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

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Introduction: Adverse effects of shivering following hypothermic cardiopulmonary bypass (CPB) include increased O_2 consumption (VO_2), 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 yrs), 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.

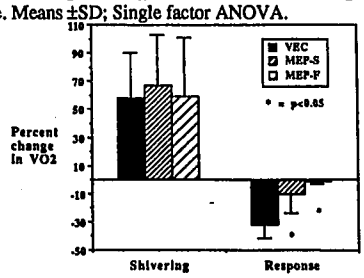
After stabilization in the (ICU), baseline data were obtained: heart rate (HR), systolic blood pressure (SBP), pulmonary artery wedge (PAW) , cardiac output (CO), continuous mixed venous oxygen saturation (SvO_2), core temp, end-tidal CO_2 ($EtCO_2$), arterial and mixed venous gases, FIO_2 and hemoglobin. Patients who shivered within 6 hours of ICU admission were randomized to receive MEP (Group I, n=10), or VEC (Group II, n=10). Shivering was scored: 0=none, 1=masseter spasm, 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 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. VO_2 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 5/5 MEP-F patients who subsequently received VEC, shivering resolved. There was no significant difference in mean VO_2, SvO_2 and $EtCO_2$ between Groups I and II at baseline and at shivering. However, with treatment VEC induced a significantly greater increase in SvO_2 and decrease in VO_2 and $EtCO_2$ than MEP ($p<0.05$, Single factor ANOVA). The % decrease in VO_2 (Fig 1) and $EtCO_2$ after treatment was significantly greater with VEC than in both MEP-S and MEP-F patients, but the % increase in SVO_2 with VEC was greater than that in MEP-F patients only. There were no significant differences in HR, SBP or CO; 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 VO_2 and $EtCO_2$. Control may be maintained by continuous VEC infusion up to 4 hrs without delaying extubation. Cessation of shivering with MEP is unpredictable, occurs in only 50% of cases, and its benefit in reducing VO_2 and $EtCO_2$ is significantly less than VEC.

Reference: 1. Journal of Cardiothoracic Anesthesia: 1:24-28, 1987

Fig.1. Percent change in VO_2 from baseline to shivering, and from shivering to response. Means ±SD; Single factor ANOVA.



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TITLE: AMRINONE INCREASES VENOUS ADMIXTURE AND DECREASES ARTERIAL OXYGENATION (PaO_2) AFTER CORONARY ARTERY BYPASS GRAFT (CABG) SURGERY

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INTRODUCTION: Amrinone (AMR) is a bipyridine used to support the circulation after CABG surgery. As a vasodilating inotrope, AMR may impair hypoxic pulmonary vasoconstriction (HPV), decreasing PaO_2 and increasing venous admixture ($\dot{Q}_s/\dot{Q}_t$).

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 O_2, and did not require vaso-active drugs. Standard hemodynamic measurements were performed; cardiac output (CO) was determined by thermodilution. Arterial and mixed venous oxygen tensions (PaO_2 and PvO_2) and saturations (SaO_2 and SvO_2) 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 I (HIGH). Measurements were repeated after B, and after 10 min of I. Cardiac index (CI) and shunt ($\dot{Q}_s/\dot{Q}_t$) were calculated. Data are reported as means ± SEM. 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 SaO_2. PaO_2 decreased following AMR (Fig 1). $\dot{Q}_s/\dot{Q}_t$ significantly increased at both doses of AMR. Surprisingly, SvO_2 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, SvO_2 remained unchanged. Thus, SvO_2 may be a poor guide for inotropic titration of AMR (1). AMR also significantly decreased PaO_2 and increased $\dot{Q}_s/\dot{Q}_t$, due, we believe, to inhibition of hypoxic pulmonary vasoconstriction (HPV). Inhibition of HPV by AMR has previously been reported in rats (2). Thus, careful PaO_2 monitoring during AMR therapy appears indicated.

REFERENCES: (1) Chest 95:1289-1294, 1989.
(2) Proc Soc Exp Biol Med. 173:205-212, 1983.

FIGURE 1.