Hemodynamic Effects of Diuresis at Rest and During Intense Upright Exercise in Patients with Impaired Cardiac Function

By Morris Stampfer, M.D., Stephen E. Epstein, M.D., G. David Beiser, M.D., and Eugene Braunwald, M.D.

SUMMARY
Although diuretic therapy appears to improve the exercise capacity of patients with moderately impaired cardiac function, the hemodynamic basis for this improvement is not clear. It is also unknown to what extent the moderate diuresis that often occurs during the first few days of hospitalization contributes to the normal or nearly normal hemodynamic measurements obtained in certain patients with cardiac impairment who are thought clinically to have signs and symptoms of pulmonary congestion. Accordingly, the circulatory response to moderate diuresis resulting in a loss of weight averaging 3.4 kg was investigated in 15 patients with heart disease. At rest in the supine position mean pulmonary arterial wedge pressure fell after diuresis from an average of 24 to 13 mm Hg. Reductions also occurred in mean pulmonary arterial pressure (42 to 26 mm Hg), mean right atrial pressure (9 to 4 mm Hg), and right ventricular end-diastolic pressure (11 to 6 mm Hg). Cardiac output decreased by an average of 20%, mean systemic arterial pressure by 12%, right ventricular stroke work by 44%, and left ventricular stroke work by 25%. Diuresis also caused similar reductions in these values in the sitting position at rest and during mild and intense levels of treadmill exercise. Despite the reductions in cardiac output, all but one of the patients studied achieved substantial clinical improvement from the diuresis. Such improvement probably resulted from the fact that the beneficial effects of lower pulmonary vascular pressures outweighed the deleterious effect of a reduction in cardiac output. Thus, moderate changes in body weight brought about by either fluid retention or fluid loss may result in substantial alterations in circulatory dynamics. These changes, if unrecognized, can lead to considerable confusion when attempts are made to correlate the hemodynamic findings with the degree of cardiac decompensation as judged clinically.

Additional Indexing Words: Congestive heart failure Heart function tests Cardiac output Cardiovascular system Exertion Heart catheterization Exercise test

With the advent of more potent oral diuretics, it has become almost routine to employ these drugs in the management of patients with heart failure not only during hospitalization or intermittently during an episode of relatively acute decompensation, but also as a chronic form of therapy. Despite the widespread use of this form of treatment, there is little information concerning its effect on the circulation. The only measurements available concerning the hemodynamic effects of diuresis were made on patients suffering from severe cardiac decompensation1-3 and have been carried out in the supine position with the patient at rest. Because the effects of oral diuretic therapy on patients with only mild to moderate degrees of cardiac decompensation have not been defined, the hemodynamic response to modest diuresis was investigated in a group of such patients.

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EFFECTS OF DIURESIS AT REST AND EXERCISE

Table 1

Description of Patients

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age (yr)</th>
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<th>Class</th>
<th>Rhythm</th>
<th>Diagnosis</th>
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<td>AF</td>
<td>MS</td>
<td>54.3</td>
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Abbreviations: BSA = body surface area in m²; weight, wet and dry = body weight before and after diuresis; AF = atrial fibrillation; NSR = normal sinus rhythm; MS = mitral stenosis; MI = mitral regurgitation; AI = aortic regurgitation; TI = tricuspid regurgitation; CM = cardiomyopathy; AS = aortic stenosis.

Patients with primary myocardial and rheumatic valvular disease at rest, and during mild and intense treadmill exercise.

Methods

Six men and nine women, aged 32 to 58 years, were studied. Fourteen had edema secondary to rheumatic valvular disease; one had primary myocardial disease. All were ambulatory and in functional classes II and III (New York Heart Association). The patients are described further in table 1.

In nine patients the first hemodynamic study was performed prior to diuresis, several days after admission to the hospital (wet weight). Dietary sodium restriction and maintenance doses of chlorothiazide or hydrochlorothiazide with potassium chloride supplementation were begun subsequent to catheterization, and occasionally, a mercurial diuretic was administered parenterally. After body weight had stabilized (dry weight), the second hemodynamic study was performed. In the remaining six patients the order of study was reversed; that is, the first catheterization was performed following diuresis. Subsequently, use of diuretics was discontinued and sodium intake was liberalized. When the patient's weight was at or near the admission weight, catheterization was repeated. If digitalis was being given, the same dosage was continued throughout the period of study. Serum electrolytes were measured frequently to ensure that no abnormalities developed. The interval between the two catheterization studies ranged from 4 to 20 days and averaged 11 days. The difference in body weight at the time of the two studies ranged from 0.7 to 5.4 and averaged 3.4 kg.

