much lower levels. It will then serve to minimize early inspiratory effort and to raise a low unassisted tidal volume to its expected value, rather than to the range of 10 to 15 ml/kg, not very different than the assist-control mode.

Rather than try to prove the superiority of PS over IMV in the general patient population, future studies should concentrate on selected groups of patients, using PS in a more rational way.

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2 Maclntyre NR: Respiratory function during pressure support ventilation. Chest, 1986, 89:677


To the Editor:

Dr. Perel emphasized that pressure support (PS) has not proved to have an advantage over other methods of assisted or controlled ventilation in the general patient population. He stated that PS can be misused so that achievement of the therapeutic goal is retarded rather than promoted. In our view, whenever PS is implemented in a certain patient, one must define the therapeutic goal. If it is meant for rapid weaning, PS should be administered cautiously, and the supporting pressure should be kept at the lowest possible level compatible with adequate gas exchange and patient comfort.

Applying these principles, we have had positive experiences with PS in our postoperative cardiac surgery patients.

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To the Editor:

The main thrust of my article was not intended to be a comparison of high level pressure support ventilation and SIMV. Rather, the major goal of the article was to describe respiratory drive and the patient’s work as a function of pressure support levels ranging from very high (ie, PSV1) to very low (ie, approaching unsupported breathing). The results clearly showed that, as pressure support is lowered toward 0, respiratory rate increases, tidal volume decreases, the patient’s work increases, and the pressure/volume change (P/ΔV) characteristics of this work increase. These observations provide a rational basis for a weaning protocol that uses decreasing levels of pressure support ventilation alone. This approach allows considerable patient control over the ventilatory pattern (ie, the rate and the inspiratory flow) during the weaning process. Moreover, this approach would appear to offer the potential benefit of supplying a more regular (ie, with every breath) workload with P/ΔV characteristics resembling normal breathing. Improved patient comfort would not be surprising under these circumstances and it can be speculated that a more appropriate muscle-conditioning workload might also result.

I certainly agree that pressure support ventilation, if maintained at too high a level for too long a period of time, can prolong the weaning process. This is true, of course, for any mode of mechanical ventilatory support. Indeed, optimal respiratory care should always be aimed at returning the work of breathing to the patient as quickly as possible. Whether this is done by reducing the number of volume-controlled breaths (as with SIMV) or by reducing the pressure assist of every tidal volume (as with PSV) is probably irrelevant in most patients who have improving lung impedances and respiratory muscle strength. Rather, the advantage to PSV over SIMV is most likely to be limited to those patients in whom weaning has been difficult either because of problems achieving patient comfort/ventilatory synchrony, or because of respiratory muscles which have not been optimally conditioned using unsupported spontaneous breathing approaches. I feel, like Dr. Perel, that further studies are clearly needed to understand the role of lung impedance characteristics, diaphragmatic muscle strength, ventilatory system reflexes and central respiratory drive in determining the optimal mode of weaning patients from mechanical ventilatory support.

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Plural Effusion and Diaphragmatic Dysfunctions

To the Editor:

Dr. Altschule’s commentary on the effects of pleural effusion on the diaphragm (Chest 1986;89:602) makes a good point. Estenne et al1 tested the effects of thoracentesis on pulmonary and inspiratory muscle mechanics. Thoracentesis produced minor and inconsistent effects on pulmonary function, lung compliance and specific airway conductance. In contrast, the maximal negative inspiratory pleural pressure increased significantly after thoracentesis, even when corrected for alterations in lung volume. Improvement in inspiratory muscle pressure is attributable to reduction of chest wall volume with concomitant lengthening of the diaphragm toward normal proportions.

Put another way, pleural effusion compresses the lung but expands the chest wall, especially the lower part. As a result, the hemidiaphragm on the affected side is displaced downward and shortened much as it would be in an acute asthma attack. In asthma, acute shortening of the diaphragm is associated with dyspnea and excessive use of accessory inspiratory muscles.2 Dyspnea in patients with chronic obstructive lung disease is evidently related to ineffective contraction because it is promptly relieved when diaphragmatic contraction is abolished as, for example, by a body respirator.3 Dr. Altschule has reminded us that diaphragmatic dysfunction is an important component of the syndrome caused by large pleural effusion.

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