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Uri Elkayam, Tien M.H. Ng, Parta Hatamizadeh, Munir Janmohamed and Anilkumar Mehra

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Renal Vasodilatory Action of Dopamine in Patients With Heart Failure
Magnitude of Effect and Site of Action

Uri Elkayam, MD; Tien M.H. Ng, PharmD; Parta Hatamizadeh, MD; Munir Janmohamed, MD; Anilkumar Mehra, MD

Background—A “renal dose” of dopamine is often used to increase renal blood flow; however, data on the magnitude of effect and site of action in patients with heart failure are scarce.

Methods and Results—Renal effects of intravenous dopamine (1, 2, 3, 5, and 10 μg·kg⁻¹·min⁻¹) were evaluated in 13 patients with chronic heart failure. Renal blood flow was calculated from renal artery cross-sectional area measured with intravascular ultrasound and renal blood flow velocity-time integral measured by the intravascular Doppler technique. Cross-sectional area increased and was significantly higher than baseline (0.30±0.04 cm²) at 5 μg·kg⁻¹·min⁻¹ (0.36±0.05 cm²) and 10 μg·kg⁻¹·min⁻¹ (0.38±0.06 cm²). The velocity-time integral was significantly higher than baseline (22±3 cm) at doses of 3 and 5 μg·kg⁻¹·min⁻¹ (both 31±4 cm). Renal blood flow increased, whereas renal vascular resistance decreased, reaching statistical significance at 2 μg·kg⁻¹·min⁻¹ through 10 μg·kg⁻¹·min⁻¹. Cardiac output gradually increased, reaching statistical significance at doses of 5 and 10 μg·kg⁻¹·min⁻¹ (5.5±0.5 and 6.1±0.7 versus 4.5±5.2 L/min at baseline), but the increase in renal blood flow appeared proportionately larger than corresponding increases in cardiac output.

Conclusions—Dopamine is associated with an increase in renal blood flow in patients with heart failure. This effect is due to dilatation of both the large conductance and small resistance renal blood vessels. Further evaluation of the efficacy and safety of dopamine for improvement of renal function in hospitalized patients with heart failure is warranted.

(Circulation. 2008;117:200-205.)

Key Words: heart failure ■ kidney ■ inotropic agents ■ vasodilation ■ blood flow ■ pharmacology

Decreased renal function during hospitalization in patients admitted for heart failure (HF) is common and has been associated with unfavorable outcomes. Further worsening of renal function during treatment of decompensated heart failure, which has been defined as the cardiorenal syndrome, occurs in a substantial number of patients and leads to undesirable consequences, including prolongation of length of stay, increased morbidity, and increased mortality after discharge. Management of the cardiorenal syndrome is difficult because of the lack of effective therapies.

Clinical Perspective p 205

Although renal dysfunction is complex and determined by a multiplicity of factors, reduction in renal blood flow (RBF), an important determinant of glomerular filtration rate, may play an important role. Enhancement of RBF in patients with decompensated HF has been challenging, and commonly used vasoactive drugs, including nesiritide, nitroglycerin, nitroprusside, and dobutamine, have failed to demonstrate a consistent effect in spite of a considerable increase in cardiac output (CO). Dopamine has been reported both in experimental animals and in a variety of patient populations to enhance RBF by multiple mechanisms, including an increase in CO and local vasodilation due to stimulation of dopamine receptors in renal blood vessels. For many years, a small dose of dopamine often has been used by clinicians to enhance RBF in critically ill patients; however, this selective effect of dopamine on the renal circulation may be eliminated or markedly attenuated by other vasoconstrictive mechanisms known to be activated in patients with HF.

In addition, the magnitude of its renal vasodilatory effects and site of action in patients with HF have not been clearly defined. The introduction of intravascular ultrasound (IVUS) and Doppler techniques provides an opportunity for further insight, because these techniques allow for an accurate assessment of vascular dimensions and blood flow velocity.
and can be used for evaluation of vasodilatory changes in both the conductance (IVUS) and resistance (Doppler) renal blood vessels.\textsuperscript{15-17} The purpose of the present study, therefore, was to gain further insight into the renal circulatory effect of dopamine in patients with HF using the above-mentioned techniques.

