# The Limited Reliability of Physical Signs for Estimating Hemodynamics in Chronic Heart Failure

Lynne Warner Stevenson, MD, Joseph K. Perloff, MD

The cardiovascular physical examination is used commonly as a basis for diagnosis and therapy in chronic heart failure, although the relationship between physical signs, increased ventricular filling pressure, and decreased cardiac output has not been established for this population. We prospectively compared physical signs with hemodynamic measurements in 50 patients with known chronic heart failure (ejection fraction, .18 ± .06). Rales, edema, and elevated mean jugular venous pressure were absent in 18 of 43 patients with pulmonary capillary wedge pressures greater than or equal to 22 mm Hg, for which the combination of these signs had 58% sensitivity and 100% specificity. Proportional pulse pressure correlated well with cardiac index ($r = .82$), and when less than 25% pulse pressure had 91% sensitivity and 83% specificity for a cardiac index less than 2.2 L/min/m$^2$. In chronic heart failure, reliance on physical signs for elevated ventricular filling pressure might result in inadequate therapy. Conversely, the adequacy of cardiac output is assessed reliably by pulse pressure. Our results facilitate decisions regarding treatment in chronic heart failure.

(*JAMA* 1989;261:884-888)

TIME-HONORED cardiovascular physical signs of congestive heart failure include a third heart sound, pulmonary rales, an abnormal jugular venous pulse (height and wave form), and peripheral edema.[1,2] These criteria, together with the history and chest roentgenogram, have been useful in identifying patients with ejection fractions of less than 40% to 50%.[3-8] Patients in whom the diagnosis of heart failure has been established require constant vigilance to identify hemodynamic deterioration that necessitates adjustment of therapy. While the relationship between physical signs and hemodynamic profile has a firm basis for *acute* congestive heart failure,[9] the *chronic* state is characterized by a host of compensatory mechanisms that may cause disparities, such as the absence of rales and peripheral edema despite symptomatic elevation of ventricular filling pressures. Because the accuracy of physical signs for the identification of elevated filling pressure and depressed cardiac output has not been established in patients with chronically depressed left ventricular systolic function, we undertook a prospective study to correlate physical signs with hemodynamic data in 50 consecutive patients so diagnosed.

From the Division of Cardiology, Department of Medicine, UCLA Medical Center, Los Angeles.

Reprints requests to UCLA Medical Center, 10833 Le Conte Ave, Rm 47-123 CHS, Los Angeles, CA 90024 (Dr Stevenson).

## PATIENTS AND METHODS

All 50 patients had secure diagnoses of heart failure at the time of elective hemodynamic measurements, which were performed as part of an evaluation for cardiac transplantation (39 patients) or an assessment of the adequacy of medical regimens (11 patients). Thirty-seven patients were men, aged 47 ± 15 years; 13 were women, aged 39 ± 13 years. Only patients with heart failure accompanied by ventricular dilation and ejection fractions of less than or equal to .30 (systolic dysfunction) were included to eliminate those with circulatory congestion arising from primary restriction of diastolic filling. Mean ejection fraction was .18 ± .06. Left ventricular dysfunction resulted from nonischemic dilated cardiomyopathy in 36 patients and from ischemic heart disease in 14 patients. The duration of congestive symptoms ranged from three to 100 months (median, 19 months). Medications being taken at the time of study included digoxin by 42 patients, furosemide by 42 patients, vasodilators by 28 patients, and milrinone by two patients.

Physical examinations were performed in the fluoroscopy suite immediately before right-sided heart catheterization. Blood pressure was determined with a sphygmomanometer and a

stethoscope applied to the right or left brachial artery. Care was taken to prevent prolonged cuff inflation to avoid venous congestion and a spurious decrease in pulse pressure.[2,3] The diastolic pressure was taken as the level at which Korotkoff's sounds disappeared. The proportional pulse pressure was defined as follows:

$$\frac{\text{systolic pressure} - \text{diastolic pressure}}{\text{systolic pressure}}$$

Pulmonary rales were graded from 0 to 4. A score of 1 indicates rales over one quarter of the posterior lung fields and 4 indicates rales over the entire posterior lung fields. Central venous pressure was assessed from the internal and external jugular veins with the patient at 30° to 45°. Central venous pressure elevation was graded as 0 if the crests of neither the internal nor the external jugular veins were visible above the clavicle in this position and as 4 if the crests were visible at the earlobe in this position and in the full upright position. Peripheral edema was graded as 0 to 4 according to the depth of indentation at the ankle. Third heart sounds were sought by identifying the left ventricular impulse in the left lateral decubitus position and applying the bell of the stethoscope just lightly enough for a skin seal.[2] Orthopnea (during the previous week) was graded as 0 to 4, with 0 indicating no need for more than one pillow on a flat bed and 4 indicating at least one night spent sleeping in a sitting position.

