Myocardial Function and Left Ventricular Volumes in Acquired Valvular Insufficiency

By Graham A. H. Miller, B.M., B.Sc., John W. Kirklin, M.D., and H. J. C. Swan, M.B., Ph.D.

End-systolic and end-diastolic volumes of the left ventricle can be determined by angiographic technics. In cases of valvular insufficiency, this information permits quantitation of the regurgitant flow and also, more important, allows investigation of the response of the ventricle to the volume load imposed by such insufficiency.

Measurements of volume of the left ventricle were made in 37 adult patients with acquired valvular insufficiency. Left ventricular volumes were measured by the angiocardioGraphic technic of Arvidsson,1 which appears to have sufficient accuracy for our purposes. The degree of insufficiency was estimated by the method of Sandler and co-workers.2 The data are interpreted to indicate that the degree of cardiac enlargement is only partially related to the magnitude of the incompetence; that the degree of cardiac enlargement is also related to the extent of impairment of myocardial function; that end-diastolic volume is uniformly increased, to a degree commensurate with the volume load in some patients but frequently to a greater degree; and that assessment of myocardial contractility in patients is possible with these technics.

Materials and Methods

The patients included in this study were referred to the cardiovascular laboratory for evaluation of incompetence of either the aortic or the mitral valve or both. A wide range of variations was represented, not only in regard to the valve lesions but also in regard to degree of disability, demonstrable myocardial ischemia, and previous history of myocardial infarction. In 18, surgical evidence was available for identification of the valve or valves involved, for evaluation of the degree of incompetence, and for recognition of the presence and significance of associated valvular stenosis. The 19 patients in whom no surgical procedure was done were classified on the basis of all the information available including the results of diagnostic cardiac catheterization. Group 1 consisted of 12 patients in whom mitral stenosis was considered to be dominant. Because of the method of selection, coexisting valvular incompetence was to be expected in this group of patients. Group 2 consisted of 15 patients in whom mitral incompetence was dominant. Group 3 consisted of 10 patients in whom aortic incompetence was dominant.

Catheterization of the right heart was carried out in all patients and retrograde catheterization of the left heart in 36. Angiocardiography was performed with the patient in the supine position and under light anesthesia (nitrous oxide and oxygen). In all, 100 per cent oxygen was administered for at least 60 seconds prior to angiocardiography. Contrast medium* was injected into the pulmonary artery in two cases and into the left ventricle in 34 cases. In three cases, the injection was made into the aortic root, and the left ventricle was opacified as a result of aortic insufficiency.

One to three minutes before angiocardiography, indocyanine green† was injected into the same chamber and through the same catheter as used for the injection of contrast medium, and indicator-dilution curves were recorded at the femoral artery. Cardiac output was calculated from these curves according to the method of Hamilton,3 and the value obtained was divided by the heart rate during inscription of the curve to give average forward stroke volume (Vs) under anesthesia immediately before angiocardio-

During angiocardiography, the electrocardiogram, arterial pressure, and times of x-ray exposures were recorded simultaneously to permit correlation of the radiographic appearances with the events of the cardiac cycle. Simultaneous an-

* Sodium and methylglucamine diatrizoates (Renovist).
† Cardio-Green, Hynson, Westcott, & Dunning, Baltimore, Maryland.

From the Mayo Clinic and the Mayo Foundation, Rochester, Minnesota.
teroposterior and lateral exposures were made at the rate of six per second with a roll film changer. Measurements of angiocardiograms were made according to the method of Avidsson as previously described. Only those films showing adequate chamber opacification were measured. No patient was included in the study in whom uncontrolled atrial fibrillation or multiple ectopic beats during filming made interpretation of chamber volume impossible. Patients were included, however, if well-controlled atrial fibrillation resulted in reasonably regular ventricular contractions.

