Chapter 7
Mechanical Ventilation Strategies

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When a decision is made to initiate invasive mechanical ventilation, the clinician is required to determine the components of the patient's respiratory system that are failing and require to be supported. It is important to recognize that mechanical ventilation does not offer cure to the underlying disease process. The goal is to buy enough time until the dysfunctional tissues recover either on their own or through pharmacologic means. The objective of mechanical ventilation is to maintain sufficient oxygenation and ventilation to ensure tissue viability and to minimize the inevitable complications of the treatment itself.

7.1 Pathophysiologic Considerations

When instituting mechanical ventilation, it is important to recognize that "one size does not fit all". The underlying pathophysiologic derangements are remarkably different in an individual patient and indeed they may change from time to time in

The original version of this chapter was revised with correct missing text at Figure 7.7. The correction to this chapter can be found at https://doi.org/10.1007/978-3-030-83738-9_14

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A. P. Sarnaik et al. (eds.), *Mechanical Ventilation in Neonates and Children*,
https://doi.org/10.1007/978-3-030-83738-9_7

the same patient. The challenge to the clinician is to tailor the mechanical ventilation strategy to suit the patient's changing respiratory function in the least injurious fashion. To accomplish this, one should consider the respiratory support as a type of a pharmacologic agent. The "dose" of the support should be titrated to the patient's need for ventilation and oxygenation and utilize the appropriate strategy not to normalize the gas exchange, but to maintain it in a safe and adequate range suitable for recovery to occur. Two major targeted components are: a) alveolar ventilation to eliminate CO_2 and 2) arterial oxygenation to maintain sufficient O_2 delivery to the tissues.

7.1.1 Alveolar Ventilation (V_A)

Overall minute ventilation is represented as a product of tidal volume and the rate (V_T X Rate). Part of the tidal volume consists of dead space which is made up of conducting airways which do not contribute to the gas exchange. The amount of air contributing to gas exchange is alveolar ventilation, calculated as $V_A = (V_T - V_D)$ X Rate. This equation assumes, as a matter of convenience, gas movement is in the form of bulk flow which suggests that the first part of the tidal volume comprises the previously exhaled gas occupying the conducting airway (V_D) and therefore useless as far as bringing the atmospheric gas into the alveoli (see Chap. 3, Fig. 3.2). The determinants of V_A, therefore, are tidal volume (V_T), dead space (V_D) and respiratory rate.

The alveolar ventilation equation can be rewritten as: $V_A = (V_T$ X Rate) - (V_D X Rate). One can surmise that the most efficient means of increasing V_A is to increase the V_T as opposed to the rate. Table 7.1 illustrates the effect of various combinations of V_T and respiratory rate on total minute ventilation and V_A. With a constant anatomic dead space, at multiple combinations of V_D and V_T that provide the same amount of overall minute ventilation, there will be greater V_A at a higher V_T and a lower rate. Both V_T and the rate have important limitations. At higher V_T, the risk of volutrauma is a concern, while at higher respiratory rate adequacy of inspiratory and expiratory times to deliver the prescribed V_T to alveoli may be compromised. Depending on the altered pathophysiology, the clinician must decide which combination is least injurious and most efficient in delivering the dose of V_A to attain a desired $PaCO_2$.

The concept of bulk flow model for V_A is not entirely accurate. This is because of frictional resistance and asymmetric velocities especially at higher respiratory rates and lower tidal volumes as in high frequency ventilation (see Chap.3, Fig. 3.2).

Table 7.1 Relationship of V_T, V_D and the Respiratory Rate on V_A

V_T (mL)	V_D (mL)	$V_T - V_D$ (mL)	Respiratory Rate/Min	Minute Ventilation (L/min)	V_A (L/Min)
200	100	100	30	6	3
250	100	150	24	6	3.6
300	100	200	20	6	4
350	100	250	17	≈ 6	4.25
400	100	300	15	6	4.5

Because of frictional resistance, gas molecules move with asymmetric velocities rather than in blocks. The molecules in the center move faster than those in the periphery. Thus, volume of gas moving in and out of alveoli is in fact higher than what can be predicted by pure bulk flow movement.

7.1.2 Time Constant

Delivery of gas occurs from a higher pressure to lower pressure. Gas flow continues as long as pressure gradient exists and it ceases after proximal pressure equilibrates with the distal end. Once proximal and distal pressures equilibrate, the flow ceases and no additional volume is delivered. The pressure equilibration does not occur instantly. It takes time. The amount of time taken for pressure to equilibrate from proximal end to distal end is directly proportional to compliance (opposite of elastance) and resistance (opposite of conductance). Pressure equilibration is faster when compliance is decreased as the alveoli oppose expansion to a greater extent and fill up quickly. On the other hand, the pressure equilibration time is longer when resistance is increased. The product of compliance and resistance (C X R) is referred to as time constant (TC) which is a reflection of the amount of time it takes for a certain percentage of the volume change. With a constant inflation pressure, 63%, 95% and 99% of the maximal tidal volume will be delivered in 1, 3 and 5 time constants, respectively. Similarly, during exhalation, 63%, 95% and 99% of the initial volume will be exhaled with 1, 3, and 5 time constants, respectively (see Chap. 2, Fig. 2.2). Normally, the expiratory time constant (TC_E) is greater than the inspiratory time constant (TC_I) since airways get narrower during exhalation resulting in increased airway resistance. With decreased compliance, TC is decreased and the TC_E is closer to TC_I. With increased resistance, both TC_I and TC_E are prolonged but TC_E is prolonged much more than TC_I. (see Chap. 2, Fig. 2.3).

Most pulmonary disorders requiring mechanical ventilatory support are of two types; a) decreased lung compliance (decreased time constant) and b) increased resistance (increased time constant). Disease states with decreased lung compliance (ARDS, pneumonia, pulmonary edema etc.) have quicker approximation of pressure for alveolar filling and emptying. However, lung disease is rarely homogenous. While the composite TC may be either decreased or increased, areas with increased and decreased TC may co-exist in the same patient and may indeed change in their respective contributions at different times. The clinician must determine which component is the dominant one. The effect of time on delivered (or emptied) volume in lungs with differing time constants is shown in Chapter 2, Fig. 2.4. Increasing the time during inspiration or exhalation will result in greater volume change in areas with increased TC (increased resistance) but will have little to no effect in changing lung volume in areas with short time constant (decreased compliance).