Radiographic information may contribute to the diagnosis of low ejection fraction and heart failure,[3-8] but chest roentgenograms were not included because this study was designed to determine the reliability of physical signs for assessment of hemodynamic status without recourse to other diagnostic procedures.

A #7 F balloon-tipped thermodilution pulmonary artery catheter was inserted percutaneously into the right internal jugular vein or the right antecubital vein, the latter when a sufficiently supine position was not tolerated. The position of the catheter tip was confirmed by fluoroscopy, and pressures were measured in the right atrium, pulmonary trunk, and pulmonary capillary wedge positions. The right atrial and wedge pressures were recorded on strip charts in the respiratory midposition. Thermodilution cardiac outputs were determined in triplicate with iced injectate.

To establish the maximum sensitivity of the physical signs, all scores greater than zero were considered positive. Pulmonary capillary wedge pressure greater than 20 mm Hg and cardiac index less than 2.2 L/min/m$^2$ were chosen as conservative estimates of hemodynamic abnormalities warranting therapy. Because no patient had a pulmonary capillary wedge pressure in the range of 19 to 21 mm Hg, the categories were divided into greater than or equal to 22 mm Hg and less than or equal to 18 mm Hg. The correlations between hemodynamics and clinical measurements were assessed with linear regression and in $2 \times 2$ tables, for which significance was determined by two-tailed Fisher's exact test.

## RESULTS

The age, etiology of left ventricular failure, and physical signs for the 50 patients are shown in Table 1. Rales were present in eight patients, elevations in mean jugular venous pressures were present in 25 patients, and peripheral edema was present in ten patients; third heart sounds were present in 48 patients.

Pulmonary capillary wedge pressures ranged from 8 to 44 mm Hg (Table 2) and were greater than or equal to 35 mm Hg in 18 (36%) of 50 patients, greater than or equal to 25 mm Hg in 38 patients (76%), and greater than or equal to 22 mm Hg in 43 patients (86%). The remaining seven wedge pressures were 8 to 18 mm Hg. The eight patients with rales all had pulmonary wedge pressures greater than or equal to 22 mm Hg. The presence of rales, however, identified only 19% of patients with pulmonary wedge pressures greater than or equal to 22 mm Hg and 11% of patients with pulmonary wedge pressures greater than or equal to 35 mm Hg. Third heart sounds were present in all but two patients (wedge pressures, 8 and 26 mm Hg) and therefore were not specific for identifying high filling pressures in our study group.

Right atrial pressures were greater than or equal to 10 mm Hg in 28 patients, all of whom had pulmonary wedge pressures greater than or equal to 22 mm Hg. Elevated right-sided filling pressures could be diagnosed from either the jugular venous pressure or from edema or from both in 21 (75%) of 28 patients with right atrial pressures greater than or equal to 10 mm Hg. All ten patients with edema also had abnormally high jugular venous pressures.

Because elevated left ventricular filling pressures were less evident clinically than the frequently coexisting elevations of right ventricular filling pressures, the reliability of the physical examination for detection of pulmonary capillary wedge pressure greater than or equal to 22 mm Hg was assessed by using jugular venous pressure and edema as criteria in addition to pulmonary rales (Table 3). Elevated jugular venous pressure was the most sensitive criterion and was present in every patient who had other physical signs of congestion. However, even using all these criteria, elevated filling pressures could not be diagnosed in 18 (42%) of 43 patients with pulmonary capillary wedge pressures greater than or equal to 22 mm Hg. Physical evidence specific for congestion was absent in eight (44%) of 18 patients with pulmonary capillary wedge pressures greater than or equal to 35 mm Hg.

To assess tolerance for the supine position during catheterization, all patients were questioned regarding orthopnea. Orthopnea within the preceding week was reported by 39 (91%) of 43 patients with pulmonary wedge pressures greater than or equal to 22 mm Hg. Recent orthopnea was absent in four patients with pulmonary wedge pressures greater than or equal to 22 mm Hg and in all seven patients with lower pressures.

