Left ventricular ejection power in coronary artery disease during atrial pacing

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SUMMARY Peak and mean left ventricular ejection power were measured during atrial pacing in 6 normal subjects (group I), 6 patients with coronary artery disease without myocardial infarction (group IIa), and 10 patients with coronary artery disease after myocardial infarction (group IIb). Pacing rates were 80 and 120/min. Power was determined by computer analysis of pressure, volume, and time. Data were normalised by end-diastolic volume and left ventricular muscle mass.

Peak left ventricular ejection power normalised by end-diastolic volume in normal subjects increased from 53.4 to 68.2 mW/ml (+28%, $P < 0.01$). No change was seen in group IIa and IIb. Peak and mean left ventricular ejection power normalised by end-diastolic volume values at a pacing rate of 120 min were significantly lower in group IIa and IIb than in normal subjects. Mean muscle mass in normal subjects was 179 g and in group IIa 216 g ($P < 0.05$). Peak power normalised by muscle mass in normal subjects tended to increase at 120/min whereas in group IIa it declined by 26 per cent ($P < 0.001$).

These data indicate that the energy output of the left ventricle at rest may be the same in patients with significant coronary artery disease as in normal subjects. Increasing the heart rate from 80 to 120/min in a normal myocardium augments power but in coronary artery disease it remains static or falls.

Left ventricular dynamics can be described in terms of pressure, volume, and time. Pressure analysis according to the principles of the force-velocity-length relation has been performed by numerous investigators (Brutsaert and Sonnenblick, 1973; Sonnenblick and Strobeck, 1977). This method has answered many questions about the contractile behaviour of the myocardium in health and disease. Nevertheless it is based upon theoretical assumptions derived from isolated muscle preparations, and the validity of data from the intact heart is limited. Furthermore, pressure analysis gives no direct information about the function of the ventricle as a pump. It has been shown in studies on ventricular volume that the ejection fraction and the velocity of circumferential fibre shortening have value in the detection of myocardial abnormality (Sonnenblick and Strobeck, 1977). However, these indices do not describe pump function adequately, whereas left ventricular power during ejection combines the time-dependent

changes in ventricular pressure and volume, and in animals has been shown to be useful as an index of cardiac performance under various haemodynamic conditions (Stein and Sabbah, 1976a, b).

This study was undertaken to determine left ventricular power in normal subjects and in patients with coronary artery disease both at rest and under stress, using a computer programme for simultaneous analysis of pressure, volume, and time developed by the group.

Subjects and methods

PATIENTS

The study was performed in 22 patients referred to our department for diagnostic cardiac catheterisation. Twenty-one were male and one (case 5) female. Six of the subjects were considered normal by noninvasive techniques, haemodynamic measurements, and coronary arteriography (group I, mean age 38 ± 12 years). These were studied because of chest pain of undetermined origin. Sixteen patients had coronary artery disease as shown by coronary

arteriography (group II, mean age 49 ± 6 years) and were potential candidates for coronary artery surgery. Six of these patients had no history of myocardial infarction (group IIa, mean age 46 ± 7 years); in the remaining 10, previous myocardial infarction had been clearly documented by electrocardiography and enzyme studies (group IIb, mean age 50 ± 5 years). Sinus rhythm with normal atrioventricular conduction was present in all subjects. Patients with significant hypertension, mitral valve regurgitation, or major left ventricular wall motion abnormalities were excluded from the study. There was no statistical difference in body surface area between the groups (mean $1.83 \pm 0.11 \text{ m}^2$).

CARDIAC CATHETERISATION TECHNIQUES

Left and right heart catheterisation was performed in the fasting and recumbent state without premedication. Catheters were introduced under local anaesthesia using the Seldinger technique via the femoral vessels. Left ventricular and aortic pressures were measured simultaneously by catheter tip manometer¹ and a fluid filled catheter (multi purpose type²), respectively. In some instances a single catheter with two manometers was used.¹ The zero reference pressure was set at 5 cm below midchest level. Left ventricular angiography was performed by injection of 60 to 70 ml of 70 per cent Na-meglumine-iotalamate into the right atrium or pulmonary artery using a pigtail catheter². With this technique premature ventricular contractions were completely avoided, and left ventricular volume remained unchanged throughout the period during which it was visible. This method has been described elsewhere in detail (Hagemann and Meyer, 1978). The ventriculograms obtained were of adequate quality for quantitative analysis by computerised videometry (Hagemann *et al.*, 1976). Only the earliest systoles providing adequate opacification of the left ventricular cavity were analysed.

