Effects of incoordination on left ventricular force-velocity relation in aortic stenosis

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Abstract

Objective—Tension development is often incoordinate in the hypertrophic left ventricle (LV). The present study aimed to elucidate the possible effects of incoordination on standard LV force-velocity relations in patients with aortic stenosis (AS). **Design**—Prospective study during aortic valve replacement with transoesophageal cross sectionally guided M mode echocardiogram, combined with high-fidelity LV pressure recorded by pressure transducer tip catheter, and thermodilution cardiac output.

Setting—Tertiary cardiac referral centre.

Patients—37 patients (mean (SD) age 63 (12)) years were studied before and 20 hours after aortic valve replacement.

Main outcome measures—LV function was assessed regionally by peak velocity of circumferential fibre shortening (peak Vcf), mean systolic wall stress, and peak myocardial power; and globally by LV stroke work index. LV coordination was quantified as cycle efficiency, derived from LV pressure-dimension loop (lower normal limit $\geq 76\%$).

Results—22 patients with a coordinate LV had significantly higher peak Vcf ($1.85 (0.47) v 1.46 (0.64) s^{-1}$) peak myocardial power ($20.8 (8.5) v 12.0 (6.1) mW.cm^{-3}$) and global stroke work index ($440 (155) v 325 (150) mJ.m^{-2}$) than those of 15 patients with an incoordinate ventricle, all $P < 0.05$; though there was no significant difference in LV end diastolic dimension, mean systolic wall stress, LV mass index, or the incidence of coronary artery disease ($P > 0.05$, respectively). Furthermore, when contraction was coordinate, mean systolic circumferential wall stress correlated inversely with peak Vcf ($r = -0.71$) and positively with peak myocardial power ($r = 0.83$), both $P < 0.01$. When contraction was incoordinate, these correlations did not apply; instead peak Vcf ($r = 0.65$) and peak myocardial power ($r = 0.73$) both correlated positively with cycle efficiency ($P < 0.02$ and 0.01 , respectively). By 20 hours after surgery, values of cycle efficiency, peak Vcf, and myocardial power were indistinguishable in the previously coordinate and incoordinate groups.

Conclusions—In aortic stenosis, incoordination causes a fall in LV peak Vcf proportional to the increase in systolic wall stress, and thus modifies the standard LV force-velocity relation to mimic

depressed contractility. However, incoordination and subsequent ventricular dysfunction were largely reversible once the aortic stenosis had been relieved.

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Keywords: valvular aortic stenosis; left ventricular systolic function; incoordination; echocardiography

Left ventricular dysfunction is common in aortic stenosis, and may take several forms. Afterload mismatch is said to have occurred when hypertrophy is not sufficient to normalise wall stress in the face of increased resistance to ejection,^{1,2} particularly if cavity size has increased. Myocardial systolic failure has been defined as a decrease in shortening velocity that is inappropriate to any increase in wall stress.^{3,4} Furthermore, a series of abnormalities of diastole may limit filling and thus the ability of an increase in preload to compensate for systolic disease.^{5–7} Though aortic valve replacement reduces the resistance to ejection, its effects on intrinsic ventricular disease are less certain.^{3,4,8} In part, this is because the definition, and thus quantification of myocardial performance, are rooted in the assumptions that ventricular function is synchronous and spatially uniform.^{9,10} However, in the diseased ventricle, neither of these assumptions need apply—for example, because of activation disturbances, regional wall motion abnormalities, fibrosis, or subendocardial ischaemia.^{11–14} All these disturbances may lead to incoordinate contraction and thus to inefficient energy transfer from the myocardium to the circulation.¹⁵ Significant ventricular incoordination is common in aortic stenosis, and it regresses after aortic valve replacement as haemodynamic state improves.¹⁶ However, the effects of incoordination on classic measurements of left ventricular systolic function have not been fully elucidated. The present study was designed to identify the effects of incoordination on standard left ventricular force-velocity relations in patients with aortic stenosis and to examine possible clinical implications.

