Echocardiographic Evaluation of the Hemodynamic Effects of Chronic Aortic Insufficiency with Observations on Left Ventricular Performance

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SUMMARY

Twelve patients with aortic insufficiency (AI) were investigated by echocardiographic methods using reflected ultrasound. No patients with associated mitral valvular disease were included. Estimates of each patient's aortic valvular total flow \( (Q_{At}) \) as computed from left ventricular cavity minor-axis dimension changes at end-diastole (\( D_1 \)) and end-systole (\( D_2 \)) were compared with their respective mitral valvular flows \( (Q_M) \) as estimated from the opening velocity of the anterior mitral leaflet. The arithmetic difference of the two valvular flows permitted calculation of the aortic regurgitant flow \( (Q_{Ar}) \) as \( Q_{Ar} = Q_A - Q_M \). In this series of patients \( Q_{Ar} \) varied from 5.2 to 12.9 liters/min, \( Q_M \) from 3.3 to 7.3 liters/min, \( Q_{Ar} \) from 1.1 to 6.5 liters/min, ejection fraction (EF) from 0.37 to 0.60 and regurgitant fraction \( (Q_{Ar}/Q_A) \) from 0.15 to 0.58. A statistically significant difference of \( Q_{Ar} \) and \( Q_M \) was not evident in a series of 20 patients without AI.

Left ventricular performance in the patients with AI was estimated by echocardiographic methods by measuring the amplitude of systolic excursion of the left ventricular midwall (PWE), the mean velocity of posterior wall motion (mean PWV), and computation of the mean velocity of circumferential fiber shortening \( (V_{CP}) \) for the left ventricular myocardium.

The data presented appear to provide a simple noninvasive method for serial observations of the hemodynamic consequences of AI and the eventual alteration of myocardial performance in such patients.

Additional Indexing Words:

- Aortic valvular total flow
- Mitral valvular flow
- Regurgitant fraction
- Ejection fraction
- Left ventricular posterior wall excursion
- Left ventricular end-diastolic volume
- Circumferential shortening velocity
- Aortic valvular regurgitant flow
- Left ventricular minor-axis dimensions
- Left ventricular posterior wall velocity
- Myocardial function

The clinical course of aortic insufficiency, discovered in children, adolescents, or young adults, usually begins as a variable period of wellbeing followed by the progressive deterioration of cardiac reserve and the appearance of frank heart failure. As in the case of the present series, such patients frequently are free of symptoms, or virtually so, and are seen because of a heart murmur. The medical evaluation of the patient usually does not prompt cardiac catheterization or consideration of surgical repair, but instead results in a decision to follow the patient's cardiac status periodically in a clinical setting.

Diastolic left ventricular volume overload due to aortic insufficiency has proven most difficult to quantitate by serial observations of patients without resorting to repeated cardiac catheterization. Such patients presumably do not progress abruptly from a position of cardiac compensation on the ascending limb of the Frank-Starling curve to one of decompensation on the descending limb of the curve. It is true from clinical observation, however, that such patients deteriorate rapidly when cardiac decompensation develops, prompting urgent surgical consideration for valve replacement. Surgical replacement of the diseased aortic valve after the onset of cardiac decompensation due to aortic
insufficiency is frequently ineffective in restoring adequate myocardial function, apparently because of an irreversible myocardial contractility impairment of the chronically dilated left ventricle.

The need for improved serial, noninvasive, objective, and quantitative assessment of the patient with aortic insufficiency is generally recognized. Echocardiographic observations from 12 patients with aortic insufficiency were prepared and are reported here. Estimates of aortic total flow (Qₐ) and regurgitant flow (Qₐ₆), computation of left ventricular end-diastolic volume (EDV), and measurement of parameters of left ventricular performance were undertaken by application of echocardiographic methods to patients with chronic aortic insufficiency of various etiologies.

