Patient-Ventilator Interaction

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Patient-ventilator interaction has been the focus of increasing attention from both manufacturers and researchers during the last 25 years. There is now compelling evidence that passive (controlled) mechanical ventilation leads to respiratory muscle dysfunction and atrophy, prolonging the need for ventilatory support and predisposing to a number of adverse patient outcomes. Although there is consensus that the respiratory muscles should retain some activity during acute respiratory failure, patient-ventilator asynchrony is now recognized as a cause of ineffective ventilation, impaired gas exchange, lung overdistention, increased work of breathing, and patient discomfort. Far more common than previously recognized, it also predisposes to respiratory muscle dysfunction and other complications, leads to excessive use of sedation, increases the duration of ventilatory support, and interferes with weaning. Appropriate recognition and management of patient-ventilator asynchrony require bedside assessment of ventilator graphics as well as direct patient observation. Among currently available ventilation modes and approaches, none has been shown to be clearly superior to all the others with respect to patient-ventilator interaction, and strongly held prefer-

ences among investigators have led to controversy and difficulties in carrying out appropriate studies evaluating them. As a result, marked practice variation exists among different specialties as well as in different institutions and geographical areas. The respected authorities on mechanical ventilation who participated in this conference differed in the modes they preferred but agreed that proper understanding and use according to the individual patient's needs are more important than which mode is chosen. Conference participants discussed the determinants, manifestations, and epidemiology of patient-ventilator asynchrony, and described and compared several ventilation modes aimed specifically at preventing and ameliorating it. The papers arising from these discussions represent the most thorough examination of this important aspect of respiratory care yet published. *Key words: mechanical ventilation; asynchrony; asynchrony; complications; acute respiratory failure; respiratory muscles; weaning; triggering; ventilation modes; noninvasive ventilation*. [Respir Care 2011;56(2):214–228. © 2011 Daedalus Enterprises]

Introduction

The purposes of mechanical ventilation in acute illness are to facilitate gas exchange, maintain lung inflation, and support the work of breathing when patients are not able to provide these functions adequately for themselves. At least a dozen previous RESPIRATORY CARE Journal conferences have dealt with ventilators and the management of patients who require ventilatory support, but none has focused specifically on patient-ventilator interaction (PVI), an area increasingly recognized and appreciated in recent years for its impact on the management and outcomes of critical illness. This 46th Journal conference brought together internationally recognized experts in mechanical ventilation from throughout North America, who met over a two-and-one-half day period to present and discuss current knowledge pertaining to 13 different aspects of PVI. This paper summarizes what I took to be the most important messages of the individual presentations and the discussions that followed them, and offers some of my own observations on this central component of the management of patients with acute respiratory failure. With a few exceptions I will not attempt to cite the most important primary work that has been done in this field; the individual papers in these 2 special issues[1-14] provide a comprehensive and authoritative review of the literature pertaining to PVI.

Table 1. Some Terms Related to Patient-Ventilator Interaction, and Their Synonyms

Term	Synonyms
Asynchrony	Dyssynchrony
Double triggering	Breath stacking
Cycle	Expiratory trigger
	Expiratory threshold
	Breath termination
	Inspiratory cycle-off
Rise time	Pressurization rate
"Cycle criteria"	Termination threshold

One challenging aspect of this topic is the use by different authors of multiple terms meaning the same thing, or nearly the same thing, as exemplified by "asynchrony" and "dyssynchrony" (Table 1). In keeping with the convention adopted at the conference, in this article I will use patient-ventilator asynchrony (PVA) to refer to "any condition where PVI is not optimal".[2]

Why Is Patient-Ventilator Interaction Important?

How a patient interacts with the ventilator, and with the process of ventilatory support, is determined by many factors (Fig. 1). These include the patient's underlying respiratory function and the superimposed effects of the acute illness, the effects of therapeutic interventions unrelated to ventilatory support, the ventilator's functional characteristics, and how it is operated by the clinician. Whether the connection between the patient and the ventilator is an endotracheal tube, a tracheostomy tube, or a mask for noninvasive ventilation (NIV), this interface plays an important role in PVI. In addi-

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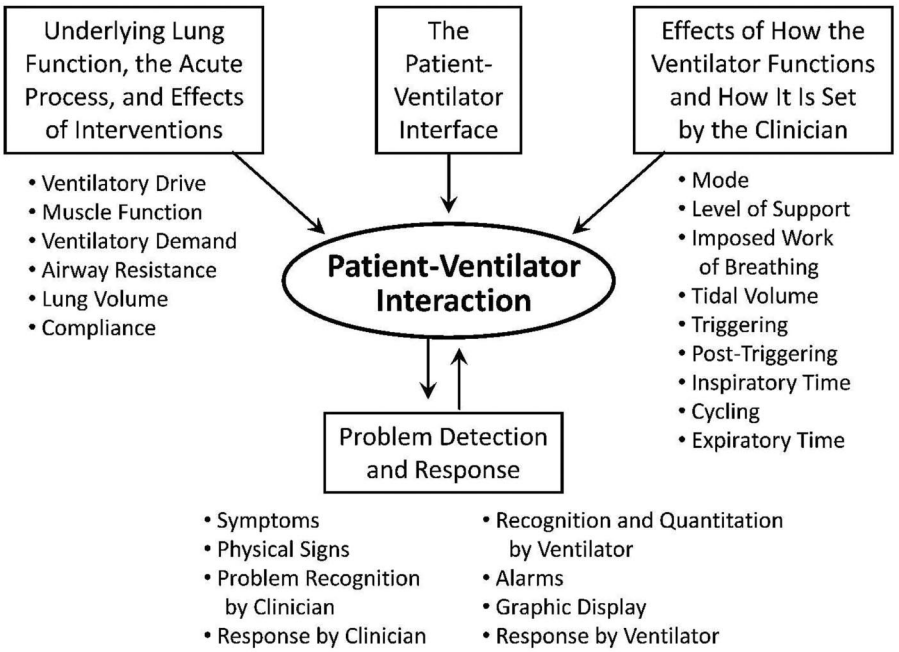

Fig. 1. Schematic representation of factors that influence patient-ventilator interaction. Each of these factors was considered during the conference.

tion, whether the patient's interaction with the ventilator is helpful or harmful is affected by how both the ventilator and the clinician respond to its manifestations. Each of the factors shown in Figure 1 was addressed during the conference.

When the delivery of gas by the ventilator does not correspond in quantity, timing, or pattern to what the patient wants, PVA is the result. Its manifestations include those of excessive work of breathing on the part of the patient, and when the mismatch between patient demand and ventilator delivery is severe, the result is respiratory distress and "fighting the ventilator," as graphically depicted in Figure 2.[15] The physical signs shown in the figure are familiar to clinicians but are often much less obvious than this—an important reason why ventilator graphics[16,17] are indispensable in detecting, monitoring, and managing PVI. Along with the bedside manifestations of excessive respiratory muscle effort is accumulating evidence that excessive stress on these muscles is both functionally and anatomically damaging to them.[12]

An understandable response to the distress patients may experience due to PVA is to try to "take them out of the equation" by means of heavy sedation—sometimes with the addition of pharmacologic paralysis—and rendering them completely passive during ventilatory support. However, passive mechanical ventilation is also now known to be detrimental to the respiratory muscles, producing detectable diaphragmatic dysfunction after as little as 48 hours of inactivity, and frank muscle atrophy when prolonged (Fig. 3).[18] A main focus of the conference was identifying and maintaining a middle ground between the adverse manifestations of PVI depicted in Figures 2 and 3. Table 2 lists a number of the adverse consequences of PVA.

