Percentage of Left Ventricular Stroke Work Loss

A Simple Hemodynamic Concept for Estimation of Severity in Valvular Aortic Stenosis

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SUMMARY

The ratio of the mean systolic transaortic valvular gradient to the mean systolic left ventricular pressure times 100 yields a percentage that represents the portion of left ventricular pressure-volume work per stroke lost because of outflow-tract obstruction. The percentage of the left ventricular stroke work loss and the area of the aortic orifice have been estimated in 49 patients with aortic valvular stenosis and two open-chest dogs with varying degrees of supravalvular aortic constriction. Left ventricular stroke work loss of 30% or greater was associated with calculated aortic valve areas of 1.0 cm² (or 0.60 cm²/m²) or less. These values are representative of serious obstruction to left ventricular outflow, that is, approximately 75% or greater reduction of normal orifice size. The calculation of the percentage of left ventricular stroke work loss, besides being simple, is based upon the most reliable measurements obtainable at left heart catheterization, that of the pressure pulses.

Additional Indexing Words:
Ventricular function
Cardiac output
Cardiac efficiency

Ventricular pressure-volume curves

Clinical methods for assessing the severity of valvular aortic stenosis are not always satisfactory, and accepted hemodynamic methods have not related the severity of the stenosis to left ventricular stroke work. Calculation of the ratio of the mean systolic transaortic valvular gradient to the mean systolic left ventricular pressure, as herein presented, yields a value that is a function of left ventricular pressure-volume work per stroke dissipated in imparting velocity to the column of blood at the site of the obstruction. The calculation is simple and dependent upon the most accurate measurements obtainable at left heart catheterization, that is, the pressure pulses. This study demonstrated that this simple ratio correlates closely with the more elaborate methods frequently employed.

Derivation of Formula for Percentage of Left Ventricular Stroke Work Loss

Mechanical work of the left ventricle can be calculated as the product of left ventricular mean systolic pressure (LV-sm), the stroke volume (SV), and a factor (0.0144) used to express the result in conventional units (grammeters); that is,

\[
\text{Total LV pressure-volume stroke work} = \text{LV-sm (mm Hg)} \times \text{SV (ml)} \times 0.0144.
\]

Effective pressure-volume stroke work of the left ventricle can be calculated by substituting aortic mean systolic pressure (Ao-sm) in the equation; that is,

\[
\text{Effective LV pressure-volume stroke work} = \text{Ao-sm (mm Hg)} \times \text{SV (ml)} \times 0.0144.
\]
When Ao-sm is substituted for LV-sm, calculation of total LV work per stroke must be increased by the kinetic energy term \( \frac{1}{2} mv^2 \) in which \( m = \) mass of fluid moved and \( v = \) velocity). Kinetic work is expended in imparting velocity to the stroke volume and is a relatively negligible portion of total LV work when the aortic orifice area is normal. However, with decreasing orifice size and also with increasing stroke volume, kinetic work increases. Torricelli's theorem states that \( v^2 = 2gh \) \((g = \) gravity acceleration factor and \( h = \) pressure differential). Thus, in valvular aortic stenosis, the pressure differential or mean systolic pressure gradient (MSG) is a function of \( v^2 \) and of kinetic work, and its magnitude is a measure of LV mechanical work expended in imparting velocity to the stroke volume,

\[
\text{Pressure-volume stroke work loss (g-m) = } [\text{LV-sm (mm Hg)} \times \text{SV (ml)} \times 0.0144] - [\text{Ao-sm} \times \text{SV} \times 0.0144] = \text{MSG} \times \text{SV} \times 0.0144.
\]

Thus, the percentage of left ventricular stroke work loss (% LVSWL) is the ratio of the pressure-volume work lost in imparting kinetic energy to the total left ventricular pressure-volume work × 100:

\[
\% \text{ LVSWL} = \frac{\text{MSG} \times \text{SV} \times 0.0144}{\text{LV-sm} \times \text{SV} \times 0.0144} \times 100.
\]

