The Effect of Dopamine on Hepatic-Splanchnic Blood Flow After Open Heart Surgery


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Summary. Dopamine (3,4 dihydroxyphenylethylamine) increases cardiac output and in particular the renal blood flow at the expense of other regional vascular beds not yet defined. Since the results of dopamine-induced changes in splanchnic perfusion are inconsistent, the effect of 6 mcg/kg/min dopamine was studied in 9 patients early after open heart surgery.

Estimated hepatic blood flow (EHBF) was calculated from the concentration-time slopes of Indocyanine Green (ICG, Cardiogreen 3R) in arterial and hepatic venous blood following single intravenous injection. Blood volume was measured using $^{51}$Cr tagged red cells. Cardiac output was determined according to the Fick method.

6 mcg/kg/min dopamine caused a mean EHBFB-increase of 82%, from 492 ± 64 to 824 ± 80 ml/min/m$^2$ ($P < 0.001$).

Related to the corresponding increase in cardiac index (CI) from 2.6 ± 0.2 to 3.8 ± 0.3 l/min/m$^2$ ($P < 0.001$), the EHBFB/CI-ratio changed from 18.5 to 21.7% ($P < 0.025$). The arterial-hepatic venous oxygen difference was reduced from 7.40 ± 0.53 to 4.91 ± 0.60 Vol% ($P < 0.001$).

It was concluded that splanchnic perfusion does not contribute to the preferential increase of renal blood flow under dopamine under the above mentioned conditions. Dopamine had the most beneficial effect on EHBFB in two cases where the latter was severely reduced.

Key words: Dopamine, Estimated hepatic blood flow, Splanchnic perfusion, Indocyanine Green single injection method.

For several years dopamine (3,4-dihydroxyphenylethylamine) has been used for the treatment of shock of various etiologies [4, 16, 23] and particularly for the treatment of low output states following cardiac surgery [8, 13, 19, 22, 25]. Compared with other catecholamines dopamine exerts positive inotropic actions with less chronotropic and arrhythmogenic effects than Isoproterenol or Orciprenaline and with less pressure effects than Epinephrine and Norepinephrine [8, 9, 13, 18]. Its main particularity, however, is its specific effect on the renal vascular bed [2, 5, 9, 10, 11, 14, 17, 23, 26]. The effect of dopamine on renal blood flow has been extensively investigated in animals[2,9,10,24, 34] as well as in man [5, 9, 10, 17, 19, 23] and a preferential increase of renal blood flow at the expense of other regional vascular beds has been firmly established. Only few studies on laboratory animals have been published concerning the effect of dopamine on splanchnic perfusion [9, 12, 20, 21, 31, 32, 34]. The reports about the specific actions of dopamine on mesenteric blood flow are contradictory [20, 31] and the effect of dopamine on hepatic perfusion in man has been investigated only in patients with liver cirrhosis [21].

Previously reported observations of ours on patients in the postabsorptive state without cardiovascular insufficiency indicated that dopamine increases cardiac output and hepatic blood flow to the same extent [1, 29]. The purpose of this present study was to investigate the effect of dopamine on splanchnic i.e. hepatic blood flow after cardiac valve operations, which often result in a low output state requiring treatment with this catecholamine.

Methods

Nine male patients with their data summarized in Table 1 were submitted to the study on the first postoperative day after open heart surgery for valve replacements. Patient selection was based on the following criteria: 1. No history of hepatic disease as judged from their own information and from simple liver function tests; absence of hepatic congestion. 2. Possibility to wean the patients off from any positive inotropic drug administration prior to the study. 3. Stable respiratory and metabolic state and absence of postoperative bleeding. 4. Informed consent.