Left ventricular volumes ($V_{LV}$) calculated from each pair of biplane angiograms were plotted against interval from the R wave of the electrocardiogram to give a composite volume curve as previously described. Values for left ventricular end-diastolic ($V_{ED}$) and end-systolic ($V_{ES}$) volumes were obtained from the composite volume curves so constructed. All volumes were expressed as milliliters per square meter of body surface area. Body surface area was determined from nomograms constructed according to the formula of Du Bois and Du Bois.

The ejection volume of the left ventricle is given by $V_{ED}-V_{ES}$, and, therefore, includes regurgitant and forward stroke volumes. Ejection volume was expressed as a fraction of end-diastolic volume, $\frac{(V_{ED}-V_{ES})}{V_{ED}}$. Previous studies in this laboratory have demonstrated that angiographically determined stroke volume ($V_{ED}-V_{ES}$) tends to exceed the value for stroke volume determined by indicator dilution ($V_R$) in patients without valvular insufficiency or left-to-right shunts. Comparison of ($V_{ED}-V_{ES}$) and $V_S$ in 30 such patients showed this difference to be of the order of 15 per cent, as indicated by the regression equation

\[
(V_{ED}-V_{ES}) = 3.3 + 1.15 V_S.
\]

The cause of this difference is uncertain but may be due to a brief increase in stroke volume caused by the injection of a large volume of fluid into the central vascular system. In the present study, this small difference is important in the calculation of valvular regurgitation. To minimize errors in ($V_{ED}-V_{ES}$)-$V_S$ the values for $V_S$ were increased, on the basis of the above regression equation, by applying the formula

\[
V_S = 3.3 + 1.15 V_S,
\]

where $V_S$ is the real forward stroke volume during the injection of contrast. The regurgitant volume ($V_R$) is given by

\[
V_R = (V_{ED}-V_{ES})-V_S.
\]

$V_R$ is reported as a percentage of ($V_{ED}-V_{ES}$).

Pressure measurements were made from directprint ultraviolet oscilloscope records; the system used catheter strain gauges (Statham P23 D series) coupled to a Heiland 40.350 galvanometer with a filter damping network giving a flat response to 12 cycles per second. Estimates of the maximal rate of increase of ventricular pressure during isometric contraction (peak dp/dt) were made from 10-cm.-per-second records of left ventricular pressure and expressed as mm. Hg per second. It is recognized that, for precise measurement of peak dp/dt, the frequency response of the system and the measurement method used leave much to be desired. Nonetheless, the estimates of peak dp/dt obtained by this measurement method yielded information of importance and significance and are reported with the limitations of our method in mind.

Left ventricular stroke work ($LVSW$) was obtained as the product of the mean left ventricular systolic pressure ($LVsm$) and the ejection volume ($V_{ED}-V_{ES}$):

\[
LVSW = (V_{ED}-V_{ES}) \times LVsm \times 0.0144.
\]

A horizontal line was drawn on the pressure record at the level of end-diastolic pressure and extended to the descending limb of the pressure tracing. The area (obtained by planimetry) beneath the pressure pulse and above this line was divided by the length of the line to give $LVsm$. The factor, 0.0144, permits expression of $LVSW$ in conventional units (gram meters) and corrects for the density of blood and the conversion of mm. Hg to cm. H$_2$O. In calculating $LVsm$, a mean value for three or four beats was obtained from 10-cm.-per-second records of left ventricular pressure made immediately before angiocardiography.

Since myocardial contractility may be defined in terms of work performed from a given fiber length and, since end-diastolic volume and fiber length are, presumably, closely related, the following index expressing contractility in numerical terms was derived:

\[
\frac{LVSW \ (Gm. \ M.)}{V_{ED} \ (ml.)}.
\]

It is referred to as the “myocardial contractility index.”

