Using Ventilator Graphics to Identify Patient-Ventilator Asynchrony

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Patient-ventilator interaction can be described as the relationship between 2 respiratory pumps: (1) the patient's pulmonary system, which is controlled by the neuromuscular system and influenced by the mechanical characteristics of the lungs and thorax, and (2) the ventilator, which is controlled by the ventilator settings and the function of the flow valve. When the 2 pumps function in synchrony, every phase of the breath is perfectly matched. Anything that upsets the harmony between the 2 pumps results in asynchrony and causes patient discomfort and unnecessarily increases work of breathing. This article discusses asynchrony relative to the 4 phases of a breath and illustrates how asynchrony can be identified with the 3 standard ventilator waveforms: pressure, flow, and volume. The 4 phases of a breath are: (1) The trigger mechanism (ie, initiation of the inspiration), which is influenced by the trigger-sensitivity setting, patient effort, and valve responsiveness. (2) The inspiratory-flow phase. During both volume-controlled and pressure-controlled ventilation the patient's flow demand should be carefully evaluated, using the pressure and flow waveforms. (3) Breath termination (ie, the end of the inspiration). Ideally, the ventilator terminates inspiratory flow in synchrony with the patient's neural timing, but frequently the ventilator terminates inspiration either early or late, relative to the patient's neural timing. During volume-controlled ventilation we can adjust variables that affect inspiratory time (eg, peak flow, tidal volume). During pressure-controlled or pressure-support ventilation we can adjust variables that affect when the inspiration terminates (eg, inspiratory time, expiratory sensitivity). (4) Expiratory phase. Patients with obstructive lung disease are particularly prone to developing intrinsic positive end-expiratory pressure (auto-PEEP) and therefore have difficulty triggering the ventilator. Bedside evaluation for the presence of auto-PEEP should be routinely performed and corrective adjustments made when appropriate. *Key words: ventilator graphics, waveforms, asynchrony, patient-ventilator interface.* [Respir Care 2005;50(2):202–232. © 2005 Daedalus Enterprises]

Introduction

Ventilator graphics are available on almost all current mechanical ventilators and have been available for evaluating the patient-ventilator interface for more than a decade. Both direct and anecdotal evidence (see below), however, suggests that bedside use of ventilators' graphics capabilities is widely underutilized, and standard approaches or guidelines for graphics interpretation are often lacking.
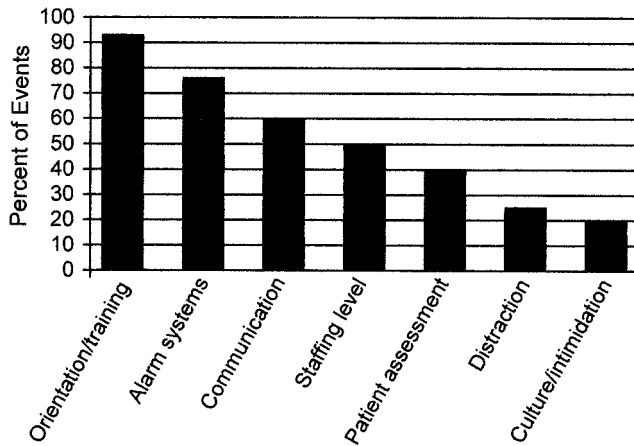
Fig. 1. The primary causes of ventilator-related deaths, 1995–2003. (Adapted from Reference 1, with permission.)

Table 1. Factors That Affect Patient-Ventilator Synchrony

Ventilator Factors
  Trigger variables: esophageal pressure, flow, or shape signal
  Sensitivity setting
  Rise-time capability
  Design, mode, and settings of the flow delivery system
  Flow pattern selected
  Design of the exhalation valve
  How positive end-expiratory pressure is generated by the software
  Extraneous flow (eg, from a nebulizer or added oxygen)
Patient Factors
  Sedation level: pain, splinting
  Inspiratory effort/respiratory drive; neural timing
  Pathology of the respiratory system or abdomen; secretions
  Intrinsic positive end-expiratory pressure
  Size and type of airway
  Presence of leaks

In a recent Joint Commission on Accreditation of Health Care Organizations report on ventilator safety,[1] human error was identified as an important factor. Further investigation of the reported errors identified "orientation and training" as the most frequent root cause of problems (Fig. 1). In addition, only about one third of the members of the respiratory care profession who are responsible for the bedside monitoring of patients receiving mechanical ventilation subscribe to the RESPIRATORY CARE Journal, which indirectly suggests that some information regarding graphics monitoring is probably not widely distributed. One of the reasons for the current Journal Conference was to gather graphical information, because it is not widely published or synthesized in the recent literature. Also, during our presentations on ventilator waveforms at the International Respiratory Congresses in recent years, we used an audience-participation feedback system to test the audience with waveform-analysis questions (unpublished data). The correct response rate ranged from 25% to 75% for fairly simple graphics analysis questions, which we believe suggests wide differences in the general understanding of waveform interpretation.

In describing the interaction between the patient and the ventilator, Kondili et al[2] offered an insightful definition of patient-ventilator synchrony/asynchrony: "During mechanical ventilation the respiratory system is affected by 2 pumps: the ventilator controlled by the physician [or clinician] and the patient's own respiratory muscle pump. Patient-ventilator interaction is an expression of these 2 controllers, which should be in harmony if the result is to be appropriate for the patient."

Though the number of additional factors affecting each of the 2 controllers is fairly substantial (Table 1), the basic description of the 2 pumps being in harmony provides a clear goal for what patient-ventilator synchrony should be; therefore, anything that falls short of that results in some degree of asynchrony, which deserves analysis and correction. Failure to make appropriate adjustments to improve synchrony can cause various complications and deleterious consequences for the patient (Table 2).

The present article reviews graphics methods for recognizing patient-ventilator asynchrony and describes how graphics can be used to improve patient-ventilator interaction.

Table 2. Deleterious Effects of Patient-Ventilator Asynchrony

Patient fights the ventilator
More sedation required
Higher work of breathing
Muscle damage
Ventilation-perfusion problems
Dynamic hyperinflation
Delayed or prolonged weaning
Longer stay
Higher costs

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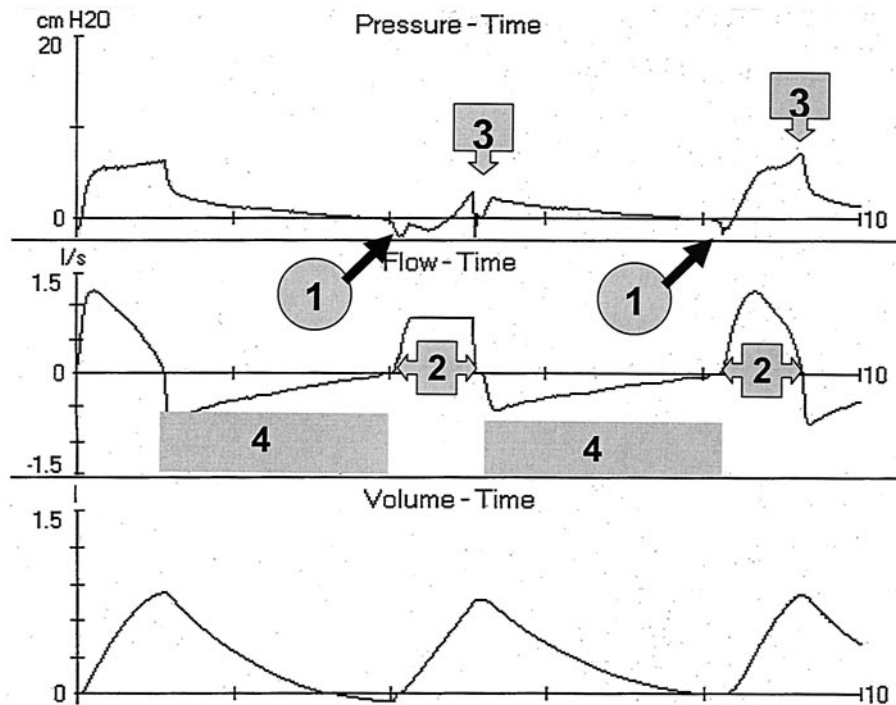
Fig. 2. Airway pressure (top), flow (middle), and volume (bottom) waveforms from a normal subject during synchronized intermittent mandatory ventilation with pressure support. The 4 phases of the breath are numbered. Phase 1 is the initiation of patient effort, which indicates achievement of the trigger threshold (2 cm H_2O) that opens the inspiratory valve. Phase 2 represents the relationship between flow delivery, as determined by the ventilator's flow algorithm, and the patient effort (the first and third breaths are pressure support breaths, in which flow is partially dependent on patient effort, and the second breath is the mandatory breath, which has a constant-flow pattern). Notice the scooped-out appearance of the pressure waveform during the mandatory breath, which indicates that the inspiratory flow was inadequate. Phase 3 is the breath-termination point, which varies based on the type of breath; for the middle breath the inspiratory time is set on the ventilator, but the inspiratory time for the pressure support breath is based on the termination criterion, which in this case is 5% of the peak flow. Phase 4 is the expiration portion of the breath. During this phase the breath should be inspected for evidence of intrinsic positive end-expiratory pressure (auto-PEEP). This expiratory flow waveform returns to zero prior to the next breath, which indicates the absence of auto-PEEP.

The use of guidelines, standards, and protocols for assessing and treating disease states improves patient outcomes.[3] Similarly, a standard approach to analysis of ventilator waveforms should improve patient comfort, reduce work of breathing (WOB), and perhaps improve outcomes. Evaluation of patient-ventilator synchrony can be broken down into 4 phases (Fig. 2): evaluation of triggering; evaluation of adequate flow delivery; evaluation of breath termination; and evaluation of intrinsic positive end-expiratory pressure (auto-PEEP), which is the primary clinical complication associated with the expiratory phase. For organization purposes and as a methodical approach for clinicians to use at the bedside, the present article is organized according to these 4 phases.

Trigger Asynchrony (Phase 1)

Definition: Trigger. "The trigger variable is defined as the variable that is manipulated to deliver inspiratory flow."[4]

"Although triggering composes only a small part of the entire inspiratory cycle, inappropriate setting or design may increase the patient's effort and inspiratory muscle work."[4,5]

"In a demand-flow system (pressure-trigger), the trigger variable is a set pressure that must be attained at the onset of inspiration for the ventilator to deliver fresh gas into the inspiratory circuit."[4] Most microprocessor-based ventilators use pressure-triggering to initiate both the mandatory breaths (assist-control and synchronized intermittent mandatory ventilation) and spontaneous breaths (continuous positive airway pressure, synchronized intermittent mandatory ventilation, pressure support ventilation).[4]

Definition: Trigger Asynchrony. This term has been defined as "muscular effort without ventilator trigger."[6] Though this definition describes the problem when patient effort fails to trigger the ventilator, we will also discuss several additional triggering problems: double-triggering,
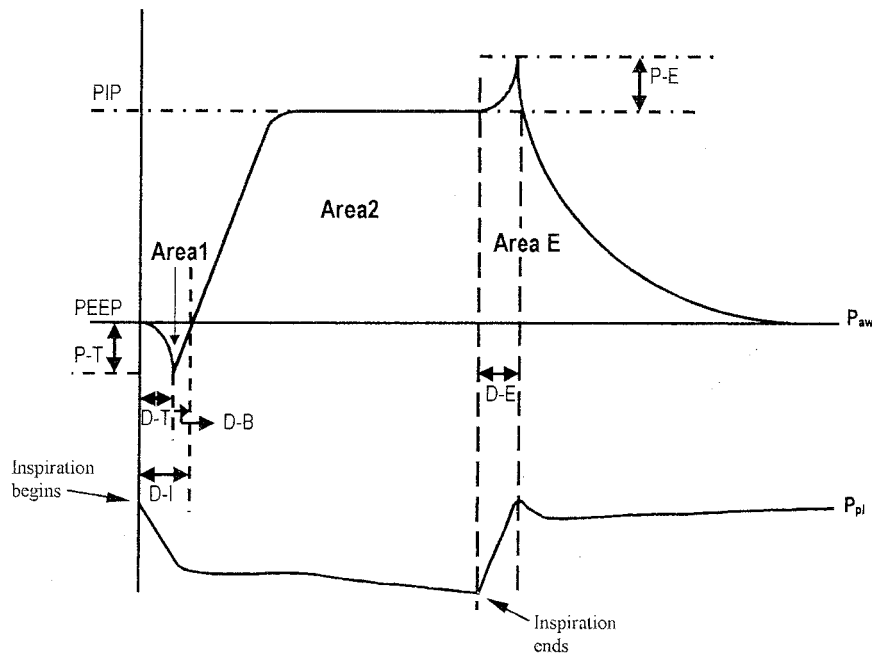
Fig. 3. Schematic of a typical breath. P-T = trigger pressure. D-T = inspiratory trigger time. D-B = time to return airway pressure to zero. D-I = inspiratory delay time. Area 1 = trigger pressure-time product. D-E = expiratory time delay. P-E = supra-plateau expiratory pressure change. Area E = expiratory pressure area. (From Reference 7, with permission.)

auto-triggering, and insensitive trigger (triggering that requires excessive patient effort).

Trigger asynchrony is only one type of problem associated with a patient fighting the ventilator. Though bedside clinicians are inclined to think of trigger problems as being associated primarily with the sensitivity setting on the ventilator, the definition has been expanded to address other variables that are influenced by the patient's inspiratory effort or respiratory drive, and the rate at which the ventilator supplies gas to the circuit (Fig. 3).[7] These variables include (1) the traditional "trigger pressure" or "valve sensitivity," which can be adjusted by the clinician; (2) the pressure maximum, which is the most negative pressure or largest downward deflection in the airway pressure waveform—this value may be more negative than the trigger pressure if the patient has a strong respiratory drive; (3) the inspiratory trigger time, which is the time elapsed between the initial patient effort and the point at which the airway pressure reaches the maximum baseline pressure— for patients with low respiratory drive, it takes longer for the airway pressure to reach the trigger pressure; (4) time to return trigger pressure to zero or baseline—this time is affected by how rapidly the ventilator is able to supply flow to pressurize the circuit, and is influenced by the slope setting; and (5) the inspiratory delay time, which is the total time delay from the initial patient effort until the pressure waveform returns to baseline—this is the sum of the inspiratory trigger time and the time to return trigger to baseline. The patient does not receive any assistance with

the breath until after the inspiratory delay time has passed. If we account for the pressure-drop across the endotracheal tube and the potential presence of auto-PEEP, the patient may not receive any positive inflation support until some time after the inspiratory delay time has passed.

Currently, there are only 2 common types of trigger mechanism available on commercial ventilators in the United States: pressure trigger and flow trigger. Though initial clinical studies indicated that flow-triggering offered some advantage in reducing trigger asynchrony, recent advances in the development of pressure transducers have resulted in nearly equivalent or comparable results.[8-10] A third type of trigger mechanism—the shape-signal or "shadow" trigger—is available in the European market and is now available in the United States as "AutoTrak" on the Respironics BiPAP ventilator.[11] This shape-signal mechanism uses a mathematical model derived from the pressure and flow signals;[12,13] it has some promise for reducing trigger asynchrony, and it may be better tolerated.[14-19] Initial clinical studies have looked at various applications of this derived signal, such as inclusion in proportional-assist algorithms, but this triggering mechanism still has some problems with repetitive auto-cycling (runaway).[15,20]

