Assessment of left ventricular function by indices derived from aortic flow velocity

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The velocity and acceleration of aortic blood flow were measured by means of a catheter velocity probe in 40 patients during routine diagnostic cardiac catheterization. Ten different variables were derived from the aortic velocity measurements, and their ability to discriminate between good and bad left ventricular (LV) function was tested.

By means of eight conventional indices of LV function derived from pressure, mean flow, and quantitative cineangiography, the patients were divided into 3 groups: group 1, good LV function; group 2, moderate LV function; group 3, poor LV function.

Aortic peak velocity and maximal acceleration correlated well with stroke volume and were thus indices of LV pump function. Aortic peak velocity also showed a significant correlation with LV stroke work. Both aortic peak velocity and maximal acceleration failed to discriminate between the three groups of patients, and correlated poorly with conventional indices of LV function. The mean values of stroke volume differed significantly between groups 1 and 2, and between groups 1 and 3, and also correlated better with the conventional functional indices.

The best discrimination between normal and abnormal LV function was provided by dividing stroke volume by maximal acceleration, but stroke volume divided by peak velocity discriminated better than stroke volume alone. Stroke volume divided by maximal acceleration also gave more significant individual correlations with the conventional functional indices than did any other variable derived from aortic velocity.

The 40 patients, all undergoing routine diagnostic right and left heart catheterization, comprised 24 men and 16 women (mean age 50 years with a range of 26 to 68 years). In order to avoid errors of flow velocity measurement caused by jet effects and turbulent flow in the aorta, all patients with more than mild aortic stenosis or regurgitation were excluded.

Cardiac index (CI), left ventricular end-diastolic volume (LVEDV), ejection fraction (EF), normalized ejection rate (NER), peak mid-wall circumferential fibre shortening velocity (peak VCF), left ventricular end-diastolic pressure (LVEDP), maximal velocity of contractile element shortening at zero load multiplied by the series elastic stiffness constant (KVmax), and KVmax after an ectopic beat with the resulting post-ectopic potentiation of contractility (KVmaxPB), were used to divide the patients into three groups. Each of the above indices of LV function was scored 1 to 3, 1 representing a normal or near-normal value, 2 a moderately impaired value, and 3 a severely impaired value. The numerical values for each index determining its score are shown in Table 1. A final score for each left ventricle was obtained.
by averaging the scores of the individual indices. Thus, the
normal case has a score of 1 for each of the 8 indi-
vidual functional indices, and therefore a final average
score of 1 (8/8). Patients were placed in group 1 if their
final average score was 1-25 or less (allowing two in-
dividual functional index scores of 2, or one of 3, all the
other scores being 1). Patients in group 2 had final
average scores greater than 1-25 but less than 2-0, and
group 3 patients all had average scores of over 2-0.
Group 1 (little or no impairment of LV function)
contained 12 patients with mild organic disease (group
1a), and 6 patients with good LV function despite more
serious cardiac lesions tending to limit the cardiac output
(group 1b). Group 2 consisted of 13 patients with
moderate impairment of LV function, and group 3 of 9
cases with poor LV function. The clinical and haemo-
dynamic details of the patients are shown in Table 2,
together with their average LV functional scores.

**Methods**

The exact nature of the investigation, including the
measurement of aortic flow velocity, was explained to all
the patients before the study, in order to obtain in-
formed consent.

All patients were studied in the post-absorptive state
after prednisone with diazepam 10 mg and atropine
0-3 mg intramuscularly.

**Cardiac output**

This was measured from duplicate indocyanine-green
dye dilution curves using a Gilford densitometer and the
fore-'n-aft triangles method of calculation (Bradley and
Barr, 1969) in 34 patients, and by the Fick method in the
remaining 6 cases.

**Left ventricular volumes**

These were measured from LV cineangiograms taken
in the right anterior oblique position at a frame speed of
48/s. The LV cavity and free wall thickness were out-
lined in successive frames covering a complete cardiac
cycle using a light pen (Varian 4551) and computer
(Varian 620 L/100). Volumes were calculated by the area-
length method (Green et al., 1967), and the computer
was programmed to display left ventricular end-diastolic
volume (LVEDV), ejection fraction (EF), and normal-
ized ejection rate (NER). The ejection rate was
computed by linear regression analysis of the ejection
phase of the LV volume/time relation, and normalized
for LVEDV so that NER was expressed in EDV/s
(Hood, Rackley, and Rolett, 1968). The computer also
calculated midwall circumference (i) for each cineframe
as l=a(M+iW), where M=minor axis and W=wall
thickness. l plotted against time was differentiated to
give midwall circumferential fibre velocity (V_{CF}).
Peak V_{CF} was read directly from the computer plot of
V_{CF} against time, and mean V_{CF} calculated by plani-
metric integration of the V_{CF} (time curve during the
ejection phase of systole. Both peak and mean V_{CF} were
normalized for end-diastolic midwall circumference and
expressed in circumferences/s (Peterson et al., 1973).

**LV pressures**

These were measured by Telco (MM52) or Millar
micromanometers. Using the St. Thomas's Hospital long
sheath technique (Brooksby et al., 1974), these instru-
ments were introduced into the LV either retrogradely
across the aortic valve following Seldinger puncture of
the right femoral artery, or antegradely across the
mitral valve after transatrial puncture. The LV
pressure and its first derivative (dp/dt) were recorded,
and the LVEDP was measured at the start of the rapid
rise in dp/dt with the onset of systole. Zero reference
was taken as the sternal angle.

