Effects of milrinone on coronary hemodynamics and myocardial energetics in patients with congestive heart failure

E. Scott Monrad, M.D., Donald S. Baim, M.D., Harton S. Smith, M.D., Alyce Lanoue, R.N., Eugene Braunwald, M.D., and William Grossman, M.D.

ABSTRACT To examine the effect of milrinone on myocardial energetics in patients with congestive heart failure, we measured systemic, pulmonary, and coronary hemodynamics in 18 patients before and after intravenous administration of milrinone (125 ± 36 μg/kg). There was a 45% increase in cardiac index (2.1 ± 0.5 to 3.0 ± 0.6 liters/min/m²; p = .0001), a 39% fall in the pulmonary capillary wedge pressure (28 ± 8 to 17 ± 8 mm Hg; p = .0001), and a 42% increase in left ventricular external work (3758 ± 1419 to 5340 ± 1598 g-m/min; p = .0001). Both the heart rate–blood pressure product (9624 ± 2272 to 9380 ± 2428 mm Hg-beats/min; p = NS) and regional left ventricular myocardial oxygen consumption (7.6 ± 2.9 to 8.1 ± 3.1 ml O₂/min; p = NS) were unchanged after milrinone, resulting in a 45% increase in calculated left ventricular external efficiency (p = .004). Although myocardial oxygen consumption did not change, regional great cardiac venous blood flow increased significantly (73 ± 32 to 85 ± 34 ml/min; p = .02) as a result of a 30% reduction in regional coronary vascular resistance (1.32 ± 0.99 to 0.93 ± 0.54 mm Hg-min/ml; p = .004), a decrease comparable to the concurrent 37% and 38% falls seen in systemic and pulmonary vascular resistances, respectively. These changes were associated with an 11% fall in the transcoronary arterial-venous oxygen difference (111 ± 24 to 99 ± 21 ml/O₂/liter; p = .0001), which is consistent with a primary coronary vasodilator effect of milrinone. Thus, milrinone enhances cardiac performance without a systematic increase in myocardial oxygen consumption, i.e., it increases left ventricular external efficiency. Furthermore, milrinone may improve coronary flow reserve by direct coronary vasodilation and/or reduction in left ventricular diastolic pressure.


MILRINONE, a bipyridine compound, causes major hemodynamic improvement in patients with advanced congestive heart failure1,2 by a combination of positive inotropic1,3-5 and systemic arteriolar vasodilator1-9 effects. Although a reduction of left ventricular systolic and diastolic pressures might be expected to improve myocardial oxygen balance through reduced myocardial work7,9 and improved subendocardial blood flow,10-12 respectively, these potential benefits may be offset by a concurrent increase in oxygen demand resulting from increases in heart rate and contractility.13 Any net increase in myocardial oxygen demand could be deleterious in patients with limited coronary vasodilator reserve due to coronary artery disease or myocardial hypertrophy, and might constitute a major limitation to the use of milrinone in such patients.

To address this issue of the effect of milrinone on the balance between myocardial oxygen supply and demand, we studied systemic and coronary hemodynamics before and after administration of intravenous milrinone in 18 patients with severe congestive heart failure.

Methods

Eighteen patients with advanced (New York Heart Association [NYHA] class III or IV) congestive heart failure that was refractory to standard medical therapy were studied. The study group included 11 patients with coronary artery disease (four of whom had undergone prior coronary artery bypass graft sur-
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gery), five patients with idiopathic dilated cardiomyopathy, one patient with dilated cardiomyopathy following acute inflammatory myocarditis, and one patient with dilated cardiomyopathy in association with surgically corrected mitral regurgitation. Clinical characteristics of this group, including NYHA class, left ventricular ejection fraction (mean 19%, range 4% to 33%), and medical regimens, were similar to those reported in our earlier group of patients treated with milrinone.1 All patients received digoxin and diuretics, and 13 patients received vasodilators as well. Informed consent was obtained from all patients under a protocol approved by the Human Studies Committee of the Beth Israel Hospital.