Methods

Echocardiographic studies were performed on 12 adult patients referred for cardiac evaluation because of the existence of a heart murmur. Eight of the 12 patients possessed clinical auscultatory and phonocardiographic characteristics of pure aortic insufficiency; the remaining four patients exhibited evidence of aortic stenosis with aortic insufficiency. In every instance, the magnitude of the aortic regurgitation was considered hemodynamically significant. One of the patients (F.A.) who had sustained a previous myocardial infarction, continued to experience symptoms of coronary insufficiency and was under treatment with quinidine and digitalis. None of the other patients with aortic insufficiency received pharmacologic agents believed to influence myocardial contractility. No patient with associated mitral valve disease was included in the series. Twenty additional patients without aortic insufficiency, mitral valve disease, intracardiac shunts or rhythm disturbances were similarly examined in order to provide simultaneous comparisons for aortic and mitral valvular flow under clinical circumstances in which a large aortic regurgitant flow did not exist. A third group of 12 normal patients without heart disease was examined to provide data pertaining to parameters of myocardial performance with which similar information in patients with aortic insufficiency might be compared. All patients were studied in normal sinus rhythm and exhibited atroventricular conduction of 0.20 sec or less.

Echocardiographic examinations were performed using a commercially available ultrasonicoscope* with 2.25 MHz transducer of 1.9 cm (0.75 in.) diameter with a repetition rate of 1,000/sec. The transducer was focused at 10 cm. The output of the ultrasonicoscope was displayed oscillographically and recorded photographically, using a Polaroid camera, and on a multichannel oscillographic recorder.† At the same time tracings were included from the ECG and the indirect carotid arterial pulse and phonocardiogram from the third left intercostal space at the sternal border. All subjects were studied in the supine position. A water-soluble gel was used to produce airless contact between skin surface and transducer.

The technique for ultrasonic display of the mitral valvular apparatus** and determination of the left ventricular cavity minor-axis dimensions†† by echocardiography have been described in detail by other investigators. Briefly, the ultrasonic transducer was placed in the fourth intercostal space at or 1 to 2 cm lateral to the left sternal border. The position of the transducer was directed posteriorly, medially, and slightly cephalad so that the characteristic motion of the anterior leaflet of the mitral valve was displayed. B-scan mode (time-motion representation) scanning procedure was followed. By utilizing the strip recorder and technique described by McDonald, Feigenbaum and Chang, the transducer was positioned to identify the anterior leaflet of the mitral valve, the interventricular septum and the posterior left ventricular wall. Modifications of beam direction and transducer position are at times necessary to obtain quality reproductions of mitral motion and good definition of the endocardial surfaces of the interventricular septum and left ventricular posterior midwall. Following the echocardiographic definition of anterior mitral valve leaflet motion, these data were applied to mitral valvular flow estimation as described by Kingsley et al.†‡ Estimates of mitral valvular flow have been computed by echocardiographic methods in the past for the Starr-Edward's mitral valve prosthesis and for the normally functioning, non diseased adult mitral valve apparatus. We used the hydraulic equation \( Q/AVT \), where \( Q = \) flow in ml, \( A = \) mitral valve cross-sectional area in cm², \( V = \) blood velocity (anterior mitral leaflet opening velocity) in cm/sec, \( T_a = \) duration of flow across the mitral valve (diastolic filling time) in seconds. By assigning a fixed cross-sectional area to the normal adult mitral valve of 5 cm², and by assuming 1) laminar flow, 2) that the opening velocity of the anterior mitral leaflet represents the linear velocity of blood flowing into the left ventricle through the mitral valve, 3) that the leaflet lacks inertia, and 4) that flow remains constant through ventricular filling, Kingsley has reported that stroke volume and cardiac output in 500 patients determined by opening anterior mitral leaflet velocity echocardiographic methods varied no more than 15% from stroke volume and cardiac output determined by dye-dilution techniques. This measurement of mitral valvular flow has been reported by Kingsley to respond to the increased cardiac output found in patients with peripheral arterio-venous shunts. Comparison of stroke volume and cardiac output in seven such patients revealed close correlation of values obtained by simultaneous dye-dilution and echocardiographic methods.


†Model DB8, Electronics for Medicine, White Plains, New York.
The left ventricular cavity minor-axis dimensions at end-diastole (Dd) and end-systole (Ds) were computed as described by Popp and associates.4 Left ventricular volumes based upon the minor-axis dimensions of the left ventricular cavity as determined by echocardiography have been estimated by many investigators.10-15 Such reports have been correlated with left ventricular volume at end-diastole and end-systole, stroke volume, ejection fraction, and/or cardiac output computed by biplane angiography, dye-dilution or by Fick principle techniques and show close comparison by linear regression analysis for each method.

