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Comparative Effects of Halothane and Ketamine on Systemic Arterial Oxygen Saturation in Children with Cyanotic Heart Disease

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Because of the occurrence of significant hypotension due to decreased myocardial contractility and peripheral vasodilation, induction of anesthesia with halothane and nitrous oxide has been avoided in children with cyanotic congenital heart disease.¹⁻⁴ The evidence for avoiding volatile agents and inhalation induction has been mostly anecdotal and based on the theoretical consideration that a decrease in blood pressure and systemic vascular resistance could favor increased right-to-left intracardiac shunting, resulting in systemic arterial oxygen desaturation and clinical decompensation during induction of anesthesia. Instead, intramuscular ketamine has been recommended as the anesthetic agent of choice for induction of these patients.⁵ Although both anesthetic techniques are widely used, insufficient data are available documenting the effects of either induction technique.

In children with cyanotic congenital heart disease the amount of oxygen that can be taken up in the lung is influenced in part by pulmonary blood flow.⁶ Pulmonary blood flow in cyanotic congenital heart lesions is that portion of systemic venous return that reaches the lung; the remainder enters the systemic circulation without being oxygenated and represents the net right-to-left shunt.⁶ The relative flows through the pulmonary and systemic circulations are determined by the resistances in the two circulations. Under resting conditions and in the absence of lung disease, it has been shown that an estimate of right-to-left shunting in these patients can be determined by measurement of systemic arterial blood oxygen saturation (Sa_{O_2}).⁷

The pulse oximeter can provide a reliable indicator of Sa_{O_2} over a wide range of oxygen saturations and is there-

fore a useful technique to evaluate Sa_{O_2} changes during induction in children.⁸⁻¹¹

Accordingly, we prospectively examined the comparative effects of a halothane/nitrous oxide inhalation induction technique and an intramuscular ketamine induction technique on Sa_{O_2} in children with cyanotic heart disease at risk for increasing right-to-left shunting and systemic oxygen desaturation.

METHODS

After Institutional Review Board approval and parental consent, 14 infants and children aged 4 months to 5 yr, scheduled for elective surgical procedures, were studied (table 1). Only patients with the potential for increasing the magnitude of right-to-left shunting and decreasing pulmonary blood flow were studied. Patients with systemic-pulmonary shunts (*e.g.*, Blalock-Taussig shunt, patent ductus arteriosus) were excluded from the study because they are unlikely to have a hypercyanotic episode and systemic oxygen desaturation. Children with complex systemic-pulmonary mixing blood flow patterns, as in transposition of the great vessels or univentricular hearts, are at similar low risk for hypercyanosis and were also excluded. Patients with lung disease were also excluded. Twelve patients had tetralogy of Fallot and two patients had a ventricular septal defect with pulmonary vascular obstructive disease and pulmonary artery hypertension.

Infants less than 9 months of age received no premedication and children greater than 9 months received meperidine 2 mg/kg, diazepam 0.1 mg/kg, nembutal 2 mg/kg, administered orally 75 min prior to the induction of general anesthesia. Following arrival of the patient in the operating room, a blood pressure cuff, precordial stethoscope, ECG, and pulse oximeter (Nellcor®) were applied. The pulse oximeter was applied to the right thumb in all cases. With the patient breathing room air and in the supine position, a set of control data were obtained. The patients were then randomized to one of two induction techniques. Anesthesia was induced in Group 1 patients with ketamine 6 mg/kg intramuscularly, followed by 100% oxygen at 5 l/min *via* a nonrebreathing system (Jackson-Rees modification of Ayre's T piece). Anesthesia was induced in Group 2 with halothane, nitrous oxide (70%) oxygen (30%); halothane was incrementally raised from 0 to 4% concentrations *via* a mask with a nonre-

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breathing system at a rate of 0.5% increments/2 respiratory breaths. In both groups heart rate (HR), mean systemic pressure (MAP), and Sa_{O_2} were monitored at baseline and then every 30 s during induction for 5 min. An intravenous catheter was placed and oral tracheal intubation performed after the 5-min observation period.