**KV\text{\text{max}}** and **KV\text{\text{maxPE}}**

A special processor, triggered by the R wave of the
electrocardiogram, continuously sampled the high fidelity
LV pressure signal from the Telco or Millar microman-
ometer from R wave to onset of ventricular con-
traction (a point selected manually by the operator). At this
point, the last sampled value of pressure was stored and
subtracted from the pressure signal for the rest of the
cardiac cycle to give LV developed pressure. The
logarithm of LV developed pressure was differentiated to
give dp/dt divided by developed pressure. This signal
(replicating velocity of contractile element shortening)
appeared on the vertical axis of an X-Y oscilloscope,
the LV pressure (representing force) appearing on the
horizontal axis. The isovolumetric portion of the
resulting 'force-velocity' loop was treated as a mono-
exponential curve and extrapolated to LVEDP (zero
developed pressure) to obtain KV\text{\text{max}} (Nejad et al.,
1971; Mirkjy, Ellison, and Hugenholtz, 1971; Grossman
et al., 1971). The pressure processor containing a unity
gain five-stage Thomson filter and differentiator had a
frequency response flat to 50 Hz, and a constant time
delay (8 ms) independent of frequency. It was also used
to differentiate pressure signals to give dp/dt, or flow
velocity signals to give acceleration.

As the calculation of KV\text{\text{max}} may be incorrect if there

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**TABLE 1 Values of indices of LV function used to assign patients to 3 functional groups**

<table>
<thead>
<tr>
<th>Index</th>
<th>Grade 1</th>
<th>Grade 2</th>
<th>Grade 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVEDV ml/m²</td>
<td>&lt;100</td>
<td>101-150</td>
<td>&gt;150</td>
</tr>
<tr>
<td>LVEDP kPa (mm Hg)</td>
<td>&lt;1.33</td>
<td>1.46-2.66</td>
<td>&gt;2.8</td>
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<tr>
<td>Cardiac index</td>
<td>2-0</td>
<td>1.2-1.99</td>
<td>&lt;1.2</td>
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<tr>
<td>1/min per m²</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ejection rate EDV/s</td>
<td>&gt;2.0</td>
<td>1.5-1.99</td>
<td>&lt;1.5</td>
</tr>
<tr>
<td>Peak VC circ/s</td>
<td>&gt;1.5</td>
<td>1.0-1.4</td>
<td>&lt;1.0</td>
</tr>
<tr>
<td>Ejection fraction</td>
<td>&gt;0.50</td>
<td>0.30-0.49</td>
<td>&lt;0.30</td>
</tr>
<tr>
<td>KV\text{\text{max}}(s⁻¹)</td>
<td>&gt;70</td>
<td>55-69</td>
<td>&lt;55</td>
</tr>
<tr>
<td>KV\text{\text{maxPE}}(s⁻¹)</td>
<td>&gt;150</td>
<td>100-149</td>
<td>&lt;100</td>
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</tbody>
</table>

For explanation of abbreviations, see text.
*In cases with diastolic overload of the LV a LVEDV ≈110 ml/m² was allowed.
†In cases of restriction a LVEDP <2.66 kPa (20 mmHg) was allowed.
### Table 2: Clinical and Haemodynamic Data

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Sex</th>
<th>Age</th>
<th>Diagnosis</th>
<th>Cardiac index</th>
<th>Stroke volume</th>
<th>LV Pressure</th>
<th>Flow probe</th>
<th>LVV</th>
<th>Ejection rate</th>
<th>Ejection fraction</th>
<th>Peak VCF</th>
<th>Mean VCF</th>
<th>dP/dt</th>
<th>Mean pressure</th>
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<td>Heart rate</td>
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<td>Diastolic</td>
<td>EDV</td>
<td>ml/m²</td>
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<td>kPa</td>
<td>EDV</td>
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<td>ml/s</td>
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<td>(mmHg)</td>
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<td>(130)</td>
<td>ml/s</td>
<td>(120)</td>
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<td>(mmHg)</td>
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<td>ml/s</td>
<td>(120)</td>
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<td>(mmHg)</td>
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</table>

**Group 1A**

1. M 60 IHD, AR 1/4
2. M 58 MS 1/4, MR trivial—AR 1/4
3. F 35 MS 1/4, AR 1/4
4. M 48 MS 1/4, AR 1/4
5. F 55 AR 2/4
6. M 34 IHD+MI
7. M 41 HOCM
8. F 50 MR 1/4, trivial MS+AR
9. M 38 IHD
10. M 39 IHD—MR 2/4 due to papillary muscle dysfunction
11. M 36 Neuromuscular chest pain
12. F 45 Essential hypertension, chest pain, MR 1/4

**Group 1B**

13. F 65 MS 2-3/4, minimal MR+AR
14. F 48 MS 2-3/4, minimal MR+AR
15. F 51 Normally functioning MVP—TR 2-3/4
16. M 57 Restrictive cardiomyopathy+MR 2-4+TR 2/4
17. M 63 Constrictive pericarditis
18. F 60 Normally functioning MVP—TR 2-3/4

**Mean values**

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<thead>
<tr>
<th></th>
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<td>Cardiac index</td>
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<td>Stroke volume</td>
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<td>0-52</td>
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<td>LV Pressure</td>
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<td>Flow probe</td>
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<tr>
<td>LVV</td>
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<td>Ejection fraction</td>
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<td>Peak VCF</td>
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<tr>
<td>Mean VCF</td>
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<td>dP/dt</td>
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<tr>
<td>Mean pressure</td>
<td>108</td>
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</table>

**Group 2**

19. M 52 CCM
20. F 57 IHD+MI+LV aneurysm
21. M 54 MS 2/4, minimal MR+AR
22. M 52 MR 3/4
23. F 50 MR 2-3/4, MS 1-2/4
Assessment of left ventricular function