Volumetric derivations in the present study have been based upon the data of Fortuin et al.,18 who have drawn close linear correlation between echocardiographic and biplane angiographic ventricular volumes. The regression equations prepared by Fortuin derived from echo-angiographic reports have been correlated with left ventricular cavity volumes in relatively normal-sized chambers. It is in this latter instance that the volume formula of a prolate ellipse has been applied.18-17 In patients with abnormally large volume left ventricular cavities, as is the case in patients with diastolic overload and aortic insufficiency, the ventricular geometry becomes more spherical, resulting in a relative increase in the minor-axis dimension as compared with the major-axis. In this situation, correlation with the linear regression equations supported by Fortuin is probably more accurate than the application of one of the several available equations based upon the cube function and the geometric configuration of an ellipse.

Measurements of amplitude of posterior wall motion (PWE) and the mean rate of anterior motion of the left ventricular posterior midwall (mean PWV) were accomplished in accordance with the description of Krausz and Kennedy.19 Echocardiographic derivation of the estimate of mean velocity of circumferential fiber shortening (mean Vcf) was based upon the method of Fortuin, Hood and Craig.19 The mean velocity of circumferential fiber shortening (Vcf) has been computed by Fortuin19 based upon the echocardiographic measurements of the left ventricular minor-axis internal dimension at end-diastole and end-systole and the duration of shortening of the axis in systole. A comparison of Vcf data obtained by echocardiographic measurements with those derived from thermodilution catheterization techniques20 reveals similar results, whereas the echo technique produces somewhat lower values for Vcf than those derived from biplane angiography.21

**Results**

The 12 patients appearing in table 1 represent the test series of patients in whom hemodynamically significant aortic insufficiency was recognized clinically. The Austin Flint murmur was audible on clinical auscultation and recorded phonocardiographically in four of the 12 patients. The existence of the Austin Flint murmur was associated with aortic regurgitant flows of 1.3 to 6.5 liters/min, and seemed to indicate that a high regurgitant flow was not essential to the production of the Austin Flint murmur.

Estimates of left ventricular hypertrophy (LVH) by electrocardiography and chest X-ray generally agreed well with the left ventricular minor-axis internal dimensions (Dd and Ds) and left ventricular end-diastolic volumes computed by echocardiography. Seven of the nine patients (table 2) possessing a Dd of 60 mm or more, a Ds of not less

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**Table 1**

**Clinical Characteristics of Twelve Patients with Aortic Insufficiency**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Sex</th>
<th>BSA</th>
<th>Valvular lesion</th>
<th>Etiology</th>
<th>Austin-Flint murmur</th>
<th>Functional class (NYHA)</th>
<th>Clinical estimate of regurgitant flow</th>
<th>ECG</th>
<th>Chest X-ray</th>
</tr>
</thead>
<tbody>
<tr>
<td>R.C.</td>
<td>51</td>
<td>M</td>
<td>2.12</td>
<td>AI</td>
<td>Rheumatic</td>
<td>Present</td>
<td>I</td>
<td>Moderate</td>
<td>LVH</td>
<td>Mild LVE</td>
</tr>
<tr>
<td>J.K.</td>
<td>29</td>
<td>M</td>
<td>1.79</td>
<td>AS, AI</td>
<td>Congenital</td>
<td>Absent</td>
<td>II</td>
<td>Moderate</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>J.P.</td>
<td>22</td>
<td>M</td>
<td>1.76</td>
<td>AS, AI</td>
<td>Rheumatic</td>
<td>Absent</td>
<td>II</td>
<td>Moderate</td>
<td>LVH</td>
<td>Normal</td>
</tr>
<tr>
<td>W.H.</td>
<td>52</td>
<td>M</td>
<td>1.83</td>
<td>AI</td>
<td>Rheumatic</td>
<td>Absent</td>
<td>I</td>
<td>Moderate</td>
<td>LVH</td>
<td>Mild LVE</td>
</tr>
<tr>
<td>E.K.</td>
<td>45</td>
<td>M</td>
<td>1.98</td>
<td>AS, AI</td>
<td>Rheumatic</td>
<td>Absent</td>
<td>II</td>
<td>Moderate</td>
<td>LVH</td>
<td>Normal</td>
</tr>
<tr>
<td>T.C.</td>
<td>18</td>
<td>M</td>
<td>1.87</td>
<td>AI</td>
<td>Rheumatic</td>
<td>Absent</td>
<td>II</td>
<td>Moderate</td>
<td>LVH</td>
<td>LVE</td>
</tr>
<tr>
<td>J.M.</td>
<td>31</td>
<td>M</td>
<td>1.77</td>
<td>AI</td>
<td>Rheumatic</td>
<td>Absent</td>
<td>I</td>
<td>Moderate</td>
<td>Normal</td>
<td>LVE</td>
</tr>
<tr>
<td>F.A.</td>
<td>63</td>
<td>M</td>
<td>2.15</td>
<td>AS, AI</td>
<td>Unknown</td>
<td>Absent</td>
<td>III</td>
<td>Moderate</td>
<td>LBBB</td>
<td>LVE</td>
</tr>
<tr>
<td>L.B.</td>
<td>47</td>
<td>M</td>
<td>1.83</td>
<td>AI</td>
<td>Unknown</td>
<td>Absent</td>
<td>I</td>
<td>Mild</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>S.M.</td>
<td>37</td>
<td>F</td>
<td>1.58</td>
<td>AI</td>
<td>Unknown</td>
<td>Present</td>
<td>I</td>
<td>Mild</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>R.B.</td>
<td>45</td>
<td>F</td>
<td>1.62</td>
<td>AI</td>
<td>SLE</td>
<td>Absent</td>
<td>I</td>
<td>Moderate</td>
<td>LVH</td>
<td>Normal</td>
</tr>
<tr>
<td>P.M.</td>
<td>20</td>
<td>M</td>
<td>1.81</td>
<td>AI</td>
<td>Congenital</td>
<td>Present</td>
<td>I</td>
<td>Moderate</td>
<td>Normal</td>
<td>Normal</td>
</tr>
</tbody>
</table>