All diuretic and vasodilator medications were held for 24 hr before the study. After premedication with 10 mg diazepam and local anaesthesia with 1% xylocaine, right heart and coronary sinus catheterization were performed in each patient with a No. 7F thermodilution Swan-Ganz catheter (Edwards Laboratories, Santa Ana, CA) and a No. 7F Baim coronary sinus catheter (Electro-catheter Corporation, Rahway, NJ),14,15 both of which were inserted via adjacent sheaths into the right internal jugular vein. An arterial line was inserted percutaneously into either the radial or femoral artery.

The coronary sinus catheter was advanced over a 0.018 Floppy guide wire (Advanced Cardiovascular Systems, Temecula, CA) into the great cardiac vein beyond the point of entry of the marginal veins, as confirmed by contrast injection. Regional great cardiac venous blood flow was measured during a 30 sec continuous injection of room temperature indicator (5% dextrose in water) through the catheter tip lumen at a rate of 50 ml/min with use of a Mark 4 angiographic injector (Medrad Co.). Turbulence during the injection completely mixes the indicator with the coronary sinus blood, allowing calculation of coronary venous blood flow by the thermodilution technique, as previously described.14,15

All pressures (right atrial, pulmonary arterial, pulmonary capillary wedge, and systemic arterial), data on regional great cardiac vein flow, and the surface electrocardiogram were recorded on an optical strip-chart recorder (VR 16, Electronics for Medicine, Honeywell). Cardiac output was measured by both the thermodilution method, in which 10 ml boluses of iced 5% dextrose in water were used, and the Fick method, in which the MRM-1 (Waters Instruments, Minneapolis) with analysis of systemic arterial and pulmonary arterial oxygen saturations in vitro on the Unisat Oximeter (American Optical, Buffalo, NY) was used. Since there was no statistically significant difference between the Fick and thermodilution determinations of the cardiac output, only the Fick determinations are reported. In all patients coronary venous blood was withdrawn from the distal lumen of the coronary sinus catheter and analyzed on the Unisat Oximeter. In six patients arterial and coronary venous oxygen content analyses were also performed with the Lex-O2-Con (Lexington Instruments, Lexington, MA). The correlation coefficient between the Unisat Oximeter and the Lex-O2-Con measurements of the transcoronary arterial-venous oxygen difference was .96, and only the data obtained by the former method are presented.

Regional myocardial oxygen consumption (MVO2) was calculated from the product of the great cardiac vein blood flow and the transcoronary arterial-venous oxygen difference. Coronary vascular resistance (CVR) (mm Hg-min/ml) was calculated as

\[
CVR = \frac{(MAP - RAP)}{GCVF} \text{ or }
CVR' = \frac{(MAP - PCWP)}{GCVF}^{16}
\]

where MAP is mean arterial pressure, RAP is mean right atrial pressure, PCWP is mean pulmonary capillary wedge pressure (in mm Hg), and GCVF is great cardiac vein blood flow (in ml/min).

Left ventricular external work (LV work) in gram-meters per minute was calculated from the following equation:

\[
LV \text{ work} = (\text{MSAP} - \text{PCWP}) \times \text{CO} \times 0.0136
\]

where MSAP is mean systolic arterial pressure [estimated by the formula MSAP = SAP - \(\frac{1}{2}\) (SAP - DAP), where SAP is peak systolic arterial pressure and DAP is diastolic arterial pressure13] in millimeters of mercury, CO is the cardiac output (cc/min), and 0.0136 is the conversion factor from millimeters of mercury times cubic centimeters per minute to gram-meters per minute.

Left ventricular external efficiency was estimated with the following equation16:

\[
\text{Efficiency} = \frac{\text{LV external work}}{\text{MVO}_2}
\]

MVO2 was converted into calories (5 calories/ml O2), and kilocalories were converted into kilogram-meters (0.425 kg-calorie). Since MVO2 was derived from a sample from the regional great cardiac vein while myocardial work was calculated from the work of the entire left ventricle, this analysis can be applied only to relative changes in external efficiency before and after milrinone, and therefore only the changes in efficiency induced by milrinone relative to the baseline value obtained in each patient are presented.