<table>
<thead>
<tr>
<th>KV&lt;sub&gt;m&lt;/sub&gt; PE&lt;sup&gt;−1&lt;/sup&gt;</th>
<th>KV&lt;sub&gt;max&lt;/sub&gt; PE&lt;sup&gt;−1&lt;/sup&gt;</th>
<th>Mean velocity cm/s</th>
<th>Peak velocity cm/s</th>
<th>Maximal Stroke work joules</th>
<th>LVPP Watts g×m/s</th>
<th>Average LV functional score</th>
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<tr>
<td>118 294</td>
<td>8.5</td>
<td>64.1</td>
<td>1040</td>
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<td>10-63</td>
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<tr>
<td>100 180</td>
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<td>28.3</td>
<td>773</td>
<td>0.616</td>
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<td>155 —</td>
<td>6.8</td>
<td>27.8</td>
<td>860</td>
<td>—</td>
<td>3.77</td>
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<td>92 152</td>
<td>9.6</td>
<td>45.5</td>
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<td>—</td>
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<td>1.125</td>
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<td>20.6</td>
<td>450</td>
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<tr>
<td>315.3 ± 0.1</td>
<td>9.85 ± 2.9</td>
<td>43.5 ± 12.8</td>
<td>788</td>
<td>6.69 ± 2.4</td>
<td>1.285</td>
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<td>50-9 ± 81-6</td>
<td>9.85 ± 2.9</td>
<td>43.5 ± 12.8</td>
<td>788</td>
<td>6.69 ± 2.4</td>
<td>1.285</td>
<td>1.0</td>
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<td>62 110</td>
<td>8.93</td>
<td>31.8</td>
<td>860</td>
<td>—</td>
<td>3.55</td>
<td>1.5</td>
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<td>120 160</td>
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<td>18.2</td>
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<td>1456</td>
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<td>67 88</td>
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<td>49</td>
<td>914</td>
<td>0.608 (62.0)</td>
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<tr>
<td>76 136</td>
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<td>57.5</td>
<td>861</td>
<td>0.971 (99)</td>
<td>6.87</td>
<td>1.625</td>
</tr>
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</table>

are major abnormalities of segmental wall motion (Sonnenblick et al., 1970), KV<sub>max</sub> and KV<sub>max</sub>PE were not used in the correlations in 5 patients with ischaemic heart disease who had obvious LV dyskinesia on cineangiography. As there is evidence that mitral regurgitation does not invalidate the calculation of KV<sub>max</sub> from pressure, presumably because the largest values of contractile element velocity occur early in systole when the inertia of the blood in the ventricle has delayed a significant volume change (Falsetti et al., 1971), patients with mitral regurgitation were included in this part of the study.

**Aortic flow velocity**

This was measured in the ascending aorta 4 to 5 cm above the aortic valve using a Carolina Medical Electronics intravascular flow velocity probe (7 FG), and the CME Model 601D 'Cliniflow' square-wave electromagnetic flowmeter. This instrument is completely patient isolated, extremely simple to use, and can be provided with factory-calibrated probes if so desired. It has a frequency response that is -2dB at 10 Hz, and -18dB at 30 Hz. The phase shift is linearly related to frequency with a time delay of 37 ms ±3 ms in the frequency range 1 Hz to 30 Hz. Because of the electrical environment in our laboratory, the velocity signal from the flowmeter was still unacceptably 'noisy', and was, therefore, passed through a 12 Hz low-pass filter (frequency response -2 dB at 10 Hz, and -18 dB at 30 Hz, with a constant time delay of 25 ms).

By passing a cosine squared pulse from a signal generator through this filter and recording the original signal and the filtered wave form for different values of base-width, together with their first derivatives, it was possible to show that 'peak velocity' was attenuated by 0, 1-5, 6-1, and 10-6 per cent, at base widths of 500, 240, and 100 ms, respectively. The corresponding attenuations of 'maximal acceleration' were 0, 2, 14, and 25-7 per cent. With the flowmeter and filter in series (both having the same frequency response), the attenuation of peak velocity and maximal acceleration are just under twice the values obtained with the filter alone. The base-widths below which the percentage attenuation exceeds 5 are 190 ms for peak velocity and 230 ms for maximal acceleration. Base-widths of under 230 ms were in fact observed in 8 of the 40 patients studied (Cases 20, 26, 31, 32, 33, 34, 35, 38), under 190 ms in only 1 patient (Case 33). In the patient with the smallest base-width (175 ms) peak velocity was attenuated by 6-1 per cent and maximal acceleration by 12-2 per cent. These results are in keeping with Gabe's work on pressure waveforms using a similar filter (Gabe, 1972).

