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Differing effects of right ventricular pacing and left bundle branch block on left ventricular function

Han B Xiao, Stephen JD Brecker, Derek G Gibson

Abstract

***Objective*—To compare the different effects of right ventricular pacing and classic left bundle branch block on left ventricular function.**

***Design*—Retrospective and prospective study of 48 patients by electrocardiography, and M mode, cross sectional, and Doppler echocardiography.**

***Setting*—A tertiary cardiac referral centre.**

***Patients*—48 patients (age range 21 to 89 years, 15 women), 24 with a VVI pacemaker implanted and 24 with classic left bundle branch block. Functional mitral regurgitation was present in all those with right ventricular pacing and 22 of those with left bundle branch block.**

***Results*—Age, RR interval, and left ventricular size were similar in the two groups, as were conventional measurements of overall systolic function: shortening fraction and pre-ejection and aortic ejection times. In right ventricular pacing, however, QRS duration ($p < 0.01$) and electromechanical delay were much longer ($p < 0.001$), whereas the time intervals from onset of mitral regurgitation to aortic opening (contraction time) and from A 2 to the end of mitral regurgitation (relaxation time) were consistently shorter ($p < 0.01$) than corresponding values in patients with left bundle branch block. Reversed splitting of the second heart sound was much commoner in left bundle branch block ($p < 0.02$), and only these patients showed an early systolic ventricular septal contraction. Its onset followed the initial deflection of the QRS complex by 40 (15) ms and preceded mitral regurgitation by a small but consistent interval of 10 ms ($p < 0.01$). The onset of posterior wall thickening was synchronous with the onset of mitral regurgitation in right ventricular pacing but much later ($p < 0.01$) in patients with left bundle branch block. The extent of incoordinate wall motion measure as relative dimension change during pre-ejection and isovolumic relaxation period was much greater ($p < 0.01$) in left bundle branch block. These major differences were not altered by left ventricular cavity size in either group, nor by the presence of previous left bundle branch block in patients who were subsequently paced.**

***Conclusions*—The left ventricle seems to be activated much more rapidly with right ventricular pacing than with left bundle branch block. This applies even when left bundle branch block is present before pacing. Electromechanical delay, contraction and relaxation times, and extent of incoordinate ventricular wall motion differ strikingly between the two conditions. The use of right ventricular pacing as an experimental model of left bundle branch block in humans must be re-examined.**

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Right ventricular pacing has been used to simulate the mechanical effects of left bundle branch block in experimental models and clinical studies.[1 2] Although clearly different in activation pathway and electromechanical delay,[3 4] the mechanical effects of the two modes of activation have not been systematically compared. We have, therefore, investigated the differences and similarities between the two, by a series of non-invasive tests.

Patients and methods

PATIENTS

We studied 48 patients; 24 of these had a VVI pacemaker implanted (with the electrode tip in the right ventricular apex) for complete heart block and 24 had normal atrioventricular conduction but classic left bundle branch block. The diagnostic criteria of left bundle branch block included QRS duration

Table 1 Clinical features

Index	*RVP (total n = 24)*	*LBBB (total n = 24)*	*p Value*
Age (mean (SD) yr)	64 (16)	58 (13)	NS
RR interval (mean (SD) ms)	825 (100)	750 (170)	NS
QRS duration (mean (SD) ms)	190 (20)	150 (15)	< 0·01
Diagnosis (n):			
Isolated conduction disturbance	8	6	
Aortic valve disease	7	6	
Dilated cardiomyopathy	5	7	
Coronary arterial disease	2	4	
Scleroderma	2	0	
Muscular dystrophy	0	1	
Functional mitral regurgitation	24	22	NS
Reversed splitting of the second heart sound	3	16	< 0·001

RVP, right ventricular pacing, LBBB, left bundle branch block.