**Methods**

**Study Population**

Patients with a history of chronic HF due to left ventricular systolic dysfunction with moderate to severe HF symptoms (New York Heart Association functional class III or IV) who were undergoing diagnostic cardiac catheterization were included in the study if they agreed and signed a consent form approved by the ethics committee of the Los Angeles County/University of Southern California Medical Center. All patients were undergoing standard therapy for HF.

**Measurements of RBF**

Changes in RBF were measured in the present study with IVUS and Doppler techniques. These methods were initially validated and used to evaluate coronary anatomy and assess coronary blood flow reserve.\textsuperscript{18,19} More recently, these techniques have also been validated for the measurement of RBF\textsuperscript{20-22} and have been found to provide accurate and reproducible results when used to measure changes in RBF in response to various vasoactive drugs and other vasmotor stimuli in patients with hypertension and renal artery stenosis.\textsuperscript{22,23}

Our group has used the technique successfully in the assessment of changes in RBF during various therapeutic interventions in patients with HF.\textsuperscript{16,17}

**Study Protocol**

Premeedication before cardiac catheterization included intravenous metoclopramide (10 mg) and diphendhydramine (30 mg). Nitroglycerin (sublingual, intravenous, or intracoronary) was not allowed throughout the study. A triple-lumen Swan-Ganz catheter was used for the performance of right heart catheterization. An 8F angioplasty multipurpose catheter or Judkins right coronary artery guiding catheter was placed in the renal artery with the tip positioned in the proximal or middle portion of the main artery. Catheter position in the renal artery was confirmed with a small amount (\textasciitilde{}3 mL) of angiographic contrast medium (Hexabrix, Mallinckrodt Medical, Hazelwood, Mo.) A 0.00018-in (0.045-mm) Doppler guidewire (Flowwire, Cardiometrics, Inc, Mountain View, Calif) was then introduced into the guiding catheter through a valved sidearm connector, and its tip was positioned under direct visualization in the main renal artery or 1 of its branches and manipulated to maintain a parallel position to flow and to record the highest possible Doppler flow signal. The wire was then locked to secure a constant position throughout the study. A commercially available 3.5F or 4.3F IVUS catheter (Mansfield, Boston Scientific Corp, Natick, Mass) was then placed through the guiding catheter and positioned under fluoroscopic guidance next to the Doppler wire. Images were obtained with a commercially available intracoronary ultrasound imaging system (Sono Intravascular, Hewlett-Packard, Palo Alto, Calif) at 30 frames per second and recorded on 0.5-in high-quality super VHS videotape for subsequent offline analysis. Renal artery lumen planimetry was performed later from the taped images, and renal artery cross-sectional area (CSA) was determined with specially designed software. Spectral Doppler RBF velocities were recorded. The velocity-time integral (VTI) was measured as the area under the outermost portion of the spectral-velocity envelope. To correct for changes related to respiration and cardiac cycle, 15 to 30 beats were used for VTI, and average values are reported. Renal artery blood flow (mL/min) was calculated as the product of the CSA in the renal artery in which the signal was recorded and the VTI, with the following formula: flow = heart rate (per minute) × VTI × CSA. Renal artery blood flow index was calculated for each patient by dividing RBF by the patient’s body surface area, and renal vascular resistance (RVR) was calculated as mean renal artery blood pressure/RBF. Renal arterial pressures were measured directly with the aid of the arterial guiding catheters, fluid-filled pressure tubing, and standard transducers.

After baseline measurements, which included hemodynamic variables, IVUS, and Doppler flow velocity, serial escalating intravenous infusions of dopamine at 1, 2, 3, 5, and 10 \( \mu \text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1} \) were begun in each patient for 3- to 5-minute intervals at each dose. Hemodynamic measurements, IVUS, and Doppler flow velocity recordings were obtained at the end of each infusion interval.

**Statistical Analysis**

Repeated-measures ANOVA was used to compare statistical differences in the various measured or calculated parameters with varying dopamine dose. Significance was determined with the Newman-Keuls post hoc test. The relationship between percentage increase in cardiac index and that of renal artery blood flow index and between the percentage increase in VTI and that of CSA was assessed at each dose of dopamine compared with the baseline by use of the Wilcoxon signed rank test. Results are expressed as mean±SD. Probability values <0.05 were considered statistically significant.

The authors had full access to the data and take full responsibility for its integrity. All authors have read and agree to the manuscript as written.