Another common discussion thread was the importance of the equation of motion as it applies to mechanical ventilation and PVI. This relationship dictates that the total pressure required for ventilation (P_{total}) is equal to the product of lung volume (V) and the elastance of the respiratory system (E), plus the product of flow ($\dot{V}$) and airways resistance (R):

$$P_{total} = (V)(E) + (\dot{V})(R)$$

When patients actively participate in ventilatory support, P_{total} has 2 components: the pressure generated by the patient's respiratory muscles, and that generated by the ventilator. For patient-ventilator synchrony to be maintained, the sum of these components has to balance the resistive and elastic loads. How the ventilator's output can be mated to the patient's efforts in meeting the demand for pressure and volume according to the equation of motion as those efforts and demand change is the challenge faced by the ventilatory modes and approaches addressed during the conference.

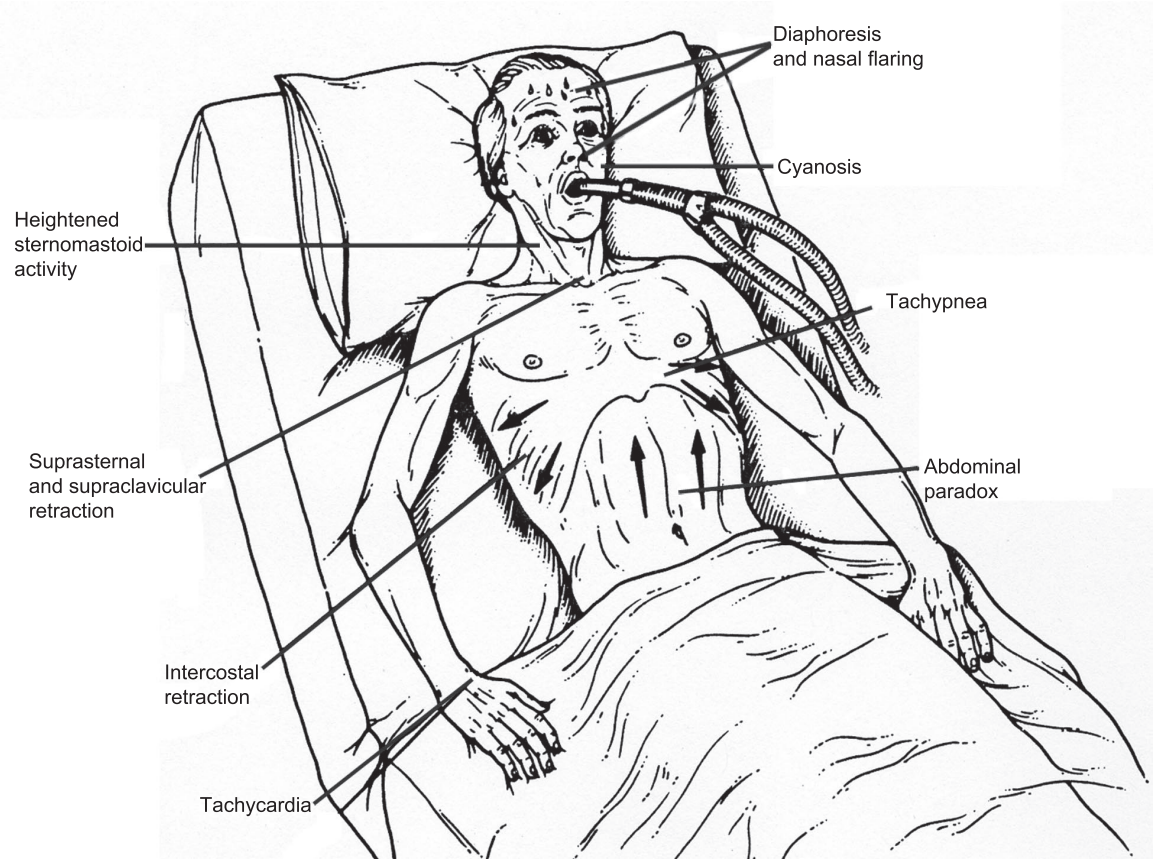

Fig. 2. Artist's depiction of a mechanically ventilated patient experiencing respiratory distress, indicating the characteristic physical signs associated with severe patient-ventilator asynchrony. Clinicians participating in ventilator management are all-too-familiar with this picture, stylized and exaggerated as it is in this cartoon. However, less dramatic patient-ventilator asynchrony may have less obvious signs and be difficult to detect by simple bedside observation. (From Reference 15, with permission.)

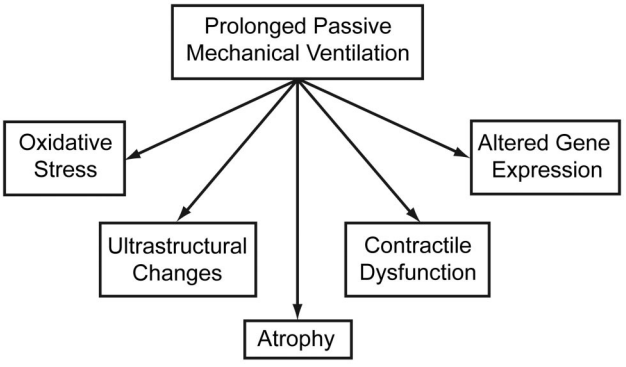

Fig. 3. Effects of prolonged passive mechanical ventilation, with no activation of the respiratory muscles, on the diaphragm. (Adapted from Reference 18.)

Table 2. Adverse Consequences of Patient-Ventilator Asynchrony

Ineffective ventilation
Hypoxemia
Lung over-distension
Dynamic hyperinflation
Increased work of breathing
Patient discomfort
"Fighting the ventilator"
Distress for family members and others at the bedside
Conflict among team members
Excessive administration of sedatives and neuromuscular blocking agents
Respiratory muscle dysfunction
Confusion with respect to readiness for weaning
Prolongation of mechanical ventilation
Neuromuscular complications of prolonged immobility

The following summaries of what I took to be the main messages of the individual presentations are not in the order in which they were presented (which was affected by travel and other logistical matters), but instead are considered in the order that makes the most sense to me in developing an overall grasp of the subject.

What We Have Learned About Respiratory Muscle Function and Critical Illness

The effects of critical illness on the muscles of respiration, and how mechanical ventilation and other interven-

tions in critical care affect these muscles, came up repeatedly during the conference and are key elements in any discussion of PVI. As part of his presentation on airway pressure release ventilation (APRV), Rich Kallet reviewed this topic for us in depth.[12] Although his presentation fell on the second day of the conference, respiratory muscle function is sufficiently important to all the topics under consideration that I will address it here.

Many studies in patients with COPD and neuromuscular disease have shown that mechanical loads in excess of what the respiratory muscles can sustain continuously will lead to electromechanical abnormalities, physical signs such as paradoxical chest and abdominal motion, and, ultimately, overt ventilatory failure. Too much load, applied over a sufficient amount of time, actually damages the respiratory muscles, as demonstrated in humans and animals by various means, including the release of pro-inflammatory cytokines into the circulation. On the other hand, putting the respiratory muscles totally to rest induces weakness and atrophy. Strong evidence now supports the concept that ventilator-induced diaphragmatic dysfunction occurs in patients with acute respiratory failure whose respiratory muscles are completely inactive during controlled mechanical ventilation.