The Judkins technique was used for coronary arteriography. Biplane cineangiography of the left ventricle was performed in the 30° RAO position. 35 mm film was used, exposed at 50 frames/second. A bipolar electrode catheter³ inserted via a femoral vein was positioned against the wall of the right atrium. Effective pacing was obtained in all patients using pulses with an amplitude of 2.0 V and a duration of 1.0 ms. There were no complications from catheterisation.

PROCEDURE

All measurements were taken before coronary arteriography. After the catheters had been placed in position, atrial pacing was started at a rate of 80/min and continued for 3 minutes. Then left ventricular cineangiography was performed during atrial pacing. Left ventricular and aortic pressures were measured simultaneously, and the data stored on digital tape. After a recovery period of at least 20 minutes and when left ventricular systolic and end-diastolic pressures had returned to the control level the rate of pacing was increased to 120/min and continued for 3 minutes. When the new steady state was achieved cineangiography during atrial pacing was repeated, care being taken to ensure that the two ventriculograms were performed under the same conditions (amount of contrast medium, injection rate, x-ray factors, patient position, and position of tube and image intensifier).

DATA PROCESSING AND CALCULATIONS

Left ventricular and aortic pressures during cineangiography were calculated on line by a computer¹ sampling at 400 times a second. This computer programme has previously been described in detail (Jensch *et al.*, 1976). In addition, pressures, the electrocardiogram, and a frame exposure signal were recorded by a photographic recorder² running at 100 mm/s. Ventricular volume was determined at 20 ms intervals by videometry after video camera scanning of the 35 mm films. Single plane contour tracings were done by light pen. Volumes were calculated according to Simpson's rule using the same computer as for pressure calculations (interactive dialogue system). Volumes obtained by videometry were calibrated by a metal sphere of known volume which had been filmed in the same position as the left ventricle. No regression equations for correction of volumes were used. Smoothing and differentiation of the volume curve was performed as described by Savitzky and Golay (1964). Left ventricular muscle mass in end-diastole was calculated according to the modified method of Rackley *et al.* (1964). Muscle mass was not assessed in patients with infarction. In the angiographic method, wall thickness is assumed to be uniform, which is not true after myocardial infarction, and thinning over the necrotic area causes muscle mass to be overestimated by an unknown amount.

Simultaneous pressure and volume data during angiography were collated by the computer to establish a basic pressure volume progression. Using a linear interpolation procedure for the

¹Millar Instruments, Houston, Texas.

²Cordis Corporation, Miami, Florida.

³USCI, Glen Falls, New York.

¹General Automation, Inc., Anaheim, Cal.

²Electronics for Medicine, Inc., White Plains, N.Y.

Fig. 1 Left ventricular pressure, volume, and ejection power in a normal subject (case 4) during atrial pacing at a rate of 80/min (left) and 120/min (right). Abbreviations: p = pressure, V = volume, P = power, ET = ejection time, t = time, A = aortic valve opening, B = aortic valve closure. Power has been plotted on the negative y axis for mathematical reasons ($-P = p \times (-dV/dt)$). The negative sign has been neglected in the Tables and in the other Figures.

volume curve calculations of instantaneous left ventricular power were made automatically at 10 ms intervals. By plotting power against time a continuous power curve was obtained (Fig. 1). Left ventricular power during ejection was calculated according to the equation

$$P = p \times dV/dt$$

where P = power, p = instantaneous left ventricular pressure, and dV/dt = instantaneous ejection blood flow. In SI units power is expressed in Watts. The conversion relation is

$$\text{power} = 1 \text{ mmHg} \times 1 \text{ ml} \times \text{s}^{-1} = 1.333 \times 10^{-4} \text{ Watt.}$$

The ejection time for calculation of mean power was identified from the aortic pressure tracing. The ejection fraction (%) was calculated as stroke volume/end-diastolic volume multiplied by 100.

STATISTICS

Statistical analysis was performed using the paired and unpaired t test (Ostle, 1963; Brownlee, 1965). A significant difference was accepted at a level of $\alpha = 5$ per cent ($P < 0.05$).

Results

The data are summarised in detail in Tables 1 and 2.

END-DIASTOLIC VOLUME

The end-diastolic volume in group I at heart rates of 80 and 120/min was lower than in group II as a whole ($P < 0.05$) and group IIb ($P < 0.05$). No significant difference however was found between groups I and IIa. When the rate was changed from 80 to 120/min end-diastolic volume decreased in groups I and IIa by 19 per cent and in group IIb by 12 per cent.