**Results**

Hemodynamic data and left heart volumes for all patients are given in tables 1, 2, and 3. Mean ejection volumes ($V_{ED}-V_{ES}$) were
substantially higher than normal \((46 \pm 8.1)\) for all three groups \((62, 112, \text{and} 113 \text{ ml./M.}^2)\) for groups 1, 2, and 3, respectively. The mean left ventricular minute volume was above normal \((3.5 \pm 0.79)\) for each of the three groups \((4.0, 9.0, \text{and} 8.0 \text{ L./min./M.}^2)\) and in only one patient was it below \(3.0 \text{ L./min./M.}^2\) (case 11)—a striking finding in view of the advanced state of disease present in many patients. In contrast, in 22 of the patients the forward stroke volumes \((V_S)\) were abnormally small (less than 30 ml./M.\(^2\)) and were less than 20 ml./M.\(^2\) in seven, of whom five had ejection fractions of less than 0.5.

The frequency distribution of values for ejection fraction in patients with acquired heart disease differs from the frequency distribution for 18 patients with no abnormality of the left heart and 32 with congenital heart disease\(^c\) (fig. 1) in that a significant proportion \((16 \text{ of} 37, \text{43 per cent})\) of the patients with acquired heart disease had values for ejection fraction of less than 0.5 (more than two standard deviations below the mean value for normal left hearts).\(^6\)

The relationship between stroke work and

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Table 1

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*Operated case.

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Figure 1

Frequency distribution (per cent of cases) of values for ejection fraction (fraction of end-diastolic volume ejected during systole) for 37 patients with acquired valvular insufficiency compared with the frequency distribution of values for 18 patients with no abnormality of the left heart and 32 with congenital heart disease.

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### Table 2

**Summary of Results: Group 2 Patients (Dominant Mitral Incompetence)**

| Case | Age (yr); sex | Body surface (M²) | VLV (ml./M²) | VED | VES | VED-VES | Vₚ (ml./M²) | Vₚ (ml./M²) | Vₚ as % VED-VES | LV minute vol (L./M²) | Cardiac index | Ejection fraction | LV peak dp/dt (mm. Hg/sec) | LVSW (Gm. meters) | LVSW/VED | Diagnosis                  |
|------|---------------|-------------------|---------------|------|-----|---------|-------------|-------------|----------------|--------------------|----------------|----------------|----------------|----------------------------|------------------|-----------|--------------------------|
| 13   | 56; F         | 1.45              | 230           | 53   | 177 | 18      | 23          | 154         | 87              | 13.4               | 1.4             | 0.77           | 670            | 135                       | 0.28             | MI (ruptured chordae)*  |
| 14   | 56; M         | 2.00              | 322           | 260  | 62  | 6       | 9           | 53          | 86              | 6.4                | 0.7             | 0.19           | 700            | 116                       | 0.18             | MI                       |
| 15   | 59; M         | 1.70              | 249           | 59   | 190 | 29      | 35          | 155         | 81              | 14.4               | 2.2             | 0.76           | 1060           | 340                       | 0.80             | MI (ruptured chordae)*  |
| 16   | 46; M         | 1.63              | 288           | 161  | 127 | 23      | 28          | 99          | 78              | 12.2               | 2.1             | 0.44           | 1060           | 194                       | 0.41             | MI                       |
| 17   | 56; M         | 1.86              | 161           | 48   | 113 | 20      | 25          | 88          | 78              | 8.8                | 1.5             | 0.70           | 700            | 248                       | 0.83             | MI (ruptured papillary muscle)* |
| 18   | 45; M         | 1.95              | 249           | 189  | 60  | 10      | 13          | 47          | 78              | 8.1                | 1.3             | 0.24           | 670            | 135                       | 0.28             | MI*                      |
| 19   | 53; M         | 2.26              | 249           | 86   | 163 | 37      | 41          | 122         | 75              | 14.0               | 1.3             | 0.65           | 1000           | 343                       | 0.63             | MI                       |
| 20   | 52; F         | 1.70              | 180           | 60   | 120 | 25      | 31          | 89          | 74              | 9.4                | 1.9             | 0.67           | 1380           | 320                       | 1.05             | MI TI*                   |
| 21   | 64; M         | 1.75              | 255           | 122  | 133 | 35      | 43          | 90          | 68              | 7.2                | 1.9             | 0.52           | 940            | 275                       | 0.62             | MI (ruptured chordae)*  |
| 22   | 46; M         | 2.06              | 155           | 54   | 101 | 31      | 37          | 64          | 63              | 9.1                | 2.8             | 0.65           | 1080           | 102                       | 0.32             | MI (myocarditis)*       |
| 23   | 53; M         | 1.70              | 164           | 65   | 99  | 30      | 36          | 63          | 63              | 4.4                | 1.3             | 0.60           | 1160           | 254                       | 0.91             | MI MS                    |
| 24   | 35; M         | 1.47              | 165           | 101  | 64  | 21      | 27          | 37          | 58              | 4.3                | 1.4             | 0.39           | 460            | 67                        | 0.28             | MI AI*                   |
| 25   | 45; F         | 1.65              | 193           | 121  | 73  | 25      | 32          | 40          | 57              | 6.0                | 2.1             | 0.38           | 820            | 109                       | 0.34             | MI MS                    |
| 26   | 31; F         | 1.60              | 225           | 100  | 125 | 48      | 57          | 68          | 55              | 10.5               | 3.9             | 0.56           | 1220           | 216                       | 0.60             | MI                       |
| 27   | 44; F         | 1.81              | 121           | 40   | 81  | 37      | 45          | 36          | 44              | 7.0                | 3.2             | 0.67           | 920             | 170                       | 0.78             | MI AI MS                  |