Trigger Asynchrony Can Occur in Any Ventilation Mode

Clinical studies indicate that ventilator-dependent patients experience trigger asynchrony in all of the common
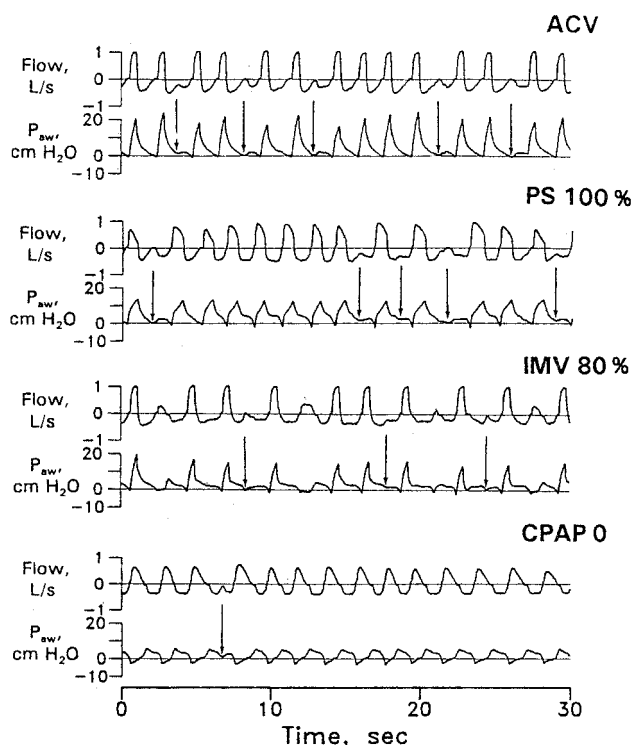
Fig. 4. Flow and pressure waveforms from a patient during assist-control ventilation (ACV), pressure support (PS) of 100%, intermittent mandatory ventilation (IMV) of 80%, and unassisted breathing through the ventilator circuit (CPAP 0). The arrows indicate inspiratory attempts that failed to trigger the ventilator. These failed efforts can also be seen in the expiratory flow waveforms, as abrupt decreases in expiratory flow. In the ASV waveform, note the progressive increase in peak airway pressure in breaths that precede nontriggering efforts, which suggests dynamic hyperinflation. (From Reference 21, with permission.)
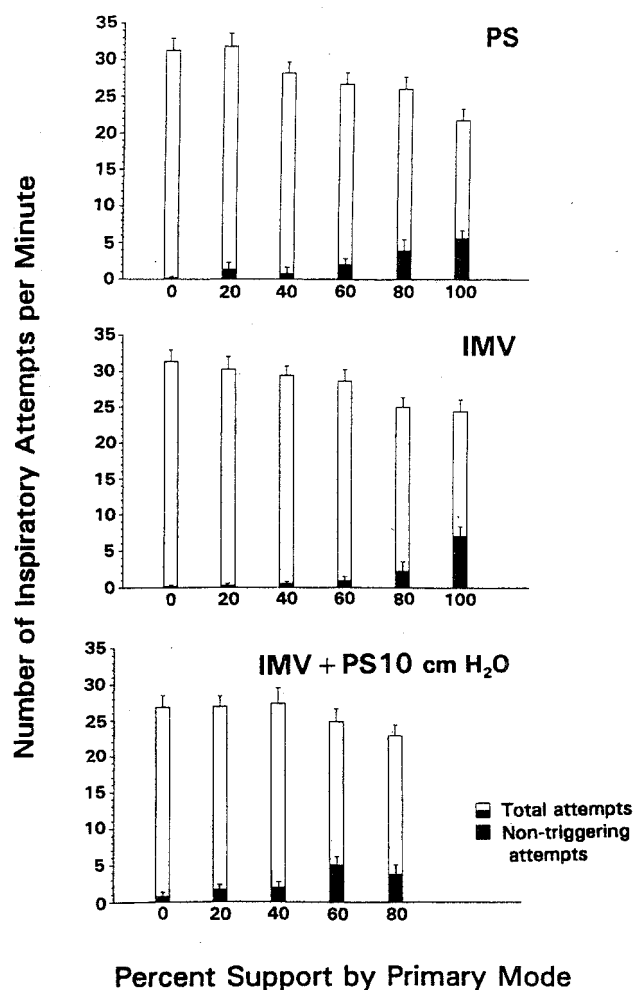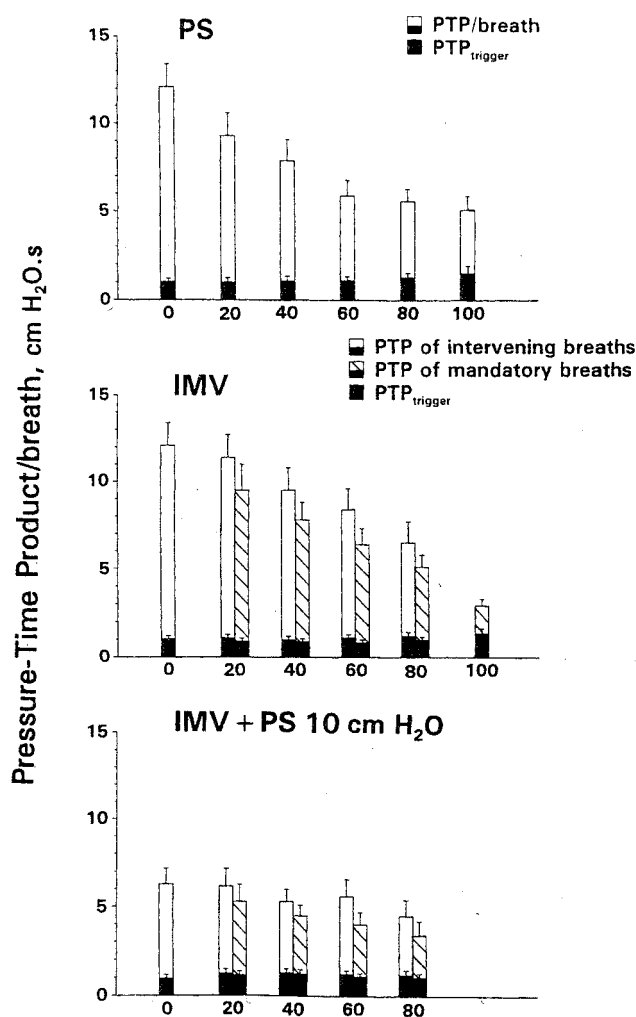

Fig. 5. Total trigger attempts (inspiratory efforts) and failed trigger attempts during pressure support (PS) ventilation, intermittent mandatory ventilation (IMV), and IMV with pressure support of 10 cm H_2O. The black portion of each bar represents the failed trigger attempts, and the overall bar height represents the total number of inspiratory attempts during 1-min. With increasing pressure support the total number of inspiratory attempts progressively decreased, but the number of nontriggering attempts increased. With increasing levels of intermittent mandatory ventilation the total number of inspiratory attempts progressively decreased, but the number of nontriggering attempts increased. With increasing levels of intermittent mandatory ventilation plus pressure support of 10 cm H_2O the total number of inspiratory attempts progressively decreased, but the number of nontriggering attempts increased. (From Reference 21, with permission.)

ventilation modes, including assist-control ventilation (continuous mandatory ventilation), pressure support ventilation, intermittent mandatory ventilation, and continuous positive airway pressure (Fig. 4).[21] Investigation into the cause or pattern associated with trigger asynchrony reveals 2 major points. The first point has to do with the effect that mechanical ventilation has on the patient's drive to breathe. As additional support is provided by the base mode of the ventilator, the patient's drive to breathe decreases, so patient effort subsides and the tendency for trigger asynchrony or failed trigger attempts increases (Fig. 5). Additional clinical evidence from esophageal pressure recordings to measure respiratory muscle activity further supports the reduction in patient effort associated with increasing ventilatory support (Fig. 6). Leung et al found that: "Progressive increases in intermittent mandatory ventilation alone and pressure support alone reduced inspiratory effort and dyspnea."[21]

Leung et al[21] also evaluated the impact of pressure support on respiratory drive and found that "graded increases

in pressure support cause a decrease in respiratory drive (dP/dt), associated with considerable increase in the triggering time" (Fig. 7). If P_{mus} [respiratory muscle effort measured with an esophageal balloon] pressure increases slowly, for example when respiratory drive is small (low P_{aCO_2}, sedation, sleep, high level of assist), the time between onset of inspiratory effort and ventilator-triggering increases, causing asynchrony. If inspiratory effort is great

Fig. 6. Pressure-time product (PTP) of the trigger phase (PTP$_{trigger}$) (black portions of the bars), post-trigger phase (white portions of the bars), and total PTP/breath (the sums of the black and white portions of the bars) during graded levels of pressure support (PS). As the level of ventilator assistance was decreased, PTP/breath and the PTP of the post-trigger phase increased, whereas PTP$_{trigger}$ remained the same. The middle panel shows the PTP$_{trigger}$ (black portions of the bars), the PTP of the post-trigger phase of the mandatory breaths (hatched bars), and the PTP of the intervening unassisted breaths (white portions of the bars) during intermittent mandatory ventilation (IMV) without pressure support. Total PTP/breath is represented by the sums of the black and white (or hatched) portions of the bars. As the level of ventilator assistance was decreased, PTP/breath and PTP of the post-trigger phase of both the mandatory and intervening breaths increased, whereas PTP$_{trigger}$ remained the same. For a given level of intermittent mandatory ventilation (without pressure support), PTP/breath was similar for the mandatory and intervening breaths. The lower panel shows PTP$_{trigger}$ (black portions of the bars), PTP of the post-trigger phase of the mandatory breaths (hatched portions of the bars), and the intervening breaths (white portions of the bars) during intermittent mandatory ventilation with pressure support of 10 cm H_2O. Total PTP/breath is represented by the sums of the black and white (or hatched) portions of the bars. As the level of
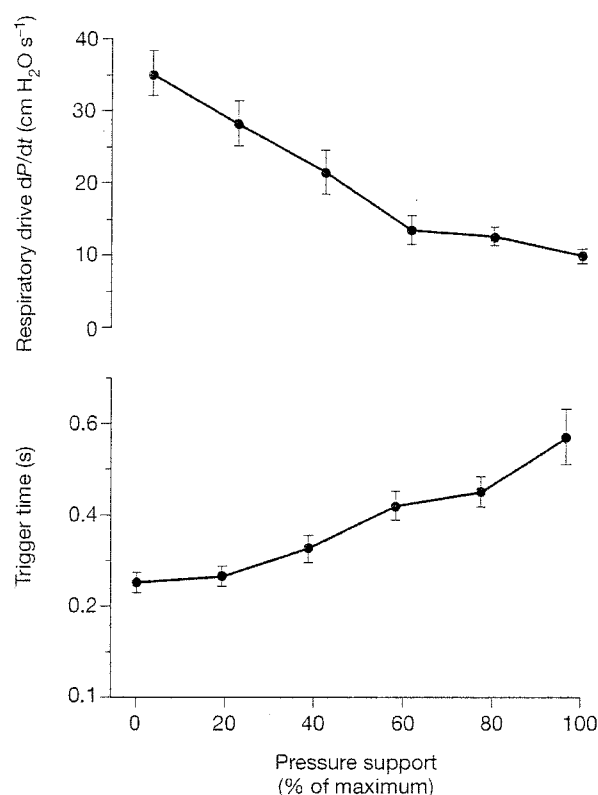

Fig. 7. Graded increases in pressure support cause a decrease in respiratory drive (dP/dt), which is associated with considerable increase in the triggering time. (From Reference 2, with permission.)

(high metabolic rate, high P_{aCO_2}, reduced sedation, reduced ventilatory assistance), then both the rate of increase in P_{mus} and peak P_{mus} will increase, allowing synchrony.[2,21]

During intermittent mandatory ventilation with pressure support, a comparison of the patient effort exerted during the mandatory, ventilator-delivered breath (mandatory breath) and during the intervening pressure support breath indicates a unique (but perhaps not surprising) relationship: the effort during the intervening breaths is carried over to the mandatory breaths (Fig. 8).[21] This raises a fundamental problem with the intermittent nature of intermittent mandatory ventilation. Patients have difficulty altering their respiratory drive between supported and unsupported breaths. Their respiratory effort seems to be equivalent for both the ventilator-assisted breath and the intervening unassisted breaths.

ventilator assistance was decreased, PTP/breath and PTP of the post-trigger phase of both the mandatory and intervening breaths increased, whereas PTP$_{trigger}$ remained the same. For a given level of intermittent mandatory ventilation with pressure support, PTP/breath was similar for the mandatory and intervening breaths. (From Reference 21, with permission.)
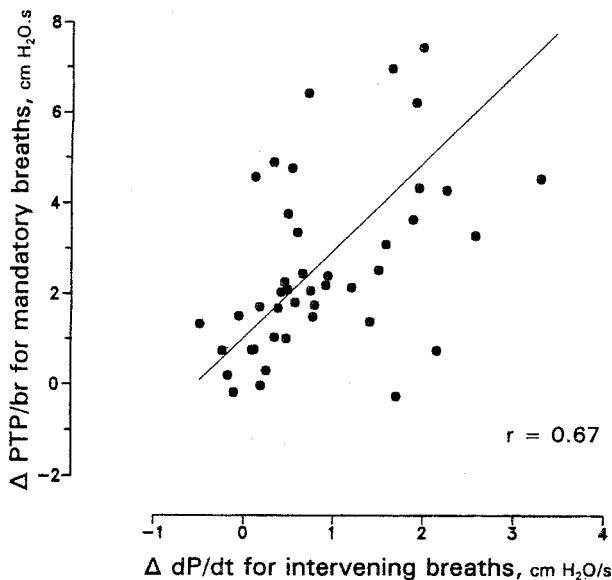
Fig. 8. The change in pressure-time product per breath (PTP/br) during mandatory breaths with the addition of pressure support of 10 cm H_2O to a given level of intermittent mandatory ventilation was correlated with the change in respiratory drive (dP/dt) effected by pressure support during the intervening breaths (r = 0.67, p < 0.001). Unloading by a given level of intermittent mandatory ventilation was increased in proportion to the ability of pressure support to decrease respiratory drive in the intervening breaths. (From Reference 21, with permission.)

> A decline in the mandatory rate of intermittent mandatory ventilation resulted in a decrease in average tidal volume, with inevitable increase in dead space. To avoid a decrease in alveolar ventilation, patients must increase inspiratory effort or respiratory frequency, both of which they did. The resulting increase in drive is carried over to the mandatory breaths, since the respiratory center does not switch off at the moment that ventilator assistance commences.[21]

The latter observation that the respiratory center does not immediately switch off is further supported by the close association between the respiratory drive (as measured by the rate of decrease of the esophageal pressure waveform) and the post-trigger pressure-time product. That means that the respiratory drive at the onset of the breath is a major determinant of the effort expended after triggering has occurred.[21]

The second major point associated with trigger asynchrony has to do with the effect of breathing-cycle dynamics (the relationship between inspiratory time, expiratory time, and total-breathing-cycle time) on the creation of auto-PEEP. While investigating the potential causes of missed trigger attempts, Leung et al[21] evaluated the cycle timing associated with the breaths preceding missed attempts.

The preceding breaths had a shorter total-breathing-cycle time, shorter expiratory time, longer inspiratory time, and a longer ratio of inspiratory time to total-breathing-cycle time than did the breaths that successfully triggered the ventilator. The consequence is a higher level of auto-PEEP at the onset of breaths that were missed than with those that successfully triggered.

This creates an interesting scenario, because the timing mechanisms during the breath substantially impact the creation and development of auto-PEEP, and it is the presence of auto-PEEP that in many instances prevents the patient's effort from being transmitted to the sensor mechanism to trigger the ventilator. Thus, auto-PEEP is a dynamic condition—in some instances occurring on a breath-by-breath basis—and is probably one of the major causes of trigger asynchrony with patients in whom we would not normally suspect air-trapping as a result of their disease state. The presence of auto-PEEP creates a larger pressure gradient (between intrinsic lung pressure and ventilator circuit pressure) that the patient must overcome to reach the trigger threshold. The addition of ventilator-applied PEEP reduces that pressure gradient and moves the circuit pressure closer to the intrinsic airway pressure, in effect reducing the pressure-drop the patient must create to trigger the ventilator.

Patients with chronic obstructive pulmonary disease (COPD) have a high prevalence of trigger problems as a result of auto-PEEP, which can be caused either by the patient's lung pathology or by manipulation of the ventilator.[2,22–24] Many of these studies have confirmed the presence of trigger asynchrony with the esophageal pressure waveform. Though use of the esophageal pressure-monitor seems to be nearly a clinical research standard for evaluation of trigger problems, it is interesting to note that in many instances the patient's missed trigger efforts can be identified in the flow waveform and often in the pressure waveform as well (Fig. 9). In the flow waveform these efforts often appear as a sudden movement of the expiratory flow waveform toward the zero baseline (convexity) and a concomitant drop in the airway pressure waveform toward baseline (concavity). The clinical identification of trigger efforts using the flow and pressure waveforms is of practical importance because commercial use of esophageal pressure-monitoring is currently of limited access; the technology is now available from only one ventilator manufacturer (Viasys, Palm Springs California).

In a study involving patients receiving long-term mechanical ventilation Chao et al[6] identified missed efforts in the esophageal-pressure waveform (see the arrows in the upper portion of Fig. 10). Improvement in the trigger capture rate was accomplished by adding ventilator-applied PEEP, as seen in the lower portion of Figure 10. As noted above, the addition of ventilator-applied PEEP moves the ventilator trigger threshold closer to the patient's intrinsic
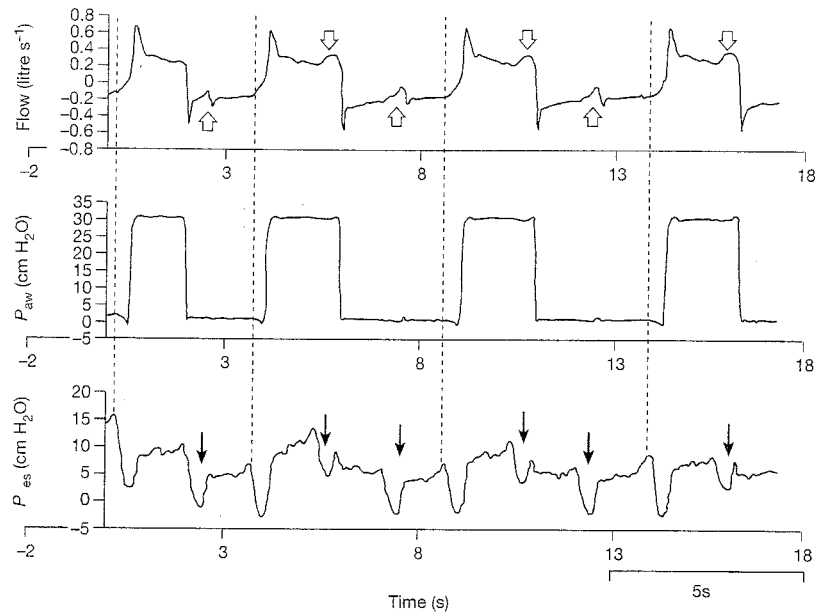
Fig. 9. Flow, airway pressure (P_{aw}), and esophageal pressure (P_{es}) in a patient with severe chronic obstructive pulmonary disease and ventilated with pressure support. The dotted lines indicate the beginning of inspiratory efforts that triggered the ventilator. The thin, black arrows indicate nontriggering inspiratory efforts. Notice the time delay between the beginning of inspiratory effort and ventilator triggering. Ineffective (nontriggering) efforts occurred during both mechanical inspiration and expiration. Those ineffective efforts can easily be identified on the flow waveform; ineffective efforts during mechanical inspiration abruptly increase inspiratory flow, whereas during expiration they result in an abrupt decrease in expiratory flow (open arrows in the flow waveform). The set respiratory frequency is 12 breaths/min, but the patient is making 33 inspiratory efforts per minute. (From Reference 2, with permission.)
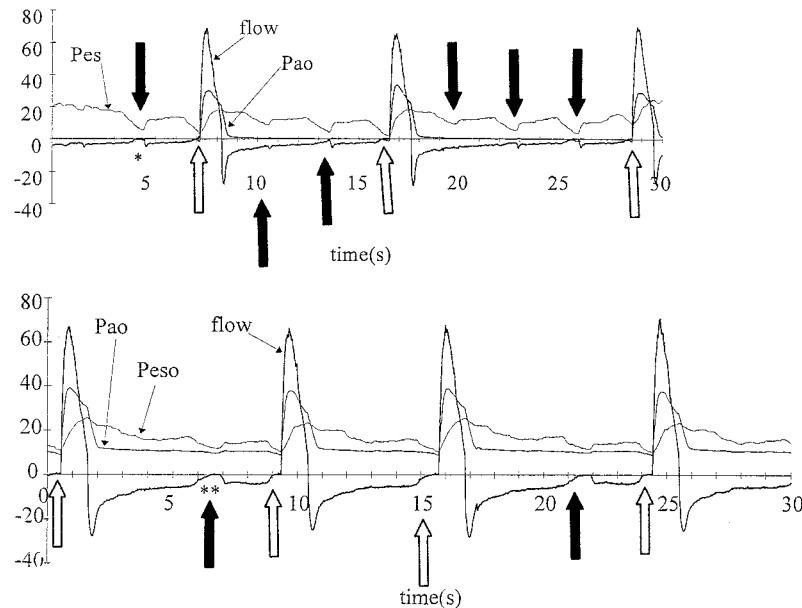

