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Brain natriuretic peptide decreases pulmonary artery pressure in rats with pulmonary hypertension

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INTRODUCTION: B-type or Brain natriuretic peptide (BNP) was first isolated from the porcine brain, but it is primarily secreted from the cardiac ventricles in humans. As a cardiac hormone it has a physiologic regulatory function in the cardiovascular system. Along with atrial, and C-type natriuretic peptides, BNP aids in the natriuretic, diuretic, and vasorelaxant responses intended to reduce blood pressure and fluid volume homeostasis. Its vasodilator properties are known to be present in the pulmonary circulation; therefore, we hypothesized that BNP can be used as a therapy to lower the pulmonary artery pressure in an animal model of pulmonary hypertension.

METHODS: Male Sprague-Dawley rats were given a one-time subcutaneous injection of 60 mg/kg monocrotaline to induce PH over a five week period. After establishment of PH, rats were anesthetized and ventilated. A catheter was placed in the right jugular vein and passed into the right ventricle to record right ventricular pressure (RVSP), an estimate of pulmonary artery pressure. A second catheter was placed in the right carotid artery to measure mean arterial pressure (MAP). RVSP and MAP were recorded before, during, and after infusions of BNP.

RESULTS: One hour infusions of 5, 25, 50 or 150 ng/kg min BNP caused 24, 31, 38, or 36% decreases in RVSP, respectively. There was no evidence of systemic hypotension at these doses of BNP.

CONCLUSION: These preliminary findings suggest that BNP causes dose dependent decreases in pulmonary artery pressure in rats with pulmonary hypertension. Further study is needed to confirm these preliminary results.