The effects of diuresis on hemodynamic findings in the supine position at rest were studied in 12 patients. In six of these patients and in three additional patients, the hemodynamic response to upright exercise was also evaluated. Pulmonary arterial and right heart pressures were measured through a Courmand catheter, and brachial arterial pressure through an indwelling needle or a short flexible Teflon catheter, which was introduced percutaneously into the brachial artery. Central venous pressure was measured through a small polyethylene catheter inserted percutaneously and advanced to the superior vena cava, or when the pulmonary arterial catheter was withdrawn to the right atrium or superior vena cava. Intravascular pressures were measured with Statham transducers, with zero reference at the midthorax in the supine position and the midright atrium in the upright position. The electrocardiogram was recorded continuously.

Cardiac output in the supine position was determined by the indicator-dilution technique with injection of indocyanine-green dye into the pulmonary artery and sampling from the brachial artery. In subjects with significant valvular insufficiency, cardiac output was determined by the Fick principle; mixed venous blood was sampled from the pulmonary artery and arterial blood from the brachial artery. Arterial and mixed venous O₂ content were measured by the method.
of Van Slyke and Neill, and CO₂ and O₂ content of expired air were determined by the micro-Scholander technique. In the studies carried out in the upright position cardiac output was determined by the Fick principle; VO₂ was measured with a continuous flow system and a paramagnetic oxygen analyzer.

Right ventricular stroke work (RVSW), in gram-meters, was calculated as the product of stroke volume (ml) and mean pulmonary arterial pressure (cm H₂O) divided by 100. Left ventricular stroke work (LVSW), in gram-meters, was calculated as the product of stroke volume (ml) and mean systemic arterial pressure (cm H₂O) divided by 100. Total peripheral vascular resistance (TPR) was calculated as the product of 1,332 and the difference between mean arterial and central venous pressures (mm Hg), divided by cardiac output (ml/sec).

The protocol for the studies carried out in the upright position was as follows: While the patient was at rest in the sitting position, intravascular pressures and VO₂ were measured and arterial and mixed venous blood withdrawn for determination of cardiac output. The patient then walked on a motor-driven treadmill at a moderate level of exertion for 5 min. Exercise was then made more intense by increasing the speed or the grade of the treadmill, or both, to a level which had been determined in previous trials to produce exhaustion in 5% to 6 min. Pressure measurements were repeated during the fourth minute of each level of exercise, and cardiac output was determined during the fifth minute. In some patients only a single level of exercise, which was a maximal effort, was possible. In each subject the second study employed the same conditions of exercise as the first.

At a separate session during which no intravascular instrumentation was employed, maximal exercise endurance was measured before and after diuresis with a standard treadmill stress test. Treadmill speed was maintained at 2.2 mph, while the grade was increased every 2.5 min to a maximum of 20%. The patient continued walking until stopped by dyspnea or fatigue; the time at which he indicated he could no longer continue was recorded and the duration of exercise is referred to as "maximal exercise endurance." The same conditions for assessing maximal exercise endurance were used before and after diuresis.

Results

Supine Rest

Mean pulmonary arterial wedge pressure was abnormally elevated in all patients prior to diuresis, ranging from 19 to 32 mm Hg (fig. 1A, table 2). The average value fell significantly after diuresis from 24 to 13 mm Hg (P < 0.001), and was within the upper limits of normal for this laboratory (12 mm Hg) in five of the eight patients in whom this measurement was made. In one subject a mean wedge pressure of 26 mm Hg before diuresis fell to 10 mm Hg after diuresis. All patients had mean pulmonary arterial pressures above the upper limit of normal for this laboratory (20 mm Hg) prior to diuresis (fig. 1B, table 2). Following diuresis, mean

Figure 1
Effects of diuresis on mean pulmonary arterial wedge pressure (A), mean pulmonary arterial pressure (B), right ventricular end-diastolic pressure (C), and mean right atrial pressure (D), measured at rest in the supine position. Values before diuresis are shown by closed circles and after diuresis by open circles. The broken lines represent the upper limits of normal values for this laboratory. Group averages before and after diuresis are shown by open circles on a horizontal line.
Table 2