Patients and methods

STUDY POPULATION

We studied 37 patients undergoing elective aortic valve replacement for valvular aortic stenosis (mean (SD) age 63 (13), 22 men). According to the NYHA classification 18 patients were in class II, 16 in class III, and three in class IV. Twenty three patients were

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taking no treatment. The three in NYHA functional class IV were on diuretics, digitalis, and ACE inhibitors. The remainder had been prescribed calcium channel blockers or nitrates for chest pain. Left ventricular mass index was $260 (90) \text{ g.m}^{-2}$ measured by M mode echocardiography.¹⁷ Sixteen patients with concomitant coronary artery disease ($\geq 50\%$ diameter stenosis in any branch on the coronary arteriograms) received full revascularisation (2 (1) grafts). Patients with atrial fibrillation either before or after surgery were excluded. Thus all patients were in sinus rhythm before valve replacement, but five patients had a QRS duration $\geq 120 \text{ ms}$, of whom, three had right bundle branch block (RBBB) and two had left bundle branch block (LBBB). All patients remained in sinus rhythm, except five who were in nodal rhythm postoperatively, and were atrially paced (80–85 beat/min). Eleven patients were given dopamine intravenously at a dosage of $\geq 5 \mu\text{g.kg}^{-1}.\text{min}^{-1}$ based on clinical haemodynamic criteria—a cardiac index $\leq 2.0 \text{ l.min}^{-1}.\text{m}^{-2}$ with a pulmonary artery wedge pressure $\geq 15 \text{ mm Hg}$ —immediately after weaning from cardiopulmonary bypass. This study is a part of an established clinical research project, approved by the ethics committee of the Royal Brompton Hospital. Written informed consent was obtained from all participants. There were no side effects of this study.

Patients were studied under general anaesthesia, maintained with fentanyl (20 to $50 \mu\text{g.kg}^{-1}$) and pancuronium oxide (0.1 mg.kg^{-1}). A Swan-Ganz thermodilution balloon tip catheter with its tip in the pulmonary artery was positioned after induction of anaesthesia and used for haemodynamic measurements. Routine cardiopulmonary bypass and current myocardial preservation techniques were used. All valve replacements were performed by the same surgeon (JRP). An aortic homograft or stentless xenograft (Toronto SPV, St Jude Medical, Minnesota, USA) was used in all patients, except two who had a stented xenograft (Carpentier-Edwards, Baxter, California, USA). The range of valve size was from 21 to 27 mm (mean $25 (2.5) \text{ mm}$). Postoperative assessment by transoesophageal echocardiography showed normal functioning of implanted valves. Cardiopulmonary bypass time was $124 (23) \text{ minutes}$, and aortic cross clamp time was $99 (20) \text{ minutes}$. After operation, patients were transferred overnight to intensive care. Postoperative measurements were obtained with the patients under sedation (morphine 1 mg.h^{-1} and propofol $50\text{--}100 \text{ mg.h}^{-1}$, intravenously) and controlled ventilation (fractional inspiratory oxygen $35\text{--}50\%$, inspiratory:expiratory ratio $1:2$, positive end expiratory pressure $1\text{--}2 \text{ mm Hg}$).

STUDY PROTOCOL

Echocardiography and left ventricular pressure recordings

A 5 MHz biplane transoesophageal echocardiographic transducer (HP 21362C) was posi-

tioned after induction of anaesthesia and interfaced with a Hewlett Packard 77025A Sonos 500 or 1500 Ultrasound System. Once the pericardium was opened, a 4F pressure transducer tip catheter (Gaeltec CTC/4F /USCI, Gaeltec, Isle of Skye, Scotland) was introduced into the left ventricle with its tip located in the mid-portion of the cavity, through the right upper pulmonary vein and across the left atrium and mitral valve. Its signal output was filtered with an upper cutoff frequency of 1 kHz , pre-amplified (Gaeltec S7b, Gaeltec, Isle of Skye, Scotland) and transferred to the auxiliary line of the echocardiographic system. The pressure transducer tip catheter was calibrated electrically before the initial measurement, and checked after its final removal with an air-operated dead-weight balance (Budenberg Gauge Company, London). The mean left atrial pressure or pulmonary artery wedge pressure was used to identify the left ventricular end diastolic pressure. Zero pressure was taken as atmospheric. In the transgastric left ventricular short axis view, a two-dimensional-image-directed M mode echocardiogram of the minor axis was recorded at the level of the tips of papillary muscles and printed on paper at a speed of 100 mm.s^{-1} , with a simultaneous left ventricular pressure trace and electrocardiogram. After the final measurement the transoesophageal probe, transducer, and catheters were removed, sedation discontinued, and the patient was weaned off ventilation within 2–3 hours.