Fig. 10. The upper panel shows waveforms of esophageal pressure (P_{es} in cm H_2O), pressure at the airway opening (P_{ao} in cm H_2O), and flow (in L/min) from a tracheostomized patient with trigger asynchrony during flow-controlled, volume-cycled (assist/control) ventilation. The patient's inspiratory efforts are identified by the negative P_{es} swings. The positive end-expiratory pressure (PEEP) is set at zero. P_{ao} appropriately drops to zero during expiration, indicating little circuit or valve resistance. Trigger asynchrony is evident; there is one triggered breath (white arrows) for every 3–4 inspiratory efforts (black arrows point to nontriggering efforts). Prolonged expiratory flow is due to airflow limitation. P_{es} swings have little effect on retarding the expiratory flow and even less effect on P_{ao}, depending on the phase of expiration. In the lower panel, PEEP was increased to 10 cm H_2O, so P_{ao} during expiration is 10 cm H_2O. There is persistent flow at end-expiration, which indicates auto-PEEP. Trigger asynchrony has improved; there is one triggered breath for every 2–3 inspiratory efforts. There is less limitation of expiratory flow, and the P_{es} swings are more effective in retarding the persistent expiratory flow. Peak inspiratory pressure and P_{es} have slightly increased (compared to the waveforms in the upper panel), which probably indicates a higher end-expiratory lung volume and total PEEP level. (From Reference 6, with permission.)
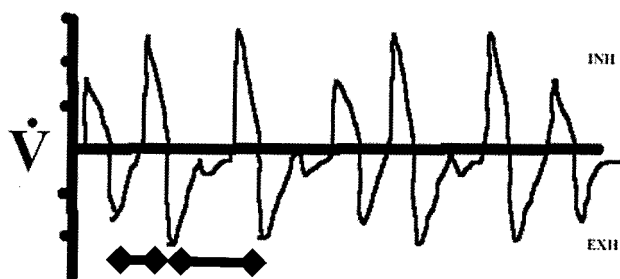
Fig. 11. Flow-time waveform from a patient receiving intermittent mandatory ventilation with pressure support. Large tidal volume (V_T) during the mandatory breath prolongs expiratory time (lengthened time constant) that exceeds the patient's neural timing mechanism. Additional patient efforts that fail to trigger the ventilator are evident in the expiratory flow waveform. $\dot{V}$ = flow. INH = inhalation. EXH = exhalation. (Courtesy of Kenneth D Hargett RRT, The Methodist Hospital, Texas Medical Center, Houston, Texas)

airway pressure, making it easier for the patient to achieve the trigger pressure.

Figure 11 illustrates the impact of large tidal volumes (V_T) provided during the mandatory breaths and the impact on expiratory timing. Following the large mandatory breath, expiratory time is slightly prolonged and interferes with the patient's internal timing mechanism, so the next inspiratory effort (seen as a sudden drop in the expiratory flow, to nearly zero) occurs prior to completion of exhalation. As in previous waveforms, this example illustrates the importance of carefully evaluating the flow waveform to identify additional patient efforts.

Figures 12–14 illustrate a different type of trigger problem: double-triggering. In complete contrast to the problems associated with missed trigger attempts, double-triggering results from patient demand or effort exceeding the volume- or flow-delivery settings on the ventilator. In other words, this is not generally a valve-sensitivity problem, but a condition in which the flow or volume settings have not been adjusted to meet the patient's demand, or a condition arises in which the patient has a sudden increase in demand that far exceeds the normal settings implemented for the resting breathing condition.

In Figure 12, in the esophageal pressure waveform we can see the continued inspiratory effort exerted by the patient despite the dramatic oscillations in the pressure and flow waveforms. In Figures 13 and 14 the airway pressure waveforms indicate inadequate flow (concave or dished out pressure waveform) and the continuation of patient effort, leading to double-triggering. Clinical situations in which double-triggering might occur include sighs, breathing efforts associated with coughing, or more serious conditions in which the volume or flow settings are set inappropriately low. Sighs or coughing may be considered normal if of short duration and without important adverse effects. They may be resolved by temporarily disconnecting the patient until the episode
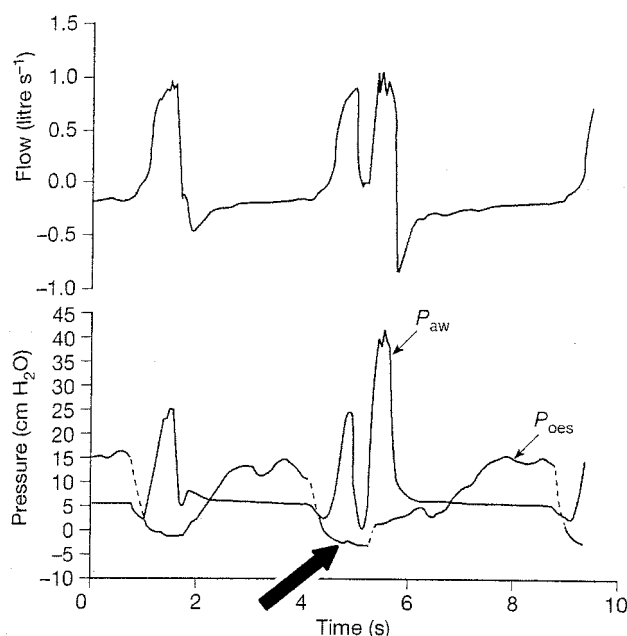

Fig. 12. Waveforms of flow, airway pressure (P_{aw}), and esophageal pressure (P_{es}) from a patient recovering from acute lung injury and ventilated with volume assist-control ventilation with constant inspiratory flow. In the second breath, tidal volume (not shown) was decreased at the same inspiratory flow, which prematurely ended mechanical inspiration. Because inspiratory muscles continued to contract (arrow), they developed pressure that overcame the elastic recoil at the end of inspiration. As a result, P_{aw} decreased below the triggering threshold and the ventilator therefore delivered a new mandatory breath. The ventilator was triggered 3 times by the 2 inspiratory efforts. Observe the high P_{aw} of the third mandatory breath, because lung volume was greater (the volume of the third breath was added to that of the second). Notice also that the total breath duration of the patient's second respiratory effort was considerably longer than that of the first, owing to activation of the Hering-Breuer reflex by the high, volume-prolonging, expiratory time. (From Reference 2, with permission.)

is over. Inadequate flow or volume, however, requires immediate intervention.

Valve sensitivity settings result in 2 additional types of trigger problems: (1) auto-triggering and (2) insensitive valve, in which the sensitivity is set so that it is too difficult for the patient to trigger. The first of these, which is auto-triggering, is possible in all current triggering methods.[2] "It may be caused by random noise in the circuit, water in the circuit (which can cause abrupt changes in circuit resistance), leaks [circuit leaks; see Fig. 15, cuff leaks, or chest tube leaks], and cardiogenic oscillations."[25,26] Setting the trigger-sensitivity requires balancing 2 objectives: to set the sensitivity as low as possible, so that it triggers with minimal patient effort, yet set it high enough to avoid auto-triggering. Imanaka et al[26] found that approximately 20% of cardiac surgery patients experienced auto-triggering episodes during
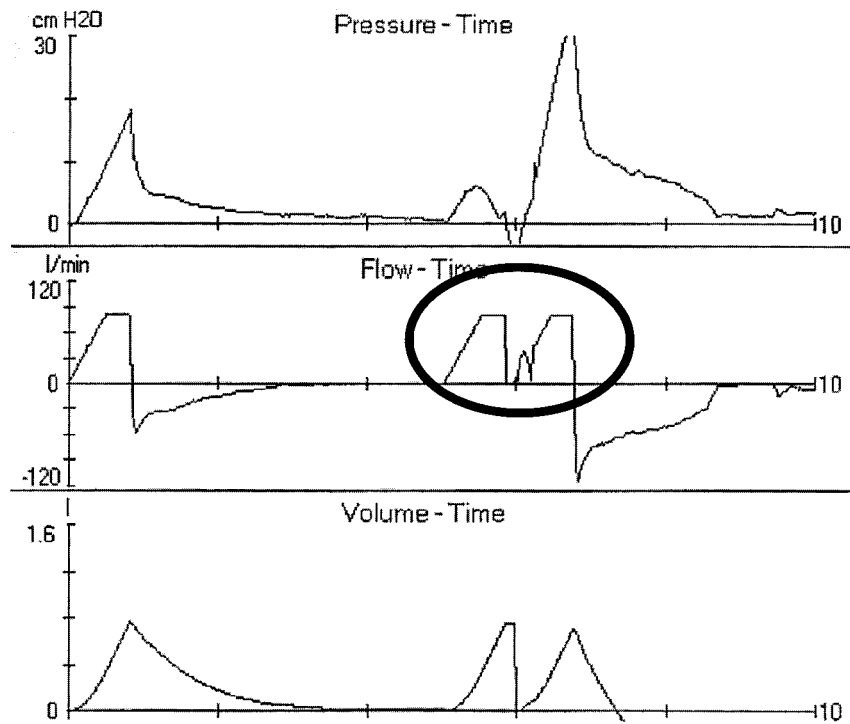
Fig. 13. Double-triggering seen in flow and volume waveforms from volume-controlled ventilation. Continued subject effort during the second breath causes the airway pressure to drop below the trigger threshold, which initiates an additional "stacked" breath. Note the large increase in peak airway pressure caused by the stacked breath and the high peak expiratory flow following the stacked breath.
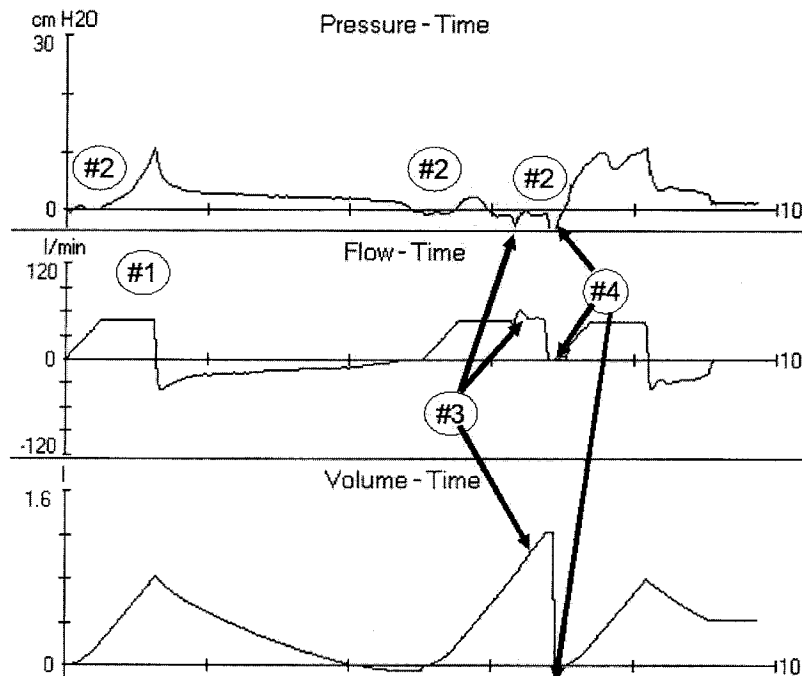

Fig. 14. Inadequate flow and volume result in double-triggering during volume-controlled ventilation. During the first breath, slow valve-opening (#1, as seen early in the flow waveform) and inadequate peak flow cause the dished-out appearance of the pressure waveform (#2). In the second breath, again the valve-opening is too slow (#2) and inadequate peak flow results in additional gas being pulled through the demand valve (#3), as seen by the appearance of the flow bump near the end of the flow waveform and the additional tidal volume beyond the set volume (in the volume waveform). Continued inspiratory effort by the subject results in a second trigger and a stacked breath (#4). The subject was breathing through a mouthpiece and filter, and was disconnected following the stacked breath, as seen in the volume waveform.
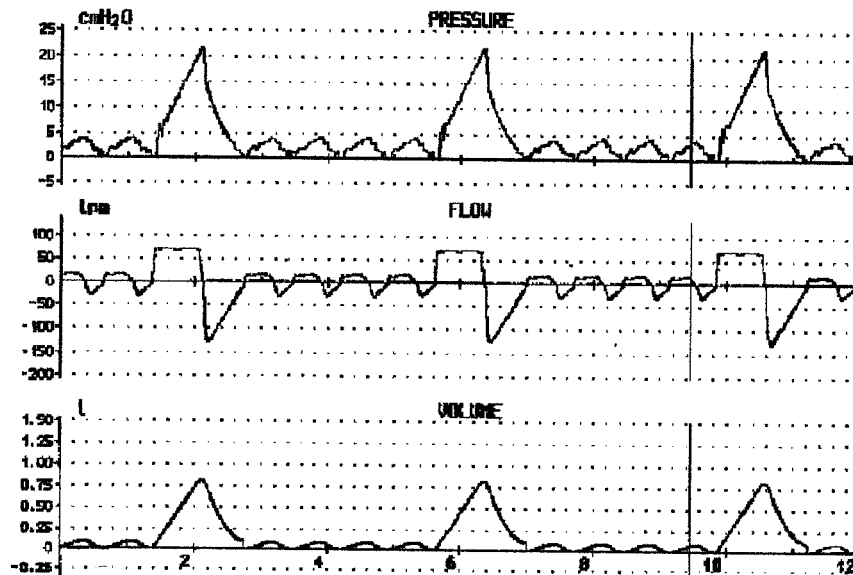
Fig. 15. Auto-triggering created by small circuit leak (tubing connection). The waveform sequence is from a Bear 1000 ventilator, set on intermittent mandatory ventilation plus 5 cm H_2O of pressure support, connected to a Michigan Instruments test lung. The small leak caused the circuit pressure to drop below the set positive end-expiratory pressure and trigger the ventilator.

flow-triggering (Fig. 16). Auto-triggering was more prevalent in patients who had acquired valve disease and had more dynamic circulatory characteristics (larger heart size, larger cardiac output, higher ventricular filling pressure, and lower respiratory-system resistance). These characteristics result in larger cardiogenic pressure oscillations, which when transmitted to the airway, can cause auto-triggering.

> Auto-triggering occurs more often with low respiratory drive and breathing frequency and when dynamic hyperinflation is absent. Such factors allow zero flow for some time during expiration before the next inspiration, making the system vulnerable to triggering from changes of airway pressure that are not caused by inspiratory effort.[2]

In contrast to auto-triggering, an inappropriately set sensitivity dial (insensitive valve) may result in triggering difficulty and unnecessary patient work (Fig. 17). Close inspection of the pressure and flow waveforms will indicate where patient effort begins and whether the valve is appropriately responding to the patient's effort. Another common cause of missed trigger attempts is the addition of extra flow to the circuit during nebulizer treatments. To avoid this, the trigger mechanism should be set to pressure-trigger (not flow-trigger), and the trigger sensitivity should be appropriately adjusted. The use of the newer vibrating-mesh nebulizers eliminates this problem but may also increase costs.

Flow Asynchrony (Phase 2)

Flow asynchrony is the second of the 4 components to evaluate when analyzing patient-ventilator synchrony. Referring back to the 2-pump model, flow asynchrony occurs whenever the ventilator flow does not match the patient flow. Flow asynchrony is a common problem, and the flow setting may be the most frequently incorrectly-set ventilator parameter.[27] The following discussion of flow asynchrony is divided into 2 sections, based on the 2 general methods for delivering gas: volume-controlled ventilation with a fixed flow pattern, and pressure-controlled ventilation with variable flow.