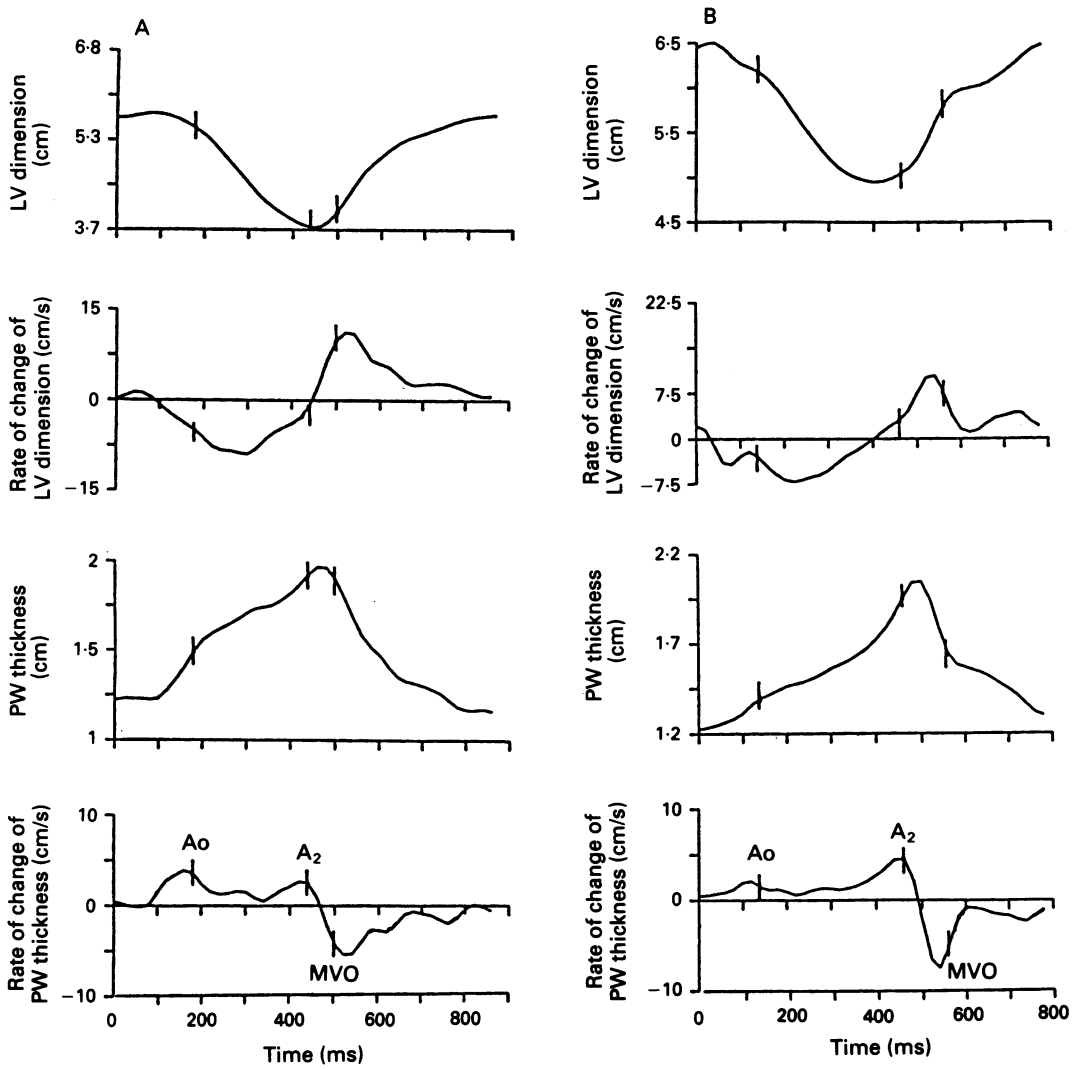

Figure 1 Digitised traces of left ventricular M mode echocardiograms from a patient with right ventricular pacing (A) and a patient with classical left bundle branch block (B). Left ventricular (LV) dimension change and rate of change, and posterior wall (PW) thickness and rate of change are shown. Zero time corresponds to onset of QRS complex; A_o, aortic valve opening; A_2, aortic valve closure; MVO, mitral valve opening. Differences between the patients in the dimension change during pre-ejection and isovolumic relaxation periods are shown.

⩾120 ms, absent septal q waves on the left precordial leads and absent R waves on the right precordial leads on a 12 lead electrocardiogram. Classic left bundle branch block had previously been present in 10 of the patients studied during right ventricular pacing. Table 1 shows clinical details. In patients with right ventricular pacing, measurements were only taken from the cardiac cycles with a PR interval > 200 ms. Only one patient had previously had a documented anterior myocardial infarction.

ECHOCARDIOGRAPHY

Cross sectionally guided M mode echocardiograms were recorded with the patients in the left lateral position, by a 3·0 MHz mechanical transducer with an ATL Mark III system or 2·5 MHz phased array transducer with a Hewlett Packard Sonos 500 system. They were recorded photographically with simultaneous electrocardiograms and phonocardiograms at a paper speed of 10 cm/s. The left ventricular M mode echocardiogram was taken just below the tips of the mitral leaflets, with endocardial echoes of both sides of the ventricular septum and the posterior wall clearly visible. We looked for early systolic transient leftward motion of the ventricular septum. End diastolic dimension was measured at the onset of the QRS complex and end ejection dimension at the onset of the first high frequency vibration of the aortic component of the second heart sound (A_2). Shortening fraction was then derived. Aortic ejection time was ascertained directly from the aortic echocardiogram or the leading edge of Doppler artefacts,[5] which were also used to identify the aortic component of the second heart sound and thus to assess whether splitting was normal or reversed.