Haemodynamic recordings

Heart rate, thermodilution cardiac output, and pulmonary wedge pressure were measured whenever an echocardiogram was recorded. The first measurements were made before cardiopulmonary bypass was started and when the haemodynamic state was stable; postoperative measurements were made at 20 hours after operation.

Digitising and calculations

From the left ventricular M mode echocardiogram and pressure trace, the minor dimension, cavity pressure, and anterior wall thickness were digitised off line (sampling frequency 100 Hz) long with depth and time calibrations.^{15 16} The onset of the QRS complex of the electrocardiogram was used to identify end diastole. The time from peak rate of left ventricular pressure rise ($+ dP/dt_{\text{max}}$) to peak rate of pressure fall ($- dP/dt_{\text{max}}$) was taken as ejection time. The time point of $- dP/dt_{\text{max}}$ was checked against the closure of the aortic valve on the M mode echocardiogram. Three successive beats were digitised at each time interval and mean values used. From the digitised output and haemodynamic data we derived:

(1) *Cavity dimension and wall thickness* (the end diastolic cavity dimension and anterior wall thickness, peak rate of dimension shortening normalised to end diastolic dimension (peak Vcf)).

(2) *Cavity pressure and wall stress* (maximum developed left ventricular pressure, mean ejection pressure, and $+ dP/dt_{\text{max}}$). Left ventricular

circumferential mid-wall stress was calculated by the method of Falsetti *et al.*,¹⁸ with long axis values derived from a previously determined regression relation.¹⁵ The mean values of wall stress during ejection were derived from the continuous measurement over the cardiac cycle.

(3) *Regional function and coordination*—Mid-wall myocardial power was given by the instantaneous product of circumferential wall stress and mid-wall dimensional shortening rate. Ventricular circumference at this point is given by $\pi (D+T/2)$, where D and T represent transverse cavity dimension and wall thickness, respectively, and mid-wall shortening rate as its first differential with respect to time. Values of myocardial power were normalised to refer to a cubic centimetre of myocardium at end diastole.^{15,16} A significant change of dimension during either isovolumic period represents incoordinate contraction and relaxation respectively. We used cycle efficiency, defined as the ratio of the left ventricular pressure dimension loop area to that of the rectangle that just encloses it—that is, the ratio of useful external work to the maximum possible for myocardium working over the same range of pressures and dimensions—to quantify these effects of ventricular incoordination in terms of their influence on energy transfer from myocardium to the circulation.^{15,16} Its lower normal limit, 76%,^{15,19} was used to divide the study population into two groups, with coordinate (cycle efficiency $\geq 76\%$) or incoordinate (cycle efficiency $< 76\%$) left ventricular function.

(4) *Global function and haemodynamics*—From thermodilution cardiac output measurements, simultaneous heart rate, and left ventricular mean ejection pressure (LVMEP) we calculated global stroke volume index (LVSVI) and thus global stroke work index (LWSWI = LVSVI $\times$ (LVMEP – LVEDP) $\times 0.0136 \times 9.8 \text{ mJ.m}^{-2}$).²⁰

STATISTICAL METHODS

Continuous data were presented as mean (1 SD). Minitab statistical software (PC Version, Release 8, 1991, Minitab Inc, USA)²¹ was used for statistical analysis. A oneway analysis of variance or a chi-squared test was used to compare quantitative or qualitative data, respectively. Stepwise regression analysis was used to identify independent predictors of left ventricular function with respect to both ventricular afterload and ventricular coordination. A $P < 0.05$ was taken as statistically significant.