Volume Ventilation With a Fixed Flow Pattern

The adequacy of flow during volume-controlled ventilation can be evaluated with the pressure-time waveform (Fig. 18).[28] Since the total work performed during the breath is the sum of the patient work and the ventilator work, we can evaluate the relative contributions of both by comparing the shapes of the pressure-time waveform during 2 different conditions: completely passive breathing, during which the waveform has a defined pattern based on the type of flow (constant-flow, descending-ramp, or sinusoidal);[29] and patient-triggered breathing, during which the additional patient effort "dishes out" (ie, makes concave) the pressure waveform, relative to the amount of patient work performed. The hatched area in Figure 18 illustrates the pressure-time product and represents the ef-
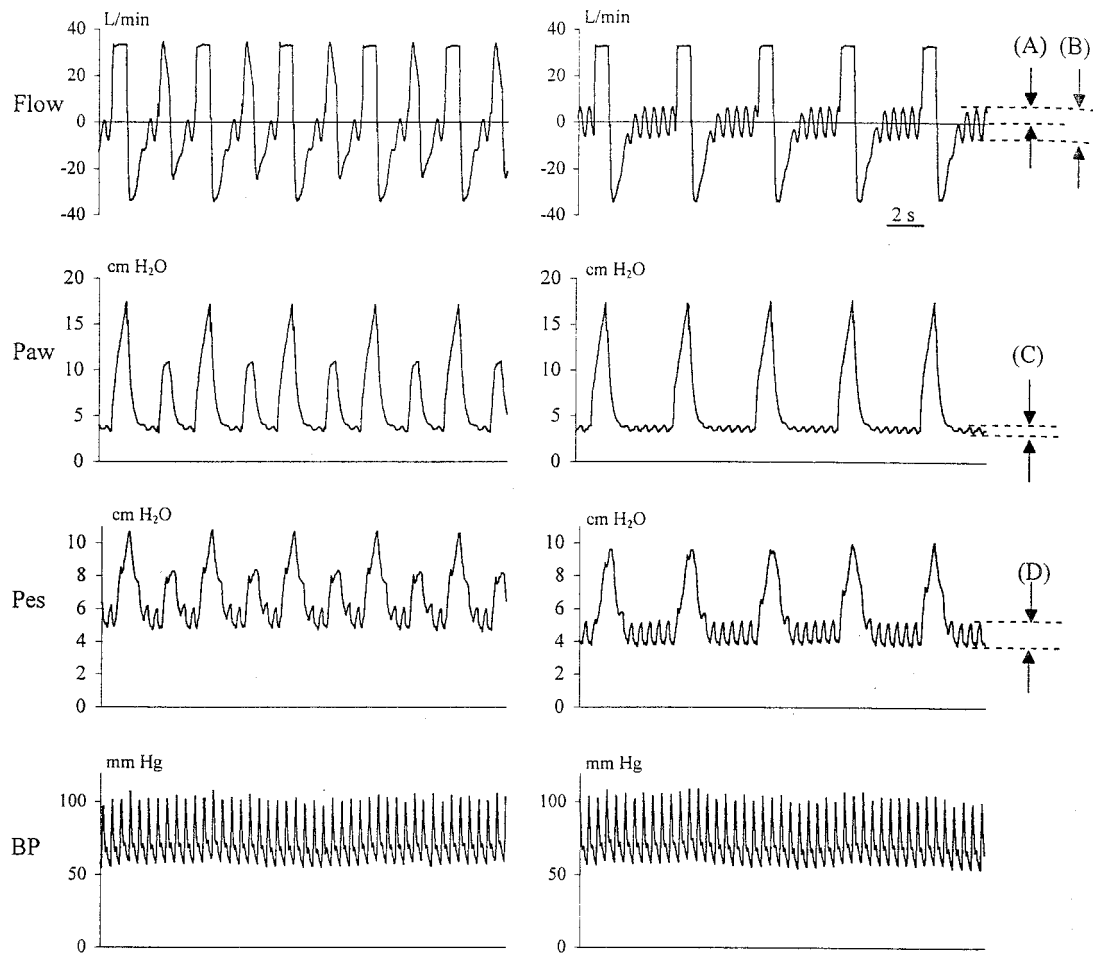
Fig. 16. Representative flow, airway pressure (P_{aw}), esophageal pressure (P_{es}), and arterial blood pressure (BP) waveforms from a patient who underwent mitral-valve replacement and tricuspid annuloplasty for mitral stenosis, tricuspid regurgitation, and aortic regurgitation. With triggering sensitivity set at 1 L/min (left), pressure support ventilation was activated between 2 synchronized intermittent mandatory ventilation breaths. When trigger sensitivity was changed to 4 L/min (right), pressure support breaths disappeared and there was marked oscillation in flow, P_{aw}, and P_{es}. Cardiogenic oscillation was evaluated as the peak inspiratory-flow fluctuation (A), amplitude in the flow oscillation (B), amplitude in airway pressure (C), and amplitude in esophageal pressure (D). Also note that the baseline of esophageal pressure was elevated when auto-triggering occurred, suggesting hyperinflation of the lungs. (From Reference 26, with permission.)

fort the patient contributed to the delivery of the breath. Figure 19 illustrates the pressure pattern associated with a constant-flow mandatory breath during passive breathing. Figures 20 and 21 illustrate breathing sequences during which the first breath is passive and during the second breath the patient took a more active role; note the dished out appearance of the second breath. Figure 22 represents a similar sequence, with increasing amounts of patient effort.

Flow asynchrony is sometimes mistaken for trigger asynchrony, and in particular trigger problems associated with incorrect sensitivity settings. The difference can be determined by carefully evaluating the vertical alignment of the pressure and flow waveforms. Figure 23 illustrates a sequence in which patient effort progressively increased. By adding vertical lines to the leading edge of the flow wave-

form, we can determine that the ventilator has sensed the beginning of patient effort and correctly initiated the breath. In this instance, however, as patient effort increases, the peak flow set on the ventilator no longer meets the patient's flow demand and the airway pressure waveform becomes progressively dished out.

The reason for evaluating flow asynchrony is to adjust the ventilator to match the patient's flow demand. This can generally be accomplished by adjusting the peak flow setting on the ventilator until the pressure waveform pattern most closely represents the condition observed during passive breathing. However, the patient's flow demand may vary considerably, based on the neural drive to breathe. The flow variability is analogous to the difference in flow demand between the resting breathing pattern and that associated with increasing athletic activity: the higher the
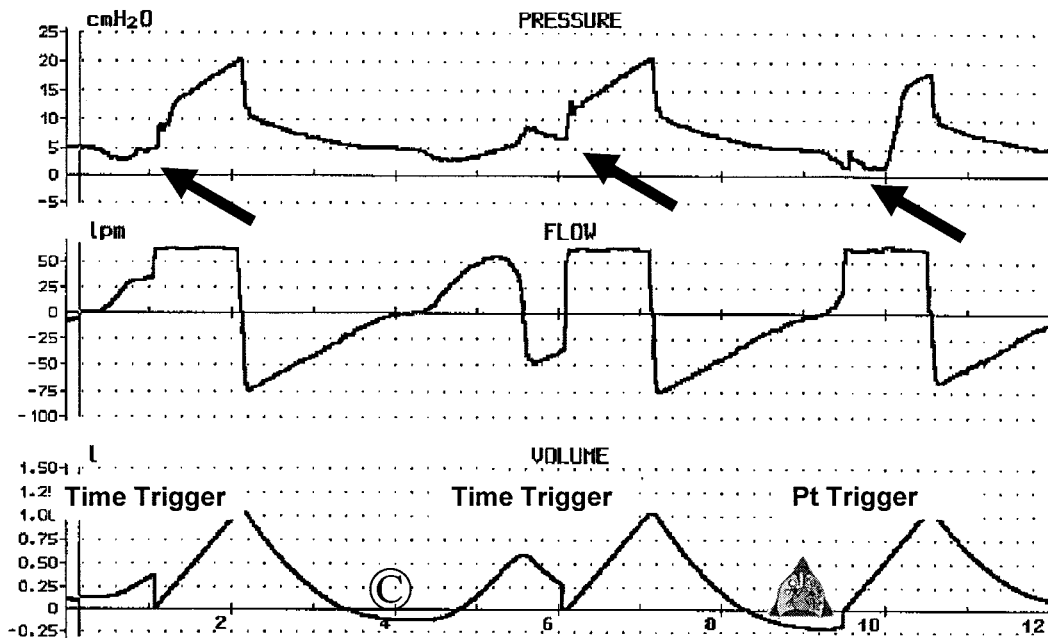
Fig. 17. Evaluating trigger sensitivity with the airway pressure and flow waveforms. The black arrows indicate initiation of mandatory constant-flow breaths. The sensitivity setting is −4 cm H_2O. During the first inspiratory effort the trigger threshold is not reached and the breath is time-triggered, but the patient effort is sufficient to pull gas through the demand valve (seen as the flow that precedes the constant-flow pattern). The next patient effort occurs before the synchronized intermittent mandatory ventilation window is open and the patient again pulls gas through the demand valve. The second mandatory breath is time-triggered (constant flow occurs during patient exhalation). Preceding the third mandatory breath, patient effort reaches the trigger threshold, but again a small amount of gas is pulled through the demand valve (the leading edge of the flow curve is slightly rounded).

exercise intensity, the greater the flow demand. As the patient's flow demand increases, the peak flow should be adjusted accordingly. As with many physiologic variables, there is no single value or flow setting that is appropriate for all patients. Studies indicate that an initial peak-flow setting of 80 L/min may be a reasonable place to start, but subsequent evaluation should be based on observation of the pressure-time waveform.[27]

Ventilator modes that deliver both volume-controlled and pressure-controlled breaths often provide additional information that is useful in evaluating adequate peak flow. As seen in Figure 24 the peak flow of the mandatory breath can be compared with the peak flow of the pressure supported breath in pressure support ventilation. The peak flow of the pressure supported breath is in part determined by the patient effort. Under ideal conditions the peak flow for both breath types should match. In this case the peak flow of the mandatory breath is considerably less than the peak flow of the pressure supported breaths, and needs to be increased. In this example, further confirmation of the inadequate peak flow during the volume-controlled breath is provided by the dished out appearance of the corresponding pressure waveform and the additional flow seen in the middle of the flow waveform (circle in Fig. 24). Current ventilators have fail-safe mechanisms that allow patients to draw flow through the circuit if the power to the
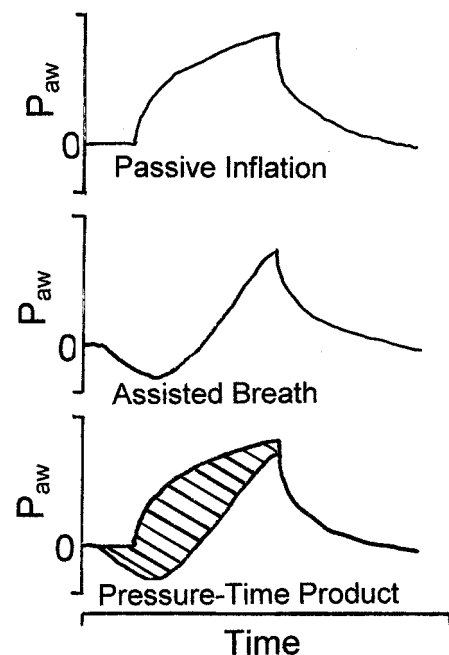

Fig. 18. Airway pressure (P_{aw}) waveforms during controlled mechanical ventilation in a completely relaxed patient (top) and during a triggered breath (middle). The shaded area in the bottom waveform is the pressure-time product of the inspiratory muscles calculated as the difference in area subtended by the P_{aw}-time curve in the presence (middle) and absence (top) of inspiratory muscle activity. (Adapted from Reference 28.)
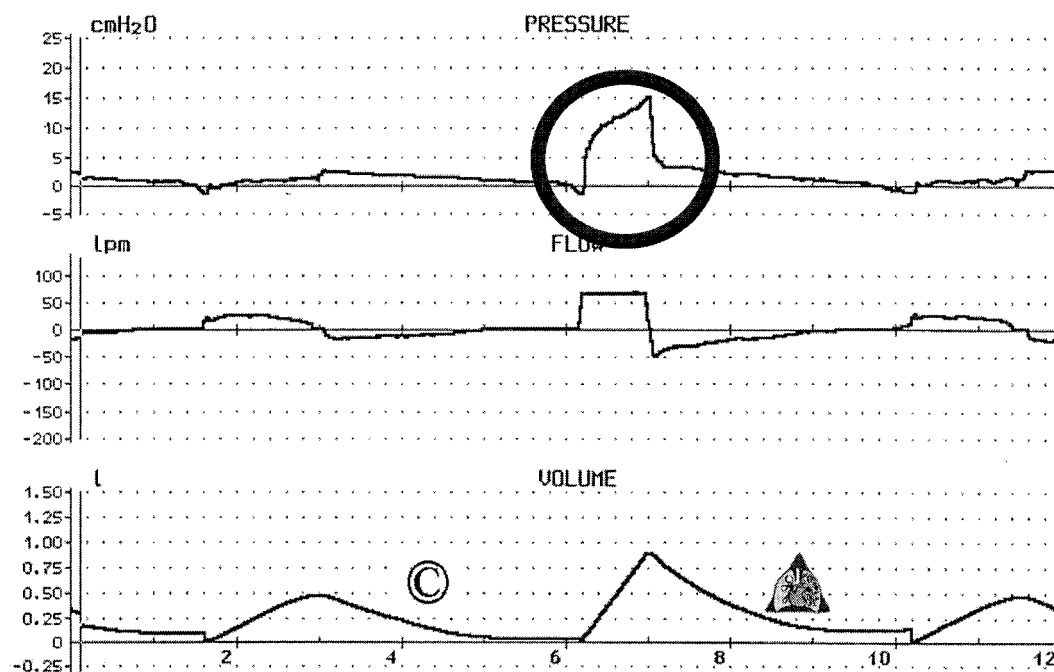
Fig. 19. These waveforms represent relaxed, passive breathing by a subject connected to a Bear 100 ventilator during intermittent mandatory ventilation plus 2 cm H_2O continuous positive airway pressure (PEEP). The circled breath is a mandatory volume breath with a constant-flow pattern.
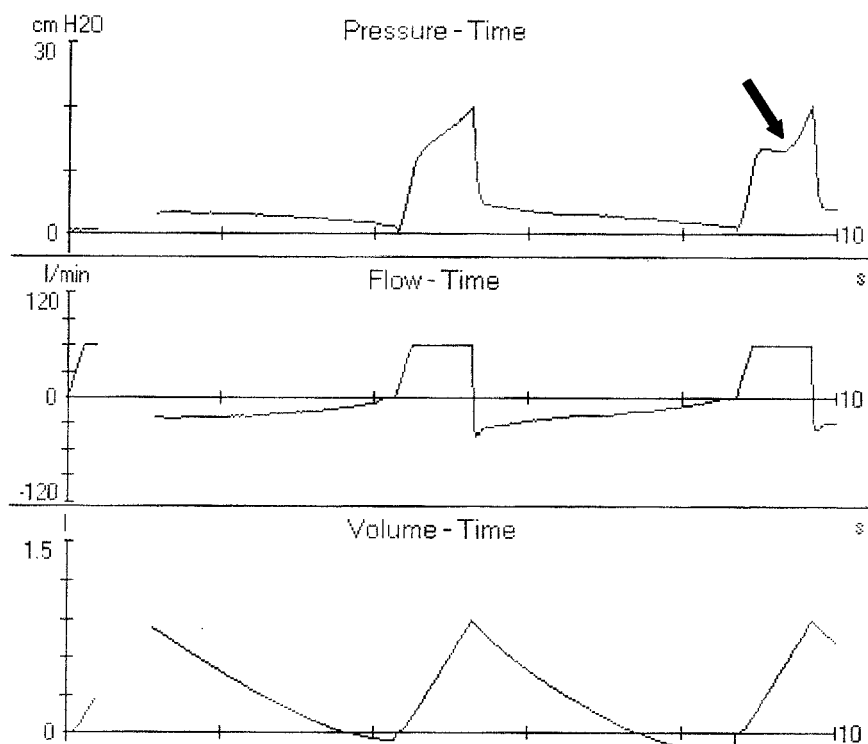

Fig. 20. Two-breath sequence during volume-controlled ventilation. The first breath is passive. During the second breath the subject exerted additional inspiratory effort, which scooped out the airway pressure waveform.
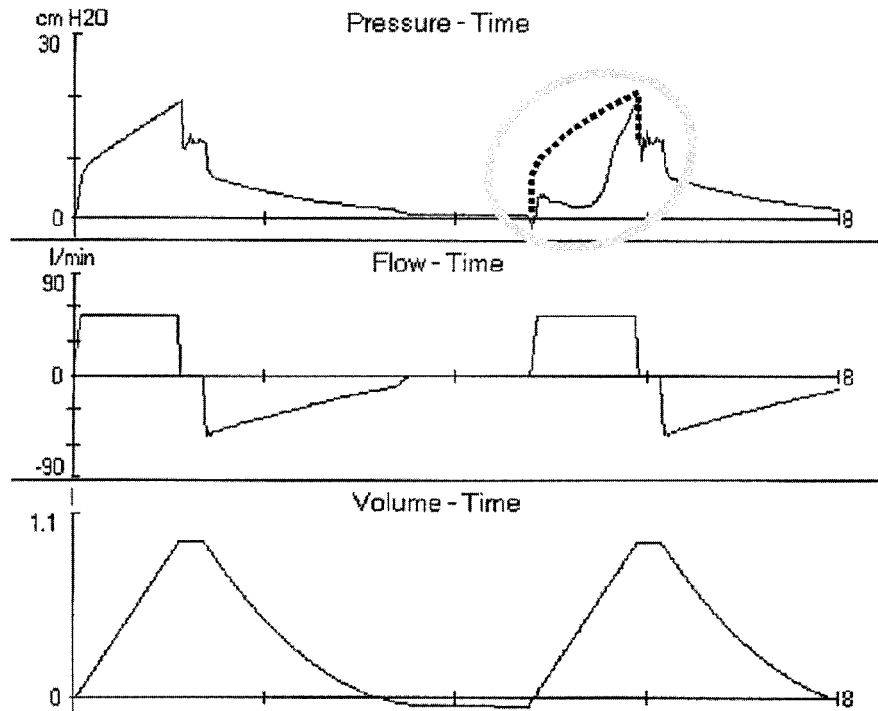
Fig. 21. Two-breath sequence during volume-controlled ventilation, created in a laboratory setting, using a Servo 300A ventilator and a Michigan Instruments test lung. Effort during the second breath was created by manually lifting the test lung. The shaded area represents the pressure-time product associated with the additional simulated inspiratory effort.