Hemodynamic Alterations at Supine Rest Produced by Diuresis

<table>
<thead>
<tr>
<th>Subject</th>
<th>Wt (kg)</th>
<th>RAP (mm Hg)</th>
<th>RVEDP (mm Hg)</th>
<th>PAP (mm Hg)</th>
<th>PAWP (mm Hg)</th>
<th>HR (beats/min)</th>
<th>CO (L/min)</th>
<th>MAP (mm Hg)</th>
<th>TPR (dynes-sec cm⁻¹)</th>
<th>RVSW (g·m⁻¹)</th>
<th>LVSW (g·m⁻¹)</th>
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<td>—</td>
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<td>—</td>
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<tr>
<td></td>
<td>Dry</td>
<td>67.8</td>
<td>—</td>
<td>18</td>
<td>10</td>
<td>57</td>
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Mean — Wet

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| Mean—Dry

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Abbreviations: Wt = body weight; RAP = mean right atrial pressure; RVEDP = right ventricular end-diastolic pressure; PAWP = mean pulmonary arterial wedge pressure; HR = heart rate; CO = cardiac output; MAP = mean brachial arterial pressure; TPR = total peripheral resistance; RVSW = right ventricular stroke work; LVSW = left ventricular stroke work; Wet = before diuresis; Dry = after diuresis; ± sd = standard deviation; P = probability that difference between measurements before and after diuresis is due to chance alone; NS = not significant (P > 0.05); — = not measured.
pulmonary arterial pressure decreased from an average of 41 mm Hg to 26 mm Hg ($P < 0.001$), and in six of the 12 patients the pressure fell to a value within the limits of normal. In one patient the pressure fell from 52 to 18 mm Hg and in another from 45 to 22 mm Hg. Right ventricular end-diastolic pressure (RVEDP) was elevated in each patient prior to diuresis and fell to values within the limits of normal ($\leq 5$ mm Hg) in six of the 10 patients after diuresis (fig. 1C, table 2); the mean value decreased from 11 to 6 mm Hg ($P < 0.001$). Ten of 11 patients had an elevated mean right atrial pressure (>5 mm Hg) prior to diuresis, and the pressure in the remaining patient was at the upper limit of normal (fig. 1D, table 2). After diuresis mean right atrial pressure fell from an average of 9 to 4 mm Hg ($P < 0.001$) and at this time the values in all except two patients were within the normal range. Cardiac output decreased from an average of 4.51 to 3.61 L/min (fig. 2A, $P < 0.01$), mean arterial pressure from an average of 98 to 86 mm Hg (fig. 2B, $P < 0.025$), RVS\(p\) by 44\% (36 to 20 g-m) (fig. 2C, $P < 0.01$), and LVSW by 25\% (79 to 59 g-m) (fig. 2D, $P < 0.01$; table 2). TPR tended to be higher after diuresis in each patient in whom it could be calculated (from an average of 1,546 to 1,974 dyne-sec cm$^{-5}$). Heart rate showed no consistent changes (table 2).

**Sitting Rest**

The circulatory changes induced by diuresis were directionally similar when the patients...
Table 3

Effects of Diuresis on Hemodynamic Response to Exercise

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<th>CO (L/min)</th>
<th>HR (beats/min)</th>
<th>( \text{VO}_2 ) (ml/min)</th>
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<th>MAP (mm Hg)</th>
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<td>D</td>
<td>W</td>
<td>D</td>
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<td></td>
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Rest—Mean

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sp 0.66 | 0.62 | 17 | 27 | 33 | 36 | 10 | 5 | 14 | 10

Mild exercise—

Mean 5.95 | 5.06 | 123 | 118 | 660 | 623.5 | 55 | 34 | 91 | 80

sp 1.09 | 1.20 | 29 | 30 | 86 | 127 | 20 | 14 | 18 | 11

Intense exercise

—Mean 7.91† | 6.89 | 157 | 144 | 955 | 953 | 59 | 40 | 96 | 87

sp 2.14 | 2.05 | 28 | 26 | 292 | 367 | 24 | 17 | 16 | 14

P < 0.005 | < 0.05 | NS | < 0.005 | < 0.05 | < 0.001 | < 0.05
### Table 3 (continued)

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<th>CVP (mm Hg)</th>
<th>RVSW (g-m)</th>
<th>LVSW (g-m)</th>
<th>TPR (dyne-sec cm⁻²)</th>
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Rest—Mean: 7, 1; 23.4, 8.8*; 76.5, 41.0*; 1625, 2269*; 11.6, 13.5
sd: 5, 1; 9.2, 2.7; 30.3, 11.6; 218, 432; 1.3, 1.4

