

## Propofol Does Not Modify the Hemodynamic Status of Children with Intracardiac Shunts Undergoing Cardiac Catheterization

D. Gozal,<sup>1</sup> A.J.J.T. Rein,<sup>2</sup> A. Nir,<sup>2</sup> Y. Gozal<sup>1</sup>

<sup>1</sup>Department of Anesthesiology and Critical Care Medicine, Hadassah University Hospital and the Hebrew University School of Medicine, Post Office Box 12000, Jerusalem 91120, Israel

<sup>2</sup>Department of Pediatric Cardiology, Hadassah University Hospital and the Hebrew University School of Medicine, Post Office Box 12000, Jerusalem 91120, Israel

**Abstract.** Immobility and cardiovascular stability are required for cardiac catheterization. Pediatric patients need a type of sedation that also allows spontaneous ventilation without supplemental oxygen. Propofol has been adequate in hemodynamically stable patients with congenital heart disease undergoing cardiac catheterization. However, mild systemic hypotension caused by propofol may increase a preexisting right-to-left shunt. The aim of this study is to evaluate, in pediatric patients scheduled for cardiac catheterization, the effects of propofol on systemic and pulmonic circulations. Fifteen patients aged 18 months to 9 years were studied. After a fast of 4–6 hours for solid food, the patient arrived at the cardiac catheterization suite, where an IV catheter was placed. Usual monitoring was used. For sedation, without supplemental oxygen, patients received 1 µg/kg of fentanyl followed by propofol (1–2 mg/kg) titrated to immobility during preparation of the groin. A continuous infusion of propofol (100 µg/kg/min) was also started to obtain immobility during the procedure. Hemodynamic data, including systemic venous, pulmonary artery and vein, aortic saturations, and pressures, were recorded;  $Q_p$  and  $Q_s$  were calculated. The same set of data was re-recorded 4 minutes after discontinuation of propofol and when the patient was responding to tactile stimuli. Despite lower pressures during propofol infusion, as compared with those pressures measured after discontinuation of propofol, the extent of the intracardiac shunt remained unchanged. Propofol seems to be an adequate sedative agent for pediatric patients undergoing cardiac catheterization, including those with intracardiac shunts.

**Key words:** Heart defects, congenital — Catheterization, cardiac — Anesthetics, intravenous — Propofol

Cardiac catheterization is required for a subgroup of patients with congenital heart disease for the purpose of hemodynamic assessment or anatomical definition. During the procedure, the pediatric patient should not be allowed to move. Movement may decrease the quality of angiography, may increase the radiation exposure, and, above all, may increase the risk of vascular trauma [2]. Thus cardiac catheterization must be performed under sedation or general anesthesia in this population. Hemodynamic stability during cardiac catheterization is usually crucial. Normal physiology is least disturbed when the patient spontaneously breathes room air. Lebovic et al. [3] have shown that propofol is an adequate sedative in hemodynamically stable patients with congenital heart disease who are undergoing cardiac catheterization. However, mild systemic hypotension caused by propofol may increase a preexisting right-to-left shunt.

The aim of this study is to evaluate the effects of propofol on systemic and pulmonary circulations in pediatric patients with intracardiac shunts.

### Patients and Methods

The study was approved by the Institutional Review Board Committee. One parent or one guardian of each child signed a written informed consent. Fifteen patients, ASA physical status II or III, who were scheduled for elective diagnostic cardiac catheterization, were included in the study. All patients presented intracardiac shunts (diagnosed by echocardiography). Patient age ranged from 18 months to 9 years. Exclusion criteria included patients requiring mechanical ventilation or inotropic support. No premedication was given, and children were fasted according to ASA guidelines: 4–6 hours for milk and solid food, and 2 hours for clear fluids. EMLA