We digitised the left ventricular M mode echocardiogram along with the times of aortic valve opening and closure and the initial separation of the mitral leaflets.[6] We defined the time interval between the onset of the QRS complex or pacing spike to aortic opening as pre-ejection period and A_2 to mitral cusp separation as isovolumic relaxation time. We plotted left ventricular dimension, posterior wall thickness, and their rates of change (fig 1). From these digitised traces we mea-

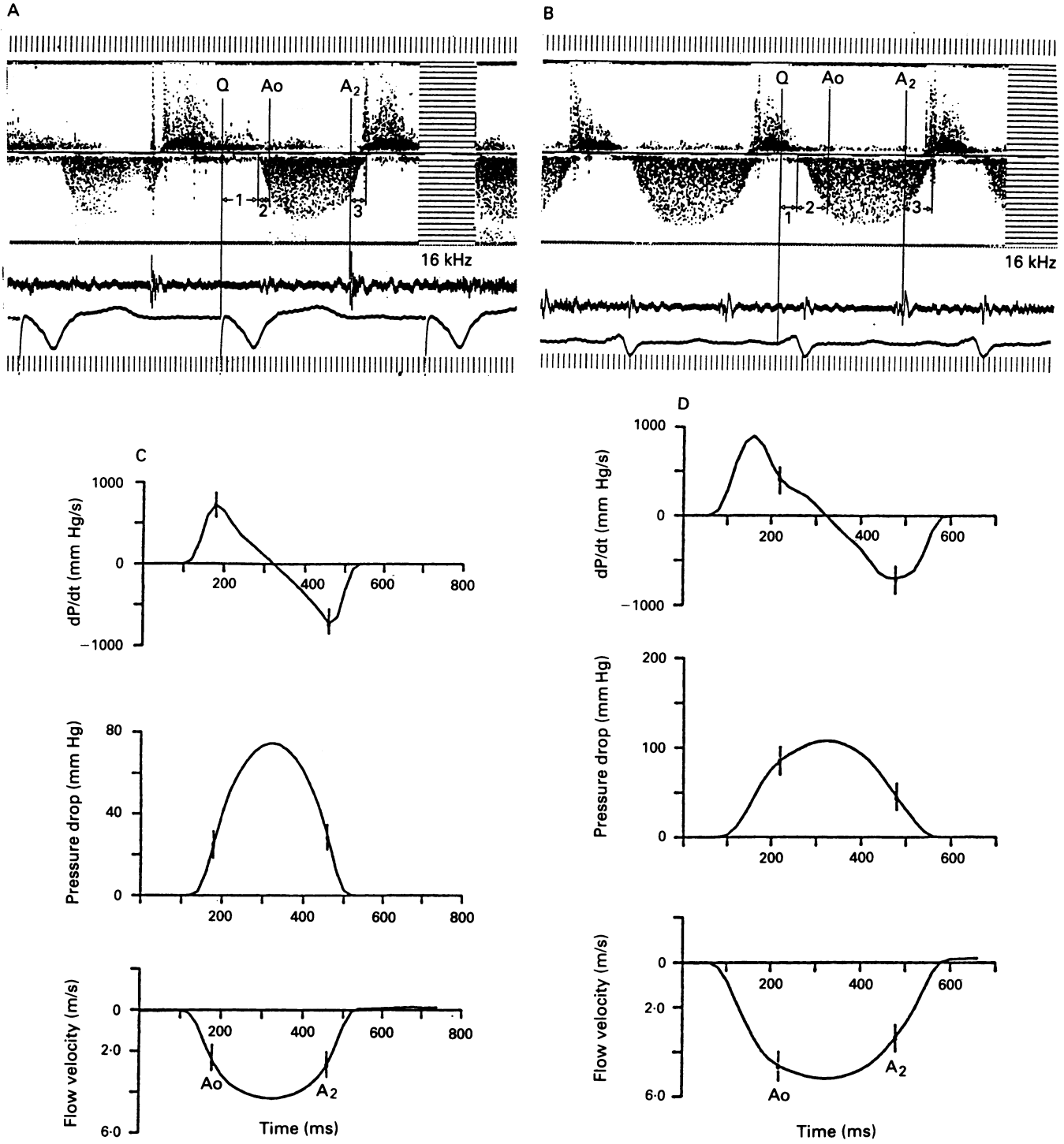

Figure 2 Continuous wave Doppler recording of functional mitral regurgitation from a patient with right ventricular pacing (A) and a patient with classic left bundle branch block (B) and the corresponding digitised traces (C and D). The Q wave or zero times on the plots represent onset of QRS complex; A_o, aortic valve opening; A_2 aortic valve closure. Differences in electromechanical delay (1), in contraction (2), and in relaxation (3) times are shown.

sured: (*a*) Any dimension change during the pre-ejection and isovolumic relaxation periods relative to the total dimension change in the cardiac cycle: (*b*) peak rates of dimension fall in systole and of increase in diastole: (*c*) peak rates of thickening and thinning of the left ventricular posterior wall.