EJECTION FRACTION

The ejection fraction at heart rates of 80 and 120/min in group II as a whole and group IIb was lower than in group I ($P < 0.001$). At 80/min group IIa showed no significant difference compared with group I, but at 120/min the ejection fraction was significantly lower in group IIa ($P < 0.02$). When the rate was increased the ejection fraction decreased in groups IIa and IIb by 17 per cent whereas no change occurred in group I.

PEAK AND MEAN EJECTION POWER

No statistical difference in these indices was found between normal subjects and patients in groups II, IIa, and IIb at heart rates of 80 and 120/min.

Table 1 Haemodynamic data on normal subjects (group I)

Case no.	End-diastolic (volume ml)		Ejection fraction (%)		LV muscle mass (g)		Peak ejection power (mW)		Mean ejection power (mW)		Peak ejection power normalised by end-diastolic volume (mW/ml)		Mean ejection power normalised by end-diastolic volume (mW/ml)		Peak ejection power normalised by muscle mass (mW/g)	
	a	b	a	b	a	b	a	b	a	b	a	b	a	b	a	b
1	162	126	72	69	198	—	10 929	9 352	5920	5870	67.5	74.2	32.5	39.2	55.2	47.2
2	160	136	55	50	197	—	8 812	7 806	4225	4382	55.0	81.0	23.2	32.0	44.7	39.6
3	172	153	65	48	189	—	6 652	7 951	4733	4495	38.7	52.0	26.1	23.4	35.2	42.0
4	198	156	58	57	170	—	9 066	10 233	5888	5404	45.8	65.6	25.9	30.5	53.3	60.2
5	124	92	64	67	132	—	5 537	5 504	4058	3558	74.3	64.4	28.9	34.7	41.9	41.7
6	172	134	56	58	185	—	6 772	9 627	5279	5192	39.3	72.0	30.3	38.9	36.6	52.0
Mean	164	133	62	58	179	—	7 961	8 412	5017	4817	53.4	68.2	27.8	33.1	44.5	47.1
± SD	± 24	± 23	± 7	± 9	± 25	—	± 1 988	± 1 715	± 809	± 833	± 14.9	± 9.9	± 3.4	± 5.9	± 8.3	± 7.8
P	< 0.001		NS		—		NS		NS		< 0.01		< 0.05		NS	

a, b = atrial pacing 80 and 120/min, respectively.

The increase in heart rate did not significantly change peak and mean ejection power in group I and group IIb but in group IIa they decreased significantly by 25 and 18 per cent, respectively (both $P < 0.001$).

PEAK AND MEAN EJECTION POWER NORMALISED BY END-DIASTOLIC VOLUME
At 80/min there was no difference in peak power normalised by end-diastolic volume between normal subjects and those in groups II, IIa, and IIb.

Table 2 Haemodynamic data on patients with coronary artery disease (group II, IIa, IIb)

