Left Ventricular Performance During Muscular Exercise in Patients with and without Cardiac Dysfunction

By John Ross, Jr., M.D., James H. Gault, M.D., Dean T. Mason, M.D., Joseph W. Linhart, M.D., and Eugene Braunwald, M.D.

The relative changes in cardiac output and total body oxygen consumption (\(\dot{V}_{\text{O}_2}\)) which occur during muscular exercise provide important information concerning the overall ability of the cardiovascular system to meet the demands of this common form of stress. It is evident, however, that when a less than normal rise in the cardiac output occurs relative to a given increase in \(\dot{V}_{\text{O}_2}\), it cannot necessarily be concluded that the performance of the ventricular myocardium is impaired. Clearly, other factors such as pericardial disease, cardiac arrhythmias, or abnormal function of the cardiac valves can markedly impair the cardiac output response to exercise, even when myocardial function is quite normal.

Although a number of clinical studies have been reported in which changes in cardiac output and \(\dot{V}_{\text{O}_2}\) during exercise have been correlated with right heart dynamics, only a few investigations have made efforts to assess the performance of the left ventricular myocardium during exercise in patients with cardiac disease. Moreover, in all but one of the latter studies, pulmonary arterial diastolic or wedge pressures were utilized as indices of left ventricular end-diastolic pressure (LVEDP), and the limitations of these indirect techniques are now generally recognized. The mean ejection rate of the left ventricle has also been employed as a measure of left ventricular performance, and while this index may be useful under certain conditions, recent studies indicate that it can be influenced profoundly by changes in heart rate and stroke volume alone. It, therefore, appeared that a better definition of left ventricular performance during exercise might be achieved by determining the effects of exercise on a more direct measure of left ventricular performance, the relation between LVEDP and stroke volume or stroke work.

Abundant evidence is now available, derived both from studies in experimental animals and man, documenting the importance of the relation between the LVEDP or volume and the stroke volume or stroke work. It is also clear that the function of the failing ventricle may be characterized by an inability to increase stroke volume in spite of an increase in LVEDP or end-diastolic volume. The relation between LVEDP and stroke volume can be affected profoundly by positive inotropic influences, such as stimulation of the adrenergic nerves or circulating catecholamines, which result in the delivery of a larger stroke volume from a constant or a lower ventricular diastolic volume and pressure. The importance of these inotropic influences is evident in studies on normal experimental animals and normal human subjects, which have shown that muscular exercise often effects an increase in the stroke volume from the same or from a lower left ventricular end-diastolic volume or dimension.

In a previous study from this laboratory, performed on patients with prosthetic aortic valves, changes in the LVEDP were used to provide an index of directional alterations in left ventricular end-diastolic volume. The results of that investigation suggested that the observed patterns of left ventricular response to muscular exercise could be interpreted in terms of the interplay between the Frank-Starling mechanism, the augmented

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### Table 1

**Clinical and Hemodynamic Findings in the Patients Studied**

<table>
<thead>
<tr>
<th>Pt.</th>
<th>No.</th>
<th>Sex, age, yr.</th>
<th>B.S.A.</th>
<th>Diagnosis</th>
<th>N.Y. H.A.</th>
<th>H.R.</th>
<th>C.I.</th>
<th>BA Pressure</th>
<th>LV</th>
<th>Mean LA</th>
<th>Gradient, (LA-LV)</th>
<th>S.W.I.</th>
<th>V.}_{o}</th>
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<td>Minimal MS</td>
<td>I</td>
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<td>123/7</td>
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<th>(LV-BA)</th>
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The first line accompanying the initials of each patient (pt.) represents data at rest, the second line that during exercise. B.S.A. = body surface area in square meters; N.Y.H.A. Class. = New York Heart Association Classification; H.R., heart rate; C.I., cardiac index in L/min/m² B.S.A.; BA = brachial arterial pressure, mm Hg; LV = left ventricular systolic/end diastolic pressure, mm Hg; LA = left atrial pressure, mm Hg; gradient = the pressure gradient (mm Hg) across the mitral valve at end diastole in patients in group 2, and the peak systolic pressure gradient across the aortic valve or the left ventricular outflow tract in patients in groups 3 and 4. Mean LA pressure was not measured (—) in patients without mitral valve disease or LV outflow tract obstruction. S.W.I. = stroke work index in g-m/m² B.S.A.; \( V_{O_2} \) total body oxygen consumption. Funct. murmur = functional heart murmur.

