and with LV pacing alone. Each patient was used as his or her own control. Data are presented as mean (SD). The variable to be changed for each patient was pacing site. The effect of pacing site for each set of data for the continuous variable was determined by a two way repeated measures analysis of variance to compare differing pacing sites, followed by Tukey's post hoc analysis to compare the effect at specific sites. Correlation was determined by Pearson's test. The statistical software used was SPSS version 7.5 (SPSS Inc, Chicago, Illinois, USA).

Table 1 Baseline clinical characteristics of patient study group

Patient	Age (years)	LVEDD (mm)	SF (%)	PR interval (ms)	QRS interval (ms)	Aetiology
1	76	62	21	192	184	Hypertension
2	63	58	19	172	149	Alcohol
3	72	81	6	224	167	Idiopathic
4	46	84	7	176	152	IHD
5	68	72	17	164	161	Idiopathic
6	56	53	21	280	145	Idiopathic
7	69	76	11	224	143	IHD
8	73	55	20	184	161	Idiopathic
9	55	75	9	198	163	Idiopathic
10	76	62	8	212	182	Idiopathic
11	68	79	7	208	200	Idiopathic
12	66	73	11	171	165	Idiopathic
13	55	61	17	144	151	Idiopathic
14	64	76	11	200	138	Idiopathic
15	49	76	13	210	140	IHD
16	51	75	12	176	208	Idiopathic
17	54	73	8	180	156	Alcohol
18	49	68	9	182	186	Idiopathic
Mean	62	71	13	194	164	
SD	10	10	5	31	24	

IHD, ischaemic heart disease; LVEDD, left ventricular end diastolic dimension; SF, shortening fraction.

Table 2 Cycle efficiency (%) at each pacing site

Patient	Baseline	RV	LV	Bi-ventricular	Premature activation
1	78	80	70	81	N
2	58	80	83	78	N
3	70	50	23	25	Y
4	78	86	82	84	N
5	68	87	77	81	N
6	67	70	86	83	N
7	72	86	57	80	N
8	94	80	47	79	Y
9	70	80	85	49	N
10	64	81	79	83	N
11	81	78	63	83	N
12	68	80	46	44	Y
13	72	80	72	83	N
14	77	76	75	69	Y
15	60	60	55	61	N
16	35	77	44	50	N
17	57	83	78	81	N
18	88	80	64	79	N
Mean	70	77*	66	71	
SD	16	9	17	17	

LV, left ventricle; N, no; RV, right ventricle; Y, yes. *p<0.05 compared with baseline and other paced sites.

RESULTS

Patient information and baseline data

Twenty three patients gave their consent to take part in the study and were taken to the catheterisation laboratory. All patients enrolled were in sinus rhythm. All sites were paced in 15 patients, four sites in three patients, two sites in two patients in whom LV pacing was technically not possible, and one site in one patient who developed transient pulmonary oedema. Patients in whom it was not possible to pace more than three sites were excluded from the analysis (n = 5). Therefore, 18 patients completed the study for data analysis.

Table 1 outlines baseline patient characteristics. Prescribed medications were loop diuretics (96%), angiotensin converting enzyme inhibitors or angiotensin receptor blockers (92%), digoxin (44%), nitrates (36%), spironolactone (25%), and β blockers (24%). The mean baseline PR interval was 194 (31) ms and QRS duration was 164 (24) ms. LBBB was present on the surface ECG in all the patients. The mean baseline LV end diastolic dimension was 71 (10) mm and the shortening fraction was 13 (5)%. The overall mean cycle efficiency was 67 (16)%. A typical mean cycle efficiency in healthy people is 83 (8)%. Baseline postejection amplitude was 9 (3) mm. Mean baseline filling time was 243 (77) seconds.

Changes in cycle efficiency with pacing

The response of cycle efficiency to pacing varied within and between patients (table 2). The best achievable cycle efficiency was associated with biventricular pacing in five patients, with LV pacing in two patients, and with RV pacing in seven patients. In four patients no pacing site improved cycle efficiency above baseline cycle efficiency. Figure 3 shows examples of changes in cycle efficiency with pacing.