Abbreviations: BSA = Body surface area, m²; AI = Aortic insufficiency, AS = Aortic stenosis, SLE = Systemic lupus erythematosus, LBBB = Complete left bundle-branch block, LVH = Left ventricular hypertrophy, LVE = Left ventricular enlargement.
than 43 mm, and EDV exceeding 185 ml exhibited chest X-ray and/or electrocardiographic criteria of LVE or LVH. Two patients (L.B. and P.M.) were considered to have moderate enlargement of the left ventricular cavity by echocardiography, with a $D_D$ of 65 and 66 mm, $D_A$ of 53 and 46 mm, and EDV of 230 and 236 ml, but had normal electrocardiograms and heart size on standard chest X-ray. One patient (E.K.) with normal minor-axis dimensions, EDV, and normal heart size by chest X-ray met the electrocardiographic criteria for LVH. This latter case had both aortic stenosis and aortic insufficiency, with the aortic stenosis clinically dominant; the patient possessed a relatively small aortic regurgitant flow of 1.6 liters/min.

Aortic valvular total flow in patients with aortic insufficiency, computed from the difference in $D_A$ and $D_D$ by echocardiography, varied from 5.2 to 12.9 liters/min. The mitral valvular flow ranged from 3.3 to 7.3 liters/min, based upon anterior mitral valve leaflet opening velocities of 214 to 432 mm/sec, and was consistently smaller than the aortic flow in each patient of this series ($P < 0.005$). Subtracting the mitral valvular flow from the total aortic flow permitted estimation of regurgitant flow and calculation of the aortic regurgitant fraction. Aortic regurgitant flows ranged from 1.1 to 6.5 liters/min and produced regurgitant fractions from 0.15 to 0.58.

To compare mitral and aortic valvular flows as estimated by echocardiographic methods, a series of 20 patients without aortic insufficiency, mitral valve disease, intracardiac shunt, cardiac rhythm disturbance or heart failure was studied. The data appear in table 3. The mean aortic valvular flow with standard deviation of the mean for these 20 patients was $5.1 \pm 1.3$ liters/min, compared with mean mitral flow and standard deviation of the mean of $4.8 \pm 1.6$ liter/min ($P > 0.05$). The data thereby reject statistically significant differences of the two measures of valvular flow in the 20 patients without aortic insufficiency. Five patients (25%) in this group (C.S., M.V., D.S., J.A., A.L.) exhibited left ventricular cavity minor-axis dimensions of 60 mm or greater at end-diastole, 43 mm or greater at end-systole, and EDV between 201 and 242 ml. This indicates an enlargement of the left ventricular cavity comparable to that of nine of the 12 patients appearing in table 2 with aortic insufficiency. Three of the five patients with echocardiographic evidence of left ventricular cavity enlargement showed slightly higher mitral flow than aortic flow as estimated by ultrasound techniques. Mean values for aortic and mitral flows for the five patients with left ventricular cavity enlargement, however, were identical and measured 4.9 liters per minute. Therefore, it would not appear that a large left ventricular cavity per se introduces an error of significant magnitude to account for aortic and mitral flow differentials found in patients with AI.