**Table 1.** Demographic data

Patients	Age (years)	Weight (kg)	Diagnosis
1	5	12.5	Pulmonary atresia-ASD
2	2	12.0	VSD
3	2	9.0	Pulmonary atresia-VSD
4	7	22.0	Pulmonary atresia-VSD
5	9	28.0	ASD
6	3	13.5	VSD
7	4	11.0	DORV-VSD
8	2	9.0	TGA-VSD
9	4	20.0	VSD
10	8	15.0	AV canal-TGA
11	1.5	10.0	VSD
12	7	22.0	TOP
13	5	11.5	VSD
14	3	11.0	ASD
15	2	8.0	TOP

ASD, atrial septal defect; VSD, ventricular septal defect; DORV, double outlet right ventricle; TGA, transposition of the great arteries; AV canal, atrioventricular canal; TOF, tetralogy of Fallot.

(eutectic mixture of local anesthetics: 2.5% lidocaine base and 2.5% prilocaine base) cream was applied on both hands 1 hour prior to the procedure. In the catheterization suite, an intravenous catheter was placed. Routine monitoring included electrocardiography, blood pressure (noninvasive and invasive when the cardiac catheter was introduced through the femoral artery), preductal pulse oximetry, end-tidal CO<sub>2</sub> for respiratory rate monitoring, and temperature. After administration of an analgesic dose of fentanyl (1 µg/kg), patients were induced with propofol (1–2 mg/kg) titrated to immobility during preparation of the groin. A continuous infusion was then started (100 µg/kg/min). If movement occurred, a bolus of one-half the induction dose of propofol was administered, and the infusion rate was increased by 50%.

The patients were kept on room air and spontaneous breathing throughout the procedure.

Systemic venous, pulmonary artery and vein, and aortic pressures were recorded. Analysis of blood gases from the same sites was performed, and oxygen saturations were recorded. Pulmonary and systemic blood flows ( $Q_p$ ,  $Q_s$ ) were calculated by the Fick principle using O<sub>2</sub> content as a marker; pulmonary and systemic vascular resistances were calculated as well. Two sets of data were obtained: the first during propofol anesthesia, and the second about 4 minutes after discontinuation of the propofol infusion and when the patient was responding to tactile stimuli.

Results are expressed as mean ± SD. Statistical analysis used the paired Student's *t*-test, *p* < 0.05 was considered significant.

## Results

Demographic data are presented in Table 1. All patients completed the study. No complications were recorded; specifically no patient needed supplemental oxygen because of arterial oxygen desaturation. Likewise, no intervention was required to treat changes in heart rate or mean arterial pressure.

Oxygen saturations were not statistically different during propofol infusion and following its discon-

**Table 2.** Oxygen saturation (%)

Site	During propofol	After propofol
SVC	70.7 ± 9.8	73.2 ± 6.4
PA	78.0 ± 11.0	80.3 ± 6.7
PV	96.2 ± 2.2	95.6 ± 2.3
Aorta	87.8 ± 12.3	90.8 ± 5.7

Data are mean ± SD. SVC, superior vena cava; PA, pulmonary artery; PV, pulmonary vein.

**Table 3.** Measured pressures (mmHg).

Site	During propofol	After propofol
SVC	7.4 ± 1.6	7.0 ± 1.4
PA syst	36.1 ± 2.5	39.1 ± 0.9
PA diast	15.4 ± 1.9	17.0 ± 1.2
PA mean	21.2 ± 0.9	24.9 ± 1.3
PV	8.8 ± 2.3	9.1 ± 1.5
Aorta syst	90.8 ± 1.6	102.4 ± 1.4
Aorta diast	53.6 ± 1.5	61.9 ± 1.1
Aorta mean	71.4 ± 1.5	81.0 ± 1.2

Data are mean ± SE. SVC, superior vena cava; PA., pulmonary artery systolic pressure; PA diast, pulmonary artery diastolic pressure; PV, pulmonary vein; Aorta syst, aorta systolic pressure; Aorta diast, aorta diastolic pressure.