Paired baseline hemodynamic measurements were made as described above. Baseline data are reported as the mean of the two consecutive hemodynamic measurements differing by less than 10%. Incremental boluses of milrinone (12.5, 25, 50, and 75 \(\mu\)g/kg) were administered at 10 min intervals and the studies described above were repeated 5 min after the final dose was given.

Data analysis and statistical comparisons were performed on Analyzer, a Hewlett-Packard 9845–based data analysis system, with paired t test analysis of measurements obtained before and after administration of milrinone. Data from patients with and without coronary disease were also analyzed separately and compared by an analysis of variance with a comparison of differences (unpaired t test).

Results

After intravenous milrinone (125 ± 36 \(\mu\)g/kg) there was a 45% increase in cardiac index (2.1 ± 0.5 to 3.0 ± 0.6 liters/min/m2; \(p = .0001\)), a 37% increase in stroke volume index (46 ± 15 to 63 ± 23 ml; \(p = .0004\)), a 39% fall in the pulmonary capillary wedge pressure (28 ± 8 to 17 ± 8 mm Hg; \(p = .0001\)), and a 38% fall in the right atrial pressure (13 ± 6 to 8 ± 4 mm Hg; \(p = .0001\) (table 1)). There was an 8% increase in heart rate (85 ± 15 to 92 ± 17 beats/min; \(p = .0001\)), an 11% fall in peak systolic arterial pressure (114 ± 19 to 102 ± 16 mm Hg; \(p = .001\)), and a 13% fall in the mean arterial pressure (84 ± 11 to 73 ± 11 mm Hg; \(p = .0001\)).

Because of these small but opposing changes in heart rate and arterial pressure, there was no significant change in the heart rate–blood pressure product (9624 ± 2272 to 9380 ± 2428 mm Hg-beats/min; \(p = \text{NS}\)). Similarly, there was no significant change in MVO2.
After milrinone (7.6 ± 2.9 to 8.1 ± 3.1 ml O₂/min; p = NS), left ventricular stroke work index increased by 31% (26 ± 11 to 34 ± 12 g-m/beat/m²; p = .002) and left ventricular external work per minute increased 42% (3758 ± 1419 to 5340 ± 1598 g-min/m; p = .0001), yielding a 45% increase in calculated left ventricular external efficiency (p = .004; table 1).

Despite the absence of a significant change in MVO₂,
for the group as a whole, there was a 16% increase in great cardiac vein flow (73 ± 32 to 85 ± 34 ml/min; p = .02). This occurred despite the 13% fall in mean aortic pressure, and was associated with a 30% fall in coronary vascular resistance (1.32 ± 0.99 to 0.93 ± 0.54 mm Hg-min/ml; p = .004). Similar reductions in coronary vascular resistance were observed when mean arterial minus capillary wedge pressure was used in place of mean arterial minus right atrial pressure as the transcoronary driving pressure. This 30% decline in coronary vascular resistance was comparable to the 37% fall observed in systemic vascular resistance (1606 ± 426 to 1016 ± 286 dynes-sec-cm²; p = .001), and to the 38% fall observed in pulmonary vascular resistance (288 ± 147 to 178 ± 72 dynes-sec-cm²; p = .001) after administration of milrinone.

The increase in great cardiac vein flow at an essentially unchanged myocardial oxygen demand resulted in an 11% decline in the transcoronary arterial-venous oxygen difference (111 ± 24 to 99 ± 21 ml O₂/liter; p = .0001), a finding that was observed in all but one of the subjects in whom the difference was unchanged.