Respiratory muscle function in critically ill patients is further deranged by such factors as severe sepsis and the administration of drugs such as neuromuscular blocking agents (NMBAs) and corticosteroids. Although the situation is far from completely settled, it is apparent that the respiratory muscles of patients with acute respiratory failure can be adversely affected by both too much and too little activity. Thus, based on current evidence, passive mechanical ventilation should be avoided, at least for periods longer than a day or two, and the respiratory muscles should be active throughout the period of ventilatory support. How active, and how best to facilitate and monitor this activity, are, however, unclear.

The History of Mechanical Ventilation and Evolution of Ventilator Technology as They Relate to Patient-Ventilator Interaction

No one has a better perspective on the historical development of mechanical ventilation from both clinical and technical viewpoints than Rich Branson. He led off the conference by showing how clinical innovations in ventilator management over the last 40 years affected PVI, and vice versa—that is, how a growing awareness of PVI prompted technical refinements and the introduction of new approaches to ventilatory support.[1] In the early days of mechanical ventilation as a form of life support, patients were passive participants in the process. They were ventilated via mask or endotracheal tube, either manually or by a positive-pressure ventilator, during anesthesia and surgery. Patients with respiratory muscle paralysis caused by polio were passively supported, first by negative-pressure ventilation (the iron lung) and later by positive-pressure ventilation via tracheostomy. In the earliest applications of mechanical ventilation in critical care, patients were either heavily sedated to ablate spontaneous breathing efforts or rendered apneic by hyperventilation.

Active participation in ventilatory support on the part of the patient became a factor in the mid-1970s, with increased use of assist control ventilation (also called continuous mandatory ventilation, with the patient able to trigger breaths from the ventilator), and especially with the introduction of intermittent mandatory ventilation (IMV, partial ventilatory support, in which the patient is required to generate a substantial portion of total ventilation, particularly during weaning). Bedside observation of the respiratory distress this produced in many patients led to studies and technical innovations aimed at mitigating excessive patient effort and rendering ventilatory support more comfortable. Investigators realized that inspiratory work by the patient during patient-triggered breaths did not cease once inspiratory flow was initiated, and that modifications of inspiratory flow and triggering sensitivity could reduce this work. As IMV became popular, mechanical ventilation became increasingly complicated for clinicians as manufacturers introduced different strategies and devices for making breaths easier for patients to trigger and terminate. In the 1980s it was shown that adding inspiratory pressure support to the spontaneous breathing circuit for IMV improved PVI, and this practice subsequently became widespread.

Improved understanding of the importance and complexity of PVI during mechanical ventilation stimulated the incorporation of microprocessors, closed-loop control, and numerous other technical advances into critical care ventilators. During the last 25 years, with different manufacturers' approaches to the triggering of inspiration, flow delivery and modulation during inspiration, the transition from inspiration to expiration, and the expiratory phase—all driven at least in part by a desire to improve PVI—the bedside application of mechanical ventilation has increasingly become a function of the brand of ventilator used. Each manufacturer's version of the different ventilation modes and features typically has its own unique proprietary name, rendering their similarities less apparent and making ventilator management more challenging for the clinician with respect to PVI.

How Often Does Patient-Ventilator Asynchrony Occur and What Are Its Consequences?

Scott Epstein next reviewed what is known about the frequency with which PVA occurs and the evidence for its adverse effects.[2] Determining the former is fraught with

difficulty, for numerous reasons. The different types of PVA—ineffective triggering efforts, delayed triggering, auto-triggering, double-triggering, premature and delayed cycling, and flow asynchrony—vary a lot with respect to how easy they are to detect and also in the degree to which they have been addressed in the literature. How often PVA occurs depends on when the patient is observed (especially with respect to sedation administration), how long the period of observation lasts, and what technique is used for detection. Because PVA is more common in some patient populations (such as those with severe COPD and those with acute lung injury or the acute respiratory distress syndrome [ARDS] who are managed with low-tidal-volume ventilation), it is important to specify the type of patient in discussing its prevalence. The frequency with which PVA occurs also varies with ventilator mode and other aspects of the ventilatory approach.

In addition, because some degree of asynchrony occurs at least transiently in virtually every ventilated patient whose drive is intact and who is capable of muscular effort, how "significant" PVA is defined becomes important in discussing its prevalence. Most investigators have used a threshold of 10% or more of all breaths being untriggered or otherwise asynchronous for "clinically important" PVA. Even considering the factors listed above, the reported prevalence varies considerably. However, it seems clear that synchrony consistently occurs in only a minority of ventilated patients.

Although many studies have shown that PVA is associated with adverse patient outcomes, direct evidence establishing causation is currently lacking. Nevertheless, it seems clear from Epstein's review of the available studies that PVA is bad for patients, and that the more it occurs, the worse it is, by numerous measures. In addition to causing respiratory distress (and thus prompting excessive sedation, which has its own set of adverse consequences), PVA makes achieving effective ventilatory support more difficult in several ways. It worsens dynamic hyperinflation in patients with obstructive lung disease, leading to hemodynamic complications and interference with weaning. Breath-stacking frustrates attempts to achieve lung-protective ventilation in acute lung injury and ARDS. It may cause unintended hyperventilation or respiratory acidosis and worsen hypoxemia. NIV may provide suboptimal support or fail altogether, requiring intubation and its associated complications. And, importantly, PVA prolongs the duration of mechanical ventilation by confusing clinicians with respect to readiness for ventilator liberation. Although the available evidence is incomplete, there is little argument that PVA makes patients uncomfortable, leads to unwarranted sedation and prolongation of hospitalization, and may even increase mortality. Understanding, detecting, and minimizing it are thus topics of great physiologic, clinical, and economic importance.

How Does Patient-Ventilator Asynchrony Develop and What Influences It?

Control of Breathing and Patient-Ventilator Interaction

The patient's drive to breathe is a primary determinant of dyspnea and also a major factor in whether PVI is helpful or detrimental. In his review of the components of the ventilatory control system, and through the use of several creative conceptual diagrams, Sai Parthasarathy helped to make more understandable the highly complex subject of how the control of breathing affects a patient's experience of, and reactions to, ventilatory support.[3]

The core of the respiratory control apparatus is a sensory-motor system made up of central controller, multiple sensors giving it input, and the respiratory muscles as effectors. This controller system responds variably to a wide array of stimulating and depressing inputs, including P_{O_2}, P_{CO_2}, pH, sensations of stiffness and irritation from the lungs and chest wall, and the effects of drugs—and the responses to these inputs are different depending on the patient's state of wakefulness or sleep. In the mechanically ventilated patient, the control system must contend with 3 separate effector pumps—the patient's inspiratory and expiratory muscles and the ventilator. This highly complex system is made further complicated by the effects of time, and especially of phase lags between the patient's controller-generated signal for something to happen (the start of inspiration, for example) and the ventilator's response to that signal.

The nature and function of the ventilatory control system explain a lot when it comes to the problems faced by both engineers and clinicians in preventing and managing PVA. Given the importance of maintaining some respiratory muscle activity, the control system needs to be active, so that dysfunction and atrophy do not develop, but not so active as to cause overt patient distress.

In his presentation Parthasarathy pointed out that technology in the field of cardiology is way ahead of what is currently available in respiratory care with respect to the detection and automatic response to events or patient changes. As someone who works in both critical care and sleep medicine, he noted that the latter is currently well ahead of the former in this respect, citing shape-signal ventilator triggering as an example as well as more complete automation in devices used in assessing and treating sleep apnea.