7.1.3 Functional Residual Capacity

While the atmospheric air is brought into the alveoli only during inspiration, gas exchange between the alveolar gas and pulmonary capillary blood is continuous. During inspiration alveolar PO_2 (PAO_2) rises as fresh atmospheric gas with higher PO_2 is brought in. During exhalation O_2 continues to be removed by deoxygenated pulmonary arterial blood. PAO_2 rises during inspiration and declines during exhalation. Alveolar PCO_2 ($PACO_2$) on the other hand, falls during inspiration as it is diluted by the atmospheric gas containing negligible amount of CO_2, and rises during exhalation as CO_2 is being added from the pulmonary circulation. The fluctuations in alveolar gas composition during inspiration and exhalation are buffered by functional residual capacity (FRC) which is the gas left in the lung at end-exhalation. The changes in PAO_2 and $PACO_2$ during the respiratory cycle are only a few torr (Fig. 7.1).

Pulmonary capillary circulation is in a state of equilibration with the FRC by the process of diffusion. Mean PAO_2 and $PACO_2$ are approximately 100 and 40 torr respectively. During inspiration PAO_2 rises to 102 torr and during exhalation it falls to 98 torr. $PACO_2$ likewise rises to 41 torr during exhalation and falls to 38 torr during inspiration. Under normal circumstances, the systemic venous blood brought to the alveoli by pulmonary capillaries will have a PO_2 of 40 torr and PCO_2 of

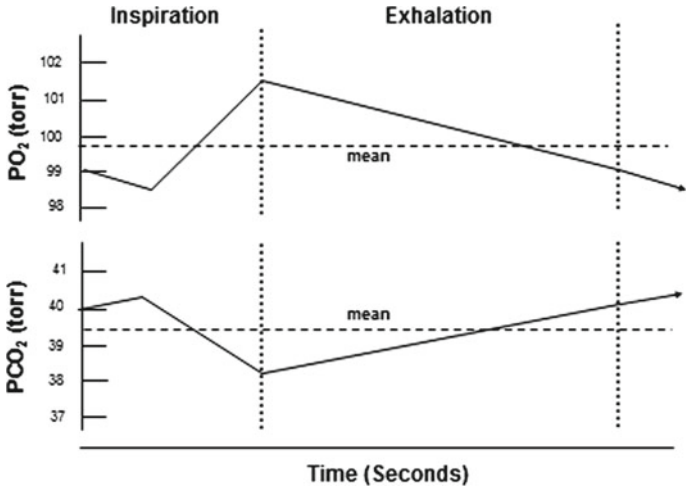

Fig. 7.1 Alveolar PO_2 and PCO_2 fluctuate throughout the respiratory cycle. During inspiration PAO_2 rises and $PACO_2$ declines as fresh atmospheric gas enters the alveoli. During exhalation the PAO_2 decreases and $PACO_2$ rises as O_2 continues to get removed and CO_2 is added by the pulmonary arterial circulation. Note that during the early part of the inspiration PAO_2 continues to decline as $PACO_2$ rises because of the entry of the dead space (previously exhaled gas). The degree of fluctuations in alveolar gas tensions is buffered by the FRC. *(Modified from Comroe JH:* Physiology of respiration, *ed 2, Chicago, 1974, Year Book Medical Publishers, p 12.)*

46 torr. FRC represents the environment available for pulmonary capillary blood for gas exchange at all times. As the pulmonary capillary blood flows across the alveoli, it gets "arterialized" to a PO_2 of 100 torr and PCO_2 of 40 torr. The major pathophysiologic effect of decreased FRC is hypoxemia. Reduction in FRC results in a sharp decline in PAO_2 during exhalation because limited volume is available for gas exchange. PO_2 of pulmonary capillary blood therefore falls excessively, approaching venous PO_2 during exhalation leading to decline in arterial PO_2. When FRC is reduced and the patient is breathing room air, any increase in PAO_2 during inspiration cannot compensate for decreased PAO_2 during exhalation due to the sigmoid shape of the O_2 dissociation curve. Since most of the O_2 in blood is combined with Hb, it is the percentage of oxyhemoglobin (SaO_2) that gets averaged rather than the PaO_2. The steep O_2 desaturation of Hb during exhalation results in overall arterial desaturation and hypoxemia. In situations where FRC is severely depleted, hypoxemia becomes resistant to administration of supplemental O_2 administration. Since CO_2 dissociation curve is relatively linear, decreased FRC does not significantly affect $PaCO_2$ as long as V_A is maintained. Two strategies can be employed during mechanical ventilation to ameliorate the hypoxemia secondary to decreased FRC. The first one is an "open lung" strategy in an attempt to increase FRC by application of positive end expiratory pressure (PEEP). The second strategy is to increase the inspiratory time (T_I) fraction of the respiratory cycle allowing for longer exposure of pulmonary capillary blood to higher O_2 and shorter exposure to lower O_2 during expiratory time (T_E). In order for the increased T_I to produce a favorable result, one must ensure that the decreased T_E is still sufficient to allow for adequate alveolar emptying. Such a strategy requires the expiratory time constant to be reduced such as in disease states with decreased compliance. Inverse ratio ventilation (IRV) and airway pressure release ventilation (APRV) are extreme examples of such a strategy.

7.1.4 Pressure–Volume (P–V) Relationship

Real time changes in volume as inflation pressure is applied is an important consideration during mechanical support of ventilation (Fig. 7.2). Collapsed or atelectatic alveoli require considerable amount of pressure to open. Once open, the alveoli require relatively less pressure for continued expansion. The process of opening collapsed alveoli is called lung recruitment and it is intended to increase the FRC. Opening the atelectatic alveoli and keeping them in an expanded state during tidal respiration is termed "open lung" strategy. Repetitive opening and closing the alveoli during tidal respiration, referred to as "tidal recruitment", is injurious to the lung and it is an important component of ventilator induced lung injury. The brunt of pressure applied to atelectatic alveoli is experienced at the delicate terminal airway-alveolar junctions causing atelectotrauma. Also, pressure delivery to maximally open alveoli cause them to over-distend and resulting in volutrauma. Most atelectatic lung diseases (e.g. ARDS) are heterogeneous in

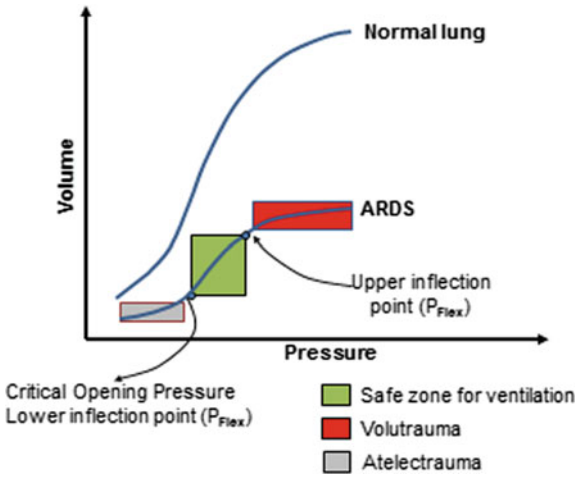

Fig. 7.2 Pressure–volume relationship in ARDS versus normal lung. Pulmonary compliance is reduced in ARDS with a narrow zone of "safe" ventilation (green rectangle) where the dynamic compliance is at its maximum. Ventilation at lung volume below the lower P_{Flex} (critical opening pressure) results in tidal recruitment and atelectrauma. Ventilation at lung volume above upper P_{Flex} results in volutrauma

nature. Each of the millions of alveoli has its own mechanical characteristics; however, a composite pressure–volume relationship for the entire lung is useful to conceptualize.

