

Title: THE CARDIOVASCULAR EFFECTS OF VERAPAMIL ADMINISTRATION DURING CORONARY ARTERY BYPASS GRAFT SURGERY

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**Introduction.** Verapamil (V), one of the slow channel calcium blockers, affects cardiac conduction, contractility, and vascular smooth muscle tone to varying degrees. Its administration to awake, cardiac patients is not associated with hemodynamic compromise unless left ventricular failure is present.<sup>1</sup> Since V is used in the perioperative period for patients with heart disease, a knowledge of its effects on the anesthetized, cardiac patient is important. This study was designed to evaluate the cardiovascular effects of V during coronary artery bypass graft surgery (CABG).

**Methods.** After approval by the human investigations committee and informed patient consent, 16 patients scheduled for elective CABG were studied. All patients had good left ventricular function (LVF) with an ejection fraction >.50, left ventricular end-diastolic pressure < 18 mmHg and no cardiac conduction defects. Preoperatively, all patients received nitrates through the morning of surgery and fifteen patients received beta blockers through the evening prior to surgery. Premedication included diazepam 0.1 mg·kg<sup>-1</sup> orally, and morphine 0.1 mg·kg<sup>-1</sup> plus scopolamine 0.2 - .4 mg intramuscularly. Intraoperative monitoring consisted of ECG (II, V<sub>5</sub> and esophageal leads), radial artery, central venous (CVP), pulmonary artery (PAP) and pulmonary capillary wedge pressures (PCWP), as well as thermodilution cardiac outputs via a Swan-Ganz catheter. Derived parameters included cardiac index (CI), systemic and pulmonary vascular resistance (SVR, PVR), and left and right ventricular stroke work index (LVSWI, RVSWI). Anesthesia consisted of morphine 0.5 - 1.5 mg·kg<sup>-1</sup>, diazepam 0.3 - 1.0 mg·kg<sup>-1</sup>, nitrous oxide 50% plus oxygen. Usual conduct of anesthesia was continued through aortic cannulation for cardiopulmonary bypass (CPB). At this point control hemodynamic measurements were made and then V 0.075 mg·kg<sup>-1</sup> or the solvent (S) was administered I.V. over 1 minute (min) in a randomized, double-blind fashion. Hemodynamic variables were measured at 1.0, 2.5, 5.0, 7.5 and 10 min after drug administration. Arterial blood analysis included V serum levels which were determined by liquid chromatography at 0.5, 1, 2, 4, 6, 8 and 10 min after V and immediately after CPB. Arterial blood gases, potassium, and ionized calcium were also measured during the study. After the study period the surgeon cannulated the atrium and proceeded with CABG as usual. The V and S groups were compared using two-way analysis of variance. Significance was assumed when p < 0.05.

**Results.** V did not cause a significant change in heart rate, CVP, PAP, PCWP, CI, PVR or RVSWI. The PR interval increased slightly but not significantly after V administration. However, mean arterial pressure (MAP), SVR, and LVSWI decreased significantly in the V group (figure). The lowest MAP was 60 mmHg and no ST-segment changes occurred during the study. None of the hemodynamic variable changed significantly in the S group. Serum V levels peaked to 202.8 ng·ml<sup>-1</sup> in 0.5 min, rapidly decayed to 17.3 ng·ml<sup>-1</sup>·kg by 10 min, and were not detected after CPB. Throughout the study arterial blood gases and electrolytes were within normal limits. During CPB the doses of phenylephrine and phentolamine necessary to maintain MAP between 50 and 100 mmHg were not significantly different between the V or S groups. All patients tolerated CABG without problems and were weaned from CPB uneventfully.

**Discussion.** These results indicate that V, 0.075 mg·kg<sup>-1</sup> can be safely administered before CPB to patients with good LVF during narcotic based anesthesia for CABG. This study group did not develop a decrease in CI or elevation in PCWP, or disturbance in cardiac conduction; however, additional studies need to include patients with compromised LVF and other anesthetic agents. Furthermore, in certain clinical situations, the predictable reduction in MAP after V administration could result in inadequate myocardial perfusion.

**References.**

1. Chew CY, Hecht HS, Collett JT, et al: Influence of severity of ventricular dysfunction on hemodynamic responses to intravenously administered verapamil in ischemic heart disease. *Am J Cardiol* 47:917-922, 1981

