15 Monitoring Interactions Between Spontaneous Respiration and Mechanical Inflations

Mark Heulitt and Steven M. Donn

Education Aims

- Understand the relationship between the two controllers involved in patients breathing spontaneously on mechanical ventilation.
- Understand the phases of mechanical ventilation and the role they play in the interactions between the patient and the ventilator.
- Identify the different types of patient-ventilator asynchrony and understand their physiologic implications for the development of asynchrony.

15.1 Introduction

Pediatric and neonatal patients breathe spontaneously during mechanical ventilation. This involves the combination of two distinct controllers: the clinician-controlled mechanical pump (the ventilator) and the patient's own respiratory muscle pump. The interactions between these two controllers can best be described by examining Newton's equation of motion.

$$P_T = P_{mus} + P_{appl} = (V \times R_{rs}) + (V \times E_{rs})$$

The equation explains the interaction of between the patient's generated pressure, $P_{mus,}$ and the ventilator's generated pressure, P_{appl}. These pressures overcome the resistance (R_{rs}) and elastance (E_{rs}) of the respiratory system. In this equation, inertia is negligible, especially in pediatric patients. This interaction is complex and involves numerous feedback pathways. For example, respiratory muscles are affected by the force-length and force-velocity relationship causing a mechanical feedback to the patient's motor center and spinal nerves from receptors in the airway, chest wall, or respiratory muscles; this has been described as the reflex feedback (Kondili et al. 2003). This relationship between the muscle feedback and reflex feedback is not well studied in mechanically ventilated pediatric patients, especially neonates, where the immaturity of the receptors, controllers, and muscle response may impact these relationships. The variables that can potentially impact the patient-ventilator interaction are also complex and include patient and ventilator factors and the patient's feedback system. These interactions create a response loop that is affected by this interaction between ventilator (controllers of the ventilator breath, including trigger, gas delivery, and how the

M. Heulitt (✉)
Department of Pediatrics, Section of Pediatric Critical Care Medicine, College of Medicine, University of Arkansas for Medical Sciences, 800 Marshall Street, Little Rock, AR 72202, USA
e-mail: heulittmarkj@uams.edu

S.M. Donn, MD, FAAP
Division of Neonatal-Perinatal Medicine, Department of Pediatrics, University of Michigan Health System, F5790 C.S. Mott Children's Hospital, 1500 East Medical Center Drive, Ann Arbor, MI 48109-0254, USA
e-mail: smdonnmd@med.umich.edu

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breath is terminated) and patient factors (mechanics of the respiratory system and muscular response), which cause a volume change with time, affecting the patient's muscular response, which in turn is affected by the force-muscle and force-velocity relationship of the respiratory muscles. This volume-time profile influences the patient's feedback system (chemical, mechanical, reflex, and behavioral) that then determines the muscular response of the patient's to the ventilator breath. The final influence on these interactions involves the clinician and his choice of the trigger, mode of ventilation, and level of support. Our discussion on monitoring the interaction of spontaneous respiration and mechanical inflation will focus on the identification of asynchrony between the patient controller and the ventilator.

15.2 Response of the Ventilator to Patient Effort

Factors affecting the response of the ventilator to the patient can be subdivided into the ventilator factors affecting the initiation of the breath (trigger variable), how the breath is sustained (gas control variable), and how the breath is terminated (cycle off criterion). Patient-related factors include the mechanics of the patient's respiratory system and characteristics of the patient-generated muscular response or pressure generated by the patient's respiratory muscles or P_{mus}. As can be seen in Fig. 15.1, the pressure generated when a patient is spontaneously breathing while receiving positive pressure ventilation is the sum of the pressure generated by the patient's respiratory muscles (P_{mus}) and that generated by the ventilator ($P_{applied}$). The proportion of each of these pressures is dependent upon the patient's respiratory drive, mechanics, and muscular response, the ventilator's trigger characteristics, and the selected ventilator mode. The mode is dependent upon the control, phase, and conditional variables. Our discussion will first focus on patient and ventilator characteristics followed by a review of when there is inability of the patient and ventilator controllers to act synchronously.

15.2.1 Ventilator-Related Factors

The response of the ventilator to a patient's effort is influenced by ventilator variables including the trigger variable that initiates the breath, pressure delivery, and the cycle variable that terminates the breath.

15.2.2 Trigger Variable

The trigger variable is controlled by either a flow or pressure signal derived from the airway. Figure 15.2 illustrates a comparison of two trigger variables. For pressure triggering, the patient must decrease the pressure in the ventilator circuit, by an isometric contraction of the respiratory muscles, to a preset value to completely open the inspiratory valve and initiate a mechanical breath. In flow triggering, the patient must generate a change in flow, sensed between the ventilator's inspiratory and expiratory pneumotachographs, by an isotonic contraction of the respiratory muscles. It has been generally believed that there are distinct advantages of flow triggering (Carmack et al. 1995; Branson et al. 1994; Giuliani et al. 1995; Sasson et al. 1994; Heulitt et al. 2000, 2003; Sanders et al. 2001). However, current ventilators are microprocessor controlled, replacing mechanical responses to patient triggering seen in older generation ventilators. The result of the microprocessor controller is a faster response time with decreased trigger delay. This improvement may negate the advantages in adult patients with larger endotracheal tubes and mature respiratory muscles. This does not appear to be true in neonatal and pediatric patients. As can be seen in Fig. 15.2 (Sanders et al. 2001), flow triggering results in a faster response time and decreased effort necessary to trigger in a pediatric-sized animal model during pressure support ventilation. These differences are important because during triggering, the initial phase of patient effort reflects essentially patient work until the inspiratory valve opens completely and delivers gas to the proximal airway. This is illustrated in Fig. 15.3, where a tracing of pressure, flow, and