Compared to the normal lung, the ARDS lung is less compliant resulting in a PV curve with a decreased slope (Fig. 7.2). At the beginning of inspiration, the atelectatic alveoli are being forced open requiring a large change in pressure for a relatively small change in volume (lower horizontal part of PV curve). Once these alveoli are opened, further increase in volume requires a relatively smaller change in pressure (middle vertical part of PV curve). After the alveoli are maximally distended, their PV curve again resumes horizontal nature indicating opposition to further volume change with added pressure. The point at which the alveoli open up to accept greater volume change for increase in pressure is termed lower inflection point (lower P_{Flex}) and the point at which added pressure yields less volume change is called the (upper P_{Flex}). The goal of mechanical support of ventilation should be to keep PEEP above the lower inflection point and to keep the peak alveolar pressure (PIP with PCV and plateau pressure with VCV) below the upper inflection point, the so called "safe zone" of ventilation. If PEEP is below the lower inflection point, the alveoli whose critical closing pressure is above the level of PEEP are likely to collapse and reinflate during subsequent inspiration, a process termed "tidal recruitment" that is injurious to lung due to the stress experienced by terminal airway-alveolar junctions. If the peak alveolar pressure is higher than the upper inflection point, overdistension of alveoli is likely to occur resulting in volutrauma and barotrauma. The strategy to avoid inflation pressure that includes lower P_{Flex} is to leave the lungs at a constant state of recruitment with appropriate amount of PEEP. Inclusion of upper P_{Flex} during inflation is avoided by delivering relatively small amount of V_T.

7.2 Planning of Mechanical Ventilation in Individual Situations

The technologic details of ventilator functioning are described in Chap. 6. Following is a suggested approach for the clinician to consider at the bedside.

Pathophysiologic considerations When strategizing mechanical ventilation for an individual patient, certain physiologic parameters need to be assessed (Fig. 7.3). These are FRC, time constant and critical opening pressure. Since reliable measurements are not readily available for any of these variables, the clinician must make reasonable assumptions based on the type of the disease process, clinical findings, blood gas analyses, and imaging studies.

7.2.1 Phases of Respiratory Cycle

Four phases of the respiratory cycle should be taken into consideration when tailoring the strategy for a given situation (Fig. 7.4). These are (1) Initiation of inspiration and a variable that is controlled, often referred to as the mode; (2) Inspiratory phase characteristics, which determine the duration of inspiration and how the pressure or volume is delivered; (3) Termination of inspiration, often referred to as the cycle; and (4) Expiratory phase characteristics which mainly consists of application of PEEP. Decision should also be made regarding the extent and the nature of patient-machine interaction that should be allowed in an individual situation.

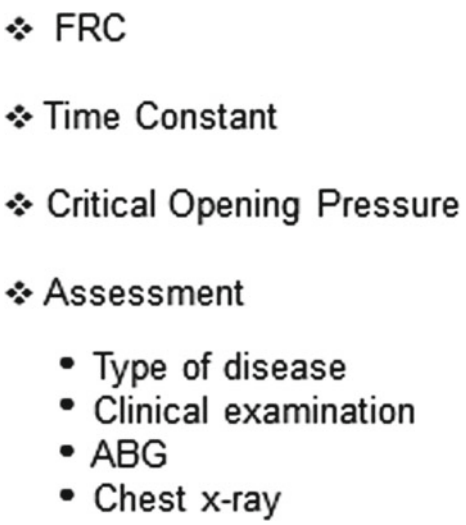

Fig. 7.3 Pathophysiologic considerations in mechanical ventilation

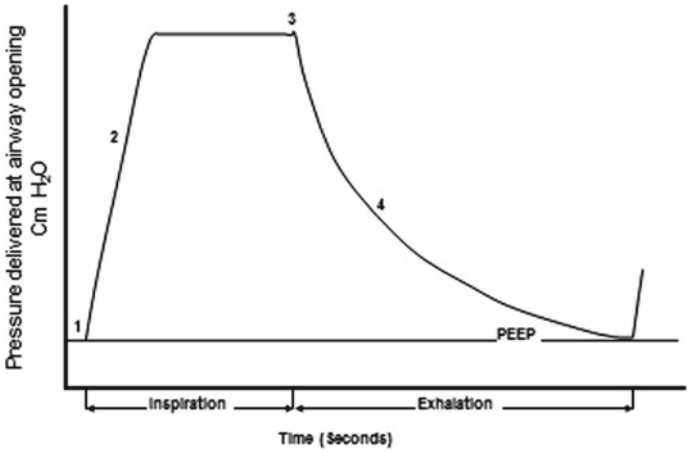

Fig. 7.4 Four phases of a pressure-controlled time-cycled mechanically delivered breath. (1) Initiation of inspiration, (2) Inspiratory phase characteristics, (3) Termination of inspiration, and (4) Expiratory phase characteristics

7.2.2 Initiation of Inspiration and the Control Variable (Mode)

The initiation of inspiration can be set to occur at a predetermined rate and time interval regardless of patient effort, or it could be timed in response to patient effort. Once the inspiration is initiated, the ventilator breath is either delivered by precisely set volume or pressure parameters (volume control or pressure control mode) or supports the patient's effort to a predetermined inspiratory volume or pressure target (volume support or pressure support mode). Advances in technology have allowed greater patient-ventilator synchrony to occur. The ventilator may be set to be "triggered" by the signal it receives as a result of the patient effort. This signal may be in form lowering of either pressure (pressure trigger) or the base flow (flow trigger) in the ventilator circuit generated by the patient's inspiratory effort. If no such signal is received, the ventilator delivers a breath at an interval selected by the operator.

Control Modes

Intermittent Mandatory Ventilation (IMV) mode In IMV, the inspiration is initiated at a set frequency. In between the machine delivered breaths a fresh source of gas is provided for the patient's spontaneous breaths. Once initiated the inspired gas is delivered at a pre-set amount of pressure (Pressure control) or volume (Volume control). Patient's respiratory system compliance and resistance determine the amount of delivered tidal volume in pressure control mode or the amount of inflation pressure generated in volume control mode. The delivered IMV breath can be synchronized to the patient's inspiratory effort (SIMV).

Assist-Control (AC) Mode In AC mode, every spontaneous breathing effort by the patient triggers a machine delivered breath, either as pressure or as volume controlled. Once initiated, the inspiratory characteristics are according to the pre-determined parameters. A back-up control rate is set to ensure the minimum number of breaths delivered if there is insufficient number of patient trigger events. Since every spontaneous breath triggers a machine delivered breath, this mode is not suitable for patients as a weaning strategy.