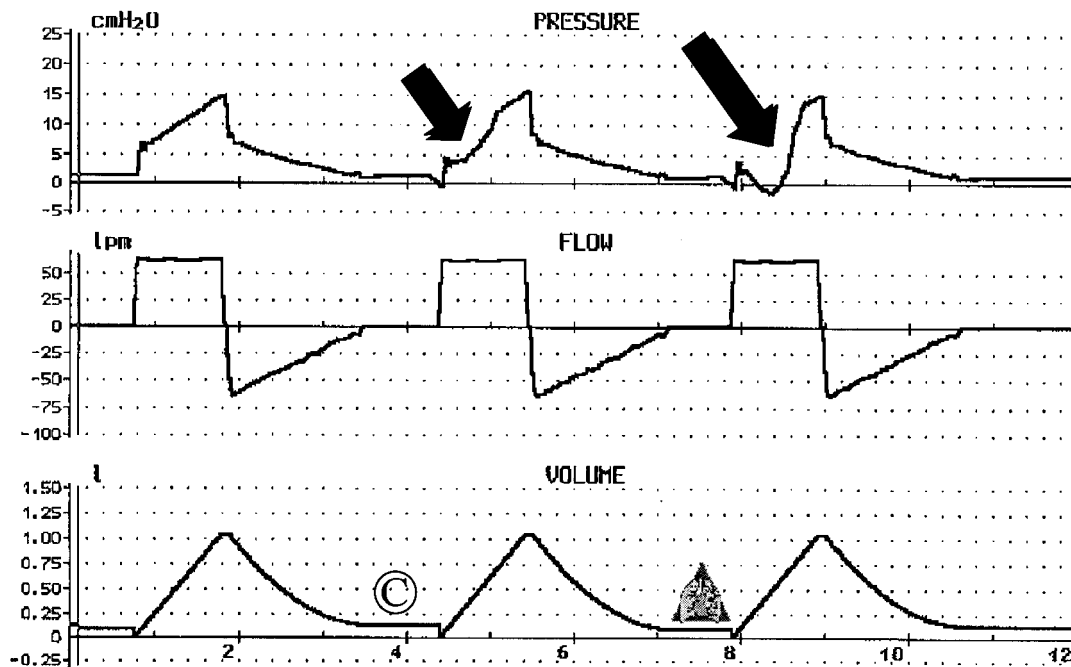Fig. 22. Three-breath sequence obtained with a Bear 1000 ventilator connected to Michigan Instruments test lung during volume-controlled ventilation. Progressive increases in patient effort during breaths 2 and 3 were created by manually lifting the test lung. The dished-out appearance of the airway pressure waveform illustrates the changes from the passive breath when flow does not meet patient demand.
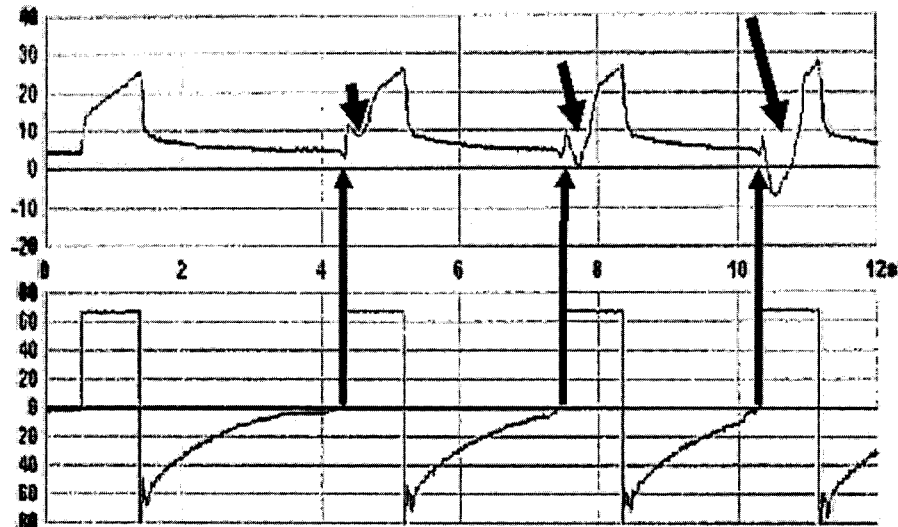
Fig. 23. Evaluation of trigger threshold and patient effort. Progressive increases in patient effort are evidenced by the increasingly scooped appearance of the airway pressure waveform (downward arrows). Trigger synchrony is evaluated by interpreting the beginning of the flow waveform, and by the time relationship to the initiation of patient effort in the airway pressure waveform (upward arrows at the beginning of the constant-flow pattern). Even though inspiratory effort progressively increased, the valve correctly opens after a small drop in airway pressure. Inadequate peak flow, however, causes the dished-out appearance of the airway pressure waveform, as the patient exerts more effort.

Fig. 24. Comparison of peak flow during a constant-flow mandatory breath (circled) and during pressure support breaths during synchronized intermittent mandatory ventilation. The peak flow of the mandatory breath is substantially less than the peak flow associated with the patient-oriented pressure support breath. The inadequacy of the ventilator peak flow is evidenced by the scooped-out appearance of the airway pressure waveform (arrow) and the additional flow that the patient pulls through the demand valve in excess of the constant-flow setting (bump in the middle of the constant-flow waveform).

Fig. 25. These waveforms illustrate inadequacy of ventilator flow while using the descending-ramp flow pattern. Upper panel: As flow decreases 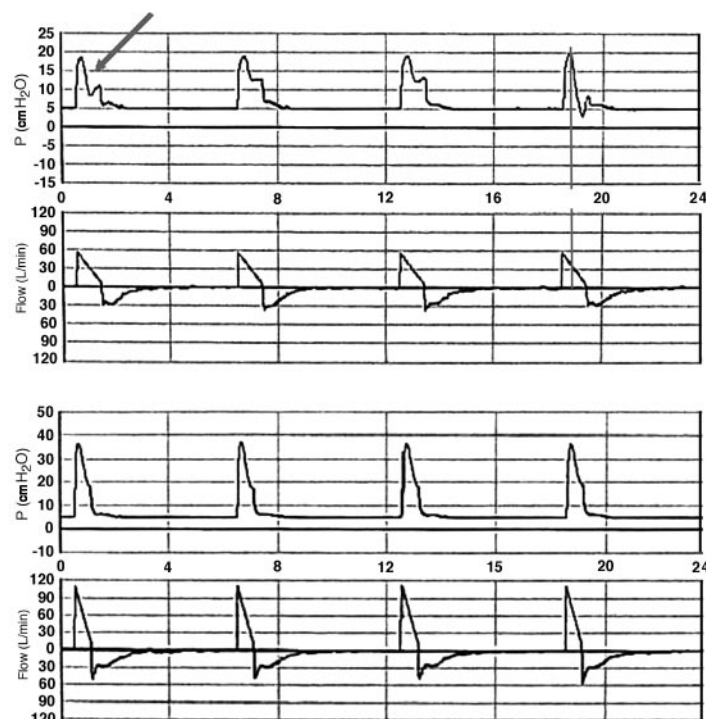during the latter portion of the breath, the patient demand for flow results in scooping of the airway pressure waveform (arrow). Lower panel: The ventilator peak-flow setting was increased from 60 L/min to 120 L/min, which eliminated the scooped appearance of the airway pressure waveform. P = airway pressure. (Courtesy of Kenneth D Hargett RRT, The Methodist Hospital, Texas Medical Center, Houston, Texas)

ventilator fails. In addition, some ventilators (eg, Bear 1000 and Servo 300A) allow the patient to draw additional flow when the patient's flow demand exceeds the ventilator flow settings.

Several studies indicate that the descending-ramp flow pattern with volume-controlled ventilation, or the variable descending-ramp flow pattern with pressure-controlled ventilation, is preferable for patients with COPD and provides better ventilator mechanics, lower peak airway pressure, better gas exchange, and less patient work.[30,31] In some respects the descending-ramp flow pattern is similar to a pressure support breath: it has a high initial peak flow, followed by a decrease to some terminal flow point. However, unlike the pressure support breath, in which the flow adjusts to patient demand, the descending-ramp flow pattern with volume control is fixed and therefore needs to be carefully monitored for flow adequacy. The upper panel of Figure 25 illustrates a patient condition in which the initial peak flow setting under volume control was inadequate and resulted in a concave appearance during the descending-ramp portion of the flow waveform. A subsequent increase in the peak-flow setting (lower panel of Fig. 25) improved the appearance of the flow waveform.

Though changes in flow pattern influence flow synchrony, they also have secondary effects on other portions of the breath and may create additional types of asyn-
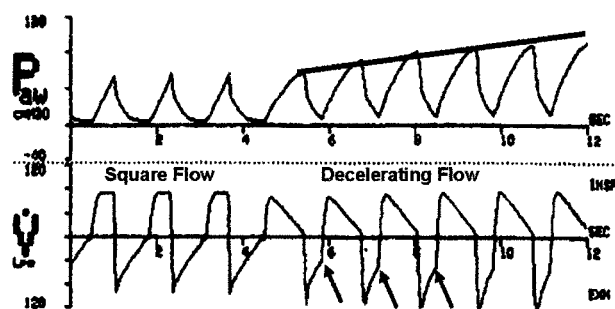

Fig. 26. These waveforms show a situation in which a change in the ventilator flow pattern caused positive end-expiratory pressure (auto-PEEP). During the initial 3 breaths the constant-flow pattern gave sufficient time for the patient to complete the exhalation to functional residual capacity (note the return of expiratory flow to zero prior to the subsequent breath). After the change to the descending-ramp pattern, inspiratory time increased to accommodate the set tidal volume, because of the reduced flow over time, which reduces expiratory time, and the patient developed auto-PEEP, as seen by failure of the expiratory flow waveform to return to zero at the ends of those breaths. Also note the increase in peak airway pressure, which indicates the development of trapped gas. (Courtesy of Kenneth D Hargett RRT, The Methodist Hospital, Texas Medical Center, Houston, Texas)

chrony. Figure 26 illustrates how a simple alteration in flow pattern can create auto-PEEP. In this case the change in the flow pattern from constant to descending ramp slightly increased inspiratory time. With a fixed cycle time,
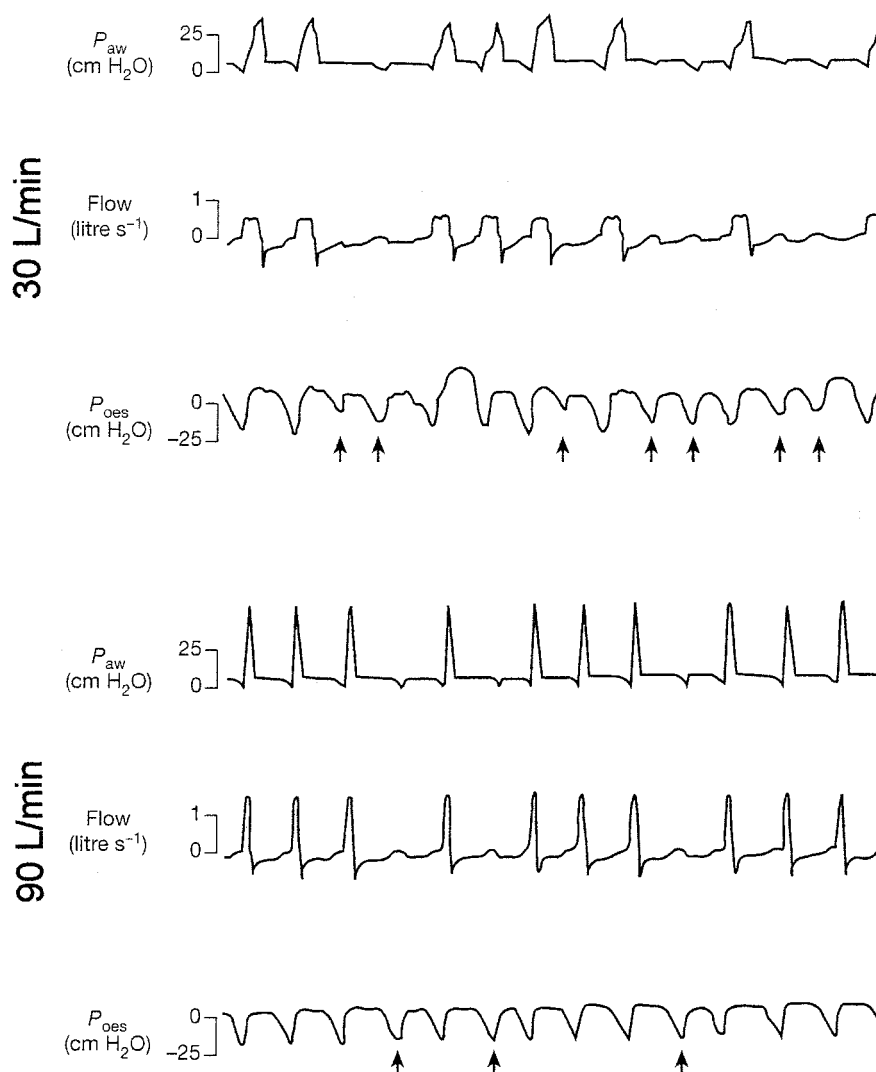
Fig. 27. Airway pressure (P_{aw}), flow, and esophageal pressure (P_{es}) waveforms from a patient with chronic obstructive pulmonary disease, ventilated with volume assist-control ventilation, with 2 inspiratory flow rates: 30 L/min and 90 L/min. With both flow rates, tidal volume was kept constant (0.55 L). Ineffective (nontriggering) inspiratory efforts are indicated by arrows. Increasing the expiratory time (by increasing inspiratory flow at constant tidal volume) decreased dynamic hyperinflation, which reduced the number of ineffective inspiratory efforts, which increased the respiratory rate. (From Reference 2, with permission.)

the prolonged inspiratory time reduced the time alotted for expiration, and resulted in air-trapping. Auto-PEEP can be positively identified in a flow waveform where expiratory flow does not return to zero prior to the onset of the next breath. In addition, the peak airway pressure increases—another indication of air-trapping.

In addition to flow patterns, V_T and peak flow settings also influence inspiratory and expiratory timing. This is particularly important with patients who have severe airflow limitation. In the literature there are examples of patients who were on fixed flow patterns and developed auto-PEEP.[2,32] The upper panel of Figure 27 illustrates a patient who had COPD and a low peak-flow setting that resulted in air-trapping and missed trigger attempts. After the peak flow setting was increased (lower panel of Fig. 27), inspiratory time decreased, expiratory time increased, and auto-PEEP decreased, which resulted in fewer missed trigger attempts. With modern ventilators the incidence of ineffective triggering does not seem to differ between flow-triggering and pressure-triggering systems,[33] but shape-signal triggering shows promise for improving synchrony for many patients with COPD.[34]

Patients with asthma (Fig. 28) can suffer deleterious effects from large V_T and flow-time relationships that result in hyperinflation and missed trigger attempts.[32] During one of the breaths (vertical lines in Fig. 28) much of the neural inspiratory time is spent in pulling through the auto-PEEP, such that the patient effort is nearly over be-
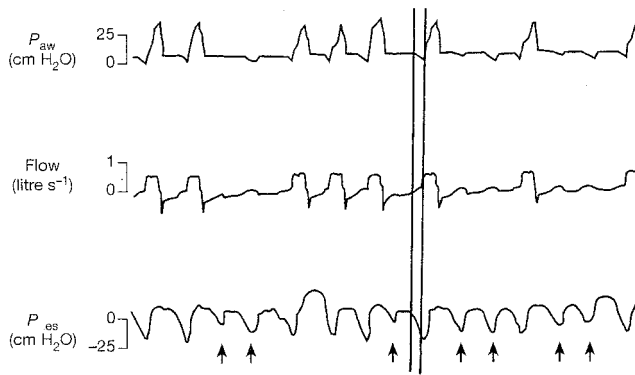
Fig. 28. Airway pressure (P_{aw}), flow, and esophageal pressure (P_{es}) waveforms from a patient with severe obstructive lung disease, ventilated on volume assist-control ventilation. With this ventilation mode, every patient effort should trigger the ventilator to deliver a breath with a predetermined volume and flow waveform, so the mandatory rate should be set by the patient, while the tidal volume and mechanical inspiratory time are determined by the ventilator. Note the substantial number of ineffective inspiratory efforts (arrows). These missed trigger efforts can be identified using the P_{aw}, flow, or P_{es} waveform. An abrupt decrease in P_{aw} and P_{es} during expiration, and hesitation in expiratory flow, which are not followed by a mandatory breath, indicate a missed trigger effort. Note also the considerable delay between the beginning of inspiratory effort (first vertical line) and ventilator triggering (second vertical line). A substantial portion of the inspiratory muscle pressure is needed to trigger the ventilator, and, in some breaths, all the muscle pressure is dissipated to trigger the ventilator, so the neural inspiratory time ends when the mandatory inspiratory time starts. In that situation there is major patient-ventilator asynchrony. Note also that in some breaths, P_{aw} is convex toward the time axis, which indicates that the flow from the ventilator is not meeting the patient's instantaneous flow demand. (From Reference 32, with permission.)

fore the ventilator inspiratory time begins. The presence of hyperinflation, by delaying the ventilator trigger, imposes a substantial delay between the beginning of patient effort and the breath delivery. Expiratory time is further delayed and results in subsequent missed trigger attempts. Though this example does not specifically address adjustments to the flow pattern, it does emphasize the importance of evaluating auto-PEEP and identifying missed trigger attempts in the flow waveform.

