Myers, Dr. Ameen Ramzy, Dr. Carl Soderstrom, Dr. John Britten, and Dr. Philip Militello for their cooperation in making this study possible.

References

1. Carlon GC, Kahn R, Howland WS, et al. Acute life-threatening ventilation-perfusion inequality: an indication for independent lung ventilation. Crit Care Med 1978; 6:380–383.
2. Powner DJ, Eross B, Grenvik A. Differential lung ventilation with PEEP in the treatment of unilateral pneumonia. Crit Care Med 1977; 5:170–172.
3. Baehrendtz S, Hedenstierna G. Differential ventilation and selective positive end-expiratory pressure: effects on patients with acute bilateral lung disease. Anesthesiology 1984; 61:511–517.
4. Geiger K. Differential lung ventilation. Int Anesthesiol Clin 1983; 21:83–98.
5. Light RB, Mink SN, Wood LDH. Pathophysiology of gas exchange and pulmonary perfusion in pneumococcal lobar pneumonia in dogs. J Appl Physiol 1981; 50:524–530.
6. Siegel JH, Giovannini I, Coleman B. Ventilation: perfusion maldistribution secondary to the hyperdynamic cardiovascular state as the major cause of increased pulmonary shunting in human sepsis. J Trauma 1979; 19:432–460.
7. Ashbaugh DG, Petty TL, Bigfelow DB, et a. Continuous positive pressure breathing (CPPB) in adult respiratory distress syndrome. J Thorac Cardiovasc Surg 1969; 57:31–41.
8. Kumar A, Falke K, Geffin B, et al. Continuous positive pressure ventilation in acute respiratory failure. N Eng J Med 1970; 24: 1430–1436.
9. Otis AB, McKerrow CB, Bartlett RA, et al. Mechanical factors in distribution of pulmonary ventilation. J Appl Physiol 1956; 8: 427–443.
10. Oppenheimer L, Craven KD, Forkert L, Woods LDH. Pathophysiology of pulmonary contusion in dogs. J Appl Physiol 1979; 47: 718–728.
11. Light RB, Mink SN, Wood LDH. The effect of unilateral PEEP on gas exchange and pulmonary perfusion in canine lobar pneumonia. Anesthesiology 1981; 55:251–255.
12. Powers SR, Mannel R, Neclerro M, et al. Physiologic consequences of positive end-expiratory pressure (PEEP) ventilation. Ann Surg 1973; 178:265–272.
13. Baehrendtz S. Differential ventilation and selective positive end-expiratory pressure. Opuscula Medica 1983; Suppl 61.
14. Baehrendtz S, Santesson J, Bindslev L, et al. Differential ventilation in acute bilateral lung disease: influence on gas exchange and central hemodynamics. Acta Anaesthesiol Scand 1983; 27:270–277.
15. Baehrendtz S, Bindslev L, Hedenstierna G, Santesson J. Selective PEEP in acute bilateral lung disease: effect on patients in the lateral lung posture. Acta Anaesthesiol Scand 1983; 27:311–317.
16. Hillman KM, Barber JD. Asynchronous independent lung ventilation (AILV). Crit Care Med 1980; 8:390–395.
17. Hedenstierna G, White FC, Mazzone R, Wagner PD. Redistribution of pulmonary blood flow in the dog with PEEP ventilation. J Appl Physiol 1979; 46:278–287.
18. Siegel JH, Stoklosa J, Geisler FH, et al. Computer-based evaluation of cardiopulmonary function for the optimization of ventilatory therapy in the adult respiratory distress syndrome. International Journal of Clinical Monitoring and Computing 1984; 1:107–126.
19. Berggren SM. The oxygen deficit of arterial blood caused by non-ventilating parts of the lung. Acta Physiol Scand 1942; Suppl II.
20. Siegel JH, Farrel EJ, Miller M, et al. Cardiorespiratory interactions as determinants of survival and the need for respiratory support in human shock states. J Trauma 1973; 13:602–619.
21. Suter PM, Fairley B, Isenberg MD. Optimum end-expiratory airway pressure in patients with acute pulmonary failure. N Eng J Med 1975; 292:284–289.
22. Suter PM, Fairley HB, Isenberg MD. Effect of tidal volume and positive end-expiratory pressure on compliance during mechanical ventilation. Chest 1978; 73:158–162.
23. West JB, Dollery CT, Matmark A. Distribution of blood flow in isolated lung: relation to vascular and alveolar pressures. J Appl Physiol 1964; 19:713–724.
24. Glazier JB, Hughes JMB, Maloney JE, West JB. Measurement of capillary dimensions and blood volume in rapidly frozen lungs. J Appl Physiol 1967; 26:65–76.
25. West JB, Dollery CT, Heard BE. Increased pulmonary vascular resistance in the dependent zone of the isolated dog lung caused by perivascular oedema. Circ Res 1965; 17:191–303.
26. Sachdeva SP. Treatment of post-operative pulmonary atelectases by active inflation of the atelectatic lobe(s) through an endobronchial tube. Acta Anaesthiol Scand 1974; 19:65–70.