The proportional pulse pressure correlated well with cardiac index ($r = .82$, $P < .001$) (Figure), with stroke volume

Table 1.—Clinical Characteristics and Physical Findings in 50 Patients With Chronic Heart Failure

| Characteristic/Finding | Present | Absent |
|---|---|---|
| Coronary artery disease | 16 | 34 |
| Male gender | 37 | 13 |
| Rales | 8 | 42 |
| Third heart sound | 48 | 2 |
| Increased jugular venous pressure | 25 | 25 |
| Peripheral edema | 10 | 40 |
| Orthopnea | 39 | 11 |

Table 2.—Hemodynamic Measurements in 50 Patients With Chronic Heart Failure

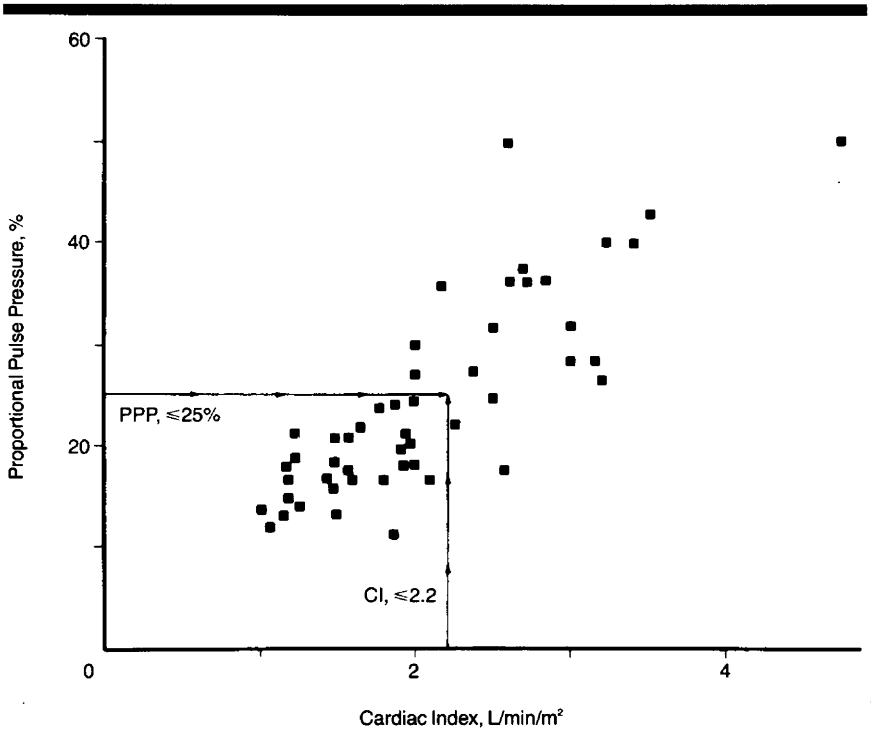
| Hemodynamic Measurement | Mean ± SD | Range |
|---|---|---|
| Ejection fraction | .18 ± .06 | .08-.29 |
| Heart rate, beats per min | 98 ± 18 | 64-130 |
| Systolic blood pressure, mm Hg | 109 ± 18 | 80-150 |
| Diastolic blood pressure, mm Hg | 82 ± 11 | 50-100 |
| Mean arterial pressure, mm Hg | 91 ± 11 | 60-113 |
| Right atrial pressure, mm Hg | 12 ± 7 | 2-38 |
| Pulmonary wedge pressure, mm Hg | 30 ± 9 | 8-44 |
| Cardiac index, L/min/m$^2$ | 2.1 ± 0.8 | 1.0-4.7 |
| Stroke volume index, cc/m$^2$ | 22 ± 9 | 10-43 |
| Systemic vascular resistance, dynes-sec-cm$^{-5}$ | 1900 ± 800 | 760-4500 |

Table 3.—Relation Between Physical Examination and Elevated Pulmonary Capillary Wedge Pressure

| Pulmonary Capillary Wedge Pressure, mm Hg | Physical Examination | |
|---|---|---|
| | Positive* | Negative |
| ≥22† | 25 | 18 |
| ≤18 | 0 | 7 |
| | P<.01 | |

*Evidence of elevated filling pressures with pulmonary capillary wedge pressure greater than or equal to 22 mm Hg included rales alone, no patients; rales and elevated venous pressure, two patients; rales, elevated venous pressure, and peripheral edema, six patients; elevated venous pressure and edema, four patients; elevated venous pressure alone, 13 patients; and edema alone, no patients.