Case no.	End-diastolic volume (ml)		Ejection fraction (%)		LV muscle mass (g)		Peak ejection power (mW)		Mean ejection power (mW)		Peak ejection power normalised by end-diastolic volume (mW/ml)		Mean ejection power normalised by end-diastolic volume (mW/ml)		Peak ejection power normalised by muscle mass (mW/g)	
	a	b	a	b	a	b	a	b	a	b	a	b	a	b	a	b
Group IIa (no myocardial infarction)																
7	167	156	57	48	191	—	10 984	8 316	4657	4574	65.8	53.3	24.2	25.6	57.5	43.5
8	194	136	56	48	220	—	12 533	9 749	5303	3954	64.6	71.1	25.8	23.6	55.9	44.3
9	217	205	47	39	256	—	9 866	8 663	6484	5204	45.5	42.3	24.6	20.5	38.5	33.8
10	208	124	45	46	252	—	9 699	5 814	5821	4391	46.6	46.8	23.4	30.0	38.4	23.0
11	183	160	48	38	165	—	7 376	5 704	4266	3501	40.3	35.7	18.6	17.0	46.5	34.5
12	153	130	70	52	213	—	9 081	6 297	5076	4440	59.4	48.4	33.2	34.2	42.6	29.5
Mean	187	152	54	45	216	—	9 923	7 424	5268	4344	53.7	49.7	25.0	25.2	46.7	34.7
± SD	± 24	± 30	± 9	± 6	± 35	—	± 1 743	± 1 706	± 800	± 577	± 11	± 12	± 4.7	± 6.3	± 8.6	± 8.2
P	< 0.05		< 0.05				< 0.001		< 0.001		NS		NS		< 0.001	
Group IIb (myocardial infarction)																
13	166	139	51	40	—	—	9 948	7 551	4825	3850	59.1	54.3	23.1	23.4	—	—
14	265	241	38	21	—	—	11 270	9 388	4645	3204	42.5	38.9	15.4	9.9	—	—
15	154	129	50	48	—	—	10 888	10 169	5185	5109	70.7	78.8	31.5	40.4	—	—
16	266	233	34	32	—	—	8 312	7 489	5136	5623	31.2	32.1	16.9	20.3	—	—
17	308	233	31	27	—	—	7 901	6 084	5219	4136	25.7	27.2	16.9	17.7	—	—
18	207	183	42	30	—	—	5 478	5 126	3885	3168	26.4	28.2	19.3	17.3	—	—
19	197	149	46	38	—	—	7 949	7 356	5077	4415	40.4	49.4	22.6	21.3	—	—
20	182	163	47	41	—	—	7 690	8 608	5246	5343	42.1	53.1	28.8	32.8	—	—
21	230	236	34	34	—	—	7 411	8 857	4067	4733	32.2	37.5	17.7	20.1	—	—
22	280	283	41	29	—	—	6 260	4 972	4277	3300	22.4	17.6	15.3	11.7	—	—
Mean	226	199	41	34			8 311	7 560	4756	4288	39.4	41.7	20.8	21.5	—	—
± SD	± 52	± 53	± 7	± 8			± 1 880	± 1 757	± 513	± 907	± 16	± 18	± 5.7	± 9.2	—	—
P	< 0.05		< 0.001				NS		NS		NS		NS		—	
Group II (all patients with CAD)																
Mean	211	181	46	38			8 913	7 509	4948	4309	44.5	44.7	22.3	22.9	—	—
± SD	± 47	± 50	± 10	± 9			± 1 942	± 1 681	± 661	± 778	± 15.4	± 15.9	± 5.6	± 8.2	—	—
P	< 0.001		< 0.001				< 0.001		< 0.001		NS		NS		—	

a, b = atrial pacing 80 and 120/min, respectively.

Fig. 2 Peak left ventricular ejection power normalised by end-diastolic volume. Normals (I) and patients with coronary artery disease without myocardial infarction (IIa) and after myocardial infarction (IIb). Atrial pacing 80 and 120/min.

At 120/min, however, it rose 28 per cent in group I but remained unchanged in both coronary groups—a significant difference (group II as a whole, $P < 0.01$, group IIa, $P < 0.02$, and group IIb, $P < 0.01$) (Fig. 2). Mean power normalised by end-diastolic volume was significantly higher at 80/min in normal subjects when compared with

group IIb ($P < 0.02$) and group II as a whole ($P < 0.05$) but not with group IIa. At 120/min the difference achieved significance for all coronary groups (IIa, $P < 0.05$, IIb, $P < 0.02$, and group II as a whole, $P < 0.02$), the increase in rate producing a 19 per cent rise in normal subjects but no change in the coronary patients (Fig. 3).

Fig. 3 Mean left ventricular ejection power normalised by end-diastolic volume. Atrial pacing 80 and 120/min. See legend Fig. 2.

EJECTION TIME

Ejection time as determined from the aortic pressure tracing at heart rates of 80 and 120/min was 273 ± 25 ms and $232 \pm$ ms respectively in group I, 283 ± 20 ms and 236 ± 12 ms respectively in group IIa, 280 ± 27 ms and 227 ± 28 ms respectively in group IIb, and 281 ± 24 ms and 230 ± 23 ms respectively in group II as a whole. The decrease in ejection time was significant in all groups ($P < 0.001$) but there was no statistically significant difference between the groups themselves.

LEFT VENTRICULAR MUSCLE MASS

End-diastolic muscle mass in normal subjects at a heart rate of 80/min was 179 ± 25 g. Muscle mass in group IIa was significantly higher than in normal subjects (216 ± 35 , $P < 0.05$).

PEAK EJECTION POWER NORMALISED BY MUSCLE MASS

Peak ejection power normalised by muscle mass at a heart rate of 80/min was 44.5 ± 8.3 mW/g in normal subjects and 46.7 ± 8.6 mW/g in patients in group IIa (NS). In normal subjects it remained essentially unchanged when the rate was altered to 120/min. In group IIa it decreased by 26 per cent to 34.7 ± 8.2 mW/g ($P < 0.001$) (Fig. 4). The difference between normal subjects and patients in

group IIa at a heart rate of 120/min was significant ($P < 0.05$).

