## A525

Title: CORONARY VASODILATION BY CONTRAST MEDIA: IONIC VS. NON-IONIC

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Introduction. The ability of ionic contrast media to cause coronary vasodilation has been attributed to their high osmolarity. Recently nonionic contrast media have been developed with lowered osmolarity and with presumably blunted coronary vasodilator effects. This study was performed to compare coronary vasodilator responses of ionic (Hypaque-76) and nonionic (Isovue-370) contrast media and to assess the roles of hyperosmolarity and metabolic autoregulation in the contrast-induced coronary vasodilator responses.

Methods. After approval from our Institutional Animal Investigation Committee, ten mongrel dogs were anesthetized with pentobarbital, mechanically ventilated and left thoractomized. The left anterior descending coronary artery (LAD) was cannulated and perfused via a pressurized reservoir at 100 mmHg. The anterior interventricular vein was cannulated for collection of local venous samples. Coronary blood flow (CBF) was measured with an electromagnetic flowmeter and its transmural distribution assessed with 15 $\mu$ radioactive microspheres. In LAD-dependent myocardium, oxygen consumption ($MVO_2$) was calculated using Fick equation. Measurements were obtained when steady state conditions were achieved (4-6 minutes) during intracoronary infusions (2 ml/min) of Hypaque and Isovue. Results during infusion of contrast media were compared to those during similar volume infusions of hyperosmotic saline solutions with osmolarities comparable to contrast media (5.0 % and 2.5 %, respectively). Aortic and left ventricular end-diastolic pressures, left ventricular dP/dt max, and heart rate, were measured.

Results. Hypaque and Isovue caused pronounced steady-state increases in CBF. The increases in CBF by Hypaque were greater than those by Isovue (99 ± 17 % vs 53 ± 10 %). During infusion of contrast media, endocardium:epicardium flow ratio remained equal to 1.0, indicating that contrast-induced increases in CBF were transmurally uniform. Infusion of contrast media had no effect on $MVO_2$ . The saline solutions caused marked, initial increases in CBF (5.0 % > 2.5 %). Initial increase in CBF caused by 5.0 % saline was similar to that caused by Hypaque, although that caused by 2.5 % saline was less than that caused by Isovue. CBF decreased rapidly during infusions of hyperosmotic saline solutions so that steady state increases in CBF were modest (22 ± 9 % and 11 ± 6 % for 5.0 and 2.5 % saline, respectively) and considerably less than those for contrast media. Intracoronary infusions had no effect on global cardiac and systemic hemodynamic parameters.

Conclusions. 1) Contrast media caused increases in CBF which, at constant perfusion pressure, reflected coronary vasodilation. The coronary vasodilating action of ionic agent, Hypaque, was greater than that of nonionic agent, Isovue. 2) Coronary vasodilation caused by contrast media was not secondary to increased metabolic demand, but was due to direct relaxation of vascular smooth muscle. 3) Hyperosmolarity can account for initial coronary dilation caused by Hypaque but not for that caused by Isovue, and it cannot account for sustained dilation caused by either medium.

## A526

TITLE: CORONARY SMALL ARTERY CONSTRICTION AND SIMULTANEOUS ARTERIOLAR DILATION DURING HYPERCAPNIA

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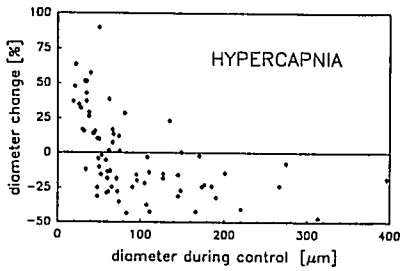
**Introduction:** Hypercapnia is a common problem in intensive care medicine and may occur accidentically during anesthesia. Among its various circulatory effects coronary vasodilation may be of special interest, since pharmacologically induced vasodilation can be detrimental to the myocardium. The aim of the present study was to investigate the effects of experimentally induced hypercapnia on coronary hemodynamics and on coronary microvessel diameters in dogs.

**Methods:** Investigations were performed in 12 mongrel dogs during general anesthesia with piritramid and halothane. Catheters were placed for hemodynamic monitoring and for measurement of myocardial blood flow by radioactive microspheres. Following thoracotomy, a 2-3 $cm^2$ surface area of the myocardium was immobilized by a heart holder. Epicardial coronary microvessels were visualized by intravital fluorescence microscopy. Arteriolar diameters were measured off-line. Recordings were obtained during a control period and during hypercapnia induced by adding approximately 6 % $CO_2$ to the inspiratory gas mixture. Left atrial and mean arterial pressures were kept constant by infusing electrolyte solution and by adjusting the inspiratory halothane concentration.

**Results:** During hypercapnia arterial $CO_2$ tension increased from 38±1 to 77±2 mm Hg. Heart rate and arterial $PO_2$ remained unchanged. Cardiac output increased by 14 %, systemic vascular resistance decreased by 19 %.

| | | control | hypercapnia |
|---|---|---|---|
| MBF | ($ml \cdot min^{-1} \cdot 100g^{-1}$) | 72 ± 6 | 123 ± 13* |
| CVR | ($mmHg \cdot min \cdot g \cdot ml^{-1}$) | 73 ± 6 | 44 ± 5* |
| cv $PO_2$ | (mmHg) | 30.9 ± 1.3 | 48.0 ± 2.0* |
| $O_2D$ | ($ml \cdot min^{-1} \cdot 100g^{-1}$) | 10.7 ± 1.0 | 16.6 ± 1.8* |
| $O_2C$ | ($ml \cdot min^{-1} \cdot 100g^{-1}$) | 6.2 ± 0.5 | 7.0 ± 0.6 |

*(MBF, myocardial blood flow; CVR, coronary vascular resistance; cv $PO_2$, coronary venous $PO_2$; $O_2D$ and $O_2C$, myocardial oxygen delivery and consumption, respectively; means ± SEM; * $p < 0.05$ vs. control.*




*In the figure % changes of coronary arteriolar diameters during hypercapnia are plotted against diameters during control (n=76).*

**Conclusion:** During hypercapnia-induced coronary dilation arterioles < 100 μm dilated, while larger vessels constricted. Preferential dilation of arterioles < 100 μm during adenosine and nitroglycerin infusion has been described in a previous study from this laboratory[1]. However, in that study arterial vessels > 100 μm dilated as well. The mechanisms responsible for the different effects of $CO_2$ on coronary arterioles of different sizes remain to be investigated.

*1. (Habazettl et al. Anesthesiology 71:A482, 1989).*