Results

CLINICAL DATA

Twenty two patients with a cycle efficiency $\geq 76\%$ (80.8(3.5)%) were considered to have coordinate left ventricular function, and the remaining 15 patients had cycle efficiency $< 76\%$ (62.7 (9.0)%) representing incoordinate left ventricular function. There was no significant difference between the two groups with respect to age (62 (12) *v* 63 (14)), gender (14/8 *v* 8/7, male/female), left ventricular mass index (235 (85) *v* 285 (90) g.m^{-2}), peak pressure gradient across the aortic valve (75 (19) *v* 79 (25) mm Hg), incidence of associated coronary artery disease (9/22 *v* 7/15, patient/patient), extent of revascularisation (2.0 (1.0) *v* 2.4 (1.0), graft), or the incidence of classic conduction abnormalities (1 LBBB, 1 RBBB *v* 1 LBBB, 2 RBBB, patient), all $P > 0.05$. However, the NYHA functional class of patients with a coordinate left ventricle was significantly better than those with an incoordinate ventricle (2.4 (0.5) *v* 2.9 (0.7), $P = 0.034$), though there was no difference in the incidence of shortness of breath (16/22 *v* 13/15, $P = \text{NS}$), angina (10/22 *v* 9/15, $P = \text{NS}$), or the requirement for medical treatment, as assessed by the prescriptions of diuretics, digitalis, ACE inhibitors, or calcium channel blockers. Aortic cross clamp (99 (19) *v* 99 (22) min) and cardiopulmonary bypass times (122 (22) *v* 127 (25) min) and postoperative requirement for positive inotropic drugs, defined as a dosage of dopamine $\geq 5 \mu\text{g/kg/min}$ (6/22 *v* 5/15, patient/patient), were all similar. The same applied to incidence of nodal rhythm (3/22 *v* 2/15 patient/patient), and conduction abnormalities (2 RBBB, 1 LBBB *v* 2 LBBB, 1 RBBB, patient) (all $P > 0.05$).

LEFT VENTRICULAR FUNCTION BEFORE AORTIC VALVE REPLACEMENT

Comparison of loading conditions—End diastolic dimension (4.5 (0.9) *v* 4.5 (0.8) cm) was identical in the two groups, as was the end diastolic wall thickness (1.8 (0.4) *v* 2.0 (0.3) cm), and end diastolic circumferential wall stress (20 (15) *v* 25 (10) g.cm^{-2}), all $P > 0.05$. During systole, there was no significant difference in mean systolic wall stress (185 (90) *v* 160 (60), g.cm^{-2} , $P > 0.05$) between the two groups.

Comparison of regional and global function—Table 1 compares myocardial function in coordinate and incoordinate ventricles. Peak Vcf was significantly lower in the incoordinate ventricles, as were peak myocardial power and myocardial stroke work. With similar heart

Table 1 Comparison of regional and global myocardial function between coordinate and incoordinate left ventricles before aortic valve replacement (mean (SD))

Variable	Coordinate LV (n = 22)	Incoordinate LV (n = 15)	P
Peak velocity of circumferential shortening (s^{-1})	1.85 (0.47)	1.46 (0.64)	0.039
Peak circumferential myocardial power (mW.cm^{-3})	20.8 (8.5)	12.0 (6.1)	0.002
Circumferential myocardial stroke work (mJ.cm^{-3})	3.3 (1.6)	1.7 (0.9)	0.001
Maximal developed pressure (mmHg)	161 (29)	142 (23)	0.035
+dP/dt _{max} (mm Hg.s ⁻¹)	1610 (370)	1545 (550)	NS
Heart rate (beat.min ⁻¹)	85 (19)	88 (20)	NS
Global stroke volume index (ml.m^{-2})	26 (6)	23 (8)	NS
Global stroke work index (mJ.m^{-2})	440 (155)	325 (150)	0.030

LV, left ventricle.

