Estimation of Aortic Regurgitation by Diastolic Pulse Wave Analysis

By Terrance P. Judge, M.D., and J. Ward Kennedy, M.D.

SUMMARY
To test the hypothesis that the fall in supravalvular aortic diastolic pressure is relative to the severity of the regurgitant flow in aortic regurgitation, 27 patients with isolated aortic regurgitation, 23 patients with combined aortic regurgitation and stenosis, and 15 normal subjects were studied. Diastolic pulse pressure (pressure at the dicrotic notch minus pressure at end diastole) and the diastolic slope (line constructed through the dicrotic notch and end diastole) were measured.

A highly significant correlation was found between the regurgitant flow in isolated aortic regurgitation and the diastolic pulse pressure when corrected for heart rate (r = 0.82, P < 0.001). The diastolic slope related similarly to the regurgitant flow (r = 0.71, P < 0.001). There was no significant relationship, however, in the presence of combined aortic stenosis and regurgitation. Aortic pressure at end diastole and pulse pressure correlated poorly with either isolated aortic regurgitation or combined aortic valve disease.

An electrical analog circuit was used to help explain these findings.

Additional Indexing Words:
Aortic stenosis Angiocardiography Circulatory electrical analog
Rheumatic heart disease

Several technics have been developed for the quantitative evaluation of aortic regurgitation. All such technics described to date have required specialized equipment, are relatively complex, or are only semiquantitative. Most of these methods have not been widely used because of these difficulties.

The angiocardiographic method for the determination of regurgitant flow has been useful in the clinical evaluation of patients, but a simpler, more widely available method is needed. This study was undertaken to test the hypothesis that the detailed analysis of the diastolic portion of the central aortic pressure would be useful in estimating severity of aortic regurgitation. The results of this analysis are compared to the regurgitant flow as independently measured from biplane angiocardiograms.

Methods
Fifty cases of aortic regurgitation, 23 of which had combined aortic stenosis, and 15 controls were selected from our catheterization files. All had complete right and left heart catheterization with clear, standardized supravalvular aortic and left ventricular pressure tracings. Pressures were measured using a fluid-filled 8 F, 100 cm Gensini catheter attached to a Statham P23Db transducer and recorded on an Electronics for Medicine or Honeywell optical recorder. This system has been tested and shown to possess less than 5% distortion to 8 to 10 Hz. Cardiac output was measured using the direct Fick method.

In the aortic regurgitation groups, all had high quality left ventricular biplane angiocardiograms. Mitral regurgitation was excluded in all cases by this technic. The total left ventricular minute output was determined by the method of Sandler and associates. The aortic regurgitant flow was quantitated by subtracting the Fick cardiac output from the total left ventricular minute output.

In the group with combined aortic stenosis and regurgitation, the aortic valve area was calculated...
using a modified Gorlin formula:\(^1\):

\[
AVA = \frac{TCO}{SEP \times HR \times 44.5 \times AVG}
\]

Where: AVA = aortic valve area; TCO = total cardiac output (as determined from biplane angiocardiograms); SEP = systolic ejection period; AVG = aortic valve gradient (during systole).

All hemodynamic measurements were within normal limits in the control group. None of these patients had a murmur of aortic regurgitation.

Pressures at the dicrotic notch and at end diastole were measured on the aortic pulse wave tracing, and the difference between these pressures was termed the diastolic pulse pressure (DPP). The diastolic period was measured and expressed as a percentage of the R-R interval (per cent diastole). In order to minimize the effect of heart rate, the DPP was corrected to a diastolic period of 60% which was found to be an approximate average for the group. The formula for this correction is as follows:

Corrected DPP (CDPP)

\[
= DPP \left(\frac{0.60}{\text{measured } \% \text{ diastole}}\right).
\]

Diastolic slope (DS) was measured by constructing a line through the nadir of the dicrotic notch and the point of end diastole. The slope of this line was expressed as mm Hg/sec. Figure 1 illustrates how these measurements were made in examples of mild, moderate, and severe aortic regurgitation.

**Results**

The hemodynamic measurements in the control group, the group with isolated aortic regurgitation, and the group with combined aortic stenosis and regurgitation are tabulated in table 1. In the group with combined aortic valve disease, the mean systolic gradient across the aortic valve ranged from 20 to 90 mm Hg (av = 56 mm Hg), and the aortic valve area ranged from 0.45 to 2.3 cm\(^2\) (av = 1.21 cm\(^2\)).

