dynamic changes and of high monomeric methylmethacrylate concentrations after cemented total condylar knee prosthesis in our small groups of patients does not, however, eliminate the potential risk of complications. A beta (type II) error is possible. Indeed, Svartling *et al.*,[10] studying the blood levels of monomeric methylmethacrylate in nine patients after total condylar knee prosthesis inserted with cement, observed low levels ranging between 0.1–1.44 μg/ml and a high level of 119.8 μg/ml in one patient who presented ventricular extrasystoles after release of the tourniquet.

In conclusion, our prospective study showed that total replacement surgery with total condylar prosthesis is followed only by acute systemic hemodynamic changes related to inflation and release of the tourniquet.

The authors thank Professor Sol Shnider for reviewing the manuscript.

REFERENCES

1. Harris NH: Cardiac arrest and bone cement (letter). Br Med J 3:523, 1970
2. Arden G: Total joint replacement of the knee in rheumatoid arthritis (letter). J Bone Joint Surg 53:150, 1971
3. Byrick RJ, Forbes D, Waddell JP: A monitored cardiovascular collapse during cemented total knee replacement. ANESTHESIOLOGY 65:213–216, 1986
4. Samii K, Elmelik E, Mourtada MB, Debeyre J, Rapin M: Intraoperative hemodynamic changes during total knee replacement. ANESTHESIOLOGY 50:239–242, 1979
5. Samii K, Elmelik E, Goutalier D, Viars P: Hemodynamic effects of prosthesis insertion during knee replacement without tourniquet. ANESTHESIOLOGY 52:271–273, 1980
6. Insall JN, Scott WN, Ranawat CS: The total condylar knee prosthesis. A report of two hundred and twenty cases: J Bone Joint Surg 61A:173–179, 1979
7. Guenier JP, Muller J: Sampling of gaseous pollutants on activated charcoal with 900 mg tubes. Ann Occup Hyg 28:61–75, 1984
8. Tordjmann G, Helmer J, Kipper R, Delagoutte JP, Vivin P: Dosage du méthacrylate de methyle dans les gaz expirés après scellement de prothèse de hanche. An Fr Anesth Réan 5:110–114, 1986
9. Lachiewicz PF, Ranawat CS: Fat embolism syndrome following bilateral total knee replacement with total condylar prosthesis: Report of two cases. Clin Orthop 160:106–108, 1981
10. Svartling N, Pfaffli P, Tarkkanen L: Blood levels and half-life of methylmethacrylate after tourniquet release during knee arthroplasty. Arch Orthop Trauma Surg 105:36–39, 1986

Anesthesiology
67:841–844, 1987

The Use of Ventilation/perfusion Lung Scans to Predict Oxygenation during One-lung Anesthesia

WILLIAM E. HURFORD, M.D.,* ANNE C. KOLKER, M.D.,† H. WILLIAM STRAUSS, M.D.‡

Selective ventilation of one lung provides a quiet surgical field and isolation of the operative bronchus during thoracic surgery.[1] Ten to forty per cent of patients undergoing this technique develop significant intraoperative hypoxemia.[2–4] It is difficult to anticipate, however, which patients will develop hypoxemia during surgery.

Kerr *et al.*[4] reported that patients undergoing one-lung ventilation for non-pulmonary surgery had a lower mean Pa_{O_2} than patients who underwent one-lung ventilation for a pulmonary resection. They postulated that the perfusion to the unventilated operative lung of patients undergoing a pulmonary resection may have been reduced chronically. This increase of pulmonary vascular resistance might protect against the development of a large pulmonary shunt and hypoxemia during one-lung ventilation. Subsequently, others have shown that, when blood flow to the operative lung is limited by intraoperative occlusion of the pulmonary artery, the Pa_{O_2} during one-lung anesthesia improves.[2,6,7] The influence of the preoperative distribution of pulmonary vascular resistance and, therefore, blood flow upon oxygenation during one-lung anesthesia has not been systematically studied.

* Instructor in Anesthesia, Harvard Medical School, and Assistant in Anesthesia, Massachusetts General Hospital.

