JACC Vol. 14, No. 5
November 1, 1989:1331–8



# Inotropic Effect of Nicardipine in Patients With Heart Failure: Assessment by Left Ventricular End-Systolic Pressure-Volume Analysis

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**Nicardipine, a new dihydropyridine calcium channel blocker, has been investigated for the treatment of coronary artery disease and heart failure. To assess the inotropic effect of nicardipine in humans independent of its vasodilator effect, equihypotensive doses of intravenous nitroprusside (mean infusion rate 65 ± 13 μg/min) and nicardipine (mean dose 5.2 ± 0.4 mg) were administered to 15 patients with heart failure (New York Heart Association functional classes II to IV, radionuclide left ventricular ejection fraction 0.15 ± 0.02). Left ventricular micromanometer pressure and simultaneous radionuclide left ventricular volume were obtained at baseline, during nitroprusside infusion, during a second baseline period and during nicardipine infusion.**

**Heart rate did not change significantly with either nitroprusside or nicardipine. Mean systemic arterial pressure decreased by an average of 21 mm Hg with both drugs. A greater decrease in left ventricular end-diastolic pressure occurred with nitroprusside (27 ± 2 to 14 ± 2 mm Hg, $p < 0.01$) than with nicardipine (27 ± 2 to 23 ± 3 mm Hg, $p < 0.05$), and pulmonary capillary wedge pressure decreased significantly only with nitroprusside. Cardiac index increased from 1.8 ± 0.1 to 2.1 ± 0.1 liters/min per $m^2$ ($p < 0.05$) with nitroprusside and to a greater extent from 1.7 ± 0.1 to 2.4 ± 0.1 liters/min per $m^2$ ($p < 0.01$) with nicardipine. Left ventricular ejection fraction increased with nicardipine (0.15 ± 0.01 to 0.19 ± 0.01, $p < 0.01$), but not with nitroprusside.**

**Peak positive first derivative of left ventricular pressure (dP/dt) decreased by 9% with both agents. Left ventricular pressure-volume loops were constructed for 14 patients. In 12 patients, the left ventricular end-systolic pressure-volume relation was shifted rightward with nicardipine, indicating a negative inotropic effect, with no shift in the remaining 2 patients.**

**Thus, nicardipine has a negative inotropic effect in patients with heart failure. Despite this effect, left ventricular ejection fraction and cardiac index increased with nicardipine. This overall improvement in ventricular systolic performance was due to a reduction in afterload.**

*(J Am Coll Cardiol 1989;14:1331–8)*

Vasodilators have assumed an important role in the short- and long-term management of heart failure due to left ventricular systolic dysfunction (1,2). As systemic vasodilators, calcium channel blockers have generated considerable interest as agents for the treatment of heart failure (3). Because calcium channel blockers are coronary as well as systemic vasodilators, they may be of particular benefit to patients with heart failure due to coronary artery disease, especially in patients with manifestations of active ischemia. The currently available calcium channel blockers, verapamil, nifedipine and diltiazem, all have potentially important negative inotropic effects (4–8). Because of these effects, efforts are underway to identify newer calcium channel blockers with minimal or absent negative inotropic effects.

Nicardipine is a dihydropyridine calcium channel blocker that has been shown to be beneficial in patients with angina pectoris (9–13). Administration of nicardipine to patients with heart failure has been reported (14–16) to improve overall left ventricular systolic performance as a result of

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Manuscript received February 7, 1989; revised manuscript received April 25, 1989, accepted May 10, 1989.

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0735-1097/89/$3.50

afterload reduction. It has been suggested that nicardipine has only a minimal negative inotropic effect, which is less important than that of nifedipine in vitro (17), in a canine model (18) and in patients (19). Clinical assessment of the inotropic effect of nicardipine, which is also a potent systemic vasodilator, has been hindered by the load dependence of most indexes of contractility. Recently, the end-systolic pressure-volume relation has emerged as a relatively load-independent measure of left ventricular contractile function (20–26) and has proved useful for the evaluation of new drugs for the treatment of heart failure (27). The present study utilized the left ventricular end-systolic pressure-volume relation to assess the inotropic effect of nicardipine in patients with heart failure.