†The criterion of 22 mm Hg was chosen because five patients had pulmonary capillary wedge pressures ranging from 22 to 24 mm Hg and the next highest value was 18 mm Hg.




Relationship between cardiac index (CI) and proportional pulse pressure (PPP) in 50 patients with chronic heart failure.

index ($r=.78$, $P<.001$), and with the inverse of systemic vascular resistance ($r=.65$, $P<.001$). There was poor correlation, however, between cardiac index or stroke volume index and systolic pressure ($r=.36$ and $r=.45$, respectively) or mean arterial pressure ($r=.05$ and $r=.08$, respectively). A proportional pulse pressure less than or equal to 25% identified 29 (91%) of 32 patients with cardiac indexes less than 2.2 L/min/m² (Table 4). The proportional pulse pressure was greater than 25% in 15 (83%) of 18 patients with cardiac indexes greater than 2.2 L/min/m². If a cardiac index of less than or equal to 2.0 L/min/m² was selected, the results were 93% and 80%. The use of proportional pulse pressure yielded an 88% predictive accuracy for low cardiac output.

Linear regression showed no correlation between the amount of pulmonary capillary wedge pressure elevation and the amount of cardiac index reduction ($r=.22$, not significant). However, the seven patients with pulmonary capillary wedge pressures less than or equal to 18 mm Hg all had cardiac indexes greater than 2.2 L/min/m² (Table 5), as did 11 (26%) of 43 patients with high pulmonary capillary wedge pressures. Right atrial pressure correlated with cardiac index ($r=.53$, $P<.01$), and 25 (89%) of 28 patients with right atrial pressures greater than or equal to 10 mm Hg had cardiac indexes less than 2.2 L/min/m², as did seven (32%) of 22 patients with right atrial pressures less than 10 mm Hg ($P<.001$).

## COMMENT

This prospective study comparing the cardiovascular physical examination to hemodynamic measurements in patients with known chronic heart failure (characterized by systolic dysfunction and ventricular dilation) showed that left ventricular filling pressure frequently exceeded 22 to 25 mm Hg despite the absence of specific evidence in the physical signs. However, cardiac indexes less than or equal to 2.2 L/min/m² were predicted reliably by the proportional pulse pressure measured at the bedside.

While other studies have reported the value of clinical information in diagnosing left ventricular dysfunction,[3-8] the purpose of this study was not to distinguish normal from abnormal ventricular function but to assess hemodynamic status among patients known to have low left ventricular ejection fractions. The primary hemodynamic abnormalities caused by left ventricular systolic dysfunction are elevation of left ventricular filling pressures and depression of cardiac output, which do not necessarily occur together. The reliability of the physical signs for identifying marked to severe elevations of ventricular filling pressure and reductions in cardiac output has not been established previously in this population, for whom inferences drawn from physical signs are important for optimal therapy with vasodilators and diuretics, especially in the outpatient setting.

A sudden elevation of pulmonary venous pressure causes rales due to extravasation of fluid into the alveoli,[10] but chronic exudation of fluid is associated with an increase in lymphatic drainage so that the alveoli remain relatively dry and rales are absent. In our patients in whom symptomatic heart failure had been present for at least three months, pulmonary rales were rarely present, even when the pulmonary capillary wedge pressures were greater than or equal to 35 mm Hg. However, accumulation of fluid in the alveolar-capillary interstitium leads to decreased lung compliance and dyspnea,[11] which also can result from an increase in physiological dead space due to ventilation-perfusion mismatching.[12] Clinical recognition of this chronic stage of pulmonary ede-

Table 4.—Relation Between Proportional Pulse Pressure and Cardiac Index in 50 Patients With Chronic Heart Failure

| Cardiac Index, L/min/m² | Proportional Pulse Pressure, % | |
|---|---|---|
| | ≤25 | >25 |
| ≤2.2 | 29 | 3* |
| >2.2 | 3 | 15 |

*Three patients with cardiac index of 2.0 to 2.17 L/min/m²; $P<.0001$.