Triggering and Patient-Ventilator Interaction

Catherine Sassoon discussed the ways in which patients may interact with the ventilator in relation to triggering.[4] Both flow asynchrony (in which patient flow demand is

not matched by the ventilator) and timing asynchrony (a mismatch between neural and ventilator inspiratory times) occur in relation to triggering. Patient-ventilator asynchrony may occur with each of the different types of timing or phase asynchrony: ineffective triggering, auto-triggering, double-triggering, premature cycling, and delayed cycling. Ineffective triggering, what happens when patient effort fails to initiate inspiratory flow or excessive effort is required, is probably the most important of these.

During the triggering phase—what happens from the onset of patient effort to the onset of inspiratory flow—PVI is impacted by both sensitivity (how much change in patient trigger signal is necessary to start inspiratory flow) and the ventilator's triggering time delay (how long it takes to start delivering flow once the change is detected). The post-triggering phase, comprised of flow delivery by the ventilator and the cycling-off variable (what causes inspiratory flow to stop), is also important in PVI, but variably in different disease processes. Aspects of the post-triggering phase that impact PVI include the overall level of ventilatory assistance, inspiratory flow rate, the cycling-off variable (in pressure support), and the level of applied PEEP.

Sassoon stressed the importance of the balance among the patient's triggering efforts, how the clinician adjusts the ventilator settings, and the fundamental characteristics of the ventilator in determining whether PVA occurs in relation to triggering and post-triggering. Excessive triggering effort is uncomfortable for the patient, yet setting the triggering threshold too low will predispose to auto-triggering. Flow asynchrony occurs when there is a mismatch between what the patient wants (that is, the intensity of the ventilatory drive and muscular effort), and what the ventilator delivers (that is, airway pressure and flow) during inspiration. Too little initial flow or a too-slow inspiratory pressurization rate (long inspiratory rise time) will accentuate air hunger and be uncomfortable for the patient, while on the other hand, if these are excessive, this will also cause discomfort. How the cycling threshold is adjusted—especially in patients with COPD—is an important determinant of total inspiratory time and hence of the tendency for air-trapping and the generation of intrinsic PEEP.

Concluding, Sassoon presented a practical algorithm (Fig. 10 in her paper)[4] for diagnosing and treating PVA related to ventilator triggering. This scheme incorporates all the elements of her presentation, permitting the clinician at the bedside to identify wasted patient efforts and to minimize or eliminate these through an orderly sequence of simple steps. In keeping with the recommendations of others at the conference, she considered an "asynchrony index" of 10% or more of patient breaths being untriggered or otherwise asynchronous to be undesirable and a threshold for intervening to try to reduce it.

Cycling and Patient-Ventilator Interaction

Cycling from inspiration to expiration (expiratory triggering), the least-studied phase of a mechanical breath with respect to PVI, was next discussed by Mike Gentile.[5] He illustrated the importance of correctly setting the "cycle criteria" in pressure support (the percentage of the peak value to which inspiratory flow must fall before inspiration is terminated) in assuring patient comfort and preventing inspiration from being either too short or too long. As was pointed out, "one size does not fit all" with respect to the cycling threshold, the common setting of 25% of peak flow being satisfactory for many patients but not for others.

Cycle asynchrony, in which the patient attempts to exhale before the end of inspiration, can occur when this threshold is set at too low a percentage, as well as when inspiratory flow is insufficient (in volume control ventilation) or inspiratory time is too long (in pressure control ventilation). An inappropriate cycling threshold can be especially important as a cause of PVA in ventilating patients with obstructive lung disease, through its facilitation of dynamic hyperinflation and worsening auto-PEEP. The escape of air through a bronchopleural fistula or a leak in the ventilator circuit can result in failure of inspiratory flow to terminate if the cycling threshold is set too low.

Detecting and Monitoring Patient-Ventilator Asynchrony at the Bedside

In her discussion of the bedside assessment and monitoring of PVI,[6] Marjolein de Wit drew primarily from the large patient series she and her colleagues have collected in their medical intensive care unit (ICU). It may be the largest and most extensively documented observational study on PVI anywhere: 80 patients (without critical hypoxemia, on no more than 8 cm H_2O of PEEP, and capable of making inspiratory efforts) who were observed and their bedside ventilator graphics recorded for more than 50 hours and 60,000 individual breaths.

Patients were observed at the bedside, and PVA was defined and categorized as follows, based on the bedside displays of flow, pressure, and volume versus time, according to the classification system of Thille et al[19]: ineffective triggering (a decrease in airway pressure, with simultaneous increase in air flow without triggering inspiration); auto-triggering (breath delivered by the ventilator because of a fluctuation in airway pressure or flow not caused by patient effort); double-triggering (2 delivered breaths separated by an expiratory time less than half the mean inspiratory time); premature cycle (any breath in which inspiratory time was less than half the mean inspiratory time); and delayed cycle (a breath in which inspiratory time was more than twice mean inspiratory time).

de Wit et al found that ineffective triggering was by far the most common form of PVA, comprising 88% of events. A finding in the study was that multiple types of asynchrony may be present simultaneously.

An important point brought out in this session was the finding that the observed frequency of PVA is a function of how long and how often during the day one looks for it. During her presentation and in the subsequent discussion, de Wit pointed out that although they were initially unfamiliar with the process, medical residents and other clinicians in the ICU were able to learn the approach she described and to use it in decreasing PVA in their patients. The key was to observe the patient and the bedside graphics simultaneously, asking whether the patient appears uncomfortable and looking specifically at such things as facial expression, "mouth breathing," the use of accessory ventilatory muscles, and signs of active expiratory effort. de Wit stressed the importance in her unit of having the residents look at the patient and at the waveforms rather than at an order sheet or the ventilator controls in deciding how to modify current settings when PVA was detected.

Ventilator management in de Wit's patients was primarily synchronized IMV (SIMV) with added pressure support. There was considerable discussion—and striking lack of agreement—among the conference participants with respect to the preferred or "best" mode for managing most patients with acute respiratory failure. However, everyone agreed that, although its manifestations might vary somewhat in the different modes, PVA was extremely common, under-recognized, and under-treated.

Ventilator-Focused Strategies for Optimizing Patient-Ventilator Interaction

Next the conference turned to discussion of PVI in the context of specific ventilation modes and approaches, much of it focused on new modes and approaches—not new, actually, in most cases, since proportional assist ventilation (PAV) and APRV have been around for decades, but still "new" in terms of widespread clinical use. Although much attention was paid at this conference, as elsewhere, to "innovations" in mechanical ventilation—new modes and approaches based mainly on engineering innovations and new commercial products—when discussing their own approaches each of the participants relied on familiar, conventional modes of mechanical ventilation, even though these varied. Sassoon,[4] de Wit,[6] and MacIntyre[7] developed their bedside approaches for patients being managed with conventional volume- or pressure-targeted ventilation, which is good for the purposes of the conference proceedings, given that the advances represented by the new modes and approaches remain somewhat hypothetical in terms of everyday patient care.

Improving Patient-Ventilator Interaction During Ventilation With Conventional Modes

Neil MacIntyre reviewed the aspects of conventional mechanical ventilation that need to be addressed in optimizing PVI, and emphasized the need to use the ventilator's pressure and flow graphics in order to do this.[7] He described the basic types of ventilator-delivered breaths in conventional modes, and showed how modifications in breath triggers, flow delivery, breath cycling, and the level of support could be applied in each case in order to improve PVI.