Table 2 Comparison of regional and global myocardial function between patients with and without concomitant coronary artery disease before aortic valve replacement (mean (SD))

Variable	Isolated AS (n = 21)	AS + CAD (n = 16)	P
Peak velocity of circumferential shortening (s^{-1})	1.66 (0.65)	1.73 (0.48)	NS
Cycle efficiency (%)	73.8 (9.8)	73.0 (12.6)	NS
Peak circumferential myocardial power ($mW \cdot cm^{-3}$)	17.1 (8.7)	17.3 (9.0)	NS
Circumferential myocardial stroke work ($mJ \cdot cm^{-3}$)	2.6 (1.6)	2.7 (1.6)	NS
+dP/dt _{max} (mm Hg s^{-1})	1595 (420)	1570 (490)	NS
Heart rate (beat min^{-1})	90 (22)	81 (15)	NS
Global stroke volume index ($ml \cdot m^{-2}$)	25 (6)	24 (8)	NS
Global stroke work index ($mJ \cdot m^{-2}$)	395 (185)	400 (155)	NS

AS, aortic stenosis; CAD, coronary artery disease.

Table 3 Stepwise regression analysis of effects of afterload and coordination on LV systolic function within each group before aortic valve replacement

Variable	Constant	Regression coefficient			SD	P
		MSS	CE	r		
Coordinate LV (n = 22):						
Peak velocity of circumferential shortening (s^{-1})	2.6	-0.0039***	NS	0.71	0.4	< 0.01
Peak circumferential myocardial power ($mW \cdot cm^{-3}$)	5.7	0.081***	NS	0.83	5.0	< 0.01
LV stroke work index ($mJ \cdot cm^{-2}$)	-180	NS	280***	0.70	10	< 0.01
Incoordinate LV (n = 15):						
Peak velocity of circumferential shortening (s^{-1})	-1.45	NS	4.6*	0.65	0.5	< 0.02
Peak circumferential myocardial power ($mW \cdot cm^{-3}$)	-24.2	NS	47**	0.73	4.5	< 0.01
LV + dP/dt _{max} (mm Hg s^{-1})	-1650	NS	5000***	0.83	320	< 0.01

LV, left ventricle; MSS, mean systolic wall stress; CE, cycle efficiency; *P < 0.02, **P < 0.01, ***P < 0.001; r, correlation coefficient.

rate and stroke volume index, global stroke work index in the incoordinate left ventricles was also significantly lower, because of the lower maximum developed left ventricular pressure, though values of +dP/dt_{max} did not differ.

Effects of coronary artery disease—Patients with concomitant coronary artery disease were significantly older (64 (12) v 53 (10) year, P < 0.01) than those without. At similar end diastolic dimension (4.3 (0.7) v 4.6 (0.9) cm, P = NS) and mean systolic wall stress (170 (75) v 180 (80) g cm^{-2} , P = NS) coronary artery disease had no consistent effect on peak Vcf, peak myocardial power, global stroke work index, or cycle efficiency (table 2).

Effects of incoordination on systolic function—Stepwise regression analysis was used to define the independent effects of systolic wall stress and cycle efficiency on myocardial systolic function in each group. In the 22 patients with coordinate ventricular contraction, peak Vcf was inversely and peak power directly proportional to mean systolic wall stress, with no additional influence of cycle efficiency detectable (table 3; figs 1, 2, and 3). Conversely, left ventricular stroke work, reflecting global function, was unrelated to mean systolic wall stress but correlated significantly with

Figure 1 Peak velocity of circumferential fibre shortening (peak Vcf) plotted against mean left ventricular (LV) systolic circumferential wall stress, before relief of aortic stenosis. Note that there is an inverse significant linear correlation between the two in coordinate ventricles (closed circles, $r = -0.71$, $P < 0.01$) but not in incoordinate ones (open circles, $r = -0.45$, $P > 0.05$).

Figure 2 Peak Vcf plotted against LV cycle efficiency, before relief of aortic stenosis. Note that there is no significant correlation between the two in coordinate ventricles (closed circles, $r = -0.27$, $P > 0.05$) but in incoordinate ventricles (open circles) peak Vcf correlated positively ($r = -0.65$, $P < 0.02$) with cycle efficiency.

Figure 3 Peak circumferential myocardial power plotted against mean LV systolic circumferential wall stress, before relief of aortic stenosis. Note that there is positive correlation between the two in those with a coordinate ventricle (closed circles, $r = 0.83$, $P < 0.01$) but not in those with incoordinate ventricles (open circles, $r = 0.28$, $P > 0.05$).