*Patients receiving digitalis.
†Patients in atrial fibrillation.
stimulation of the heart by the adrenergic nervous system which occurs during exercise, and the functional state of the left ventricle. It was proposed that correlation of the change in the LVEDP induced by exercise, with the associated alteration in the stroke volume was a practical means of evaluating left ventricular function in man. To assess the usefulness of this technique in detecting disturbances in the performance of the left ventricular myocardium, in the present study we determined the hemodynamic responses to exercise of patients with mechanical valvular obstruction, patients with clinical evidence of myocardial disease, and subjects considered to have no significant left ventricular dysfunction.

Methods

Thirty-eight patients were studied in the post-absorptive state, following premedication with 100 mg of sodium pentobarbital. The age, sex, diagnosis, and New York Heart Association classification in each patient are shown in table 1. The patients were divided into four groups on the basis of clinical and hemodynamic findings.

Group 1 consists of seven patients considered to have normal, or almost normal, left ventricular function. Two of these patients had functional heart murmurs and three had minimal mitral stenosis with end-diastolic left atrioventricular pressure gradients at rest of 4 mm Hg or less, and normal mean left atrial pressures (<12 mm Hg) before and during exercise. The remaining two patients had atrial septal defects with pulmonary to systemic flow ratios of 2.3:1.0 and 2.4:1.0, respectively, and normal pulmonary arterial pressures. All seven subjects were asymptomatic at the time of the study; three were receiving digitalis because of previous episodes of paroxysmal arrhythmias.

Group 2 consists of 13 patients with mitral stenosis, all of whom had elevated mean left atrial pressures at rest or during exercise. They also had exertional dyspnea, and seven patients were receiving digitalis. None of these patients had associated mitral regurgitation.

Group 3 is composed of six patients with valvular aortic stenosis without associated regurgitation. In patients W.S. and K.M., the lesion was shown at subsequent operation to be congenital valvular stenosis, while in the remaining four patients it was thought to be rheumatic in origin; in patient M.K. the aortic valve was calcified. Patients E.W. and L.R. had histories of angina pectoris on exertion, although no angina occurred during the present studies. Two patients in this group were receiving digitalis.

Group 4 consists of 12 patients considered to have left ventricular myocardial disease, and in all of whom the left ventricle was enlarged radiographically. Eight patients were considered to have left ventricular myocardial disease without evidence of obstruction, while four were considered to have idiopathic hypertrophic subaortic stenosis with minimal obstruction at rest and during exercise; in the resting state, the pressure gradients across the ventricular outflow tract in these four patients ranged from 0 to 21, and during exercise the maximum gradient was 3 mm Hg. Seven of the patients in group 4 had dyspnea on exertion, four (patients D.S., E.L., M.R., and B.W.) had histories of angina pectoris, and three had had nocturnal dyspnea; five patients were receiving digitalis (table 1).

Catheterization of the left ventricle was performed either by the transseptal approach or by retrograde arterial catheterization through a brachial arteriotomy. A needle was placed into the opposite brachial artery for pressure determinations, and cardiac output was measured by the indicator-dilution technique, using indocyanine green injected into the left ventricle with brachial arterial sampling. V̇O₂ was determined by measuring ventilation with a Tissot spirometer and analyzing the expired gas with a micro-Scholander apparatus.

Left ventricular and systemic arterial pressures, V̇O₂, and the cardiac output were first measured in the resting state. The patients then pedalled a bicycle ergometer for 4 to 7 minutes in the supine position. The workload ranged from 250 to 500 ft·lb/min, depending upon the functional status of the patient. During the final 2 minutes of exercise, V̇O₂ was redetermined, and during the final minute, the measurements of left ventricular pressure, arterial pressure, and cardiac output were repeated. Pressure gradients across the mitral valve were determined by withdrawing the transseptal catheter across the valve immediately upon cessation of exercise, or by simultaneous measurement of left atrial pressure and left ventricular pressure, when the latter was measured by means of a retrograde catheter. The LVEDP was measured at the onset of isovolumic contraction in the LV pressure tracing; in the absence of a significant isoelectric segment in the initial portion of the electrocardiogram, this event occurs from 40 to 60 msec after the onset of the QRS complex.