MacIntyre made the observation that a volume-targeted breath is more difficult to synchronize with the patient than a pressure-targeted breath, as supported by some but not all studies. He explained his concept of "sculpting the breath," as depicted at the bedside by the flow-, pressure-, and/or volume-time waveform, manipulating the magnitude and shape of inspiratory flow, inspiratory time and end-inspiratory pause time, delivered volume, and the proportion of machine-triggered and patient-triggered breaths, in order to find the combination of these best adapted to the individual patient. He echoed the observation of several others during the conference that there is no "one size fits all" with respect to PVI, and that different disease processes (for example, ARDS vs COPD), as well as the reactions of individual patients, require adjusting different variables and to different degrees. Particularly in the presence of dynamic hyperinflation, he pointed out, the necessity for the patient to generate changes in pressure or flow at the airway opening in order to trigger the ventilator can be problematic, and moving the triggering sensor into the lower airway (such as at the distal tip of the endotracheal tube) might make it easier to improve PVI in this context.

With respect to "unconventional" conventional modes such as pressure-regulated volume control, volume support, and adaptive support ventilation, available as proprietary features on different ventilators, MacIntyre observed that these appear to work less well in practice than they should in theory, and many patients do not seem to tolerate them very well. He also offered the cogent observation that a patient with a strong respiratory drive is more difficult to synchronize with the ventilator, such that "synchrony begets synchrony, and asynchrony begets asynchrony." This insight emphasizes the importance of adjusting the ventilator so that it comes as close as possible to providing what the patient wants, and it also acknowledges the frequent need to blunt that drive pharmacologically while not ablating it altogether.

As noted by others, the *level* of support provided by the ventilator is clearly important, with more generally being better for patient comfort but with the caveat that excessive support can lead to Cheyne-Stokes respiration, apnea, and other forms of periodic breathing. However, the *pat-*

tern of support is also a key determinant of optimal PVI. When IMV was first popularized, it was asserted that patients would easily and naturally adapt to a mixture of mandatory and spontaneous breaths. This seems to be wrong, with the weight of evidence indicating that patient-ventilator synchrony is easier to maintain with similar support of every breath than with multiple breath types with different loading patterns. Just as patients on continuous mandatory ventilation continue to generate muscular efforts throughout inspiration, heavier loading of occasional spontaneous breaths during IMV tends to increase the drive to breathe and to induce PVA.

Proportional Assist Ventilation

Bob Kacmarek next described PAV and NAVA, comparing and contrasting these approaches to improved PVI via partial ventilatory support based on the ventilator's direct responses to the patient's needs—as gauged by changes in work performed by the respiratory muscles and diaphragmatic electrical activity, respectively.[9] Almost 20 years ago, Magdy Younes introduced PAV, a novel approach to ventilatory support that aimed to reduce a patient's work of spontaneous breathing, according to the equation of motion, depending on how much effort the patient exerted.[20] As a clinician and non-engineer, I have always liked the analogy between PAV and power steering in my car. Driving down the interstate at the speed limit, it takes very little work to steer the car, and the power steering provides only minimal assistance to the driver, while during parallel parking, which takes a lot of effort, the power steering contributes a much greater amount of the total work required. With PAV, the clinician determines the fraction of overall work the ventilator is to contribute; according to that set proportion, if the patient's needs are minimal and accompanied by only modest effort, the ventilator provides little assistance, whereas if the work of breathing is high and the patient is struggling harder, the ventilator chips in with much greater assistance.

The concept as I have just explained it seems simple enough, but the complexities involved in making PAV work accounted in part for the long time that passed between its initial description and its availability to clinicians as a feature on a commercially available ventilator. To determine the necessary pressure to generate in order to unload the patient's respiratory muscles by the specified proportion (say, 60%), according to the equation of motion, total respiratory system resistance and elastance as well as tidal volume and inspiratory flow must be measured. This requires a brief end-inspiratory pause, which the ventilator can perform either prior to initiating PAV or intermittently as it is applied. From these determinations the ventilator can calculate the total airway pressure required, and can add in the specified proportion of that total pressure that is not being generated by the patient. The more effort the patient exerts, the larger the deficit between measured and target airway pressure, and the more work the ventilator adds to the system. As ventilatory mechanics and demand improve during the course of illness, the ventilator automatically adjusts its pressure output to match the patient's changing needs.

A substantial literature has accumulated about PAV.[9] The mode has been used successfully in both intubated and nonintubated patients, in those both clinically stable and acutely ill, during exercise (as in pulmonary rehabilitation), and also during sleep. In studies comparing PAV to PSV in stable patients with acute respiratory failure, the incidence of PVA has been lower with the former. Because PAV is purely a patient-triggered mode, it requires that the patient have an intact respiratory drive and be capable of inspiratory muscle effort. Further, because it relies on measured deflections in airway pressure, there must be no leaks in the system, and there must be nothing to interfere with the patient's inspiratory efforts being translated into changes in airway pressure, as occurs with auto-PEEP. This mode would be less appropriate than more conventional modes for use in severely hypoxemic patients and those who are hemodynamically unstable.

Neurally Adjusted Ventilatory Assist

Like PAV, NAVA is designed to provide partial ventilatory support tailored to the patient's need.[9] Here again the clinician does not set tidal volume, flow, pressure, or timing. However, instead of generating airway pressure in proportion to patient-generated volume and flow signals, NAVA generates airway pressure in proportion to a signal based on the electrical activity of the diaphragm. Inspiration is triggered by changes in the electrical signal from the diaphragm (E_{di}). Inspiration is triggered by a clinician-preset E_{di} amplitude change, and cycled when the E_{di} decreases to a default percentage of peak E_{di} amplitude. This process requires placement of a special nasogastric tube that incorporates a series of electromyographic electrodes situated above and below the diaphragm. The clinician sets the amount of airway pressure to be applied for each millivolt of diaphragmatic electrical activity. The larger the signal, presumably indicating greater demand for ventilation, the more pressure the ventilator provides. Inspiration terminates when the electromyographic signal diminishes to a set proportion of its peak value.

Reported experience with NAVA is much more limited than with PAV, although it has been shown capable of supporting critically ill adult patients. Like PAV, when adjusted according to its intended use, NAVA tends to result in patients having smaller tidal volumes and faster respiratory rates than those generally employed by clini-

cians. So far, no clinical trials have systematically compared NAVA to conventional ventilation with respect to outcome.

Based on his theoretical analysis and the available literature, Kacmarek provided the following observations about these modes. Both PAV and NAVA are available for application in both invasive and NIV, although PAV is not currently approved for NIV in the United States. Because it uses airway pressure, flow, and volume, PAV requires no additional, special equipment. However, PAV does require an intact respiratory drive and the ability to generate an inspiratory effort. It is not recommended for use in children weighing less than 20 kg, and is not effective in the presence of either auto-PEEP or substantial air leaks. Although it requires a special nasogastric catheter, NAVA has the potential advantages of use in patients of all sizes, and effective ventilation in the presence of both endogenous PEEP and air leaks in the system. Like PAV, NAVA relies on the patient's respiratory drive, in this case as manifested by efferent electrical signals to the diaphragm. Neither mode is well suited for use in acutely unstable patients, in those with severe hypoxemia, or in critically ill patients requiring prolonged ventilatory support.

As Kacmarek pointed out, PAV and NAVA are designed to accomplish the same goals, just driven by different inputs from the patient. Both are intended to improve patient-ventilator synchrony by allowing patients to establish a ventilatory pattern consistent with their ventilatory demands. He noted that, because of their closed-loop function and thus their ability to respond to changes in the patient's condition independently of the clinician, both modes have a potential advantage over conventional ventilation in institutions whose clinicians are too busy to tend to PVI at the bedside of every ventilated patient. Whether clinical trials will bear this out, and whether PAV and/or NAVA will prove sufficiently advantageous over conventional modes for widespread adoption by clinicians, remain to be seen.