Doppler traces were recorded with a 2 MHz pencil transducer and Doptek equipment at a paper speed of 10 cm/s. Simultaneous lead II electrocardiograms and phonocardiograms were also recorded. Doppler recording of functional mitral regurgitation was made in continuous wave mode, with a 1 kHz filter, and the measurements taken were[7]: (*a*) QRS duration: (*b*) electromechanical delay measured from the onset of the pacing signal or native QRS complex to onset of mitral regurgitation. No patient had a presystolic component to the mitral regurgitation signal: (*c*) overall duration of mitral regurgitation: (*d*) left ventricular contraction

Table 2 Characteristics of M mode echocardiograms (mean (SD))

Index	*RVP*	*LBBB*	*p Value*
Left ventricular end diastolic dimension (cm)	6·1 (1·2)	6·5 (1·3)	NS
Total dimension change (cm)	1·5 (0·5)	1·1 (0·5)	< 0·05
Shortening fraction (%)	23·5 (10·5)	19 (7·0)	NS
Abnormal septal motion	0	18	< 0·001
Dimension change in pre-ejection period (%)	7·8 (7·0)	29·5 (20)	< 0·01
Dimension change in isovolumic relaxation period (%)	10·0 (7·0)	21·0 (15)	< 0·01
Peak rate of dimension decrease (cm/s)	8·5 (2·5)	6·4 (2·5)	< 0·01
Peak rate of dimension increase (cm/s)	12·0 (4·5)	8·5 (3·5)	< 0·01
Peak rate of posterior wall thickening (cm/s)	3·5 (1·3)	3·7 (2·0)	NS
Peak rate of posterior wall thinning (cm/s)	6·3 (3·0)	5·0 (2·5)	NS

RVP, right ventricular pacing, LBBB, left bundle branch block.

Table 3 Characteristics of mitral regurgitation and left ventricular pressure pulse (mean (SD))

Index	*RVP*	*LBBB*	*p Value*
Electromechanical delay (Q to onset MR) (ms)	115 (25)	45 (20)	< 0·001
Pre-ejection time (ms)	185 (30)	170 (25)	NS
Overall duration of MR (ms):	415 (40)	480 (65)	< 0·001
Contraction time (ms)	70 (25)	120 (25)	< 0·001
Aortic ejection time (ms)	265 (35)	245 (60)	NS
Relaxation time (ms)	80 (20)	115 (25)	< 0·01
Peak pressure drop (mm Hg)	85 (20)	80 (20)	NS
Peak +dP/dt (mm Hg/s)	765 (230)	680 (190)	NS
Peak −dP/dt (mm Hg/s)	595 (215)	595 (200)	NS
Onset MR to onset of posterior wall thickening (ms)	3 (35)	80 (25)	< 0·001
Onset of MR to peak +dP/dt (ms)	55 (25)	80 (40)	< 0·05

MR, functional mitral regurgitation.

time, from the onset of mitral regurgitation to aortic valve opening: (*e*) aortic ejection time, from aortic opening to its closure (A_2): (*f*) left ventricular relaxation time, from A_2 to the end of mitral regurgitation: (*g*) time interval from the onset of mitral regurgitation to the onset of septal and posterior wall motion.

We digitised the mitral regurgitation signals along with time and velocity calibrations, and thus derived the time course of pressure drop from left ventricle to left atrium and its rate of change (fig 2) with the modified Bernoulli equation. From these plots, we measured (a) peak left ventricle to left atrium pressure drop, (b) peak rate of pressure rise (+dP/dt) and fall (−dP/dt), (c) times to peak +dP/dt, with the onset of the QRS complex and mitral regurgitation as zero.

DATA ANALYSIS

The average values were taken from three successive beats and expressed as mean (SD) for group measurements. Differences between mean values were assessed by the unpaired Student's *t* test and in group frequencies by Fisher's exact probability test.

Table 4 Characteristics of M mode echocardiograms in patients with right ventricular pacing (mean (SD))

Index	*LV size ≤ 5·6 cm (n = 11)*	*LV size > 5·6 cm (n = 13)*
Total dimension change (cm)	1·5 (0·4)	1·5 (0·7)
Dimension change in pre-ejection period (%)	10·3 (6·7)	5·7 (7·1)
Dimension change in isovolumic relaxation period (%)	7·4 (7·5)	12·5 (7·0)
Peak rate of dimension fall (cm/s)	8·5 (2·8)	8·3 (3·0)
Peak rate of dimension increase (cm/s)	12·0 (3·8)	12·0 (5·1)
Peak rate of posterior wall thickening (cm/s)	3·0 (0·8)	3·8 (11·5)
Peak rate of posterior wall thinning (cm/s)	6·3 (3·4)	6·5 (3·0)

LV, left ventricle. There were no statistically significant differences.

Results

GENERAL

Table 1 shows that age, RR interval, and left ventricular size were similar in the two groups, as were conventional measurements of overall systolic function: shortening fraction, pre-ejection, and aortic ejection times. QRS duration was significantly longer ($p < 0·01$) in right ventricular pacing, and reversed splitting of the second heart sound was much more often present in patients with left bundle branch block ($p < 0·001$).

PRE-EJECTION PERIOD

Tables 2 and 3 show that the mean pre-ejection time was similar in the two groups, as was peak rate of rise of left ventricular pressure (+dP/dt). In right ventricular pacing, however, electromechanical delay was much longer ($p < 0·001$), whereas the time from onset of mitral regurgitation to aortic opening (contraction time) was consistently shorter ($p < 0·01$) than corresponding values in patients with left bundle branch block (fig 2).