The "exercise factor" was calculated as the in-
increase in cardiac output in ml/min per 100 ml/min increase in \( V_{o_2} \). The stroke work index of the left ventricle was calculated using the formula:

\[
\frac{SV \times (LVS - LVEDP) \times 1.36}{100 \times B.S.A.}
\]

where \( SV \) = stroke volume in milliliters, \( LVS \) = mean left ventricular pressure during ejection determined by planimetric integration and expressed in millimeters of Hg, \( LVEDP \) = left ventricular end-diastolic pressure expressed in millimeters of Hg, and B. S. A. = body surface area in square meters.

**Results**

**Group 1**

The cardiac index at rest was normal in five of the seven patients in group 1 (2.70 to 3.09 L/min/m\(^2\)), and slightly elevated in two (3.66 and 4.88 L/min/m\(^2\)). The LVEDP at rest was within normal limits (12 mm Hg or less\(^{20}\)) in all seven patients. During exercise, the cardiac indices increased appropriately for the increases in \( V_{o_2} \), as evidenced by normal exercise factors in all patients

\[
\left( \frac{600 \text{ ml/min}}{100 \text{ ml } V_{o_2}/\text{min}} \right) \text{ or greater}
\]

(fig. 1). The LVEDP fell during exercise in five patients, and increased by 2 mm Hg or less in two (fig. 1); in none of the subjects did the LVEDP exceed 12 mm Hg during exercise. The stroke work index was usually increased by exercise (five patients); it remained essentially unchanged in one patient and diminished markedly in one (table 1).

The relationships between the change in the LVEDP and the change in the stroke volume index (\( \Delta SVI \)) induced by exercise are plotted in figure 2. In four patients a slight increase or fall in LVEDP was accompanied by a rise in the SVI. In one subject both SVI and LVEDP remained almost constant, and in two a reduction in LVEDP was associated with a fall in SVI. It is of interest that the last two patients (M.R. and J.P.) exhibited high resting cardiac indices, and their SVIs at rest were elevated (60 and 70 ml/m\(^2\) B.S.A., respectively).

**Group 2**

The cardiac indices at rest were normal in seven of the patients with mitral stenosis (> 2.5 L/min/m\(^2\)), slightly reduced in three patients, and markedly reduced in three patients. The LVEDPs were normal at rest in 10 patients, and mildly elevated in three (14 mm Hg). Although the cardiac indices always increased during exercise, the exercise factors were abnormally low

\[
\left( \frac{600 \text{ ml/min}}{100 \text{ ml } V_{o_2}/\text{min}} \right) < 5
\]

in eight of the 13 patients (fig. 1). The LVEDPs fell during exercise by 1 to 5 mm Hg in seven patients, and increased by 1 or 2 mm Hg in six (fig. 1). In all three of the patients in whom the LVEDP was elevated at rest, it fell to within normal limits during exercise, and in two of these patients the exercise factors were normal. The stroke work indices declined in four patients and increased in eight (table 1).

The relationships between \( \Delta SVI \) and the \( \Delta LVEDP \) generally resembled those observed in the patients in group 1 (fig. 2). Thus, a slight increase or a fall in LVEDP was accompanied by no change or a rise in SVI in eight patients, and two patients exhibited a fall in LVEDP (1 to 2 mm Hg) and a fall in the SVI.

**Group 3**

In four of the six patients with aortic stenosis, the cardiac index was normal at rest, in one patient it was reduced, and in one it was elevated. In all of the patients, the LVEDP was elevated at rest, the values ranging from 14 to 26 mm Hg. The exercise factors were normal in two patients, and in one patient (M.K.) the exercise factor was slightly reduced (fig. 1). In three patients the exercise factors were clearly reduced (L.R., E.N., and K.M.); in one of these (K.M.) the resting cardiac index was elevated. In all six patients, the LVEDP increased during exercise, the increases ranging from 3 to 29 mm Hg, and the levels of LVEDP during exercise ranging from 18 to 44 mm Hg (fig. 1). In four patients the stroke work indices increased and in two (K.M. and L.R.) they diminished (table 1).
Figure 1

Relations between cardiac index and left ventricular end-diastolic pressure (LVED Pr.) with the patient at rest (closed circles) and during supine muscular exercise (open circles). The exercise factor for each patient is indicated adjacent to the open circles. Values given are as milliliter per minute increase in cardiac output per 100 ml/min increase in \( V_{O_2} \).