In table 4, data pertaining to myocardial performance of the 12 patients with aortic insufficiency are displayed in terms of systolic excursion (PWE) and mean velocity of motion (PWV) of the left ventricular posterior myocardial midwall. In addition, the mean velocity of circumferential fiber shortening ($V_{CF}$) has been derived in accordance with Fortuin's equation for each case and appears.
CHRONIC AORTIC INSUFFICIENCY

Table 3

Aortic and Mitral Valvular Flow Computed by Echocardiographic Methods in Patients Without Aortic Insufficiency, Mitral Valve Disease or Intracardiac Shunt

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Sex</th>
<th>BSA</th>
<th>HR</th>
<th>D1 (mm)</th>
<th>D2 (mm)</th>
<th>EDV (ml)</th>
<th>Qa (liters/min)</th>
<th>Qv (liters/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>J.A.</td>
<td>53</td>
<td>M</td>
<td>2.05</td>
<td>59</td>
<td>52</td>
<td>30</td>
<td>154</td>
<td>5.6</td>
<td>5.8</td>
</tr>
<tr>
<td>R.L.</td>
<td>55</td>
<td>M</td>
<td>1.93</td>
<td>53</td>
<td>51</td>
<td>42</td>
<td>148</td>
<td>3.8</td>
<td>3.0</td>
</tr>
<tr>
<td>S.N.</td>
<td>45</td>
<td>M</td>
<td>1.90</td>
<td>81</td>
<td>51</td>
<td>43</td>
<td>148</td>
<td>5.3</td>
<td>4.9</td>
</tr>
<tr>
<td>C.S.</td>
<td>68</td>
<td>M</td>
<td>1.79</td>
<td>71</td>
<td>67</td>
<td>63</td>
<td>242</td>
<td>4.8</td>
<td>4.9</td>
</tr>
<tr>
<td>M.J.</td>
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<td>F</td>
<td>1.63</td>
<td>56</td>
<td>55</td>
<td>35</td>
<td>171</td>
<td>7.1</td>
<td>6.9</td>
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<tr>
<td>G.J.</td>
<td>48</td>
<td>M</td>
<td>1.91</td>
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<td>45</td>
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<td>112</td>
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<td>3.5</td>
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<td>M</td>
<td>2.07</td>
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<td>53</td>
<td>36</td>
<td>160</td>
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<td>6.1</td>
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<td>M</td>
<td>1.82</td>
<td>61</td>
<td>55</td>
<td>44</td>
<td>172</td>
<td>5.2</td>
<td>4.8</td>
</tr>
<tr>
<td>M.V.</td>
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<td>M</td>
<td>2.02</td>
<td>80</td>
<td>60</td>
<td>53</td>
<td>201</td>
<td>3.7</td>
<td>5.5</td>
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<tr>
<td>O.D.</td>
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<td>M</td>
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<td>36</td>
<td>165</td>
<td>9.3</td>
<td>9.5</td>
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<tr>
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<td>M</td>
<td>2.01</td>
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<td>50</td>
<td>38</td>
<td>142</td>
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<td>3.8</td>
</tr>
<tr>
<td>D.S.</td>
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<td>M</td>
<td>2.05</td>
<td>60</td>
<td>60</td>
<td>49</td>
<td>201</td>
<td>5.5</td>
<td>4.3</td>
</tr>
<tr>
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<td>F</td>
<td>1.51</td>
<td>75</td>
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<td>154</td>
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<td>3.7</td>
</tr>
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<td>M</td>
<td>1.89</td>
<td>52</td>
<td>52</td>
<td>44</td>
<td>154</td>
<td>3.5</td>
<td>3.7</td>
</tr>
<tr>
<td>V.L.</td>
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<td>M</td>
<td>1.38</td>
<td>79</td>
<td>52</td>
<td>44</td>
<td>145</td>
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<td>4.8</td>
</tr>
<tr>
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<td>M</td>
<td>1.81</td>
<td>62</td>
<td>54</td>
<td>45</td>
<td>166</td>
<td>4.6</td>
<td>4.7</td>
</tr>
<tr>
<td>M.S.</td>
<td>39</td>
<td>M</td>
<td>2.05</td>
<td>59</td>
<td>51</td>
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<td>148</td>
<td>4.2</td>
<td>2.4</td>
</tr>
<tr>
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<td>F</td>
<td>1.68</td>
<td>65</td>
<td>43</td>
<td>33</td>
<td>101</td>
<td>4.3</td>
<td>4.0</td>
</tr>
<tr>
<td>J.A.</td>
<td>55</td>
<td>M</td>
<td>1.77</td>
<td>67</td>
<td>60</td>
<td>51</td>
<td>201</td>
<td>5.4</td>
<td>3.9</td>
</tr>
<tr>
<td>A.L.</td>
<td>79</td>
<td>M</td>
<td>1.71</td>
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<td>61</td>
<td>53</td>
<td>207</td>
<td>5.2</td>
<td>6.0</td>
</tr>
</tbody>
</table>