Mild exercise—Mean:
- Mean: 9, 2; 36.8, 20.5; 62.8, 48.3; 1282, 1491; 11.8, 13.9
- sp: 4, 3; 11.5, 9.4; 21.0, 15.1; 385, 297; 1.7, 1.8

Intense exercise—Mean:
- Mean: 10, 2; 36.8†, 23.3; 67.5†, 56.9; 1021, 1157; 12.5, 14.5
- sp: 4, 1; 12.6, 10.4; 19.2, 16.2; 292, 249; 1.4, 1.5

Abbreviations: CO = cardiac output; HR = heart rate; VO₂ = oxygen consumption; PAP = mean pulmonary arterial pressure; MAP = mean brachial arterial pressure; CVP = mean central venous pressure; RVSW = right ventricular stroke work; LVSW = left ventricular stroke work; TPR = total peripheral resistance; Hgb = hemoglobin; W = before diuresis; D = after diuresis; R = rest; E₁ = mild exercise; E₂ = intense exercise; sp = standard deviation; P = probability that difference between measurements before and after diuresis is due to chance alone; NS = not significant (P > 0.05); — = not measured.

* = excluding H.K.
† = excluding G.T.
EFFECTS OF DIURESIS AT REST AND EXERCISE

were studied at rest in the sitting position. Thus, mean pulmonary arterial pressure fell from an average of 29 to 18 mm Hg ($P < 0.005$), central venous pressure from 7 to 1 mm Hg ($P < 0.05$), cardiac output from 4.18 to 3.02 L/min ($P < 0.001$), and mean arterial pressure from 94 to 82 mm Hg. RVSW fell 62% from 23.4 to 8.8 g-m ($P < 0.001$), and LVSW 46% from 76.5 to 41.0 g-m ($P < 0.01$). $\text{V}O_2$ averaged 250 ml/min before and 225 ml/min after diuresis ($P < 0.005$). Heart rate was not significantly changed, and TPR increased from an average of 1,025 to 2,269 dynes-sec cm$^{-5}$ ($P < 0.005$). Arterial hemoglobin concentration increased significantly after diuresis from an average of 11.6 to 13.5 g/100 ml (table 3).

Treadmill Exercise

Despite subjective and objective evidence of clinical improvement after diuresis in eight of the nine patients, cardiac output fell in each. The mean value decreased from 5.95 to 5.06 L/min ($P < 0.05$) during mild exercise, and from 7.91 to 6.89 ($P < 0.005$) during intense exercise (figs. 3A and 5A, table 3). Mean pulmonary arterial pressure fell from an average of 55 to 34 mm Hg ($P < 0.005$) and from 59 to 40 mm Hg ($P < 0.001$) during mild and intense exercise, respectively (figs. 3B and 5B, table 3). Similarly, mean central venous pressure fell from an average of 9 to 2 mm Hg during mild exercise ($P < 0.02$) and from 10 to 2 mm Hg during intense exercise ($P < 0.005$) (fig. 3C, table 3). Significant reductions in both right and left ventricular stroke work were also observed after diuresis. RVSW fell by an average of 44% from 36.8 to 20.5 g-m during mild exercise ($P < 0.005$), and 37% from 36.8 to 23.3 g-m ($P < 0.001$) during intense exercise (fig. 3D, table 3). LVSW fell by an average of 23% during mild exercise from 62.8 to 48.3 g-m ($P > 0.05$), and 16% during intense exercise from 67.5 to 56.9 g-m ($P < 0.02$, table 3). Mean systemic arterial pressure decreased from an average of 91 to 80 mm Hg ($P < 0.05$) during mild exercise and from 96 to 87 mm Hg during intense exercise ($P < 0.05$, table 3). Heart rate was not changed significantly by diuresis during mild exercise, but during intense exercise it decreased from an average of 157 to 144 ($P < 0.05$, table 3). No significant changes in TPR or $\text{V}O_2$ occurred after diuresis either during mild or intense exercise. Following diuresis, the arterial hemoglobin concentration during intense exercise increased significantly from an average of 12.5 to 14.5 g ($P < 0.001$).