**Results**

**General Information**

Thirteen patients were included in the study. There were 10 men and 3 women (22 to 67 years old). The underlying cause of HF was coronary artery disease in 5 patients and nonischemic, dilated cardiomyopathy in 8. All patients had severe depression of left ventricular systolic function as documented by contrast or radionuclide ventriculography or by echocardiography (3 patients). Left ventricular ejection fraction ranged from 14% to 32% (mean 24±2%). Medical therapy included diuretics in all patients, ACE inhibitors in 10, digoxin in 9, \( \beta \)-blocking agents in 7, and organic nitrates in 7. Mean baseline hemodynamic values as measured during cardiac catheterization were as follows: heart rate 86±5 bpm, mean blood pressure 92±4 mm Hg, right atrial pressure 8±1 mm Hg, mean pulmonary artery pressure 31±3 mm Hg, pulmonary artery wedge pressure 20±2 mm Hg, cardiac index 3.3±0.2 L·min\(^{-1}\)·m\(^{-2}\), stroke volume index 39±4 mL·min\(^{-1}\)·m\(^{-2}\), and systemic vascular resistance 1158±21 dyne·s\(^{-1}\)·cm\(^{-5}\).

**Systemic Hemodynamics**

The dose-dependent systemic hemodynamic effects of dopamine are summarized in the Table. There was no significant change in heart rate, blood pressure, or right atrial pressure at any dopamine dose used in the present study. Mean pulmonary artery pressure showed no significant response to doses between 1 and 5 \( \mu \text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1} \) but was significantly increased from 32±4 mm Hg at baseline to 40±4 mm Hg during the infusion of 10 \( \mu \text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1} \). CO demonstrated a gradual increase, which reached statistical significance compared with baseline at doses of 5 and 10 \( \mu \text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1} \) (5.5±0.5 and 6.1±0.7 versus 4.5±5.2 L/min at baseline). Calculated systemic vascular resistance was reduced significantly compared with baseline at all dopamine doses.
Renal Hemodynamics

The renal hemodynamic effects of dopamine are summarized in the Table. There were no significant differences in renal artery pressure with dopamine at any dose. Mean renal artery CSA (Figure 1A) was 0.30±0.04 cm² at baseline before the administration of dopamine and demonstrated a progressive increase with increasing dopamine dose, reaching statistical significance, compared with baseline, at 5 μg·kg⁻¹·min⁻¹ (0.36±0.05 cm²) and at 10 μg·kg⁻¹·min⁻¹ (0.38±0.06 cm²).

Table. Dose-Dependent Systemic and Renal Hemodynamics of Dopamine

<table>
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<td>HR bpm</td>
<td>90±4</td>
<td>93±5</td>
<td>93±5</td>
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<td>SBP mm Hg</td>
<td>126±7</td>
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<td>121±8</td>
<td>125±8</td>
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<td>DBP mm Hg</td>
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<td>76±7</td>
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<tr>
<td>MBP mm Hg</td>
<td>96±5</td>
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<td>94±5</td>
<td>97±5</td>
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<td>RAP mm Hg</td>
<td>10±1</td>
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<tr>
<td>MPAP mm Hg</td>
<td>32±4</td>
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<td>34±4</td>
<td>34±4</td>
<td>40±5*</td>
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<tr>
<td>CO L/min</td>
<td>4.5±0.6</td>
<td>5.2±0.6</td>
<td>5.0±0.3</td>
<td>5.0±0.5</td>
<td>5.5±0.5*</td>
<td>6.1±0.7†</td>
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<tr>
<td>CI L·min⁻¹·m⁻²</td>
<td>2.5±0.4</td>
<td>2.9±0.4</td>
<td>2.8±0.2</td>
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<td>3.1±0.3</td>
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<td>SV mL</td>
<td>52±8</td>
<td>58±8</td>
<td>56±4</td>
<td>62±7</td>
<td>63±7</td>
<td>66±8†</td>
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<td>SVI mL/m²</td>
<td>29±5</td>
<td>35±5</td>
<td>31±3</td>
<td>34±4</td>
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<tr>
<td>VTI cm</td>
<td>22±3</td>
<td>25±3</td>
<td>28±5</td>
<td>31±4</td>
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<td>28±4</td>
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<tr>
<td>CSA cm²</td>
<td>0.30±0.04</td>
<td>0.32±0.04</td>
<td>0.34±0.04</td>
<td>0.35±0.05</td>
<td>0.36±0.05*</td>
<td>0.38±0.06†</td>
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<tr>
<td>RBF mL/min</td>
<td>558±61</td>
<td>677±84</td>
<td>762±94*</td>
<td>831±103*</td>
<td>879±92*</td>
<td>898±131*</td>
</tr>
<tr>
<td>RBFi mL/min</td>
<td>308.2±32.6</td>
<td>373.8±46.2</td>
<td>420.0±50.0</td>
<td>461.7±59.1*</td>
<td>488.3±53.8*</td>
<td>501.1±82.0*</td>
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<tr>
<td>RVR dyne·s⁻¹·cm⁻²</td>
<td>13615±1317</td>
<td>11681±1384</td>
<td>10200±1310*</td>
<td>10018±1324*</td>
<td>9540±1269*</td>
<td>9408±1620*</td>
</tr>
<tr>
<td>SVR dyne·s⁻¹·cm⁻²</td>
<td>1734±180</td>
<td>1444±137*</td>
<td>1417±129*</td>
<td>1402±122*</td>
<td>1431±131*</td>
<td>1326±200*</td>
</tr>
</tbody>
</table>