Only RV pacing (cycle efficiency 77 (9)%) led to a significant increase in baseline cycle efficiency (70 (16)%). Patients were divided into two groups based on the baseline cycle efficiency.[7] Patients in group 1 had baseline incoordination (cycle efficiency ⩽ 72%, n = 12) and those in group 2 had baseline coordination (cycle efficiency > 72%, n = 6). RV pacing led to a significant increase in baseline cycle efficiency in group 1, whereas LV pacing worsened cycle efficiency in group 2 (table 3).

Premature activation of the ventricle

Premature activation (fig 4) was defined as the peak inward movement of the minor axis posterior wall > 20 ms before closure of the aortic valve (peak −dP/dt). This pattern was never seen at baseline or with RV pacing, but was seen with LV and biventricular pacing in four patients each (table 2).

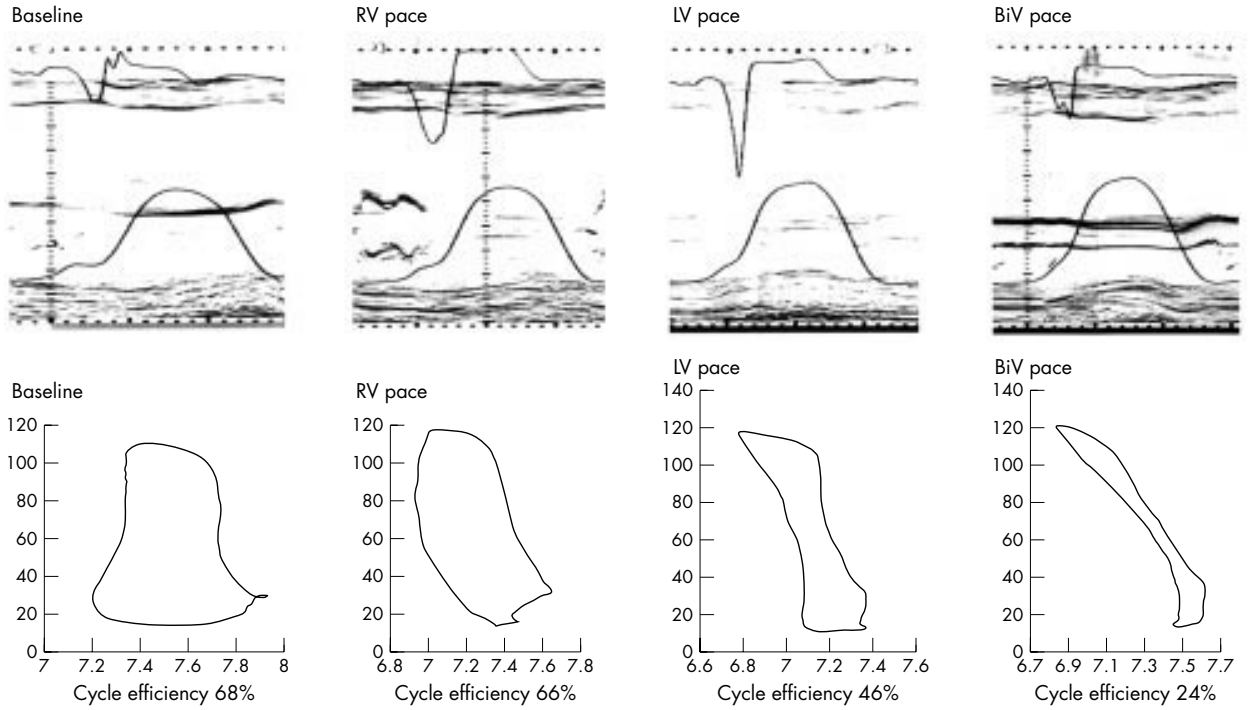

Figure 3 Four pressure dimension loops are shown from the same patient. At baseline and with right ventricular (RV) pacing there was mild incoordination. With left (LV) or biventricular (BiV) pacing incoordination was greater—note the increase in cavity size while the ventricular pressure generated was still relatively high, and when the ventricular pressure was low at the end of the recording the posterior wall moved inwards.

