The Effect of Various Fenoldopam Doses on Renal Perfusion in Patients Undergoing Cardiac Surgery

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Background. **The hypothesis that fenoldopam mesylate, by increasing renal flow, could reduce renal damage in patients undergoing cardiac surgery with cardiopulmonary bypass has gained great interest. The aim of the current study was to quantify the relationship of the increase of the renal blood flow as a function of the fenoldopam dose in these patients and to evaluate renal flow distribution within the renal parenchyma using Doppler.**

Methods. **Twenty-five patients admitted to cardiac surgery have been enrolled. We used the Doppler technique to measure renal blood flow at the level of the renal, segmental, interlobar, and interlobular arteries. We calculated both the resistive and pulsatility indexes in all the renal segments. Moreover, we calculated echographically all the variables of preload, afterload, and cardiac index. Measurements were performed at baseline and after the infusion of fenoldopam mesylate at the doses of 0.05, 0.1, 0.2, and 0.3 $\mu g \cdot kg^{-1} \cdot min^{-1}$.**

Results. **Fenoldopam infusion at doses more than 0.1 $\mu g \cdot kg^{-1} \cdot min^{-1}$ significantly increases blood flow in all renal compartments, thus improving the resistive and pulsatility indexes starting at a dose of 0.1 $\mu g \cdot kg^{-1} \cdot min^{-1}$. The highest renal flow increase is observed with 0.3 $\mu g \cdot kg^{-1} \cdot min^{-1}$. Fenoldopam seems to increase the renal flow directed to the most external kidney areas. Systemic hemodynamically significant changes are observed only in patients receiving doses more than 0.1 $\mu g \cdot kg^{-1} \cdot min^{-1}$.**

Conclusions. **In hemodynamically stable patients undergoing cardiac surgery with preserved renal function, fenoldopam shows a pharmacodynamic dose-dependent profile: it significantly increases renal flow and reduces the resistances of the renal circulation starting at a dose of 0.1 $\mu g \cdot kg^{-1} \cdot min^{-1}$.**

(Ann Thorac Surg 2010;89:497–504)
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Fenoldopam mesylate is a benzazepine derivative that is a potent short-acting dopamine A1 receptor agonist that decreases systemic vascular resistance (SVR) while, at the same time, increases renal blood flow (RBF) [1, 2]. The hypothesis that fenoldopam mesylate could reduce the renal damage in several clinical conditions has recently gained high interest, for example, for patients undergoing cardiac surgery in cardiopulmonary bypass conditions (CPB) [3], for the protection against radiocontrast-induced nephropathy, and for several conditions of intensive care medicine.

The studies conducted so far have been performed with fenoldopam mesylate at various doses, although the most commonly used is 0.1 $\mu g \cdot kg^{-1} \cdot min^{-1}$. Currently, there is no study highlighting the dose-relationship effect of fenoldopam mesylate on RBF in patients undergoing cardiosurgery with CPB.

The aim of the current study was to evaluate the effects of various increasing doses (0.05, 0.1, 0.2 and 0.3 $\mu g \cdot kg^{-1} \cdot min^{-1}$) of fenoldopam mesylate infusion on RBF and on ventricular afterload. The effects on systemic hemodynamic were also evaluated.

Accepted for publication Sept 29, 2009.

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Material and Methods

Patient Population

The study protocol was approved by the Ethics Committee of our institution, and all enrolled patients provided their written informed consent to participate. The study was conducted in accordance with the ethical standards of human investigation and with the Declaration of Helsinki (1975, revised in 1983).

Patients undergoing myocardial revascularization were asked to participate in the current study. All patients had preserved presurgical cardiac function, normal creatinine values, and age less than 75 years without concomitant major diseases. On arrival at the intensive care unit, after volemia normalization, patients considered hemodynamically stable, with no need of inotropic substances or mechanical supports, were included. All patients were sedated with propofol (0.5 $mg \cdot kg^{-1} \cdot h^{-1}$) and mechanically ventilated (FiO_2 0.5, tidal volume 7 mL/kg, positive end-expiratory pressure 5 cm/H_2O, 10 breaths per minute).

Echocardiographic Evaluation

After hemodynamic stabilization, mivacurium (0.1 mg/kg) was administered and a probe for transesophageal echocardiography was inserted (Omniplane II, Hewlett-Packard 5-MHz probe; Hewlett-Packard, Andover, MA).