HR indicates heart rate; SBP, systolic blood pressure; DBP, diastolic blood pressure; MBP, mean arterial blood pressure; RAP, renal artery pressure; MPAP, mean pulmonary artery pressure; CI, cardiac index; SV, stroke volume; SVI, stroke volume index; RBFi, renal blood flow index; and SVR, systemic vascular resistance.

*P<0.05 vs baseline (0.0 μg·kg⁻¹·min⁻¹).
†P<0.05 vs 1 μg·kg⁻¹·min⁻¹.

Failure of this parameter to change significantly with lower doses may be related to a type II error due to the relatively small number of patients.

The mean value of VTI (Figure 1B) also showed an increase and was significantly higher than baseline (22.3 cm) at doses of 3 and 5 μg·kg⁻¹·min⁻¹ (both 31.4 cm). An increase in dopamine dose to 10 μg·kg⁻¹·min⁻¹ resulted in a reduction in VTI to 28.4 cm, a value not statistically different from baseline. The percentage increase in VTI, significant difference between values recorded at 2 to 10 μg·kg⁻¹·min⁻¹. RABF indicates renal artery blood flow. D, RVR and dopamine dose. RVR was lower than baseline at all doses of dopamine. The fall in RVR reached statistical significance at 2 through 10 μg·kg⁻¹·min⁻¹. There was no statistically significant difference in mean values of RVR between 2 and 10 μg·kg⁻¹·min⁻¹. *P<0.05.
compared with baseline, was generally greater than the corresponding increase in CSA, especially at 3, 5, and 10 μg · kg⁻¹ · min⁻¹. This difference, however, was not statistically significant.

The increase in this parameter started early and became apparent vasoconstriction, possibly because of the short duration of the infusion. The dopamine-mediated increase in RBF has been reported to be multifactorial and includes renal vasodilatation due to activation of dopamine receptors, β-receptors, and α-receptors. Generally, at doses <3 μg · kg⁻¹ · min⁻¹, dopamine has been found to activate dopamine A1 receptors, which cause vasodilation of the renal arteries and other vascular beds, including mesenteric, coronary, and cerebral beds. In addition, there is stimulation of dopamine A2 receptors that leads to inhibition of norepinephrine release from sympathetic nerve endings. Activation of dopamine A1 and A2 receptors also results in a decline in systemic vascular resistance and an increase in RBF, which was observed in the present study. Dopamine infused at approximately 3 to 5 μg · kg⁻¹ · min⁻¹ activates β1- and β2-adrenergic receptors, conferring a positive inotropic effect that is responsible for the increase in CO. At a dose >5 μg · kg⁻¹ · min⁻¹, dopamine has been reported to exert clinically relevant activation of α1- and α2-adrenergic receptors, which results in arterial vasoconstriction. In the present study, however, the use of 10 μg · kg⁻¹ · min⁻¹ did not result in apparent vasoconstriction, possibly because of the short duration of the infusion.