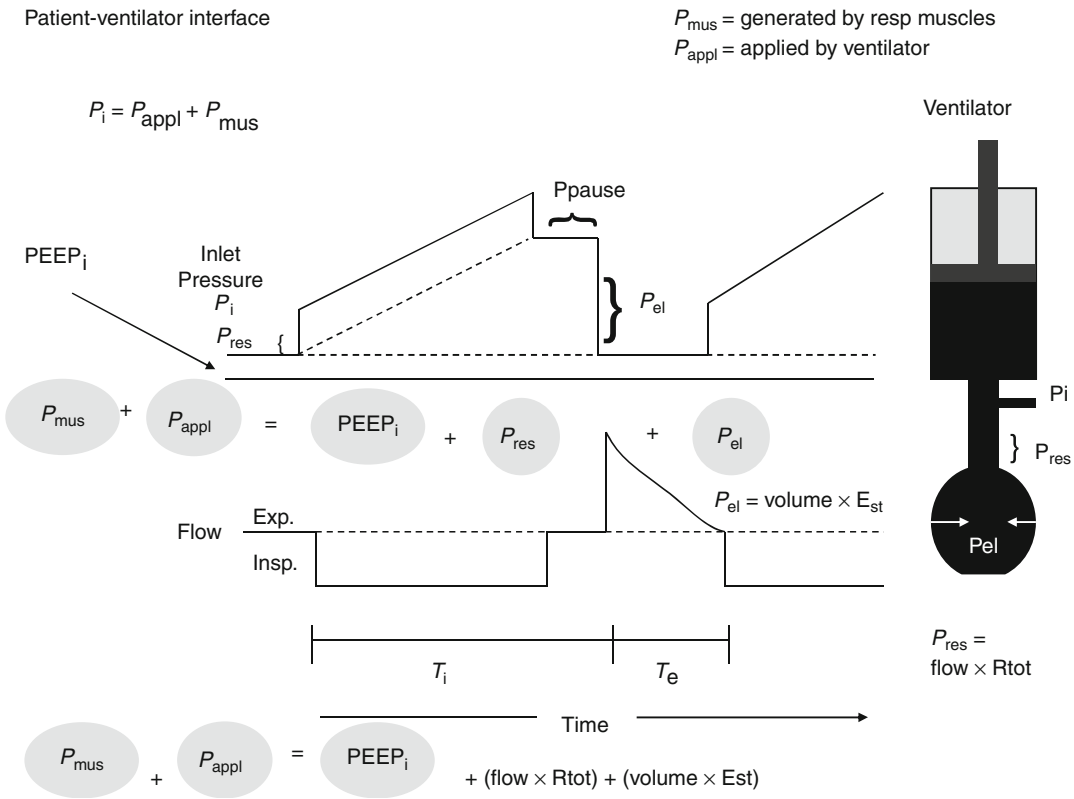

Fig. 15.1 The model on the right is of the respiratory system with the resistive elements represented by the straight tube and the elastic elements represented by the balloon connected to a ventilator represented as a piston. During inflation with a constant flow demonstrated in the lower waveform, there is a stepwise increase in the inlet pressure (P_i) that equals the loss of pressure across the resistive elements (P_{res}). Thereafter, P_i increases linearly and reflects the mechanical properties of the elastic elements (P_{el}). P_i is the sum of P_{res} and P_{el}. P_{res} is the product of the total resistive components and flow. P_{el} is the product of the volume delivered and elastance of the respiratory system. At the end of inspiration, when flow has ceased which reflects the pressure at P_{pause} (insp. pause), P_i decreases by an amount equal to P_{res}, P_i equals P_{el} during Insp (Modified from figure used with permission from Hubmayer et al. (1990))

volume is recorded at an animal's airway during pressure support. In this example, there is a waveform illustrating the opening and closing of the inspiratory valve with a tracing of the animal's muscular response, illustrated by an EMG tracing of the diaphragm. In a study of pediatric-sized lambs, WOB during flow triggering was reduced by 47 % during pressure support and 19 % in CPAP (Carmack et al. 1995). However, there have been no controlled studies in pediatric and neonatal patients to determine if these differences affect outcome measures such as length of ventilation.

It is important to note that despite the differences illustrated in the type of triggering, there are also differences related to the design characteristics of the ventilator. These differences relate to ventilator control algorithms that can affect trigger delay. Trigger delay is the time from the beginning of inspiratory muscle activity and the beginning of mechanical inflation (increase in pressure at the proximal airway). Increased trigger delay has been associated with the design characteristics of the pneumatics and electronics of ventilator system and correlated with respiratory drive (Leung et al. 1997) more time to trigger with less drive. This is especially important in small preterm infants, who have intrinsically short inspiratory times. For instance, if the inspiratory time is 0.2 s and the trigger delay is 100 ms, the patient will be halfway through the inspiratory phase before mechanical assistance is appreciated.

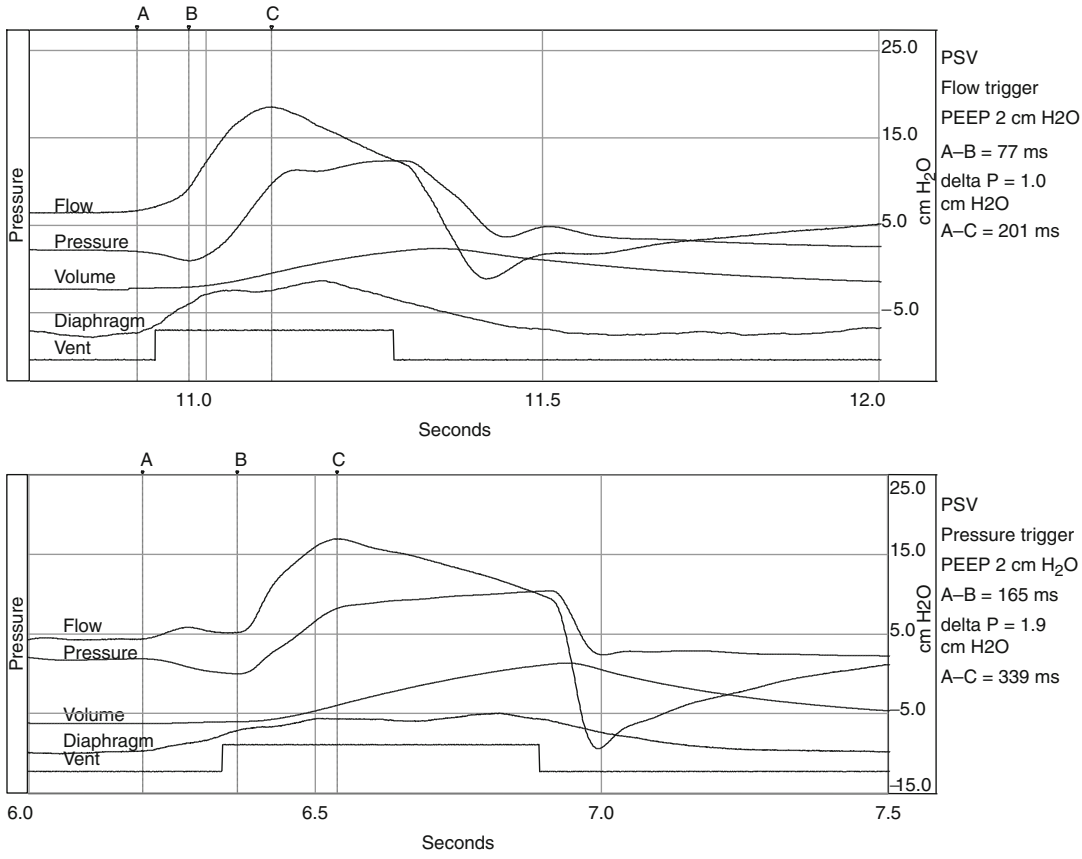

Fig. 15.2 Represents two waveforms. The *upper* waveform represents a single breath of a spontaneously breathing animal on pressure support being flow triggered. The *bottom* waveform represents a pressure-triggered breath. The negative pressure generated by the animal during triggering of the pressure-triggered breath is almost double of the pressure necessary to trigger the flow-triggered breath. Also there is evidence of increased trigger delay in the pressure-triggered breath. *A* initiation of breath, *B* most negative pressure, *C* peak flow

In a study between two microprocessor-controlled neonatal ventilators, it was found that there was a significant difference in trigger delay and work of breathing between these two ventilators (Heulitt et al. 2000) illustrating the role of ventilator design on both work of breathing and triggering. Once triggering occurs, there is pressurization of ventilator circuit and subsequently the patient.

15.2.3 Factors Affecting Pressure Delivery

Control variables include pressure, flow, and volume. Once the trigger variable is met and the inspiratory valve opens fully, there is pressurization of the ventilator circuit by the delivery of fresh gas flow. This pressurization of the system is illustrated in Fig. 15.4. The phase of this pressurization can be subdivided into the inspiratory positive pressure area or area 2 on Fig. 15.4 (Chatmongkolchart et al. 2001), which follows area 1, and is the amount of effort expended to activate the mechanical breath. Area 2 is defined by the start of the inspiratory pressure curve with the return of pressure to baseline and ending at the onset of expiration. Area 2 represents the ability of the ventilator to pressurize the system or the actual area of pressure versus time applied during inspiration. The variables that control the delivered pressure depend upon the