## Methods

**Study patients.** The study group comprised 14 men and 1 woman, aged 63 ± 2 years, with chronic heart failure and a left ventricular ejection fraction <0.40. Heart failure was due to coronary artery disease in 12 patients and to idiopathic dilated cardiomyopathy in 3. All patients remained symptomatic despite treatment with digoxin, diuretic drugs and vasodilators. Six patients were in New York Heart Association functional class II, four were in class III and five were in class IV. All patients had sinus rhythm. The experimental protocol was approved by the Subcommittee for Human Studies of the Massachusetts General Hospital. Informed consent was obtained from all patients.

**Hemodynamic measurements.** Patients underwent left and right heart catheterization without premedication 8 to 24 h after discontinuation of digoxin, diuretic drugs and vasodilators. Left heart catheterization was performed from the femoral approach using a micromanometer catheter (Millar Instruments) in 14 patients and a fluid-filled catheter in 1 patient. Systemic arterial pressure was measured from the sidearm of a femoral artery introducer. The following hemodynamic variables were recorded: heart rate, right atrial pressure, pulmonary artery pressure, pulmonary capillary wedge pressure, high fidelity left ventricular pressure, mean systemic arterial pressure and, by electronic differentiation, the first derivative of left ventricular pressure (dP/dt). Left ventricular dP/dt was not measured in the one patient without micromanometer catheterization. Cardiac index, stroke volume, systemic vascular resistance and pulmonary vascular resistance were calculated from the cardiac output obtained by the thermodilution technique.

**Drug administration.** After hemodynamic and radionuclide data were obtained at baseline study, nitroprusside was infused at an initial dose of 25 μg/min and titrated upward with the goal of achieving a 15 to 20 mm Hg decrease in mean systemic arterial pressure. After an equilibration period of 10 min, hemodynamic data were acquired and radionuclide angiography performed. Nitroprusside administration was then discontinued, and data were acquired 15 min later when mean arterial pressure had returned to its baseline value. Nicardipine was then infused at an initial dosage of 1 mg/min to achieve a 15 to 20 mm Hg reduction in mean arterial pressure ("bolus") and titrated downward to maintain this reduction over a 10 min period ("infusion"), during which data were acquired.

**Gated blood pool imaging.** Left ventricular volume was calculated from supine gated blood pool images, as previously described (27,28). Briefly, scans were acquired in the anterior (16 frames) and left anterior oblique (32 frames) views after labeling of red blood cells in vivo with 30 mCi of technetium-99m. With use of the left anterior oblique view, a time-activity curve of the left ventricle was constructed by a semiautomated edge detection method with a variable region of interest. During nitroprusside infusion, the second baseline period and nicardipine infusion, images were acquired in only the left anterior oblique view, and care was taken to avoid patient and camera movement during the study. Absolute left ventricular end-diastolic volume at baseline study was derived from a previously validated geometric biplane area-length method (29). Volumes at other time points in the cardiac cycle and during the nitroprusside infusion, second baseline and nicardipine infusion periods were calculated as the baseline end-diastolic volume multiplied by the ratio of counts in a particular frame to counts in the baseline end-diastolic frame. Counts were smoothed by using a three point weighted moving average (coefficients: 0.25, 0.50, 0.25). Counts in scans obtained during nitroprusside infusion, second baseline and nicardipine infusion periods were corrected for differences from the baseline scan in acquisition time and frame interval and for physical and biologic decay of the isotope. Volumes, and hence pressure-volume loops, were obtained in 14 patients.

**Data analysis.** High fidelity left ventricular pressure tracings from four consecutive beats at the midpoint of the gated scan acquisition were traced and digitized on a Summagraphics Bitpad interfaced to a VAX 780 computer. Pressure-volume loops were then constructed by plotting these pressures with the corresponding radionuclide ventriculographic volumes, starting at a simultaneous time point (the peak of the R wave on the electrocardiogram).

To generate the baseline end-systolic pressure-volume relation, nitroprusside, a "pure" vasodilator with no inotropic effect, was administered to alter end-systolic pressure. The baseline end-systolic pressure-volume relation was determined by the method of Kass et al. (30) from the pressure-volume loops (baseline, nitroprusside infusion and second baseline periods) obtained for each patient. Points from each loop for which the ratio of pressure to volume was maximal were selected, and linear regression analysis of these points was performed. The extrapolated volume-axis intercept at zero pressure was determined from the regression analysis. Points on each loop for which pressure divided by the

difference between corresponding volume and the volume-axis intercept was maximal were then selected, and a new pressure-volume line (with a new volume-axis intercept) was generated. This process was iterated until there was no change in slope (end-systolic elastance) or volume-axis intercept. The coefficients used for weighting the end-systolic points derived from the baseline, nitroprusside and second baseline pressure-volume loops were 0.25, 0.50 and 0.25, respectively.