Regional wall motion also differed between the two groups. Only the patients with left bundle branch block showed an early systolic ventricular septal contraction; its onset followed the initial deflection of the QRS complex by 40 (15) ms. The onset of posterior wall thickening started at a similar time in both groups with respect to the onset of the Q wave. It was synchronous with the onset of mitral regurgitation in right ventricular pacing, but was much later ($p < 0·01$) in patients with left bundle branch block. The onset of mitral regurgitation followed that of the septal dip by a small but consistent interval of 10 ms ($p < 0·01$). The extent of incoordinate wall motion, measured as the relative dimension change during the pre-ejection period, was much greater ($p < 0·01$) in patients with left bundle branch block than in those with right ventricular pacing.

EJECTION PHASE

Tables 2 and 3 show that aortic ejection time and the peak pressure drop from left ventricle to atrium were the same in both groups. Peak rate of dimension fall was greater in patients with right ventricular pacing ($p < 0·01$) although peak rate of posterior wall thickening was the same in both. In eight patients with left bundle branch block (but none in right ventricular pacing ($p < 0·05$) peak rate of wall thickening occurred after closure of the aortic valve—that is, during the period of isovolumic relaxation.

AFTER EJECTION

Tables 2 and 3 show that the time from A_2 to the end of mitral regurgitation (relaxation time) was greatly prolonged in patients with left bundle branch block compared with those with right ventricular pacing ($p < 0·001$). Peak rates of pressure fall (−dP/dt) and posterior wall thinning were the same in both groups, but peak rate of dimension increase was greater ($p < 0·01$) in patients with right

Table 5 Characteristics of mitral regurgitation and the left ventricular (LV) pressure pulse in patients with right ventricular pacing (mean (SD))

Index	LV size ≤ 5·6 cm (n = 11)	LV size > 5·6 cm (n = 13)	p Value
RR interval (ms)	860 (95)	795 (110)	NS
Electromechanical delay (Q to onset MR) (ms)	100 (20)	125 (15)	< 0·01
Pre-ejection time (ms)	175 (20)	190 (25)	NS
Overall duration of MR (ms):	420 (25)	410 (45)	NS
Contraction time (ms)	75 (20)	65 (25)	NS
Aortic ejection time (ms)	265 (30)	265 (40)	NS
Relaxation time (ms)	80 (15)	80 (20)	NS
Peak pressure drop (mm Hg)	98 (20)	75 (20)	< 0·05
Peak +dP/dt (mm Hg/s)	915 (265)	670 (140)	< 0·05
Peak −dP/dt (mm Hg/s)	675 (245)	540 (180)	NS
Onset MR to onset of posterior wall thickening (ms)	10 (35)	− 5 (35)	NS

MR, functional mitral regurgitation.

ventricular pacing and the extent of dimension change during isovolumic relaxation period was much greater in patients with left bundle branch block ($p < 0·01$).

THE EFFECT OF THE LEFT VENTRICULAR CAVITY SIZE

We divided each group of patients into two subgroups according to the left ventricular end diastolic dimension, comparing the patients with a left ventricular cavity size of greater than 5·6 cm with those in whom it was less. Tables 4 to 7 show that in patients with right ventricular pacing, electromechanical delay was shorter and peak pressure drop and its rate of increase were higher when left ventricular size was normal, whereas patients with left bundle branch block the contraction time and the dimension change during isovolumic relaxation were both slightly greater in those with a dilated left ventricle. Apart from these minor differences cavity size had no noticeable effect on any of the variables we measured.

THE EFFECT OF PREVIOUS LEFT BUNDLE BRANCH BLOCK IN RIGHT VENTRICULAR PACING

We compared the patients who had had left bundle branch block before pacemaker implantation with those who had not. Tables 8 and 9 show that QRS duration was longer in those with previous left bundle branch block, but there was no other difference between the two patient groups.

Table 6 Characteristics of M mode echocardiograms in patients with left bundle branch block (mean (SD))

Index	LV size ≤ 5·6 cm (n = 6)	LV size > 5·6 cm (n = 18)	p Value
Total dimension change (cm)	1·2 (0·4)	1·1 (0·5)	NS
Dimension change in pre-ejection period (%)	26·5 (13·8)	30·5 (21·3)	NS
Dimension change in isovolumic relaxation period (%)	13·0 (6·5)	24·0 (16·0)	< 0·05
Peak rate of dimension fall (cm/s)	6·7 (2·5)	6·3 (2·2)	NS
Peak rate of dimension increase (cm/s)	10·5 (4·5)	7·5 (3·0)	NS
Peak rate of posterior wall thickening (cm/s)	3·7 (1·5)	3·7 (2·0)	NS
Peak rate of posterior wall thinning (cm/s)	6·4 (3·0)	4·5 (2·0)	NS
Q to early systolic septal dip (ms)	45 (10)	37 (15)	NS

LV, left ventricle.