The catheter velocity probe was passed to the ascending aorta through a long sheath after Seldinger puncture of the femoral artery. Previous workers (Gabe et al., 1969; Bennett et al., 1974) were unable in some patients to pass the flow probe to ascending aorta from brachial artery. In no case in the present series did it prove impossible to position the probe in ascending aorta. Particular care was taken to avoid angulation and undue movement of the probe, and the position and stability of the velocity sensor were verified from cineradiographs.
<table>
<thead>
<tr>
<th>Case No.</th>
<th>Sex</th>
<th>Age</th>
<th>Diagnosis</th>
<th>Heart rate</th>
<th>Stroke volume</th>
<th>Cardiac index</th>
<th>Systolic pressure</th>
<th>Diastolic pressure</th>
<th>LV EDV</th>
<th>Ejection fraction</th>
<th>Peak VCF</th>
<th>Mean VCF</th>
<th>dP/dt</th>
</tr>
</thead>
<tbody>
<tr>
<td>24</td>
<td>M</td>
<td>54</td>
<td>CCM; complete AV block</td>
<td>62</td>
<td>33-3</td>
<td>4-3</td>
<td>13-3</td>
<td>0-53</td>
<td>106</td>
<td>1-02</td>
<td>0-36</td>
<td>1-44</td>
<td>—</td>
</tr>
<tr>
<td>25</td>
<td>F</td>
<td>62</td>
<td>MS 2-3/4</td>
<td>94</td>
<td>33-0</td>
<td>6-6</td>
<td>16</td>
<td>1-33</td>
<td>74</td>
<td>1-78</td>
<td>0-32</td>
<td>1-25</td>
<td>—</td>
</tr>
<tr>
<td>26</td>
<td>F</td>
<td>58</td>
<td>Angina with normal coronary arteries</td>
<td>95</td>
<td>20-6</td>
<td>9-2</td>
<td>16</td>
<td>2-66</td>
<td>138</td>
<td>1-91</td>
<td>0-44</td>
<td>0-9</td>
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</tr>
<tr>
<td>27</td>
<td>M</td>
<td>43</td>
<td>IHD—MI</td>
<td>60</td>
<td>49-0</td>
<td>5-0</td>
<td>16-3</td>
<td>0-66</td>
<td>89</td>
<td>2-87</td>
<td>0-60</td>
<td>1-0</td>
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</tr>
<tr>
<td>28</td>
<td>M</td>
<td>57</td>
<td>MS 1/4 rheumatic myocardial involv.</td>
<td>100</td>
<td>47-5</td>
<td>3-9</td>
<td>21-3</td>
<td>2-93</td>
<td>102</td>
<td>2-05</td>
<td>0-53</td>
<td>1-45</td>
<td>0-74</td>
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<tr>
<td>29</td>
<td>F</td>
<td>46</td>
<td>MS 1/4</td>
<td>75</td>
<td>56</td>
<td>2-6</td>
<td>17-3</td>
<td>0-66</td>
<td>169</td>
<td>1-53</td>
<td>0-36</td>
<td>0-8</td>
<td>—</td>
</tr>
<tr>
<td>30</td>
<td>M</td>
<td>57</td>
<td>IHD—MI</td>
<td>120</td>
<td>20-6</td>
<td>2-2</td>
<td>16</td>
<td>0</td>
<td>74</td>
<td>2-78</td>
<td>0-55</td>
<td>1-1</td>
<td>0-62</td>
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<tr>
<td>31</td>
<td>M</td>
<td>26</td>
<td>Early CCM</td>
<td>88</td>
<td>44-3</td>
<td>2-6</td>
<td>16-3</td>
<td>1-32</td>
<td>34-7</td>
<td>0-58</td>
<td>0-141</td>
<td>0-24</td>
<td>0-09</td>
</tr>
<tr>
<td>Mean</td>
<td>51-4</td>
<td>9-2</td>
<td>17-6</td>
<td>0-91</td>
<td>17-4</td>
<td>16-3</td>
<td>1-32</td>
<td>34-7</td>
<td>0-58</td>
<td>0-141</td>
<td>0-24</td>
<td>0-09</td>
<td>504</td>
</tr>
<tr>
<td>±1SD</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
</tbody>
</table>

**Group 3**

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Sex</th>
<th>Age</th>
<th>Diagnosis</th>
<th>Heart rate</th>
<th>Stroke volume</th>
<th>Cardiac index</th>
<th>Systolic pressure</th>
<th>Diastolic pressure</th>
<th>LV EDV</th>
<th>Ejection fraction</th>
<th>Peak VCF</th>
<th>Mean VCF</th>
<th>dP/dt</th>
</tr>
</thead>
<tbody>
<tr>
<td>32</td>
<td>M</td>
<td>54</td>
<td>CCM</td>
<td>91</td>
<td>20-7</td>
<td>2-5</td>
<td>14-4</td>
<td>2-77</td>
<td>257</td>
<td>0-6</td>
<td>0-18</td>
<td>0-6</td>
<td>0-16</td>
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<tr>
<td>33</td>
<td>F</td>
<td>65</td>
<td>Essential hypertension</td>
<td>93</td>
<td>17</td>
<td>1-8</td>
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<td>100</td>
<td>2-03</td>
<td>0-52</td>
<td>0-78</td>
<td>0-39</td>
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<tr>
<td>34</td>
<td>M</td>
<td>52</td>
<td>MR 2/4 AR 1-2/4 past history of SBE</td>
<td>101</td>
<td>16</td>
<td>1-6</td>
<td>20-7</td>
<td>2-53</td>
<td>84</td>
<td>1-62</td>
<td>0-25</td>
<td>0-9</td>
<td>0-42</td>
</tr>
<tr>
<td>35</td>
<td>M</td>
<td>55</td>
<td>Normaly functioning MVP; ASD (? dilated foramen ovale) PH of SBE</td>
<td>98</td>
<td>20-7</td>
<td>1-7</td>
<td>12-6</td>
<td>3-33</td>
<td>280</td>
<td>0-48</td>
<td>0-22</td>
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<tr>
<td>Mean</td>
<td>51-2</td>
<td>12-5</td>
<td>17-8</td>
<td>0-67</td>
<td>13-2</td>
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<td>3-33</td>
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</table>

**Statistical significance of difference of mean values**

<table>
<thead>
<tr>
<th>Group 1 v. 2</th>
</tr>
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<tbody>
<tr>
<td>NS</td>
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<tr>
<td>Group 2 v. 3</td>
</tr>
<tr>
<td>NS</td>
</tr>
<tr>
<td>Group 1 &amp; 3</td>
</tr>
<tr>
<td>NS</td>
</tr>
</tbody>
</table>

**Footnote:** AF—Atrial fibrillation; A.Fl.—atrial flutter; AR—aortic regurgitation; AS—aortic stenosis; ASD—atrial septal defect; CCM—congestive cardiomyopathy; HOCM—hypertrophic obstructive cardiomyopathy; IHD—ischaemic heart disease; MI—myocardial infarction; MR—mitral regurgitation; MS—mitral stenosis; MVP—mitral valve prosthesis; SBE—subacute bacterial endocarditis; SR—sinus rhythm; TR—tricuspid regurgitation; TVP—tricuspid valve prosthesis. F denotes Fick estimation.
Stabilization of the probe was facilitated by its tapering tip extending 6 cm beyond the velocity sensor (Jewitt et al., 1974).