Discussion

DR. LEWIS FLINT, JR. (Buffalo, New York): I enjoyed listening to the data, and, because I have been frustrated on several occasions by this problem, I find that I have several questions to ask Dr. Siegel that do not necessarily relate to the feasibility or the technology of his system but to what we might learn about the pathophysiology of the condition from such a system.

First of all, it has seemed to me that clinically identifiable barotrauma to the lung in a patient receiving high levels of PEEP and adjuvant ventilator therapy has been more or less randomly distributed in patients who have identifiable unilateral worse ARDS *versus* bilateral severe ARDS, and I would ask Dr. Siegel if this system, when applied to patients with bilateral disease, does, in fact, lower the frequency of clinically identifiable barotrauma by the ability to identify the worse lung.

The second question I have has to do with the differences between ARDS hypoxemia produced as a result of pulmonary contusion, which I think is the clinical example he showed us, *versus* ARDS produced by systemic sepsis, and whether the measurements of lung mechanics can help us to differentiate any differences in pathophysiology between these two processes.

And the third question has to do with the explanation for the reduced shunt. In the two examples that were presented in the abstract, there was one marked reduction in intrapulmonary shunting and one relatively modest reduction in intrapulmonary shunting. I would ask whether Dr. Siegel has made some measurements with regard to separating pulmonary blood flow and pulmonary vascular resistance to identify the mechanism that might result in reduced shunt.

DR. RICHARD M. PETERS (San Diego, California): I want to congratulate Dr. Siegel for showing us again what we have come to expect from him—the use of nicely designed, complex methods of analysis—in this presentation of sophisticated pulmonary physiology. I guess that the computer processing involves long and complex programs. Certainly the front end instrumentation is both expensive and intricate.

My first question is, how much is your analysis and decision making dependent on the mechanical measurements and how much requires a gas analysis system? The mechanical sensors are cheaper and more generally available.

My second question is about therapy. You use two ventilators for the patients with one stiff and one compliant lung. These must be coordinated and more than double the cost of and skill required to manage the ventilators. How well would you expect the poor man's ventilators to work? Namely, to position the patient with the good lung down to (1) increase its perfusion, and (2) compress it with the weight of the mediastinum and abdomen so that it is protected from hyperexpansion.

This is a very nice study and demonstrates a combination of measurement of coordination of ventilation and perfusion and analysis of

mechanical properties of the lungs of patients with ARDS that has not been done. Such studies are difficult on patients that can cooperate. To carry them out on sick ICU patients is an impressive feat.

DR. LOUIS DEL GUERCIO (Valhalla, New York): I would like to ask Dr. Siegel a question. I have always believed that the best PEEP is that level of PEEP that provides the best level of oxygen transport to the body cell mass. In other words, oxygen transport is the product of the cardiac output and the arterial blood oxygen content, and the best PEEP reflects a favorable balance of the two variables.

Dr. Siegel has shown very nicely how well he has improved the oxygen content and reduced shunt but did not indicate what happened to oxygen transport. It is obvious that he has the data within his computer system to calculate that, and I would like to ask him how closely his improvements correlated with improved oxygen transport and utilization.

DR. JOSEPH M. CIVETTA (Miami, Florida): We were interested in the same problem in the late 1970s, and although we did not have access to the sophisticated ventilators or computerized calculations, we were able to combine a Bird Mark II® time cycling device with inspiratory and expiratory flow cartridges and produce a ventilator that could achieve independent lung ventilation.

We, too, were also intrigued with the efficacy in a few clinical cases and then designed a prospective randomized study. Over the last 4 years we have not found a patient to enter. There have been three reasons that I can identify, and I wonder what the future role will be for this technique.