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**TABLE 1**

(Continued)

<table>
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<tr>
<th>LV ext. work</th>
<th>MVO₂ (ml O₂/min)</th>
<th>ΔEffíc.</th>
<th>CSF</th>
<th>A-CS O₂</th>
<th>CVR</th>
<th>CVR'</th>
<th>SVR</th>
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3758 ± 1419  7.6 ± 2.9  73 ± 32  111 ± 24  1.32 ± 0.99  1.03 ± 0.79  1,606 ± 426  288 ± 147
5340 ± 1598  8.1 ± 3.1  45 ± 56  85 ± 34  99 ± 21  0.93 ± 0.54  0.80 ± 0.50  1,016 ± 286  178 ± 72

.0001 NS .004 .02 .0001 .004 .02 .0001 .0001
No patient developed either angina or ST changes suggestive of ischemia while receiving milrinone.

Discussion

An understanding of the effects of inotropic drugs on MVO₂ is important to their safe use in patients with refractory congestive heart failure, many of whom have a reduction in coronary reserve due to coronary atherosclerosis and/or elevation of resting myocardial oxygen demand because of myocardial hypertrophy and increased wall stress. In such patients, any further increase in myocardial oxygen requirements may exceed the coronary vasodilator reserve and precipitate myocardial ischemia, with potential exacerbation of underlying myocardial dysfunction and arrhythmias.

Experimental studies in a variety of animal preparations have demonstrated that MVO₂ is determined by several variables, including heart rate, myocardial contractility, and systolic wall stress, the last of which is in turn a function of systolic pressure, chamber diameter, and wall thickness. In studying pharmacologic agents that may have multiple and partially opposing actions on these variables, it is difficult to predict net changes in MVO₂ from simple indexes such as external cardiac work, the heart rate–systolic pressure product, or even the tension-time index, all of which are insensitive to changes in contractility and chamber dimension. Moreover, the effect of an inotropic drug may depend critically on the hemodynamic condition of the subject being tested. In normal subjects positive inotropic drugs such as the digitalis glycosides may increase MVO₂, while in patients with congestive heart failure the effects of increases in contractility on stimulation of MVO₂ may be offset by reductions in chamber diameter and, therefore, wall stress. Thus, the effect of a new inotropic drug on myocardial energetics must be assessed directly in patients with congestive heart failure.

Milrinone, a derivative of the earlier bipyridine amrinone, is a new agent for the therapy of refractory congestive heart failure. Its mechanism of action appears to be related at least in part to phosphodiesterase inhibition and a consequent increase in intracellular cyclic AMP, which results in improved calcium handling (uptake, storage, and release) by the sarcoplasmic reticulum during excitation-contraction coupling, presumably due to phosphorylation of the sarcoplasmic reticular protein phospholamban. Because its inotropic effect exceeds that observed with more potent phosphodiesterase inhibitors such as the methylxanthenes, additional direct effects on transmembrane calcium fluxes seem likely, and a stimulatory effect of milrinone on calcium ATPase has been demonstrated. There is no evidence that milrinone exerts its salutary effects by conventional inotropic mechanisms such as stimulation of β-adrenergic or histamine receptors or inhibition of sodium-potassium ATPase.

The significant beneficial effects of milrinone on systemic hemodynamics in patients with advanced congestive heart failure are associated with complex and partially opposing actions on the determinants of MVO₂. In the current study there was no significant change in the heart rate–blood pressure product because of modest offsetting increases in heart rate and reductions in systolic arterial pressure. As an inotropic drug, milrinone causes a significant increase in myocardial contractility that is reflected in increases in peak positive dP/dt and in afterload-independent increases in the slope of the left ventricular end-systolic pressure-dimension relationship. Since contractility is a major determinant of MVO₂, this would be expected to increase oxygen consumption.

Milrinone, however, is also a potent vasodilator. By reducing systemic arteriolar tone, the drug lowers peak developed left ventricular systolic pressure, and thereby tends to reduce systolic wall stress. Although left ventricular end diastolic volume does not fall significantly after the administration of milrinone, the increase in left ventricular stroke volume observed after it is administered is associated with a fall in left ventricular end-systolic volume, and thereby a further reduction in mean left ventricular systolic wall stress. This reduction in systolic wall stress may offset the increase in MVO₂ that would otherwise be anticipated with increased contractility.

