

In Vivo Response of Air-filled Balloon-tipped Catheters to Nitrous Oxide

P. M. H. du Boulay, M.B.B.S., F.F.A., R.C.S.*, and Michael L. Nahrwold, M.D.†

Four balloon-guided pulmonary artery catheters from each of two manufacturers were placed in eight anesthetized dogs, and balloon volumes were measured at randomized time intervals from 1 to 30 min during (1) ventilation with 100% oxygen and (2) ventilation with 70% nitrous oxide in oxygen. Catheters from one manufacturer showed no increase in balloon volume in either condition. The balloon volumes of catheters from the second manufacturer increased up to 17% during ventilation with nitrous oxide. Balloons of these catheters required less inflation pressure than those from the first manufacturer. It was concluded that the effects of nitrous oxide on the balloon volumes of pulmonary artery catheters *in vivo* are of little clinical significance. (Key words: Anesthetics, gases: nitrous oxide. Equipment: catheters, flow-directed. Monitoring: vascular.)

THE ABILITY of nitrous oxide to diffuse into the balloon of a balloon-guided pulmonary artery (PA) catheter has been described by Kaplan *et al.*¹ These authors demonstrated a maximal increase of 125% in the volume of gas within the balloon following 10-min exposure to a mixture of 75% N₂O/25% O₂. They suggested that expansion of the PA catheter balloon might account for difficulty in passing a PA catheter through the pulmonary outflow tract in pediatric patients. Furthermore, they claimed that their data support the concepts of inflating the balloon of PA catheters with the same gas mixture inspired by the patient or of deflating the balloon every few minutes if air is used to inflate the balloon during N₂O anesthesia.

The study of Kaplan *et al.*¹ was performed *in vitro* by placing the PA catheter in a vessel containing the gas mixture under study. This study is an attempt to confirm these findings *in vivo*, and to compare the responses of PA catheters supplied by two different manufacturers.

* Instructor, Department of Anesthesiology, The University of Michigan. Present address: Senior Registrar, Shackleton Department of Anaesthetics, Level E, Centre Block, Southampton General Hospital, Shirley, Southampton, England.

† Professor, Department of Anesthesiology, The University of Michigan.

Received from the Anesthesia Research Laboratories at the University of Michigan, Ann Arbor, Michigan. Accepted for publication June 2, 1982.

Address reprint requests to Dr. Nahrwold: Department of Anesthesiology, The University of Michigan Hospital, Ann Arbor, Michigan 48109.

Materials and Methods

The investigation was carried out in eight mongrel dogs of either sex, weighing 19–26 kg. Anesthesia was induced with 30–40 mg/kg pentobarbital, iv, and maintained with incremental doses of pentobarbital. Following endotracheal intubation, the animals were paralyzed with 0.1 mg/kg pancuronium bromide and ventilated using an Air Shields Ventimeter (Ohio Medical Products, Madison, Wisconsin) to give a PaCO₂ of 30–38 mmHg. The right femoral artery was cannulated for monitoring of systemic pressure and measurement of blood gases using an ABL-2 blood-gas analyzer (Radiometer). Fresh gas supply was delivered via calibrated flowmeters (Ohio Medical Products), and the inspired gas composition was confirmed using an oxygen analyzer (Critikon Division, McNeil Laboratories). During placement of the catheters, the animals were ventilated with 100% O₂.

Eight 7F PA catheters were used, four from Edwards Laboratories Inc. and four from Electronics for Medicine (E for M). In each animal, a new PA catheter was placed via cut-down to the right internal jugular vein, and using pressure waveform display as a guide, the catheter was advanced into the pulmonary artery and secured so that there was no wedging at maximal balloon inflation (1.5 ml). Each animal was assigned randomly to either an Edwards or E for M group. The balloon port was connected, via an epoxy-sealed three-way stopcock, to both a calibrated gas-tight syringe (Hamilton) and a pressure transducer (Gould Statham P23Gb). Intra-balloon pressure was displayed via amplifier and Gould 2600 recording system. With the balloon open to the atmosphere, the pressure display was set at zero, which thus served as a reference point to prevent excessive aspiration from the PA catheter.