We recorded the following transesophageal views:

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0003-4975/10/$36.00
doi:10.1016/j.athoracsur.2009.09.071

Abbreviations and Acronyms

AVA = aortic valve area
BMI = body mass index
CI = cardiac index
CPB = cardiopulmonary bypass
CSA = cross-sectional area
CVP = central venous pressure
DAP = diastolic arterial pressure
EDA = end diastolic area
EDID = end diastolic internal dimension
EDV = end diastolic velocity
EF = ejection fraction
ESID = end systolic internal dimension
ESMWS = end systolic meridional wall stress
ESPWT = end systolic posterior wall thickness
FDS = fractional diameter shortening
HR = heart rate
LAP = left atrial pressure
LVEDV = left ventricle end diastolic volume
LVSWT = left ventricle systolic wall tension
MAP = mean arterial pressure
MV = mean velocity
PI = pulsatility index
PSV = peak systolic velocity
RBF = renal blood flow
RF = renal failure
RI = resistive index
RRBFI = right renal artery blood flow index
SAP = systolic arterial pressure
SV = stroke volume
SVR = systemic vascular resistances
TDI = tissue Doppler imaging
TVI = time velocity integral

mid-esophageal four chamber, mid-esophageal long axis, mid-transgastric short axis, and deep transgastric. The tissue Doppler imaging was measured on the lateral wall of the mitral annulus in the four-chamber projection. All the measurements were done by a single operator (MM). The spectral Doppler signal settings were adjusted at the lowest wall filter and at the minimum optional gain. The measurements were carried out during expiration, using the mean of three consecutive cardiac cycles.

The preload was evaluated by left ventricle end-diastolic volume (LVEDV), end-diastolic area (EDA), and central venous pressure (CVP). Left atrial pressure (LAP) was calculated according to Nagueh and colleagues [4], using the formula: LAP = 2 + 1.3 (E/E′). The afterload was evaluated by end-systolic meridional wall stress (ESMWS) = 0.33∗SAP∗(ESID/ESPWT)∗(1 + ESPWT/ESID), where SAP is systolic arterial pressure, ESID is end-systolic internal dimension, and ESPWT is end-systolic posterior wall stress; left ventricle systolic wall tension (LVSWT) = 1.33∗SAP∗(ESID/2); and systemic vascular resistance (SVR) indexed (SVRI) = MAP − CVP/CI∗80, where MAP is mean arterial pressure and CI is cardiac index. Cardiac index was calculated as SV∗HR/BMI, where SV is stroke volume, HR is heart rate, and BMI is body mass index; and SV was calculated as SV = TVI∗AVA, where TVI (cm/s) is the time velocity integral of the flow across the aortic valve obtained with a continuous-wave Doppler in a deep transgastric view, and AVA (cm^2) is the mean effective aortic valve area throughout the ejection phase. The cross-sectional area was calculated as $\pi * r^2$, where r represents half of the annular diameter measured immediately proximal to the point of insertion of the aortic leaflet at the time of maximal separation. Cardiac contractility was calculated by measuring ejection fraction and fractional diameter shortening (FDS) = EDID − ESID/EDID∗100, where EDID is end-diastolic internal dimension.

Renal Perfusion

Renal blood flow in the renal artery was evaluated by the technique described by Garwood and colleagues [5]. From the transgastric position with a depth regulated at 12 cm, the probe was deflexed in a neutral position and turned 180 degrees clockwise. If the kidneys were not visible in the bottom left-hand sector of the screen in this position, the probe was moved 5 to 10 cm, turning the probe by 30 to 45 degrees to the patient's right to picture the kidney. By using the color Doppler in this position, the operator could identify the main renal, segmental, interlobar, and interlobular arteries.

It was not possible to apply this technique to all patients. Because the renal artery was not clearly identifiable with the use of transesophageal Doppler in 12 of the patients, the transabdominal approach—the so-called flank approach—was used [6]. The flow velocity in the renal artery was measured at 1 cm from its entrance to the kidney and the flow velocity in the segmental artery, at 0.5 cm from its origin, as well as the flow velocities in the interlobar and interlobular arteries. Peak systolic velocity (PSV), end-diastolic velocity (EDV), and mean velocity (MV) were recorded.

The pulsatility index (PI) was calculated using the formula PI = PSV − EDV/MV; and the resistive index (RI) was calculated using the formula RI = PSV − EDV/PSV [7]. Time velocity integral (TVI) was measured as the area under the outermost portion of the spectral velocity envelope. The right renal artery blood flow index (RRBFI [mL/min]) was calculated as the product of the right renal artery cross-sectional area (CSA) and the TVI, according to the following formula: flow = HR∗TVI∗CSA/BSA, where BSA is body surface area.

Table 1. Characteristics of the Patients

Age, years	61 ± 7
Sex	
Male	14 (56%)
Female	11 (44%)
Body surface area, m^2	2.26 ± 0.15
Preoperative ejection fraction, %	60 ± 6
Surgery	
Coronary artery bypass graft surgery × 3	13 (52%)
Coronary artery bypass graft surgery × 4	12 (48%)
Cardiopulmonary bypass time, minutes	65 ± 9
Aorta cross-clamp time, minutes	52 ± 10

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