Control Variable Once initiated, either the V_T or the inflation pressure is controlled. The mechanical breath is described as either volume controlled when a predetermined machine-delivered tidal volume is delivered or pressure controlled when a predetermined inflation pressure is generated at the airway opening. Adjusting the flow rate determines the inspiratory time (T_I) over which V_T is delivered in volume-controlled ventilation (VCV) whereas in pressure-controlled ventilation (PCV) T_I is directly pre-set as the time over which the inflation pressure will be administered. The inflation pressure generated during VCV and V_T delivered during PCV are secondary variables dictated by the respiratory system compliance and resistance.

VCV and PCV have their own advantages and disadvantages (Table 7.2). In patients with un-uniform time constants where some lung units fill up quickly (diseases with reduced compliance) and some take much longer for pressure equilibration to occur (diseases of increased resistance), PCV offers an advantage of raising the inflation pressure quickly, allow the areas of short time constants to fill up in early part of inspiration and let the areas of prolonged time constant fill in the later part of inspiration (Fig. 7.5).

Table 7.2 Characteristics of PCV and VCV

	Pressure-Controlled Ventilation	Volume-Controlled Ventilation
Control variables	- Inflation Pressure - Inspiratory time - Rise time	- Tidal volume - Flow rate - Inspiratory flow pattern (constant vs decelerating)
Machine-delivered volume	Depends on respiratory system compliance and resistance	Constant
Inflation pressure	Constant	Depends upon respiratory system compliance and resistance
Inspiratory time	Precisely set	Depends on flow rate adjustment
Endotracheal tube leak	Somewhat compensated	Part of delivered volume leaked during inflation
Distribution of ventilation	More uniform in lungs with varying time constant units	Less uniform in lungs with varying time constant units
Weaning process	Inflation pressure adjustment required to deliver desired tidal volume	Tidal volume remains constant, inflation pressure automatically weaned
Patient comfort	Possibly compromised	Possibly enhanced

Table 7.3 General recommendations for mechanical ventilation for patients with severely decreased respiratory system compliance (e.g., ARDS, pneumonia)

Age (Years)	Rate ("Rapid")	Tidal volume ("Shallow")	I:E ratio
0.1–2	30–40/min	6 mL/Kg	(1.5–1):2
2–4	25–30/min	6 mL/Kg	(1.5–1):2
5–12	20–25/min	6–7 mL/Kg	(1.5–1):2
13–18	20/min	6–7 mL/Kg	(1.5–1):2

Titrate PEEP ($\geq$6 cm H_2O) for improving PaO_2/FiO_2. Monitor C_{dyn}

Table 7.4 General recommendations for mechanical ventilation for patients with severe obstructive lung disease (e.g., status asthmaticus).

Age (Years)	Rate ("Slow")	Tidal volume ("Deep")	I:E ratio
1–4	18–20/min	10 mL/Kg	1:3
5–8	14–18	10–12 mL/Kg	1:3
9–12	12–14	12 mL/Kg	1:3.5
13–18	8–12	12 mL/Kg	1:4

Add sufficient PEEP to counteract Auto-PEEP. Monitor C_{dyn}

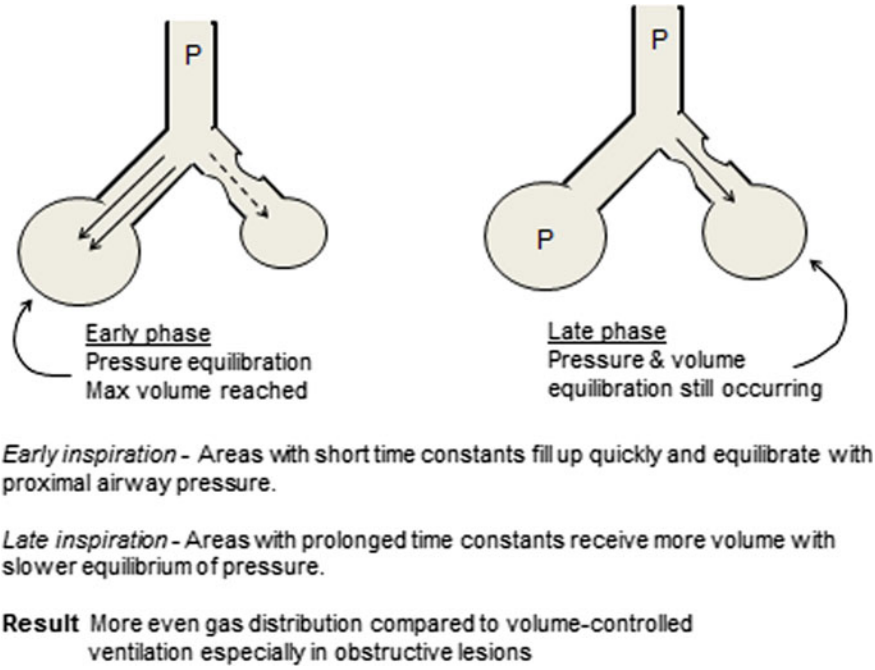

Fig. 7.5 Pressure-control Ventilation

Fig. 7.6 Volume-control Ventilation

This limits excessive rise in inflation pressure and forces more uniform distribution of tidal volume. In VCV, the areas of shorter time constant will be preferentially filled with the delivered volume throughout the inspiration resulting in uneven distribution of ventilation and a rise in inflation pressure and a decrease in C_{DYN} (Fig. 7.6). VCV is more advantageous in patients with relatively normal or recovering lungs as a reliable V_T can be delivered. In such situations, the inflation pressures fall automatically with improving compliance and resistance. With PCV, the delivered V_T can change significantly with rapid changes in compliance and resistance requiring frequent manipulations of inflation pressure.

Pressure Regulated Volume Control (PRVC)

The advantage of PRVC over PCV is a more consistent tidal volume delivery over time since it targets a set tidal volume per breath. When the delivered tidal volume is lower than the set tidal volume, the ventilator automatically increases the peak inspiratory pressure required to meet the target tidal volume up to a certain limit. When the delivered tidal volume is larger than the set tidal volume, when there is an improvement in compliance, the peak inspiratory pressure is automatically decreased to maintain the set tidal volume. The disadvantage is that in PRVC, with severe lung disease, the peak inspiratory pressure may be increased above the upper inflection point to maintain the tidal volume. Therefore, in PRVC, peak inspiratory pressure changes need to be monitored more closely.