Discussion

The left ventricle works as, and is broadly similar to, a mechanical hydraulic pump. In mechanics, pumps are characterised by their peak and mean hydraulic power for a given cycle length and the data needed for the determination of pump power are pressure, volume, and time. However, since ventricular power changes dynamically throughout the cardiac cycle, these indices have to be analysed simultaneously in adequate detail for its study. Furthermore, it is essential to relate ventricular power to a constant cycle length, that is to heart rate.

Bearing these points in mind this investigation was designed to determine the levels of ventricular power during ejection at rest and under stress in normals and in patients with coronary heart disease. Chronic ischaemia of the myocardium as a result of coronary artery stenosis leads to impairment of ventricular pump function, and while this has been shown for other ejection phase indices of ventricular performance (ejection fraction, stroke volume, cardiac output, stroke work, etc. (Sonnenblick and Strobeck, 1977) these do not allow for time, which is an important variable in myocardial dynamics.

Studies of ventricular power in animals and in man have been reported by Chapman *et al.* (1959), Bunnell *et al.* (1962), Hernandez *et al.* (1964), Greenfield *et al.* (1968), and Snell and Luchsinger (1965). Russel *et al.* (1971) and Stein and Sabbah (1976 a, b) investigated left ventricular ejection power in various cardiac abnormalities at rest, but their data can only partly be compared with our own; in particular the heart rate was not kept constant. In our patients, however, this was achieved by the use of atrial pacing, both for basal measurements (at 80/min) and for stress testing (at 120/min). It has been shown previously that rapid atrial pacing is an acceptable method of stressing the heart (Forrester *et al.*, 1971) and in our opinion it is superior to others, such as treadmill testing, pharmacological intervention, or volume loading, for it interferes very little with the systemic circulation in terms of peripheral resistance, mean arterial pressure, and intravascular volume. In addition, it gives results of high reproducibility, which aids the direct comparison of different subjects.

Our findings showed that measured peak left ventricular ejection power under basal conditions was essentially the same in both normal subjects and patients with coronary artery disease, with

Fig. 4 Peak left ventricular ejection power normalised by left ventricular muscle mass. Normal subjects (I) and patients with coronary artery disease without myocardial infarction (IIa). Atrial pacing 80 and 120/min.

and without myocardial infarction, though the latter tended to have higher values. This may be explained by the myocardial hypertrophy (increased muscle mass) which was found in this group. However, under stress, peak power fell appreciably in both coronary groups (while in normal subjects it tended to rise) and this fall was statistically significant in the patients without infarction, even though there was no statistical difference in peak left ventricular power at either heart rate between the groups. Similar results were obtained for measured mean left ventricular power and it seemed necessary, therefore, to 'normalise' power by relating it to indices which represent the dimension of the ventricle and the mass of its wall. End-diastolic volume, as a measure of muscle fibre length before contraction, was higher in the patients with coronary artery disease than in the normal subjects. This finding is well known (Dwyer, 1970); chronic myocardial ischaemia induces ventricular hypertrophy and dilatation. At the basal heart rate, peak left ventricular power normalised by end-diastolic volume was in the same range in all the 3 groups, though patients with infarction tended to have lower values. When the heart rate was increased, this rose significantly in normal subjects but remained unchanged in both groups of patients with coronary artery disease. Mean left ventricular power normalised by end-diastolic volume, however, was significantly lower at both heart rates in patients with infarction than in normal subjects, and the rate increase led to a rise in this index in normal subjects but not in either coronary group. These results indicate that whereas, normally, ventricular power rises with heart rate, in coronary disease the limited oxygen supply prevents this.

Left ventricular muscle mass was determined in normal subjects and the group without infarction only; it was significantly higher in the latter. Peak power values normalised by muscle mass at rest were in the same range in both, but under stress decreased significantly in the coronary group alone. Similar results were obtained for mean power/g muscle mass. Thus, myocardial hypertrophy in chronic ischaemic heart disease without infarction compensates only in part for loss of pump function. Under moderate stress (here, tachycardia at 120/min) the energy output per unit of muscle mass not only cannot be maintained but indeed falls to critical levels, indicating impending heart failure. This study, then, proves left ventricular power to be of value in delineating myocardial performance at rest and under stress. Further work is needed to determine the influence of the extent and localisation of coronary artery lesions, of arrhythmias, and of drugs, on this measurement.

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