Table 5.—Hemodynamic Status for 50 Patients With Chronic Heart Failure

| Pulmonary Capillary Wedge Pressure, mm Hg* | Cardiac Index, L/min/m² | |
|---|---|---|
| | ≤2.2 | >2.2 |
| ≥22 | 32 | 11 |
| ≤18 | 0 | 7 |

*No patients with pressure of 19 to 21 mm Hg; $P<.001$.

ma is difficult[13] and should not depend on the presence of rales.

Third heart sounds have been considered reliable signs of heart failure and elevated ventricular filling pressure[2-8] and are helpful for the initial diagnosis of ventricular failure. Nevertheless, in our patients already known to have severely reduced ejection fractions, third heart sounds were too common to be specific for identifying major elevations of left ventricular filling pressure.

Physical signs of high right ventricular filling pressure usually were evident when the right atrial mean pressure was greater than 10 mm Hg. Variations in loading conditions of the right ventricle are reliably transmitted directly into the systemic venous system, which can be assessed visually in the jugular pulse.[2] Elevated right atrial pressure, in this setting of left ventricular failure, was always accompanied by elevated left ventricular filling pressure, although the converse was not true in this and previous studies.[9,14] Elevated jugular venous pressure and peripheral edema, therefore, were specific but not sensitive for high left ventricular filling pressure. The correlation between high right ventricular filling pressure and low cardiac output may reflect the effect of poor left ventricular function on right ventricular performance, impairment of left ventricular filling by right ventricular distention, or the importance of right ventricular function as a determinant of cardiac output, at least during physical exercise.[15,16]

Exertional dyspnea may result more from inadequate tissue oxygen delivery than from pulmonary venous congestion,[17] but orthopnea results from positional volume changes that increase left ventricular filling pressure and extravascular pulmonary fluid.[18] In our study, a history of recent orthopnea was the most reliable clinical indicator of high left ventricular filling pressure.

Cuff blood pressure has been used in the diagnosis of a low ejection fraction[4] but previously has not been evaluated specifically for assessment of cardiac output in patients with chronically low ejection fractions. In our patients, neither the systolic blood pressure nor the mean arterial pressure alone predicted cardiac index or stroke volume index, which were predicted by the proportional pulse pressure. The pulse pressure reflects the complex interplay between stroke volume, heart rate, aortic distensibility, and peripheral vascular tone and their relative contributions to incident and reflected waves.[19-21] While measurement of cardiac output from pulse pressure was first suggested in 1904,[22] even the introduction of multiple adjustment factors did not allow precise quantitation of cardiac output from pulse pressures for a varied population.[23-25]

In this study of simple physical assessment of hemodynamic status, pulse pressure was used primarily to identify the presence of severely reduced cardiac index rather than to estimate an exact value. As many patients with severe heart failure achieve their best cardiac outputs at systolic blood pressures below 100 mm Hg,[26] pulse pressure as a proportion of total systolic pressure was analyzed. A proportional pulse pressure less than or equal to 25% identified 91% of patients with cardiac indexes less than or equal to 2.2 L/min/m$^2$, while a higher pulse pressure identified 83% of patients with higher cardiac indexes. For comparison, proportional pulse pressures derived from blood pressure recordings in 42 patients with noncardiac disease (without hypertension) and Fick cardiac outputs of $6.3 \pm 0.4$ L/min/m$^2$ were $39\% \pm 8\%$, with only one value less than or equal to 25% in an early study on cardiac output measurement.[25] It should be emphasized that the current study validates proportional pulse pressure only for patients with known chronic dilated left ventricular failure (ejection fraction, $.18 \pm .06$) and may not be applicable to patients with other cardiac diseases, including hypertension.

The role of the physical examination in the management of chronic heart failure is coupled closely to the hemodynamic goals of therapy. Pulmonary capillary wedge pressure usually can be lowered to near-normal levels by diuretic and vasodilator therapy in the chronically dilated ventricle without compromising cardiac output.[26] Such therapy not only improves symptoms of congestion but also reduces the left ventricular dilation and mitral regurgitation that increase ventricular work and further contribute to decompensation.[27] A cardiac index of 2.0 to 2.2 L/min/m$^2$ represents a reasonable estimate of the minimum necessary for adequate organ perfusion with normal hemoglobin level and resting oxygen consumption.[28] The efficacy of many vasodilator agents for increasing lower cardiac outputs has been shown.[29,30] Pulmonary wedge pressure less than 18 mm Hg and cardiac index greater than 2.2 L/min/m$^2$, therefore, represent conservative hemodynamic goals for this patient population.