Measurements were made in each animal both during ventilation with 100% O₂ and during ventilation with a mixture of 70% N₂O/30% O₂. The order in which these two conditions occurred was randomized, and adequate time was allowed for near-equilibration of arterial blood with the inspired gases. This was approximately 20–30 min, and was confirmed by observing a stabilization of the arterial partial pressure of oxygen during at least three consecutive measurements. Room

air (1.5 ± 0.025 ml) was used to inflate the balloons for either 1, 2, 3, 4, 5, 10, 15, or 30 min, measured by stopwatch. The order in which these time periods occurred was randomized. At the end of each time period, the same gas-tight syringe was used to aspirate the system to a pressure of zero, and the volume of gas aspirated was measured. The balloon, transducer dome, and syringe were flushed with air between each measurement.

At the end of each *in vivo* experiment, the PA catheter was removed, and using the same measuring system, an inflation pressure vs. inflation volume curve for the balloon was determined in air. The balloon was inflated in increments of 0.1 ml from 0 to 1.5 ml, with complete deflation between each pressure recording. Measurements were then repeated for inflations starting at 1.5 ml and decreasing to zero in increments. The mean values of the two pressures measured at each incremental volume were those used in figure 1.

The data were subjected to analysis of variance followed by Student's paired and unpaired *t* tests when appropriate.

Results

Balloon volumes of E for M PA catheters slowly decreased with time, regardless of whether the dogs were ventilated with oxygen or nitrous oxide (table 1). At each time of inflation, there was no statistically significant difference in volume when dogs breathed nitrous oxide as compared with oxygen. The balloon volumes of Edwards PA catheters also decreased slowly with time when the animals were ventilated with 100% oxygen. However, ventilation with 70% nitrous oxide caused the Edwards balloon volumes to increase to a maximum mean volume of 1.76 ml at 5 min, after which the volumes gradually decreased. Even at maximum balloon distension, pulmonary artery pressure tracings did not show a wedge pattern. With these catheters, differences in volumes between 100% oxygen and 70% nitrous oxide were statistically significant for all time periods of balloon inflation except 30 min.

Balloon inflation pressure vs. inflation volume curves for the catheters are shown in figure 1. In all cases, as inflating volume increased, pressure also increased up to a point at which pressure rapidly decreased as the balloon began to fill. It is evident that the inflation volume at which this occurred was earlier for Edwards PA catheters than for E for M PA catheters. Furthermore, once balloon inflation had started, pressures within Edwards catheter balloons were lower for a given injected volume than they were for E for M balloons.

Discussion

Using Edwards PA catheters *in vitro*, Kaplan *et al.*¹ showed an alarming increase in balloon volume of more

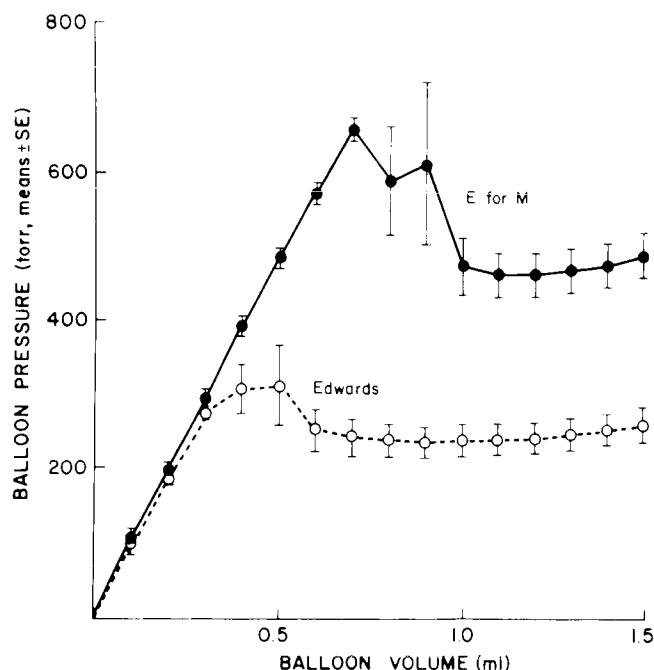


FIG. 1. Inflation pressure vs. inflation volume curves for Edwards and E for M pulmonary artery catheters.