*Stroke work index* was determined from the integrated area of the pressure-volume loops as stroke work index (g-m/m$^2$) = 0.0136 × pressure-volume area (mm Hg-ml)/body surface area (m$^2$).

**Statistics.** Results are expressed as mean values ± SEM. Comparisons between two measurements were made by paired *t* test. Comparisons among treatment periods were made by analysis of variance, with differences between group mean values assessed by the Newman-Keuls test. The cumulative binomial distribution test was used to assess the significance of the shift of the end-systolic point derived during the nicardipine infusion from the baseline end-systolic pressure-volume relation. Differences for which the p value was <0.05 were considered significant.

## Results

**Baseline hemodynamic data (Table 1).** Baseline heart rate was 87 ± 3 beats/min, and mean arterial pressure 94 ± 4 mm Hg. Baseline pulmonary capillary wedge (24 ± 3 mm Hg) and left ventricular end-diastolic (27 ± 2 mm Hg) pressures were elevated, and cardiac index (1.8 ± 0.1 liters/min per m$^2$) was depressed. There were no significant differences between the two baseline periods in any of the measured or derived variables.

**Hemodynamic responses to nitroprusside and nicardipine (Table 1, Fig. 1).** During nitroprusside infusion (65 ± 13 μg/min), systemic arterial, right atrial, pulmonary artery and pulmonary capillary wedge pressures decreased, as did systemic and pulmonary vascular resistance. There was no significant change in heart rate. Left ventricular end-diastolic pressure decreased from 27 ± 2 to 14 ± 2 mm Hg ($p < 0.01$), whereas cardiac index increased from 1.8 ± 0.1 to 2.1 ± 0.1 liters/min per m$^2$ ($p < 0.05$).

Nicardipine administration (bolus dose 1.7 ± 0.2 mg, infusion rate 0.27 ± 0.03 mg/min, total dose 5.2 ± 0.4 mg) caused a decrease in systemic arterial pressure and systemic and pulmonary vascular resistance. Heart rate was not affected. There was no change in pulmonary artery or pulmonary capillary wedge pressures, although left ventricular end-diastolic pressure decreased slightly (27 ± 2 to 23 ± 3 mm Hg, $p < 0.05$). Cardiac index increased from 1.7 ± 0.1 to 2.4 ± 0.1 liters/min per m$^2$ ($p < 0.01$). Nicardipine caused stroke volume to increase in all patients. Left ventricular end-diastolic pressure increased by 3 mm Hg in Patient 12 and by 1 mm Hg in Patient 13 and decreased or did not change in the other 13 patients.

**Table 1.** Hemodynamic Response to Nitroprusside and Nicardipine in 14 Patients

| | Baseline | Nitroprusside | Baseline | Nicardipine |
|---|---|---|---|---|
| HR | 87 ± 3 | 84 ± 3 | 85 ± 3 | 84 ± 3 |
| MAP | 94 ± 4 | 73 ± 2* | 91 ± 4 | 72 ± 2* |
| RAP | 6 ± 1 | 3 ± 1* | 6 ± 1 | 7 ± 1† |
| PAP | 33 ± 3 | 19 ± 2* | 33 ± 3 | 31 ± 3† |
| PCWP | 24 ± 3 | 11 ± 2* | 24 ± 3 | 22 ± 3† |
| LVEDP | 27 ± 2 | 14 ± 2* | 27 ± 2 | 23 ± 3*† |
| LVESP | 94 ± 4 | 74 ± 3* | 91 ± 4 | 71 ± 3* |
| CI | 1.8 ± 0.1 | 2.1 ± 0.1* | 1.7 ± 0.1 | 2.4 ± 0.1*† |
| SWI | 40 ± 6 | 32 ± 4* | 39 ± 5 | 41 ± 5 |
| SVR | 2,131 ± 150 | 1,486 ± 130* | 2,130 ± 129 | 1,170 ± 66*† |
| PVR | 230 ± 39 | 167 ± 23* | 234 ± 36 | 157 ± 18* |
| Peak +dP/dt | 1,156 ± 95 | 1,030 ± 64* | 1,070 ± 85 | 959 ± 66*† |
| LVEDV | 412 ± 20 | 370 ± 21* | 409 ± 19 | 401 ± 21† |
| LVESV | 352 ± 20 | 312 ± 19* | 347 ± 19 | 328 ± 20*† |
| LVEF | 0.15 ± 0.02 | 0.16 ± 0.01 | 0.15 ± 0.01 | 0.19 ± 0.01*† |