Table 7 Characteristics of mitral regurgitation and left ventricular (LV) pressure pulse in patients with left bundle branch block (mean (SD))

Index	LV size ≤ 5·6 cm (n = 6)	LV size > 5·6 cm (n = 18)	p Value
RR interval (ms)	765 (150)	745 (180)	NS
QRS duration (ms)	145 (8·5)	155 (15)	NS
Electromechanical delay (Q to onset MR) (ms)	55 (20)	45 (15)	NS
Pre-ejection time (ms)	170 (20)	170 (25)	NS
Overall duration of MR (ms):	470 (45)	485 (70)	NS
Contraction time (ms)	110 (10)	125 (30)	< 0·05
Aortic ejection time (ms)	250 (35)	240 (65)	NS
Relaxation time (ms)	110 (15)	115 (25)	NS
Peak pressure drop (mm Hg)	80 (15)	78 (20)	NS
Peak +dP/dt (mm Hg/s)	620 (75)	690 (200)	NS
Peak −dP/dt (mm Hg/s)	590 (125)	595 (215)	NS
Onset MR to onset of posterior wall thickening (ms)	85 (15)	80 (25)	NS

MR, functional mitral regurgitation.

Discussion

Despite the obvious differences in the pathways of left ventricular activation, right ventricular pacing has conventionally been used as an experimental model of left bundle branch block, possibly because of some resemblance in the surface electrocardiogram.[8] Its use in this way however, has not been systematically validated. A previous study noted that in patients with VDD pacing, electromechanical delay and filling time were both longer compared with patients who had left bundle branch block, although these differences were not pursued in detail.[3] Evidence increases for the deleterious mechanical effects of disordered activation on left ventricular function in patients with severe heart disease,[7] and it becomes possible that conventional indications for pacing might be extended in these circumstances.[9] Thus we have used a series of non-invasive techniques to undertake a formal study of the mechanical consequences of these two modes of activation.

Irrespective of the diagnosis, our patients were similar in terms of age, left ventricular size, and orthodox measures of systolic function including shortening fraction, pre-ejection period, ejection time, and peak rate of rise in left ventricular pressure. There were, however, major differences between the two patient groups. We confirmed that the early systolic septal dip was commoner in left bundle branch block than that in right ventricular pacing.[4] Although electromechanical delay, taken as the interval from the onset of the QRS complex to that of mitral regurgitation was longer in patients with right ventricular pacing, once contraction began, its overall duration was much less than in left bundle branch block. This was not because of any difference in ejection time, but because both the contraction and relaxation times were much shorter (fig 2). Also, incoordinate left ventricular wall motion, defined as dimension change before aortic opening or after mitral closure, was much more striking in patients with left bundle branch block. There were, therefore major differences, independent of cavity size, in left ventricular contraction pattern between the two modes of activation.

Not only were we able to detect these differences, we also gained information about

Table 8 Comparison of M mode echocardiograms between patients, with right ventricular pacing with and without previous left bundle branch block (LBBB) (mean (SD))

Index	Patients with previous LBBB (n = 10)	Patients without previous LBBB (n = 14)	p Value
Left ventricular end diastolic dimension (cm)	6·1 (1·3)	6·0 (1·2)	NS
Total dimension change (cm)	1·3 (0·5)	1·6 (0·5)	NS
Shortening fraction (%)	21·3 (12·5)	25·0 (10·0)	NS
Dimension change in pre-ejection period (%)	6·0 (7·5)	8·8 (7·0)	NS
Dimension change in isovolumic relaxation period (%)	9·0 (5·5)	11·0 (9·0)	NS
Peak rate of dimension fall (cm/s)	7·0 (2·5)	9·4 (2·5)	NS
Peak rate of dimension increase (cm/s)	11·0 (3·5)	12·5 (5·0)	NS
Peak rate of posterior wall thickening (cm/s)	3·3 (1·5)	3·5 (1·1)	NS
Peak rate of posterior wall thinning (cm/s)	6·1 (2·8)	6·7 (3·5)	NS