Table 4 Stepwise regression analysis of effects of loading and coordination on LV systolic function in all 37 patients after aortic valve replacement

Variable	Constant	Regression coefficient			r	SD	P
		EDD	MSS	CE			
Peak velocity of circumferential shortening (s^{-1})	5.3	-0.64***	NS	NS	0.59	0.6	< 0.01
Peak circumferential myocardial power ($mW.cm^{-3}$)	11	NS	0.062*	NS	0.35	7.2	< 0.05
Maximal developed LV pressure (mm Hg)	142	-9.4**	NS	NS	0.39	15	< 0.05
LV stroke work index ($mJ.cm^{-2}$)	39	-5.2*	0.140***	NS	0.57	9.3	< 0.01

LV, left ventricle; EDD, end diastolic dimension; MSS, mean systolic wall stress; CE, cycle efficiency; *P < 0.05, **P < 0.01, ***P < 0.001; r, correlation coefficient.

cycle efficiency (table 3); $+dP/dt_{max}$ and peak developed left ventricular pressure showed no significant correlation with either cycle efficiency or mean systolic wall stress. In the 15 patients with incoordinate ventricular contraction, peak Vcf and myocardial power did not correlate with mean systolic wall stress (table 3, figs 1 and 3) but instead peak Vcf was directly related to cycle efficiency (fig 2). Cycle efficiency also correlated with $+dP/dt_{max}$, but neither peak developed pressure nor left ventricular stroke work index correlated with cycle efficiency or mean systolic wall stress in a stepwise regression analysis (table 3).

CHANGES IN LEFT VENTRICULAR FUNCTION AFTER AORTIC VALVE REPLACEMENT

Twenty hours after aortic valve replacement, mean systolic wall stress ($115 (45) v 115 (40) g.cm^{-2}$, $P > 0.05$) had fallen similarly in each group, though the end diastolic dimension remained unchanged in both. Mean cycle efficiency in the group with previously incoordinate ventricular function had returned to normal ($77.4 (6.9)\%$) and no longer differed from that in the previously coordinate group (now $76.0 (5.8)\%$), $P > 0.05$. With unchanged end diastolic dimension ($4.5 (0.7) v 4.6 (0.7) cm$, $P > 0.05$) and similar fall in mean systolic wall stress, peak Vcf increased to similar values in both groups ($2.45 (0.73) v 2.30 (0.8) s^{-1}$, $P > 0.05$), as did peak myocardial power ($19.6 (8.2) v 16.4 (6.3) mW.cm^{-3}$, $P > 0.05$). Maximal developed left ventricular pressure ($92 (6) v 104 (19) mm Hg$, $P < 0.05$), however, was still lower in the previously incoordinate ventricles. After relief of aortic stenosis, cycle efficiency was no longer correlated with any measurement of left ventricular systolic function (table 4).

Discussion

This study demonstrates that significant left ventricular incoordination impairs regional and global systolic function in a way that cannot be predicted by classic force-velocity relations during excessive afterload. Once incoordination had regressed after successful valve replacement, these effects on left ventricular force-velocity relations were no longer present.

DEFINITION AND QUANTIFICATION OF VENTRICULAR INCOORDINATION

Even the normal left ventricle shows some physiological heterogeneity, which does not

interfere with normal function, and may even contribute to it. However, with the development of ventricular disease, asynchrony affects systole and diastole.²²⁻²⁴ In order to quantify it, we used a definition based on its main effect, namely reduced efficiency of energy transfer from myocardium to circulation. The shape of the pressure-dimension loop depends on the interrelation between local function—that is, the dimension—and overall function represented by the pressure. It relates useful external work to the maximal possible that could have been performed by myocardium working over the same range of pressure and dimension, and so allows the significance of regional asynchrony to be determined.^{15 16} The physiological significance and clinical practical value of measurements of cycle efficiency have been reported in previous studies.^{15 16 19 25 26}