Abbreviations: As in tables 1 and 2.

in the table. As might be anticipated, six patients exhibited PWE exceeding 1.00 cm and four cases exceeded 1.20 cm as compared with 0.85 ± 0.18 cm observed in 12 normal patients without known heart disease. The mean PWV was considered to be normal in ten of the aortic insufficiency cases and probably minimally reduced in two patients (R.C. and E.K.) when compared with the 12 normal patients in whom a value of 4.1 ± 1.1 cm per second was observed. Computation of Vcf yielded small reductions in three cases (W.H., T.C., and L.B.) and more substantial impairment of this parameter of myocardial performance in two additional cases (R.C. and R.B.) when compared with Fortuin’s normal range of 0.91 ± 0.15 cm/sec or that of the 12 normal cases from our laboratory in whom 1.01 ± 0.19 cm/sec was found.

These data appear to confirm the expected increased systolic excursion of the left ventricular posterior midwall in patients with aortic insufficiency. It is hypothesized that this phenomenon may contribute to the basic mechanism whereby stroke volume is increased and is therefore of importance as a means of compensation in patients with large aortic regurgitant flows. The echocardiographic measurement of the velocity of motion of the wall and estimation of the velocity of contraction of the left ventricular chamber circumferentially about its long axis has been suggested as a method of providing detailed information pertaining to the effective contractile performance of the left ventricular myocardium. In the physiologic circumstance of chronic diastolic volume overload associated with aortic insufficiency, these measurements may possess similar significance and aid in recognition of the migration of a patient’s myocardial function

Table 4

Echocardiographic Parameters of Left Ventricular Performance in Patients with Aortic Insufficiency

<table>
<thead>
<tr>
<th>Patient</th>
<th>PWE (cm)</th>
<th>Mean PWV (cm/sec)</th>
<th>Vcf (cm/sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>R.C.</td>
<td>0.85</td>
<td>2.8</td>
<td>0.44</td>
</tr>
<tr>
<td>J.K.</td>
<td>1.13</td>
<td>4.5</td>
<td>1.12</td>
</tr>
<tr>
<td>J.P.</td>
<td>1.13</td>
<td>3.8</td>
<td>0.89</td>
</tr>
<tr>
<td>W.H.</td>
<td>0.92</td>
<td>3.7</td>
<td>0.76</td>
</tr>
<tr>
<td>E.K.</td>
<td>0.76</td>
<td>2.7</td>
<td>0.90</td>
</tr>
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<td>T.C.</td>
<td>1.46</td>
<td>3.9</td>
<td>0.74</td>
</tr>
<tr>
<td>J.M.</td>
<td>1.21</td>
<td>4.8</td>
<td>1.15</td>
</tr>
<tr>
<td>F.A.</td>
<td>1.62</td>
<td>5.3</td>
<td>0.85</td>
</tr>
<tr>
<td>L.B.</td>
<td>1.45</td>
<td>4.7</td>
<td>0.71</td>
</tr>
<tr>
<td>S.M.</td>
<td>0.81</td>
<td>3.6</td>
<td>1.36</td>
</tr>
<tr>
<td>R.B.</td>
<td>0.75</td>
<td>3.0</td>
<td>0.66</td>
</tr>
<tr>
<td>P.M.</td>
<td>0.88</td>
<td>3.0</td>
<td>0.92</td>
</tr>
</tbody>
</table>

Abbreviations: PWE = Left ventricular posterior wall systolic excursion, PWV = Left ventricular posterior wall systolic velocity, Vcf = Mean velocity of circumferential fiber shortening.

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from the ascending to the descending limb of the Frank-Starling curve.