Support Modes

Pressure-support ventilation (PSV) and Volume support ventilation (VSV) Support modes are designed to support patient's spontaneous respiratory efforts. With PSV, the patient's spontaneous respiratory effort is supported by a rapid rise in in ventilatory pressure to a pre-selected level. The inspiration is continued until the inspiratory flow falls to a pre-set level (generally 25%) of peak flow rate as the lungs fill up. Thus the T_I is controlled by the patient's own efforts and the pulmonary mechanics. With VSV, all spontaneous breaths are supported by generation of inflation pressure to deliver a pre-set tidal volume. They are frequently combined

with SIMV so that any breath above the SIMV rate is supported by either PSV or VSV.

Inspiratory phase characteristics Once initiated, the T_I, the inspiratory flow waveform, and the pressure rise time can be adjusted to suit the pulmonary mechanics. In PCV, T_I is directly set in seconds. In VCV, the T_I is adjusted by adjusting the inspiratory flow (volume over time). Increasing the flow rate will decrease T_I and decreasing the flow rate will increase it. I:E ratio depends on the respiratory rate which determines the duration of the total respiratory cycle. Both T_I and T_E should be considered individually. Increase in T_I will increase MAP and also the duration of time the pulmonary capillary blood is exposed to higher PO_2 resulting in improved oxygenation. This strategy is helpful in situations where FRC is decreased such as in ARDS or pulmonary edema. Increasing T_I will also increase V_T in PCV without increasing the inflation pressure if inspiratory flow has not ceased at the end inspiration. T_E must be sufficient to allow for expiratory flow to return to baseline or close to it. Decrease in respiratory rate may be required if T_E is insufficient for adequate exhalation.

Inspiratory flow waveform can be adjusted in VCV mode as either a constant flow (square waveform) or a decelerating flow (descending ramp waveform). With a square waveform the flow is kept constant throughout inspiration. In a descending waveform, flow is maximal at the start of inspiration and steadily declines to zero at end inspiration.

In PCV and PSV, the predetermined inflation pressure is achieved through delivery of airflow. Pressure rise time reflects the rapidity with which the ventilator achieves the target pressure. Rise time is adjusted to a value that is most comfortable for a patient who is awake and also to prevent a rapid rise and pressure overshoot that could be injurious to the lung.

Termination of inspiration (Cycle) Cessation of inspiration is effected by 3 mechanisms depending on the mode used; time-cycling, volume-cycling, or flow-cycling. PCV is "cycled off" when a predetermined T_I elapses (time-cycled), VCV inspiration is terminated after the prescribed volume is delivered, and PSV is cycled off after inspiratory flow declines to a pre-selected percentage of peak flow. Volume cycled breath can be pressure-limited to prevent a rise in pressure beyond a certain limit. In such a situation, an inspiratory hold is created as the excess volume is popped off in the expiratory circuit. While this strategy prevents undesirable elevation in the inflation pressures, less than predetermined volume is delivered to the patient.

Expiratory phase maneuvers The most useful expiratory phase maneuver is the application of PEEP. The most important clinical benefits of PEEP are to recruit atelectatic alveoli and to increase FRC in patients with alveolar-interstitial lung disease thereby improving oxygenation. Even briefly disconnecting the ventilator and allowing the alveolar pressure to reach zero results in substantial de-recruitment and loss of FRC that takes a period of time to recover from after reapplication of PEEP. In patients with obstructive disease where insufficient exhalation time results in auto-PEEP, application of extrinsic PEEP, can delay airway closure and improve ventilation. Other salutary effects of PEEP include displacement of alveolar fluid to

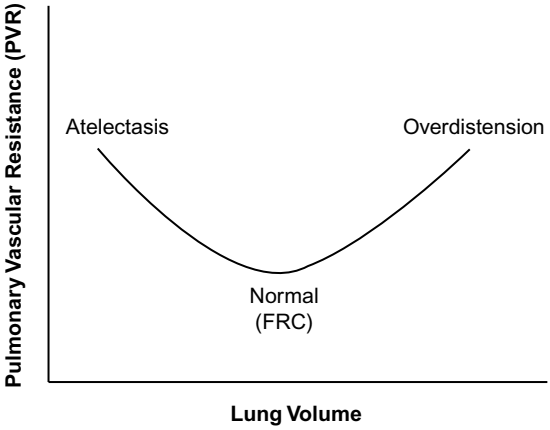

Fig. 7.7 Effect of FRC on PVR. Pulmonary vascular resistance (PVR) is the lowest at normal FRC. At too low (atelectasis) and too high (overdistension) FRC, the PVR is proportionately elevated

extra-alveolar spaces, decrease in left to right shunt and stabilization of chest wall. Effect of PEEP on lung compliance is variable and it depends on the patient's pulmonary mechanics. By shifting the ventilation to a more favorable part of the pressure–volume curve, PEEP may recruit more alveoli and improve lung compliance. Excessive PEEP on the other hand will result in alveolar overdistension and reduction in compliance. (Fig. 7.2). The effect of FRC on pulmonary vascular resistance (PVR) is parabolic. PVR is at the lowest at a normal FRC. PVR is increased at excessively low (atelectasis) and excessively high (overdistention) FRC. (Fig. 7.7). Elevated PVR increases right ventricular afterload and impairment of cardiac output. At low FRC, there is increased intrapulmonary right to left shunting past the hypoventilated, atelectatic alveoli whereas at high FRC, there is increase in dead space ventilation because of decreased pulmonary perfusion. Efforts directed at achieving normal FRC is the goal of the mechanical ventilation strategies.

Airway Pressure Release Ventilation (APRV*)* APRV is utilized to improve oxygenation in patients with diffuse atelectatic process such as ARDS. This modality delivers high level continuous positive pressure (P_{high}) to the respiratory system through most of the breath cycle (3–5 s) with intermittent release of the pressure without allowing it to return it zero (P_{low}) for brief periods (0.3–0.5 s) of time. P_{high} is aimed at recruiting alveoli and maintaining satisfactory lung volume while P_{low} is to allow alveolar gas to escape for CO_2 elimination while still maintaining positive alveolar pressure at end expiration to maintain satisfactory FRC. P_{high} is akin to PIP and P_{low} is similar to setting PEEP. The patient is allowed to breathe during both the P_{high} and P_{low} phases. The long P_{high} phase is tolerated because of the active expiratory valve in the ventilatory circuit making spontaneous respiration possible.