underlying mechanisms. We believe that the time course of functional mitral regurgitation, in being so closely related to the left ventricular pressure pulse,[10 11] reflects the time course of mechanical systole. We were reassured to find that even the septal dip in patients with left bundle branch block, which is widely attributed to early unopposed contraction of a localised region of the septum,[12 13] was consistently followed within 10 ms by the onset of mitral regurgitation (fig 3). In patients with right ventricular pacing, the onset of mitral regurgitation and that of thickening of the posterior wall, which we took as an index of local function, were effectively synchronous, whereas in those with left bundle branch block the onset of mechanical activity of the posterior wall was delayed by a mean of 80 ms. This was not due to local disease, as when wall thickening did occur, its time course and extent were the same as in patients with right ventricular pacing. If we assume that the close relation between regional activation and regional wall motion noted experimentally[14] also applies to our patients, we must conclude that the delayed posterior wall motion in patients with left bundle branch block reflects delayed activation. The septum is likely to have been the earliest part of the left ventricular myocardium to be activated in both groups of patients, so the posterior wall on M mode echocardiogram, one of the most distant regions from it, is likely to have been one of the last. By contrast with left bundle branch block, effective left ventricular activation time does not therefore seem to have been significantly prolonged in patients with right ventricular pacing, where there was no measurable difference between the onset of mitral regurgitation and that of posterior wall motion. We conclude, therefore, that in patients with left bundle branch block, left ventricular activation was very abnormal, whereas in those with right ventricular pacing, access had been gained at an early stage to a much more rapid pathway on the left side, presumably the Purkinje system. This still applies in patients with previous left bundle branch block, suggesting that even in these circumstances, the Purkinje system route is still accessible. We did not determine why this is the case in patients with right ventricular pacing and not in those with naturally occurring activation as seen in left bundle branch block. Possibly the difference is related to the higher activating energy associated with pacing.

Table 9 Comparison of mitral regurgitation (MR) and left ventricular pressure pulse between patients with right ventricular pacing, with and without previous left bundle branch block (LBBB) (mean (SD))

Index	Patients with previous LBBB (n = 10)	Patients without previous LBBB (n = 14)	p Value
RR interval (ms)	845 (100)	815 (110)	NS
QRS duration (ms)	200 (20)	180 (30)	< 0·05
Electromechanical delay (Q to onset MR) (ms)	115 (25)	113 (25)	NS
Pre-ejection time (ms)	190 (30)	180 (25)	NS
Overall duration of MR (ms):	425 (35)	410 (40)	NS
Contraction time (ms)	75 (25)	70 (20)	NS
Aortic ejection time (ms)	265 (45)	265 (20)	NS
Relaxation time (ms)	90 (20)	75 (15)	NS
Peak pressure drop (mm Hg)	87 (25)	82 (22)	NS
Peak +dP/dt (mm Hg/s)	780 (235)	750 (235)	NS
Peak −dP/dt (mm Hg/s)	625 (180)	570 (245)	NS
Onset MR to onset of posterior wall thickening (ms)	10 (35)	− 3 (40)	NS

This conclusion, based on the mechanical response of the ventricle, seems to differ from previous ones in which left ventricular endocardial activation time was measured directly.[15 16] The apparent discrepancy arises from the low incidence of patients with previous myocardial infarction in our study. Recalculation of the results of these previous studies, taking this additional factor into account, indicates that left ventricular activation consistently starts earlier, and is completed more rapidly in patients with right ventricular pacing compared with those with left bundle branch block. Both figures were greater in the presence of previous myocardial infarction. It is clear, therefore, that left ventricular activation time in patients with right ventricular pacing also depends on the degree of integrity of the conducting system surviving in the left ventricle, a question our study did not specifically attempt to answer.

In spite of these differences in overall activation time, we confirmed that peak rate of increase of left ventricular pressure (+dP/dt) is low in patients with right ventricular pacing.[12 17] It did not differ significantly from that in patients with left bundle branch block, despite the portion of mitral regurgitation before opening of the aortic valve being so much shorter. These two findings are not incompatible. They indicate that the mean rate of pressure rise in patients with right ventricular pacing is normal, whereas the peak value is reduced. We explain this apparent discrepancy by suggesting that in patients with right ventricular pacing, the impulse did not gain access to the Purkinje system by the physiological route from the atrioventricular node and bundle of His, but across the septum in the neighbourhood of the pacing electrode at the apex of the right ventricle. For the duration of mechanical systole to be normal, therefore, all that seems to be necessary is that the time for spread of ventricular activation should be normal. To achieve the normal peak rate of pressure rise, almost double that seen in our patients, seems to be a more specialised function.[2 18] We suggest that this also requires that the sequence of activation be normal, spreading in the bundle of His from the base of the heart and not from