Sa_{O_2} determined noninvasively by the pulse oximeter was compared with oxygen saturation measured directly by co-oximetry from an arterial blood sample from the patient at the conclusion of the study. Between group and within groups comparisons were determined by one-way analysis of variance. Paired measurements for one variable between groups were then analyzed by two-sample *t* tests. Statistical significance was assumed at $P < 0.05$. Data are presented as mean \pm standard error of the mean.

RESULTS

Both groups had similar heart disease and there was no difference in age, starting MAP or Sa_{O_2} (table 1). Hemoglobin concentrations were similarly elevated in both groups of patients; 17.9 g/dl in the halothane group versus 17.6 g/dl in the ketamine group. During the induction of anesthesia, Sa_{O_2} increased significantly in both the halothane and the ketamine groups (fig. 1). At 5 min there was no difference in the Sa_{O_2} between the two groups. No significant change in MAP from baseline was seen with ketamine; however, induction with halothane and nitrous oxide was associated with a significant decrease in MAP (fig. 1). Simultaneous correlation of Sa_{O_2} determined directly by co-oximetry and noninvasively by the

TABLE 1. Demographic Data*

	Group 1†	Group 2‡
Number	7	7
Age (month)	11 \pm 4	12 \pm 3
Hemoglobin (g/dl)	17.6 \pm 1.1	17.9 \pm 1.5
Congenital heart lesion		
Tetralogy of Fallot	6	6
Ventricular septal defect		
Pulmonary artery hypertension	1	1
Baseline Sa_{O_2}	76 \pm 4	72 \pm 6

* Mean \pm SEM. No significant differences between groups.

† Intramuscular ketamine group.

‡ Halothane/nitrous oxide inhalation group.

pulse oximeter at the conclusion of the study revealed similar measurements over a broad range of oxygen saturations ($r = 0.94$).

DISCUSSION

Published reports are inconclusive and often conflicting with regard to the effects of various sedative and anesthetic agents on pulmonary blood flow in cyanotic heart disease. Rudolph and Danilowig cautioned against sedation, proposing that the supine position and inactivity decrease systemic vascular resistance and venous return, increasing right-to-left shunt.¹² Other authors have shown that general anesthesia with an inhalation agent, or intravenous morphine are effective treatments of hyper-