† Instructor in Anesthesia, Harvard Medical School, and Assistant in Anesthesia, Massachusetts General Hospital. Current address: Memorial Sloan-Kettering Cancer Center, New York, New York.

‡ Professor of Radiology, Harvard Medical School, and Radiologist, Massachusetts General Hospital.

Received from the Anesthesia and Radiology Services of the Massachusetts General Hospital, Boston, Massachusetts; and the Departments of Anesthesiology and Radiology, Harvard Medical School, Boston, Massachusetts. Accepted for publication June 16, 1987. Supported by NHLBI SCOR grant HL23591. Dr. Hurford is a recipient of USPHS National Research Service Award HL07416-07. Presented at the Annual Meeting of the American Society of Anesthesiologists, Las Vegas, Nevada, October, 1986.

Address reprint requests to Dr. Hurford: Department of Anesthesia, Massachusetts General Hospital, Boston, Massachusetts 02114.

Key words: Anesthesia: thoracic; Hypoxia; Measurement techniques: ventilation and perfusion lung scintigraphy; Ventilation: one-lung.

TABLE 1. Preoperative Pulmonary Function Measurements

	Mean ± SEM	Range
Arterial blood gas values while breathing air		
Pa_{O_2}, mmHg	74 ± 2	61–91
Pa_{CO_2}, mmHg	42 ± 1	36–47
*p*Ha	7.42 ± 0.01	7.38–7.47
Spirometry (% predicted)		
FEV_1	67 ± 5	28–116
FVC	72 ± 4	29–117
FEV_1/FVC	92 ± 3	58–122
Lung volumes (% predicted)		
RV	115 ± 8	57–189
TLC	87 ± 3	52–114
RV/TLC	132 ± 8	83–192

The goal of our study was to test the clinical impression that the greater the preoperative blood flow to the operative lung, the more likely it is that intraoperative hypoxemia might occur from persistent pulmonary blood flow during one-lung anesthesia. Accordingly, we estimated the relative perfusion of the operative and non-operative lungs using preoperative ventilation and perfusion lung scans, and retrospectively compared this value to arterial blood gas values during one-lung anesthesia. Our results suggest that unimpaired perfusion of the operative lung (>45% of total pulmonary blood flow) is a major risk factor for developing hypoxemia during one-lung anesthesia.

MATERIALS AND METHODS

We examined the records of 30 consecutive patients who had received quantitative ventilation/perfusion lung scans, and subsequently underwent thoracic operations. Data collected included age, sex, arterial blood gas tensions during room air breathing, the results of preoperative pulmonary function tests by spirometry, and lung volume by plethysmography. All patients had a ventilation and perfusion lung scan performed as a part of their clinical evaluation for surgery and medical management.

Regional ventilation was assessed after 740 MBq (20 mCi) of ^{133}Xe gas was inhaled by each patient while sitting. Initial breath, equilibrium, and elimination images were recorded with a large field-of-view camera fitted with a low-energy all-purpose collimator and positioned behind the patient. The pulse-height analyzer was set at 81 keV with a 20% window. Following the ventilation study, the patient was placed supine and 148–160 MBq (4–4.3 mCi) of ^{99m}Technetium-labeled human albumin microaggregates, averaging 25 μ in diameter, were injected intravenously. Approximately 200,000–700,000 particles were administered. Following injection, the patient was again studied in the same position and perfusion images were obtained using the same instrument with the pulse-height analyzer set at 140 keV with a 20% window. The activity over each lung field in the equilibrium ventilation and perfusion images were expressed as percentage of total lung activity, producing a relative measure of the ventilation and perfusion of each lung.