The near balance between factors tending to augment or reduce MVO₂ is reflected by the fact that MVO₂ did not change significantly for the group as a whole after administration of milrinone. However, there was some patient-to-patient variability: MVO₂ rose in 11 patients (by ≥30% in four patients) and fell in the remaining seven patients. These individual changes in MVO₂ did not correlate closely with the changes in the heart rate–blood pressure product after milrinone (r = .48), attesting to the importance of the nonrate-pressure determinants of MVO₂ in patients receiving agents with complex effects on afterload and contractility, and there was no difference between patients with and without coronary artery disease (figure 1).

Taking into consideration the relative constancy of MVO₂ and the marked increase in external left ventricular work after milrinone, there was a 45% increase in
FIGURE 1. The percent change (Δ) in MVO₂ after administration of milrinone in patients with coronary artery disease (CAD), patients with coronary artery disease who have undergone coronary artery bypass graft surgery (CAD + CABG), and patients with dilated cardiomyopathy and normal coronary arteries (CMP). There were no significant differences between patients with and without coronary artery disease (see text).

FIGURE 2. The percent change (Δ) in O₂ supply (arterial oxygen content times great cardiac vein blood flow) compared with the changes in MVO₂ (according to the format of Mohrman and Feigl10). After milrinone there was a significant (p = .0001) upward shift in this relationship relative to the line of identity (the dotted line), reflecting increased oxygen delivery above that required to meet the changes in oxygen consumption due to a primary coronary vasodilator effect of milrinone. In contrast, increases in blood flow resulting from autoregulation alone would lead to changes in oxygen supply and demand falling along the line of identity.

calculated external efficiency, i.e., the quantity of external work performed per unit oxygen consumed. Afterload reduction in the dilated and failing heart might shift the myocardial pressure-efficiency relationship to a more optimal level.27 Indeed, similar increases in efficiency have been demonstrated after administration of pure vasodilators.28,29 However, we cannot exclude the possibility that a milrinone-induced increase in true cellular efficiency was a contributing factor to our current observations.

One observation of particular importance was the increase in great cardiac vein blood flow despite a reduction in arterial pressure and a relatively constant MVO₂. This increase in great cardiac vein blood flow resulted from a 30% fall in calculated coronary vascular resistance, and appeared to be the result of primary rather than secondary (i.e., autoregulatory12) vasodilation, since it was accompanied by a reduction in the transcoronary arterial-venous oxygen difference (figure 2).30 Moreover, the fall in coronary vascular resistance was similar to the fall in systemic and pulmonary vascular resistances seen after administration of milrinone, suggesting a direct vasodilator action in all three vascular beds.

Since coronary blood flow is influenced by left ventricular diastolic pressure11,12 according to the vascular waterfall model, another explanation for the apparent primary coronary vasodilatation could be a reduction in the zero-flow coronary pressure (P_{r=0})31 after administration of milrinone. This is of particular interest in view of the recent demonstration that milrinone appears to improve left ventricular diastolic relaxation and compliance in patients with heart failure.26 Although there is no way to measure P_{r=0} in intact human subjects, we attempted to account for its influence by calculating coronary vascular resistance using the difference between mean systemic arterial pressure and pulmonary arterial wedge pressure (rather than that between mean arterial pressure and right atrial pressure, or mean arterial pressure alone) as the “driving pressure” for coronary blood flow. A reduction in calculated coronary vascular resistance after milrinone was evident with each formulation. Similar coronary vasodilation has been observed with milrinone in a canine heart-lung preparation.*

The clinical significance of this primary coronary vasodilator effect is unclear. A reduction in intramyocardial diastolic pressure may improve subendocardial