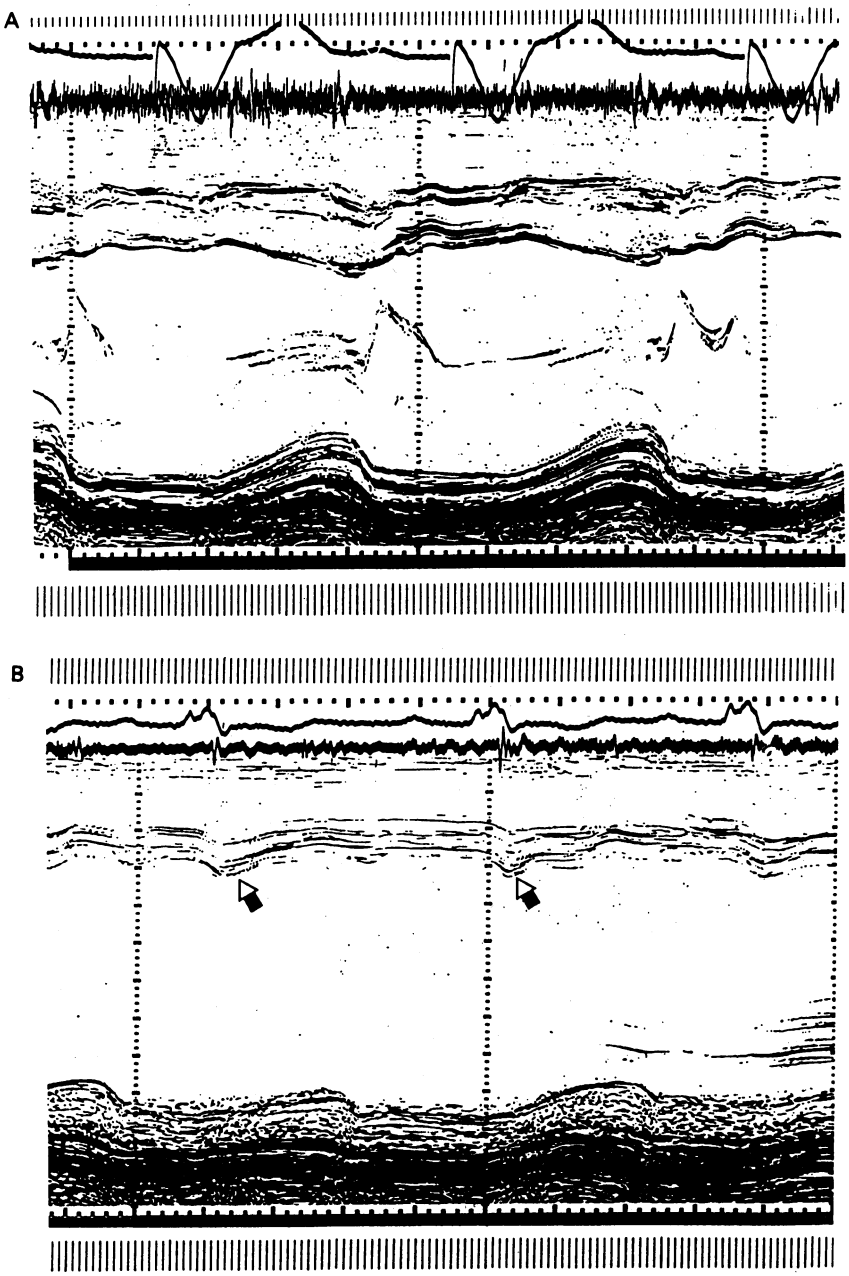

Figure 3 M mode echocardiograms of the left ventricle showing normal septal motion in a patient with right ventricular pacing (A) and an early septal dip (arrow) in a patient with classic left bundle branch block (B).

some apicoseptal site.

Our study had limitations. As in all previous studies, we used the pacing spike as the onset of the QRS complex, and thus did not allow for a possible delay in local activation.[3 16] In patients with left bundle branch block the time interval between the onset of the QRS complex and septal dip or the onset of mitral regurgitation was sometimes $\leqslant 10$ ms. This very short electromechanical delay raises the possibility that activation may sometimes start during what seems to be the PR interval on the surface electrocardiogram, so that overall activation time would be underestimated by a simple measure of QRS duration. If it were confirmed, this concealed activation would be of some theoretical interest, but it is unlikely to have been > 40 ms, and thus will not have affected our overall conclusions. We studied patients in whom pacemakers had been implanted for standard clinical indications. The tip of the electrode was at the apex of the right ventricle in all of them, so we were unable to investigate the site of pacing as a further variable. It is possible that our patients with left bundle branch block had more severe intrinsic myocardial disease than those with right ventricular pacing, but even if this were the case, the presence of cavity dilatation does not affect our conclusions. We were careful to exclude patients with significant mitral regurgitation from our study, as a severe lesion may shorten the duration of regurgitant flow.[19] Again, any such difference would have been reflected in the extent of dimension changes. There was no reason to suggest that left atrial pressure was consistently higher in one or the other group of our patients, and it is only when a high "v" wave develops that atrial pressure can cause the time course of regurgitation to dissociate from that of ventricular pressure.

We conclude that right ventricular pacing and left bundle branch block in humans are not identical in their effects on left ventricular function and the use of right ventricular pacing an experimental model for left bundle branch block must be re-examined. It seems that when the heart is paced from the right ventricle, the impulse can, at an early stage, gain access to a rapid left ventricular conduction pathway so as to make it unlikely that the pathway is anything other than the Purkinje system. This applies even when left bundle branch block had previously been present. For normal mechanical function of the left ventricle, however, more than a normal activation time is necessary. To achieve high values of peak $+dP/dt$, we suggest that this additional factor is a normal activation sequence, which is likely to have been disturbed in our patients. There have been few studies on mechanical consequences of subtle variations in the timing of onset of tension development in components of the ventricular myocardium implicit in the normal activation sequence. In view of the deleterious mechanical consequences of abnormal activation, we believe that these interrelations are a potentially fertile field of future study.

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