than 100% after a 10-min exposure to 75% nitrous oxide in oxygen. Based on these findings, the authors recommended that "... if balloons are filled with air during placement under N₂O anesthesia, they should be deflated every few minutes. This may be particularly critical in children."¹ The present study tried to confirm these results in a more clinically relevant experimental situation. To our surprise, we were unable to do this. In dogs ventilated with nitrous oxide, the balloons of Edwards catheters did expand significantly with time, but the maximal increase over the original volume was

TABLE 1. Balloon Volumes of Pulmonary Artery Catheters in Dogs Breathing 100% Oxygen or 70% Nitrous Oxide in Oxygen

Time (min)	Balloon Volume (ml, Means ± SE)			
	E for M Catheters		Edwards Catheters	
	100% O ₂	70% N ₂ O	100% O ₂	70% N ₂ O
0	1.50	1.50	1.50	1.50
1	1.45 ± 0.03	1.48 ± 0.05	1.48 ± 0.01	1.58 ± 0.02*
2	1.41 ± 0.04	1.46 ± 0.06	1.47 ± 0.01	1.62 ± 0.01*†
3	1.36 ± 0.06	1.43 ± 0.07	1.44 ± 0.01	1.66 ± 0.03*†
4	1.32 ± 0.04	1.39 ± 0.08	1.42 ± 0.01†	1.66 ± 0.02*†
5	1.25 ± 0.08	1.32 ± 0.10	1.39 ± 0.02	1.76 ± 0.05*†
10	1.05 ± 0.07	1.10 ± 0.14	1.28 ± 0.03†	1.75 ± 0.10*†
15	0.86 ± 0.15	1.08 ± 0.18	1.16 ± 0.05	1.68 ± 0.07*†
30	0.52 ± 0.10	0.65 ± 0.18	0.97 ± 0.09†	1.38 ± 0.14†

* Different from 100% O₂ using the same catheter (*P* < 0.05).

† Different from E for M in dogs ventilated with the same gas mixture (*P* < 0.05).

only 17%, far less than the more than 100% reported by Kaplan *et al.* If as a "worst case" it is assumed that at maximal balloon inflation there is no further pressure increase associated with balloon volume increase, and that the balloon is a sphere, one can show that a 17% increase in volume will lead to an increase in balloon diameter of 5.5%. Data from Edwards Laboratories, as presented by Kaplan *et al.*, indicate that at an inflation volume of 1.5 ml, the balloon diameter is 12.7 mm. An increase in this diameter of 5.5% would represent an increase of only 0.7 mm.

We were disturbed by the discrepancy between our study and that of Kaplan *et al.* Therefore, we approximated their study *in vitro* and, in fact, confirmed their findings (data not presented). Eisenkraft and Eger have performed a similar *in vitro* study using gas-equilibrated saline rather than a gaseous medium to surround the balloons.² Their results were qualitatively similar to those of Kaplan *et al.* We have no clearcut explanation for this. However, there are a number of additional factors present in an *in vivo* model. These would include

presence of a gas-balloon-blood interface, intravascular pressure, and a temperature of 37° C.

In animals ventilated with 100% oxygen, diameters of the balloons did not increase, regardless of the brand of catheter utilized. In addition, in animals ventilated with 70% nitrous oxide, the diameters of E for M catheters did not increase with time. This suggests that these catheters may be somewhat safer under these conditions if the balloon inadvertently is left inflated. However, it is important to realize that the pressure within an E for M balloon is potentially twice that of an Edwards balloon. This may be a much more hazardous factor in terms of damage to the pulmonary arterial wall than a small increase in balloon diameter.

References

1. Kaplan R, Abramowitz MD, Epstein BS: Nitrous oxide and air-filled balloon-tipped catheters. *ANESTHESIOLOGY* 55:71-73, 1981
2. Eisenkraft JB, Eger EI II: Nitrous oxide and Swan-Ganz catheters. *Anesth Analg (Cleve)* 61:308-309, 1982