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ASA ABSTRACTS

TITLE: VECURONIUM INFUSION VS. MEPERIDINE IN THE TREATMENT OF SHIVERING FOLLOWING CARDIOPULMONARY BYPASS


Introduction: Adverse effects of shivering following hypothermic cardiopulmonary bypass (CPB) include increased O2 consumption (VO2), hypercarbia, acidosis and hemodynamic instability.1 We hypothesized that vecuronium (VEC) abolishes shivering and its adverse effects more reliably and effectively than meperidine (MEP).

Methods: After institutional approval and consent, 20 male patients (age 59±9 yr), undergoing hypothermic CPB (CABG, n=18; AVR, n=2), were randomized and prospectively studied. Patients undergoing reoperation, with CHF or COPD, hemodynamic instability, or those who did not shiver, were excluded. Patients were anesthetized with a standard fentanyl-relaxant-benzodiazepine technique. Postop sedation was assured by a continuous infusion of fentanyl in the intensive care unit (ICU) for the study duration.

Standard monitoring was used, plus an Oximetrix® pulmonary artery catheter (Abbott Inc), neuromuscular blockade monitor, and capnograph.

After stabilization in the ICU, baseline data were obtained: heart rate (HR), systolic blood pressure (SBP), pulmonary artery wedge pressure (PAWP), cardiac output (CO), continuous mixed venous oxygen saturation (SvO2), core temp, end-tidal CO2 (EtCO2), arterial and mixed venous gases, FiO2 and hemoglobin. Patients who shivered within 3 h of ICU admission were randomized to receive VEC (Group I, n=10), or VEC (Group II, n=10). Shivering was scored: 0=none, 1=muscle spasms, 2=localized, 3=generalized, and 4=violent. MEP was given as 25 mg IV q15 min until shivering score = 0 or 75 mg was given. If shivering persisted, patients received VEC. Group II patients received VEC 0.1 mg/kg q IV followed by 1.0 µg/kg/min continuous infusion, adjusted q15 min to maintain 1 twitch on the train-of-4 blockade monitor. Data were repeated 15 min after each intervention and at 1, 2 and 4 hrs post treatment. VO2 was derived by Fick equation. Data were analyzed by ANOVA and Fisher's LSD.

Results: In 50% of Group I patients (5/10) MEP successfully abolished shivering (MEP-S), but in 50%, (5/10) MEP failed (MEP-F). In 100% of Group II patients (10/10) shivering ceased, and remained absent during VEC infusion. In 55 MEP-F patients who subsequently received VEC, shivering resolved. There was no significant difference in mean VO2, SvO2 and EtCO2 between Groups I and II at baseline and at shivering. However, with treatment VEC induced a significantly greater increase in SvO2 and decrease in VO2 and EtCO2 than MEP (p<0.05, Single factor ANOVA). The % decrease in VO2 (Fig 1) and EtCO2 after treatment was significantly greater with VEC than in both MEP-S and MEP-F patients, but the % increase in SvO2 with VEC was greater than that in MEP-F patients only. There were no significant differences in HR, SBP or CO2; however, MEP patients frequently required reduction of vasodilator or sedative infusions to maintain stability. There was no difference in time to postoperative wakening or extubation.

Discussion: VEC following CPB provides assurance of abolishing shivering with marked improvement in VO2 and EtCO2. Control may be maintained by continuous VEC infusion up to 4 hrs without delaying extubation. Cessation of shivering with VEC is unpredictable, occurs in only 50% of cases, and its benefit in reducing VO2 and EtCO2 is significantly less than VEC.


FIGURE 1

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TITLE: AMINORONE INCREASES VENOUS ADMIXTURE AND DECREASES ARTERIAL OXYGENATION (Pao2) AFTER CORONARY ARTERY BYPASS GRAFT (CABG) SURGERY

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INTRODUCTION: Aminorone (AMR) is a bipyridine used to support the circulation after CABG surgery. As a vasodilating inotrope, AMR may impair hypoxic pulmonary vasoconstriction (HPV), decreasing Pao2 and increasing venous admixture (Qv/Qa).

METHODS: After IRB approval, 24 consenting patients with good left ventricular function were studied 24 hrs after CABG surgery. All patients were extubated, received supplemental O2, and did not require vasodilator drug therapy. Standard hemodynamic measurements were performed; cardiac output (CO) was determined by thermodilution. Arterial and mixed venous oxygen tensions (Pao2 and PvO2) and saturations (SaO2 and SvO2) were measured.

After baseline hemodynamic measurements, AMR was administered at one of two doses: 0.75 mg/kg bolus (B) + an infusion (I) of 10 µg/kg/min (LOW), or 2.25 mg/kg B + 20 µg/kg/min (HIGH). Measurements were repeated after B, and after 30 min of I. Cardiac index (CI) and shunt (Qv/Qa) were calculated. Data are reported as means ± SE. Data were analyzed by ANOVA for significant differences (P<0.05).

RESULTS: Low- and high-dose AMR increased CI by 10.5% and 22.6%, respectively. AMR at both doses decreased arterial and pulmonary pressures and SaO2. Pao2 decreased following AMR (Fig 1). Qv/Qa significantly increased at both doses of AMR. Surprisingly, SvO2 was unchanged at both AMR doses despite the significant increase in CI.

DISCUSSION: AMR increased CI and decreased systemic vascular resistance consistent with its inotropic and vasodilatory effects. Despite the increases in CI, SvO2 remained unchanged. Thus, SvO2 may be a poor guide for inotropic titration of AMR (1). AMR also significantly decreased Pao2 and increased Qv/Qa due, we believe, to inhibition of hypoxic pulmonary vasoconstriction (HPV). Inhibition of HPV by AMR has previously been reported in rats (2). Thus, careful Pao2 monitoring during AMR therapy appears indicated.


FIGURE 1