Table 3 Cycle efficiency (%) by pacing site compared according to cycling efficiency at baseline (group 1, ≤72%; group 2, >72%)

Group	n	Baseline	RV pacing	LV pacing	Biventricular pacing
All	18	70 (16)	77 (9)*	66 (17)†	71 (17)†
1	12	63 (10)	76 (11)*	65 (20)†	67 (20)
2	6	83 (7)	80 (4)	67 (12)*	79 (5)
1, excluding patients with premature activation	10	62 (11)	78 (8)*	72 (15)*	73 (14)*

Data are mean (SD). *p < 0.05 *v* baseline; †p < 0.05 *v* RV.

Table 4 Postejection long axis amplitude (mm) by pacing site compared according to cycling efficiency at baseline (group 1, ⩽ 72%; group 2, > 72%)

Group	n	Baseline	RV pacing	LV pacing	Biventricular pacing
All	18	9.0 (3.0)	9.4 (3.0)†	7.6 (2.0)*	8.8 (3.0)
1	12	9.3 (3.3)	9.8 (3.6)	8.0 (2.4)	9.4 (3.0)†
2	6	8.4 (0.5)	8.6 (0.9)†	6.8 (1.0)*	7.4 (2.7)

Data are mean (SD).
*p < 0.05 *v* baseline; †p < 0.05 *v* LV.

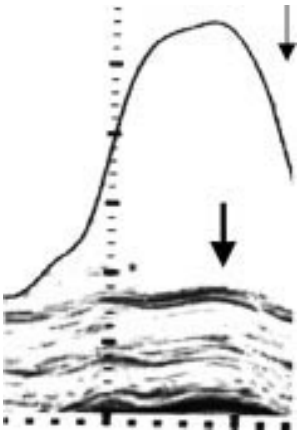

Figure 4 Left ventricular pacing causing premature activation. A "double hump" can be seen, which is the echocardiographic feature of abnormal premature activation. The thin arrow indicates peak –dP/dt, a surrogate for closure of the aortic valve. The thick arrow indicates the point of peak inward motion. Before this inward motion, earlier inward motion also occurs, indicated by the asterisk.

Premature activation was associated with incoordination: mean cycle efficiency (in patients with premature activation) with LV pacing 48 (21)% and with biventricular pacing 54 (24)%. Removal of the four patients with premature activation from group 1 improved cycle efficiency at all pacing sites in the remaining patients compared with baseline (table 3).

Postejection long axis amplitude

The amplitude of long axis motion after peak –dP/dt (a surrogate for the aortic component of the second heart sound (A2); fig 2), was reduced with LV pacing (7.6 (2.0) mm) compared with baseline (9.0 (3.0) mm). The postejection long axis amplitude (after A2) was consistently less with LV pacing regardless of whether there was baseline incoordination (table 4).

Table 5 Delay in onset of long axis inward movement (ms) from the Q wave at each pacing site in patients with and those without premature activation of the LV posterior wall

Premature activation	Baseline	RV pacing	LV pacing	Biventricular pacing
Yes	84 (4)	85 (30)	105 (26)	145 (100)*
No	78 (34)	76 (40)	70 (32)	63 (30)

Data are mean (SD). *p < 0.05 *v* baseline.

Time of onset of inward motion of the long axis

There was no significant difference in the timing of the onset of long axis contraction between baseline and pacing sites (fig 2). This was similar in patients with baseline incoordination (baseline 74 (38) ms, RV 71 (41) ms, LV 79 (41) ms, biventricular 98 (27) ms; ns).

The onset of long axis activation was delayed in patients with pacing induced premature activation (as defined above) but not in those without (table 5).

LV filling characteristics

Pacing at all sites significantly increased filling time from baseline (table 6). The effect on filling time of pacing at the various sites was similar in patients with a baseline filling time⩽ 200 ms and with> 200 ms. Deceleration time was similar for all pacing sites.

DISCUSSION

In heart failure, QRS duration is often increased[11] and this disturbed activation makes ventricular function incoordinate. Long axis shortening, manifest by the onset of left atrioventricular ring motion, is delayed, and isovolumic contraction and relaxation times are both increased.[12] During ventricular pacing a wave of activation spreads away from the pacing site and creates an array of temporally dispersed sequences of contraction and relaxation results in a complex interaction between individual fibres, which is difficult to assess. Studies have investigated the effects of pacing on electromechanical sequence,[13] pressure volume loops,[14] and the interventricular relation.[15] We studied the effect of pacing on intraventricular coordination, long axis function, and ventricular filling characteristics.