High Frequency Ventilation (HFV) HFV is another approach used especially in children with hypoxemic respiratory failure. The strategy is to recruit lung volume using supraphysiologic respiratory rates at high mean airway pressure (MAP) and

relatively minor fluctuations in pressure around it to deliver small V_T. The aim is to protect the lung from excessive tidal stretch which is responsible for ventilator induced lung injury. Two forms of HFV are most commonly in vogue. Both are commonly used as rescue therapies when conventional ventilation is likely to be ineffective or injurious. High frequency oscillation (HFO) employs to and fro oscillations with a parallel bias flow from which air is entrained. Air is pushed in during inspiration and sucked out during exhalation. The main determinants of oxygenation are MAP and FiO_2 whereas changes in pressure (amplitude) determine the ventilation. High frequency jet ventilation (HFJV) tiny amounts of gas (jets) at high velocity are introduced into the bias flow causing entrainment of additional gas. Unlike HFO, the exhalation in HFJV is passive due to the elastic recoil of the lungs and the chest wall. Major determinants of oxygenation are FiO_2 and PEEP while PIP determines the ventilation. In our experience, HFOV is an effective strategy for predominantly alveolar interstitial disease such as ARDS and pneumonia. However, in patients with significant airway obstruction (e.g. bronchiolitis, pertussis), the use of HFOV may exacerbate airway collapse during exhalation (Fig. 6.13) because of the generation of negative pressure in the airway. In such situations, HFJV may be the preferred option.

Conventional Ventilator Settings

Fraction of inspired oxygen (FiO2) The shape of the hemoglobin-O_2 dissociation dictates that most of the oxygen attaches to the hemoglobin at PaO_2 of around 70 torr at which hemoglobin-O_2 saturation (SO_2) is 94% under normal circumstances. Higher PaO_2 levels contribute little to increase O_2 content while exposing the patient to the risk of O_2 toxicity. Unless higher PaO_2 is required temporarily for select situations such as CO poisoning, pulmonary hypertensive crisis and severe anemia, a PaO_2 that yields SaO_2 in the mid 90's should be adequate. In most situations, a PaO_2 value around 70 torr is a reasonable goal. Whenever possible, FiO_2 should be decreased to < 0.50 as long as SaO_2 remains in the mid 90's. FiO_2 concentrations less than 0.5 are generally considered safe.

Mode The choice of mode of ventilation depends upon the disease entity that is treated and how much ventilator-patient interaction is desired. Patients with normal lungs with ability to trigger the machine are best managed with SIMV in a volume control mode or either PSV or VSV as a support mode. The emphasis should be placed on the mode that provides maximum comfort. Patients with abnormal lungs, either obstructive or restrictive nature, will require more precise ventilatory strategies. Generally, VCV is preferable since it is the V_T that determines the alveolar ventilation rather than inflation pressure which effects a change in volume as a secondary variable. In more severe alterations with respiratory units of different time constants, PCV is the preferred choice for effecting a more uniform distribution of V_T. Regardless of the choice of the mode, the exhaled VT (VT_E) and PIP should be monitored on an ongoing basis.

Tidal Volume and Rate The dose of alveolar ventilation (V_A) is calculated as ($V_T - V_D$) X Rate. The anatomic V_D is generally estimated to be 2.2 mL/kg. The manner in which V_A is administered should be based according to the patient's

pathophysiologic alterations. The most important consideration is that of time constant, a product of compliance and resistance.

In patients with normal lungs, time constant can be assumed to be normal. V_T can be chosen as 8–10 mL/kg with age appropriate respiratory rate. In such patients (such as those being ventilated for status epilepticus, traumatic/metabolic encephalopathies, neuromuscular dysfunction etc.), alveolar ventilation should be adjusted to maintain a desired $PaCO_2$ as monitored by $ETCO_2$ or blood gas analysis.

In patients with reduced lung compliance (e.g. ARDS, pulmonary edema, interstitial pneumonia), the time constant is shorter than normal. Pressure equilibration occurs more rapidly. Also, larger tidal volumes and higher inflation pressures are injurious to the lungs. Such patients are best ventilated at relatively lower V_T (6–7 mL/kg) and higher respiratory rates to maintain adequate V_A. When using PCV, an appropriate inflation pressure (PIP-PEEP) should be selected to deliver the desired volume. If VCV or PRVC is used, the end inspiratory pressure should be kept under less than 30 cm H_2O if possible.

In patients with increased airway resistance (e.g. asthma), the time constant is prolonged, necessitating longer time for pressure to be equilibrated and volume to be delivered to the distal end. Such patients should be ventilated at slower rates providing sufficient time for inflation and deflation to occur. To compensate for slower rates, V_T needs to be increased up to 12 mL/kg as necessary (Tables 7.3 and 7.4).

Inspiratory Time (TI) and Expiratory Time (TE) Both T_E and T_I need to be set to allow for satisfactory inflation and deflation to occur. In addition to considering the I:E ratios, T_I and T_E should also be considered independently of each other in terms of adequacy for pressure equilibration to occur. This most certainly depends on the time constant. The total time available for each respiratory cycle is determined by the respiratory rate (RR). Thus $T_E + T_I$ is 5 s at RR of 12/min and 3 s for RR of 20/min. In a normal respiratory pattern the exhalation is twice as long as inspiration with pressure equilibration occurring at the end of either of these phases. With diseases of compliance, as the time constant is short, pressure equilibration is rarely a problem allowing for faster rates and smaller V_T. Prolongation of T_I is often practiced to improve oxygenation by (a) increasing MAP and (b) allowing greater duration for pulmonary capillary blood to be exposed to higher PAO_2. In diseases of increased airway resistance the time constant is prolonged. However, the TC_E is prolonged much more than TC_I necessitating not only a slower rate but prolongation of exhalation with I:E ratios of 1:3 or more. The best way of determining the effective T_I, T_E and I:E ratios is to observe flow time relationship on ventilator wave form and the VT_E. The flow should be nearly complete especially at end exhalation and VT_E should be sufficient for adequate V_A. T_I is set directly on the ventilator setting in PCV and PRVC while in VCV, it is a function of inspiratory flow rate which is set to distribute the V_T over the duration of T_I.

Positive End-Expiratory Pressure (PEEP) The most important application of PEEP is in alveolar-interstitial disease to increase FRC above the critical opening pressure or lower P_{Flex} and allowing ventilation to occur in the relatively safe zone,

improving C_{dyn} and avoiding tidal recruitment (Fig. 7.2). Increase in FRC results in improved oxygenation and decrease O_2 requirements. Choosing the ideal PEEP is often based on PaO_2/FiO_2 values. The adverse effects of PEEP on venous return compromising cardiac output should also be taken into account. In diseases of increased resistance, application of PEEP delays airway closure and reduce air-trapping. This effect can be monitored by measuring the auto-PEEP before and after application of PEEP. A decrease in auto-PEEP is desired after application of PEEP through the ventilator. Presence of an ET tube prevents the patient's ability to grunt by exhaling with a partially closed glottis. Grunting results in maintaining positive pressure at end expiration to maintain alveolar volume. All intubated patients, even those with normal lungs, should therefore have, at a minimum, a small amount of PEEP (2–4 cm H_2O) to maintain FRC.

The optimum PEEP is the level at which there is an acceptable balance between the desired goals and undesired adverse effects. The desired goals are (1) reduction in inspired oxygen concentration to "nontoxic" levels (usually < 50%); (2) maintenance of PaO_2 or SaO_2 (arterial oxygen saturation) of more than 60 mmHg or more than 90%, respectively; (3) improvement of lung compliance; and (4) maximal oxygen delivery.

