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In Reply:—Dr. Marshall has raised a number of points that require a response. First, Dr. Marshall states that nondependent lung CPAP pressurizes and inflates the lung and, thereby, eliminates one of the fundamental advantages of one-lung ventilation (1LV).[1] However, in most reports, only 5 cm H_2O are required to eliminate hypoxemia. Because lung compliance near residual volume is only 10 ml/cm H_2O, 5 cm H_2O CPAP creates only a 50-ml volume. This is clinically a trivial volume in an open 3-liter hemithorax. Second, Dr. Marshall concludes that "the real interest of the data obtained by Benumof *et al.*[2] lies in what clues they may offer about causes of variability" in Pa_{O_2} during 1LV and draws four conclusions concerning interpatient and intergroup variability. The first conclusion was that the use of halothane during one-lung ventilation is associated with a large probability of arterial hypoxemia (defined as Pa_{O_2} < 100 mmHg). Our data do not support that conclusion. The individual Pa_{O_2} values during halothane anesthesia and 1LV were 236, 116, 102, 92, 82, and 69 mmHg. Defining arterial hypoxemia as Pa_{O_2} < 80 mmHg, only one of our patients was mildly hypoxemic (Pa_{O_2} = 69 mmHg). Ordinarily, 5 cm H_2O CPAP would have easily corrected the situation.

The second conclusion was that halothane inhibited hypoxic pulmonary vasoconstriction (HPV) more variably than isoflurane. This conclusion was based on the Pa_{O_2} data in our paper[2] and that of a few studies that employed different experimental preparations, different species, type of hypoxia (atelectasis *versus* nitrogen ventilation), degree of hypoxia, distribution of anesthesia, etc. On the other hand, there are many studies that demonstrate the same variability for halothane and isoflurane.[3] Finally, our halothane group was only more variable than the isoflurane group with respect to Pa_{O_2} values; it was just the opposite with respect to shunt values.

The third conclusion was that the choice of inhalation anesthetic *versus* intravenous anesthetic is most important in patients who might have the lowest Pa_{O_2} during inhalation anesthesia and one-lung ventilation. Our data do not support that conclusion; the patients who had the lowest Pa_{O_2} during halothane and one-lung ventilation had the smallest increase in Pa_{O_2} when switched to intravenous anesthesia and one-lung ventilation, and the patients who had the highest Pa_{O_2} during halothane anesthesia and one-lung ventilation had the largest increase in Pa_{O_2} when switched to intravenous anesthesia and one-lung ventilation.

Dr. Marshall reaches a tentative fourth conclusion that may be valid, but is speculative in nature. However, this conclusion was reached by dismissing two thoughts, each of which, in turn, may be valid. The first thought was that the patients receiving halothane and isoflurane may not be entirely comparable, and there are data to support this contention. For example, our patients in the halothane group had a Pa_{O_2} = 116 mmHg, while those in the isoflurane group had a Pa_{O_2} = 232 mmHg. I do not know why there are these differences, but subtle factors such as variable double-lumen tube position may make otherwise comparable groups dissimilar. Again, we were interested in having each patient serve as their own control rather than make interpatient and intergroup comparisons. Second, intravenous anesthetics may inhibit HPV, in which case the interpretation of the results is either inhalation and intravenous drugs both do not inhibit HPV, or they both inhibit HPV to the same extent.

Finally, Dr. Marshall comments that our calculations of $\dot{Q}_s\dot{Q}_t$ may be in error because the anesthesia circuit may not have contained 100% oxygen. The patients were receiving high flows of 100% oxygen for prolonged periods of time, no other gas other than either halothane or isoflurane was used, and the FI_{O_2} and FET_{O_2} (mass spectrometer) were typically 1.00 and 0.96–0.97, respectively.

In summary, our primary goal was to have each patient serve as his or her own control, and compare arterial oxygenation during inhalation *versus* intravenous anesthesia with the same set of one-lung ventilation conditions. Within this latter context, we[2,4] and others[5] have not found much of a difference. I agree that the problem of hypoxemia during one-lung ventilation is not unidimensional, and I join in Dr. Marshall's call for continued effort to understand the determinants of arterial oxygenation during anesthesia.

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