Since stroke volume and the conversion factor appear both in the numerator and the denominator, they can be algebraically cancelled from the equation. Thus,

\[
\% \text{ LVSWL} = \frac{\text{MSG}}{\text{LV-sm}} \times 100.
\]

Methods

Clinical Studies

Forty-nine patients with pure valvular aortic stenosis and without aortic insufficiency were studied during diagnostic cardiac catheterization. Two patients had associated severe mitral stenosis: one patient had moderate mitral insufficiency, and the other had severe systemic hypertension. The ages of the 49 patients ranged from 2 months to 73 years, and with four exceptions (ages 25, 30, 38, and 39 years), the series was composed of two age groups. Twenty-six patients were less than 16 years of age (21 males and five females) and 19 were more than 50 years of age (13 males and six females).

Left ventricular (LV) and ascending aortic (Ao) pressures were recorded simultaneously or by "pull back" from the left ventricle to the aorta, on photographic paper using a Visicorder (Minneapolis Honeywell Co., Model 1012), and strain-gauge transducers (Statham, Model P23Db). The simultaneous pressures were recorded at equal sensitivities on the same base line. The catheter-manometer systems used have frequency responses that are uniform to greater than 12 cycles per second.

Cardiac output was calculated by the indicator-dilution technique, with the use of indocyanine green and a cuvette oximeter (Waters Corp., Model XC50B), according to the method of Hamilton and associates. The systems employed and the technique used have been described in detail. Dilution curves for calculation of cardiac output were obtained within 1 to 3 minutes of the pressure-pulse record. The stroke volume (SV) was obtained by dividing the cardiac output by the heart rate.

Experimental Studies

Two mongrel dogs were anesthetized with pentobarbital and restrained in the supine position. Femoral arteriovenous fistulas were established bilaterally. The chest was opened through a sternum-splitting incision. A carotid artery and jugular vein were exposed and 6-F catheters (Goodeal-Lubin) were manipulated under fluoroscopic and manometric control into the superior vena cava for the injection of indocyanine green and into the aortic arch for sampling of indicator. Pressure pulses were recorded simultaneously, at the same sensitivities and on the same base line, directly from the sinus of the left ventricle and from the aorta 3.0 cm above the aortic valve. Special catheter-manometer systems were used. These consisted of flanged, 19-gauge, thin-walled needles (1.5- and 1.0-cm long); nylon tubing with an internal diameter of 0.039 inch and a length of 12 cm; strain-gauge transducers (Statham, Model P-23Db); and galvanometers (Heiland, Model 40-350) which were electrically damped by a 300-ohm resistance. The frequency response of this system is uniform to 35 cps and has a variability of 10%. Cardiac output was measured by the indicator-dilution technique, with the use of a densitometer (Waters Corp., Model XC100). Calculations of cardiac output and SV were done as in the clinical studies. The descending limb of the dilution curves was not significantly affected by recirculated indicator particles when
the fistulas were open, and thus reasonable estimates of cardiac output could be calculated. Inclusion of recirculated indicator particles in the calculations would have lowered the estimated cardiac output.

When relative stability was obtained, control observations that included the recording of indicator-dilution curves and pressure pulses were made when arteriovenous fistulas were closed (control I), when one fistula was open (control II), and when two fistulas were open (control III). Then, the aorta was constricted between the aortic valve and the needle that recorded aortic pressure by tightening an encircling tape until a pressure gradient was detected. After 5 minutes, indicator-dilution curves and pressure pulses were again recorded with fistulas closed (I), one fistula open (II), and both fistulas open (III). The aorta was progressively constricted in stages, and the variables recorded for each orifice size. Orifice area in square centimeters was calculated from the length of the circumferential tape minus the thickness of the aortic wall.

Calculations

The left ventricular mean pressure and the mean aortic pressure during systole were obtained by planimetric integration. The mean systolic pressure gradient was obtained by subtraction. Aortic valve closure was considered to coincide with the dicrotic notch of the aortic pressure pulse contour.