The dopamine-mediated increase in RBF has been reported to be multifactorial and includes renal vasodilatation due to activation of dopamine receptors in the renal vasculature, augmentation of CO due to β-adrenergic activity that leads to both inotropic and chronotropic effects, and peripheral vasodilatation mediated by stimulation of dopamine receptors in the systemic vasculature. The present study provides information regarding the magnitude and site of dopamine action in the renal circulation. Intravenous infusion of dopamine resulted in an early vasodilatory effect on the small resistance renal arteries, as reflected by a significant fall in RVR and an increase in RBF velocity. At the same time, a gradual dose-dependent increase in renal artery CSA indicated a

**Discussion**

The findings of the present study provide new insight into the magnitude and site of action of the renal vasodilatory effect of dopamine and the resulting renal circulatory effects in patients with HF treated with standard HF therapy, including diuretics, neurohormonal blockers, and vasodilators. The present findings show a marked effect of dopamine on RBF. The increase in this parameter started early and became statistically significant at a dose as low as 2 μg · kg⁻¹ · min⁻¹. RBF continued to rise, achieving its highest value at 10 μg · kg⁻¹ · min⁻¹, which was the maximum dose used in the present study. Although the difference between RBF values at a dose of 2 μg · kg⁻¹ · min⁻¹ and higher doses did not reach statistical significance, this may be due to the limited number of patients included in the study. On an individual basis, the present data also demonstrated a high interpatient variability, with a number of subjects achieving maximum response at doses greater than 1 to 3 μg · kg⁻¹ · min⁻¹, a dose range traditionally used by clinicians to augment RBF. These findings support previous reports indicating that the dose of dopamine that produces the maximal increase in RBF in patients with HF is higher than the dose commonly thought of as the low “renal” dose.

The present results reflect the known pharmacology of dopamine, which has been shown to exhibit a graded pharmacological response, with a dose-dependent predominant activation of dopaminergic receptors, β-receptors, and α-receptors. Generally, at doses <3 μg · kg⁻¹ · min⁻¹, dopamine has been found to activate dopamine A1 receptors, which cause vasodilation of the renal arteries and other vascular beds, including mesenteric, coronary, and cerebral beds. In addition, there is stimulation of dopamine A2 receptors that leads to inhibition of norepinephrine release from sympathetic nerve endings. Activation of dopamine A1 and A2 receptors also results in a decline in systemic vascular resistance and an increase in RBF, which was observed in the present study. Dopamine infused at approximately 3 to 5 μg · kg⁻¹ · min⁻¹ activates β1- and β2-adrenergic receptors, conferring a positive inotropic effect that is responsible for the increase in CO. At a dose >5 μg · kg⁻¹ · min⁻¹, dopamine has been reported to exert clinically relevant activation of α1- and α2-adrenergic receptors, which results in arterial vasoconstriction. In the present study, however, the use of 10 μg · kg⁻¹ · min⁻¹ did not result in apparent vasoconstriction, possibly because of the short duration of the infusion.

The dopamine-mediated increase in RBF has been reported to be multifactorial and includes renal vasodilatation due to activation of dopamine receptors in the renal vasculature, augmentation of CO due to β-adrenergic activity that leads to both inotropic and chronotropic effects, and peripheral vasodilatation mediated by stimulation of dopamine receptors in the systemic vasculature. The present study provides information regarding the magnitude and site of dopamine action in the renal circulation. Intravenous infusion of dopamine resulted in an early vasodilatory effect on the small resistance renal arteries, as reflected by a significant fall in RVR and an increase in RBF velocity. At the same time, a gradual dose-dependent increase in renal artery CSA indicated a
concomitant vasodilatory effect of dopamine on the large
conductance blood vessels, especially with the use of larger
(5 and 10 μg · kg⁻¹ · min⁻¹) doses, which may also contribute
to an increase in RBF. This increase in CSA may be due to
the direct effect of dopamine but could also represent a
flow-mediated, endothelium-derived vasodilation.

An increase in CO with either vasodilators or inotropes is
often assumed by clinicians to augment RBF in hospitalized
patients with acute decompensated HF; however, a marked
increase in CO with drugs such as nitroglycerin, nitroprusside,
and nesiritide has resulted in small or even no effect on
RBF.²⁸ The present study demonstrates a dopamine-
mediated increase in both CO and RBF. The change in RBF,
however, appeared to be proportionally larger than the change
in CO, which suggests that dopamine-mediated renal vaso-
regulation in patients with HF is predominantly due to its
local vascular action rather than its central hemodynamic
effect.