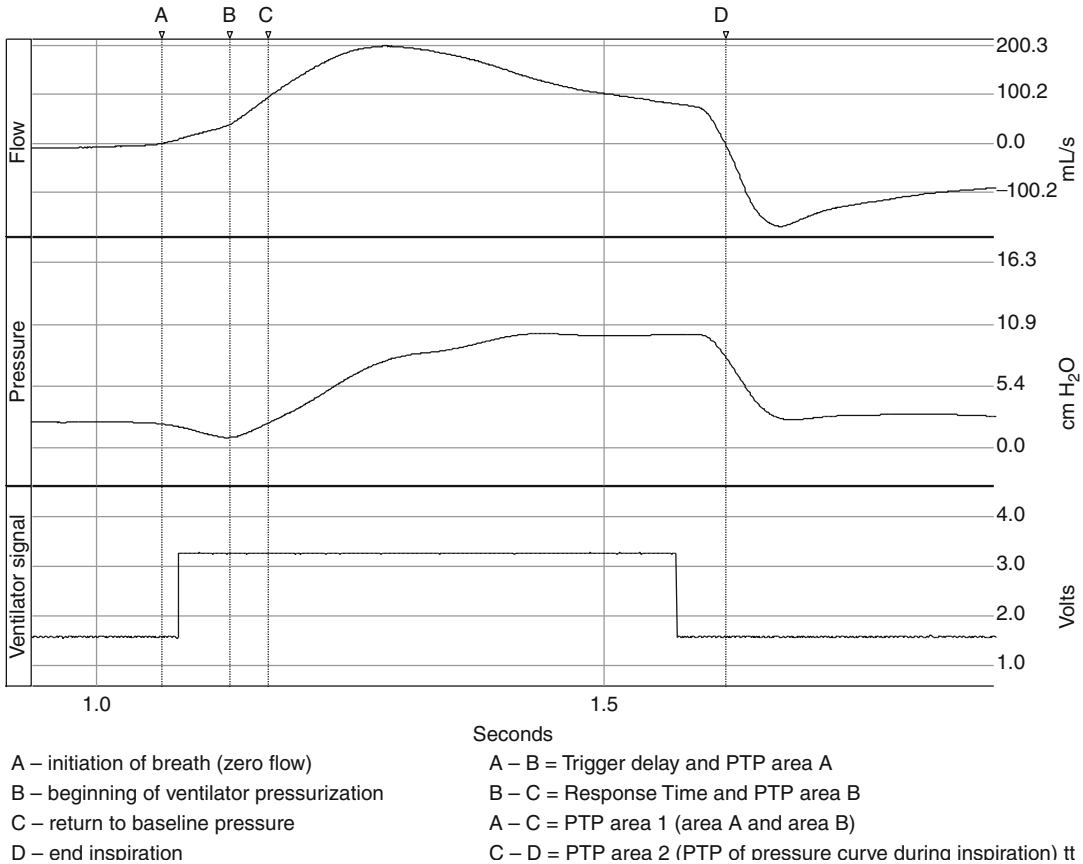

Fig. 15.3 Waveforms of flow, pressure, ventilator signal, and diaphragmatic (edi) signal are displayed. *A* is the begging of the edi signal, *B* the initiation of the breath where flow is a zero, *C* is the peak inspiratory flow, *D* the end of the edi signal. From point B to C, the line represents the slope of the inspiratory flow

mode of ventilation and the controller utilized in that mode. For example, in a mode with a preset tidal volume (e.g., volume assist-control), upon triggering the ventilator operates under a preset flow-time profile for the delivery of the tidal volume, and the ventilator determines the mechanical inflation time. In contrast, in a mode where there is a preset pressure (e.g., pressure support ventilation), the inflation time is influenced by both the patient and the ventilator. MacIntyre et al. (1990) demonstrated that if a rise in flow is not commensurate with the patient's demand during pressure support ventilation, there may be a too rapid rise in flow, or flow may be inadequate to meet the patient's effort. In either case patient-ventilator asynchrony can result. Current ventilators also have dual control capability. Some modes (e.g., PAV, PRVC, VS) offer a theoretical compensation for these limitations. Breaths are regulated by one variable to meet a target variable. For example, in PRVC, the clinician sets a target volume and then regulates the delivered pressure between each breath to reach that volume target. In contrast, in PAV the ventilator delivers pressure that is proportional and set by the clinician to instantaneous flow and volume and thus the patient's own P_{mus}. Thus, depending upon the mode, it may or may not reflect corresponding changes in patient's effort.

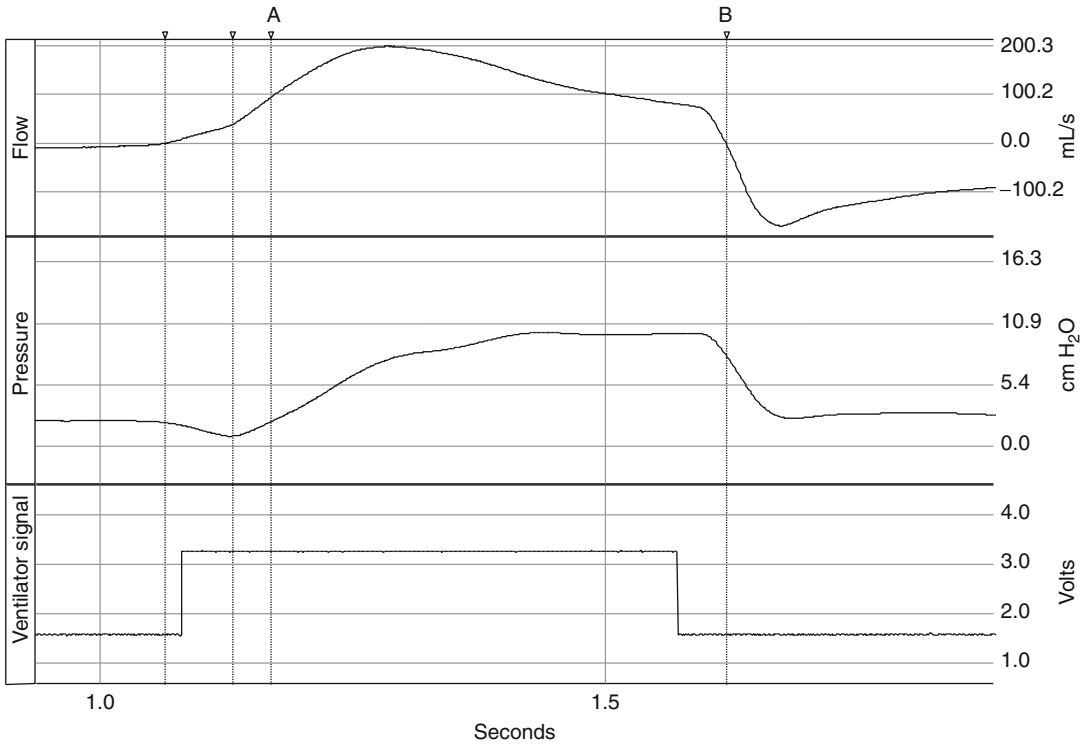

Fig. 15.4 The pressurization of the respiratory system is illustrated in the figure. The phase of this pressurization can be subdivided into the inspiratory positive pressure area or area 2 in the figure (Chatmongkolchart et al. 2001), which follows area 1, and is the amount of effort expended to activate the mechanical breath. Area 2 is defined by the start of the inspiratory pressure curve with the return of pressure to baseline and ending at the onset of expiration. Area 2 represents the ability of the ventilator to pressurize the system or the actual area of pressure versus time applied during inspiration

15.2.4 Cycle Off Variable

The cycle off variable is the variable that controls the end of inspiration. This can be a clinician-controlled variable. As can be seen in Fig. 15.5, the timing of this trigger signal may not correspond to the end of neural inspiration and the peak of diaphragmatic activity or contraction of inspiratory muscles after the close of the inspiratory valve. Thus, if flow stops either before or after the patient's own inspiratory flow, expiratory flow occurs before the end of inspiratory effort. In this situation, P_{mus} continues to increase even though inspiratory flow is zero (inspiratory valve closed) or is revered, and the muscle tension is applied to the elastic recoil of the respiratory system rather than obtaining further inspiratory flow. Thus, at the end of mechanical inspiration, P_{mus} continues to increase the muscle tension applied to overcome elastic recoil of the respiratory system causes a short mechanical inflation and low elastic recoil at end-inspiration and can promote re-triggering or ineffective triggering. The effects of asynchrony depend on the type of asynchrony present (Georgopoulos and Roussos 1996) and will be discussed further below. In the newborn, use of flow cycling is important in achieving expiratory synchrony because of the rapidity with which the respiratory time constant can change.