Table 6 Filling time (ms) characteristics by pacing site

	n	Baseline	RV pacing	LV pacing	Biventricular pacing
All patients	18	243 (77)	318 (109)*	327 (124)*	344 (140)*
Baseline filling time ⩽200 ms	8	175 (19)	232 (35)*	240 (35)*	253 (56)*
Baseline filling time >200 ms	10	290 (65)	377 (96)*	379 (130)*	402 (159)*
Deceleration time (ms)	18	90 (27)	123 (55)*	113 (75)	121 (68)

*p < 0.05 *v* baseline.

Ventricular coordination

The advantage of pressure dimension loops is that any changes in dimension during the isovolumic periods are easily visualised. Incoordination, represented by loss of the optimal relation between pressure and dimension, is reflected in a reduction in mechanical efficiency resulting in reduced efficiency of energy transfer to the circulation.[7] This is measured as a reduction in the ratio of observed external work to the maximum possible for a ventricle working over the same range of pressure and dimension—that is, the ratio of the area of the generated loop to that of the ideal loop. We dichotomised the group based on baseline cycle efficiency with the cut off at 72%. This was chosen as a physiological value based on the original work on coordination.[7] Previously data have been reported on cycle efficiency in groups with valve disease leading to heart failure[16 17] or requiring revascularisation without heart failure.[18]

In our patients the site of pacing leading to the greatest increase of cycle efficiency varied. However, RV pacing improved coordination significantly more than LV or biventricular pacing, especially in patients with baseline incoordination. This was unexpected considering the widely held view that biventricular pacing leads to improved coordination or so called "resynchronisation". However, the normal conduction system after the bundle of His divides to the right and left branching bundles. The right bundle persists to the apex of the RV before dividing into the terminal Purkinje fibres, whereas in the LV free wall the terminal Purkinje fibres distant from the left bundle are present. Therefore, pacing at the RV is more likely to cause conduction of the organised right bundle with further conduction along the usual conduction pathways. In contrast, with pacing from the LV free wall, pacing induced myogenic electrical propagation may actually be more delayed. This may explain why premature activation seen with LV or biventricular pacing may be associated with a delay in the onset of long axis contraction. The effect of pacing perhaps depends on the relative proximity of organised conducting tissue. We targeted the posterolateral branch of the LV—the site usually selected when implanting permanent systems—because this is usually the site of greatest delay in mechanical activation with LBBB.

The unusual pattern of premature activation of the posterior wall of the LV with pacing is seen only with LV or biventricular pacing. Although there were few patients in whom premature activation occurred, when they were excluded from the comparison of the effect of pacing site on cycle efficiency, the cycle efficiency of all pacing sites increased to a similar degree. Biventricular pacing may cause up to three electrical wavefronts: endogenous, from the RV, and from the LV. Future programming to alter RV and LV timing may allow greater improvement of coordination with biventricular pacing.

Filling characteristics and long axis characteristics

Pacing led to similar improvement in filling time regardless of site. Therefore, the site of pacing is not the main determinant of alteration in filling time, and filling characteristics did not relate to changes of coordination.

Long axis amplitude (after A2) was reduced with LV pacing. This was not altered even when patients with premature ventricular activation were excluded, implying that the reduction in amplitude was probably not related to incoordination. Decreased LV filling may explain a reduction in long axis amplitude but in this small population no such relation was determined. Although overall no difference in time to the onset of inward motion of the long axis after the Q wave on the ECG was found, paradoxically a consistent delay in the onset of inward motion was seen in patients in whom premature activation occurred in the short axis. It seems that the premature activation of the short axis fibres causes contraction to be "desynchronised" from the long axis fibres contributing to incoordination, a novel finding.

Study design limitations

Patients with normal QRS duration were not studied, because those with LBBB are most likely to benefit from LV based pacing and to have a biventricular pacemaker for heart failure implanted permanently. Patients with more severe heart failure (NYHA IV), right bundle branch block, or non-specific intraventricular conduction delay and atrial fibrillation were not studied and the results may not apply to them. Atrioventricular delay has been shown to be a less important influence than site,[19 20] and we selected 100 ms based on our own experience and previously published data.[21 22] It was not possible to pace all patients recruited at all sites because of the vulnerable health of several patients, but this was considered in the statistical analysis. Variables examined in this study were assessed only at rest and effects of pacing on ambulatory patients may not be the same. Small sample size could have produced a type II statistical error and greater caution should be applied to the subgroup analysis.