V_T also has important affects on hyperinflation. Figure 29 illustrates the impact of V_T and mean expiratory time on the amount of trapped gas in patients with asthma. Larger V_T and shorter expiratory time substantially increase the amount of trapped gas. The volume of trapped gas is significantly correlated with the incidence of barotrauma and hypotension in patients with asthma.[35,36] Strategies to reduce dynamic hyperinflation include: (1) reduce minute ventilation (ie, permissive hypercapnia), (2) increase expiratory time by increasing inspiratory flow, and (3) decrease resistance to expiratory flow by using bron-
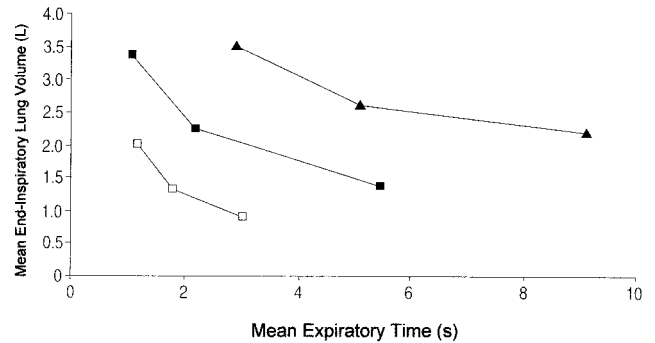

Fig. 29. Mean end-inspiratory lung volume (V_{EI}) (above passive functional residual capacity) plotted against mean expiratory time in patients with severe airway obstruction. The measurements were obtained at 3 tidal volumes: 0.47 L, 0.88 L, and 1.44 L. Note the profound effect of V_T and expiratory time on V_{EI}. Note also the values of V_T and expiratory time associated with a safe range of V_{EI} (ie, 1.4 L) (From Reference 32, with permission.)

chodilators, by using the largest-diameter endotracheal tube appropriate for the patient, and by reducing circuit resistance (eg, connectors and valves).[32]

Dynamic hyperinflation may be identifiable in the pressure-time waveform (Fig. 30), although this requires adjusting the scale on the time axis, or it may be displayed on the pressure-volume loop as an over-distention "beak" (Fig. 31). The latter is more likely to display obvious changes in pattern, but is often missed because the clinician must select a different display option than the standard waveforms. Clinically, the V_T should be reduced until the beak on the pressure-volume loop disappears.

Fig. 30. Airway pressure as a function of time during constant-flow inflation in a patient with status asthmaticus and severe dynamic hyperinflation. Note that the pressure curve is convex downward (arrow), indicating that the system, because of dynamic hyperinflation, approaches total lung capacity at the end of inspiration, during which compliance is relatively low. In this patient the intrinsic positive end-expiratory pressure was 17 cm H_2O. (From Reference 32, with permission.)

Fig. 31. Over-distention "beak" on a pressure-volume loop. In this example, volume in excess of 350 mL abruptly increases pressure, indicating over-distention. V_T = tidal volume. P_{circ} = circuit pressure. (Courtesy of Kenneth D Hargett RRT, The Methodist Hospital, Texas Medical Center, Houston, Texas)

Pressure Ventilation With Variable Flow

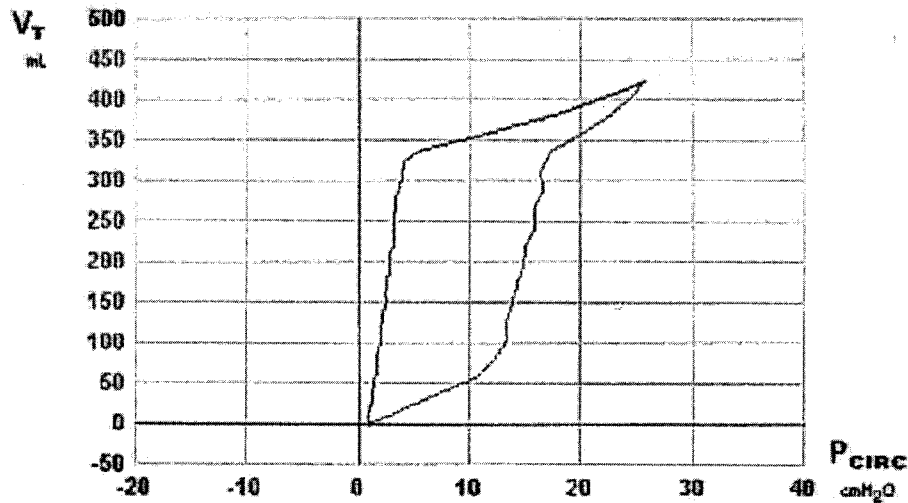Evaluation of flow asynchrony during pressure-controlled ventilation also requires viewing the pressure-time waveform to determine the adequacy of flow delivery. As with volume-controlled ventilation, the flow delivery is assessed by evaluating the presence or lack of concavity in the pressure-time waveform. During pressure-controlled ventilation, however, the peak flow is no longer fixed, but is variable and depends on various factors, including; (1) set target pressure, (2) patient effort, and (3) respiratory-system compliance and resistance. Of those, only the pressure can be set by the clinician. The pressure should be set based on the lowest pressure setting that achieves the desired V_T. The main flow parameter that can be adjusted during pressure-controlled ventilation is

Fig. 32. These waveforms illustrate pressure support breaths using different rise time (pressure slope, or rate of valve-opening) settings, ranging from very slow (1) to very fast (5) valve opening, performed with a nearly constant or stable inspiratory effort (flow demand). The airway pressure waveform appears scooped-out initially and then progresses to a more constant-pressure pattern as the valve setting is changed to open faster. Breath 4 has the best valve-opening setting for these conditions. In breath 5 the valve opens a little too fast and causes a slight pressure-overshoot early in the airway pressure waveform.

Fig. 33. Effect of minimum (mn), medium (md), and maximum (mx) rise times on trigger pressure-time product (T-PTP) during pressure support ventilation (white bars) and pressure assist/control modes (black bars) with 5 ventilator brands. T-PTP decreased as rise time increased, with all the ventilators, in both pressure support and pressure assist/control mode. (From Reference 7.)

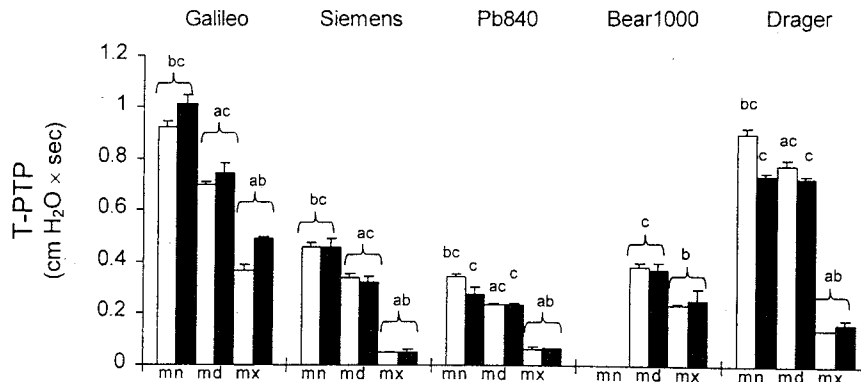the rate of valve-opening (also called rise time, pressure slope, or flow acceleration). Each manufacturer has its own algorithm to control the valve responsiveness or rate of valve-opening, but the basic settings on the ventilator either increase or decrease how rapidly the valve opens from the initial closed position to the open position. In more specific terms, under passive breathing conditions, the rate of valve opening affects the time-to-return-trigger-pressure-to-zero (D-B in Fig. 3) and the time for the pressure to rise from zero to the set peak pressure. Figure 32 shows examples of how different rise time settings adjusted during active breathing affect the pressure, flow, and volume waveforms.

Chatmongklochart et al analyzed various ventilators, with different minimum and maximum rise-time settings.[7] They evaluated the effect of rise time on the total time delay, the trigger pressure, and the trigger pressure-time product (Area 1 in Fig. 3). Slower rise times caused increases in all of the above-named parameters, including the trigger pressure-time product (Fig. 33). They found that "patient effort and WOB are affected by the ventilator's ability to meet patient peak inspiratory demand."[37,38] During pressure-controlled ventilation the rise time should be adjusted to meet the patient's inspiratory flow demand and is ideally set when the pressure waveform has a minimal negative deflection on the front end, a smooth and rapid rise to near peak pressure (while avoiding pressure-overshoot), and the appearance of a short plateau prior to termination (almost-constant-pressure waveform)[39] (see the breath marked by an asterisk in Fig. 32). If the flow demand is not met, the pressure waveform is concave on the front end and the patient performs unnecessary work, resulting in flow asynchrony.

Studies of flow asynchrony during pressure-controlled ventilation suggest that many patients require a rapid rise time because of increased ventilatory demand.[40–42] Bonmarchand et al found reductions in inspiratory WOB in

Fig. 34. Effects of rise time (pressure slope) on the work performed per liter of ventilation (W_I/L) and expressed as a percentage of the values for work observed under spontaneous ventilation (SV). The slope was modulated so that the same plateau pressure (15 cm H_2O) was reached after a period ranging from 0.1 second (T 0.1) to 1.5 seconds (T 1.5). Each curve represents 1 patient ($n = 12$ patients). The bold line indicates variation of the means. Five of the curves are shorter than the rest because the last sequence could not be performed with those patients. (From Reference 43, with permission.)

patients with obstructive[42] and restrictive[43] lung disease (Fig. 34) when rise time was reduced (rapid valve-opening). When rise time is prolonged (slow valve-opening), it is easy to see the increase in patient work, demonstrated by the area under the esophageal pressure waveform (see Fig. 35). Johnson et al reported similar changes in the esophageal pressure waveform and noted the dished-out appearance of the pressure waveform in a case study involving acute lung injury.[39] Figure 36 illustrates a situation in which

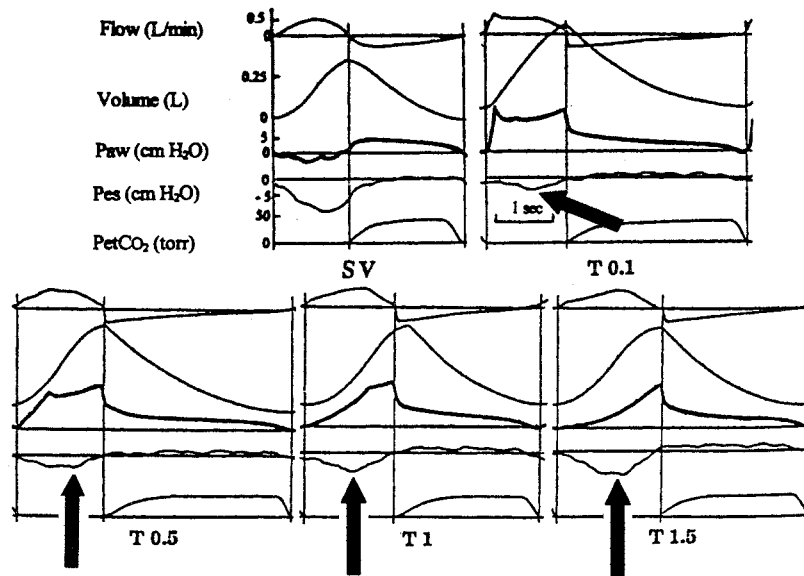Fig. 35. Representative flow, volume, airway pressure (P_{aw}), esophageal pressure (P_{es}), and end-tidal carbon dioxide pressure (P_{ETCO_2}) waveforms from a patient with spontaneous ventilation (SV), and one undergoing pressure support ventilation (pressure support of 15 cm H_2O). The rise time was modulated so that the plateau pressure was reached after a time ranging from 0.1 second (T 0.1) to 1.5 seconds (T 1.5). Each reduction of the pressure ramp slope was associated with an apparently dose-dependent progressive increase in P_{es} swings (arrows). Tidal volume appeared to remain constant. (From Reference 43, with permission.)
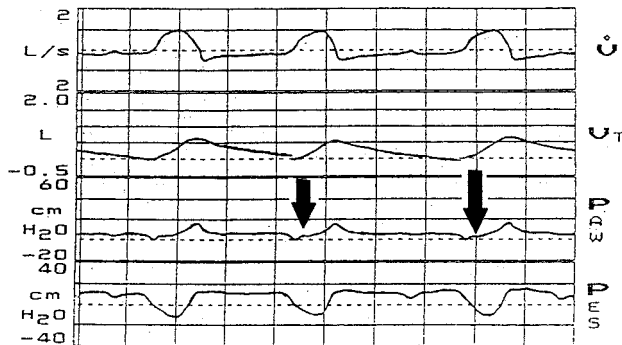

Fig. 36. Waveform recorded by the freestanding respiratory monitor on postoperative day 9. The rise-time setting is 10%. Note the scooped-out appearance of the airway pressure (P_{aw}) waveform (arrows), which indicates inadequate flow. When the initial flow output is lower than patient demand, the rise in airway pressure may be delayed or prolonged, or in some case may prohibit attaining the set pressure support level. $\dot{V}$ = flow. V_T = tidal volume. P_{es} = esophageal pressure. (Adapted from Reference 39.)
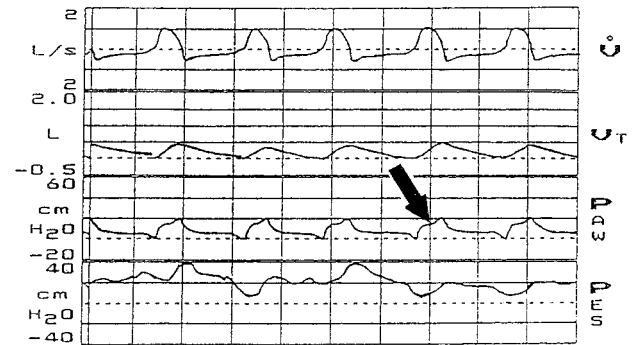

Fig. 37. These waveforms are from the same subject as the waveforms in Figure 36, in which the rise time was 10% (slow valve-opening). In the above figure, the rise time is 1% (rapid valve-opening), which causes a much steeper slope in the beginning of the inspiratory flow waveform. The pressure support level is achieved earlier in the inspiratory phase (arrow), and there is a pressure plateau. $\dot{V}$ = flow. V_T = tidal volume. P_{es} = esophageal pressure. (Adapted from Reference 39.)

the initial ventilator rise-time setting did not provide adequate flow for the patient's flow demand (slow valve-opening). The rise-time setting was increased to meet the patient's demand (Fig. 37), which improved the pressure-time waveform (pressure rises more rapidly to the target pressure) and decreased the esophageal pressure swing.

In contrast, there are both patient and ventilator conditions in which rapid valve-opening causes pressure-overshoot early in the breath, in which case a slower valve-opening setting may be preferable.[40–42] The pressure-overshoot occurs when there is a mismatch between the patient's flow demand and the rate of flow delivery determined by the rise-time setting (see Fig. 38). Rapid rise time can also cause flow asynchrony, patient discomfort, and premature termination of inspiration. Premature termination can be caused by either of 2 mechanisms: (1) early in flow delivery, the initial pressure-overshoot may exceed the target pressure and
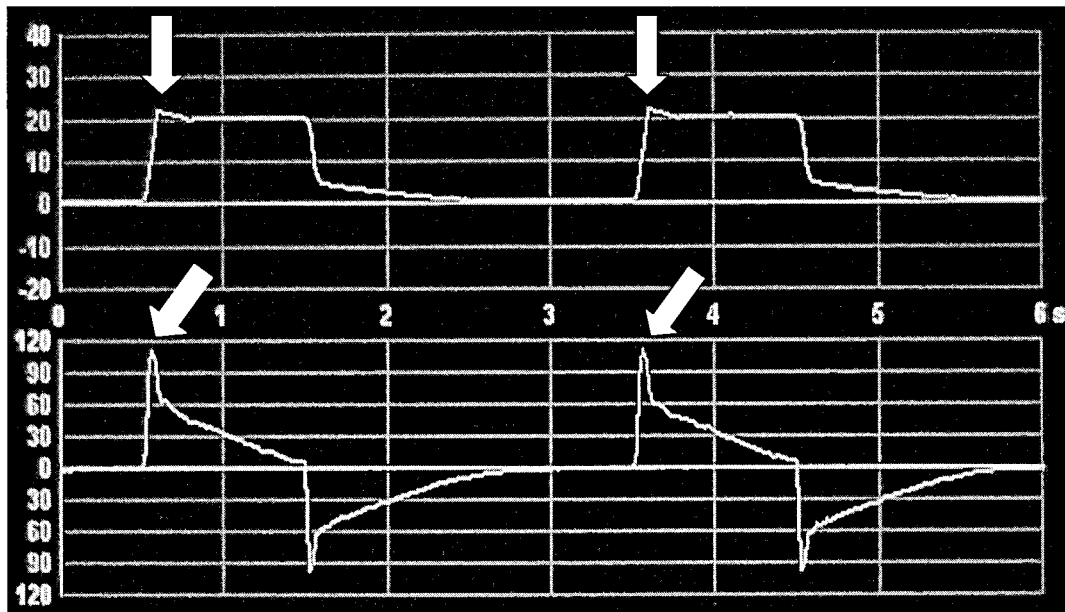
Fig. 38. Rapid valve-opening (fast rise time) results in pressure-overshoot in the airway pressure waveform (arrows). In addition, the initial flow in excess of patient demand creates a high peak flow and a steeper flow decrease, which can cause premature termination of inspiration. (Courtesy of Kenneth D Hargett RRT, The Methodist Hospital, Texas Medical Center, Houston, Texas)

terminate inspiration as part of the secondary breath-termination criteria (pressure termination), or (2) late in the flow delivery, the flow-termination point may occur sooner (percentage of peak flow is now a higher flow-termination value because of the higher initial peak flow) (Fig. 39). The Servo 300A ventilator allows adjustment

of rise time, even during volume-controlled ventilation (Fig. 40). In the example in Figure 40, the rise-time setting (inspiratory rise-time percent) was set at its lowest point (ie, most rapid valve-opening setting, which can be accomplished only by depressing the knob and turning it to the zero position, which the manufacturer

Fig. 39. Airway pressure (above) and flow (bottom) waveforms during 2 different rise time settings. The faster rise time (second breath) results in a higher peak flow and pressure-overshoot (arrow). In addition, the flow-termination criterion (percent of peak flow) is reached sooner because of the higher peak flow, and the breath terminates sooner. I = inspiratory. (Courtesy of Kenneth D Hargett RRT, The Methodist Hospital, Texas Medical Center, Houston, Texas)

Fig. 40. With these waveforms the rise-time (on the Servo 300A ventilator) during volume-controlled ventilation was set to its fastest position (shortest rise time), which causes pressure oscillations early in the pressure waveform and also on the pressure-volume loop. On the Servo 300 ventilator the rise time can be set in any mode. Accessing the fastest rise-time setting requires depressing the setting knob and turning it to the zero position, which the manufacturer does not recommend under normal conditions.
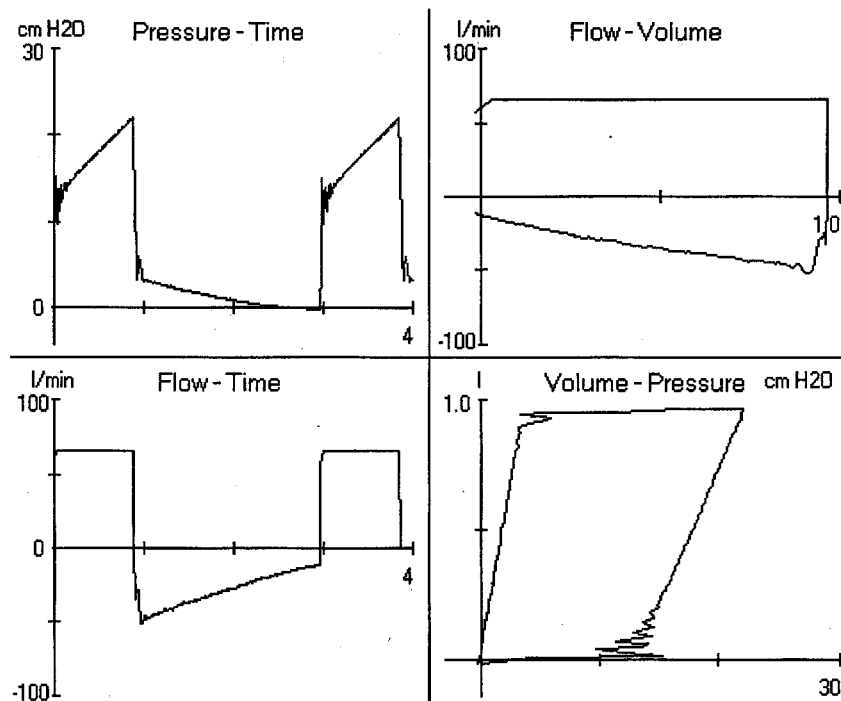
does not recommend under normal circumstances), which causes pressure oscillations early in the pressure waveform.

