A139

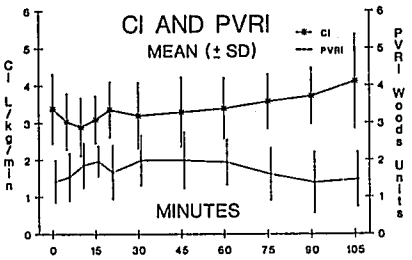
TITLE: HEMODYNAMIC EFFECTS OF OKT3 DURING RENAL TRANSPLANT

AUTHOR: S.T. Robinson, MD
AFFILIATION: Anes. Dept, O.H.S.U., Portland, Oregon 97201

Perioperative administration of a monoclonal antibody to cytotoxic T cells, muromonab CD3 (Orthoclone OKT3), is a major advancement in the prevention of rejection in renal transplant recipients (RTR).[1] However, it may cause pulmonary edema in awake patients whose weight is elevated above their dry baseline and often causes nausea, vomiting, malaise and fever. Administration of OKT3 during anesthesia would reduce the occurrence of the unpleasant symptoms. However, pulmonary edema was a concern and we sought to observe whether it occurred under anesthesia and if so why.

Methods: 12 patients undergoing cadaveric renal transplant at baseline dry weight and without evidence of CHF were studied intraoperatively. The anesthetic management varied somewhat according to the attending anesthesiologist. Patients were sedated with midazolam, induced with thiopental, intubated with the assistance of succinylcholine or vecuronium and maintained with O_2/N_2O/isoflurane and fentanyl. A pulmonary artery catheter was inserted. Azathioprine 2mg/kg, cephapirin 1gm, methylprednisolone 500mg and diphenhydramine 50mg were administered IV. Following surgical incision the patient was stabilized and baseline HR, BP, PAP, PCWP, CVP, CO, nasal temp, F_iO_2, SaO_2, and $EtCO_2$ were measured. OKT3, 5mg, was administered IV and these measurements were repeated at 5, 10, 15, 20, 30, 45, 60, 75, 90 and 105 minutes. Newman-Keuls and Dunnett's tests were applied to positive ANOVA results to determine if the differences noted were from baseline.

Results: All patients had uneventful anesthetic courses, remained euthermic and maintained hemodynamic and respiratory stability. There were no statistically significantly differences in any of the variables from their pre-OKT3 baseline except for an increase in the CI at 105 minutes ($p<.05$) which was probably caused by lighter anesthesia at the end of the case. Although not statistically significant, PVRI tended to be elevated during the first hour post OKT3. The correlation of CVP and PCWP was r=0.80 described as PCWP= 0.92 x CVP + 3.2.



Discussion: We conclude that OKT3 can be safely administered intraoperatively to RTR who are in stable fluid balance. We found no signs of hemodynamic or pulmonary compromise in these patients. The early increase in PVRI supports further study into the practice of avoiding OKT3 in volume loaded patients. In lieu of a PA catheter, CVP monitoring in euvolemic RTR who will be receiving intraoperative OKT3 is adequate.

Reference

1. Nephron 46: suppl., pp. 12-18, 1987.

A140

CARDIOVASCULAR EFFECT OF DESFLURANE IN OUTPATIENTS

JE Fletcher, MB, PS Sebel, MB, MR Murphy, MD
CS Smith, MD, M Flister, CRNA, S Mick, RN

Department of Anesthesiology, Emory University School of Medicine
Crawford Long Hospital, Atlanta, Georgia

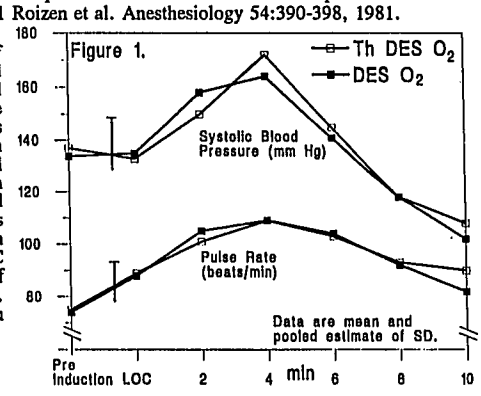
INTRODUCTION: We sought to determine whether, in outpatients, the cardiovascular effects of: a) induction of anesthesia with thiopental differed from that with desflurane (DES) inhalation; b) (DES) supplemented anesthesia differed from isoflurane (ISO) supplemented anesthesia.

METHOD: After Human Investigation Committee approval, 80 consenting ASA I - III patients (age 40.7 ± 12.0 yr, weight 77.0 ± 14.0 kg) were randomized into four groups: 1) Thiopental induction followed by DES in N_2O and O_2. 2) Thiopental induction followed by ISO in N_2O/O_2. 3) Thiopental induction followed by DES/O_2. 4) DES inhalational induction followed by DES/O_2. Premedication was ranitidine 150 mg and metoclopramide 10 mg. No opioids were given; succinylcholine was used to facilitate intubation. Anesthesia was maintained with 1.3 MAC DES (no N_2O) (age adjusted MAC in O_2) or 1 MAC ISO or DES (with N_2O) with normocapnic ventilation. Patients received intravenous crystalloid, 0.5 to 1 ml/kg/hr fasting prior to induction. Blood pressure (Dinamap) and pulse (pulse oximeter) were recorded automatically every minute. Data are mean ± SD. Statistical analysis was with repeated measures ANOVA and t test.

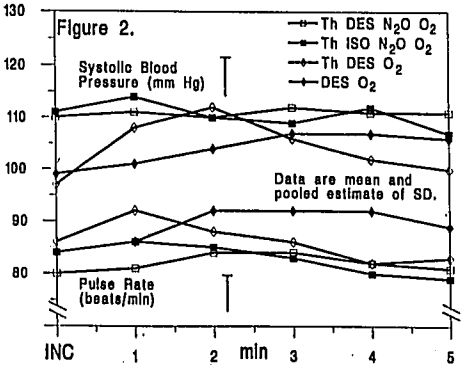
RESULTS: Groups were not different for age or weight. There were no differences at either induction or incision in systolic or diastolic blood pressures, or pulse rate between groups 1 and 2, or groups 3 and 4. An inhalational induction resulted in excitation, including hypertension and tachycardia, shortly after loss of consciousness. Similar cardiovascular changes occurred in the group of patients induced using thiopental despite the absence of excitation [Fig 1]. One patient in each of group 3 and 4 required treatment for hypertension/tachycardia and were excluded from the analysis. Airway complications were minimal. Patients were pleased with both techniques of induction. Prior to incision ephedrine was required to support the blood pressure in 3 (group 1) and 2 (group 2) patients. Three minutes after incision systolic blood pressure increased by over 10 mm Hg in 36% of patients maintained with 1.3 MAC DES in O_2 [Fig.2].

DISCUSSION: We conclude that the cardiovascular effects of DES show no clinically important differences from those of ISO and that DES inhalational induction has similar cardiovascular effects to thiopental induction. As 1.5 MAC anesthetic concentration is associated with a 10 mm Hg rise of systolic BP in 50% of patients after incision[1], compared to 36% patients at 1.3 MAC in our study, we conclude that 1.3 MAC of DES in O_2, without opioids, is adequate to block the cardiovascular response to incision.

REFERENCE: 1 Roizen et al. Anesthesiology 54:390-398, 1981.



Comparison of systolic blood pressure and pulse rate between patients receiving an inhalational induction with desflurane and an intravenous induction with thiopental. LOC = loss of consciousness. Times are min from LOC.



Comparison of systolic blood pressure and pulse rate between all groups just prior to (INC), and in the 5 min after commencing surgery.