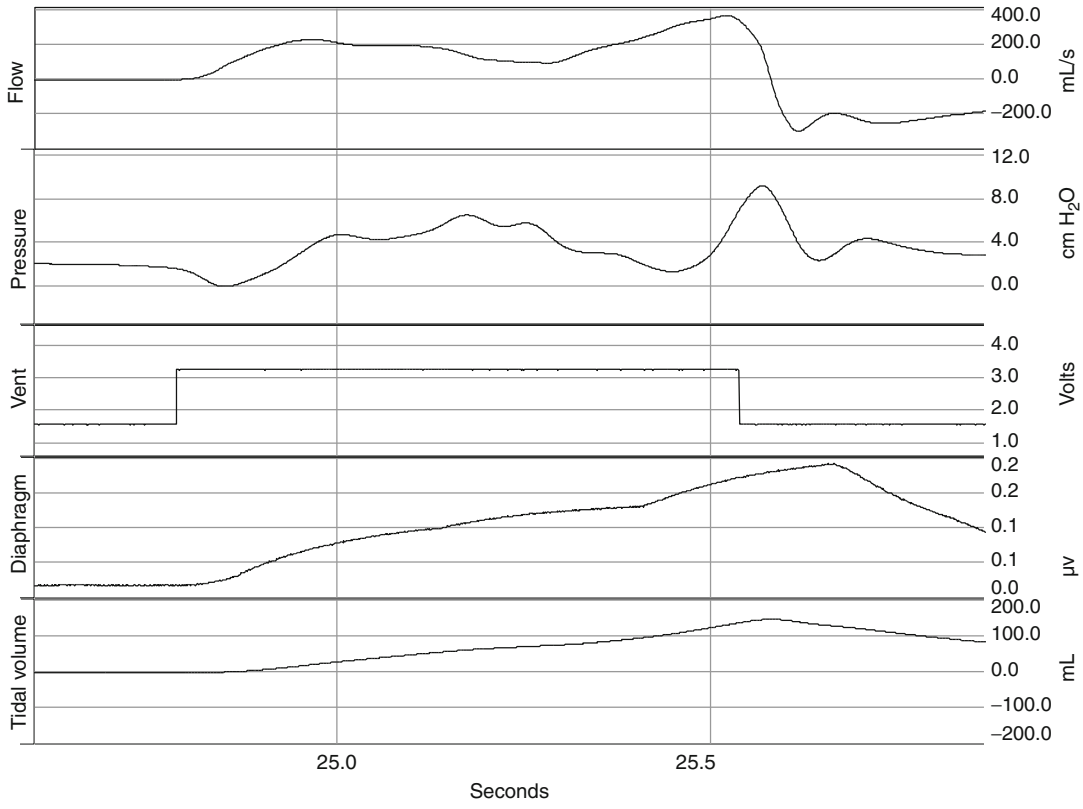

Fig. 15.5 Signals for flow, pressure, ventilator trigger, diaphragm EMG, and delivered tidal volume are displayed. The timing of this trigger signal may not correspond to the end of neural inspiration and the peak of diaphragmatic activity or contraction of inspiratory muscles after the close of the inspiratory valve. Thus, if flow stops either before or after the patient's own inspiratory flow, expiratory flow occurs before the end of inspiratory effort. In this situation, P_{mus} continues to increase even though inspiratory flow is zero (inspiratory valve closed) or is revered, and the muscle tension is applied to the elastic recoil of the respiratory system rather than obtaining further inspiratory flow. Thus, at the end of mechanical inspiration, P_{mus} continues to increase the muscle tension applied to overcome elastic recoil of the respiratory system which causes a short mechanical inflation and low elastic recoil at end-inspiration and can promote re-triggering or ineffective triggering

15.2.5 Patient-Related Factors

15.2.5.1 Mechanics of the Respiratory System and Characteristics of P_{mus} Waveform

Factors that affect flow because of the mechanical properties of the respiratory system and tubing can affect the pressure delivered by the ventilator (P_{aw}) independent of P_{mus} and may lead to asynchrony. This is usually seen when there is dynamic hyperinflation, where ineffective triggering, increased trigger delay, or prolonged inflation are common.

However, the pattern of the P_{mus} waveform can affect P_{aw} in several ways, depending upon factors related to both the patient and the ventilator. If the patient has decreased drive, P_{mus} increases slowly, and the time between the onset of the patient's inspiratory effort and ventilator triggering increases, causing trigger delay with subsequent asynchrony. In contrast if the patient's inspiratory effort is vigorous and longer than mechanical inflation time, double triggering can occur.

15.2.5.1.1 Response of the Patient Effort to the Ventilator-Delivered Breath

Normal reflex responses to changes in chemical, mechanical, or receptor stimulation may cause physiologic changes that may be difficult to interpret by the clinician. For example, it is important to understand the role that mechanical feedback plays in the patient's response to a mechanically delivered breath. The mechanical feedback is related to the delivered lung volume (length of muscular contraction) and flow (velocity of the contraction) delivered. Thus, when lung volume and flow are greater, P_{mus} will be less. The exact role of mechanical feedback is not well understood and may play only a small role in patient-ventilator interactions, but in a situation of high ventilator demands with hypercapnic hyperventilation, P_{mus} may underestimate neural output to respiratory muscles and can be reduced by up to 15 % (Georgopoulos and Roussos 1996).

Another feedback mechanism involves the response of the respiratory system to PaO_2, $PaCO_2$, and pH or chemical feedback. In normal subjects in both wakefulness and sleep, chemical feedback determines respiratory motor output. In mechanical ventilation, it is theorized that neuromuscular output is tightly linked to carbon dioxide tension and not to load reduction on the respiratory system (Georgopoulios 1997). Thus, during mechanical ventilation, chemical feedback remains an important determinant of P_{mus}. However, these effects may differ substantially between wakefulness and sleep or sedation during mechanical ventilation.

During mechanical ventilation in a subject who is conscious, the effects of $PaCO_2$ cause an increased P_{mus} (respiratory effort) with no change in respiratory rate. However, respiratory rate increases if $PaCO_2$ increases considerably. In contrast, when the drive to breathe from wakefulness is reduced during sleep or sedation, the dependence of the respiratory rhythm upon $PaCO_2$ is increased (Skatrud and Berssenbrugge 1983; Younes 1989). Thus, any increase in V_T may induce periodic breathing and apnea. In patients with lung diseases such as pneumonia or ARDS, other inputs to the respiratory controller may prevent chemical feedback from diminishing the tendency to increase neural inspiratory time and decrease neural expiratory time to a greater extent, resulting in a higher breathing frequency.

In addition to mechanical and chemical reflexes, other reflexes related to lung volume or flow changes – and mediated by receptors located in the respiratory tract – the lung and chest wall are important in controlling breathing (Shannon 1989; Younes 1981). These changes in volume and flow may elicit a P_{mus} response caused by other reflexes such as the Hering-Breuer reflex. The ultimate response is dependent upon the interplay of the magnitude and type of lung volume change, the level of consciousness, and the relative strength of the reflexes involved. In the premature infant, the chest wall is often more compliant than the lung, contributing to an increased work of breathing. An example of this interplay resulting in a misinterpretation by the clinician is demonstrated in a patient with decreasing levels of pressure support with the concomitant reduction in V_T and inspiratory flow. These changes cause a reflex feedback to increase neural inspiratory time and decrease neural expiratory time to a greater extent, resulting in an increase in breathing frequency. The resultant increase in respiratory rate may be interpreted by the clinician as intolerance by the patient to the attempt to wean ventilator support and thus delay further weaning by the clinician.

Finally, behavioral feedback is affected by changes in sedation, the sleep-wake state, and other aspects of the patient's environment and may play a role in the patient's response to ventilatory changes made by the clinician. For example, in an awake patient with increased airway resistance, ventilatory changes to compensate for hyperinflation may reduce inspiratory flow to values less than the spontaneous level causing perceived patient discomfort resulting in dyspnea with rapid shallow breathing and resultant patient-ventilatory asynchrony.