The relationship between \( \Delta \text{LVEDP} \) and \( \Delta \text{SVI} \) was abnormal in every patient in this group (fig. 2). There were three patients in whom increases in LVEDP of 4 to 10 mm were accompanied by increases in SVI. In one patient (K.M.) only a slight increase in LVEDP occurred (3 mm Hg), and this was associated with a decrease in the SVI; as mentioned, this patient had a high cardiac index and SVI at rest. Two patients exhibited marked increases in LVEDP with little change (E.N.) or a fall in the SVI (L.R.).
EXERCISE AND THE LEFT VENTRICLE

Figure 2

Relations between changes in stroke volume index (ΔSVI) and changes in left ventricular end-diastolic pressure (ΔLVED) induced by exercise in each group of patients. Each arrow points to the changes in SVI and LVED (closed circle) that occurred during exercise. The cross-hatched area encompasses the range of normal responses.

Group 4

The cardiac indices were normal at rest in 11 of the 12 patients considered to have left ventricular myocardial disease, and reduced in one patient. In 10 of the 12 patients the LVEDP was elevated at rest, values ranging from 16 to 29 mm Hg (fig. 1). During exercise, the cardiac indices rose in 11 of the 12 patients (fig. 1). The exercise factors were normal in five patients, and reduced in seven (fig. 1); in one of the latter patients (J.L.), the cardiac index actually declined during exercise. The LVEDPs increased during exercise in all patients, being augmented by 5 mm Hg or more in 10 of the 12 patients. During exercise the LVEDPs were abnormally high in all 12 patients, ranging from 15 to 45 mm Hg (fig. 1). The stroke work indices fell in seven patients and increased in five (table 1).

The relationships between ΔLVEDP and ΔSVI in this group were always abnormal and resembled the patterns observed in group 3 (fig. 2). In three patients the LVEDPs
increased abnormally, and these increases were accompanied by increases in the SVI. In eight patients abnormal increases in LVEDP were associated with little change or a fall in the SVI. In patient W.G., as with patient K.M. in group 3, the resting cardiac index (4.43 L/min/m²) and stroke volume index were high, the SVI fell with exercise, and the LVEDP rose slightly.

Discussion

The assessment of left ventricular function during exercise by the technique described herein is complicated by the changes in intrathoracic pressure which accompany the hyperventilation of exercise. It appears reasonable, however, to consider that increases in LVEDP indicate increases in left ventricular end-diastolic volume. Thus, mean intrapleural pressure tends to fall during exercise, and this influence alone would act to lower the LVEDP in relation to an external zero reference point. Since significant changes in ventricular compliance probably do not occur acutely with changes in heart rate, or with catecholamine stimulation of the heart, any increase in LVEDP occurring with exercise should reflect an increase in the left ventricular end-diastolic volume. Although some patients had a considerable degree of tachycardia with exercise (table 1), the levels were always below those found associated with acute alterations in the apparent ventricular distensibility in experimental animals. It is appreciated that accurate measurements of left ventricular end-diastolic volume before and during exercise would be desirable. However, such measurements would have required placement of an additional catheter containing a thermistor or other sensing device in the aortic root; moreover, considerable controversy exists concerning the accuracy of indicator-dilution techniques relative to the angiographic methods for determining ventricular volume.

It is well known that the level of LVEDP can be acutely influenced by the force as well as by the timing of atrial contraction. However, by altering the LVEDP, these factors also acutely influence the force of ventricular contraction and the stroke volume through the Frank-Starling mechanism. Thus, the ventricular end-diastolic pressure or volume, like the preload or resting fiber length in isolated cardiac muscle, provides a reference point from which it is possible to begin an analysis of changes in the level of contractile state of the ventricle.