EFFECTS OF INCOORDINATION ON MEASURES OF VENTRICULAR SYSTOLIC FUNCTION

In isolated heart muscle, shortening velocity is inversely related to the force opposing it. This force-velocity relation has been widely applied to the intact ventricle over two decades^{9 10} and used to investigate patients with impaired myocardial function because of pressure or volume overload.¹⁻⁴ However, possible ways in which force-velocity relations might be modified by coexistent incoordination have received little attention. Our results suggest that ventricular incoordination greatly modifies these principles, and when it is severe the expected inverse force-velocity relation can no longer be demonstrated. Instead, peak Vcf relates inversely to the degree of incoordination itself. This relation is not affected either by cavity size or afterload. In a coordinate ventricle, therefore, a low value of peak Vcf is caused by increased afterload, but this no longer applies when incoordination is severe.

The reciprocal relation between myocardial force and shortening velocity can also be studied in terms of peak myocardial power, the product of the two.^{9 27} In the present study, peak myocardial power was directly proportional to wall stress over the range of 100 to $430 g.cm^{-2}$, provided that left ventricular function was coordinate (fig 3), so that higher power output was achieved at low shortening velocities. This relation was also lost in the presence of incoordination. As might be expected, cycle efficiency correlated with $+dP/dt_{max}$ in incoordinate ventricles,¹⁵ again suggesting that indices based on peak rate of rise of pressure²⁰ are of limited value in assessment of contractility in these circumstances.

By 20 hours after valve replacement, cycle efficiency had improved and its relation with systolic function was no longer apparent.

LIMITATIONS

Our study has a number of limitations. Left ventricular function was assessed during and after anaesthesia and cardiac surgery, which have effects, particularly opening the pericardium and possible changes in loading conditions that may have affected the measurements. However, these factors should have operated to a similar extent between the two groups and so are unlikely to have led to consistent differences. Any effect of chest closure on postoperative functional assessment was likely to have been very small, because the pericardium was left open and drains were in situ until the end of study. The incidence of preoperative drug treatment and intraoperative positive inotropic drug administration was similar in each group, so neither the difference between the groups nor the improvement of previously incoordinate ventricles can be explained on this basis. In fact, our previous study has shown that a higher dose of positive inotropic drug causes a greater ventricular incoordination early after aortic valve replacement.²⁸ Calculation of wall stress based on Laplace's law has well recognised limitations, particularly in the presence of hypertrophy, when wall thickness is large compared with cavity dimension. Our use of Falsetti's method in this study was based on the close relation between major and minor ventricular axes, and justified in a previous study.¹⁵ As there was no difference in minor dimensions between the two groups, systematic error is unlikely to have been significant. The same applies to any effect of overestimation of the circumferential fibre shortening velocity from endocardial measurements.²⁹ The lower limit of normal range we used for cycle efficiency, 76%, was based on previous studies. However, our conclusions would not have been altered by taking any value in the range of 70–76%. The position of the transoesophageal echo transducer was checked with respect to mitral valve anatomy on the two dimensional display, so possible transducer displacement was minimised. The reproducibility of computer digitising has been reported elsewhere,¹⁶ and is adequate to support our conclusions with the sample size we used. Finally, the present study was designed to investigate the consequences rather than the causes of ventricular incoordination. Statistically though neither coronary artery disease nor major conduction abnormalities were significant causes.

CLINICAL IMPLICATIONS

Left ventricular dysfunction remains an important cause of morbidity and mortality in patients with aortic valve disease, so its successful management is likely to improve surgical outcome, particularly in high risk cases. A reduction in myocardial shortening velocity that is inappropriate for the level of afterload present with aortic stenosis is usually interpreted as being caused by severely impaired

myocardial contractility, and thus theoretically may be a high risk condition if surgical option is considered. However, our results suggest that this picture may be associated with significant incoordination, which responds well to surgical treatment. It is by no means certain that the optimal medical management of incoordinate contraction is the same as that of uniformly depressed myocardial contractility. Indeed, there is every prospect that inhomogeneous myocardial disease may respond to a positive inotropic drug in such a way as to make the inhomogeneity worse.^{28–30} Ventricular hypertrophy remains a major cause of heart failure; recognition of the role of ventricular incoordination in such patients may avoid inappropriate drug treatment and may form the basis for deriving more successful approaches to its management.

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