From these data, the following values were calculated:

1. The area of the aortic orifice (AVA) was calculated using the Gorlin equation:

\[
AVA = \frac{CO/SEP}{C \times 44.5 \times \sqrt{\text{LV-sm}} - \text{(Ao-sm)}}
\]

in which

- \(CO\) = cardiac output (ml/min),
- \(SEP\) = systolic ejection period (systolic sec/min),
- \(LV-sm\) = left ventricular mean systolic pressure (mm Hg),
- \(Ao-sm\) = aortic mean systolic pressure (mm Hg),
- \(44.5\) = \(\sqrt{2g}\) = gravity acceleration factor,
- \(C\) = empiric constant = 1, and
- \(AVA\) = aortic valve area (cm²).

2. Left ventricular stroke work loss was calculated as outlined previously.

3. Left ventricular stroke work loss was calculated using peak pressures (LVSWL-p).

\[
% \text{LVSWL-p} = \frac{\text{PSG}}{\text{PLVsp}} \times 100
\]

in which the peak systolic gradient (PSG) was obtained by subtraction of the average peak aortic pressure from the average peak left ventricular systolic pressure (PLVsp).

4. Since the mean systolic pressure gradient (MSG) is a function of \(v^2\), the quotient (LVSWL-K) of the square root of MSG divided by LV-sm was calculated.

\[
(LVSWL-K) = \sqrt{\frac{\text{MSG}}{\text{LV-sm}}} \times 100.
\]

Results

Hemodynamic Data

Values at Rest

Table 1 lists the hemodynamic data obtained by diagnostic catheterization from which indices of severity of valvular aortic stenosis were calculated. The aortic valve area is not listed in five instances, since no temporal relationship existed between pressure-pulse records and measurements of cardiac output. Mean systolic gradients ranged from 11 to 105 mm Hg, aortic valve areas ranged from 1.9 to 0.3 cm² or 1.3 to 0.2 cm²/m² values for left ventricular stroke work loss ranged from 12 to 58%, and values for LVSWL-K ranged from 3.6 to 6.0. The correlation between MSG and the aortic valve area was \(-0.31\) (fig. 1). In
all but five patients, MSG values greater than 40 mm Hg were associated with aortic valve areas of less than 1.0 cm². The correlation between the left ventricular stroke work loss and the aortic valve area in cm² and cm²/m² was −0.79 and −0.80, respectively (fig. 2). With five exceptions (cases 5, 6, 9, 10, and 47), patients who had aortic valve areas of 1.0 cm² or less had greater than 30% left ventricular stroke work loss, and with two exceptions (cases 9 and 47) patients who had aortic valve areas (AVA) of 0.6 cm²/m² or less also had left ventricular stroke work loss greater than 30% (fig. 3). Two patients (cases 3 and 4) had AVA of greater than 0.6 cm²/m² and LVSWL of greater than 30%. With peak systolic pressures used to calculate left ventricular stroke work loss, aortic valve areas of 0.6 cm²/m² or less were associated with greater than 33% LVSWL-p (fig. 4) in all but five cases, and in another five cases, LVSWL-p of greater than 33% was associated with AVA of greater than 0.6 cm²/m² (r = −0.64). The correlation between values for LVSWL obtained when mean pressures and peak pressures were used was + 0.88. The correlation between the left ventricular stroke work loss constant and the aortic valve area is seen in figure 5. Each individual, with two exceptions (cases 48 and 49), who had an aortic valve area of 0.6 cm²/m² or less had a left ventricular stroke work loss constant (LVSWL-K) greater than 4.65. Four patients (cases 3, 4, 6, and

![Figure 2](image1.png)

**Figure 2**

Relationship of per cent left ventricular stroke work loss to aortic valve area in cm²/m².

![Figure 3](image2.png)

**Figure 3**

Relationship of 30% loss of left ventricular stroke work to aortic valve areas of 0.6 cm²/m².

![Figure 4](image3.png)

**Figure 4**

Relationship of 33% loss of left ventricular stroke work, calculated by using peak pressures, to aortic valve areas of 0.6 cm²/m².