The same probe was used in all patients, and was calibrated in normal saline (NaCl 0-15 mol/l) using a special flow rig. The probe was inserted through a water-tight sleeve into a glass tube of internal diameter 1-42 cm. An electric pump circulated the saline from a reservoir through a rotameter, and then via the glass tube containing the probe back to the reservoir. The volume of flow could be adjusted by a tap downstream to the rotameter, the accuracy of which was confirmed by timed volume collections. After establishing a true zero reading, flow was adjusted to give velocities of 10, 20, and 40 cm/s in the glass tube, and the 'probe factor' control on the flowmeter adjusted until the appropriate mean velocity appeared on the digital meter. Flow was then reversed in the tube and the process repeated. Over this range of velocities, and with this probe, the 'probe factor' control setting varied by not more than 8 per cent. Before each insertion of the probe into a patient, it was zeroed in a bowl of sterile saline, and the zero obtained in this way did not differ from the assumed zero velocity occurring late in diastole (Hoffman et al., 1965).

Using this zero, peak velocity (PV) was measured directly from the velocity record in 10 consecutive beats. The velocity signal was also differentiated to give acceleration, and maximal acceleration (MA) measured for the same 10 beats. Mean velocity (MV) was derived by planimetric integration of the area under the velocity curve for the 10 beats. The flow probe cardiac output was obtained by multiplying MV by 60 x aortic cross-sectional area. The aortic diameter was measured from aortograms, and cross-sectional area obtained after correction for magnification from πr². The flow probe stroke volume (SV) was the cardiac output/heart rate. Peak aortic flow (ml/s) was given by aortic peak velocity (PV) x aortic cross-sectional area. LV peak power (LVPP) was calculated from the formula:

\[
\text{LVPP} (\text{g m/s}) = \frac{\text{Peak aortic flow} \times \text{peak LV developed pressure (mm Hg)} \times 1.36}{100}
\]

in SI units:

\[
\text{LVPP (watts) = Peak aortic flow} (\text{ml/s}) \times \text{peak LV dev. press. (kPa)} \times 10^{-3}
\]

This value for LVPP is a close approximation, since peak aortic flow occurs just before peak LV or aortic pressure. Fig. 1 shows simultaneous aortic flow and pressure, and Fig. 4 simultaneous aortic flow and LV pressure. The flow signal has a total transit time of 62 ms (37 ms for the flowmeter, and 25 ms for the filter), and if allowance is made for this then peak flow precedes peak LV or aortic pressure by 20 to 90 ms.

In 18 patients in whom simultaneous LV and aortic pressures were recorded, LV stroke work (LVSW) was calculated:

\[
\text{LVSW (g m) = Flow probe SV} \times (\text{LVSP-LVEDP mm Hg}) \times 1.36
\]

100
In SI units:

\[ \text{LVSW (joules)} = \text{SV (ml)} \times (\text{LVSP} - \text{LVEDP} \text{ kPa}) \times 10^{-3} \]

Where LVSP is the mean LV pressure during ejection obtained by planimetric integration of the area of the LV pressure curve contained between the beginning of the upstroke and the incisura of the aortic pressure curve (Covell et al., 1966).

All pressure and flow signals were recorded on a Philips 'Analog-Seven' tape recorder together with the electrocardiogram. Selected periods of tape-recorded data were replayed onto a Cambridge twelve channel photographic recorder.

The mean values of the various indices of LV function obtained in each of the three groups of patients were compared using Student's t test for unpaired data. Correlations between different indices of function were carried out by least squares regression analysis. The statistical significance of the correlation coefficient (r value) was tested by the formula:

\[ t = \sqrt{\frac{r^2 \times (N-2)}{1-r^2}} \]  

(Croxton, 1953).

**FIG. 1** Aortic flow velocity (third tracing from top) recorded simultaneously with aortic pressure (Millar micromanometer—second tracing from top). Flow acceleration (top) and electrocardiogram (bottom) also shown. Flow velocity tracing delayed by 62 ms so peak velocity precedes peak aortic pressure by some 80 ms.

**Results**

The clinical and haemodynamic data of the 40 cases studied are shown in Table 2. Aortic peak velocity (PV) and aortic maximal acceleration (MA) showed an excellent linear correlation \( (r=0.71; P<0.001) \). There was also good correlation between PV and stroke volume (SV) \( (r=0.73; P<0.001) \)—Fig. 2, and between MA and SV \( (r=0.755; P<0.001) \)—Fig. 3. This dependence of PV and MA on SV is further illustrated in Fig. 4. Fig. 4 shows aortic flow velocity, LV pressure, and dP/dt in a patient with congestive cardiomyopathy and complete heart block. PV and MA in this patient were low, averaging 20.4 cm/s and 443 cm/s², respectively. It can be seen, however, that whenever the PR interval fell between 100 and 280 ms, PV was obviously higher with a concomitant increase in LV peak pressure and max. dP/dt. Thus, the increase in SV resulting from a well-timed atrial contraction was reflected by an increase in PV. In 2 patients, LV pressure alternans developed during pacing. Alternation of both PV and MA accompanied the alternation of SV and LV peak pressure. PV also correlated well with LVSW \( (r=0.70; P<0.005) \) in the 18 patients in whom it was possible to calculate LVSW. That this was not entirely because of the good correlation between PV and SV is suggested by the fact that there was a small but significant positive correlation in all 40 cases between PV and LV peak developed.

**FIG. 2** Peak velocity plotted against flow probe stroke volume in 40 patients. \( R=0.73, P<0.001, Y=0.23X+22.27 \).

**FIG. 3** Maximal acceleration plotted against flow probe stroke volume in 40 patients. \( R=0.755, P<0.001, Y=9.15X+306.8 \).
pressure \((r=0.383; \, P<0.02)\). Fig. 5a shows SV, Fig. 5b PV, and Fig. 5c MA values obtained in patients considered to have normal or near-normal LV function (group 1), moderate impairment of function (group 2), and severe impairment of function (group 3).

