airway pressure in the nonventilated lung to vary less than 1 cmH_2O from the 10 cmH_2O indicated on the valve (with an 8 $l \cdot min^{-1}$ oxygen flow rate). Other O_2 flow rates were not tested clinically; however, bench testing of the circuit with O_2 flow rates varying from 1 to 10 $l \cdot min^{-1}$ produced less than 10% (1 cmH_2O) variation from the indicated pressure at all O_2 flow rates tested. Clinically, use of this circuit to provide CPAP and O_2 insufflation to the nonventilated lung during continuous OLV significantly improved Pa_{O_2} in patients undergoing thoracotomy, as shown in figure 2.

Capan *et al.*[1] have described a similar system to provide CPAP and O_2 insufflation to the nonventilated lung during which they used an adjustable pressure relief valve to limit the escape of oxygen from the insufflation circuit. This system has the disadvantage that an inadvertent occlusion of the valve or an increase in flow rate could create high airway pressures. The system we describe utilizes reusable materials that are readily available within most anesthesiology departments, which gives it the combined advantages of availability and simplicity. Over-pressure is highly unlikely, since 10 cmH_2O pressure relief (independent of O_2 flow rate from 1 to 10 $l \cdot min^{-1}$) is designed into the system. We observed no difficulty with the circuit; furthermore, 10 cmH_2O CPAP does not appear to interfere with surgical exposure and may even facilitate intralobar dissection.

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Does Isoflurane Really Preserve Baroreflex Responsiveness Better than Halothane or Enflurane?

To the Editor:—Kotrly *et al.* assert that "the data indicate isoflurane preserves baroreflex regulation of heart rate better than either halothane or enflurane" and suggest that isoflurane may be the preferred inhalational anesthetic in clinical situations where baroreflex responsiveness would be important, *e.g.*, acute hypovolemia or position changes under anesthesia.[1] There are four reasons why I believe these conclusions to be premature.

First, if the pressor slopes (R-R interval *vs.* blood pressure) for isoflurane from Duke *et al.*[2] are plotted *versus* MAC multiples as in figure 6 of reference 1, the line for isoflurane can be superimposed on the halothane–N_2O one and is parallel to those for halothane, enflurane, and enflurane–N_2O. Even if the concentration of isoflurane is reduced but the level of anesthesia maintained with N_2O, the isoflurane–N_2O line can be superimposed on the isoflurane one. Thus, the data of Duke *et al.* do not show the degree of sparing of baroreflex responsiveness that Kotrly observed.

Given the above, the question is raised as to how comparable are the experimental methods from the two different laboratories. Duke controlled ventilation to keep the arterial P_{CO_2} about 35 mmHg.[3] It is not clear from Kotrly's article whether ventilation was controlled and what the actual arterial P_{CO_2}s were. A statement is made that "all subjects resumed spontaneous ventilation prior to the tests carried out during anesthesia."[1] Thus, the arterial P_{CO_2} may have been 50 mmHg.[4] An elevated CO_2 tension could increase sympathetic outflow from the vasomotor center, one component of the baroreflex arc.

Thirdly, in two elegant studies in dogs, Kampine's group determined that both halothane and isoflurane, with a thiopental background, attenuated in a dose-related manner all the individual components of the baroreceptor reflex arc except the baroreceptor itself, which was sensitized.[5,6] When the intact reflex arc was studied, isoflurane at 1 MAC appeared to be less depressant than halothane at 1 MAC. However, the control slopes (R-R interval in ms *vs.* mean BP in mmHg) in the conscious dogs are so different (12.38 ± 3.6 ms/mmHg for halothane and 59.4 ± 16.5 ms/mmHg for isoflurane). If the halothane data were normalized to the same

starting slope as for isoflurane, the slope depression for 1.3% isoflurane and 0.75% halothane may not be significantly different.

Lastly, the clinical situations in which baroreceptor responsiveness would be important are equivalent to the depressor tests of the baroreflex, *i.e.*, the increase in heart rate (decrease in R-R interval) in response to a fall in blood pressure produced by sodium nitroprusside. When the depressor responses are compared, little sparing is seen (figure 3 of reference 1).

In conclusion, isoflurane may depress the arterial baroreflex heart rate responses less than halothane or enflurane in humans, but the studies to establish this are difficult to perform, and the available data are not interpreted easily.

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In reply:—Dr. Roy raises some important points in his letter and I appreciate the opportunity to answer them.

The pressor slopes in the abstract reported by Duke *et al.*[1] indeed indicate a greater depression of baroreflex function than we have observed.[2] It would be erroneous, however, to draw conclusions from their abstract alone. Neither baseline arterial blood pressures nor heart rates are reported and awake control pressor slopes of Group 1 subjects in our study[2] were different from the pressor slopes of Duke *et al.*[1] The data from the larger group of patients we have presented (n = 23) also may be more reliable than preliminary data by Duke *et al.*[1]

In our study, spontaneous ventilation was allowed for a short period of time after intubation in order to demonstrate that the hydrolysis of succinylcholine has terminated its effect prior to collecting data. As stated in the study procedures, all tests were performed 10–15 min following introduction of isoflurane. During this time period, ventilation was kept constant (assisted or controlled) to assure normocarbic conditions the Pa_{CO_2} ranging from 35 to 42 mmHg (determined by arterial blood gas analysis).

The studies of Seagard *et al.*[3,4] have shown that both halothane and isoflurane depress baroreceptor reflex function at multiple sites of the baroreflex arc. The overall depression at 1.0 MAC isoflurane was less, compared with 1.0 MAC halothane. At 2.0 MAC of isoflurane there was further progressive depression of baroreflex slopes while no further depression was seen with halothane. This seems to indicate that 1.0 MAC isoflurane appears to be less depressant than halothane with some of the baroreflex function spared. Dr. Roy points out the difference between the control slopes in the two studies. Not only were the control slopes different, but the anesthetic technique used in the halothane study differed from the isoflurane study in that it employed 50% N_2O in O_2 in addition to halothane and thiopental. Isoflurane, on the other hand, was used with 100% O_2 and no N_2O. Duke and Trosky[5] have observed that substitution of N_2O for a portion of halothane MAC resulted in significantly smaller baroreflex depression when compared with halothane in 100% O_2. It therefore is clear that N_2O itself alters baroreceptor reflexes. Seagard *et al.*[3,4] elected to compare absolute baroreflex slope values in the two studies because a different population of animals was used each time and normalizing such different control slopes may present additional problems (personal communication).

The role of anesthetics in human baroreflex mediated tachycardia in response to the depressor test has been reported in literature for isoflurane only.[1,2] Figure 3 of our study reveals depression of sodium nitroprusside baroreflex slopes in Groups 1, 2, and 3 with isoflurane.