acutely using present pharmacologic agents. Since the stable atrial cycle in atrial flutter length does not result in concealed conduction in the AV node, control of ventricular rate via induction of a stable, higher degree of AV block is possible only rarely. Short-term administration of an intravenous antiarrhythmic agent such as procainamide will convert less than 25 percent of patients to a stable sinus rhythm. Paradoxically, such a trial may slow the atrial cycle length, permit 1:1 AV conduction, and may actually exacerbate the situation.

For these reasons, electrical cardioversion has long been the principal mode of therapy for patients with persistent atrial flutter. Transsthoracic cardioversion is effective in >90 percent of patients with atrial flutter, but the requirement for general anesthesia to reduce discomfort makes it relatively contraindicated in some patients. Rapid atrial pacing using either epicardial or endocardial electrodes is also highly effective, but except in some postsurgical patients, cannot be employed immediately at the bedside. In this issue (see page 110), Falk and Werner describe the use of the esophageal pill electrode for rapid atrial pacing in patients with atrial flutter. Although the method described resulted in conversion to a stable sinus rhythm in only six of 14 patients, six others were converted to atrial fibrillation or type 2 atrial flutter with a well-controlled ventricular rate. In only two patients was the technique a total failure.

There are several factors that recommend consideration of this approach in the patient who presents with atrial flutter. First, a recording of the atrial electrogram permits confirmation of the diagnosis. Second, the technique is potentially applicable in almost any patient able to swallow without concern for recent food ingestion, pulmonary status or venous access. Finally, the procedure itself is well tolerated with minimal risk or discomfort. Although, as stressed by Falk and Werner, special equipment is necessary to permit appropriately filtered recordings to be made and to allow painless atrial capture during pacing, this technique is easy to learn and employ. It offers a rapid bedside alternative to alternate forms of therapy and physicians likely to encounter patients with atrial flutter should acquaint themselves with it.

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Mechanical Cardiac Support by Synchronous, Cardiac Cyclespecific High-frequency Jet Ventilation
More Than a Matter of Timing?

Since the studies of Cournand et al1 and others2,3 documenting that inspiratory increases in intrathoracic pressure (ITP) during intermittent positive-pressure ventilation (IPPV) may adversely affect cardiac output and systemic hemodynamics, a central goal during mechanical ventilatory support of critically ill patients has been to provide adequate gas exchange without impairing oxygen transport. The general concept remains valid that sustained increases in ITP decrease the pressure gradient for systemic venous return and, consequently, ventricular preload. Recent studies have confirmed earlier observations4 that equivalent positive swings in ITP can reduce the instantaneous transmural left ventricular (LV) wall stress, or afterload to LV systolic ejection.5 This effect is disproportionate in a severely impaired, volume-overloaded LV and creates a relatively preload-independent hemodynamic state.6 By this mechanism, sustained increases in ITP associated with positive end-expiratory pressure (PEEP) or continuous positive airway pressure (CPAP) have been exploited to improve cardiac performance.7-9 If prolonged, however, such beneficial heart-lung interactions may have unanticipated and potentially harmful consequences in extrapulmonary organ systems in the critically ill. Increases in the back pressure to cerebral, renal, and hepatic outflow can jeopardize vital organ function even as oxygen delivery is improved.10-12

Pinsky and colleagues13 reported their clinical experience with an alternative method of using increases in ITP to influence ventricular loading conditions, to augment cardiac output. In that study, single positive-pressure respiratory cycles were placed within single cardiac cycles using synchronous, cardiac cycle-specific high-frequency jet ventilation (HFJV). The investigators found that phasic inspiratory increases in ITP significantly improved cardiac performance in a group of patients with severe LV dysfunction awaiting cardiac transplantation. Independent of mean ITP and heart rate, synchronous increases in ITP improved cardiac output approximately 30 percent more than
either asynchronous HFJV or conventional IPPV. What a remarkable tool this may be! Perhaps the hemodynamic benefit of intra-aortic balloon counterpulsation can be achieved with minimal intravascular invasion while gas exchange is supported.

Before routine application of this technique can be justified, several important questions must be answered. Patients must be carefully selected on the basis of specific changes in pathophysiology. First, if small changes in intrapleural pressure are to influence LV systolic ejection, both sufficient compromise of LV function and a relatively preload-independent state must be present. Second, reductions in lung compliance in patients with acute lung injury may limit the extent to which positive airway pressure is transmitted to the pleural space, thereby modifying the hemodynamic responses. In patients with abnormally reactive airways, from asthma or chronic obstructive lung disease, air-trapping may result in inadvertent PEEP. Such effects appear to be minimal at ventilatory frequencies less than 150/min (the current FDA limit) in patients without increased airway resistance. Sustained increases in ITP associated with air-trapping might impair systemic venous return and negate any hemodynamic advantage gained. As with intra-aortic balloon pumping, synchronization of ITP pulses with each heart beat may not be practicable in patients with significant cardiac arrhythmias.

Widespread clinical use of this form of high-frequency ventilation must also await improvements in the safety features of ventilators, such as reliable alarms for power failure or patient disconnection, together with pressure monitors and pressure-responsive cutoff systems, and functional circuitry for synchronization. In the past, providing adequate humidification has been a general problem with HFJV delivery systems. It is not certain that new advances in humidification have resolved this issue. In addition to monitoring the performance of the ventilator, we must clarify which variables in the patient should be monitored during the application of HFJV.

Finally, we need more information on the long-term effects of this type of ventilatory support on nonventilatory aspects of pulmonary function, such as surfactant physiology, lung healing, and clearing of secretions.

It will be very interesting to see if the beneficial cardiopulmonary interaction that Pinsky and associates have clearly shown in their patient population will work equally well for longer periods in similar patients with severe heart failure. Whether this technique might be applicable in treating other types of patients with impaired cardiopulmonary function to ultimately improve oxygen supply/demand in multiple extra-pulmonary organ systems is an exciting prospect, but currently unclear. Since mechanical cardiac support by synchronous cardiac cycle-specific increases in ITP is therefore more than simply a matter of timing, the clinical challenge will be to define more rigorously its indications/contraindications, and effects on systemic homeostasis in critically ill patients.

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