*Operated case.
end-diastolic volume is shown in figure 2.* Data for eight patients with normal left hearts for whom LVSW could be calculated are included in figure 2 for comparison. In general, patients with low values for ejection fraction have low values for peak dp/dt and form a separate population in that they perform substantially less work from any given end-diastolic volume than do the remainder of the patients.

Figure 3a* shows the relationship between peak dp/dt and ejection fraction. There is a significant correlation \((p < 0.001)\) between peak dp/dt and ejection fraction such that, of the 12 patients with ejection fraction <0.5, nine had values for peak dp/dt <900 mm Hg per second. Of the 18 patients with ejection fraction >0.5, only one had a value for peak dp/dt <900 mm Hg per second. Figure 3b* shows the relationship between peak dp/dt and LVSW/V_{ED} (myocardial contractility index). There is a significant \((p < 0.001)\) correlation between peak dp/dt and LVSW/V_{ED}. Of the 10 patients with values for peak dp/dt <900 mm Hg per second only one had a value for LVSW/V_{ED} >0.4. Of the 20 patients with values for peak dp/dt >900 mm Hg per second only one patient (case 22), who had surgical and necropsy evidence of myocarditis, had a value for LVSW/V_{ED} <0.4. Consideration of figures 2 and 3 suggests that peak dp/dt, ejection fraction and LVSW/V_{ED} are all related to myocardial function. When this is depressed, low values (<900 mm Hg per second, <0.5 and <0.4 for peak dp/dt, ejection fraction and LVSW/V_{ED}, respectively) are to be expected. Among a group of 18 patients with normal left hearts, we have encountered none with <0.5 for LVSW/V_{ED}.

For all patients, V_{ED} is related to V_{R} (fig. 4). In general, at any level of V_{R}, those patients with the high values for V_{ED} had low values for ejection fraction, peak dp/dt, and

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*Cases of aortic stenosis have been excluded from this plot, since pressure overload increases myocardial contractility.\(^8\)

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Figure 2
Relationship between left ventricular stroke work (LVSW, vertical axis) and end-diastolic volume (VED, horizontal axis) for 30 patients with acquired valvular insufficiency (circles) and eight with no abnormality of the left heart (triangles). Patients with aortic stenosis have been excluded from this plot. Left. Data separated according to whether ejection fraction is greater (solid) or less (open) than 0.5. Right. Data separated according to whether peak dp/dt is greater (solid) or less (open) than 900 mm. Hg/sec.