**Table 4.** Calculated hemodynamic values.

	During propofol	After propofol
$Q_p$ (L/min/m <sup>2</sup> )	6.0 ± 1.6	6.3 ± 1.5
$Q_s$ (L/min/m <sup>2</sup> )	4.3 ± 0.9	4.5 ± 0.7
$Q_p/Q_s$	1.43 ± 0.5	1.43 ± 0.5
SVR (Wood units/m <sup>2</sup> )	16.5 ± 2.3	16.6 ± 3.3
PVR (Wood units/m <sup>2</sup> )	2.1 ± 1.2	3.1 ± 1.5

Data are mean ± SE.  $Q_p$ , pulmonary blood flow;  $Q_s$ , systemic blood flow; SVR, systemic vascular resistance; PVR, pulmonary vascular resistance.

tinuation (Table 2). The average time interval between discontinuation of propofol and response to tactile stimuli was 4 minutes. All measured pressures tended to be lower during propofol infusion as compared with those recorded after the cessation of the infusion. However, the difference was not significant (Table 3). As shown in Table 4, there was no change in the extent of the intracardiac shunt.  $Q_p/Q_s$  remained unchanged in both states.

## Discussion

This study shows that, despite lower systemic and pulmonary pressures, propofol did not modify the

characteristics of the intracardiac shunt. Propofol seems to have a favorable profile for cardiac catheterization. Lebovic et al. [3] have shown that propofol infusion with fentanyl analgesia was associated with significantly shorter recovery times than ketamine/midazolam anesthesia in pediatric cardiac catheterization procedures. Propofol, used as the sole anesthetic, may be not sufficient since it lacks an analgesic effect [5]. At our institution, cardiologists always infiltrate the puncture site with lidocaine, so that the main indication of the sedation is to keep the patient still. Every effort is made to maintain spontaneous ventilation and to minimize inspired oxygen concentration so that the hemodynamic data obtained by the cardiologist will be meaningful. The sedation technique used in this study responded well to the latter criteria. Another important issue, raised by Lebovic et al. [3], was that the mild decrease in arterial oxygen saturation in their study, during induction with propofol, was due to hypotensive episodes and thus to an increased right-to-left shunt. Our study specifically assessed this point and does not support this assumption.

Although we did not measure plasma concentrations of propofol during the continuous infusion and when the patient was waking up (responding to tactile stimuli), we may reasonably assume that the level was low and was not clinically significant 4 minutes after discontinuation of the infusion. The reported pharmacokinetic profile of propofol is such that after 4 minutes, a low level remains [4]. The rapid

decline in propofol concentration to a level below the one required for hypnosis permits rapid awakening [1]. The increase in all pressures, as shown in Table 3, supports this interpretation. We did not think it ethical to keep the cardiac catheter *in situ* until the child was fully awake just to obtain measurements without any trace of propofol. It may also be dangerous to keep the catheter in place; the child would start moving, and the risk of perforation would be great.

In conclusion, this study demonstrates that propofol sedation, with the addition of an analgesic dose of fentanyl, is a satisfactory method for pediatric patients undergoing cardiac catheterization, including those with intracardiac shunts.

## References

1. Adam HK, Kay B, Douglas EJ (1982) Blood disopropofol levels in anaesthetized patients. *Anaesthesia* 37:536–540
2. Javorski JJ, Hansen DD, Laussen PC, Fox ML, Lavoie J, Burrows FA (1995) Paediatric cardiac catheterization: innovations. *Can J Anaesth* 42:310–329
3. Lebovic S, Reich DL, Steinberg LG, Vela FP, Silvay G (1992) Comparison of propofol versus ketamine for anesthesia in pediatric patients undergoing cardiac catheterization. *Anesth Analg* 74:490–494
4. Saint-Maurice C, Cockshott ID, Douglas EJ, Richard MO, Harmey JL (1989) Pharmacokinetics of propofol in young children after a single dose. *Br J Anaesth* 63:667–670
5. Smith I, While PF, Nathanson M, Gouldson R (1994) Propofol: an update on its clinical use. *Anesthesiology* 81:1005–1043