Maximal Exercise Endurance

In five of the six patients for whom it was determined, maximal exercise endurance was substantially greater after diuresis (fig. 4). The three subjects who did not undergo the

\begin{figure}[h]
\centering
\includegraphics[width=0.5\textwidth]{maximal_exercise_endurance.png}
\caption{Maximal exercise endurance time before diuresis (closed circles) and after diuresis (open circles).}
\end{figure}
standard endurance test also improved with diuresis as manifest by their ability to achieve higher levels of treadmill exercise during the hemodynamic study. All eight patients also felt stronger subjectively, and had less orthopnea and exertional dyspnea. The ninth subject, T. W., who had moderately severe mitral regurgitation, was atypical in that his exercise tolerance was considerably lower after diuresis than before. After the first study on him, which was carried out after diuresis, diuretic therapy was discontinued, and unlimited salt intake was permitted. Following a weight gain of 3.3 kg his maximal exercise test showed marked improvement. Diuretics and salt restriction were reinstituted. Deterioration in endurance followed diuresis, and the endurance times for this third study were similar to those obtained during the first study (fig. 5C). Serum electrolytes remained normal throughout these studies.

**Discussion**

Although John C. Warren stated over 150 years ago that “blood-letting affords more speedy and compleat relief (from the symptoms of heart failure) than any other remedy,” little was known regarding the mechanisms responsible for this clinical improvement until the 1940’s when cardiac catheterization began to be used commonly in the evaluation of patients with heart disease. At that time and in subsequent years it was demonstrated that interventions which decreased filling pressure in patients with severe cardiac decompensation led to a reduction in resting pulmonary arterial pressures, and in many but not all patients, resting cardiac output increased. These findings suggested that the hearts of such patients may have been functioning on the descending limb of the Starling curve.

From these results it might be anticipated that the increased exercise tolerance produced by diuretic therapy might be related, at least in part, to an increase in the cardiac output. However, in this investigation the patients studied at rest in the supine and sitting positions responded to a reduction in
filling pressure produced by a relatively modest diuresis by a decrease rather than increase in cardiac output. In addition, the cardiac output during exercise was also lower in every patient following diuresis. It would thus seem that in patients with mild to moderate cardiac decompensation, as in normal subjects, the heart functions in the ascending limb of the Starling curve both at supine and sitting rest and during mild and intense exercise. The measurements in this study, however, were made over the relatively short interval of several weeks. It is conceivable that the impressive decrease in filling pressures and in right and left ventricular stroke work may, over a longer period of time, have a beneficial effect on the contractile state of the heart and result in a significant improvement in the cardiac output response to exercise.

Although it seems reasonable to attribute the decrease in cardiac output following diuresis to reductions in cardiac filling pressures, other causes must be considered. The observation that VO₂ was unchanged after diuresis at comparable levels of mild and intense exercise demonstrates that the decrease in output cannot be attributed to a decrease in metabolic demands. It might also be argued that since hemoconcentration occurs as a result of diuresis (table 3), the greater O₂-carrying capacity of the blood would enable the same VO₂ to be achieved at a lower cardiac output. Thus, the decrease in cardiac output would not necessarily be secondary to a decrease in filling pressure, but rather to an increase in the arterial O₂ capacity. While this argument might apply to the changes in cardiac output that occurred at rest or during mild levels of exertion, it would not explain the decrease in output that occurred during maximal exercise. At this level of exertion the O₂ demands of exercise exceed the capacity of the body to take up O₂, a situation which results in the progressive accumulation of an O₂ debt. That cardiac output fell after diuresis despite the fact that the level of output was inadequate to meet the metabolic requirements suggests that the increase in arterial O₂-carrying capacity was not primarily responsible for the decreased output.

An interesting finding in this investigation was that exercise capacity improves strikingly after diuresis despite a decrease in the cardiac output in response to exercise. The explanation of this paradox probably lies in the fact that the fall in cardiac output is associated with a lower pulmonary arterial, and presumably lower pulmonary venous pressure. It is this decrease in an excessively high pulmonary venous pressure present during exercise that is probably the critical factor responsible for the symptomatic improvement, overriding the deleterious effects of a reduced cardiac output.