In all patients, anesthesia was induced with iv thiopental and maintained by inhalation of enflurane or halothane. Laryngoscopy and tracheal intubation were facilitated by the iv administration of succinylcholine. Subsequently, neuromuscular blockade was provided by the iv administration of a nondepolarizing muscle relaxant. The non-operative, dependent bronchus was then intubated with a Robertshaw tube in all patients but one, who had a permanent tracheostomy and underwent endobronchial intubation with a single lumen tube. The patients were operated upon in the lateral jacknife position. The position of the endobronchial tube was confirmed by inspection and auscultation of the chest in the supine and lateral positions.[8] Fiberoptic bronchoscopy was performed to confirm the tube's position in one patient. All patients received controlled ventilation with 100% oxygen at a rate and volume appropriate to maintain the Pa_{CO_2} at approximately 40 mmHg.

Samples of arterial blood were obtained *via* a radial artery catheter at least 10 min after the operative lung was collapsed, confirmed by observing lung atelectasis, and prior to ligation of the pulmonary vessels. Pa_{O_2}, Pa_{CO_2}, and *p*Ha were measured with an automated blood gas analyzer (Corning® 175) calibrated by blood tonometered with gases of known concentration.

The data were subsequently analyzed using single or multiple linear regression, *t* tests, or Chi squares.[9] A *P* value of less than 0.05 was considered significant.

RESULTS

Nineteen men and 11 women were studied. Their ages averaged 60 ± 2 yr (mean ± SEM, range 38–78 yr of age). Twenty-four of the patients had lung cancer, two had empyema, two had recurrent pleural effusion, one had a bronchogenic cyst, and one had a bronchial stenosis. Nineteen had right-sided lesions and 11 had left-sided lesions. Their preoperative pulmonary function measurements are summarized in table 1. Fifteen of the patients underwent lobectomy; seven, segmentectomy or wedge resection; three, pneumonectomy; three, total decortication; and two, exploratory thoracotomy.

The Pa_{O_2} measured during one-lung anesthesia correlated inversely with the relative perfusion of the oper-

ative lung as measured by the perfusion scans (r = −.72; $P < 0.001$; fig. 1). Those patients with relatively little flow to the operative lung had only a small shunt and a high Pa_{O_2}. Patients with normal or excessive blood flow to the operative lung were often hypoxemic, which we defined as a $Pa_{O_2} < 75$ mmHg during one-lung ventilation with 100% oxygen.

With preserved preoperative perfusion, *i.e.*, greater than 45% of the cardiac output going to the operative lung,[10,11] the Pa_{O_2} averaged 115 ± 15 mmHg (mean ± SEM) during one-lung ventilation. When reduced preoperative perfusion, *i.e.*, 45% or less of the cardiac output going to the operative lung, was documented, the Pa_{O_2} averaged 290 ± 43 mmHg ($P < 0.002$).

Eight of the patients had a Pa_{O_2} of less than 75 mmHg during one-lung anesthesia, despite ventilation with 100% oxygen (mean Pa_{O_2} 55 ± 4 mmHg). The likelihood of developing this degree of hypoxemia increased progressively with additional perfusion of the operative lung, as determined by the preoperative perfusion scan (fig. 2). This is not a specific predictor, however, since many patients with preserved preoperative perfusion maintained an adequate level of oxygenation during one-lung anesthesia.

Since preoperative regional ventilation was well matched to perfusion (r = .87, $P < 0.001$), the per cent of total ventilation to the operative lung was also correlated with the Pa_{O_2} during one-lung anesthesia (r = .73, $P < 0.001$).

Neither age, sex, side of operation, preoperative analysis of arterial blood gas tensions while breathing room air, the values recorded during preoperative pulmonary function testing (FEV_1, etc.), nor preoperative lung volume correlated with oxygenation during one-lung anesthesia. Multivariate regressions using these variables did not improve the correlation between preoperative lung perfusion or ventilation and oxygenation during one-lung anesthesia.

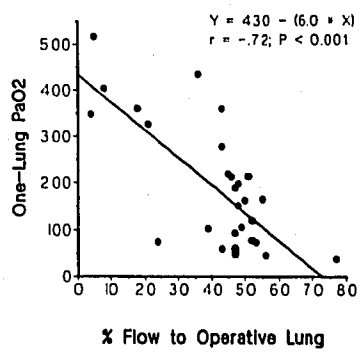

FIG. 1. The relative preoperative perfusion to the operative lung correlated with the Pa_{O_2} after 10 min of one-lung anesthesia. Relative perfusion to the operative lungs was measureed by scintigraphy performed after the intravenous injection of ^{99m}Technetium macroaggregated human albumin.