Figure 3
a. Relationship between peak dp/dt (vertical axis) and ejection fraction (horizontal axis) for 30 patients with acquired valvular insufficiency. Lower limits of normal values for peak dp/dt (900 mm. Hg/sec.) and ejection fraction (0.50) are indicated by horizontal and vertical lines, respectively. b. Relationship between peak dp/dt (vertical axis) and myocardial contractility index (LVSW/VED, horizontal axis) for 30 patients with acquired valvular insufficiency. Lower limits of normal values for peak dp/dt (900 mm. Hg/sec.) and myocardial contractility index (0.4) are indicated by horizontal and vertical lines, respectively. (Cases with aortic stenosis have been excluded from the plots in both parts of this figure.)

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LVSW/V_{ED}, suggesting that the end-diastolic volume is determined by both the volume load and myocardial function.

In figure 5 is shown the comparison between V_{R} estimated angiographically and the severity of regurgitation estimated at operation for the 15 patients who underwent operation and in whom such an estimate was made.

**Discussion**

Measurement of known volumes in vitro\(^{10}\) indicates that the angiographic technic has a high degree of accuracy, and a close agreement between stroke volumes determined by the angiographic method and by classic methods\(^{11, 12}\) supports a similar conclusion concerning the ability of this technic to measure a wide range of volumes in vivo. The several restrictions and limitations of the technic have been discussed previously\(^{6, 12}\) but we concur with the opinion of Gleason and Braunwald\(^{13}\) that "...these technics probably provide the most accurate means available at this time for the determination of left ventricular volume in man."

Values obtained by the indicator "wash-out" technic of Bing\(^{14}\) and Holt\(^{15}\) are significantly higher for V_{ED} and significantly lower for ejection fraction than values obtained by angiographic methods. Resolution of these discrepancies has not yet been achieved. Reported herein are comparisons of data for different patients obtained by means of an identical technic; general conclusions from such com-
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Comparisons are presumably valid. Average values for end-diastolic and end-systolic ventricular volumes for each patient were required and satisfactory measures of these volumes can be obtained from a composite curve representing several cardiac cycles, with a degree of precision adequate for the purposes of this study.

Incompetence of the mitral and aortic valves increases the volume load on the left ventricle but the quantitative interrelations of the resulting cardiac enlargement and the severity of valve incompetence have not been established. Also, it is uncertain whether increased ejection volume always results from increased left ventricular end-diastolic volume or can be achieved by a more complete emptying of the ventricle. Most importantly, the relative contribution to heart size of volume load and myocardial function has not been assessed.

No patient in this series with a significantly increased volume load had a normal end-diastolic or end-systolic volume. No patient had a value for ejection fraction two standard deviations in excess of the mean value of 0.67 determined in this laboratory for 18 patients with normal left hearts. Therefore, under chronic conditions the ventricle responds to a volume load by increasing both end-diastolic and end-systolic volume rather than by a more complete emptying of the ventricle than occurs normally.

A significant proportion (43 per cent) of patients had values for ejection fraction more than two standard deviations below the mean determined for 18 patients with normal left hearts, in contrast to a group of 32 patients with congenital heart disease causing volume or pressure overload of the left ventricle (fig. 1). The ejection fraction should give some indication as to the functional status of the myocardium of the left ventricle. Myocardial function may be expressed in terms of “contractility”; when less work is performed from a given fiber length, a reduction in contractility may be said to have occurred. When the ejection fraction is low, the ejection volume is small compared to end-diastolic volume and, since end-diastolic volume reflects end-diastolic fiber length, it follows that (neglecting for the moment the effect of pressure) with low ejection fractions less work is being obtained from a given fiber length than is obtained with higher ejection fractions; that is to say, contractility is impaired. From figure 2 left it will be seen that patients with low ejection fractions do indeed perform less work from any given end-diastolic volume than do patients with normal ejection fractions. (The two patients in the low ejection fraction group [<0.5] who appear from figure 2 left to have normal myocardial contractility had ejection fractions of 0.49 and 0.47, respectively.)