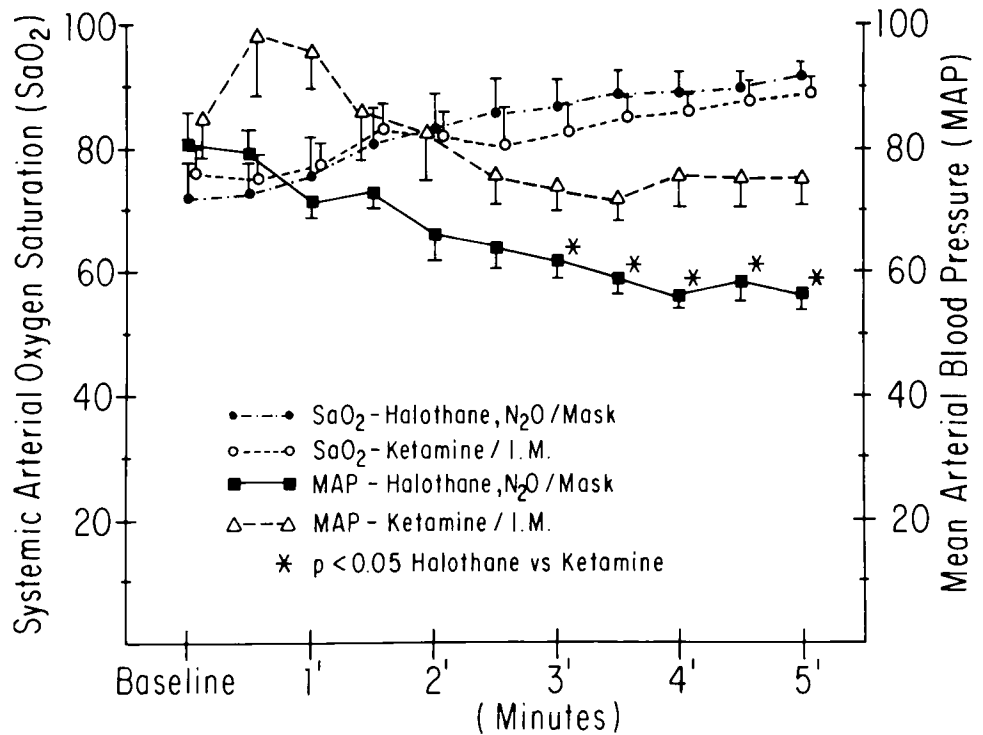


FIG. 1. Comparative changes in arterial oxygen saturation (Sa_{O_2}) and mean arterial blood pressure (MAP) during mask halothane nitrous oxide/oxygen and intramuscular ketamine/oxygen induction techniques.

cyanotic spells due to right-to-left shunting.^{13,14} Still others have recommended avoidance of a halothane/nitrous oxide induction due to risks of hypotension, which may worsen right-to-left shunting and decrease pulmonary blood flow.^{4,5} Many of these authors have advocated the use of an intramuscular ketamine induction in children with cyanotic heart disease because systemic vascular resistance and overall cardiovascular stability are maintained.

Using the pulse oximeter during the induction of anesthesia in children with selective cyanotic congenital heart defects, we were able to study the effects of two hemodynamically diverse induction techniques on Sa_{O_2} in patients at high risk for potential increases in right-to-left shunting and systemic desaturation. All of the patients in both groups had a history of hypercyanotic spells and had a significant elevation of hemoglobin concentration, suggesting chronic decreased pulmonary blood flow.

Under the conditions of our study, both induction techniques were associated with a significant increase in Sa_{O_2} . Following administration of ketamine in our subjects, MAP initially increased because of patient agitation due to the intramuscular injection. After 2 min MAP returned to baseline and was stabilized at that level.

In the halothane group the increase in Sa_{O_2} occurred despite a significant decrease in MAP. A large decrease in MAP would be expected to result in a net increase in right-to-left shunting, resulting in systemic desaturation. However, decreases in systemic arterial oxygen saturation were not seen in any of these high-risk patients studied. There are several explanations for our observations. Depending on the magnitude of pulmonary blood flow, breathing 30% oxygen during induction may have increased, in a limited manner, Pa_{O_2} and thus Sa_{O_2} in those children with large pulmonary blood flows. Second, in our patients tissue oxygen utilization was decreasing as the children progressed from an awake, premedicated state to the anesthetized state. This trend may have resulted in both a decrease in pulmonary blood flow requirement and an increase in mixed venous oxygenation. Keeping the pulmonary blood flow constant, a rise in mixed venous oxygenation has been shown to increase Sa_{O_2} significantly in cyanotic heart disease.¹⁵ Another explanation may be the decrease in dynamic infundibular pulmonary stenosis in those patients with tetralogy of Fallot due to the negative inotropic effect of halothane on the myocardium resulting in a decrease resistance to right ventricular outflow, as has been described for other negative inotropic drugs.¹⁶ Finally, the decrease in blood pressure might have been due to decreased cardiac output rather than a decrease in systemic vascular resistance. Under these conditions pulmonary blood flow was sufficient to increase Sa_{O_2} . This possibility is the most likely because cardiac output was not measured, and halothane is known to produce venodilation and a decrease in myo-

cardial contractility while maintaining systemic vascular resistance in adults and children.¹⁷

In conclusion, induction with intramuscular ketamine or inhalation of halothane and nitrous oxide with oxygen was accompanied by increased arterial oxygen saturation in children with cyanotic congenital heart disease at risk for right-to-left shunting. We believe that either technique is safe and effective in cyanotic patients where the development of further hypoxemia is a concern.

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