The mean values of SV obtained in the three groups (group 1: 58.8 ± 15.0 ml—mean ± SD; group 2: 44.3 ± 17.4 ml; group 3: 31.6 ± 13.2 ml) show a barely significant difference \((P<0.025)\) between groups 1 and 2, no significant difference between groups 2 and 3, and a highly significant difference \((P<0.001)\) between groups 1 and 3. The mean values of PV (group 1: 43.5 ± 12.8 cm/s; group 2: 37.4 ± 14.9 cm/s; group 3: 33.3 ± 11.8 cm/s) do not differ significantly. The differences of the mean values of MA (group 1: 788 ± 171 cm/s²; group 2: 767 ± 291 cm/s²; group 3: 631 ± 200 cm/s²) just reach statistical significance \((P<0.05)\) between groups 1 and 3 only.

In order to see if these results were influenced by the inclusion in group 1 of 6 patients with significant organic heart disease but relatively unimpaired LV function (group 1b), these patients were excluded and the results recalculated. The mean SV for group 1a was 62.4 ± 16.3 ml, mean PV 46.3 ± 12.3 cm/s, and mean MA 820 ± 148 cm/s². The differences in SV became more significant \((P<0.01)\) between groups 1a and 2, and remained highly significant \((P<0.001)\) between groups 1a and 3.

The differences in the mean PV just reached statistical significance \((P<0.05)\) between groups 1a and 3, and those for MA did not change. Fig. 5a, 5b, and 5c show the large overlap in values of SV, PV, and MA between the three groups of patients, and suggests that in the individual case PV and MA (and to a lesser extent SV) are insensitive indices of LV function.

In an attempt to correct for the dependency of PV and MA on SV, the SV was divided by either PV or MA. Fig. 5d shows the values of SV/MA, and Fig. 5c those for SV/PV in the three groups of patients studied. In particular, SV/MA distinguished well between patients with good and bad LV function, the difference of the mean values of this ‘flow fraction’ being highly significant between groups 1 and 2 \((P<0.005)\), and between groups 1 and 3 \((P<0.001)\). The fraction SV/PV was less sensitive, since the difference between groups 1 and 2 did not reach statistical significance, though the difference between groups 1 and 3 was again highly significant \((P<0.001)\). The use of these fractions clearly allowed a much better separation of the patients in the three functional groups than did either PV or MA alone. SV/MA also proved superior to SV alone in discriminating between patients with good and bad LV function.

Fig. 6 and 7 show the distribution of the other indices of LV performance between the three groups of patients. Using any single index of function, there is some overlap between individual patients in the three groups, despite the fact that each index (except max. \(dP/dt\) and mean \(V_{CF}\)) shown in these figures was used to determine the functional group to which the patient was assigned. The best single discriminatory index in this array of tests of LV function was the ejection fraction.

In order to evaluate further the ability of the velocity measurements to assess LV performance, each of them was separately correlated against seven indices of LV function derived from LV pressure or LV volume measurements using least squares regression analysis. Table 3 gives the results of this analysis, the number of patients, the \(r\) value, and the \(P\) value for each correlation. Mean velocity \((MV)\) just correlated significantly with mean \(V_{CF}\), max \(dP/dt\), and ejection fraction, but a more significant correlation was obtained with \(K_{V_{max}}\), in particular with \(K_{V_{max}PE}\).

PV showed a barely significant correlation with max \(dP/dt\), and a slightly better correlation with \(K_{V_{max}PE}\). The other correlations were not significant.

MA showed no significant correlation with any of the other indices of LV function tested.

Flow probe stroke volume (SV) correlated signi-
FIG. 5 Values of flow probe stroke volume, peak velocity, maximal acceleration, stroke volume/ maximal acceleration, and stroke volume/peak velocity obtained in 40 patients divided into 3 groups. Group 1: patients with near-normal LV function. Group 2: moderate impairment of LV function. Group 3: poor LV function. Also shown are mean ± SD for each variable and each group. NS = not significant. P values indicated at bottom.

FIG. 6 Values of peak $V_{CP}$, mean $V_{CP}$, normalized ejection rate, and ejection fraction obtained by quantitative LV cineangiography in 40 patients divided into 3 functional groups. Also shown are mean ± SD for each variable and each group. NS = not significant. P values indicated at bottom.
Assessment of left ventricular function

FIG. 7 Values of Max dP/dt, \( KV_{\text{max}} \), and \( KV_{\text{max}} \)PE obtained from LV micromanometer pressure records in 40 patients divided into 3 functional groups. Also shown are mean±SD for each variable and each group. NS = not significant. P values indicated at bottom.

TABLE 3 Correlation coefficients between indices of LV function derived from flow velocity and those derived from pressure and quantitative cineangiography