*$p < 0.05$ drug vs. preceding baseline; †$p < 0.05$ nicardipine vs. nitroprusside. Values shown are mean values ± SEM. CI = cardiac index (liters/min per m$^2$); HR = heart rate (beats/min); LVEDP = left ventricular end-diastolic pressure (mm Hg); LVEDV = left ventricular end-diastolic volume (ml); LVEF = left ventricular ejection fraction; LVESP = left ventricular end-systolic pressure (mm Hg); LVESV = left ventricular end-systolic volume (ml); MAP = mean systemic arterial pressure (mm Hg); PAP = mean pulmonary arterial pressure (mm Hg); PCWP = pulmonary capillary wedge pressure (mm Hg); Peak +dP/dt = peak positive left ventricular dP/dt (mm Hg/s); PVR = pulmonary vascular resistance (dynes-s/cm$^5$); RAP = right atrial pressure (mm Hg); SVR = systemic vascular resistance (dynes-s/cm$^5$); SWI = stroke work index (g-m/m$^2$).

*Comparison of the effects of nicardipine with those of nitroprusside (Fig. 1)* shows that for a similar decrease in afterload, as measured by mean arterial pressure or left ventricular end-systolic pressure, nicardipine caused a larger increase in cardiac index (42% compared with 17% with nitroprusside, $p < 0.01$). The greater increase in cardiac index during nicardipine administration was attributed to the relative maintenance of preload with this drug because left ventricular end-diastolic volume decreased from 412 ± 20 to 370 ± 21 ml during nitroprusside infusion ($p < 0.01$), but remained unchanged during the nicardipine infusion (409 ± 19 to 401 ± 21 ml; p = NS).

**Assessment of left ventricular contractile function (Fig. 2).** Peak positive dP/dt decreased by similar amounts with nitroprusside (1,156 ± 95 to 1,030 ± 64 mm Hg/s, $p < 0.01$) and nicardipine (1,070 ± 85 to 959 ± 66 mm Hg/s, $p < 0.01$). Left ventricular ejection fraction was unchanged with nitroprusside, but increased with nicardipine (0.15 ± 0.01 to 0.19 ± 0.01, $p < 0.01$). Stroke work index, derived from the integrated area of the left ventricular pressure-volume loops, decreased with nitroprusside (40 ± 6 to 32 ± 4 g-m/m$^2$, $p < 0.05$), but was unchanged with nicardipine.

*Left ventricular pressure-volume loops are shown in*

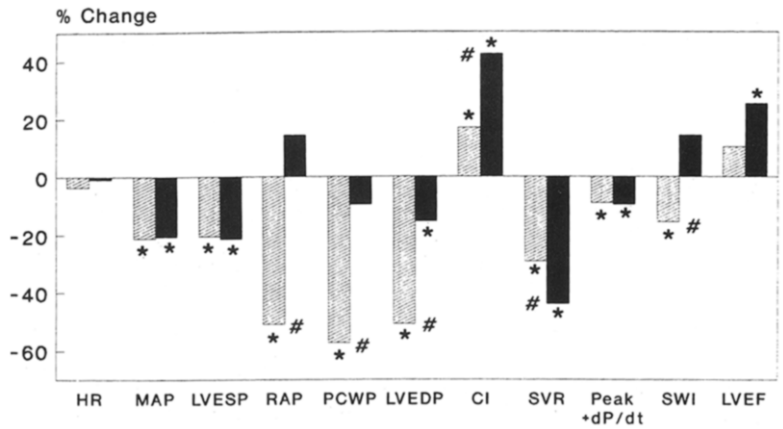


**Figure 1.** Relative changes in hemodynamic indexes with infusion of nitroprusside (**hatched bars**) and nicardipine (**solid bars**). CI = cardiac index; dP/dt = first derivative of left ventricular pressure; HR = heart rate; LVEDP = left ventricular end-diastolic pressure; LVEF = left ventricular ejection fraction; LVESP = left ventricular end-systolic pressure; MAP = mean arterial pressure; PCWP = pulmonary capillary wedge pressure; RAP = right arterial pressure; SVR = systemic vascular resistance; SWI = stroke work index. * $p < 0.05$ versus preceding baseline; # $p < 0.05$ versus nitroprusside.