![Figure 5](image4.png)

**Figure 5**

Relationship of the left ventricular stroke work loss constant to aortic valve in cm²/m².
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<tr>
<th>Case</th>
<th>Age (yr)</th>
<th>LV-sm* (mm Hg)</th>
<th>Ao-sm* (mm Hg)</th>
<th>MSG* (mm Hg)</th>
<th>Stroke volume (mL)</th>
<th>Systolic ejection period (sec)</th>
<th>Aortic valve area (cm²)</th>
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10) had AVA of greater than 0.6 cm²/m² in association with LVSWL-K greater than 4.65 (r = -0.54).

Values Obtained During Other Physiological States

In nine of the 49 patients studied, left ventricular and aortic pressure pulses and indicator-dilution curves for measurement of cardiac output were recorded at rest and during at least one other physiological state (table 2). Alteration of the physiological status of the patient induced changes in cardiac output that ranged from +58% (exercise) to -34% (anesthesia) and in calculated indices of severity that ranged from 2 to 38 mm Hg for MSG, from 0.1 to 0.2 cm² for aortic valve area, from 0 to 11% for left ventricular stroke work loss, and from 0.1 to 0.4 for left ventricular stroke work loss constant. The correlation between the calculated left ventricular stroke work loss at rest and that during other physiological states was +0.82 (fig. 6). The correlation between the calculated left ventricular stroke work loss constant at rest and that during other physiological states was +0.92 (fig. 6).

Experimental Studies

Table 3 lists the hemodynamic data derived from experiments in two open-chest dogs and demonstrates the effects of progressive, stepwise, supravalvular constriction of the aorta on the left ventricular stroke work loss and the left ventricular stroke work loss constant. In both animals, systolic gradients appeared abruptly when the aorta was constricted to approximately 35% of normal and increased precipitously with further constriction (fig. 7). Constriction of the aorta to less than 0.7 cm² (40 to 47% LVSWL) in one animal and to less than 1.0 cm² (37 to 43% LVSWL) in the other was followed by ventricular fibrillation. The effects of variation in the cardiac output and stroke volume, induced by opening one or two femoral arteriovenous fistulas, on left ventricular stroke work loss are listed in table 3 at each degree of constriction. These data indicate that an increase
Table 2
Effects of Changes in Cardiac Output on Aortic Orifice Area, Per Cent of Left Ventricular Stroke Work Loss, and Left Ventricular Stroke Work Loss Constant

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<th>Physiological state</th>
<th>Cardiac output (L/min)</th>
<th>Stroke volume (ml)</th>
<th>Change in SV (%)</th>
<th>LV-sm (mm Hg)</th>
<th>MSG (mm Hg)</th>
<th>Systemic resistance (mm Hg L/min)</th>
<th>Aortic valve area (cm²)</th>
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*Change over initial value.
†Left ventricular stroke work loss = \( \frac{\sqrt{\text{MSG}}}{\text{LV-sm}} \times 100 \).
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**Table 3**

Effects of Progressive Decrease in Orifice Area and Changes in Cardiac Output on Percentage of Left Ventricular Stroke Work Loss

1 = Fasts closed; II = one A-V fistula open; III = both A-V fistulas open.

Change from fistulas closed.

Left ventricular stroke work loss constant = \( \frac{\sqrt{MS}}{L.V.sm} \times 100 \).

Circulation, Volume XXXV, May 1967
in changes in left ventricular stroke work loss constant that do not exceed the control value by more than 0.3.

**Discussion**

Several methods are used to evaluate the severity of valvular aortic stenosis. The peak systolic pressure gradient between the left ventricle and the aorta or a peripheral artery is an accepted standard of severity, and a peak pressure gradient greater than 50 mm Hg is considered to indicate a "significant" degree of outflow obstruction.\(^1\)\(^8\)\(^9\)\(^10\) There are valid objections to the use of these pressure gradients as standards of severity: (1) The pressure gradient across a fixed-orifice obstruction is dependent upon blood flow. Thus, the abnormally low cardiac outputs that may be present with incipient left ventricular failure or anesthesia may be associated with peak pressure gradients less than the above "standard," despite the presence of severe aortic stenosis. (2) Many investigators derive peak pressure gradients from simultaneously recorded left ventricular and peripheral artery pressure pulses. Since there are significant amplitude and phase differences between the

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**Figure 6**

Comparison of per cent of left ventricular stroke work loss (Left Panel) and left ventricular stroke work loss constant (Right Panel) during states of altered cardiac output with values obtained at rest. Note the absence of significant changes from the resting values despite the intervention of anesthesia, exercise, or isoproterenol.

