Exercise testing is used to determine the patient’s cardiovascular reserve, as it measures the ability of the cardiac output to increase in response to stress. In addition, it assesses the autonomic control of the peripheral circulation and the ability to redistribute blood flow to the tissues in need. Thus, the use of exercise testing with gas exchange measurements is indeed a logical approach for evaluating cardiovascular reserve.

There are limitations to the use of exercise tests for determining the cardiovascular reserve to postoperative stress. For example, the bowel wall rather than the muscle may be the organ requiring more blood (hence, oxygen) flow; the former may not have the same tolerance to hypoxia as the latter. Also, postoperative edema would require that capillary PO₂ be maintained at a higher value than in the nonedematous state to overcome the increased distance between red cells and mitochondria for oxygen diffusion. That the AT determined during exercise reflects the ability of the circulation to maintain oxygen delivery for homeostatic regulation of aerobic regeneration of adenosine triphosphate in the perioperative period, as described by Older et al, is certainly an important and practical observation.

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Pressure-controlled Inverse Ratio Ventilation

What Have We Learned?

In this issue of Chest (see page 871), Mercat et al carefully evaluate the cardiorespiratory effects of pressure-controlled ventilation with and without inverse-ratio ventilation. They conclude that physiologically, at least in their patient population, there was no benefit over volume-controlled ventilation in terms of oxygenation, and note an increase in mean airway pressure during inverse-ratio ventilation that induced a decrease in cardiac index and oxygen delivery.

It is of interest, however, that Abraham and Yoshihara⁴,⁵ performed studies similar to that of Mercat et al, but had very different conclusions. They found that pressure control, with or without inverse-ratio ventilation, actually increased \( \Delta \text{O}_2 \), decreased peak inspiratory pressure, and had no effect on hemodynamics.

Since our original report of increased oxygenation and decreased peak airway pressure when using inverse ratio ventilation,⁶ I have read with great interest all of the follow-up studies involving this mode, and have been fortunate enough to review manuscripts and communicate with investigators and clinicians around the world. With due respect to all who have diligently worked on evaluating these modalities, the question remains: why do we still get variable answers to what might otherwise seem fairly straightforward questions?

My observation is the fact that there really are no guidelines for the setup of these modalities. Nowhere is it better described than by East et al.,⁴ who state there is no standard approach to the management of PC-IRV; the elaborate Table 2 in their article supports that conclusion.

In the current article by Mercat et al, it should be noted that the investigators kept the respiratory rate and tidal volume the same in pressure-controlled ventilation as in volume-controlled ventilation. They adjusted the "dialled-in" level of positive end-expiratory pressure (PEEP) to keep the total PEEP the same as it was in volume-controlled ventilation. This probably accounts for their findings. Other investigators⁵,⁶ achieved the "total PEEP" effect by adjusting tidal volume (really, the inspired ventilatory pressure) and respiratory rate, which makes the "dialled-in PEEP" noncontributory since the total PEEP effect exceeds the dialled-in amount, which is not reached during the short expiratory phase. It should also be noted that in the current study by Mercat et al, eight of the ten patients who were considered "hemodynamically stable" were supported with vasoactive drugs, which may have made them more susceptible to changes in mean airway pressure, thereby causing a more obvious drop
in cardiac output.

Rather than being a definitive study to close the door on pressure-controlled inverse-ratio ventilation, this article fuels the fire of controversy regarding its appropriate use. It becomes more evident with each published study that the method by which this technique is applied is probably just as important, if not more so, than the modality itself.

I propose that what may be needed in our pulmonary/critical care literature is a set of international criteria or standards by which ventilator comparison studies should be done. This certainly would make comparisons more meaningful, and would simplify study design so that specific areas of concern could be addressed without “reinventing the wheel” each time. This has the potential to be a valuable project for the American College of Chest Physicians.

So what about the future for inverse-ratio ventilation? There is little doubt that this modality has a place. It is imperative, however, that users understand the controversies and complexities that surround it, so that when it is attempted, appropriate parameters are followed and the risks and benefits are repeatedly assessed.

One of our future goals must be to better understand the “hocus-pocus” that now surrounds how we select and employ ventilator modalities as well as to match them to specific patient groups so as to affect the most successful outcomes. We have only scratched the surface.

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Excessive Daytime Sleepiness, Upper Airway Resistance, and Nocturnal Arousals

Excessive daytime sleepiness (EDS) is increasingly being recognized as an important cause of motor vehicle and industrial accidents, with important socioeconomic consequences. While there are multiple causes of EDS, insufficient nocturnal sleep, obstructive sleep apnea, periodic leg movements during sleep, and narcolepsy are the major diagnostic considerations. Approximately 5 to 10 percent of patients evaluated for EDS will have no specific etiology and will, therefore, be classified as having idiopathic hypersomnia.1

In this issue of Chest (see page 781), Guilleminault and colleagues report a study in which they carefully evaluated 48 subjects (20 men and 28 women) with a diagnosis of idiopathic hypersomnia for subtle evidence of sleep-disordered breathing. They found that 15 (31 percent) of these subjects demonstrated frequent arousals from sleep in relation to increased upper airway resistance. This was proved by directly recording esophageal pressure (Pes) with a balloon catheter and airflow with a pneumotachograph. Electroencephalographic evidence of arousal, manifested by the transient appearance of alpha activity in the EEG, was documented to follow sequential breaths characterized by increasing respiratory effort (negative Pes), airflow limitation, and decreased tidal volume.

Since episodes of increasing respiratory effort with decreased airflow and oxyhemoglobin desaturation are widely recognized and classified as obstructive apneas, the reader may legitimately ask, what is new? The answer is that these 15 subjects did not show evidence of oxyhemoglobin desaturation and that 5 (33 percent) of the subjects either did not snore or had only intermittent light snoring. Because a 2 to 4 percent fall in oxyhemoglobin saturation is generally required to score a disordered breathing event as an episode of hypopnea, the mean respiratory disturbance index for their group was normal at 2.1 ± 1.7 per hour. In contrast, the arousal index was moderately elevated at 31.3 ± 12.4 per hour. The fact that arousals can occur as the result of increased upper airway resistance without significant oxyhemoglobin desaturation has been previously observed and reported in both snorers and patients with apnea.2,3 This phenomenon may result in false-negative overnight oximetry studies when employed to screen patients for sleep disordered breathing.4 Although the determinants of oxyhemoglobin desaturation are complex, oxygen stores within the lung play the dominant role.5,6 Consequently, thin subjects with normal lung function are less likely to demonstrate oxyhemoglobin desaturation with disor-