Conclusions

Despite the widely held view that biventricular pacing causes resynchronisation, LV coordination has never been directly measured. In this study we have shown that cycle efficiency can be improved by pacing but that the site of pacing associated with greatest improvement differs greatly between patients. The only consistent benefit we could show was with RV based pacing in those with a baseline incoordinate ventricle. In a subset of patients, LV based pacing induced premature short axis contraction and this was associated with a delayed onset of long axis contraction. This, therefore, seems to induce asynchrony of short and long axis LV fibres, which was associated with worsening incoordination.

The clinical significance of these findings remains to be defined, but these observations challenge the view that biventricular pacing induces beneficial "resynchronisation" in all patients. Attempts to predict the electromechanical effect of pacing will likely be crucial in selecting those patients who would benefit the most from this treatment.

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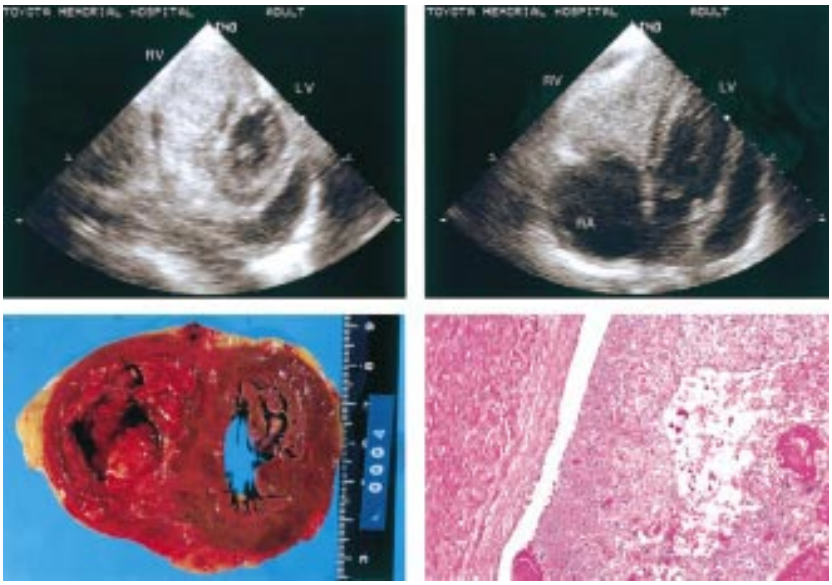
IMAGES IN CARDIOLOGY

Single large metastatic tumor growing progressively and occupying right ventricular cavity

A 70 year old woman was admitted because of breathlessness and chest discomfort. An ovarian tumour had been resected six months before admission, and was diagnosed as a mature cystic teratoma with squamous cell carcinoma. On physical examination, hypotension, peripheral oedema, and jugular vein dilatation were evident. Transthoracic echocardiography revealed a high echoic large mass in the right ventricular (RV) cavity, an enlarged right atrium (RA), and pericardial effusion (top left, parasternal short-axis view; top right, apical four chamber view; LV, left ventricle). No masses were detected elsewhere. Contrast computed tomographic scans showed a large filling defect in the RV cavity. The patient died of developmental cardiogenic shock two weeks later.

Necropsy revealed that a cardiac tumour arising from the RV free wall occupied the RV cavity almost completely (bottom left). Pathological diagnosis of the tumour was squamous cell carcinoma, suggesting metastasis of the ovarian cancer previously resected (bottom right). Surprisingly, metastatic lesions were not found in the other major organs, including brain, lung, liver, kidney, as well as adrenal gland. The pericardium is the most common site of cardiac metastasis of malignant tumours and the resultant cardiac tamponade is the most frequent cause of hemodynamic compromise. However, the involvement in endocardium is extremely rare. In contrast,



only a few cases with intracavitary metastatic lesions causing haemodynamic compromise have been reported. In the present case, the sole metastatic tumor grew progressively without any other metastatic lesions in the whole body.

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