Title : CARDIOVASCULAR EFFECTS OF DIMETHYL TUBOCURARINE IN PATIENTS WITH AORTIC VALVULAR DISEASE DURING MORPHINE-DIAZEPAM-OXYGEN ANESTHESIA

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Introduction: Muscle relaxants are an integral part of the anesthetic management of patients undergoing valvular heart surgery. Dimethyl tubocurarine (DMTC), a nondepolarizing muscle relaxant, has been available since 1948, but only recently has been shown to have minimal cardiovascular effects in healthy patients during nitrous-narcotic,[1,2] enflurane,[3] and halothane[4] anesthesia. Patients with coronary artery disease taking propranolol also were observed to have minimal cardiovascular changes associated with DMTC's use.[5] The intent of this study was to evaluate the hemodynamic effects of DMTC in patients with aortic valve disease after a morphine-diazepam induction.

Methods: Seven consecutive patients with aortic stenosis (mean gradient 90 torr) who gave their written consent were premedicated with morphine 0.1 mg/kg and scopolamine 0.2 mg. The patients were monitored with an arterial line, a pulmonary artery catheter, an EKG, a chest phonocardiogram, and a train-of-four nerve stimulator. Anesthesia was induced with morphine and diazepam until they no longer responded to verbal stimuli (morphine 26.7 ± 12.2 mg, diazepam 17.5 ± 4.8 mg). After 10 minutes on 100% oxygen to allow stable hemodynamics after the induction, the control measurements were made. Fifteen mg/m^2 of DMTC were given over 2 minutes and measurements were performed at 2½,5,7½,and 10 minutes after the DMTC was administered. Arterial blood gases were drawn at the control measurement and at the 5 and 10 minute measurements to assure adequate oxygenation and ventilation. Direct measurements consisted of mean arterial blood pressure ($\overline{BP}$), mean pulmonary artery pressure ($\overline{PA}$), heart rate (HR), pulmonary capillary wedge pressure (PCWP), central venous pressure (CVP) cardiac output (CO), systolic time intervals, and arterial blood gases while derived variables involved pulmonary vascular resistance (PVR), systemic vascular resistance (SVR), right ventricular minute work index (RVMWI), and endocardial viability ratio (EVR). For each ventricle, 1/PEP^2 was calculated separately ($1/PEP^2_{RV}$, $1/PEP^2_{LV}$). Neuromuscular blockade was evaluated by observing the number of twitches evoked by the electrical stimuli in the train-of-four. The measurements were analyzed statistically by an analysis of variance.

Results: The results demonstrate that the patients were hemodynamically stable during the study period and that the administration of DMTC to patients with aortic stenosis after a morphine-diazepam induction caused no significant change in $\overline{BP}$ (64.1 ± 8.9 control to 73.4 ± 7.0 at 10 minutes), $\overline{PA}$ (16.7 ± 2.9 to 19.0 ± 5.1), PCWP (12.6 ± 2.8 to 14.5 ± 4.5), CVP (9.3 ± 2.0 to 11.0 ± 3), HR (57.3 ± 10.4 to 63.0 ± 10.1), CI (2.13 ± .32 to 2.50 ± .48), PVR (93.3 ± 20.0 to 83.6 ± 16.6), SVR (1229 ± 240 to 1217 ± 312) or RVMWI (.219 ± .099 to .279 ± .146). (see Fig.) Muscle relaxation was clinically adequate with the train-of-four revealing ≥ 80% block (≥ 2 twitches abolished).

Discussion: These patients were classified as having "tight" aortic stenosis because of the high gradient. The use of muscle relaxants in these patients is necessary but, at times, dangerous. Pancuronium and gallamine can result in tachycardia and hypertension and d tubocurarine can result in hypotension with reflex tachycardia. Obviously both of these situations are undesirable when dealing with a patient with aortic stenosis. DMTC, when given over a 2 minute period at a dose of 15 mg/m^2 produces no significant hemodynamic changes and results in adequate muscle relaxation.

References:

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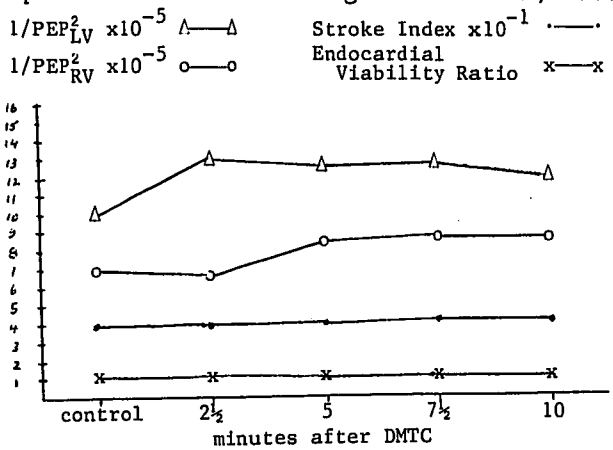

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