All patients were mechanically ventilated and premedicated with Morphine 0.1 mg/kg 50 - 60 min prior to the study. Each patient had two central venous catheters, one arterial catheter, one pulmonary artery catheter, the latter introduced either by percutaneous puncture (Swan-Ganz Size F 5) or directly into the pulmonary artery by the surgeon, and one Goodale-Lubin catheter Size F 6 placed
in a main right hepatic vein [28] by puncture of the subclavian or internal jugular vein under fluoroscopic control immediately after surgery in a way to avoid wedging and yet minimize vena cava reflux [3, 28]. The position of all catheters was confirmed by x-ray prior to the study.

Hepatic blood flow (EHBF) was estimated from the concentration-time slopes of peripheral arterial and hepatic venous blood following a single intravenous injection of 0.75 mg/kg Indocyanine Green (ICG, CardioGreen®) [3, 6, 7, 28, 33] after first withdrawing a sample of blood for a plasma blank. Blood samples were taken from the radial artery at 2-minute intervals during 14 minutes and from the hepatic vein at 1-minute intervals during 10 minutes, with a delay of 10 to 15 seconds after arterial sampling to allow for the transit time [28]. Samples were centrifuged and the dye concentrations read against the plasma blank on a spectrophotometer (Specronic 710, Bausch and Lomb). The values of optical density, if plotted against the withdrawal time on a semilog scale, fell in a straight line, from which the half-time (t1/2) of dye clearance and the ICG-disappearance rate constant (K) were derived. The extraction rate (ER) was calculated from the ratio of the difference in arterial and hepatic venous dye concentration: ER = Aa/Aa - Ahv [3, 28].

Blood volume (BV) was measured using 51Cr tagged red cells (Packard Auto-Gamma-Scintillation Spectrometer). These parameters allowed an estimate of hepatic blood flow to be made from the formula: EHBF = K x BV/ER. This method of estimation of hepatic-splanchnic blood flow does not distinguish between blood, that reaches the liver by the hepatic artery or by the portal vein. The most critical step of this method is the sampling of mixed hepatic venous blood, which is used for the determination of the extraction rate of Cardiogreen. The relative lack of homogeneity of the mixed hepatic venous blood sample is unlikely to be influenced by the effects of dopamine and therefore this method of estimating the hepatic blood flow was deemed adequate for our investigation. Arterial, mixed venous and hepatic venous oxygen saturations and Hb-concentrations were also measured in each study (CO-Oxymeter Model 182, Instrumentation Lab. Inc.). Cardiac output was derived from the Fick equation and total oxygen consumption (VO2) directly estimated with a Mijnhardt Oxycon device (Oxycon, Mijnhardt Brothers B.V., Okijk, Holland) [15, 30]. In addition the following hemodynamic parameters were measured in each study phase: Heart rate (HR) by continuous ECG-monitoring, mean arterial pressure (MAP) in the radial artery and diastolic pulmonary artery pressure (PAdiast.) by a Statham Transducer SP 37 and Hellige monitoring equipment with analog and digital display. Central venous pressure (CVP) was measured by a saline manometer.

Studies were performed before and during the constant infusion (Braun infusion pump) of dopamine (6 mcg/kg/min) for at least twenty minutes.

In 3 of the 9 patients the measurements were repeated 20–30 minutes after stopping the dopamine infusion. The values obtained coincided well with the control values as listed in Table 4. For medical and logistic reasons the study could not be extended for each patient in such a way.

In order to control stability of pH, PaCO2 and PaO2 throughout the study arterial blood gas analyses were performed in each phase.

Results

The mean arterial and central venous pressure, heart rate, pulmonary arterial diastolic pressure, cardiac index, stroke index, systemic and pulmonary vascular resistance of all patients studied before and during dopamine administration are indicated in Table 2. A significant increase of arterial pressure from 75 ± 4 to 83 ± 4 mmHg, of the heart rate from 82 ± 5 to 111 ± 6 per minute1 and of the cardiac index from 2.6 ± 0.2 to 3.8 ± 0.3 l/min/m², as well as a significant decrease of systemic vascular resistance were seen.