Figure 2 demonstrates the relationship between the magnitude of the regurgitant flow in the group with isolated aortic regurgitation and the CDPP (r = 0.82, P > 0.001) and the DS (r = 0.71, P < 0.001). Although there is some overlap with the control group, this only occurs in patients with small regurgitant flows. Without correction for diastolic period, the DPP correlated similarly (r = 0.75).

When aortic stenosis was present the DPP, CDPP, and DS were abnormally elevated compared to the control group but were not significantly related to the magnitude of the regurgitant flow (table 2).

**Figure 1**

Supravalvular aortic pressure waves in mild (A, regurgitant flow = 1.4 L/min), moderate (B, regurgitant flow = 5.9 L/min), and severe aortic regurgitation (C, regurgitant flow = 10.5 L/min). Lines representing the diastolic slope have been drawn through the dicrotic notch and end diastole in each case and their respective numerical values in mm Hg/sec noted. Technic for measuring the diastolic pulse pressure is also illustrated and the values in mm Hg are noted. Time lines are 0.2 sec.
Aortic pressure at end diastole and pulse pressure tended to be abnormal in the groups with aortic regurgitation, but there was considerable overlap with the controls, even in subjects with marked aortic regurgitation. No significant relationship was found between the regurgitant flow and the end-diastolic pressure. Pulse pressure was related significantly to the regurgitant flow with a low coefficient of correlation (fig. 3).

Table 2 summarizes the correlation coefficients with their P values between the pressure measurements and the regurgitant flow in both groups.

### Discussion

A simple and accurate technic for estimating the regurgitant flow in aortic regurgitation would have great usefulness. Supravalvular cineangiography, the most commonly used method, is highly subjective. Cohn and associates \(^2\) compared this method to the magnitude of aortic regurgitation observed at surgery. The correlation was good when the regurgitant flow was either absent or severe but held up poorly in the intermediate groups. Regurgitation graded as 2+ by cineangiography was shown to vary between trivial and severe at surgery. Sandler’s group, \(^8\) and Arvidsson and Kornell \(^9\) developed the relatively accurate technic using biplane angiocardiography; however, this method is tedious, requires expensive radiographic equipment, and cannot distinguish between regurgitation and stenosis.

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**Table 1**

<table>
<thead>
<tr>
<th>Hemodynamic data</th>
<th>Group</th>
<th>Range</th>
<th>Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left ventricular minute flow (L/min)</td>
<td>Controls*</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td>AR†</td>
<td>5.3 - 25.7</td>
<td>12.5</td>
</tr>
<tr>
<td></td>
<td>AR + AS‡</td>
<td>4.6 - 12.4</td>
<td>8.7</td>
</tr>
<tr>
<td>Fick cardiac output (L/min)</td>
<td>Controls</td>
<td>4.5 - 10.3</td>
<td>7.0</td>
</tr>
<tr>
<td></td>
<td>AR</td>
<td>4.1 - 7.1</td>
<td>5.5</td>
</tr>
<tr>
<td></td>
<td>AR + AS</td>
<td>3.8 - 7.0</td>
<td>5.1</td>
</tr>
<tr>
<td>Regurgitant flow (L/min)</td>
<td>Controls</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td>AR</td>
<td>1.1 - 20.5</td>
<td>7.0</td>
</tr>
<tr>
<td></td>
<td>AR + AS</td>
<td>0.5 - 7.8</td>
<td>3.6</td>
</tr>
<tr>
<td>Aortic systolic pressure (mm Hg)</td>
<td>Controls</td>
<td>100 - 132</td>
<td>119</td>
</tr>
<tr>
<td></td>
<td>AR</td>
<td>107 - 212</td>
<td>145</td>
</tr>
<tr>
<td></td>
<td>AR + AS</td>
<td>94 - 187</td>
<td>131</td>
</tr>
<tr>
<td>Aortic pressure at end diastole (mm Hg)</td>
<td>Controls</td>
<td>62 - 90</td>
<td>75</td>
</tr>
<tr>
<td></td>
<td>AR</td>
<td>37 - 96</td>
<td>57</td>
</tr>
<tr>
<td></td>
<td>AR + AS</td>
<td>37 - 80</td>
<td>59</td>
</tr>
<tr>
<td>Diastolic pulse pressure (mm Hg)</td>
<td>Controls</td>
<td>7 - 29</td>
<td>21</td>
</tr>
<tr>
<td></td>
<td>AR</td>
<td>23 - 57</td>
<td>40</td>
</tr>
<tr>
<td></td>
<td>AR + AS</td>
<td>21 - 49</td>
<td>32</td>
</tr>
<tr>
<td>Corrected diastolic pulse pressure (mm Hg)</td>
<td>Controls</td>
<td>7 - 29</td>
<td>21</td>
</tr>
<tr>
<td></td>
<td>AR</td>
<td>22 - 66</td>
<td>44</td>
</tr>
<tr>
<td></td>
<td>AR + AS</td>
<td>23 - 45</td>
<td>34</td>
</tr>
<tr>
<td>Diastolic slope (mm Hg/sec)</td>
<td>Controls</td>
<td>18 - 56</td>
<td>45</td>
</tr>
<tr>
<td></td>
<td>AR</td>
<td>42 - 142</td>
<td>87</td>
</tr>
<tr>
<td></td>
<td>AR + AS</td>
<td>43 - 98</td>
<td>67</td>
</tr>
</tbody>
</table>