The information presented in tables 2 and 4 shows five of the patients with aortic insufficiency (R.C., W.H., T.C., L.B., and R.B.) were found to possess values for \( V_{CP} \) in the range of 0.44 – 0.76 circ/sec and were the only patients in the series with ejection fractions below 0.50. The EDV in two of the five patients exceeded 300 ml and was 278 ml in a third patient. Three of the five patients exhibited PWE in the range of 0.75 to 0.92 cm; although in the normal range, this is in contradistinction to the trend observed in half the patients in this series with aortic insufficiency in whom PWE is increased, presumably as a compensatory mechanism. The mean PWV data exhibited no discernible trends, although one of the two instances in which this parameter of myocardial performance was low occurred in R.C., whose aforementioned myocardial performance data were also abnormal. It is probable that serial observations of aortic total and regurgitant flow data will be required in order to assign prognostic significance to this information in individual cases.

The five patients with aortic insufficiency to whom reference has been made, and represented by patient R.B. in figure 1, showed evidence by echocardiographic methods of impaired left ventricular performance. These individuals could not be recognized by routine clinical examination as differing significantly in the severity of their disease from their seven counterparts. The echocardiograms of E. K. appear in figure 2 as representative of the patient with AI, but without evidence of impair-

![Echocardiogram](image)

**Figure 1**

Case R.B. (Left) Echocardiogram from left ventricular posterior midwall and interventricular septum showing normal amplitude of PWE, normal mean PWV, significantly reduced mean \( V_{CP} \), significantly increased EDV, and low EF. (Right) Echocardiogram of anterior mitral valve leaflet motion. \( Q_M \) of 5.7 liters/min based upon the leaflet opening velocity compares with \( Q_{At} \) of 7.6 liters/min computed from the cavity study on the left, resulting in \( Q_{At}/Q_{At} \) of 0.25. Calculation: Calibration dot intervals equal 1 cm vertical axis and 0.5 sec horizontal axis.

\[
\hat{V}_M = AVT_o = 5 \cdot 36.7 \cdot 0.402 = 74 \text{ ml (mitral stroke volume)} = 74 \cdot 77 = 5.7 \text{ liters/min (mitral minute flow)}.
\]

\[
\hat{Q}_{At} = (EDV) - (ESV) = [(59 \cdot 67-153) - (47 \cdot 56-120)] = 99 \text{ ml (aortic stroke volume)} = 99 \cdot 77 = 7.6 \text{ liters/min (aortic minute flow)}.
\]
CHRONIC AORTIC INSUFFICIENCY

Figure 2

Case E.K. (Left) Echocardiogram from left ventricular posterior midwall and interventricular septum showing normal amplitude of PWE, slightly reduced mean PWV, normal mean VCP, normal EDV, and normal EF. (Right) Echocardiogram of anterior mitral valve leaflet motion. QM of 4.2 liters/min based upon the leaflet opening velocity compares with QA of 5.8 liters/min computed from the cavity study on the left, resulting in QA/QM of 1.6 liters/min and QA/QM of 0.28.

Calculation:

\[ Q_M = AVT_s = 5 \times 25.8 \times 0.500 = 64.5 \text{ ml (mitral stroke volume)} = 64.5 \times 65 = 4.2 \text{ liters/min mitral minute flow).} \]

\[ QA = [(EDV)-(ESV)] = [(59 \times 51-153) - (47 \times 38-120)] = 89 \text{ ml (aortic stroke volume)} = 89 \times 65 = 5.8 \text{ liters/min (aortic minute flow).} \]

Figure 3 illustrates the fluttering of the leaflet in diastole in two patients of this series.

Discussion

The volume of aortic regurgitant blood flow has been estimated by a variety of indirect methods under circumstances of clinical aortic insufficiency in man. Indicator-dilution techniques, contrast radiography, and analysis of aortic or peripheral arterial pulses have been used in the clinical assessment of the magnitude of aortic regurgitant flow. More direct approaches to the problem have been applied under cardiac surgical circumstances in man and, in the experimental laboratory, in animals by use of flowmeters of various types. Popp and Harrison have estimated regurgitant flow through mitral and aortic valves by comparing

mment of myocardial performance. Four of the five patients were graded N.Y.H.A. functional class I. It was noted that all cases with impaired myocardial performance possessed pure aortic insufficiency rather than a combined lesion; the patients with one exception tended to fall into the older age category; four of the five patients showed LVH in their ECGs; and three exhibited abnormal chest X-rays. The character of the aortic insufficiency murmur by clinical auscultatory and phonocardiographic methods did not differ recognizably in these patients from the remaining patients in the series, although of the four patients in whom an Austin Flint murmur was heard, two exhibited impaired myocardial performance as defined by this study.