Though adjustment of the rise time is considered fine-tuning of the flow parameter, it is fairly easy to accomplish by observing the flow and pressure waveforms simultaneously and adjusting the rise time to maximize the appearance of the waveform (almost square appearance of the pressure waveform, no concavity, and no overshoot; see asterisk in Fig. 32). In comparison to volume-controlled ventilation, the ability to more nearly match the patient flow waveform during pressure-controlled ventilation, and the ventilator's ability to increase or decrease flow in response to patient demand are desirable traits that ultimately improve flow synchrony.

In general, the pressure-controlled modes (pressure support and pressure control) are better capable of meeting inspiratory demand than is volume-controlled ventilation.[44,45] In addition, dual-control modes, which combine the attributes of pressure limiting and volume control, *might* improve patient-ventilator synchrony.[11,46]

Termination Asynchrony (Phase 3)

The first 2 types of asynchrony (missed trigger attempts or trigger delay and flow asynchrony) are asso-

ciated with the inspiratory cycle. The remaining types of asynchrony are associated with expiratory events. These include premature or delayed breath termination, and problems associated with overlap of expiratory muscle activity into the inspiratory cycle and simultaneous contraction. While the latter are difficult to evaluate without neural recordings, evaluation of breath-termination is possible with the use of the standard pressure and flow waveforms, and it has also been documented with neural recordings.[47,48] Figure 41 illustrates the difference between premature termination (ventilator cycles off before the end of expiratory-muscle neural activity) and delayed termination (ventilator does not cycle off until after expiratory-muscle activity is initiated).

Delayed Termination

Parthasarathy et al studied delayed termination in a group of healthy subjects in whom airflow limitation was simulated with a Starling resister.[47] As seen in Figure 42, an increase in pressure support resulted in a greater number of missed trigger attempts. In evaluating the data, the nontriggered attempts were preceded by breaths with larger peak flow, higher V_T, and prolonged inspiratory time. Nontriggering attempts involved weaker inspiratory efforts
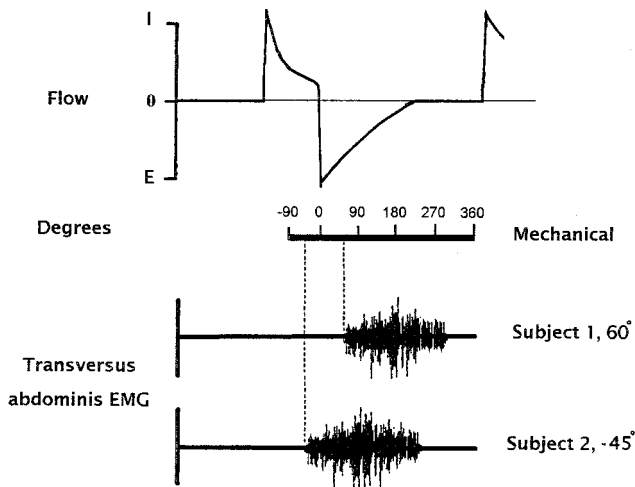
Fig. 41. The relationship of neural expiratory time to ventilator expiratory time was assessed by measuring the phase angle, expressed in degrees. If neural activity began simultaneously with the ventilator, the phase angle (0) was zero. Neural activity beginning after the offset (termination) of inflation by the ventilator resulted in a positive phase angle (60 degrees for subject 1). Neural activity beginning before the offset of inflation by the ventilator resulted in a negative phase angle (-45 degrees for subject 2). (From Reference 48, with permission.)
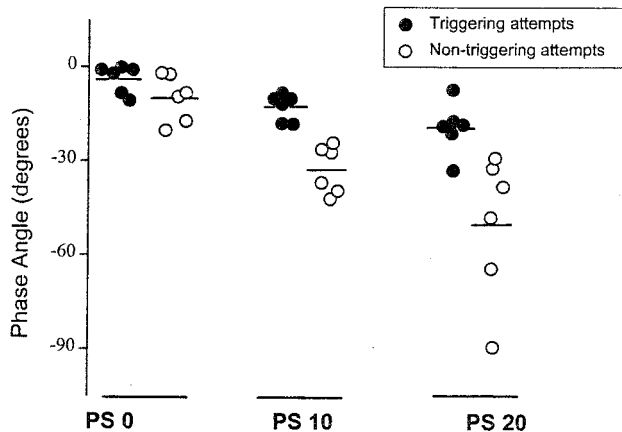

Fig. 42. Phase angle (see Fig. 41) between neural and mechanical expiratory times before triggering (closed circles) and nontriggering (open circles) inspiratory attempts. At pressure support (PS) of 10 cm H_2O and 20 cm H_2O, the phase angle before nontriggering attempts exceeded that before triggering attempts, indicating that neural expiratory time during late mechanical inflation preceded the onset of mechanical expiratory time and was longer before nontriggering attempts than before triggering attempts. (From Reference 48, with permission.)

(smaller pressure-time product) than did efforts that triggered the ventilator. The decreased inspiratory pressure-time product might have been caused by increased lung volume, which shortens the inspiratory muscles and thus decreases inspiratory pressure generation.[47] Increased lung volume also increases vagal afferent traffic, which via the

Fig. 43. Waveforms of flow, airway pressure (P_{aw}), and transversus abdominis electromyogram in a critically ill patient with chronic obstructive pulmonary disease receiving pressure support of 20 cm H_2O. Expiratory muscle activity (vertical dotted line) began when mechanical inflation was only partly completed. Note there is also a small airway pressure spike near the end of mechanical inflation, which coincides with the patient's neural expiratory activity. (From Reference 48, with permission.)

Hering-Breuer reflex, decreases respiratory motor output[49] and therefore decreases inspiratory pressure-time product.

In subjects in this study, "the continuation of mechanical inflation into neural expiration not only directly countered expiratory flow, it also decreased the time available for unopposed expiratory flow."[47] This leads to an increase in elastic recoil, which necessitates a greater inspiratory effort to trigger the ventilator.[50,51]

Similar results are seen in patients with COPD (Fig. 43).[47] Though the neural waveform verifies the presence of expiratory-muscle activity prior to the end of mechanical inflation, the consequences of the expiratory effort can be seen in the flow and airway pressure waveforms. The airway pressure waveform shows a small spike near the end of the inhalation. The flow waveform drops dramatically at the end of the breath, indicating pressure termination. Jubran et al[52] also noted that 5 of 12 patients with COPD appeared to recruit expiratory muscles during mechanical inflation. In that study they used P_{es} measurements (as opposed to direct expiratory-muscle electromyo-

Fig. 44. These waveforms illustrate a subject being ventilated with a pressure support of 5 cm H$_2$O. The subject's neural timing prece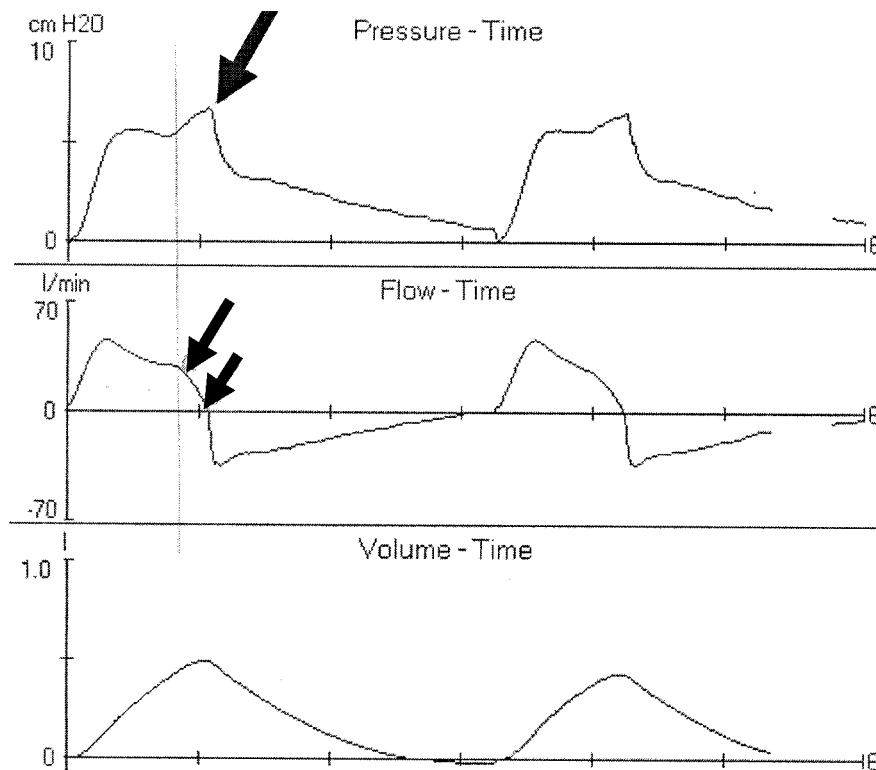des the end of mechanical inflation and results in a pressure spike (large arrow) on the airway pressure waveform. Also note the rapid decline in the inspiratory flow waveform near the end of the breath (small arrows) as a result of the subject's expiratory effort.

gram recordings) to evaluate expiratory-muscle activity. Parthasarathy et al[47] used electromyography to confirm similar observations in normal subjects.

Delayed termination generally results in dynamic hyperinflation, which causes trigger-delay and increases the number of missed trigger attempts. Though there may be differences between patient groups, most patients seem to have rather weak compensatory responses to acute delays in breath-termination[52,53] and generally do not make acute changes in their expiratory timing following a single inspiration.

Even in normal healthy subjects the effects of delayed termination can be assessed by carefully evaluating the pressure and flow waveforms. Figure 44 illustrates pressure and flow waveforms during pressure support ventilation of a healthy respiratory care student. Those waveforms demonstrate the same delayed breath-termination characteristics as in the COPD patient noted above. There is an obvious pressure spike near the end of the breath, which coincides with a rapid decline in flow, indicating the subject's attempt to exhale. A similar pattern is seen in the waveform in Figure 45, which was obtained during pressure-regulated volume-control ventilation (on a Servo 300A ventilator). Again, there is an obvious pressure spike at the same time as the zero-flow plateau in the flow waveform. The zero-flow plateau indicates achievement of

the target pressure and the end of mechanical inflation. In pressure-controlled modes, however, the inspiratory time is set by the clinician. In this case, subsequent reduction in inspiratory time removed the pressure spike and the zero-flow plateau. Figure 46 illustrates another case study involving delayed termination in the pressure-control mode. Inspiratory time is prolonged excessively, and the pressure waveform illustrates repeated expiratory attempts by the patient.

Data from diseased subjects, with a variety of disorders, indicate that inspiratory times are often in the range of 1 second or less.[54] Though there are clearly instances in which variations occur, the less-than-1-second guideline should be used as an approximate starting point for evaluating and setting inspiratory time.[47,54] During pressure-controlled ventilation the initial inspiratory time can be set with the less-than-1-second guideline, and then subsequent adjustments should be based on the time for the inspiratory flow waveform to decay to zero, but not beyond, and to prevent a pressure spike near the end of inspiration. During pressure-support ventilation the expiratory sensitivity can be adjusted within this range, while fine tuning can occur in 2 directions: if the breath is too long, there will be a pressure spike near the end of the breath; if the breath is too short, the patient may continue to inhale, resulting in
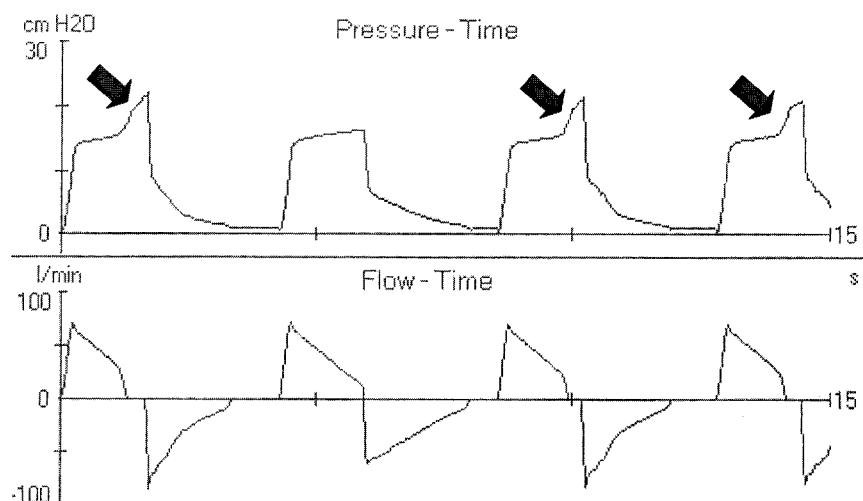
Fig. 45. Waveforms from a normal subject ventilated with pressure-regulated volume control on a Servo 300A ventilator. The subject's expiratory effort begins just prior to the end of the mechanical inspiratory time, which causes pressure spikes (arrows). Also note the small inspiratory zero flow plateau at the end of the inspiratory flow waveform, which indicates that flow into the lung has stopped just prior to mechanical expiration. In pressure-controlled modes the inspiratory time is set on the ventilator, and in this instance the ventilator inspiratory time setting needs to be reduced slightly.
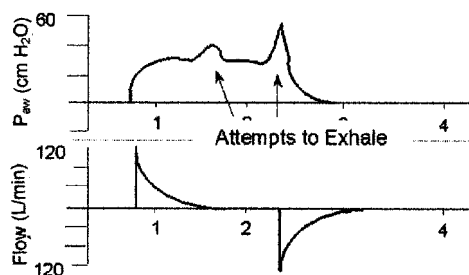

Fig. 46. Airway pressure and flow waveforms from a patient ventilated with a Puritan Bennett 7200 ventilator, in pressure-control mode. Note the multiple pressure spikes (arrows), which indicate expiratory efforts, and the appearance of the zero-flow plateau beginning at mid-inspiration. The initial inspiratory-time setting on the ventilator was far in excess of the patient's neural timing, and was subsequently reduced. P_{aw} = airway pressure. PCV = pressure control ventilation. (Courtesy of Kenneth D Hargett RRT, The Methodist Hospital, Texas Medical Center, Houston, Texas)

double-triggering (also known as a "stacked breath"; see the section below on early termination). During volume-controlled ventilation the less-than-1-second guideline can be used to determine the peak inspiratory flow necessary to achieve the desired V_T while keeping inspiratory time around 1 second. Subsequent fine-tuning should be based on interpretation of the pressure waveform.

Premature Termination

Premature breath-termination also has deleterious effects and causes asynchrony in patients with acute respiratory distress syndrome. Tokioka et al[55] evaluated the

effects of different inspiratory termination criteria (1%, 5%, 20%, 35%, and 45% of peak flow) during pressure support ventilation. In Figure 47 the waveforms in the upper 2 panels were obtained with the 5%-of-peak-flow termination criterion, and the waveforms in the lower 2 panels were obtained with the 35% (left) and the 45% (right) criteria, both of which cause earlier termination than the 5% criterion. Note several differences between the waveforms. In the flow waveform, earlier termination causes an abrupt initial reversal in the expiratory flow waveform (the expiratory flow slope has a rapid return to zero), indicating the continuation of patient effort. Evaluation of the airway pressure waveform also indicates an abrupt drop from the peak pressure toward baseline, again associated with the continuation of patient inspiratory effort (concave appearance rather than a normal gradual decay). Similarly, there is a continued drop in the esophageal pressure waveform, even after inspiratory flow termination, which indicates that muscular effort has not yet stopped. During the exaggerated condition (termination at 45% of peak flow) the continued patient inspiratory effort results in 1 incidence of double-triggering (the circled breaths in Fig. 47). Analysis of the waveform (Fig. 48) indicates that early termination substantially reduced V_T, increased respiratory rate, decreased inspiratory time, and increased WOB.