*Figure 2.* The loop obtained during infusion of nicardipine was shifted downward and rightward relative to the baseline in all but two patients (Cases 6 and 11). This shift in 12 of 14 patients indicates depression of left ventricular contractile function during nicardipine administration compared with baseline values ($p = 0.012$ by the cumulative binomial distribution test).

**Side effects.** One patient (Case 8) experienced an increase in baseline ventricular ectopic activity during nicardipine infusion. Sinus bradycardia developed during both nitroprusside and nicardipine infusions in one patient (Case 14), and pronounced transitory hypotension occurred with nitroprusside in another (Case 5). No patient suffered from hemodynamic decompensation during nicardipine administration, none required specific therapy for side effects and all completed the study protocol.

## Discussion

Many patients with coronary artery disease have manifestations of both active ischemia and heart failure. Others have left ventricular systolic dysfunction without symptoms and signs of heart failure. Calcium channel blockers may be beneficial for both groups of patients because both myocardial oxygen supply-demand balance and left ventricular performance may be improved by the coronary and systemic vasodilator effects of these agents. Administration of the available calcium channel blockers to patients with systolic dysfunction has been limited, however, by theoretical and practical concerns related to their negative inotropic effects.

**Effects of available calcium channel blockers on myocardial contractility and ventricular performance.** The negative inotropic properties of calcium channel blockers have been demonstrated in isolated cardiac muscle preparations (31,32). Of the currently available agents—verapamil, diltiazem and nifedipine—verapamil offers the least favorable balance between vasodilator and negative inotropic effects because of its relatively weak effects as a vasodilator (32). Although administration of verapamil to patients with reduced left ventricular ejection fraction has been reported to improve overall systolic performance in many patients (33), it may result in hemodynamic decompensation in others (4).

*Nifedipine, a dihydropyridine derivative*, is a more potent systemic and coronary vasodilator than is verapamil (32) and has generally been reported to bring about an improvement in ventricular performance in patients with chronic left ventricular dysfunction (34–37). A negative inotropic effect of nifedipine, however, has been demonstrated in vivo after intracoronary injection in patients with coronary artery disease (34,38) and after sublingual administration in patients with chronic stable heart failure (6). The clinical importance of this negative inotropic effect is underscored by the demonstration of acute hemodynamic deterioration after administration of the drug to some patients (7,8).

*Of the three available agents, diltiazem* has undergone the least thorough investigation of its inotropic and hemodynamic effects in patients with heart failure. Although a negative inotropic effect has been demonstrated in an animal model (5), a study (39) in a small group of patients with heart failure did not show such an effect. The negative *chronotropic* effect of diltiazem, like that of verapamil, may be disadvantageous in patients whose stroke volume is relatively fixed because of myocardial dysfunction.

**Effects of nicardipine on myocardial contractility and ventricular performance.** Nicardipine, like nifedipine, is a dihydropyridine derivative with potent vasodilating and minimal negative chronotropic effects. In an in vitro study (17), the negative inotropic potency of nicardipine was demonstrated to be 10% of that of nifedipine, whereas the vasodilator potency of the two drugs was similar. In open chest dogs, the cardiodepressant effects of nicardipine were less than those of equimolar doses of nifedipine, and the differences were accentuated by pretreatment with propranolol (18). Rousseau et al. (19) found that intracoronary administration of nicardipine to patients with angina pectoris did not alter left ventricular peak positive dP/dt, in contrast to the decrease in dP/dt observed after intracoronary administration of nifedi-