Left heart catheterization is not ordinarily performed in normal individuals, and it is recognized that most of the patients in group 1 were not entirely normal. However, the existing abnormalities were mild, and their nature would not be expected to compromise left ventricular function. With these reservations, the "normal" response of the left ventricle to exercise (group 1) is characterized by a fall, no change, or a minimal increase (≤ 2 mm Hg) in the LVEDP. This response was accompanied by an increase in cardiac index, an exercise factor exceeding 600 ml/min and usually by an increase in the SVI. In addition, while the quantitative effect of a reduction in mean intrapleural pressure on LVEDP could not be determined, the minimal alteration in LVEDP and the increases in SVI which occurred in most of the subjects suggest that exercise exerted a positive inotropic influence, improvement in left ventricular performance being evidenced by a larger stroke volume, delivered from a lower or minimally changed LVEDP (fig. 2). In two of the patients without evidence of left ventricular dysfunction, both the LVEDP and the SVI fell. This finding is probably related to lack of a resting basal state in these subjects, and may be consistent with normal left ventricular function; since resting cardiac and stroke volume indices were elevated in these patients, moderate exercise induced a less than normal rise in the cardiac index, while the increase in heart rate caused a reduction in the elevated stroke volume. Gorlin and associates also noted only small changes in LVEDP during exercise in 20 patients without heart disease or with mild valvular lesions. Fifteen
of them exhibited no change or a fall in ventricular end-diastolic volume, estimated by the thermal dilution method, while five exhibited some increase in the volume.

In most of the patients in all four groups, systemic arterial pressure did not change strikingly with exercise. Alterations in the stroke work were, therefore, similar to the changes in the stroke volume (table 1). However, in patient M.R. (group 1), the systemic arterial pressure fell and the stroke work was markedly reduced during exercise, while in patient B.W. (group 4) marked hypertension during exercise was associated with an increase in the stroke work and a fall in stroke volume.

The usefulness of measuring the LVEDP in conjunction with the cardiac output and stroke volume becomes apparent when the effects of exercise on these variables are analyzed in the patients with various types of cardiac disease. It should be emphasized that patients who had regurgitant lesions of the mitral or aortic valves were not included in this study. While the exclusion of these patients places a limit on the applicability of the present method of analysis, it is apparent that since only the forward stroke volume can be measured accurately in such patients, correlations cannot be made between the total stroke volume and the LVEDP.

Although the resting cardiac indices and the exercise factors were low in the majority of the patients with mitral stenosis, the cardiac indices exhibited relatively large increases per unit increase in the LVEDP (fig. 1). The relations between ΔLVEDP and ASVI also resembled the normal pattern in most patients (fig. 2), and in the three patients in whom the LVEDP was slightly elevated at rest the directional alterations in LVEDP with exercise were normal. Although three patients exhibited a minimal rise in LVEDP pressure and a reduction in the SVI, the increases in LVEDP were small (≤2 mm Hg). It is not possible to predict to what level the LVEDP might have risen in some of these patients if mechanical obstruction had not limited the filling of the left ventricle. During this relatively mild stress, however, the level of LVEDP during exercise was always normal (fig. 1) and this together with the relationship between the ΔLVEDP and ΔSVI (fig. 2), suggests that most of these patients with pure mitral stenosis had normal left ventricular function. This conclusion supports other studies in which a different form of stress, the infusion of angiotensin, was used and in which some patients with relatively severe mitral obstruction exhibited normal responses to increasing the resistance to left ventricular ejection.19

The LVEDPs were elevated at rest and became further elevated during exercise in all of the patients with aortic stenosis (group 3). This resulted in larger increases in LVEDP per unit increase in the cardiac index than were observed in groups 1 and 2 (fig. 1), although in two patients these findings were associated with normal exercise factors and in one the exercise factor was only mildly reduced (fig. 1). In comparing the changes in LVEDP with those in the SVI (fig. 2), it is of interest that those patients who exhibited large increases in SVI had normal or near-normal exercise factors (patients W.S., E.W., and M.K.). Since LVEDP increased in these three patients, it is possible that the increase in stroke volume originated from a little changed end-diastolic volume through a positive inotropic effect, the elevated diastolic pressure reflecting chronically diminished left ventricular compliance; alternatively, it could have resulted from an increase in LVEDP volume through the Frank-Starling mechanism. Either of these interpretations would be consistent with the hypothesis that the left ventricles of these three patients met the metabolic demands of exercise, but in an abnormal manner. We have termed this type of response "abnormal left ventricular dynamics," as opposed to "depressed left ventricular function" defined below. Two of the remaining three patients in group 3 (L.R. and E.N.) exhibited large increases in LVEDP with exercise while the SVI either fell or remained unchanged. This finding strongly
suggests depression of left ventricular function. In the last patient (K.M.), the resting cardiac index was high (5.0 L/min/m²) and, as in several patients in group 2, the SVI fell with exercise; LVEDP exhibited a minimal increase, and as mentioned previously it is possible that this pattern is consistent with a minimal disturbance of left ventricular function.