DISCUSSION

The most important finding of this study is that preoperative ventilation/perfusion scans appear to identify those patients at greatest risk of hypoxemia during one-lung anesthesia. The extent of perfusion or ventilation to the operative lung, measured prior to operation, correlates inversely with the level of oxygenation achieved during one-lung anesthesia. Other preoperative tests, such as analysis of arterial blood gases while breathing room air or routine pulmonary function tests, are not significantly correlated with arterial oxygenation during one-lung anesthesia. These results provide evidence that hypoxemia during one-lung ventilation usually results from persistent blood flow to the operative lung.[2,12,13]

Preoperative lung scans, while demonstrating the effect of tumors upon ventilation and pulmonary blood flow, do not reflect the influence of posture, anesthesia, and surgery upon pulmonary shunt and ventilation/perfusion matching. The correlation of the results of preoperative lung scans to oxygenation during one-lung anesthesia probably would have been increased if the scans had been performed with the patient in the appropriate lateral decubitus position. In this way, the effect of posture upon regional ventilation and perfusion would have been assessed.

Benumof[14] estimated that, in the lateral decubitus position, approximately 40% of the cardiac output perfuses the normal, ventilated nondependent lung. During one-lung anesthesia, the effect of gravity and hypoxic pulmonary vasoconstriction could reduce the expected pulmonary shunt to 20% of the cardiac output.[14] The level of Pa_{O_2} measured during one-lung anesthesia in some of our patients with normal preoperative perfusion to the operative lung suggests a total pulmonary shunt of greater than 50%, although we did not place

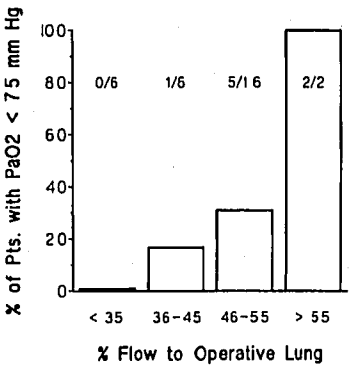

FIG. 2. The proportion of patients with a $Pa_{O_2} < 75$ mmHg during one-lung anesthesia grouped according to the relative preoperative perfusion to the patients' operative lung. The proportion of patients with a $Pa_{O_2} < 75$ mmHg during one-lung anesthesia increased with increasing preoperative perfusion of the operative lung. The ratios within the figure refer to the number of patients with $Pa_{O_2} < 75$ mmHg during one-lung anesthesia, as compared to the number of patients within each of the four groups.

pulmonary artery catheters, and thus could not directly measure the shunt fraction. An increase of pulmonary shunt normally accompanies general anesthesia and thoracic surgery. An increased shunt fraction may be caused by the presence of an open chest, mediastinal compression of the lower lung, elevation of the inferior hemidiaphragm, and interstitial edema in lung regions lying below the level of the heart.[13] An increase of pulmonary vascular resistance in the ventilated lung or a decrease of cardiac output would also increase the pulmonary shunt during one-lung ventilation.[15] In addition, intraoperative hypoxemia may result from improper location of the double lumen tube or the accumulation of secretions.

In summary, we used quantitative ventilation/perfusion lung scans to estimate the relative perfusion and ventilation of each lung in 30 patients who subsequently underwent one-lung anesthesia for thoracic surgery. We found that the degree of preoperative perfusion and ventilation of the operative lung correlated inversely with intraoperative oxygenation during one-lung anesthesia. Unimpaired preoperative perfusion and ventilation of the operative lung appeared to be major risk factors for developing intraoperative hypoxemia during one-lung anesthesia.