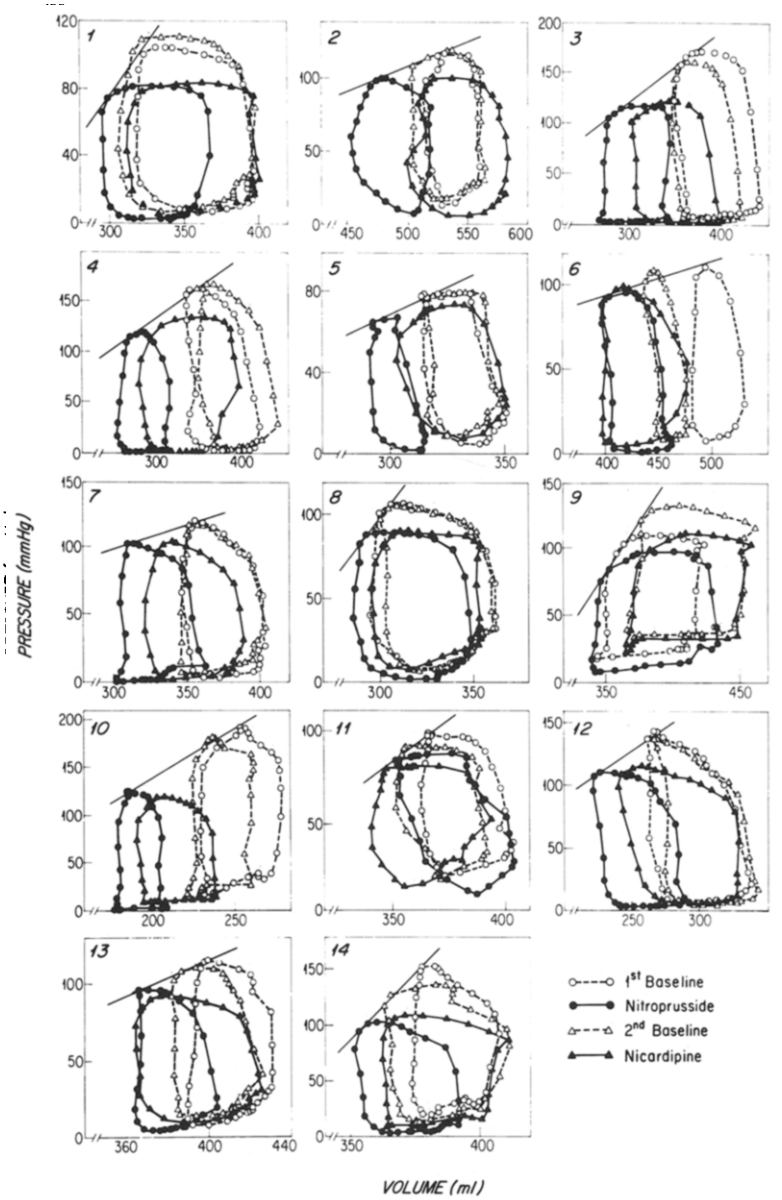


**Figure 2.** Pressure-volume loops demonstrating that nicardipine infusion was associated with a rightward shift from the baseline end-systolic pressure-volume relation, indicating a negative inotropic effect, in 12 of 14 patients (Cases 6 and 11 are exceptions). The **line** connecting the end-systolic points of the first and second baseline and nitroprusside pressure-volume loops, the baseline end-systolic pressure-volume relation, was derived by an iterative method (see text).

pine to patients after coronary artery bypass surgery (34,38). In a preliminary report (40), a decrease in dP/dt after intracoronary injection of nicardipine was seen in patients with coronary artery disease, but this decrease was significantly less (13% versus 34%) than that observed for nifedipine at equal doses of the two drugs (which produced equivalent augmentation of coronary blood flow).

*These findings have spurred investigation of the hemodynamic effects of nicardipine in patients with heart failure.* Several studies (14–16) in such patients have shown that cardiac output increases in response to nicardipine. Pulmonary capillary wedge pressure was observed to decrease by a modest amount (14,16) or remain unchanged (15); left ventricular end-diastolic pressure was not directly measured in these studies. In the present study, we also demonstrated an improvement in left ventricular pump performance, that is, an increase in cardiac output with a small decrease in left ventricular end-diastolic pressure (although pulmonary capillary wedge pressure, a less reliable measure of left ventricular filling pressure, did not decrease significantly).

Although we observed left ventricular peak positive dP/dt to decrease slightly in our patients, this index of contractility decreased by a similar amount with nitroprusside, a vasodilator without known inotropic effect. Peak positive dP/dt has indeed been demonstrated to be both preload- and afterload-dependent (41). Even though left ventricular ejection fraction did *increase*, as also observed in a previous study (42), this effect was most likely attributable to afterload reduction rather than to improvement in inotropic state of the ventricle. Differentiation of the inotropic effect of nicardipine from

its vasodilator effects in these patients required a load-independent measure of contractile function.