Gleason and Braunwald have presented evidence that the maximal rate of rise of ventricular pressure during isometric contraction (peak dp/dt) is a measure of myocardial contractility, the lower limit of values for patients with volume overload of the left heart being 900 mm. Hg per second. Low values are found in patients with cardiac failure and high values in patients with pressure overload of the left ventricle. Figures 3 and 2 right demonstrate a relationship between peak dp/dt and ejection fraction, and demonstrate that both have low values when contractility is impaired. The data thus suggest that both peak dp/dt and ejection fraction probably are valid indicators of myocardial contractility. A better index of contractility is LVSW/V_{ED}, which, while requiring measurement of LV_{sm} and from this LVSW, relates work to end-diastolic volume and thus provides a measure of myocardial contractility in numerical terms.

For the cases reported here, the amount of external work performed by the left ventricle was often very large by reason of the large ejection volumes. Thus, those patients with low values for ejection fraction may truly be considered to be examples of high-output failure of the left ventricle. Age, duration of disease, and coronary atherosclerosis all may be factors leading to such impaired myocardial function.

In a previous communication a linear relationship was demonstrated between V_{ED}
and \(V_{ES}\) for patients with normal hearts and patients with congenital heart disease; ejection fraction averaged 0.67 for normal and 0.66 and 0.61 for subjects with congenital heart disease causing pressure and volume overload, respectively. A similar linear relationship between end-diastolic volume and the volume load \(\left(V_{ED}-V_{ES}\right)\) would follow from this. Insofar as \(V_R\) reflects volume load, we would expect a linear relationship between \(V_R\) and \(V_{ED}\) for those patients with normal myocardial contractility, and this is found to be so (fig. 4 left). When myocardial contractility is depressed, however (as evidenced by low values for ejection fraction, peak \(dp/dt\) or LVSW/\(V_{ED}\)), we would expect larger values for \(V_{ED}\) at any level of \(V_R\), and again this is found to be so (fig. 4).

Thus, two factors determine the relationship between severity of regurgitation and end-diastolic volume: regurgitant volume and myocardial contractility. It follows that a simple estimate of heart size alone does not permit conclusions about severity of regurgitation or about myocardial failure, since each causes an increase in heart size \(\left(V_{ED}\right)\). Determination of volume and volume change, however, permits an estimate of these two factors and also reveals that the relation between volume load \(\left(V_{ED}-V_{ES}\right)\) and end-diastolic volume is primarily determined by myocardial contractility. Certain patients appear to eject a large volume \(\left(\text{regurgitant plus forward flow}\right)\) in the same proportion to the end-diastolic ventricular volume as normal subjects \(^6\) and in a manner similar to children with congenital mitral insufficiency. \(^4\) Such patients have normal myocardial contractility and can achieve very large minute volumes and maintain a normal forward flow with only moderate increases in end-diastolic ventricular volume (for example, case 15 in table 2). Other patients have a disproportionately reduced ejection fraction and can only maintain the increased ejection volume imposed by regurgitation at the expense of further and considerable increase in end-diastolic (and end-systolic) volume (for example, case 29 in table 3). Such patients have impaired myocardial contractility. When the ability of the ventricle to eject its content (contractility) is impaired, a normal forward flow can only be maintained by increasing ventricular volumes even when there is no regurgitation and thus no increased volume load (for example, case 11 in table 1).