**Figure 7**

Supravalvular aortic constriction in open-chest dogs showing effect of progressive decrease in orifice area and changes in cardiac output in percentage of left ventricular stroke work loss. For each rectangle (orifice size), measurements were made with differing cardiac output values caused by the release of arteriovenous fistulas. In general, for a given degree of constriction, the value for per cent of LVWSL increased inconsistently in relation to blood flow.

\(y = 0.86x + 5.87\)

\(S_{yx} = 7.24\)

\(r = +.82\)

\(y = 1.13x - 0.63\)

\(S_{yx} = 0.37\)

\(r = +.92\)
peripheral pressure pulse and the central aortic pressure pulse, the peak pressure gradient thus obtained may be inconsistent among laboratories, among patients, or among records of a single patient. Recognizing the errors introduced by the use of peak pressure gradients alone in the evaluation of the severity of semilunar valvular stenosis, Dow and associates,11 Silber and co-workers,12 and Gorlin and Gorlin7 evolved formulas to derive indices of severity that included blood flow. The hydraulic formula of Gorlin and Gorlin7 has been widely accepted in spite of significant criticism.13 When aortic valve area is estimated, maximum opportunity for error is found in the calculation of the mean rate of blood flow per unit time. In this study, aortic valve area has been used as the standard for comparison, although the probability of significant error is recognized in such a standard.

The normal aortic valve area (AVA) in man ranges from 3.0 to 4.0 cm².14 In experimental studies, the effective orifice must be reduced to 25% or less of its natural size before serious hemodynamic effects are seen15,16; this was also found in our animal experiments (table 3). Although knowledge obtained from experimental studies is not strictly applicable to valvular aortic stenosis in man, narrowing an orifice to 25% of original size would be equivalent to aortic valve areas of 0.7 to 1.0 cm² or 0.4 to 0.6 cm²/m² (assuming an average body surface area of 1.75 m²) and should be indicative of hemodynamically serious obstruction of the outflow tract. Braunwald and associates8 considered an AVA of 0.5 to 0.75 cm²/m² as indicative of severe outflow-tract obstruction. Figure 2 shows an inverse relationship between the aortic valve area and the per cent LVSWL. Aortic valve areas of 0.6 cm²/m² or less, were found in 27 of 29 patients with LVSWL of 30% or greater. Each of these values can be considered to describe hemodynamically serious valvular aortic stenosis.

The inverse relationship between the per cent LVSWL and the orifice area demonstrated in figure 2 is predictable from consideration of the terms of each formula. The denominator of the LVSWL formula (LV-sm) has a functional relationship to the numerator of the orifice equation (stroke volume),17 and the mean systolic gradient (MSC) is the numerator of the LVSWL formula whereas the square root of MSG is a factor in the denominator of the orifice equation. Thus, despite the implications of figure 2, the inverse relationship between per cent LVSWL and the orifice area should be second order and nonlinear. MSG is a measure of the square of the velocity of blood flow which varies inversely with orifice size and directly with stroke volume. To assess the relative importance of variation in stroke volume upon per cent LVSWL, percentage changes in stroke volume (tables 2 and 3) were plotted against percentage changes in MSG and LVSWL (r = +0.24 and +0.02, respectively). Thus, decreasing orifice size apparently is the major factor in the genesis of MSG and LVSWL.