In conclusion, the interactions between the patient and the ventilator during assisted mechanical ventilation are complex. It may be difficult for the clinician to balance the clinician-controlled ventilator and the patient's own muscular or reflex response because of the

difficulty in interpreting ventilator changes and the patient's physiologic response. The resultant patient's response may either be a normal physiologic reflex or indicative of patient-ventilator asynchrony requiring further changes. Proper interpretation of patient-ventilator asynchrony requires accurate identification.

15.3 Patient-Ventilator Asynchrony

Patient-ventilator asynchrony is the failure of two controllers to act in harmony. The patient's work of breathing and effort are affected by the ventilator's ability to meet the patient's peak inspiratory demand (Marini et al. 1985, 1986). The factors that affect patient-ventilator asynchrony are listed in Table 15.1 and can be subdivided into equipment factors, patient factors, and decision-making (clinician) factors. The evaluation of patient-ventilator asynchrony can also be subdivided into four phases. These phases consist of triggering, flow delivery, breath termination, and the effects of $PEEP_i$. For each phase, waveforms will be reviewed to demonstrate how the clinician can detect patient-ventilator asynchrony.

Table 15.1 Factors that affect patient-ventilator asynchrony

Patient factors	Hering-Breuer reflexes
	Respiratory muscle
	Weakness
	Respiratory system mechanics
	Pathology
	Leaks
Ventilator factors	Ventilator algorithms and control
	Trigger signal
	Cycling off
	Rate and character of inspiratory flow
	Intrinsic PEEP
	Leaks
Decision-making factors	Mode
	Level of support
	Level of sedation
	Nutritional support
	Other treatments

Asynchrony results in ineffective gas exchange and has been associated with gas trapping, thoracic air leaks, increased work of breathing, inconsistent tidal volume delivery, and even intraventricular hemorrhage in the preterm infant.

15.3.1 Trigger Asynchrony

Trigger asynchrony is defined as the presence of muscular effort without effective ventilator triggering. The incidence and occurrence of patient-ventilator asynchrony is not well studied in pediatric patients. Clinical studies in adults have demonstrated trigger asynchrony in all common ventilator modes. In studies by Jubran et al. (1995) and Parthasarathy et al. (1988), cycle dyssynchrony during the PS mode occurs because of activation of abdominal musculature during the inspiratory phase, increasing patient effort and the number of failed trigger efforts.

In a recent study by Heulitt et al. (2009) of mechanically ventilated pediatric-sized animals receiving PSV, it was found that trigger asynchrony in healthy animals was evident in 13 % of breaths. After creating lung injury by saline lavage followed by lung recruitment with pulmonary compliance recovered to 60–70 % of baseline, evidence of trigger asynchrony increased to 60–70 %. Also there was evidence of increased trigger delay and increased work of breathing measured as pressure time product (PTP) in the pneumatically triggered breaths as compared to neurally triggered breaths. The most common form of trigger asynchrony is trigger delay. Trigger delay is important in infants and children because of their increased respiratory rate. For example if an infant is breathing 40 times per minute, their respiratory cycle is 1500 ms's. If there is a trigger delay of 200 ms then this represents 13 % of their respiratory cycle where they are exerting an effort without a response.

The reduction in trigger delay is important when it is considered in regard to timing of actual triggering. These results demonstrate an advantage in neurally triggered breaths. In both healthy and recruited lungs, we found it took less time (ms) from the initiation of the breath to the beginning of the ventilator trigger and that the percent

of PIF at the beginning of the ventilator signal was less for neurally triggered breaths. In the recruited animals, this could also be explained by a higher respiratory drive exemplified by a difference in P0.1. These findings are consistent with a faster response to effort.

The actual differences between trigger delay between the healthy and recruited animals are interesting. The triggered delay in the recruited animals is less than in the healthy animals in neurally triggered NAVA breaths. Since P0.1 and delta time from initiation of breath to opening of the ventilator valve are less in the neurally triggered breaths, this may represent a difference in the flow control algorithm allowing for a more rapid response to the animals' attempt to receive flow from the ventilator system. However, the peak inspiratory flow is less in the neurally triggered breaths which is consistent with the finding of a higher rate of flow asynchrony in these breaths.

Trigger delay is a delay in the time from the beginning of inspiratory muscle activity to the beginning of mechanical inflation. Causes of trigger delay are listed in Table 15.2. Trigger delay and ineffective effort can be easily detected by recording esophageal pressure or monitoring diaphragmatic activity with its EMG signal. Inserting an esophageal catheter is, however, a relatively invasive procedure, but there are commercially available catheters that combine EMG sensors with a nasogastric tube to justify its placement. Unfortunately, only one ventilator manufacturer has the ability to monitor the EMG signal. The EMG catheter may be superior to an esophageal balloon because it more closely reflects neural events (Parthasarathy et al. 2000; Sinderby et al. 1997). An alternative to an invasive catheter may be found by inspecting the flow waveform. Identifying the abrupt decrease in expiratory flow from the flow trajectory established earlier indicates either the beginning of inspiratory muscle contraction or relaxation of expiratory muscles during active expiration (Fig. 15.5). In either case, the point of expiratory flow deviation signifies the beginning of the triggering phase. This can clearly be seen in Fig. 15.5, where the ventilator trigger signal and diaphragmatic EMG are included (these signals enhance the flow signal but are not required). The time lag between this point and the point at which P_{aw} starts to increase is the trigger delay. If there is no mechanical breath following an abrupt fall in expiratory flow, this can be classified as an ineffective triggering. However, the clinician must not confuse changes in the flow signal caused by cardiac oscillations with ineffective efforts.

Table 15.2 Trigger delay

Ventilator characteristics and settings	Type and setting of trigger Site of signal recording Valves Level of pressure assistance Ventilator modes
Patient characteristics	Dynamic hyperinflation Respiratory drive during trigger phase Upper airway resistance (during NIV)
Circuit characteristics and interfaces	Additional resistance (ET, ventilator circuit, airway sensor, HME)

It is important to note that trigger delay is not always caused by poor inspiratory effort. In adult patients, it has been found that effort is more than a third greater when the threshold for triggering the ventilator is not reached than when it is. Breaths that do not trigger the ventilator have higher V_T and shorter expiratory time (Leung et al. 1997).

Trigger asynchrony can cause breaths to be stacked. This is defined by when the delta time between the ventilator's trigger is one half of the mean inspiratory time of the patient. These breaths are classified as stacked breaths. Stacked breaths occur with or without expiratory flow between triggers. Figure 15.6 illustrates stacked breaths. Auto-triggering, which refers to the phenomenon of the ventilator being triggered in the absence of patient effort, may also occur. This phenomenon may be caused by improper setting of the trigger threshold or P_{aw} distortions caused by circuit leak, presence of water in the ventilator circuit, or patient cardiac oscillations. Auto-triggering occurs frequently in neonatal patients because uncuffed endotracheal tubes are used. In small premature infants, clinicians often set a low trigger threshold to avoid ineffective triggering, and thus even small leaks will result in auto-triggering. It may be difficult to distinguish auto-triggering from rapid