The four hemodynamic profiles defined for acute myocardial infarction[9] were adapted to our patients with chronic heart failure. Although high pulmonary capillary wedge pressure generally accompanies low cardiac index, the converse is not always true. There were seven patients with the first profile, cardiac index greater than 2.2 L/min/m$^2$ and pulmonary wedge pressure less than or equal to 18 mm Hg, consistent with adequate therapy. Pulmonary capillary wedge pressure was high without low cardiac index in 11 patients (second profile). Unlike patients with acute infarction,[9] none of our patients with chronic heart failure presented with low filling pressure *and* low cardiac index (third profile). Pulmonary capillary wedge pressure was high with low cardiac index in 32 patients (fourth profile). Despite the use of digoxin, diuretics, and vasodilators in most patients prior to evaluation, 43 (86%) of 50 patients referred thus had hemodynamic profiles with targets for further therapy with diuretics, vasodilators, or both.

Physical evidence of pulmonary congestion (rales) was specific for profile 2 or 4, but rare. Detection of elevated mean jugular venous pressure was more common, but still not very sensitive. Narrow proportional pulse pressure indicated profile 4 (or 3). When the hemodynamic profile and targets for therapy can be identified from the physical examination, therapy often can be adjusted effectively in the outpatient setting, without invasive quantitation of left ventricular filling pressures or cardiac output. However, the frequency of undetected high left ventricular filling pressures, with or without low cardiac index, indicates that patients with low ejection fractions who describe dyspnea and particularly orthopnea should not be doubted merely because specific physical signs of pulmonary and systemic venous congestion are absent. These patients may require invasive study to detect and treat severely elevated left ventricular filling pressures. In addition, patients in whom empiric therapy based on pulse pressure, venous pressure, or orthopnea is limited by hypotension or declining renal function may benefit from insertion of an indwelling pulmonary arterial catheter to facilitate more precise design of treatment. Initial administration of intravenous diuretics and vasodilators tailored to reduce pulmonary capillary wedge pressure and systemic vascular resistance often allows the subsequent design of effective oral therapy in patients previously considered clinically refractory.[31]

By prospective comparison of cardiovascular physical signs with hemodynamic measurements in patients with chronic congestive heart failure from dilated cardiomyopathy or end-stage coronary artery disease, our study has

shown that marked to severe elevations of left ventricular filling pressure frequently are undetected, while the adequacy of resting cardiac output is assessed reliably by the proportional pulse pressure. These observations allow more effective adjustment of outpatient medical therapy for chronic heart failure and more judicious use of invasive hemodynamic monitoring in this population.

This study was supported in part by the Streisand/American Heart Association Endowment and was carried out during Dr Stevenson's tenure as a clinician-scientist of the American Heart Association, Greater Los Angeles Affiliate.

We wish to express appreciation to the staff of the UCLA 4 West Special Procedures Room, supervised by Richard Pandolfi, and to thank Diane Miller and Michelle Moravec for secretarial assistance.