Airway Pressure Release Ventilation

APRV, first introduced in the 1980s, is a form of pressure-control IMV employing very large I:E ratios (eg, up to 10:1) and unrestricted spontaneous breathing during mandatory inflations.[13] This mode maximizes mean airway pressure while simultaneously encouraging the purported physiologic advantages of spontaneous ventilation. It may be thought of as supporting the patient on 2 different levels of continuous positive airway pressure (CPAP). The higher CPAP level is intended to maintain lung inflation (but without higher peak airway pressure spikes during inspiration), and abrupt release to the lower CPAP level permits tidal exhalation for CO_2 removal; the difference between the 2 CPAP levels and the frequency of pressure-release determine overall alveolar ventilation and arterial P_{CO_2}. If more than half of the total respiratory cycle is spent at the higher CPAP level, the mode is referred to as APRV, whereas if most of the time is spent at the lower CPAP level, it is called "BIPAP" (not to be confused with the more common use of that acronym in NIV) or biphasic positive airway pressure. This method of mechanical ventilation does not require active breathing efforts by the patient (as with PAV and NAVA), or synchronization with the ventilator when these are present. If no spontaneous breathing efforts are made, APRV and BIPAP are functionally the same as pressure control ventilation.

In discussing APRV, Rich Kallet pointed out that enthusiasm for the use of this mode reflects decades-long, strongly held advocacy by some proponents of spontaneous breathing throughout the course of acute respiratory failure. The proposed clinical advantages of APRV over conventional ventilation include reduced patient work of breathing, better synchrony with the ventilator, diminished requirements for the use of sedatives and NMBAs, and decreased duration of mechanical ventilation. That these advantages are borne out by evidence is, however, far from clear. Although APRV is intended to eliminate PVA, Kallet showed us several patient examples illustrating its occurrence. He reviewed the published studies comparing APRV to conventional ventilation, pointed out that such comparisons have been difficult to carry out in a balanced, unbiased fashion, and indicated that the available outcome studies have been particularly affected by design problems. Whether patient work of breathing and PVA are consistently decreased by APRV is by no means clear. By analogy to IMV, this may be due in part to the simultaneous imposition of 2 different breathing patterns—spontaneous patient breaths and time-cycled pressure releases—with attendant stimulation of ventilatory drive and air hunger.

Another problem raised in the discussion of APRV was the potential for larger tidal excursions with this mode than those recommended for lung-protective ventilation in managing patients with acute lung injury and ARDS. Kallet pointed out that if the average minute ventilation requirement for a normal arterial P_{CO_2} in such patients is 13–15 L/min, and the cycling frequency between the 2 CPAP levels as recommended is 10–15 cycles/min, the effective tidal volume will substantially exceed 6–8 mL/kg predicted body weight.

Despite their theoretical advantages, all 3 of the unconventional modes addressed at the conference (PAV, NAVA, and APRV) appear at present to be less well suited to the management of the most critically ill patients than conventional pressure- or volume-targeted modes. The appropriate role for each of them in everyday management of ventilated patients remains to be established, but for the

present it is difficult to conclude that any of them is clinically superior to conventional mechanical ventilation.

Conceptualizing and Assessing Ventilator Modes With Respect to Patient-Ventilator Interaction

Forty years ago, when I first began caring for patients with acute respiratory failure, we had ventilators that provided a set inflation pressure (although the pressure and flow were pretty limited) and other, newer, somewhat more powerful machines that delivered a pre-set tidal volume, and we could either set a rate or allow the patient to trigger the breaths. That was it. Today most critical care ventilator manufacturers provide about a dozen different modes, and Dräger offers 26 of them. In addition to the modes already discussed in this summary, the clinician can now choose from AutoMode, mandatory rate ventilation, pressure-controlled synchronized mandatory ventilation, proportional pressure support, and SmartCare—among many others. As pointed out by Rob Chatburn in his presentation on the classification of ventilator targeting schemes,[8] one current respiratory equipment book lists 56 unique mode names, as compared to 3 in a comparable book from 1973. As a clinician and educator, although I used to understand generally how ventilators worked, faced with this overwhelming complexity I no longer do—and this is a big problem when it comes to understanding and optimizing PVI.

After discussing the concept and taxonomy of closed-loop control of mechanical ventilation, which most current critical care ventilators employ, Chatburn attempted to bring some logic and clarity to the welter of brand names and descriptive terms applied to current ventilator modes. He classified them according to 6 basic targeting schemes applied in today's ventilators, and further with respect to the 3 general areas of safety, patient comfort, and the facilitation of weaning (liberation) from ventilatory support. For variables related to comfort, perhaps most directly applicable to PVI, in his presentation he also considered 6 potential capabilities: adaptation to the patient's spontaneous breathing pattern; neural control of trigger and cycle; prevention of dynamic hyperinflation; capability of flow synchrony; coordination of mandatory and spontaneous breaths; and scaling of delivered work to work demand.

It is clear that closed-loop control of mechanical ventilators has had a major impact on their design and function, and also that we will be seeing even more sophisticated and complex ventilation schemes and modes in the near future. Whether clinicians will be able to comprehend and effectively use these innovations, and whether their implementation will improve PVI and safely permit better matching of ventilatory support to patient needs, remain to be seen.

Patient-Focused Strategies for Optimizing Patient-Ventilator Interaction: Sedation and Paralysis

Along with the realization that it is better for patients to be active participants in ventilatory support than to be totally passive recipients has come the understanding that more sedation is not better for patients, but worse, in numerous ways. Bill Hurford reviewed the evidence supporting this understanding and provided insight into how best to approach sedation in mechanically ventilated patients.[11] He also discussed the use of NMBAs in relation to their potential indications and adverse effects.

A large and expanding evidence base shows that patients who receive more sedation spend more time on the ventilator, and also suffer a range of complications, from neuromuscular weakness to post-traumatic stress disorder. The adverse effects of benzodiazepines and opioids are increased when these drugs are administered via continuous infusion, and can be reduced if given on an intermittent, as-needed basis. In addition, the use of standardized assessment scales and unit-based protocols for sedation and analgesia reduces the amount of drugs administered, with associated benefits in stay and the incidence of complications.

A key aspect of Hurford's presentation was the observation that pain, anxiety, and delirium are distinct processes, with different causes, assessments, and treatments, all of which, however, are manifested by agitation. To the extent that these 3 separate problems can be recognized and managed individually, patient care can be made more rational and effective, and important outcomes improved. Sedatives and analgesics should be given for specific indications, rather than as a kind of generic chemical restraint for patient agitation; patients should be kept calm and comfortable but should also be easily arousable.

Pain is as prevalent in critically ill medical patients as in those in surgical or trauma ICUs, and studies show that it is commonly under-treated. Opioids, not sedatives, are most effective for pain, and successful management may call for a multimodality approach, as, for example, with the inclusion of epidural analgesia. In contrast, anxiety is best treated with benzodiazepines such as midazolam or lorazepam, yet these agents are frequently overused and adverse effects are common. Several studies have shown that administration of less sedation is associated with shorter duration of mechanical ventilation and hence fewer complications such as ventilator-associated pneumonia. Sedatives should thus be weaned as aggressively as ventilatory support.