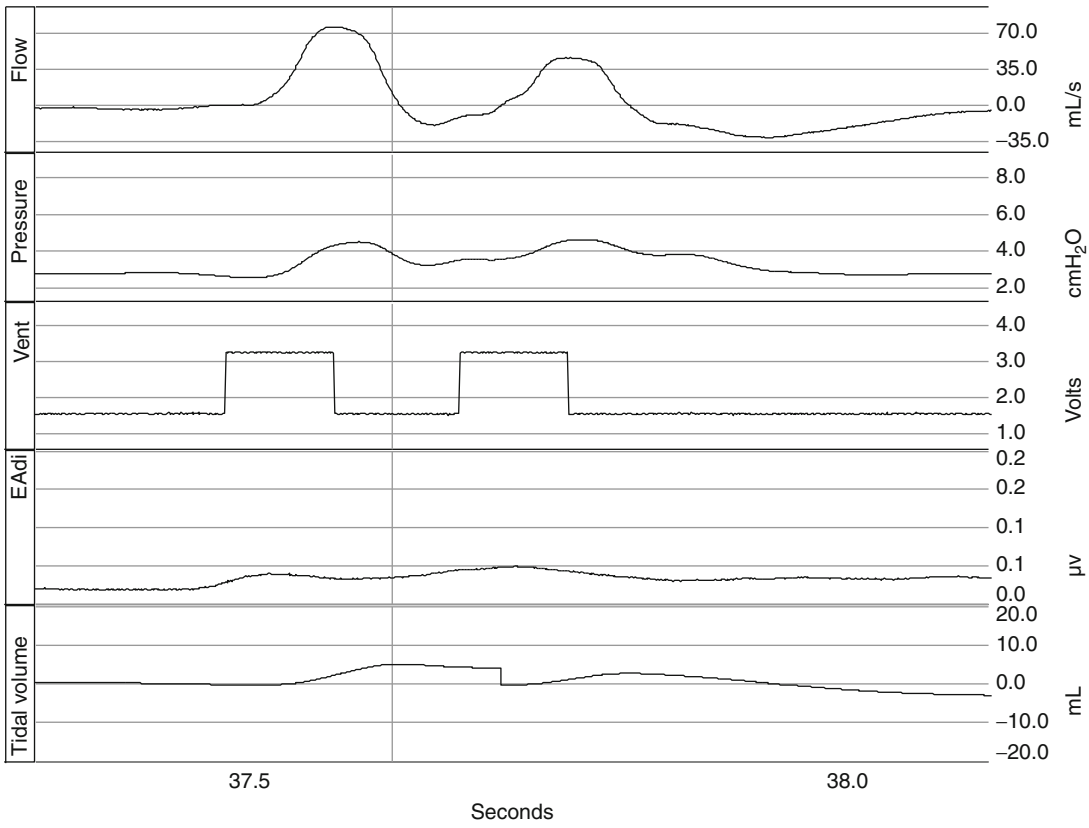

Fig. 15.6 Signals for flow, pressure, ventilator trigger, diaphragm EMG, and delivered tidal volume are displayed. This figure illustrates auto-triggering with evidence of ventilator response without evidence of patient effort in a spontaneous breathing, patient-triggered mode. Auto-triggering can also be detected by inspection of the pressure and flow waveforms by identifying the absence of the initial pressure drop below end-expiratory pressure which would be required in patient-triggered breath

breathing in these patients. Auto-triggering should be suspected when each breath looks identical and occurs at regular intervals. Rapid breathing will show some variability in rate and the appearance of the waveforms. Figure 15.7 illustrates auto-triggering with evidence of ventilator response without evidence of patient effort in a spontaneous breathing, patient-triggered mode. Auto-triggering can also be detected by inspection of the pressure and flow waveforms by identifying the absence of the initial pressure drop below end-expiratory pressure which would be required in patient-triggered breath.

Another issue that can influence triggering of the ventilator leading to ventilator asynchrony is the presence of increased airway resistance leading to the presence of inadvertent positive end-expiratory pressure, $PEEP_i$. Mechanically ventilated patients with obstructive lung disease who develop $PEEP_i$ have to generate a negative intrapleural pressure to match the value of $PEEP_i$ in addition to the ventilator sensitivity threshold level before triggering occurs and a ventilator breath is initiated. When inspiratory effort by the patient is less than the threshold value, the ventilator will not deliver a breath, causing effort without response from the ventilator. This is illustrated in Fig. 15.8, where there is clear evidence of muscular activity but no evidence of ventilator response. Therefore, dynamic hyperinflation (PEEPi) leads to frequent non-triggering of breaths in patients with obstructive lung disease. Such non-triggered breaths represent wasted effort on the part of the patient and lead to patient-ventilator asynchrony. In any spontaneous breathing mode, the ventilator must be set to

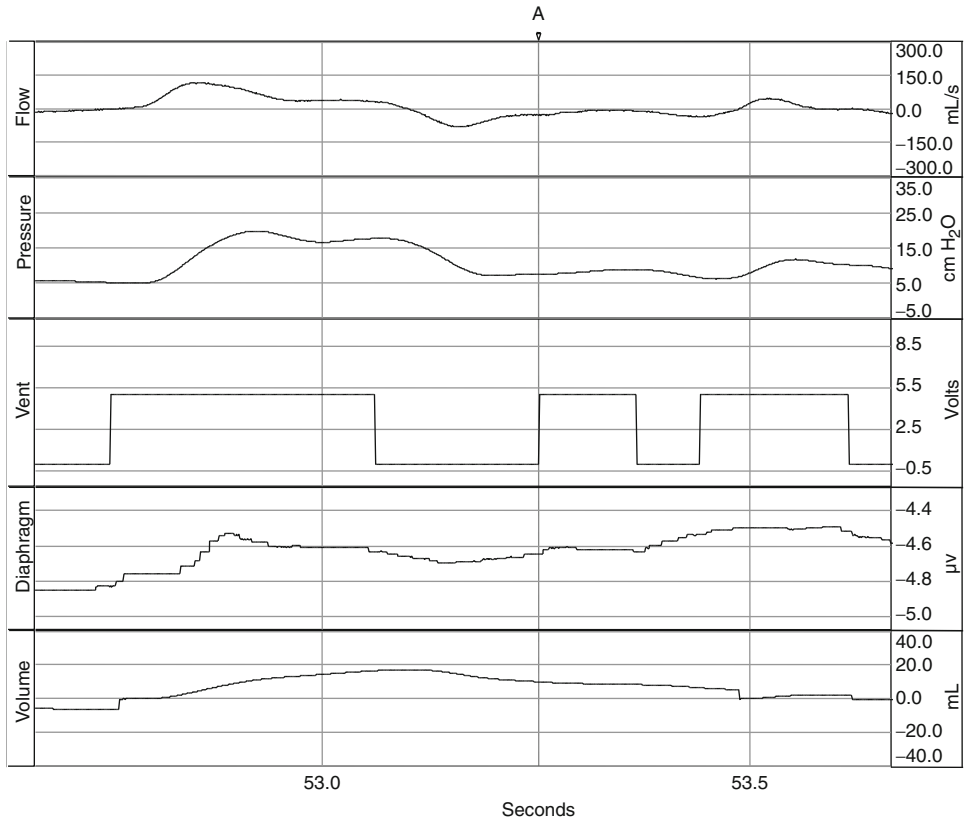

Fig. 15.7 Signals for flow, pressure, ventilator trigger, diaphragm EMG, and delivered tidal volume are displayed. There is clear evidence of muscular activity but no evidence of ventilator response. *A* indicates the point where flow crosses the zero flow line in a positive direction indicating effort to trigger. As indicated by A above, an effort was made to trigger the ventilator; however, there was no corresponding response. Mode is volume support ventilation

respond to the patient's breathing effort in order to provide adequate support. In addition, application of external PEEP could reduce the elastic threshold load and WOB, particularly in patients with flow limitations during tidal expiration.