Expiratory Asynchrony (Phase 4)

We have discussed termination asynchrony as a separate consideration to emphasize the importance of performing a stepwise analysis of the breathing pattern. The
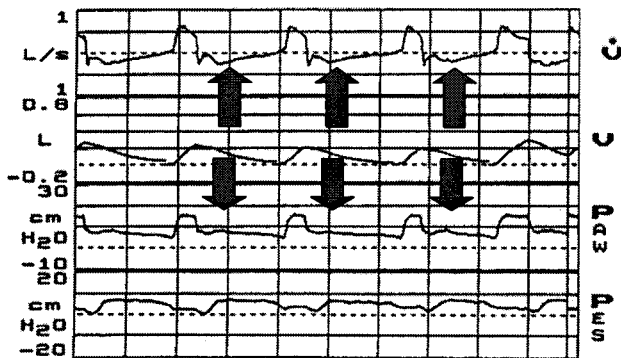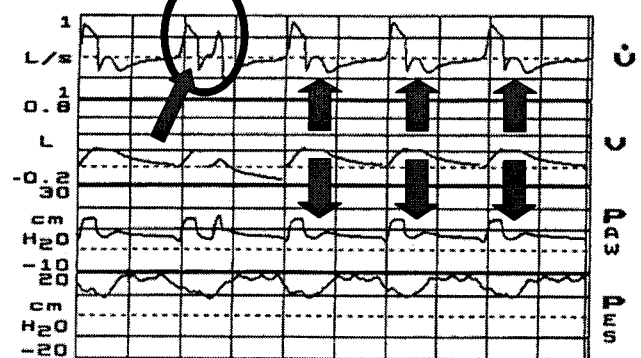
Fig. 47. Left Panel: Flow ($\dot{V}$), volume (V), airway pressure (P_{aw}), and esophageal pressure (P_{es}) waveforms with termination criteria (TC) of 5% and 35% of peak flow, during ventilation with pressure support of 10 cm H_2O. With TC 5% the breathing pattern was regular. Tidal volume was 390 mL and respiratory frequency was 17 breaths/min. The negative deflection of P_{es} during inspiration was minimal. With TC 35%, tidal volume decreased to 281 mL, and respiratory frequency increased to 23 breaths/min. The inspiratory flow terminated despite continuous negative deflection of P_{es}. Work of breathing increased from 0.20 J/L with TC 5% to 0.32 J/L with TC 35%. The arrows indicate continued patient inspiratory effort on the expiratory flow waveform (convex pattern) and the airway pressure waveform (concavity). Right panel: TC 5% versus TC 45% during ventilation with pressure support of 10 cm H_2O, with case 2. With TC 5%, inspiratory flow terminated simultaneously with the cessation of the patient's inspiratory effort, estimated by P_{es}. In contrast, premature termination with double-breathing (circled) occurred with TC 45%. Work of breathing also increased, from 0.42 J/L with TC 5% to 0.64 J/L with TC 45%. Note the larger P_{es} deflection during TC 45%. Also during TC 45% note the more pronounced changes (arrows) in the expiratory flow curve and the expiratory portion of the airway pressure waveform, caused by continued patient inspiratory effort despite early termination of the mandatory breath. (From Reference 55, with permission.)

remaining portion of the breath to consider is the expiratory time and the potential clinical consequences of shortened or prolonged expiratory time.

Shortened expiratory time has major clinical implications because of the risk of causing auto-PEEP. Prolonged expiratory time is of little consequence, unless the expiratory time is so long as to cause hypoventilation. Expiratory asynchrony can occur in conditions in which there is delay in the relaxation of the expiratory-muscle activity prior to the next mechanical inspiration[47] or overlap between expiratory and inspiratory muscle activity,[56–58] but those conditions are verified with neural measurements and are beyond the scope of our discussion about use of the standard ventilator waveforms to evaluate patient-ventilator synchrony.

Shortened expiratory time creates the potential for air trapping and auto-PEEP, which can cause trigger asynchrony because of the additional effort required to pull through the auto-PEEP to reach the trigger threshold.[21,47,59,60] Breaths preceding missed breaths typically have longer inspiratory times and shorter expiratory times, and are associated with auto-PEEP. In completing the breath analysis it is perhaps circuitous to note that the final consideration of auto-PEEP has as one of its main impacts the creation of trigger asynchrony, and that each of the following portions of the breath (inspiratory flow and termination) also have as a consequence of asynchrony the creation of auto-PEEP. In other words, flow patterns that increase inspiratory time (eg, lower peak flow during control ventilation, switch from constant-flow to descending-
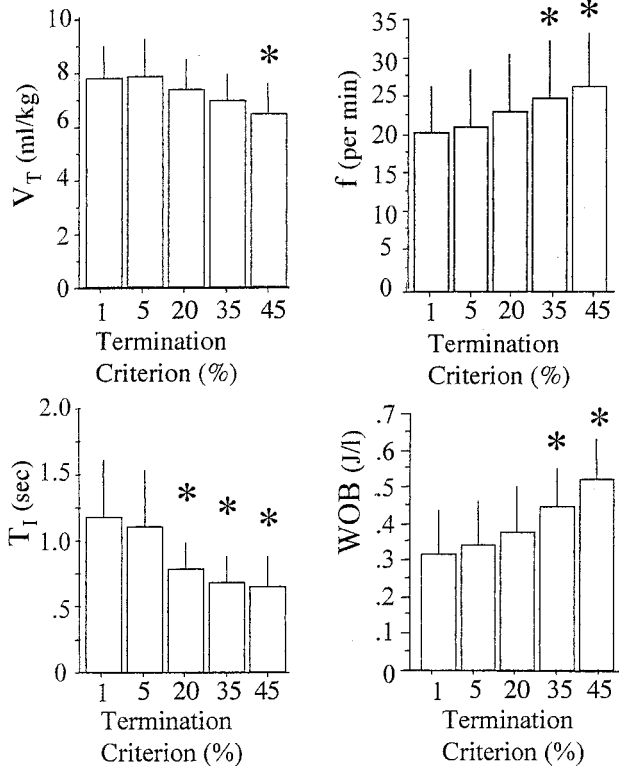
Fig. 48. Tidal volume (V_T), respiratory frequency (f), inspiratory time (T_I), and work of breathing (WOB) with 5 different termination criteria (TC) during pressure support ventilation of 8 patients. V_T was significantly lower with TC 45% than with TC 1%. With increasing TC, f steadily increased and T_I markedly decreased. WOB was significantly higher with TC 35% and TC 45% than with TC 1%. (From Reference 55, with permission.)

ramp flow, inadequate pressure slope during pressure-controlled ventilation, termination criteria that prolong expiratory time during pressure support or pressure-control) or decrease expiratory time can cause auto-PEEP. In addition, delayed flow termination offsets neural timing and reduces expiratory time, which can also create auto-PEEP.

Summary

Asynchrony is an important and common problem that can occur at several points in the breathing cycle. Conceptually, we have divided the breath into 4 phases to simplify the analysis of patient-ventilator asynchrony.

Trigger Asynchrony (Phase 1)

1. Trigger asynchrony is fairly easy to identify on the flow and pressure waveforms. 2. The flow waveform in particular should be evaluated to detect failed trigger attempts, and it should be correlated with patient observa-

tion. 3. Auto-PEEP is a common cause of failed attempts and should be routinely evaluated. 4. Auto-PEEP can be identified in the expiratory flow waveform.

Recommendations. 1. Including the trigger portion of the signal (figure-8) on the pressure-volume loop would help clinicians evaluate trigger effort. 2. Including a scrollable vertical line in the graphics package would help clinicians evaluate the correlation between pressure and flow waveforms and the synchrony of patient effort with initiation of ventilator flow. 3. Enhanced graphics capabilities should be available to allow clinicians to better analyze information (eg, automate calculations).

Flow Asynchrony (Phase 2)

Evaluating Flow Asynchrony During Volume-Controlled Ventilation. 1. Inadequate flow is evidenced by a scooped-out appearance on the pressure waveform, and in some instances by observation of additional flow in the flow waveform (eg, additional flow from the demand valve). 2. To evaluate flow adequacy, the clinician needs to be familiar with the normal, relaxed, pressure waveforms associated with the various flow options.[29] 3. Peak flow is frequently set too low, which prolongs inspiratory time and decreases expiratory time, causing auto-PEEP and asynchrony. 4. Both peak flow and the flow pattern affect flow asynchrony. 5. Careful evaluation of the pressure waveform may reveal over-distention, which should be confirmed with the pressure-volume loop.

Evaluating Rise Time. 1. Rise time can be evaluated by observing the pressure and flow waveforms. 2. Usually the pressure waveform should achieve near target pressure early in the inspiratory cycle. Short (fast) rise time may cause a pressure spike or pressure oscillations near the beginning of the pressure waveform. 3. The flow waveform should smoothly reach the peak flow and then appropriately decrease. 4. In general, faster rise time should reduce patient WOB.

Termination Asynchrony (Phase 3)

Delayed Termination. Delayed breath-termination is evidenced by a pressure spike at the end of the breath and rapid decrease in the flow, which can cause subsequent failed trigger attempts.

Premature Termination. Premature breath-termination can be identified on the pressure waveform, which will show a post-termination concavity (indicating continued patient effort) and on the flow waveform, which will show prolonged high flow during the breath, followed by rapid

deceleration and a convex pattern in the expiratory flow waveform, which indicates continued patient effort.

Expiratory Asynchrony (Phase 4)

The major consideration during the expiratory phase is the presence or creation of auto-PEEP, which can be caused by insufficient expiratory time or by asynchrony created during the flow or termination phases. 1. Routinely check for auto-PEEP by inspecting the end of the expiratory flow waveform and by measuring auto-PEEP (most commonly performed at the bedside with the expiratory-hold technique). 2. If auto-PEEP is identified, adjust, if possible, trigger sensitivity, peak flow, flow pattern, pressure slope, inspiratory time, breath-termination criteria, expiratory time, or respiratory rate, depending on the ventilation mode. 3. Trigger problems associated with auto-PEEP may improve with applied PEEP, approximately equal to the auto-PEEP.[61]

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Discussion

Sanborn: I'm somewhat confused about the concept of breathing through the demand valve. In the past there was an actual demand valve—a scuba-type valve. In today's ventilators there is no equivalent of that. The valve either triggers or it doesn't trigger, and in most of the ventilators I know of, there is no way you can breathe *through* that valve.

Nilsestuen: You are absolutely right. Modern ventilators have reduced the patient's ability to do that. The sample waveform that this refers to is from the Bear 1000. In this ventilator there was a backup demand valve that allowed the patient to breathe through the circuit in case the machine shut down.

Benditt: This harkens back to Dean Hess's "eyeball test." If the patient seems to be synchronous with the ventilator, if you look at the flow waveform, will you pick up abnormalities that you would not suspect with the eyeball test?

Nilsestuen: If you carefully evaluate the flow waveform? Is that what you're asking? Or does the eyeball test mean observing the patient to determine if he or she is synchronous with the ventilator?

Benditt: No. Say I go to the bedside and I look at the patient, and the patient looks relaxed, isn't using accessory muscles, the abdominal muscles aren't contracting, and everything looks good to me as a clinician. Then I turn on the flow waveform. Will I see these things popping out at me that I didn't suspect?

Nilsestuen: My take on that is that sometimes, yes, you will see some things that you didn't suspect, because the patient is not yet exhibiting a strong enough apparent effort or obvious muscular activity that counters what the ventilator is doing. The subtleties start showing up in the waveforms before it's obvious at the bedside.

Hess: Regarding what Josh Benditt just brought up, I teach the respiratory therapists, fellows, residents—anyone who will listen to me—to look at the patient, listen to the ventilator, and understand the physiology. I teach them to look at the patient and listen to the ventilator, and if you see that the patient is making inspiratory efforts and you hear no response from the ventilator, there's auto-PEEP. Then look for chest recoil, and if you see that the thorax is still recoiling when the next breath starts, there's auto-PEEP—the same thing you'll see on the flow waveform. So first look at the patient and listen to the ventilator, then look at the graphics and do all these other assessments to try to understand the underlying physiology that is producing the clinical problem. So, personally, I like graphics a lot, but I still rely a lot on the eyeball test.

Benditt: I think the reason that's important, I would say, is because I don't routinely on rounds go to the bedside and say, "OK, as a part of my physical examination I'm going to look at the flow waveform to make sure that things are OK here." Personally, I would do as you say—just examine the patient, and if there's a problem then investigate it with these tools. But if it's the other way around, then I might have to change my practice and say that to maximize synchrony I should include the flow waveform as part of my routine rounds. Sounds like we've got a *little* difference of opinion as to whether that's part of the eyeball test.

Nilsestuen: My sense is that there are some very obvious things that you can see the patient do. But, for instance, if the neural timing is off just a little bit, and there's just a tiny little bump in the pressure waveform, you won't see that just by looking at the patient, because the patient will look fairly relaxed, whereas in fact he's just starting to recruit muscular activity right at the end. That's fine-tuning, definitely, but, I think looking at the graphics makes asynchrony very apparent, and then you can document that there is a subtlety there that may improve patient comfort.

Pierson:* I think this stuff is pretty hard. I'm hearing Dean say that the eyeball test is valuable, and that how the patient looks in general should be your overall guide as to how things are going. But I'm also hearing Jon saying that you can see things on the graphics displays that are early warnings of important things you haven't picked up by your observation. Now, I suspect that Jon would agree that there are things that one might see on the waveforms that are not clinically important. On the other hand, though, the eyeball test depends very much on whose eyeball it is. At the very beginning of his presentation Jon told us

about the experienced practitioners he has had in his classes who have come back for advanced training, and how confident and familiar they are with all of these things, but how poorly they do on actual examinations that reflect their understanding of what they do.

It's been my observation for a long time that clinicians—critical care nurses for example—are often very familiar and comfortable with all the hemodynamic data they deal with, but may have incomplete understanding of what those data represent physiologically and what they actually mean. Likewise, physicians who have been in practice for a while may have developed routines and familiarity and comfort and fluency with the data that they deal with every day, but may not interpret that data in a manner consistent with our current understanding of what it ought to mean. So I think this is a very complex issue that involves both the clinician as subjective observer—the eyeball test—but also knowledge of the technical aspects of what the waveforms are telling us. I think neither of them is 100% the right answer, and they *may* sometimes be in conflict with one another.

Hess: I am reminded, David, of a picture that you published in one of Martin Tobin's books,[1] which shows the respiratory therapist peering at the ventilator, and the nurse peering at the monitor, and the residents all looking at the laboratory results, and nobody's looking at the patient!

REFERENCE

1. Pierson DJ. Goals and indications for monitoring. In: Tobin MJ, editor. Principles and practice of intensive care monitoring. New York: McGraw-Hill; 1998:33–44. [Figure 2–2 on page 40]

Campbell: I agree with what you just said. I think it's very important. The patient's appearance may lead you to look at the graphics to diagnose and maybe fine-tune, but certainly with

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advanced settings (such as rise time and expiratory cycle sensitivity or termination criterion), there's no way you can set those appropriately just by your view of what the patient's doing. With lots of the graphs that Jon showed, if you didn't have the line to line it up. . . . So I would never think you could put your hand on the patient's chest or diaphragm and put your other thumb on the graphic and say, "Ah-ha, I think we're in synchrony." I don't think that's a possibility, and I think you really need those graphics to perfectly set it. Respiratory therapists ask what to do with the ventilator's expiratory sensitivity setting and I tell them to take the default, and if you see a pressure spike, adjust it. That's the problem; we don't have the equivalent of a PDR [Physicians Desk Reference] for the ventilator that would tell us, "if this, then that," and "this is the dose," and "this is how to do it."

Nilsestuen: Another example of something that is far better done using the graphics than with just the visual technique of looking at the patient is setting the correct inspiratory time during pressure-controlled ventilation. There's almost no way you can do that visually by just looking at the patient, but it's very easy to do if you understand the graphics and you just look at the flow waveform. Because then you can see the plateau if it's too long, you can see it chop off if it's terminating too soon. That's maybe one of the best examples where it is very clear that the graphics have a huge advantage.

Hess: Call me old-fashioned. I look at graphics a lot and I like graphics a lot, but I have many times set PEEP with patients who have auto-PEEP by simply looking at the patient, looking for failed triggering efforts, and just turning up the PEEP until every inspiratory effort triggers the ventilator.

Pierson: But that's *your* eyeball, Dean, and not necessarily any random eyeball.

Harris: This sort of reminds me of the physical examination versus, say, echocardiography for the heart. If you have a very experienced person who can understand what's going on or has a lot of experience looking at a patient, then the physical examination is very helpful. If you have somebody who is *not* that experienced, it might not be so helpful.

Jon, you described asynchrony as 2 pumps that are not working together, not in harmony, and I'm thinking that we have a lot of information about one of the pumps, the ventilator, but we don't have a lot of information about what's driving the *human* pump. Should we be using things that Jon was talking about? Should we be looking at diaphragm function? Should we be looking at neural time? Is it overkill, or would it be helpful to have that information to synchronize the 2 pumps?

Campbell: We're spending an awful lot of time talking about secondary settings, such as setting the inspiratory time during pressure control. Let's

not forget that the primary setting is the pressure control, and the setting of pressure control is going to affect the flow waveform, or whatever waveform you decide to look at. So what we really need is some tool that would tell us if we have the base setting correct before we take that next leap to the secondary settings. The volume-support slides that you showed were classic; they were showing all the missed triggers. Well, maybe they just didn't have a high enough volume set. Maybe it wasn't the cycle criterion that was the problem.

Benditt: I have a comment about Dr Pierson's question, "Whose eyeball is it?" If you think about it, it is much more common to teach the physical examination. Maybe it's easier to get people to understand what muscle effort is in a patient who is asynchronous with the ventilator than it is to evaluate waveforms, set up the line to look at the waveform overlap, and so forth. I think you need at least as much, and probably more, expertise to look at the waveforms as to look at the muscle function at the bedside. So, unless you are going to have an echocardiogram or have the therapist or some expert person do a daily analysis of these waveforms for you and then put it in a report and send it to you, I think it's probably better to teach them the physical examination.

Hess: Glad to know I'm not the only old curmudgeon in the room.