References

1. Robertshaw FL: Low resistance double lumen endobronchial tubes. Br J Anaesth 34:576–579, 1962
2. Kerr JH, Crampton Smith A, Prys-Robert C, Meloche R, Foex P: Observations during endobronchial anaesthesia. II: Oxygenation. Br J Anaesth 46:84–92, 1974
3. Weinreich AI, Silvay G, Lumb PD: Continuous ketamine infusion for one-lung anaesthesia. Can Anaesth Soc J 27:485–490, 1980
4. Tremper KK, Konchigeri HN, Cullen BF, Kapur PA, Thangathurai D, Percival C: Transcutaneous monitoring of oxygen tension during one-lung anesthesia. J Thorac Cardiovasc Sug 88:22–25, 1984
5. Johnson B, Benumof JL, Gibbons J: Predicting the degree of shunt during one-lung ventilation (abstract). ANESTHESIOLOGY 63:A565, 1985
6. Alfery DD, Zamost BG, Benumof JL: Unilateral lung lavage: Blood flow manipulation by ipsilateral pulmonary artery balloon inflation in dogs. ANESTHESIOLOGY 55:376–380, 1981
7. Alfery DD, Benumof JL, Trousdale FR: Improving oxygenation during one-lung ventilation in dogs: The effects of positive end-expiratory pressure and blood flow restriction to the nonventilated lung. ANESTHESIOLOGY 55:381–385, 1981
8. Wilson RS: Endobronchial intubation, Thoracic Anesthesia. Edited by Kaplan JA. New York, Churchill Livingstone, 1983, pp 389–402
9. Brown BW Jr, Hollander M: Statistics: A Biomedical Introduction. New York, John Wiley and Sons, 1977
10. Wulff KE, Aulin I: The regional lung function in the lateral decubitus position during anesthesia and operation. Acta Anaesthesiol Scand 16:195–205, 1972
11. Rehder K, Wenthe FM, Sessler AD: Function of each lung during mechanical ventilation with ZEEP and with PEEP in man anesthetized with thiopental-meperidine. ANESTHESIOLOGY 39:597–606, 1973
12. Benumof JL: One-lung ventilation and hypoxic pulmonary vasoconstriction: Implications for anesthetic management. Anesth Analg 64:821–833, 1985
13. Capan LM, Turndorf H, Patel C, Ramanathan S, Acinapura A, Chalon J: Optimization of arterial oxygenation during one-lung anesthesia. Anesth Analg 59:847–851, 1980
14. Benumof JL: Isoflurane anesthesia and arterial oxygenation during one-lung ventilation (editorial). ANESTHESIOLOGY 64:419–422, 1986
15. Cheney FW, Colley PS: The effect of cardiac output on arterial blood oxygenation. ANESTHESIOLOGY 52:496–503, 1980

Anesthesiology
67:844–846, 1987

Exacerbated Spinal Neurologic Deficit during Sedation of a Patient with Cervical Spondylosis

RICHARD A. MILLER, M.D.,* GREGORY CROSBY, M.D.,† PENNATHUR SUNDARAM, M.D.*

New or worsened neurologic deficits associated with "awake" tracheal intubation in patients with instability or narrowing of the cervical spinal canal are generally attributed to injudicious movement of the neck. However, we recently encountered a patient with cervical spondylosis who became substantially weaker after receiving diazepam and droperidol prior to intubation, despite neck stabilization, normal arterial blood pressure, and normal arterial blood gases. Consequently, we speculate that, under some conditions, sedative-hypnotics and tranquilizers can exacerbate or unmask underlying spinal cord dysfunction.

* Instructor in Anaesthesia.

† Assistant Professor of Anaesthesia.

Received from the Anesthesia Services of the Massachusetts General Hospital and the Department of Anaesthesia, Harvard Medical School, Boston, Massachusetts. Accepted for publication June 16, 1987.

Address reprint requests to Dr. Crosby: Department of Anesthesia, Massachusetts General Hospital, Boston, Massachusetts 02114.

Key words: Anesthetics, intravenous: diazepam; droperidol. Complications: neurologic. Spinal cord: weakness.

Report of a Case

A 74-yr-old woman with a long history of osteoarthritis (treated with aspirin), type II diabetes (well-controlled on NPH and CZI insulin),