Percentage of LVSWL and values for LVSWL-K were calculated from hemodynamic data both at rest and during another physiological state (table 2). The plots in figure 6 indicate that in the individual patient, LVSWL-K values are more constant than per cent LVSWL. The data in table 3 also indicate that at any degree of aortic constriction, the LVSWL-K value was relatively constant despite alterations in the cardiac output. Thus, some data support the concept that in an individual patient with valvular aortic stenosis, the LVSWL-K may tend to remain constant regardless of the physiological state and may estimate the severity of aortic valvular stenosis more accurately than does the percentage of LVSWL. This concept was tested in 49 individuals with valvular aortic stenosis (fig. 5), but the use of LVSWL-K values did not improve the negative correlation with aortic valve area. Percentage of LVSWL rather than LVSWL-K seems to correlate better with aortic valve area, but both ratios can be expected to have imperfect correlations with aortic valve area, since the relationships between MSG, LV-sm, and AVA in valvular aortic stenosis cannot be simply described for the following reasons: (1) The
physical laws used are exact only in a rigid system with constant laminar flow of a nonviscous solution, whereas, the heart and aorta are a two-chambered system in which the flow is pulsatile, the walls are visco-elastic, the solution is viscous and, with valvular stenosis, the flow beyond the orifice is turbulent. In addition, the aorta is an elastic volume container ("Windkessel"),\textsuperscript{18} and outflow from the aortic container is dependent upon the peripheral resistance. Aortic mean systolic pressure is dependent upon left ventricular systolic ejection, vascular elasticity, and peripheral resistance. Thus, the peripheral resistance, which is variable under the influence of nervous, humoral, and local-tissue factors and usually changes inversely with blood flow (table 2), is also a determinant of the mean systolic pressure gradient. (2) Inherent errors in calculations of aortic valve area also contribute to imperfections in the demonstrated relationships between the percent LVSWL, LVSWL-K, and the AVA. In this hydraulic formula, the square root of the mean systolic valvular gradient is used to estimate velocity. Thus, the previous statements, which place limitations on the use of the MSG in the estimation of velocity, also apply to AVA. Other sources of error that exist in the hydraulic formula result from errors in the estimation of blood flow per systolic second and in the limitations that exist in the theoretic derivation of the orifice coefficient C.\textsuperscript{3}

Despite theoretic imperfections, estimations of the severity of valvular aortic stenosis based on aortic valve area, percentage of left ventricular stroke work lost, and left ventricular stroke work loss-constant are of practical value in the management of patients, and in this study, these estimates were in good agreement. Left ventricular stroke work loss of 30% or greater or left ventricular stroke work loss-constants of 4.65 or greater were associated with aortic valve areas of 1.0 cm\textsuperscript{2} (or 0.6 cm\textsuperscript{2}/m\textsuperscript{2}) or less. Percentage of left ventricular stroke work loss calculated using peak pressures had a high positive correlation with values obtained using mean pressures (fig. 4) and provides a useful value quickly.

Acknowledgment
The assistance of Dr. H. M. Schoolman and Mr. J. M. Bechtel of the Biostatistics Research Support Center at Veterans Administration Hospital, Hines, Illinois, is acknowledged.

References
12. SILBER, E. N., PREG, O., GROSSMAN, N., AND
LEFT VENTRICULAR STROKE WORK


Angina Pectoris and Claudication from a Tourniquet, 1809

In health, when we excite the muscular system to more energetic action than usual, we increase the circulation in every part, so that to support this increased action, the heart and every other part has its power augmented. If, however, we call into vigorous action, a limb round which, we have with a moderate degree of tightness applied a ligature, we find that then the member can only support its action for a very short time; for now its supply of energy and its expenditure, do not balance each other; consequently, it soon, from a deficiency of nervous influence and arterial blood, fails and sinks into a state of quiescence. A heart, the coronary vessels of which are cartilaginous or ossified, is in nearly a similar condition; it can, like the limb, be girt with a moderately tight ligature, discharge its functions so long as its action is moderate and equal.—ALLAN BURNS: Observations on Some of the Most Frequent and Important Diseases of the Heart (1809). New York Academy of Medicine, History of Medicine Series. New York, Hafner Publishing Co., 1964, p. 138.
Percentage of Left Ventricular Stroke Work Loss: A Simple Hemodynamic Concept for Estimation of Severity in Valvular Aortic Stenosis

JOHN R. TOBIN, JR., SHAHBUDIN H. RAHIMTOOLA, PETER E. BLUNDELL and H. J. C. SWAN

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