However, that this is not always the case, and that a decrease in the capacity to perform exercise can occur after diuresis, is illustrated by the findings in one patient who was studied. This patient (T. W.) was referred with a diagnosis of mitral regurgitation for consideration for mitral valve replacement. His exercise tolerance was severely restricted despite salt restriction and diuretic treatment, and hemodynamic studies during treadmill exercise revealed a severely depressed cardiac output response (fig. 5A). Despite the marked impairment of cardiac function, however, his central venous and pulmonary arterial pressures were within normal limits (fig. 5B, table 3). It was suspected that he might have been relatively hypovolemic and that the depression of cardiac output was due, in part, to an inadequate filling pressure. Administration of diuretics was therefore discontinued; the patient received a high sodium diet; and after a weight gain of 3.3 kg he was restudied. During the second study pulmonary arterial and central venous pressures were slightly increased at rest and during exercise, and the cardiac output response to exercise approached the normal range, a marked increase from his previous performance. In addition, his symptoms and the standard maximal endurance showed considerable improvement (fig. 5C). To exclude the possibility that this improvement was due to pro-
longed rest in the hospital, sodium restriction and diuretic therapy were reinstituted. After a loss of 3.6 kg, the patient was restudied. Cardiac output and mean pulmonary arterial and central venous pressures were decreased to levels similar to those observed at the first study, and performance during the endurance test fell (fig. 5).

The results in this patient demonstrate that occasionally the disadvantages of a decrease in cardiac output following diuresis may outweigh the advantages of a decrease in pulmonary vascular pressures, resulting in a net decrease in the patient's exercise capacity. The frequency of this latter occurrence is presently unknown, but it suggests that the efficacy of a potent diuretic regimen must be carefully evaluated for each individual patient.

In addition, the results of this investigation indicate that the pressure and flow measurements obtained during hemodynamic studies can be markedly altered by relatively small changes in body weight brought about by either fluid retention or fluid loss. Thus, while pulmonary vascular and right heart pressures were lower in each patient following diuresis, in five of eight patients the moderately to severely elevated pulmonary arterial wedge pressures present in the supine position actually fell to values within the normal range. A similar decrease from abnormal to normal values occurred in mean pulmonary arterial pressure in six of 12 patients, in right ventricular end-diastolic pressure in six of 10 patients, and in mean right atrial pressure in nine of 11 patients.

The significance of this observation centers on the fact that the results of catheterization often are among the more important factors used in the assessment of patients with reduced cardiac reserve. For example, while the clinical evaluation of patients with mitral valve disease is usually the most important consideration in determining whether or not operative correction is recommended, the finding of normal or only slightly elevated pulmonary wedge or left atrial pressures often may cause considerable confusion concerning the mechanisms responsible for the production of the symptoms, and therefore uncertainty as to whether correction of the anatomic abnormality will result in symptomatic improvement. Such uncertainty is not uncommon, and much discussion in the literature revolves around this problem. For example, it has been suggested that patients with mitral stenosis but with normal or near normal resting pulmonary pressures should not be operated upon since, if their pressures are already normal, operative correction of the mechanical obstruction would produce neither hemodynamic nor symptomatic improvement. The results of this study suggest that in at least some of these patients, the explanation for the relatively low left atrial and pulmonary vascular pressures may be related to diuresis that frequently occurs during the first few days of hospitalization. If the patient were studied at the body weight present prior to this diuresis, appreciable elevations in these pressures might have been observed.

Thus, in patients who are severely limited by symptoms indicative of pulmonary venous hypertension, and whose valvular lesions are amenable to surgical repair, the finding of low pulmonary vascular pressures could lead to either of two courses of action. It could serve as a stimulus to tighten the patient's medical regimen further, especially in relation to diuretic therapy and dietary salt restriction in the hope of maintaining the low pulmonary vascular pressures after discharge from the hospital. However, while the reduction of pulmonary vascular and left atrial pressures by diuresis is desirable insofar as it usually decreases the incidence of symptoms attributable to pulmonary venous hypertension, an actual deterioration in exercise capacity may be produced by diuresis (fig. 5). If the measures employed to lower filling pressure have such deleterious effects, the second course of action, operative intervention, should be considered despite the presence of normal pulmonary arterial wedge or left atrial pressures. Such a decision would be further strengthened.
by the finding that pulmonary vascular pressures increase markedly following small increments in body weight produced by fluid accumulation, a condition that may exist outside the hospital and which would thus be responsible for many of the patient's symptoms.

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References

Hemodynamic Effects of Diuresis at Rest and During Intense Upright Exercise in Patients with Impaired Cardiac Function
MORRIS STAMPFER, STEPHEN E. EPSTEIN, G. DAVID BEISER and EUGENE BRAUNWALD

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