7.3 Weaning and Extubation

Mechanical ventilation, while life-saving, can be associated with undesirable side effects and complications. Therefore, it is important that, as soon as the patient is capable of comfortably sustaining adequate gas exchange with spontaneous breathing, mechanical ventilation is discontinued. When weaning the patient off mechanical ventilation, it is important to consider decreasing the most injurious part of the support. Often it is the FiO_2, inflation pressure (or V_T), and respiratory rate in that order. Also, greater reliance on support modes over mandatory modes is preferred for patient comfort and safety.

7.3.1 The Weaning Process

Weaning from mechanical ventilation is the transition from ventilatory support to complete spontaneous breathing. During this transition, the patient assumes increasing responsibility for effective gas exchange while positive pressure support is reduced. Weaning is complete and is defined as a success when a patient maintains adequate gas exchange with complete spontaneous breathing, while remaining comfortable without any mechanical assistance. Weaning is defined as a failure when spontaneous efforts are incapable of sustaining effective gas exchange without mechanical support. The timing of extubation generally coincides with an assessment that the patient is capable of maintaining acceptable gas exchange

without ventilator support. Extubation failure is defined as the need for reintubation within 48 h of extubation.

7.3.2 Initiation of Weaning

Weaning should start when: (1) the underlying disease process is improving; (2) the gas exchange is adequate; (3) no conditions exist that impose an undue burden on the respiratory muscles, such as cardiac insufficiency, severe malnutrition, and muscle weakness; and (4) the patient is capable of sustaining spontaneous ventilation as ventilator support is decreased without expending an excessive amount of energy. Improvement of the underlying disease process can be assessed by measurement of gas exchange, respiratory system mechanics, and x-ray findings. Patients cannot be arbitrarily forced to wean because it is the patient who dictates the pace of the weaning process. Patient's ability to breathe effectively depends on several factors: (1) respiratory muscle strength, (2) stability of the cardiovascular system, (3) work of breathing, (4) general nutritional status of the patient, and (5) the absence of an underlying hypercatabolic state (e.g., sepsis).

7.3.3 Weaning Techniques

Weaning practice ranges from abrupt withdrawal to gradual withdrawal from ventilatory support. Most common practice is gradual reduction in ventilator settings which involves reducing the mechanical minute ventilation, FiO_2, and PEEP in steps while assessing the patient's ability to tolerate the change. Currently, most children are weaned with SIMV with or without added pressure support, or with pressure support alone. At the end of weaning, patients may or may not be tested with an extubation readiness test (ERT) before liberation from mechanical ventilation. An ERT is defined as a trial that tests the ability of complete spontaneous breathing to maintain gas exchange with or without minimal assistance. The criteria for passing and terminating an ERT are shown in Table 7.5.

Studies have shown that in children, when the criteria to initiate weaning are met, 50–75% of the patients can be liberated from mechanical ventilation after passing an ERT without undergoing a gradual reduction in ventilator settings. The premise underlying the more rapid withdrawal is that many patients are ready to be liberated as soon as they meet the criteria to initiate weaning and therefore, do not require a prolonged weaning process. If patients meet the criteria as outlined in Table 7.6, they can be subjected to an ERT. If the ERT is successful, then the patient can be liberated from mechanical ventilation. If a patient fails an ERT, mechanical ventilation can be continued at a level that keeps the patient comfortable with no increased work of breathing. If invasive mechanical ventilation in continued, the ERT can be repeated in 24 h. Alternatively, the patient can be extubated

Table 7.5 Criteria for initiating an Extubation Readiness Test (ERT)

Eligibility for an ERT
1. Improvement or resolution of the underlying cause of acute respiratory failure
2. Adequate oxygenation
• PaO_2 > 65 mmHg
• SpO_2 > 95%
• F_IO_2 < 0.4
• PEEP < 7 cmH_2O
3. Adequate ventilation
• $PaCO_2$ < 45 mmHg with no chronic respiratory failure
• $PaCO_2$ > 45 mmHg with acceptable arterial pH with chronic respiratory failure
4. A core temperature below 38.5 °C
5. Alert mental status after removal of sedative agents
6. Minimal to no vasoactive agent requirement
• There should be no planned procedures for next several hours
• Enteral feeds should be stopped for 4 h before ERT
• Maintenance fluid are being given IV in infants and small children

Table 7.6 Criteria for passing an extubation readiness test

• Can maintain SpO_2 > 95% during the test
• Is breathing comfortably without significant retractions or paradoxical breathing
• Respiratory rate within the range: (a) age < 6 months - 20–60/min
(b) 6 months-2 years - 15–45/min
(c) 2–5 years - 15–40/min
(d) > 5 years - 10–35/min
• Spontaneous tidal volume > 5 mL/kg
• No hemodynamic instability during the trial

to noninvasive mechanical ventilation, if appropriate and eligible. If the decision is to extubate to noninvasive positive pressure support, then it would be prudent to test the patient on a level of pressure support that is necessary to maintain gas exchange and decrease the work of breathing (Table 7.6).

7.3.4 ERT Trials

There are three commonly employed ERT methods: CPAP provided through the ventilator, T-piece breathing, and minimal PSV with PEEP. During a CPAP trial, the patient is placed on a low level of CPAP (usually 5 cm H_2O) with or without supplemental oxygen and without any pressure support. With a T-piece trial, the patient is removed from the ventilator and humidified supplemental oxygen is provided to the airway. In this system, a corrugated tubing from the nebulizer/humidifier attaches to one end of the T-piece, and an extension of corrugated tubing attaches to the other end of the T-piece. The flow rate is adjusted to produce a

constant mist coming from the extension piece on the T-tube both during inspiration and expiration so that the patient's minute ventilation is matched by the device. This corresponds to approximately at least three times the patient's minute ventilation. There is some concern that the endotracheal tube increases the work of breathing. Therefore, a low-level PSV (5–10 cm H_2O) has been advocated as an ERT based on the hypothesis that this overcomes the resistance to breathing through the artificial airway. Several studies have shown that an appropriately sized endotracheal tube with an adequate inspiratory flow through a T-piece circuit does not increase the inspiratory work of breathing. The duration of an ERT can range from 30 min to 2 h. An ERT is terminated if any of the criteria listed in the Table 7.6 is not present. Instead of a specific level of pressure support, some ventilators such as the Servo-I have a volume-targeted pressure-support mode called volume support that can guarantee a minimal tidal volume and minute ventilation. When the pressure support level decreases to a predetermined low level usually < 8 cm H_2O while maintaining a tidal volume of at least 5 mL/kg, then the patient can be liberated from mechanical ventilation. Most clinicians prefer either a CPAP or a pressure support with PEEP trial.

