

Another use for this display technique is related to the EEG processor display—display of the spectrum analysis of other physiologic variables. Possibilities include the phonocardiogram, the electromyogram, the arterial pressure waveform, and the ECG. Still another application of density modulation is to display annotations across the strip-chart channel (optimally 10–20 characters) by using raster alphanumeric characters similar to television displays. Events

concerned with surgical procedures and anesthesia, such as drug injection, position change, or hemorrhage, could be displayed, along with physiologic variables, by use of a small keyboard.

#### REFERENCES

1. Fleming RA, Smith NT: An inexpensive device for analyzing and monitoring the electroencephalogram. *ANESTHESIOLOGY* 50:456–460, 1979

Anesthesiology  
50:546–547, 1979

## Halothane–Nitrous Oxide Anesthesia in a Patient Receiving High-dose Propranolol

ROBERT K. STOELTING, M.D.\*

Kopriva *et al.*<sup>1</sup> reported that maintenance of propranolol therapy until five to six hours before anesthetic induction in patients with coronary-artery disease did not produce adverse hemodynamic changes during nitrous oxide–halothane anesthesia. In their cases the doses of propranolol averaged 140 mg/day and never exceeded 240 mg/day. They pointed out that hemodynamic responses might be different in patients receiving higher doses of propranolol. Therefore, we felt it important to describe uneventful nitrous oxide–halothane anesthesia in a patient receiving propranolol, 960 mg/day.

#### REPORT OF A CASE

A 50-year-old, 55-kg, 157-cm woman with renovascular hypertension was scheduled for an elective right-renal-artery-bypass graft. Medications included propranolol, 960 mg/day, triamterene–hydrochlorothiazide, and chlorthalidone. Resting supine and standing blood pressures were 180–220/100–120 torr. Heart rate was 66–72 beats/min. Preoperative electrolytes, chest radiograph and electrocardiogram were normal.

Intravenous infusion of physiologic saline solution 200 ml/hr, was started at 6:30 PM the evening before operation. The last dose of propranolol, 240 mg, was given at 10:00 PM. Preanesthetic medication was morphine, 10 mg, and atropine, 0.4 mg, im. After arrival of the patient in the operating room, diazepam, 10 mg iv, was administered prior to placement of a radial-artery catheter and a Swan-Ganz catheter.

Isoproterenol, 4 µg, iv, did not change awake heart rate or blood pressure. Subsequent anesthetic induction was with thiopental–succinylcholine, followed by tracheal intubation and controlled ventilation. Sodium nitroprusside, 100 µg, iv, was administered 15 sec before beginning direct laryngoscopy for tracheal intubation. Anesthesia was maintained with 60 per cent nitrous oxide

and 0.25–0.75 per cent inspired halothane, plus *d*-tubocurarine for skeletal muscle relaxation. Residual *d*-tubocurarine effect at the conclusion of the operation was reversed with neostigmine, 2.5 mg, combined with atropine, 1.0 mg. Total operative time was 200 min. Intraoperative fluids were lactated Ringer's solution, 2,800 ml, and 5 per cent dextrose in water, 500 ml. Urinary output was 1,200 ml and estimated blood loss 300 ml.

Hemodynamic responses to induction, tracheal intubation, and anesthetic maintenance are summarized in table 1. Arterial blood propranolol concentration just before anesthetic induction was 118 ng/ml (40–85 ng/ml considered therapeutic).

#### DISCUSSION

This report describes uneventful anesthesia of a patient maintained on high doses of propranolol until near the time of operation. The presence of significant beta-adrenergic receptor blockade at the time of anesthetic induction was suggested by the absence of heart rate or blood pressure changes in response to a bolus injection of isoproterenol. Romagnoli and Keats<sup>2</sup> reported this dose of isoproterenol increased heart rate 30 beats/min and systolic blood pressure 30 torr in patients who had not received propranolol or in patients from whom propranolol was withdrawn 24–48 hours preoperatively whose blood concentrations were less than 23 ng/ml. The lack of response to isoproterenol in our patient was consistent with the concentration of propranolol in blood, 118 ng/ml, at the time of anesthetic induction. Coltart and Shand<sup>3</sup> have suggested that complete beta-adrenergic receptor blockade will be produced by more than 100 ng/ml propranolol in the blood.

Despite the presence of significant beta-adrenergic receptor blockade, the circulatory responses during anesthetic induction and maintenance were not different from responses observed in patients not receiving propranolol. The decreases in mean arterial

\* Professor and Chairman, Department of Anesthesia, Indiana University School of Medicine, Indianapolis, Indiana 46202.

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Address reprint requests to Dr. Stoelting.