In all 12 patients with left ventricular myocardial disease (group 4), a rise in LVEDP occurred during exercise (fig. 1), and the increases in LVEDP per unit increase in cardiac index were large. In five patients the exercise factors were normal; in all of these patients the resting cardiac indices were also normal, and in one the LVEDP at rest was normal. The relations between ΔLVEDP and ΔSVI (fig. 2) revealed three patients in whom increases in LVEDP were associated with increases in stroke volume. These patients may be considered to have abnormal left ventricular dynamics, and it is of interest that they, as did the patients in group 3 with similar responses, had normal or only mildly reduced exercise factors. Seven patients in group 4 exhibited increases in LVEDP with little change or actual decreases in SVI, findings consistent with depressed left ventricular function.

In the remaining two patients in group 4 the resting LVEDP and the exercise factors were abnormal, but minimal increases in the LVEDPs were accompanied by reductions in the SVI (fig. 2). In one of these patients (W.G.) the resting cardiac index and SVI were high, and as discussed previously, the fall in SVI with exercise may be explained on this basis. In the second patient (M.H.), the resting cardiac index was reduced, and the reason for the observed response remains uncertain.

The interpretations of the relations between ΔLVEDP and ΔSVI which occurred with exercise are summarized diagrammatically in figure 3. Three general patterns of response are illustrated by the shaded sectors: normal left ventricular function (quadrants I and II); abnormal left ventricular dynamics (quadrant III), reflected in an elevation of LVEDP and an increase in SVI; and depressed left ventricular function (quadrant IV), characterized by an increase in LVEDP and no change or a fall in SVI. In the areas between the shaded sectors fall those responses in which minimal increases in LVEDP, or small alterations in the SVI prohibit definitive classification.

In conclusion, the situations in which measurement of the changes in LVEDP and SVI during exercise proved to be useful may be summarized as follows: (1) in detecting normal left ventricular function in patients with mitral stenosis and abnormal resting hemodynamics; (2) in distinguishing patients with abnormal left ventricular dynamics from those with depressed left ventricular function among patients having elevated resting LVEDP; (3) in detecting abnormal left ventricular function in patients with normal resting hemodynamics.

**Summary**

The LVEDP, cardiac output, and \( \dot{V}_{O2} \) were measured before and during supine muscular

Figure 3

The patterns of left ventricular response to supine muscular exercise. Normal LV function (quadrants I and II, hatched area) includes a variable change in stroke volume, usually an increase, and a fall or no change in LVEDP. Abnormal left ventricular dynamics (quadrant III, stippled area) is associated with an increase in stroke volume index (SVI) and an increase in LVEDP. Depressed LV function (quadrant IV, hatched area) is characterized by no change or a fall in SVI and an increase in LVEDP. The areas between the shaded sectors include responses that cannot be definitively classified.
exercise in 38 patients. The normal pattern, established in seven patients without left ventricular dysfunction, consisted of an exercise factor $\frac{600 \text{ ml/min}}{100 \text{ ml } V_O_2/\text{min}}$, an LVEDP during exercise of less than 12 mm Hg, and little change or a decrease in LVEDP, which was accompanied in most instances by an increase in the stroke volume. In the majority of 31 patients with various cardiac lesions, but without valvular regurgitation, measurement of the LVEDP before and during exercise permitted the definition of normal or abnormal left ventricular function when the status of the left ventricle could not have been ascertained from other measurements. Thus, in most of the patients with mitral stenosis and abnormal resting hemodynamics, the pattern of left ventricular function during exercise was considered to be normal. Among the patients with aortic stenosis or left ventricular myocardial disease and elevated resting LVEDP, two types of abnormal performance of the left ventricle were identified. In some patients, an increase in LVEDP was accompanied by an increase in stroke volume, and this response was termed "abnormal left ventricular dynamics"; in the remaining patients, an increase in LVEDP and no change or a fall in stroke volume occurred and this response was termed "depressed left ventricular function." It is concluded that determination of the LVEDP before and during exercise adds importantly to measurements of the changes in cardiac output and $O_2$ consumption in characterizing left ventricular performance. The method employs standard catheterization techniques and appears to provide a practical and useful means for evaluating the functional status of the left ventricle in patients with and without myocardial dysfunction.

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Left Ventricular Performance During Muscular Exercise in Patients with and without Cardiac Dysfunction

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