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean VCF circ/s</th>
<th>Peak VCF circ/s</th>
<th>Ejection rate EDV/s</th>
<th>Ejection fraction n=40</th>
<th>Max dP/dt N=40</th>
<th>KV(_{\text{max}}) N=36</th>
<th>KV(_{\text{max}})PE N=34</th>
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</thead>
<tbody>
<tr>
<td>Flow probe stroke volume</td>
<td>R=0.484 P&lt;0.025</td>
<td>R=0.47 P&lt;0.005</td>
<td>R=0.556 P&lt;0.001*</td>
<td>R=0.376 P&lt;0.001</td>
<td>R=0.389 P&lt;0.025</td>
<td>R=0.387 P&lt;0.001*</td>
<td></td>
</tr>
<tr>
<td>Peak velocity</td>
<td>R=0.272 NS</td>
<td>R=0.122 NS</td>
<td>R=0.272 P=0.02</td>
<td>R=0.318 P&lt;0.001</td>
<td>R=0.307 R&lt;0.01</td>
<td>R=0.439 P&lt;0.001*</td>
<td></td>
</tr>
<tr>
<td>Maximal acceleration</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>R=0.289 NS</td>
<td>P&lt;0.02</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean velocity</td>
<td>R=0.03 NS</td>
<td>R=0.2 NS</td>
<td>R=0.34 NS</td>
<td>R=0.393 R&lt;0.001</td>
<td>R=0.491 R&lt;0.001</td>
<td>R=0.527 R&lt;0.001*</td>
<td></td>
</tr>
<tr>
<td>Peak power</td>
<td>R=0.291 NS</td>
<td>R=0.449 NS</td>
<td>R=0.222 NS</td>
<td>R=0.476 R&lt;0.001</td>
<td>R=0.475 R&lt;0.001</td>
<td>R=0.495 R&lt;0.001*</td>
<td></td>
</tr>
<tr>
<td>Mean velocity/max. acceleration</td>
<td>R=0.558 NS</td>
<td>R=0.235 NS</td>
<td>R=0.424 NS</td>
<td>R=0.293 R&lt;0.001</td>
<td>R=0.498 R&lt;0.001</td>
<td>R=0.570 R&lt;0.001*</td>
<td></td>
</tr>
<tr>
<td>Peak velocity/max. acceleration</td>
<td>R=0.475 NS</td>
<td>R=0.154 NS</td>
<td>R=0.135 NS</td>
<td>R=0.136 R&lt;0.001</td>
<td>R=0.261 R&lt;0.001</td>
<td>R=0.517 R&lt;0.001*</td>
<td></td>
</tr>
<tr>
<td>Stroke volume/max. acceleration</td>
<td>R=0.719 P&lt;0.001*</td>
<td>R=0.651 NS</td>
<td>R=0.396 NS</td>
<td>R=0.565 R&lt;0.001</td>
<td>R=0.543 R&lt;0.001</td>
<td>R=0.514 R&lt;0.001*</td>
<td></td>
</tr>
<tr>
<td>Stroke volume/peak velocity</td>
<td>R=0.473 R&lt;0.001*</td>
<td>R=0.522 NS</td>
<td>R=0.262 NS</td>
<td>R=0.453 R&lt;0.001</td>
<td>R=0.252 R&lt;0.001</td>
<td>R=0.10 R&lt;0.001*</td>
<td></td>
</tr>
<tr>
<td>Peak power/max. acceleration</td>
<td>R=0.418 NS</td>
<td>R=0.552 NS</td>
<td>R=0.209 NS</td>
<td>R=0.484 R&lt;0.001</td>
<td>R=0.454 R&lt;0.001</td>
<td>R=0.296 R&lt;0.001*</td>
<td></td>
</tr>
</tbody>
</table>

*Highly significant correlation.
Kolettis, Jenkins, and Webb-Peploe

significantly with mean $V_{CF}$, peak $V_{CF}$, NER, ejection fraction, $KV_{max}$, and $KV_{maxPE}$; the best correlation being with ejection fraction.

Peak power (LVPP) correlated significantly with peak $V_{CF}$, ejection fraction, max dP/dt and $KV_{maxPE}$.

$MV/MA$ just correlated significantly with mean $V_{CF}$, NER, and somewhat better with $KV_{max}$ and $KV_{maxPE}$.

PV/MA proved disappointing, correlating only with mean $V_{CF}$ and $KV_{maxPE}$.

The 'flow fractions' SV/MA, and SV/PV did considerably better. In particular, SV/MA correlated well with all other indices of LV function except max dP/dt (Table 3) LVPP/MA correlated well with peak $V_{CF}$, EF, max dP/dt and $KV_{maxPE}$.

Discussion

Despite much progress in the past 15 years, the search for better methods of assessing left ventricular function continues. In intact man, the performance of the left ventricular pump depends not merely on the state of the myocardium and the efficiency of the heart valves, but also on sympathetic nervous impulses, circulating catecholamines, the filling pressure of the pump (preload), and the load against which it has to eject (afterload). It is not surprising, therefore, that measurements of pressure and mean flow (cardiac output, stroke volume, stroke work, etc.), though useful in detecting directional changes in the same patients, are of limited value in comparisons between patients (Braunwald and Ross, 1964; Braunwald et al., 1969). The rate of rise of LV pressure (dP/dt) also has limitations in the evaluation of myocardial contractility in man (Mason, 1969).

With the application of Hill's work on skeletal muscle (Hill, 1938) to isolated heart muscle (Abbott and Mommaerts, 1959; Sonnenblick, 1962; Brady, 1965), and subsequently to the intact human heart (Hugenholtz et al., 1970; Mason, Spann, and Zelis, 1970; Falsetti et al., 1971), it was hoped that $V_{max}$—the velocity of contractile element shortening at zero load—would provide a valid measure of LV contractility. The determination of $V_{max}$ in the intact human heart is, however, based on a number of assumptions that are open to question, and also presents certain practical difficulties (Mirsky et al., 1971, 1974). Its independence of preload has also been challenged (Noble, Bowen, and Hefner, 1969; Pollack, 1970; Nejad et al., 1971). The use of quantitative angiography to assess LV function is based on assumptions about ventricular geometry that may not be valid, especially in the presence of LV dyskinesia. The contrast medium temporarily depresses the function being measured so that continuous or even repetitive measurements are not possible. LV volume measurements are, moreover, extremely time consuming.

Experimental studies in dogs led Rushmer (1964) to suggest that MA was a sensitive index of LV performance. MA, PV, and SV have all been shown to reflect changes in LV contraction induced by positive inotropic stimulation (Noble et al., 1966; Ross et al., 1966) or by coronary artery ligation (Noble et al., 1966) and experimental heart failure (Ross, Covell, and Sonnenblick, 1967).