Study Limitations
In the present study, we used the antiemetic drug metoclo-
pramide for premedication before cardiac catheterization.
Metoclopramide possesses a dopamine A2 receptor antago-
nistic effect and has been shown to slightly attenuate the
effect of dopamine on renal plasma flow in healthy volunteers
but not in patients with renal failure²⁷ and only at high doses
in patients receiving chemotherapy.²⁸ Although no informa-
tion is available on the effect of metoclopramide on RBF at
the dose used in the present study in patients with heart
failure, it is possible that the effect of dopamine on RBF was
somewhat attenuated by metoclopramide.

In addition, the dose of dopamine was increased at 3- to
5-minute intervals without washout periods between the
doses. Although the half-life of the drug is relatively short, a
carryover effect of the previous dose to the next, and thus
augmentation of effect, cannot be excluded. At the same time,
however, it is possible that the relatively short duration of
infusion of each dose did not allow enough time for equili-
bration and achievement of maximum effect.

The method used in the present study was not designed to
accurately measure total RBF or RVR but rather to assess the
relative changes in these parameters. Because it is not always
possible to position the tip of the Doppler wire in the main
renal artery owing to early artery division, and because
positioning of the flow wire absolutely parallel to blood flow
is not always possible, especially in larger vessels such as the
renal artery, the method used here is likely to often underes-
timate total RBF. However, this method provides accurate
and reproducible information and is highly suitable for
assessment of changes in renal hemodynamics, which are
likely to be proportional to the change in total RBF.¹⁵,²²,²³ The
study was performed in clinically stable HF patients as part of
diagnostic cardiac catheterization, with an unknown effect of
contrast used on the vascular effect of dopamine. For all these
reasons, the present findings may not be completely applica-
table to patients with acute decompensated HF who are treated
with renal-dose dopamine.

The present study was also limited to evaluation of the
magnitude and site of vasodilatory action of various doses of
dopamine on the renal vasculature and was not designed to
assess renal function. Although RBF is an important deter-
minant of glomerular filtration rate,³ more information will be
needed to determine whether the rise in RBF documented in
the present study may also influence parameters of renal
function in patients with HF. In addition, the study cannot
provide safety information related to a potential effect of the
drug on either short- or long-term outcome. Tachycardia,
cardiac arrhythmias, myocardial ischemia, and infarction are
well-documented potential side effects of dopamine. Blunting
of hypoxic ventilatory drive, increasing pulmonary shunt
fraction, and gut ischemia have also been described in
critically ill patients receiving dopamine.²⁹ Recent studies
have demonstrated a detrimental effect of other inotropes
used for the hemodynamic improvement in hospitalized
patients with HF on both short- and long term outcome.³⁰,³¹

In summary, the present study demonstrated a significant
increase in RBF with intravenous dopamine in patients with
HF. This effect was due to dilatation of both the large
conductance renal blood vessels and the small resistance
vessels, although the latter appeared to be affected earlier.
The increase in RBF appeared proportionally larger than the
increase in CO, which indicates that augmentation of flow is
mainly due to reduction in RVR mediated by the local
vasodilatory effect of dopamine. These data, coupled with
previous reports indicating a lack of RBF response to other
vasoactive medications and an increased risk of both short-
and long-term outcomes with other inotropic drugs, suggest
that further evaluation of the efficacy and safety of dopamine
for improvement of renal function in hospitalized patients
with acute decompensated HF is warranted.

Disclosures
None.

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**CLINICAL PERSPECTIVE**

Decreased renal function is common in hospitalized patients with heart failure, and attempts to improve it with a number of vasoactive medications have not resulted in a consistent favorable effect. A “renal dose” of dopamine has been used by clinicians for many years to enhance renal function in critically ill patients. The renal circulatory effect of dopamine, however, has not been well studied in patients with heart failure. The results of the present study demonstrated a significant vasodilatory effect of dopamine in doses between 2 and 10 μg · kg⁻¹ · min⁻¹ on both resistance and conductance renal arteries. This effect resulted in a significant reduction in renal vascular resistance and a rise in renal blood flow. In addition, intravenous dopamine resulted in a marked effect on cardiac output. These beneficial effects of intravenous dopamine both on the systemic and renal circulation are encouraging and suggest that further research on the effect of dopamine on renal function and outcome in hospitalized patients with heart failure is warranted.

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