Peculiarities of anterior mitral leaflet behavior in patients with AI have been reported previously. Figure 3 illustrates the fluttering of the leaflet in diastole in two patients of this series.
stroke volume computed by echocardiography with effective forward stroke volume determined simultaneously by the standard Fick method. The data appearing in the present report, derived from echocardiographic methods, confirm the magnitude of regurgitant flows previously described in aortic insufficiency of mild to moderate clinical severity by a number of investigators\textsuperscript{24-33} using other methods.

In addition to providing a description of left ventricular stroke volume and aortic minute flow in patients with aortic insufficiency, the present investigation has permitted an assessment of the regurgitant volume in this clinical situation by application of noninvasive methods. Despite the necessary acceptance of several assumptions inherent in Kingsley's method of estimating mitral valve flow and its limited application to those adult patients with normal mitral valves, the usefulness of the mitral valve flow data is apparent in the evaluation of patients with aortic insufficiency, in which instance it permits by simple subtraction from total aortic flow the estimation of regurgitant flow through the aortic valve.

The influence of aortic regurgitation upon atrial transport has been recognized more frequently as experience with cardiac catheterization has increased. Occasional examples of reversed pressure gradient across the mitral valve, premature closure of the valve in late diastole, and rarely, diastolic mitral insufficiency have been reported in severe clinical aortic insufficiency\textsuperscript{37-41} and in acute experimental aortic insufficiency in calves\textsuperscript{42, 43}. Impedance, in late diastole, of atrial transport and left ventricular filling via the mitral valve might therefore be expected, in the more severe cases of aortic insufficiency, to yield spuriously elevated estimates of forward mitral valve flow as determined from the opening velocity of the anterior leaflet. Echocardiographic recognition of early mitral valve closure is feasible,\textsuperscript{44} but has not been reported to our knowledge, and has not been recognized in the present series of patients with aortic insufficiency.

The physiologic effect of increased preload in aortic valvular insufficiency becomes manifest in time by progressively increased left ventricular end-diastolic volumes associated with increasing end-diastolic tension and increasing myocardial contractile velocity to achieve the greater total stroke
volumes needed to maintain effective forward aortic flow. The Frank-Starling mechanism, through which an increase in the ventricle’s end-diastolic volume and tension produces increased force of myocardial contraction, provides the failing heart with a major compensatory mechanism. In aortic insufficiency, the dilated left ventricle ejects a total stroke volume significantly exceeding the normal. To maintain an effective forward stroke volume at near normal levels, the heart dilates and the Frank-Starling mechanism results for a time in increasing the force of contraction and total stroke volume. An increase in the end-diastolic volume of the ventricle permits the ejection of a larger stroke volume, even when the extent of shortening of the muscle fibers remains constant. In the patient with aortic insufficiency, the onset of reduced total stroke volume with constant or increasing left ventricular end-diastolic volume clearly indicates that it is at this point that a decrease in the degree of muscle fiber shortening must have occurred. Such observations are now feasible in patients with aortic insufficiency by serial echocardiographic measurements. Increasing left ventricular end-diastolic volume associated with decreasing stroke volume and echocardiographic evidence of progressive impairment of myocardial performance as indicated by diminished PWE, mean PWV, or Vef may delineate the onset of an important myocardial mechanical defect in the patient with aortic insufficiency. It is proposed that availability of such data prior to appearance of the more blatant symptoms of clinical heart failure would then prompt further evaluation by cardiac catheterization and surgical consideration.

We have described a simple, accurate, non-invasive and reproducible method of estimating the regurgitant volume in patients with aortic insufficiency without associated mitral valve disease. Echocardiographic methods have been applied to a group of such patients with AI in an effort to assess the effect of chronic left ventricular diastolic volume overload. Five of the 12 patients studied were found to exhibit evidence of impaired myocardial performance. The implications of progressive deterioration of left ventricular performance as demonstrated in serial studies should alert the clinician to the magnitude of the myocardial disability found in patients with aortic insufficiency in advance of clinical signs and symptoms of heart failure. As experience is gained with these procedures and data, more precise information is anticipated relevant to their correlation with surgical indications in aortic insufficiency.

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