Propofol, an anesthetic agent with sedative but not analgesic effects, is widely used in ventilated patients; it has the advantages of rapid onset and offset but requires continuous infusion and is associated with an increasingly recognized propofol infusion syndrome. The more recently introduced dexmedetomidine may have advantages over

benzodiazepines in maintaining sedation targets and shortening the duration of mechanical ventilation, although studies on this are limited at present.

Benzodiazepine use predisposes to delirium, which is exceedingly common among critically ill patients and has highly unfavorable prognostic implications. Delirium increases ICU and hospital stay, predisposes to multiple complications both acutely and long-term, and is associated with increased overall mortality. While agents such as haloperidol are helpful, the primary treatment for delirium is not pharmacologic but rather consists of interventions such as increased orientation and interaction with caregivers and family members, distraction and diversion, avoidance of interruptions, early mobilization, and efforts to preserve a normal sleep-wake cycle.

The administration of NMBAs during mechanical ventilation is difficult to control appropriately and interferes with patient assessment. Use of these agents is associated with longer duration of mechanical ventilation, longer ICU stay, and higher mortality. While NMBAs may improve arterial oxygenation in patients with critical hypoxemia unresponsive to other measures, the need for this use occurs infrequently. To facilitate PVI the use of NMBAs should be the exception, and then for as short a period as possible—less than 24 hours in all but the most exceptional circumstances. However, as with sedation, this is an area of great practice variation in American ICUs, one in need of greater examination and standardization.

Noninvasive Ventilation and Patient-Ventilator Interaction

Discussing what is known about PVI during NIV for acute respiratory failure, Dean Hess concluded that PVA is both as prevalent and as important in NIV as it is in invasive mechanical ventilation.[10] He reviewed the studies that have examined this problem, and showed examples of ineffective triggering, auto-triggering, double-triggering, late cycling, and premature cycling in patients on NIV. The frequency of PVA is correlated with patient comfort during NIV, although at present it is not known whether this factor affects NIV's overall success or failure.

The biggest factor in PVI during NIV appears to be air leak. That is, the larger the leak, the more asynchrony, particularly with higher levels of pressure support. Hess emphasized the importance of understanding the function of the ventilator used for delivering NIV. While some ventilators have excellent leak compensation, such may not be the case with others, predominately ICU ventilators, with which triggering and cycling may be markedly affected by varying leaks. It was pointed out that the triggering and cycling functions of the system should be checked when switching to a different mask with different leak characteristics. The point was also made that pressure-control mode, with a fixed mandatory rate, may be more effective than pressure-support modes in assuring appropriate cycling in the presence of large or variable leaks.

Other factors that may be more important with respect to PVI during NIV than with invasive mechanical ventilation are effects of the patient's sleep-wake state on such things as ventilatory drive and airway resistance, which may lead to over-ventilation and periodic breathing as well as under-ventilation. New modes such as average volume-assured pressure support and adaptive support ventilation, which are now being applied in managing sleep-disordered breathing, may have advantages in NIV during acute respiratory failure; this was cited as an example of greater recent technological progress in the realm of sleep medicine than in critical care.

Patient-Ventilator Interaction in Prolonged Mechanical Ventilation

Unexpectedly and at short notice, Alex White was unable to attend the conference. Two unfortunate results of this were that aspects of PVI pertaining to prolonged mechanical ventilation received less attention in all the discussions than they should have, and that the material reviewed by White and his colleagues in their paper[14] could not be subjected to real-time discussion by the attendees.

Prolonged mechanical ventilation is most often defined as the need for invasive ventilatory support for more than 21 days, although an alternative, pragmatic definition is ventilatory support after placement of a tracheostomy, typically after several failed weaning attempts. Most of what we know about PVI comes from the acute-care setting, and few primary data have been generated in long-term acute-care hospitals (LTACHs) during the care of patients requiring prolonged mechanical ventilation. However, PVI is as important in the LTACH setting as it is in the ICU, in terms of its potential to affect patient comfort, work of breathing, and other determinants of successful weaning and other outcomes. Improving PVI shortens weaning time, permits liberation from ventilatory support in some individuals who would otherwise remain ventilator-dependent, reduces complications, improves sleep quality, and reduces costs in the LTACH setting.[14]

A large proportion of patients receiving prolonged mechanical ventilation in LTACHs have COPD. Because the use of pressure support is widespread in the LTACH setting, recognition and control of triggering asynchrony and other problems associated with this mode in patients with COPD and dynamic hyperinflation are especially important in that context. White and his colleagues point out that delirium is extremely common among patients transferred

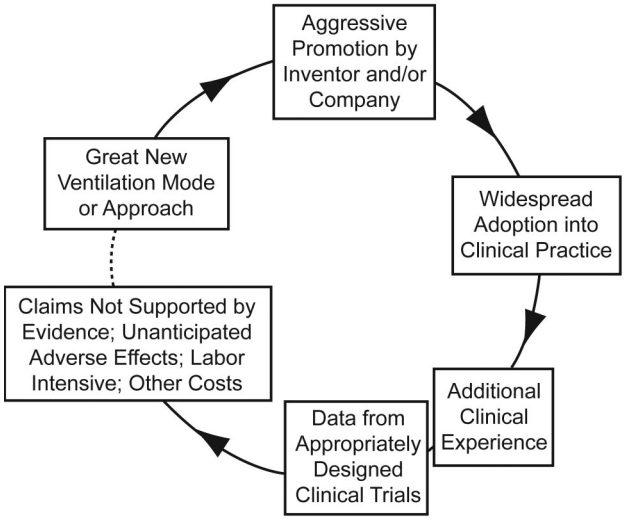

Fig. 4. The cycle of entrepreneurial innovation in mechanical ventilation. New modes and approaches tend to be promoted vigorously either by their inventors or by manufacturers before being evaluated in appropriately designed clinical studies. As a result of this advocacy they become popularly known and are often widely adopted into clinical practice. Only years later, after extensive practical experience in real-world application and/or the availability of the results of rigorous clinical trials, is a more objective appraisal made, typically resulting in much reduced enthusiasm for the new approach. In the last 35 years this "vicious circle" has played out numerous times with ventilator modes and strategies, and in only a minority of instances has the innovation proved truly beneficial for patients.

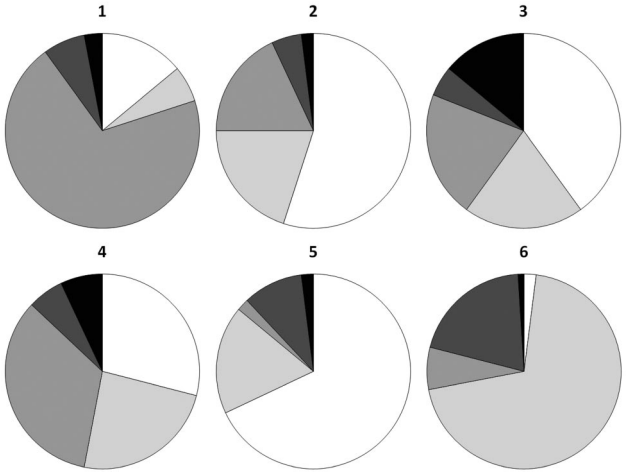

Fig. 5. Magnitude of practice variation in the management of invasive mechanical ventilation, as illustrated by the relative proportions of the different modes used in 6 different geographic regions, as of 2002. The regions shown are: 1 Australasia; 2 Central and South America; 3 Central and Western Europe; 4 Eastern Europe; 5 North America; and 6 Northern Europe. The proportion of cases managed with volume control and/or assist control ventilation (shown in white), with pressure control ventilation (in light gray), and with synchronized intermittent mandatory ventilation, with or without added pressure support (medium gray), each ranged from a very small percentage to more than two thirds in the different areas of the world. Pressure-support ventilation alone (dark gray) was used in from about 5% to roughly 20% of cases, with other modes (black) representing a small fraction. (Data from Reference 23.)

to LTACHs from acute-care hospitals, and that its effective management is a key prerequisite for successful outcomes.[14] They also discuss the potential effects of tracheostomy on PVI, both favorable in the form of possible improvements in work of breathing and patient comfort, and unfavorable when tubes are too small, malpositioned, or obstructed.

