in the patient's position or amount of pleural suction varied the amount of PEEP applied as well as alveolar ventilation, apparently because of further changes in the air leak and degree of lung collapse.

The expired tidal volume and minute ventilation were not reliable in detecting under- or overventilation. Frequent arterial blood gas determinations were the only means of determining changes in oxygenation and alveolar ventilation. The air leak made vital capacity, inspiratory force, and dead space to tidal volume ratio inaccurate or unobtainable as criteria for weaning. Only the AaDO₂ and clinical observation could be used as guides. Since high levels of pleural suction decreased measured ventilation it was decreased prior to weaning. A high oxygen flow and large reservoir were also needed during spontaneous respiration in order to prevent undue air dilution due to pleural suction.

Under similar circumstances in the future, we would recommend that an attempt be made to achieve full lung expansion. If the patient is being supported with PEEP, it should be removed if possible in an attempt to achieve this goal. Strong pleural suction should probably be avoided because of the disturbances in ventilator control caused by the massive air leak. When faced with progressive widening of the AaDO₂ suggesting the development of the adult respiratory distress syndrome, a trial of PEEP along with multiple pleural tubes and gentle pleural suction can be a life-saving temporizing measure.

REFERENCES


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Editorial Expression

This article represents a report of a very well managed, difficult problem in respiratory therapy. In a patient with a large bronchopleural fistula, the adult respiratory distress syndrome developed. When the need for high inspired oxygen concentrations continued despite the dehydration regimen, the authors felt that positive end-expiratory pressure was necessary to lower inspired oxygen concentrations. The use of positive end-expiratory pressure was noted to decrease the volume of the lung on the side of the fistula and to increase the apparent functional residual capacity of the other lung. It may be postulated that with increasing atelectasis of the pneumonic lung there was an increased resistance to blood flow through that lung and a shift of blood flow to the aerated lung with its lower vascular resistance. The technique was valid in this patient at least temporarily, in that the inspired oxygen concentrations could then be lowered and the changes of the adult respiratory syndrome improved. An important question must be asked in this type of situation as to the effect upon the pneumonic lung itself of increased atelectasis and decreased blood flow. It is probable that decreased aeration and diminished perfusion tend to increase the severity of pneumonitis within the lung by impaired cellular metabolism, and decreased tissue antibiotic levels. This technique then, if used in the critical situation, must be used with the understanding and knowledge that it is probably a two-edge sword; improvement in arterial oxygenation which occurs must be balanced against the probable deleterious effects upon the collapsed lung itself.

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Acute Myocardial Infarction in Idiopathic Hypertrophic Subaortic Stenosis

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A 61-year-old man with idiopathic hypertrophic subaortic stenosis (IHSS) and an acute anteroseptal wall myocardial infarction is described. Hemodynamic study after recovery from the infarction revealed complete loss of the outflow tract obstruction, and a coronary arteriogram demonstrated the left anterior descending artery to be occluded. Acute myocardial infarction, not previously described in IHSS, must now be added to the clinical spectrum of this disorder.

Although angina pectoris, sudden death, and electrocardiographic abnormalities suggestive of coronary heart disease are common clinical manifestations of idiopathic hypertrophic subaortic stenosis (IHSS), acute myocardial infarction is not recognized as part of

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