**Left ventricular end-systolic pressure-volume relation.** In this study, we utilized the left ventricular end-systolic pressure-volume relation, a relatively load-insensitive index of left ventricular contractile function, to assess whether nicardipine has a measurable negative inotropic effect in patients with heart failure. While it has been recognized for many years that the relation between end-systolic pressure and volume is independent of end-diastolic volume (43), this concept has only recently been applied to the assessment of contractility. Suga et al. (20) showed that the end-systolic pressure-volume relation in the canine left ventricle is insensitive to changes in preload, and that the slope of a line connecting end-systolic pressure-volume points is sensitive to changes in contractility. End-systolic pressure-volume analysis was subsequently extended to humans, and was shown to be a sensitive indicator of inotropic state independent of loading conditions (22,25,26). We previously demonstrated (44) the linearity of the end-systolic pressure-volume relation in patients with severe heart failure.

In the present study, we demonstrated a downward and rightward shift of the end-systolic pressure-volume relation in the majority (86%) of our patients with heart failure due to left ventricular systolic dysfunction. Because of the time constraints inherent in a clinical study incorporating left heart catheterization, we did not generate pressure-volume loops under multiple afterload conditions during nicardipine infusion. As a result of this limitation, we could not construct the full end-systolic pressure-volume relation for the left ventricle with nicardipine and, thus, did not determine whether the change in the relation was characterized by alterations in slope or intercept (that is, a parallel shift), or both. Indeed, inotropic interventions have been shown (45) to have variable relative effects on the slope and intercept of the end-systolic pressure-volume relation. Because end-systolic volume was higher with nicardipine than that predicted from the baseline end-systolic pressure-volume relation for the same end-systolic pressure, we conclude that left ventricular contractile performance was depressed by nicardipine.

**Limitations of the study.** Several other limitations of the present study must be acknowledged. First, because of time constraints, only one nitroprusside dosage and, thus, only one diminished afterload condition were utilized to generate the baseline end-systolic pressure-volume relation. Construction of the relation using only two afterload conditions is based on the assumption that the relation is linear in this range of afterload. Although theoretical considerations (46) and experimental data (47,48) indicate nonlinearity of the end-systolic pressure-volume relation under certain conditions, we have shown (44) that the relation is accurately represented by a straight line in the "physiologic" range of afterload in patients with heart failure.

*Second,* it has been suggested that infusion of nitroprusside alters the end-systolic pressure-volume relation (49,50). To obviate this limitation by using mechanical rather than pharmacologic means to generate the relation would have required use of balloon occlusion of the inferior vena cava. We have found (51) that such steady state alteration of loading conditions can be achieved by inflating a balloon in the inferior vena cava below the diaphragm, but that systemic arterial pressure is not sufficiently reduced by this technique to reliably construct the end-systolic pressure-volume relation.

*Third,* digoxin, diuretic drugs and vasodilators were withdrawn only 8 to 24 h before the study. It is possible that residual effects of these medications were present. It is unlikely, however, that their effects changed between treatment periods by an amount sufficient to alter our conclusions.

*Finally,* reduction in blood pressure by nitroprusside or nicardipine may cause a reflex increase in sympathetic tone, associated with a positive inotropic effect. Evidence for blunting or absence of this effect in heart failure has been demonstrated in a canine model (5) and in humans (6). In the present study, we did not measure levels of circulating catecholamines, but we observed no increase in heart rate associated with the reductions in systemic arterial pressure.

**Summary and implications for therapy.** We have shown that the acute intravenous administration of nicardipine to patients with heart failure is accompanied by measurable impairment of left ventricular contractile function. Despite this negative inotropic effect, overall left ventricular systolic performance is usually enhanced by nicardipine because of afterload reduction. Long-term studies enrolling greater numbers of patients with oral administration of nicardipine will be required to test its clinical efficacy and safety in patients with heart failure and myocardial ischemia. In patients with heart failure due to causes other than coronary artery disease, there is little rationale for prescribing nicardipine because vasodilators without any negative inotropic effect are available.

We gratefully acknowledge the advice of John B. Newell regarding statistical methods.

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