15.3.2 Flow Asynchrony

Flow asynchrony occurs whenever the patient and the ventilator flows do not match. Flow from the ventilator can be a fixed or constant flow pattern (such as volume-controlled ventilation) or can be variable (PC, PS, or PRVC). In VC, flow is fixed so that a set level of flow is delivered with each breath. Because WOB is the sum of the work performed by the ventilator and the work performed by the patient, reduction in ventilator support or work will reduce the level of support. During ventilation with variable flow, the peak flow depends upon on the set target pressure, the patient's effort, and the respiratory system compliance and resistance. During PC, the clinician can set the target pressure and the rate of flow acceleration or rise time. Ideally, in pressure ventilation, the rise in gas flow should match the patient's demand for flow. The control of flow acceleration varies according to the manufacturer of the ventilator,

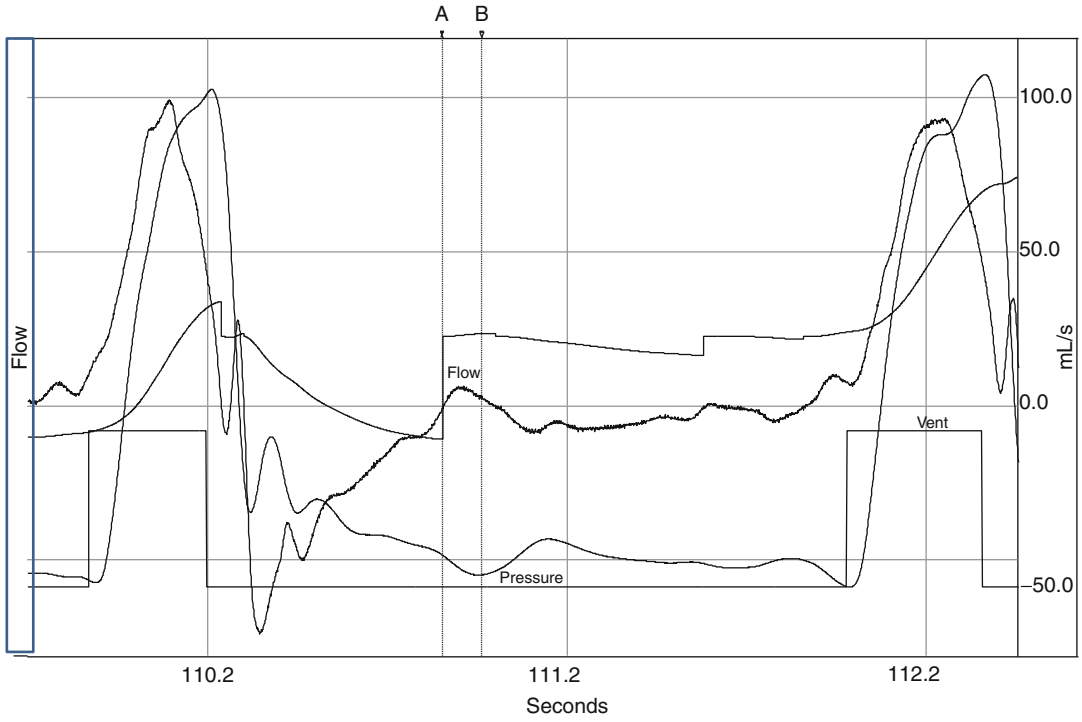

Fig. 15.8 On most pressure-volume loops, the pressure is plotted on the *x*-axis and volume and on the *y*-axis. Patient-triggered breaths will look different from time-triggered or machine-triggered breaths on the pressure-volume loops as the patient generates a negative pressure at the beginning of inspiration. *A* shows a patient-triggered breath and the resulting pressure-volume loop that traces the inspiration and exhalation. Pressure-volume loop. *B* represents a single breath with excessive triggering effort reflected by the classic "Figure 8" pattern

but the principles remain the same. Changing the rise time can have a profound effect on the flow-time waveform (Mancebo et al. 1995). If the rise time is set to the fastest setting (rise time 0), a sharp increase in inspiratory flow is dictated by the interaction between P_{mus}, P_{aw}, and elastic recoil at end-expiration (Bonmarchand et al. 1996). It has been reported that a very high pressurization rate has been associated with presence of a pressure overshoot causing a sense of dyspnea in the patient. A slower rise time may limit the ability of the ventilator to meet the patient's demands. Studies of flow asynchrony during PC or PSV have implied that many patients require a rapid rise time to match increased ventilatory demand. MacIntyre et al. (1997) assessed whether adjustments in the initial flow or breath termination criteria affect patient-ventilator synchrony. The ventilator pattern response to PSV in 33 adult patients was studied under conditions with two parameters: seven different levels of delivered initial PSV flow and during PSV termination at 50 and 25 % of peak flow. They found an optimal initial flow could be defined for a given PSV level, which resulted in the patient gaining a maximal pressure and volume from the ventilator. In addition, the initial PSV flows above and below this optimal flow were associated with faster breathing rates (or minute ventilation), shorter inspiratory times, smaller tidal volumes, and a tendency for airway pressure to meet the preset value. In pediatric patients who have smaller endotracheal tubes, increased flow may lead to increased turbulence and, possibly, increased asynchrony.

Figure 15.6 illustrates flow asynchrony, with the common finding seen as concavity in the

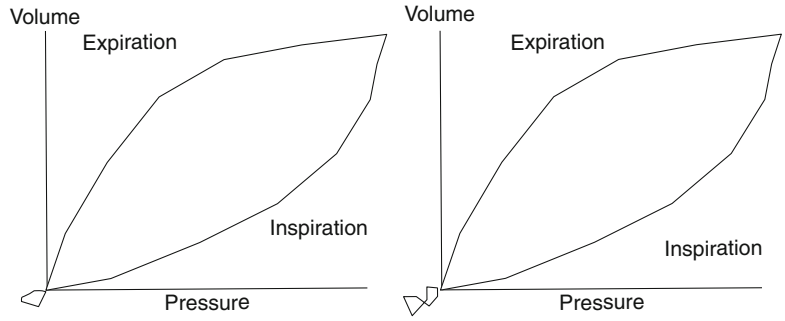

Fig. 15.9 Pressure volume curves in a spontaneous breathing subject. The portion of the curve extending beyond the origin of the figure represents the patient's work of breathing. The second figure demonstrates increased work of breathing because the area, thus effort is greater that the first figure with a "Fig. 15.8" configuration

pressure waveform illustrating inadequate flow to meet the patient's needs. Also this can be illustrated in a pressure-volume loop with evidence of the classic "Figure 8" due to increased triggering effort by the patient and turbulence (Fig. 15.9).

15.3.3 Termination Asynchrony

Termination asynchrony occurs when neural inspiratory time and ventilator inspiratory time do not coincide. The ability of the ventilator mode to terminate a breath when the patient desires a longer inspiratory time constitutes an important factor in reducing the incidence of dyssynchrony. For example, in pressure support inspiration is terminated by one of three mechanisms. The primary method is a decrease in flow. The second is a rise in pressure above the target setting. The third is inspiratory time exceeding a specific maximum duration. Termination asynchrony can be caused by delayed termination or premature termination. Termination asynchrony is defined as an increase in the expiratory portion of the airway pressure waveform (e.g., >2 cm H_2O). This occurs when the peak of muscle activity occurs before the inspiration valve is closed. This is illustrated when there is a peak in PIP beyond the close of the inspiratory valve but after the peak of respiratory muscle activity exemplified by the EMG of the diaphragm or Edi signal. The most common type of termination asynchrony is delayed termination. Generally, delayed termination results in dynamic hyperinflation, with resultant trigger delay and increased missed trigger attempts. Premature termination can also have deleterious effects with resultant asynchrony. In a study by Tokioka et al. (2001), premature termination led to substantially reduced V_T, increased respiratory rate, decreased inspiratory time, and increased WOB. In addition, in a study by Yamada and Du (2000) that mathematically modeled the transition from inspiration to expiration and determined that the relationship of flow at the end of a patient's neural inspiratory time to peak inspiratory flow is related to two factors, the ratio of the respiratory time constant to the patient's neural inspiratory time and the ratio of the set PS level to the maximum inspiratory muscle pressure. Thus, with set inspiration termination criteria, a patient can end inspiration before or after the ventilator reaches it termination flow. This variability in inspiration termination criteria clearly increases the probability of patient-ventilator synchrony.