## References

1. Mattingly TW: Clinical and hemodynamic features of primary myocardial disease. *Trans Am Clin Clim Assoc* 1958;70:132-139.
2. Perloff JK: The arterial pulse, in *Physical Examination of the Heart and Circulation.* Philadelphia, WB Saunders Co, 1982, p 50.
3. Mattleman SJ, Hakki A-H, Abdulmassih AS, et al: Reliability of bedside evaluation in determining left ventricular function: Correlation with left ventricular ejection fraction determined by radionuclide ventriculography. *J Am Coll Cardiol* 1983; 1:417-420.
4. Cease KB, Nicklas JM: Prediction of left ventricular ejection fraction using simple quantitative clinical information. *Am J Med* 1986;81:429-439.
5. Echeverria HH, Bilsker MS, Myerburg RJ, et al: Congestive heart failure: Echocardiographic insights. *Am J Med* 1983;75:750-755.
6. Eagle KA, Quertermous T, Singer DE, et al: Left ventricular ejection fraction: Physician estimates compared with gated blood pool scan measurements. *Arch Intern Med* 1988;148:882-885.
7. Marantz PR, Tobin JN, Wassertheil-Smolker S, et al: Relationship between left ventricular systolic function and congestive heart failure diagnosed by clinical criteria. *Circulation* 1988;77:607-612.
8. Harian WR, Oberman A, Grimm R, et al: Chronic congestive heart failure in coronary artery disease: Clinical criteria. *Ann Intern Med* 1987;86:133-138.
9. Forrester JS, Diamond G, Chatterjee K, et al: Medical therapy of acute myocardial infarction by application of hemodynamic subsets. *N Engl J Med* 1976;295:1356-1362.
10. Staub NC, Nagano H, Pearce ML: Pulmonary edema in dogs, especially the sequence of fluid accumulation in lungs. *J Appl Physiol* 1967;22:227-240.
11. Straub NC: Pulmonary edema. *Physiol Rev* 1974;54:679-700.
12. Sullivan MJ, Higginbotham MB, Cobb FR: Increased exercise ventilation in patients with chronic heart failure: Intact control despite hemodynamic and pulmonary abnormalities. *Circulation* 1988;77:552-559.
13. Szidon JP, Pietra GG, Fishman AP: The alveolar-capillary membrane and pulmonary edema. *N Engl J Med* 1972;286:1200-1204.
14. Mangano DT: Monitoring pulmonary arterial pressure in coronary-artery disease. *Anesthesiology* 1980;53:364-370.
15. Baker BJ, Wilen MW, Boyd CM, et al: Relation of right ventricular ejection fraction to exercise capacity in chronic left ventricular failure. *Am J Cardiol* 1984;54:596-599.
16. Barber D, DiSessa T, Child JS, et al: Hemodynamic responses to isolated increments in heart rate by atrial pacing after a Fontan procedure. *Am Heart J* 1988;115:837-841.
17. Fink LI, Wilson JR, Ferraro N: Exercise ventilation and pulmonary artery wedge pressure in chronic stable congestive heart failure. *Am J Cardiol* 1986;57:249-253.
18. Perera GA, Berliner RW: The relation of postural hemodilution to paroxysmal dyspnea. *J Clin Invest* 1943;22:25-28.
19. O'Rourke MF: The arterial pulse in health and disease. *Am Heart J* 1971;82:687-702.
20. Pepine CJ, Nichols WW, Conti R: Aortic input impedance in heart failure. *Circulation* 1978;58:460-465.
21. Abrams J: Arterial pulse and pressure. *Cardiovasc Rev Rep* 1985;6:1055-1073.
22. Erlanger J, Hooker DR: Studies in blood pressure. *Johns Hopkins Hosp Bull* 1904;15:179-180.
23. Remington JW, Noback CR, Hamilton WF, et al: Volume elasticity characteristics of the human aorta and prediction of the stroke volume from the pressure pulse. *Am J Physiol* 1948;153:298-308.
24. Brotmacher L: Evaluation of derivation of cardiac output from blood pressure measurements. *Circ Res* 1957;5:589-593.
25. Starr I: Clinical tests of the simple method of estimating cardiac stroke volume from blood pressure and age. *Circulation* 1954;9:664-681.
26. Stevenson LW, Tillisch JH: Maintenance of cardiac output with normal filling pressures in patients with dilated heart failure. *Circulation* 1986;74:1303-1308.
27. Stevenson LW, Bellil D, Grover-McKay M, et al: Effects of afterload reduction on left ventricular volume and mitral regurgitation in severe congestive heart failure secondary to ischemic or idiopathic dilated cardiomyopathy. *Am J Cardiol* 1987;60:654-658.
28. Grossman W, Braunwald E: High cardiac output states, in Braunwald E (ed): *Heart Disease.* Philadelphia, WB Saunders Co, 1984, p 807.
29. Cohn JN, Franciosa JA: Vasodilator therapy of cardiac failure. *N Engl J Med* 1977;297:27-31, 254-258.
30. Chatterjee K, Parmely WW: Vasodilator therapy for acute myocardial infarction and chronic congestive heart failure. *J Am Coll Cardiol* 1983; 1:133-153.
31. Stevenson LW, Dracup KA, Tillisch JH: Efficacy of medical therapy tailored for severe congestive heart failure in patients transferred for urgent cardiac transplantation. *Am J Cardiol,* in press.