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Ketamine Does Not Increase Pulmonary Vascular Resistance in Children with Pulmonary Hypertension

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Introduction: Presence of pulmonary hypertension (PHT) significantly increases the perioperative risk of morbidity and mortality.[1] Ketamine is an useful anesthetic agent for children with diminished cardiopulmonary reserve. Use of ketamine in children with PHT is controversial because of concern about escalating pulmonary vascular resistance.[2,3] The aim of this prospective study of intravenous ketamine anesthesia in children with PHT was to evaluate its effects on pulmonary vascular resistance during spontaneous ventilation.

Methods: Patients between 3 months and 18 years of age, undergoing cardiac catheterization for evaluation of PHT were studied after institutional and parental consent. Patients were premedicated with oral midazolam (0.5-0.75 mg/kg). After mask induction with air and sevoflurane, venous and arterial access was established. Baseline systemic and pulmonary arterial and venous pressure data and blood gases were obtained on 0.5 to 1 MAC of sevoflurane in air. Patients then received an IV ketamine bolus (2mg/kg) over 5 minutes followed by an IV ketamine infusion at 10mcg/kg/min. Sevoflurane/air was continued at 0.5 to 1.0 MAC and all patients were breathing spontaneously. Hemodynamic and blood gas data were obtained at 5, 10, and 15 minutes after end of ketamine bolus. Mean (SD) data are presented and paired t test performed; significance set at $P<0.05$.

Results: Ten patients of mean age =9 yr (range=3 mos –15 years) and weight=31.5 kg (range: 6.5-68 kg) were enrolled in this ongoing study. Five subjects had associated congenital heart defects. No adverse effects have been associated with the use of ketamine thus far. Hemodynamic and respiratory data are shown in Table 1. Measurements at 5, 10 and 15 minutes after ketamine load did not differ significantly from baseline values.

Discussion: In this study of children with severe PHT receiving 0.5-1 MAC of sevoflurane anesthesia, a 2mg/kg bolus of ketamine followed by a 10mcg/kg/min ketamine infusion did not increase the pulmonary vascular resistance. Ketamine may be valuable for preserving coronary perfusion to the at-risk right ventricle as it did not increase pulmonary vascular resistance. In addition it maintained systemic vascular resistance and systemic mean arterial pressure without causing tachycardia*:* Aggravations in pulmonary vascular resistance related to airway instrumentation have been reported in patients with PHT.[1] Conversely, hypercapnea may develop during anesthesia with spontaneous ventilation and lead to an increase in pulmonary artery pressure.[4] The addition of ketamine to low dose sevoflurane did not increase $PaCO_2$ during spontaneous ventilation and allowed us to avoid instrumentation of the airway.
Children with PHT often present for interventions requiring anesthesia and have an increased risk of anesthetic complications.[1] Interim analyses suggest ketamine may be an appropriate anesthetic agent for such patients.

Table 1: Mean (SD) values at baseline and 5, 10 and 15 minutes after ketamine load.

	Baseline	5 mins	10 mins	15 mins
Heart rate (bpm)	105 (22)	103 (22)	100 (22)	101 (22)
MAP (mm Hg)	65 (7)	72 (12)	69 (12)	70 (11)
Mean PAP (mm Hg)	56 (18)	54 (16)	56 (17)	56 (17)
PVR (Wood units)	11.2 (4.8)	11.2 (4.5)	11.4 (4.5)	11.5 (4.4)
SVR (Wood units)	13.1 (4.1)	14.7 (4.5)	14.5 (4.8)	14.7 (4.2)
SaO_2 (%)	95 (6)	94 (7)	95 (6)	95 (5)
$PaCO_2$ (mm Hg)	51 (7)	50 (6)	50 (6)	50 (7)

MAP = mean arterial pressure, PAP = pulmonary artery pressure, PVR = pulmonary vascular resistance, SVR = systemic vascular resistance, SaO_2 = arterial oxygen saturation, $PaCO_2$ = arterial carbon dioxide partial pressure.

References:

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