* N = 15.
† N = 27.
‡ N = 23.

Abbreviations: AR = isolated aortic regurgitation; AR + AS = combined aortic regurgitation and stenosis.
Table 2

<table>
<thead>
<tr>
<th>Hemodynamic data</th>
<th>Isolated aortic regurgitation</th>
<th>Aortic regurgitation and stenosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aortic pressure at end diastole</td>
<td>( r = 0.15 )</td>
<td>( r = 0.29 )</td>
</tr>
<tr>
<td>Aortic pulse pressure</td>
<td>( r = 0.36 )</td>
<td>( r = 0.17 )</td>
</tr>
<tr>
<td>(peak systolic-end diastolic pressure)</td>
<td>( P = 0.05 )</td>
<td>( NS )</td>
</tr>
<tr>
<td>Diastolic pulse pressure</td>
<td>( r = 0.75 )</td>
<td>( r = 0.19 )</td>
</tr>
<tr>
<td>(pressure at dicrotic notch- aortic pressure at end diastole)</td>
<td>( P &lt; 0.001 )</td>
<td>( NS )</td>
</tr>
<tr>
<td>Corrected diastolic pulse pressure</td>
<td>( r = 0.82 )</td>
<td>( r = 0.16 )</td>
</tr>
<tr>
<td>(slope of line through dicrotic notch and end diastole)</td>
<td>( P = 0.001 )</td>
<td>( NS )</td>
</tr>
<tr>
<td>Diastolic slope</td>
<td>( r = 0.71 )</td>
<td>( r = 0.20 )</td>
</tr>
</tbody>
</table>

*NS = not significant.

Figure 2

Relation between aortic regurgitation (L/min) and both the corrected diastolic pulse pressure and the diastolic slope in isolated aortic regurgitation. The sloping line represents the regression equation. The bar on the abscissa represents the range of our controls.

occurring at the mitral and aortic valves. Hill's sign has been advocated as an accurate and simple clinical tool for estimating aortic regurgitation,\(^{13}\) but Pascarelli and co-workers,\(^{14}\) showed by direct measurement that the sign is an artifact of the indirect technic for determining blood pressure.

Many variables determine the aortic pulse pressure and aortic pressure at end diastole in aortic regurgitation including stroke volume, heart rate, force of systolic contraction, aortic compliance, peripheral resistance, regurgitant valve area, and the presence or absence of aortic stenosis. Previous authors\(^{6, 12, 13}\) have
ESTIMATION OF AORTIC REGURGITATION

Relation between aortic regurgitation (L/min) and both aortic pressure at end diastole and pulse pressure. The bar on the abscissa represents the range of our controls.

