Title: COMPARISON OF HEMODYNAMIC RESPONSES DURING HYPOTENSION INDUCED BY HALOTHANE AND ADENOSINE TRIPHOSPHATE.

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INTRODUCTION: Halothane is widely used, yet severe cardiac depression and inadequate tissue perfusion is well known to occur during halothane-induced hypotension. In this study, hypotension was achieved either by deepening halothane anesthesia or by giving an intravenous infusion of adenosine triphosphate (ATP) at a constant halothane concentration. The hemodynamic effects of hypotension produced by ATP and by halothane were compared.

METHOD: Six dogs were induced with sodium thiopental (20 mg/kg, IV). After tracheal intubation, they were ventilated with 1% halothane in oxygen to maintain normal PCO_2. Animals were instrumented and the following measurements were recorded: ECG, heart rate (HR), mean arterial blood pressure (MAP), left ventricular end diastolic pressure (LVEDP) and dP/dt. Central venous pressure (CVP), mean pulmonary arterial pressure (MPAP) and pulmonary capillary wedge pressure (PCWP) were measured through a Swan-Ganz catheter which was also used to determine thermodilution cardiac output (CO). After 1 hour of stabilization, control measurements were recorded at end-tidal anesthetic concentration (ET) of 0.85%. Thereafter, halothane concentration was increased to produce a MAP of 70, 50 and 30 mmHg (ET were 1.17±0.15, 1.66±0.13 and 2.57±0.09% respectively) which was maintained for 30 minutes each, after which time repeat measurements were taken. Halothane ET was then returned to 0.85% and another set of control measurements was obtained 40 minutes later. Thereafter, the same hypotension steps were produced by continuous intravenous infusion of ATP (1.7±0.9, 4.7±1.0 and 8.2±0.9 mg/kg/min respectively) and measurements were repeated. Paired t-test was used to compare progressive changes between the two periods.

RESULTS: The dose response and hemodynamic parameters are summarized in figures 1 and 2. There was no significant difference between the two control periods. CO and stroke volume (SV) significantly decreased during halothane and consistently increased during the ATP induced hypotension. Left ventricular stroke work (LVSW) decreased significantly in both periods. SVR was significantly decreased only during ATP induced hypotension. CVP increased significantly with halothane, but was unchanged during ATP period.

CONCLUSION: The findings during deep halothane induced hypotension confirm earlier studies that the major cause of arterial hypotension during halothane anesthesia is the depression of myocardial contractility[1], characterized by a failure to empty rather than a failure to fill. HR and SVR was reduced during ATP induced hypotension and this resulted in increased CO and SV. On the basis of this study ATP is a mild cardiac depressing agent (decreased LVSW) and is also a vasodilator which has the advantage of maintaining sufficient cardiac function during controlled hypotension.

REFERENCE:

1. Prys-Roberts C, Gersh BJ, Baker AB: The effects of halothane on the interactions between myocardial contractility, aortic impedance, and left ventricular performance I. Brit. J. Anaesth. 44:634-648, 1972.

Fig 1: Dose response

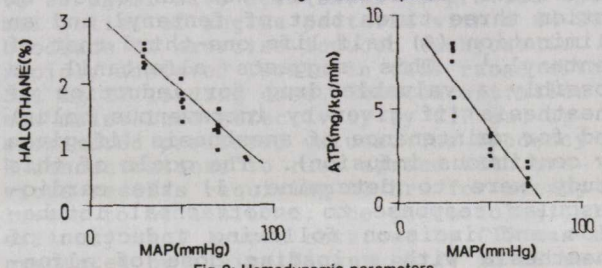

Fig 2: Hemodynamic parameters