15.3.4 Expiratory Asynchrony

Expiratory asynchrony results from a shortened or prolonged expiratory time and the patient attempting effort during expiration when the ventilator is unresponsive. Shortened expiratory time creates the potential for hyperinflation secondary to air trapping with generation of $PEEP_i$. This can occur in ventilator systems with and without active expiratory systems. It is assumed that the incidence is decreased in an active expiratory system, where ventilator response to patient effort in expiration can occur.

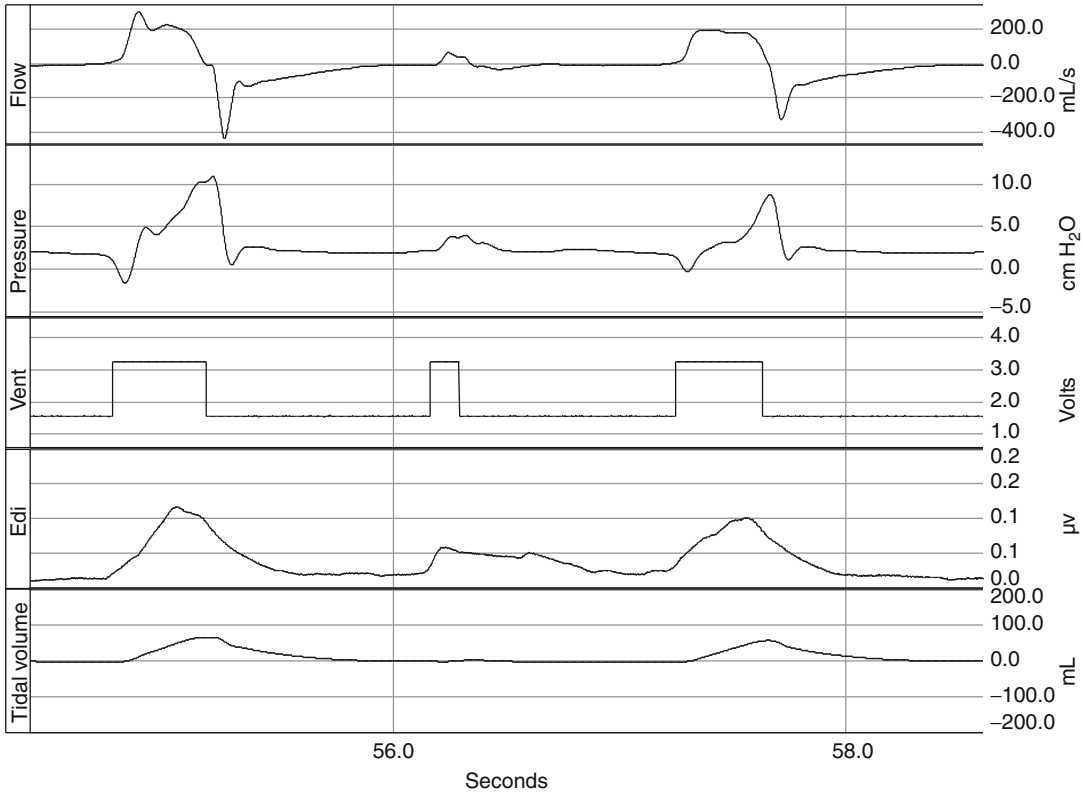

Fig. 15.10 This figure illustrates short cycle asynchrony. Short cycle is when inspiratory time is < ½ the mean inspiratory time for the breathe studied

15.3.5 Short and Prolonged Cycled Asynchrony

Further asynchrony can be defined as short cycled or prolong cycled. Short cycled is when inspiratory time is <1/2 the mean inspiratory time for breaths studied. This is illustrated in Fig. 15.10. Prolonged cycle is when the inspiratory time is >2 times the mean inspiratory time for breaths studied (Fig. 15.11).

Future Perspectives

Current clinical trends require a better understanding of the interactions between the spontaneously breathing patient and the ventilator because of the recommendations for patients to be maintained at a higher level of wakefulness during mechanical ventilation. Research in the future needs to define the advantages and disadvantages of this practice and further investigate the effects of sleep in these patients. Future advances in mechanical ventilation may require a closed-loop system that would allow the patient and ventilator to interact independently of the clinician to allow for better patient-ventilator synchrony.

Essentials to Remember

- Newton's equation of motion explains the interaction between the patient's generated pressure represented by P_{mus} and the ventilator's generated pressure P_{appl} and represents the pressures necessary to overcome t\ system.
- Factors affecting the response of the ventilator to the patient can be subdivided

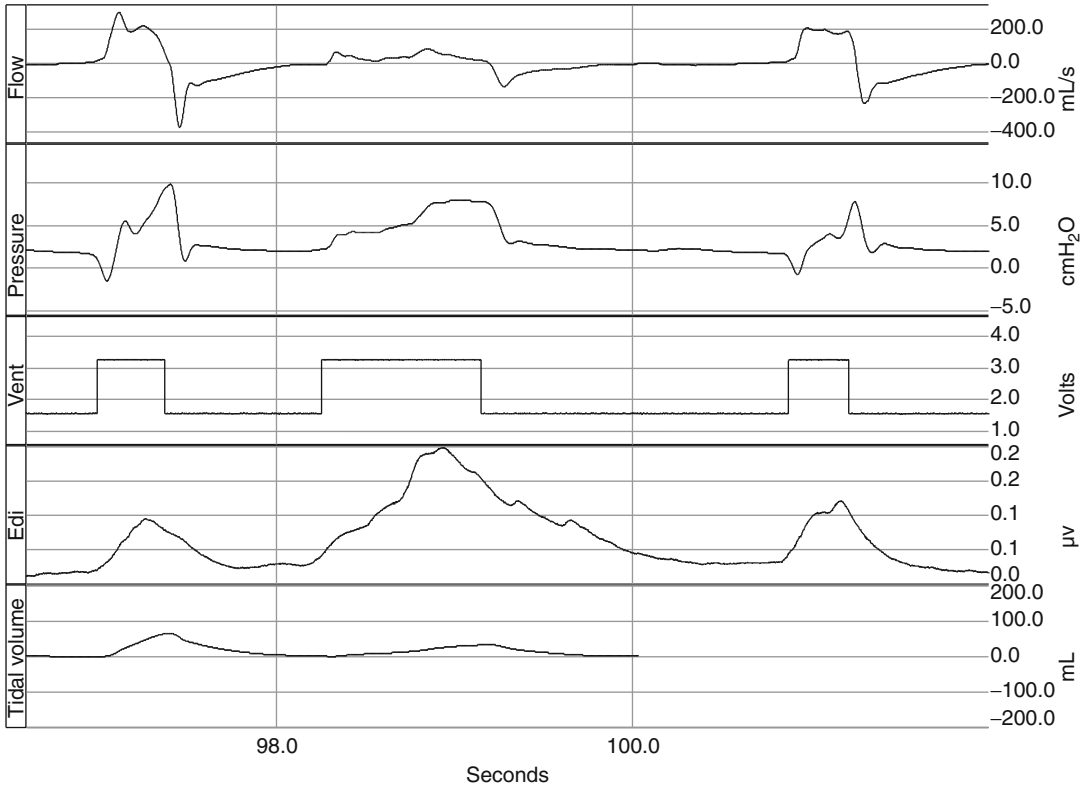

Fig. 15.11 This figure illustrates prolonged cycle asynchrony. Prolonged cycle asynchrony is when the inspiratory time is > 2 times the mean respiratory time for the breaths studied

into the ventilator factors affecting the initiation of the breath (trigger variable), how the breath is sustained (gas control variable), and how the breath is terminated (cycle off criterion).

- Patient-related factors include the mechanics of the patient's respiratory system and characteristics of the patient-generated muscular response or pressure generated by the patient's respiratory muscles or P_{mus}.
- Patient-ventilator asynchrony is the failure of two controllers to act in harmony.
- The factors that affect patient-ventilator asynchrony can be subdivided into equipment factors, patient factors, and decision-making factors.

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