The first reason was that we have not been able to identify patients in whom there is such significant unilateral lung disease to justify the complicated technique.

The second is, I think, the real crux of the matter. We have finished a prospective study comparing end points of oxygenation and found no difference if inspired oxygen remains at nontoxic levels (less than 50%) while arterial oxygenation is maintained over 65 mmHg). The patients in this study started at values of arterial oxygenation that were in this range while breathing 40 to 50% oxygen. We failed to show a real improvement in outcome based on titration of PEEP so that we no longer consider these patients for this form of treatment.

The third is the advent of high frequency jet ventilation, which is quite satisfactory and avoids the long-term problems of trying to maintain the bronchocath in correct position. I would like to ask Dr. Siegel, although this technique is definitely useful and possible and the information is very interesting, what do you consider to be the future role of this technique?

DR. STANLEY R. FRIESEN (Kansas City, Kansas): Dr. Siegel, I do not know whether or not you said how long you can leave these tubes in the bronchi.

DR. JOHN H. SIEGEL (Closing discussion): Thank you very much, Dr. Friesen and discussants. I would like to try and answer some of the questions.

The first question I will try to answer is Dr. Friesen's. We have tried not to leave the tube in the bronchus longer than about 90 hours. We are not sure whether it can be left longer, but certainly 90 hours seems to be safe, and, frankly, it appears to us that in most instances the value of this technique is in the relatively short period.

We have had to discontinue it on one occasion. Because of the smaller lumen, the aspiration of secretions became a problem. This seems to be one of the limitations of the technique and may limit its use in patients with tremendous degrees of exudative lung disease.

On the other hand, to go back and answer one question again that Dr. Flint raised about ARDS trauma *versus* sepsis, I think that one of the things that has struck us is the fact that many patients who begin with ARDS posttraumatic in one lung may become infected, and then very rapidly the infective process spreads to the other. It is very hard to determine how much of that is in endobronchial spread and how much is hemotogenous, but we feel that endobronchial spread may be quite important. It may well be that the use of this technique earlier in the course of patients' disease process may enable us to isolate and protect the better lung. For example, one of the two surviving patients had aggressive necrotizing pneumonitis in the bad lung due to Pseudomonas. We put the tube in and were able to maintain the patient by selectively ventilating the two lungs until we could, in fact, do an emergency pneumonectomy on that patient; this is one of the two patients who survived.

Obviously, a technique of ventilation can do no more than ventilate the lung, and when you get into late septic processes with destruction of lung tissue, you reach a point where no ventilatory technique is going to be successful. That in the end is the limiting factor for all of these patients. Those who died, with the exception of the two patients whose major cause of death was related to their underlying head injury, died because the septic necrosis in the lungs could not be controlled.

With regard to Dr. Peters' questions about mechanical *versus* gas analysis, at the present time the mechanical component seems to be the one that is the best guide if used in conjunction with the blood gases. It is a very sensitive measure. It tells you whether the patient has an adequate compliance curve and where on the point of the pressure-volume curve an increase in volume of ventilation causes the pressure to rise to a point where barotrauma becomes significant.

In addition, since a large number of patients with posttraumatic injuries have some degree of pneumothorax, it becomes very important to be able to adjust the techniques. That is the value of the servoventilator as opposed to standard controlled ventilation. You can adjust for leak, and therefore, compensate for the leak by using a combination of pressure *versus* volume control. Thus, the level of ventilation may be made optimal by obtaining higher mean pressure in the worse lung relative to the pressure in the other lung. This is one of the values of the technique.

This is one thing that is similar to high frequency ventilation, but, on the other hand, we have many patients whom we have not been able to ventilate with high frequency ventilation alone because it may not exceed critical opening pressure. To exceed the critical opening pressure requires the institution of a pressure level in the ventilator, and that is one of the advantages of being able to use a volume-controlled or pressure-controlled component rather than high frequency alone.

Finally, to the question of making this available, it is clear that this is a complicated technique and that one has to simplify it. We are trying to revise it for a microcomputer system so that all the aspects of using the pressure flow relationships will be readily available to individuals without having to go through a complex, large computer system. We hope that, when this occurs, it will make this general approach to quantitative control of ventilation generally applicable to surgeons and intensivists everywhere.