Additional circuit for aortic insufficiency

Additional resistor for aortic stenosis

Simplified electrical analog of the cardiovascular systems with circuits for aortic insufficiency and stenosis. Electrical symbols and their cardiovascular equivalents (given in parentheses): G = pulse wave generator (left ventricle); D = diode (unidirectional valve) (aortic valve); C = capacitor (vascular compliance); L = inductor (blood mass [inertia]); R1 = resistor (systemic resistance); R2 = resistor (regurgitant valve resistance); R3 = resistor (systolic resistance with aortic stenosis).

demonstrated the poor correlation between aortic regurgitation and both the aortic pressure at end diastole and pulse pressure. Our data further confirm their findings. Examination of the aortic diastolic pulse wave as a means of estimating aortic regurgitation seems more reasonable since it is less dependent on systolic events. The fall in aortic diastolic pressure is mainly determined by the regurgitant valve area, and to a lesser extent

Circulation, Volume XLI, April 1970
on peripheral resistance, aortic compliance, and heart rate. The effect of heart rate can be minimized by considering the rate of fall of the diastolic pressure (diastolic slope) or by correcting the diastolic pulse pressure for diastolic period (corrected diastolic pulse pressures). Rees and associates examined the rate of decline in the aortic diastolic pressure and found it was not of value in estimating the regurgitant flow. His series, however, consisted of eight patients, five of whom had associated aortic stenosis. Libanoff measured the time required for the diastolic aortic-ventricular pressure difference to fall to one half its initial value. He found this half time to correlate well with the regurgitant flow with or without associated aortic stenosis. It was not stated how the reflux was independently measured. Although our measurements on the diastolic pressure wave are not exactly similar, they showed poor correlation with the regurgitant flow in the presence of aortic stenosis. However, in isolated aortic regurgitation, our diastolic pressure wave measurements correlated well with the regurgitant flow.

Since these diastolic measurements are in part determined by peripheral resistance and aortic compliance they may be affected by systemic hypertension, severe arteriosclerosis, or other states which alter these parameters.

A simplified electrical analog model of the cardiovascular system is useful to help explain these findings (fig. 4). Normally, during systole, the pulse generator charges the capacitor and causes flow through L and R1. During diastole the capacitor discharges causing flow to continue through L and R1. In aortic regurgitation the situation during systole is similar; however, during diastole the capacitor now discharges across the parallel resistors R2 (regurgitant flow into the left ventricle) and R1. The rate of the capacitor discharge (rate of pressure fall) is inversely proportional to the resistance in the circuit. Since the sum of parallel resistors is always less than either resistor considered separately, the rate of pressure fall will increase. If the values of C, L, and R1 are relatively constant, then the rate of pressure fall is mainly determined by the value of R2 (regurgitant orifice size), and the correlation between the regurgitant flow and the aortic diastolic pressure fall should be good. Our data suggest that this is the case.

When aortic stenosis is also present, an additional resistor (R3) must be added to the circuit. In this case, during systole a voltage drop will occur across R3 (pressure gradient) which will affect the charge imposed on the capacitor. The higher the resistance at R3, the less charge the capacitor will receive. The charge on the capacitor thus becomes highly variable and affects the rate of pressure fall in diastole. We think this is at least one explanation for the lack of correlation between the rate of diastolic pressure fall and the regurgitant flow in combined aortic valvar disease.

Further research may provide methods for quantitating the values of capacitance, inductance, and resistance, thereby enabling the calculation of regurgitant flow from diastolic pressure wave analysis in the presence of aortic stenosis.

References

7. Leeman JS, Boyle JJ, Debas JN: Quantitation of aortic valvar insufficiency by catheter
Estimation of Aortic Regurgitation


The Double Murmur of Aortic Insufficiency

Duroziez (1861)

The double murmur can be produced in two ways, by means of the stethoscope or by means of the hand. With the stethoscope pressure is exerted to completely compress the artery; at a certain moment the double murmur will appear; only when the second murmur can be readily produced is it possible to place the stethoscope on the artery without pressure and then gradually slight pressure can be exerted with the hand above and below the stethoscope. Pressure above will produce the first murmur, while pressure below will produce the second murmur; it is evident that the second murmur is produced by the arteries of the legs, which propel the blood backwards and in some manner empty the capillaries.—P. Duroziez: The Double Intermittent Murmur over the Femoral Arteries as a Sign of Aortic Insufficiency. In Willis F A, and Keys T E: Classics of Cardiology, vol. 2. New York, Dover Publications, Inc., 1941, p. 495.
Estimation of Aortic Regurgitation by Diastolic Pulse Wave Analysis
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Circulation. 1970;41:659-665
doi: 10.1161/01.CIR.41.4.659
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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