Cardiac output (forward flow) is determined by the relationship between regurgitant and ejection volumes. Ejection volume in turn is determined by the relationship between \(V_{ED}\) and ejection fraction, which latter is primarily related to myocardial function. For example, in case 15 the patient had the most severe volume load calculated (14.4 L./min./M.\(^2\)) but maintained a nearly normal forward stroke volume in spite of a regurgitant volume of 155 ml./M.\(^2\)/beat—the largest calculated—and from an only moderately increased end-diastolic volume. (For this patient, knowledge of total left ventricular stroke volume and mean diastolic mitral valve gradient permitted calculation of the mitral valve area as 2.8 cm.\(^2\), which is well within the range considered possible for subjects with pure mitral insufficiency and cardiac enlargement.) \(^17\)

It seems unlikely that valvular insufficiency would often impose a more severe volume load, yet, in this series, 10 patients (of whom nine had ejection fractions of less than 0.5) had larger end-diastolic volumes. Such patients may maintain a normal forward flow, despite impaired myocardial contractility, but at the expense of further increase in end-diastolic volume (for example, case 29 in table 3); however, the patients with the largest end-diastolic volumes do not necessarily have the largest volume load or the most severe regurgitation, since a decrease in the fraction of end-diastolic volume ejected may reduce the total ejection volume despite the effect of increasing end-diastolic volume. A point may thus be reached where the ejection fraction is so low that, despite a very large end-diastolic volume, the ventricle cannot maintain an adequate ejection volume (regurgitant plus forward flow) and forward flow may decrease to very low levels (for example, case 14 in table 2).

Within the limits of errors of technic and
interpretation of the angiographic method previously described, left ventricular volume determinations permit a reasonable estimate of the magnitude of valvular incompetence (fig. 5). More importantly, determination of the ejection fraction, or better, the fraction LVSW/V_{ED} may permit an estimate of myocardial function, which is a significant part of the cardiac problem in such patients. The nature of this problem is not readily evident on usual clinical grounds. Thus, no relationship was apparent between symptoms and signs of pulmonary and systemic venous hypertension and fluid retention and myocardial function—as might be expected from the large number of factors involved in causing such symptoms and signs in patients with valvular heart disease; nor was the nature of the problem readily apparent solely on the basis of heart size, as clearly demonstrated in this study.

**Summary**

The effect of valvular insufficiency on left ventricular volumes was studied by an angiographic method in 37 patients. Ejection of the additional volume load imposed by valvular insufficiency was achieved by an increase in end-diastolic volume and not by increasing the proportion of end-diastolic volume ejected. Of the 37 patients, 16 (43 per cent) had a significant reduction in the fraction of end-diastolic volume ejected per beat. These patients performed significantly less stroke work from a given end-diastolic fiber length (end-diastolic volume) than did the others and evidence is presented that they had impaired myocardial function. An index of myocardial contractility is derived which relates stroke work to end-diastolic volume.

The magnitude of left ventricular volumes is determined in part by the severity of the volume load but a further increase in volume without necessarily a further increase in ejection occurs in those patients with impaired myocardial function. Valvular insufficiency is quantitated from the difference between left ventricular ejection volume determined angiographically and forward stroke volume determined from indicator-dilution curves. The results show good agreement with subsequent surgical findings in the 15 patients who underwent operation.

**References**

12. Gribbe, P.: Comparison of the angiocardiograph-


James Mackenzie—General Practitioner and Cardiologist

Mackenzie's work and example are of the greatest value today. His researches did not depend upon the laboratory nor upon hospital facilities but upon that continuous and careful observation of patients which is only possible in general practice. There is no doubt that his work led to the recasting of our views on cardiac disease. Sir Thomas Lewis said of him: "He did more perhaps than any other man before him to place upon a rational basis forecasts of the course of heart disease in individual patients, and the treatment of heart disease by digitalis." That is high praise by one who was himself an expert in that line. But in quite another way Mackenzie deserves to be acclaimed. He is the best modern example of achievement in general practice, of high endeavour under difficult circumstances leading to results which must for ever be an encouragement to everyone engaged in that front line of the National Health Service.—ZACHARY COPE, KT. Some Famous General Practitioners and other Medical Historical Essays. London, Pitman Medical Publishing Co., Ltd., 1961, p. 26.
Myocardial Function and Left Ventricular Volumes in Acquired Valvular Insufficiency

GRAHAM A. H. MILLER, JOHN W. KIRKLIN and H. J. C. SWAN

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