Some Concluding Observations

Much of the discussion during the conference focused on efforts by clinicians and ventilator manufacturers to detect the patterns and quantities of ventilation patients want and to provide them as much as possible. Something that received relatively little attention is the fact that what patients may want in terms of ventilation and what we know about the adverse effects of ventilatory support may conflict. The most obvious example is lung-protective ventilation for patients with ARDS. Compelling evidence supports the use of small tidal volumes in order to prevent ventilator-induced lung injury, yet "respiratory distress" is part of the name of the syndrome, and patients may experience greater comfort with larger tidal volumes. Just as the better oxygenation associated with larger tidal volumes and higher PEEP levels has not correlated with better outcomes in ARDS,[21,22] resisting the temptation to deliver larger tidal volumes in order to improve PVI may be counterintuitive but necessary for optimum patient outcomes. As in the case of patients with severe COPD, in whom increasing ventilation in response to dyspnea may result in dangerous levels of auto-PEEP, this typically requires the judicious use of sedation. These examples also illustrate the notion that "one size does not fit all" in terms of ventilator modes and other settings when it comes to optimizing PVI.

The history of PVI in relation to the technology of mechanical ventilation illustrates a recurring theme in critical care and in respiratory therapy (Fig. 4). The availability of definitive, clinically relevant data on the proposed benefits of new ventilation modes and approaches tends to lag many years behind their initial introduction into clinical practice. One result of this lag, with multiple available approaches and strongly held opinions among investigators and clinicians alike, is striking practice variation in the day-to-day management of ventilated patients. Depending on where one trained, whether one manages primarily medical or surgical patients, the culture of institutional or regional practice, and where in the world one lives, the

Table 3. Patient-Ventilator Interaction: Main Take-Home Messages From the Conference

PVA is extremely common, is frequently unrecognized, and needs to be looked for in all ventilated patients.
Using direct observation of the patient and simultaneous assessment of ventilator graphics at the bedside, clinicians can improve PVI in everyday management.
Patients' needs and responses to the ventilator may change rapidly, so that assessment of PVI needs to be an ongoing activity throughout the period of ventilatory support.
Ventilator graphics are no longer optional as tools for use in everyday patient care.
Ineffective triggering is the most common form of PVA, and is especially affected by dynamic hyperinflation and auto-PEEP, emphasizing the importance of how the triggering function is handled by the ventilator and set by the clinician.
Given the improvements in ventilator technology, how the clinician uses the technology has become a more important determinant of PVI.
The complexity of today's ventilators and the multiplicity of proprietary mode names are barriers to clinician understanding, communication, and optimum patient care, emphasizing the need for interdisciplinary communication and cooperation on an ongoing, continuous basis.
The lack of equipoise resulting from strongly held positions on the part of researchers and clinicians with respect to modes and approaches to mechanical ventilation has been detrimental to progress in the field as well as to patient care.
Acknowledging the dramatic practice variation among different clinicians, institutions, and geographic regions, it is more important that the mode used be understood and applied correctly than that any particular mode or approach be chosen.
Different disease processes in patients with acute respiratory failure (such as COPD and ARDS) present different challenges to PVI that must be considered in the approach to mechanical ventilation.
Although they may not be a large part of the process initially leading to acute respiratory failure, the respiratory muscles and how they are affected by the ventilator are primary determinants of PVI, complications, and clinical outcomes.
Passive mechanical ventilation, with no respiratory muscle activity, should be avoided except for brief periods; ventilatory support needs to involve some work on the part of the patient, but not too much.
Achieving and maintaining synchrony is generally a greater challenge with volume-targeted than with pressure-targeted ventilation.
A consistent breath pattern is generally more acceptable for patients with respect to PVI than a mixture of breath types.
With noninvasive ventilation, better patient synchrony is associated with more successful application, and this is primarily a function of how the ventilator detects and compensates for leaks.
Pain, anxiety, and delirium are distinct processes manifested by patient agitation, each playing a different role in PVI and each having a different optimal management approach.
Using less sedation and neuromuscular blocking agents during mechanical ventilation improves patient outcomes, emphasizing the importance of non-pharmacologic measures for optimizing PVI.

PVA = patient-ventilator asynchrony
PVI = patient-ventilator interaction
COPD = chronic obstructive pulmonary disease
ARDS = acute respiratory distress syndrome

modes and approaches used for mechanical ventilation differ enormously (Fig. 5).[23,24]

Practice variation generally correlates inversely with adherence to best evidence and clinical practice guidelines.[25] However, in the field of mechanical ventilation, even the most evidence-driven, respected authorities have divergent opinions and personal practices with respect to mode. This was evident around the table at this conference. Several times I attempted to poll the participants about their personal approaches to points being debated. Most of these attempts failed (perhaps because I was unable to frame the questions in an unambiguous manner), but I succeeded in obtaining shows of hands on a few issues. Everyone agreed that bedside graphics should be used in adjusting ventilator settings for optimal PVI. There was also unanimous agreement with the statement that there is no single best mode for all patients under all circumstances. The majority of those present (11 out of 13) agreed with Neil MacIntyre's statement that, in general, volume-targeted breaths are more difficult to synchronize with the patient than pressure-targeted breaths. However, when I asked how many of the speakers used each of the available modes, during the first 24 hours, for the majority of their patients with acute respiratory failure, the vote was 6 for volume-targeted continuous mandatory ventilation to 7 for pressure-targeted continuous mandatory ventilation. There was general agreement, though, that appropriate use of whatever mode is selected is probably more important in the long run than which mode that turns out to be.

Summary

Table 3 lists, in no particular order, what I consider to be the most important take-home messages from this conference. These messages emerged not only from the speakers' formal presentations but also from the sometimes heated discussions that followed them. It is now firmly established that patients with acute respiratory failure require some ventilatory support, but not too much (Fig. 6).

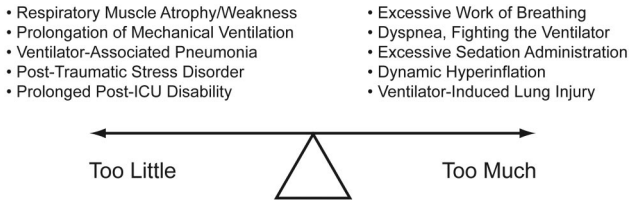

Fig. 6. How to achieve the right balance between too little and too much patient-ventilator interaction is an important challenge for future research in mechanical ventilation as well as for the clinician at the bedside. The balance varies according to the interplay of all the factors depicted in Figure 1, and is probably somewhat different in different patients as well as in different clinical settings.

The challenge is to know how much is the right amount, and to tailor it to the patient's needs for maximum comfort without providing injurious tidal volumes or transpulmonary pressures. Today, in addition to bedside observation and physical examination, graphical analysis and other input from the ventilator are crucial for detecting PVA and optimizing PVI. This conference and the papers it has generated go a long way toward knowing how to use this information most effectively.

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