*Farah AE: Personal communication.
blood flow in patients whose coronary flow reserve is compromised by marked hypertrophy and wall stress, with or without significant associated epicardial coronary atherosclerosis. On the other hand, excessive primary arteriolar vasodilation in the setting of restricted coronary inflow could produce ischemia via a "coronary steal" mechanism.32, 33 This latter effect is unlikely given the magnitude of milrinone-induced coronary vasodilation, and we have noted no clinical or electrocardiographic evidence of myocardial ischemia precipitated by short-term milrinone infusion, even in the four patients with a greater than 30% increase in MVO$_2$ after milrinone. It should be noted, however, that three of these four patients had normal coronary arteries and the fourth had undergone coronary artery bypass graft surgery; if this increase had occurred in a patient with marginal coronary flow reserve, ischemia might have developed or been exacerbated. A similar increase in MVO$_2$ may have contributed to the increase in angina observed during long-term oral milrinone therapy in three of our previously reported patients,1 but this appeared to result primarily from increased activity made possible by the improvement in heart failure.

The effects of intravenous milrinone on myocardial energetics compare favorably with those of other inotropic and/or vasodilating drugs used in the therapy of heart failure. Digitalis glycosides reduce left ventricular diastolic pressures in animal preparations of heart failure and potentially improve subendocardial blood flow; concurrent reductions in left ventricular systolic chamber size tend to reduce MVO$_2$. However, more potent inotropic drugs such as dobutamine tend to increase MVO$_2$ in patients with congestive heart failure,35 and may result in myocardial ischemia in patients with limited coronary reserve.36 In contrast, pure vasodilators such as nitroprusside,34 nitroglycerin,37 and captopril38, 39 reduce systolic wall stress and thereby MVO$_2$, while also reducing left ventricular diastolic pressures and potentially improving subendocardial blood flow.38 Amrinone, the parent compound of milrinone, appears to cause a slight decrease in MVO$_2$ in patients with congestive heart failure,39 and has been shown to reduce MVO$_2$ in one animal preparation of acute, ischemia-mediated left ventricular failure.40 In a second animal preparation without heart failure, however, amrinone worsened ischemia in the zone of a transiently occluded coronary artery.41 While the effects of amrinone and milrinone in patients with congestive heart failure are similar, small apparent differences may reflect differences in the balance between the positive inotropic and vasodilator activities of these two drugs.

There are several potential limitations to this study. First, regional measurements of flow and oxygen content in the great coronary vein may not be representative of energetics in the entire left ventricle, particularly in patients with coronary artery disease. This site, rather than the coronary sinus, was selected because it provided greater catheter stability and avoided contamination of data by right atrial reflux. Furthermore, parallel changes in MVO$_2$ would be expected in all noninfarcted territories after milrinone, whether or not coronary artery disease was present, and there was no difference in results when the data on patients with and without coronary artery disease were analyzed separately. Second, we did not evaluate myocardial lactate metabolism, which may have shown evidence of ischemia despite constant MVO$_2$ and increased regional coronary blood flow. Third, our overall findings do not preclude individual differences in response, and may not predict the responses of patients with milder degrees of myocardial dysfunction, of patients with acute myocardial infarction, or of patients receiving long-term oral milrinone.

In summary, we have demonstrated that the substantial improvements in hemodynamics that follow the short-term administration of milrinone to patients with refractory heart failure are not associated with a significant increase in MVO$_2$, despite the increase in contractility and the substantial increase in the external work of the left ventricle. This appears to be due to milrinone’s vasodilator activity, which causes a compensating reduction in MVO$_2$. Coronary blood flow increases, and this is associated with a decline in coronary vascular resistance and a fall in myocardial oxygen extraction, which appears to represent a primary arteriolar vasodilator action of milrinone. Thus, milrinone can generally improve the condition of the patient with advanced congestive heart failure without worsening the balance between myocardial oxygen supply and demand, although some individual variability in response is evident. Since milrinone has not been studied in patients with ischemic heart disease and milder heart failure or in patients with acute myocardial infarction,continued caution must be advised in these situations.

We are indebted to the technical and nursing staffs of the cardiac catheterization laboratory, and to Dr. Bernard J. Ransil for his assistance with the statistical analysis.

References


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