TABLE 1. Hemodynamic Responses

	Blood Pressure (torr)	Heart Rate (Beats/Min)	Pulmonary-artery Occlusion Pressure (torr)	Cardiac Output (l/min)	Temperature (C)	F <sub>I</sub> O <sub>2</sub>	PaO <sub>2</sub> (torr)	PaCO <sub>2</sub> (torr)
Preinduction	200/100	72	9	6.0		.21	91	41
One minute after thiamylal-succinylcholine	180/90	72	7	5.2				
Tracheal intubation	225/100	76	10	5.2				
N <sub>2</sub> O-halothane for 5 min	154/80	68	6	6.2	36.0	.4	139	29
Five minutes after dTc, 24 mg	120/64	66	5	5.8				
Just before incision of skin	132/60	66	6	5.6				
Five minutes after incision of skin	148/74	64	5	4.9				33
After one hour of operation	152/88	64	7	4.7	35.2	.4	123	35
End operation	160/100	72	7	4.8	34.6	.4	114	31

pressure and cardiac output following rapid-sequence administration of thiamylal-succinylcholine were similar to those described to occur in normotensive patients in the absence of propranolol.<sup>4</sup> Direct laryngoscopy for tracheal intubation took 30 sec and was associated with a 25-torr increase above awake levels in mean arterial pressure. This is similar to the increases we have observed in patients not receiving propranolol undergoing laryngoscopy of similar duration with prior sodium nitroprusside. The heart rate did not change during the induction-intubation sequence, probably reflecting propranolol effect.

Institution of controlled ventilation with 60 per cent nitrous oxide in 0.25-0.75 per cent halothane initially reduced blood pressure without a change in heart rate or cardiac output. The lowest systolic blood pressure occurred 5 min after the administration of *d*-tubocurarine. The magnitude of this decrease was similar to that previously found in the absence of propranolol.<sup>5</sup> Circulatory responses to incision of the skin were minimal. Pulmonary-artery occlusion pressures did not reveal severe left ventricular depression.

Sprague<sup>6</sup> reported bradycardia following administration of neostigmine-atropine to an anesthetized patient receiving propranolol. It was speculated that parasympathetic predominance produced by propranolol accentuated the muscarinic effects of neostigmine. He recommended that atropine precede neostigmine instead of being administered simultaneously. Nevertheless, bradycardia was not observed in our patient, who received a neostigmine-atropine mixture.

Animal studies have confirmed that excessive circulatory depression did not occur when halothane anesthesia was administered to dogs receiving 20 mg/kg/day propranolol.<sup>7</sup> It was suggested from these data that complete beta-adrenergic blockade was equivalent to increasing the inspired halothane concentration 0.5 per cent—*i.e.*, blood pressure reduction with 1.5 per

cent halothane was similar to that produced by 1.0 per cent halothane plus beta-adrenergic receptor blockade. Circulatory changes in our patient during low-dose administration of halothane did not differ from those observed in normal patients. In addition to minimal anesthetic concentrations, preoperative hydration and minimal intraoperative blood loss probably contributed to circulatory stability.

Careful monitoring of the cardiovascular system is important in the anesthetic management of patients maintained on propranolol until the time of operation. Appropriate monitors during major operations on patients receiving high-dose propranolol may include systemic and pulmonary-artery catheters. This report describes a case in which such a patient underwent uneventful anesthesia and operation despite maintenance of high-dose propranolol until near the time of operation.

#### REFERENCES

1. Kopriva CJ, Brown ACD, Pappas G: Hemodynamics during general anesthesia in patients receiving propranolol. *ANESTHESIOLOGY* 48:28-33, 1978
2. Romagnoli A, Keats AS: Plasma and atrial propranolol after preoperative withdrawal. *Circulation* 52:1123-1127, 1975
3. Coltart DJ: Plasma propranolol levels in the quantitative assessment of  $\beta$ -adrenergic blockade in man. *Br Med J* 3:731-734, 1970
4. Stoelting RK: Circulatory changes during anesthetic induction: Impact of *d*-tubocurarine pretreatment, thiamylal, succinylcholine, laryngoscopy and tracheal intubation. *Anesth Analg (Cleve)* 55:77-81, 1976
5. Stoelting RK: The hemodynamic effects of pancuronium and *d*-tubocurarine in anesthetized patients. *ANESTHESIOLOGY* 36:612-615, 1972
6. Sprague DH: Severe bradycardia after neostigmine in a patient taking propranolol to control paroxysmal atrial tachycardia. *ANESTHESIOLOGY* 42:208-210, 1975
7. Roberts JG, Foex P, Clarke TNS, et al: Haemodynamic interactions of high dose propranolol pretreatment and anesthesia in the dog. I: Halothane dose response studies. *Br J Anaesth* 48:315-324, 1976