Normal values for PV and MA in man have not yet been established. PV values averaging 56.3 cm/s (Rudewald, 1962), ranging from 54–80 cm/s (Gabe et al., 1969) and of 67 and 83 cm/s (Bennett et al., 1974) have been reported in patients with apparently normal LV function. MA values averaging 1969 cm/s² (Rudewald, 1962) and of 1894, 1556, and 1510 cm/s² (Bennett et al., 1974) were obtained in the same patients; Gabe and co-workers (1969) did not measure MA.

The values for PV and MA obtained in our group 1 patients were lower than those quoted above. Electrical isolation from the patient in the CME 601D flowmeter has been achieved at the cost of some reduction in frequency response compared to their other models and to the instruments used by earlier workers (Gabe et al., 1969; Bennett et al., 1974; Jewitt et al., 1974). This is, we believe, a small price to pay for the increase in safety and ease of use of this flowmeter. As stated in the methods section, PV attenuation exceeding 5 per cent occurred in one patient only (6-1% attenuation), and MA attenuation over 5 per cent in 8 patients (the worst attenuation was 12.2%). It seems unlikely, therefore, that the lower values for PV and MA obtained in our group 1 patients compared with the 'normal subjects' of other workers is because of the frequency response of the CME 601D flowmeter. Only 1 of the 18 patients in group 1 had no demonstrable heart disease, and the relatively low CI and SV values obtained in many of them may have been in part because of diuretic therapy before study. Routine premedication with atropine also resulted in some increase in resting heart rate with a concomitant decrease in SV.

Both PV and MA proved to be highly dependent on SV (Fig. 2 and 3), and this was well illustrated in the patients with complete heart block (Fig. 4) and pulsus alternans. Benchimol et al. (1969), using a Doppler flow probe, have reported similar findings, and have also shown that PV correlates well with the length of the preceding RR interval in patients in atrial fibrillation, an observation since confirmed by Jewitt et al. (1974).
To determine SV continuously using the intra-vascular velocity probe requires a measurement of aortic cross-sectional area which, in intact man, can only be made either by aortography or by echocardiography. The good correlation between velocity probe SV and both PV and MA in the present study means that these two simple velocity measurements satisfactorily reflect the performance of the LV pump, and can be used to follow the course of the critically ill patient without the need to measure aortic cross-sectional area in order to calculate SV. PV and MA have indeed been shown to be superior to aortic pressure in assessing the progress of patients in the intensive care unit (Jewitt et al., 1974; Cross and Light, 1974).

PV and MA in the individual patient are, however, determined by the complex relation between the velocity and extent of shortening of each myocardial fibre, the size and geometry of the LV, and the cross-sectional area of the aorta. Thus, the failure of PV and MA to separate the three functional groups of patients in the present study is not altogether surprising. The dependence of these two variables on SV (itself greatly influenced by changes in preload and heart rate) limits their value in comparisons between patients. Noble et al. (1966) reported that MA in dogs was independent of postural changes, and, therefore, suggested that it was independent of preload. The good correlation between MA and SV in the present study makes this very unlikely. Van den Bos et al. (1973) have suggested that changes in MA are also inversely related to changes in afterload, and such a relation has indeed been demonstrated for PV and after-load (Ross et al., 1966).

The poor correlations between PV and MA and the other indices of LV function in the present study contrast with the report of Bennett et al. (1974) that both PV and MA correlated well with ejection fraction, and clearly separated their 3 normal subjects from the 9 cases with coronary artery disease. Their data show that in their relatively few patients, deterioration of LV function was accompanied by an increase in heart rate and a decrease in cardiac index. Stroke index calculated from their data was thus progressively reduced as ejection fraction fell, and correlated well with MA (r = 0.729), with PV (r = 0.744), and with ejection fraction itself (r = 0.728).

Mirsly et al. (1974) have suggested that ‘normalization’ of blood velocity measurements may result in better functional indices. From Fig. 5d and 5e it will be seen that SV/MA was the most satisfactory normalized velocity measurement in the separation of the good from the bad ventricles, followed by SV/PV. These results support the hypothesis that severe impairment of LV function may be associated with a relatively good initial acceleration of blood in the aorta, which, however, is not sustained by the failing ventricle (Cross and Light, 1974). This results in a reduction of PV, and in particular SV, compared to MA.

The failing ventricle, through an increase in its end-diastolic volume, is capable of ejecting a normal or near-normal stroke volume despite a fall in its ejection fraction. This ventricular dilatation is theoretically advantageous in that each individual myocardial fibre needs to shorten less to expel the same quantity of blood (Burch, Ray, and Cronvich, 1952). This advantage is, however, offset by the increased wall tension that the dilated ventricle must develop in order to generate the same pressure within its cavity (application of Laplace’s law to the ventricle—Burton, 1957). This increased wall tension results in increased myocardial oxygen demand. In a normal ventricle, which ejects at least 50 per cent of its end-diastolic volume, the change in wall radius is considerable. The decreasing radius offsets the increasing intracavitary pressure with the result that wall tension is not maintained at peak levels for long, but decreases early in the ejection phase. Dilatation of the LV with reduction of its ejection fraction means that higher wall tensions must be maintained for longer in order to eject the same stroke volume. The metabolic disadvantage of prolonged and increased wall tension, however, may result in failure to sustain the initial relatively good acceleration, as indicated by our findings in patients with poor LV function. One possible reason for this is that myocardial oxygen demand exceeds the available oxygen supply, particularly towards the latter part of systole. Such an explanation is in keeping with the results of cell fractionation and enzymatic analysis of left ventricular endomyocardial biopsies, showing a reduction in mitochondrial oxidative enzyme activity in patients with poor myocardial function, together with evidence of enhanced anaerobic glycolysis in patients with congestive cardiomyopathy who have dilated ventricles (Peters et al., 1975).

References


Assessment of left ventricular function


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