7.3.5 Extubation

The patient must be awake, alert, and have airway protective reflexes. Breathing must be effective and without undue exertion. Adequate gas exchange with a relatively low FiO_2 must be established. Cardiovascular function with satisfactory perfusion is a prerequisite. When the ERT is deemed successful, the patient can be extubated and be liberated from mechanical ventilation to entirely spontaneous breathing without any positive pressure support. When the patient fails an ERT, he/she can still be extubated to noninvasive ventilation if they are suitable candidates. For patients to be candidates for non-invasive mechanical ventilation, they must have adequate airway protective reflexes and tolerate the necessary nasal mask or facemask. In infants, extubation to nasal CPAP may be an option. It is important to remember that while they have been liberated from endotracheal intubation, they have not been liberated from positive pressure support. Noninvasive ventilation, therefore, can be viewed as a transitional step toward complete liberation from positive pressure support.

Extubation failure is defined as the requirement for reintubation within 48 h after extubation.

Factors that prolong the weaning process are (1) slow resolution of the underlying disease process, (2) ventilatory pump failure, and (3) psychological factors. Ventilatory pump failure can be due to increased respiratory work load, decreased respiratory muscle capacity, or a combination of both. Decreased ventilatory drive may result from respiratory center dysfunction caused by sedative agents; neurological dysfunction, and metabolic alkalosis. Phrenic nerve injury, chest wall

splinting and instability are contributing factors after cardiovascular surgery. They increase the work of breathing.

7.4 Ventilator Induced Lung Injury

Ventilator-induced lung injury (VILI) is a term that encompasses many aspects of injury caused by mechanical ventilation. Alveolar rupture can occur from over-distended alveoli. Pneumothorax, pneumomediastinum, pneumoperitoneum, pneumopericardium, interstitial emphysema and subcutaneous emphysema are all examples of overt ventilator-induced lung injury. These examples of extra-alveolar air can be life-threatening due to cardiovascular compromise. A bronchopleural fistula is a track that develops between the bronchus and the pleural space resulting in an almost continuous flow of air from the airway into the pleural space. Ventilator-induced lung injury can be minimized by protective lung ventilation.

7.4.1 Airway Injury

An endotracheal tube traversing the upper airway can be associated with significant airway injury. Tight taping can cause pressure injury leading to ulceration of the angle of the mouth (orotracheal tube) or ala nasi (nasotracheal tube). Palatal injury can range from simple ulceration to deep grooves including traumatic cleft palate in severe cases. Newborns and young infants are especially vulnerable because of their softer tissues. Laryngeal injury may extend from minor swelling to ulceration of the mucosa involving supraglottic structures and the vocal cords. A common injury seen in infants and children is in the subglottic region and may range from minor swelling to major ulceration. Scarring and granuloma formation can result in significant airway obstruction. The factors that increase the risk of tracheal injury are the size of the endotracheal tube, high cuff pressure, decreased tissue perfusion, upper respiratory tract infection, duration of intubation, and head/neck movement. Suctioning to keep the airways patent and clear secretions can also cause injuries if done vigorously. Tracheal injuries can lead to tracheal stenosis and/or tracheomalacia.

7.4.2 Biotrauma, Atelectrauma, Oxytrauma

Large V_T delivered at pressures with increased frequency cause cyclic strain, which may lead to disruption of the tight junctions between the alveolar epithelial and capillary endothelial cells and intracapillary blebs. The resultant biotrauma may cause the release of proinflammatory cytokines that further injure the lung and enter

the systemic circulation, leading to multiorgan failure. Evidence shows that in patients with ARDS, avoidance of $V_T \geq 10$ mL/kg and $P_{plat} \geq 30$ cm H_2O limits diffuse alveolar damage. Atelectrauma is a shear stress on the alveolar walls caused by cyclic opening and closing of the alveoli. Keeping PEEP above the lower P_{Flex} prevents repetitive alveolar collapse. It is important that alveolar units are neither overdistended nor collapsed. Careful adjustments of PEEP are also useful in lowering the FiO_2 another source of lung injury (oxytrauma). Although the FiO_2 value below which there is no risk of O_2 toxicity is unknown, a value < 0.6 is prudent.

7.4.3 Ventilator-Associated Pneumonia

The pathophysiology of ventilator-associated pneumonia (VAP) is multifactorial. Aspiration of oral and/or gastric secretions, colonization of ET tube, suppression of cough and impediment to mucociliary clearance play a collective role. New-onset fever and leukocytosis accompanied by demonstration of a newly observed infiltrate on chest radiograph are consistent with a diagnosis of VAP. Occurrence of VAP results in worsened gas exchange, increased duration of ventilation, and even death. Elevation of the head of the bed to 30 degrees after initiation of mechanical ventilation and oral decontamination measures during mechanical ventilation are effective means of reducing the risk for VAP. The most effective strategy to minimize any of the aforementioned complications is regular assessment of extubation readiness and liberation from mechanical ventilation as soon as clinically possible.

7.5 Heart–Lung Interactions

While anatomically the heart lies between two lungs, functionally the lungs are in between two types of heart circulations; systemic and pulmonary. It is hardly a surprise that alteration in one organ influences the function of the other. Heart-lung interactions can be classified as neural, humoral, functional, and mechanical. Neural interactions refer to the changes in the respiratory or cardiovascular system when the other system is perturbed due to neural connections between the two systems. For example, hypoxemia stimulates peripheral chemoreceptors and cause hyperpnea and hyperventilation. Lung inflation can induce reflex changes in heart rate. Humoral interactions are mediated through substances that are released by the lung during lung inflation which affects the cardiovascular system. Functional interactions refer to the effect of dysfunction of one system on the other. Heart failure can affect gas exchange and breathing. Chronic lung disease can result in pulmonary hypertension which can affect right ventricular function. These are referred to as functional heart–lung interactions. Mechanical interactions are due to the changes in lung volume and intrathoracic pressure affecting cardiovascular function.

7.5.1 Mechanical Heart–Lung Interactions

During inspiration, there is change in lung volume as well as a change in intrathoracic pressure. Intrathoracic pressure is negative during spontaneous breathing and negative pressure ventilation, while positive pressure ventilation increases the intrathoracic pressure during inspiration. Heart–lung interactions involve changes in lung volume or intrathoracic pressure affecting heart rate, preload, contractility and afterload of one or both ventricles. Lung inflation at normal tidal volumes increases heart rate by inhibiting the vagus nerve.

Spontaneous breathing increases venous return and right ventricular preload by increasing the gradient for venous return. Positive pressure ventilation on the other hand, decreases right ventricular preload by decreasing the gradient for venous return. The effect of positive pressure on venous return is exacerbated in shock, especially due to hypovolemia. This effect can be mitigated by bolus fluid administration. Pulmonary vascular resistance (PVR) is lowest at normal functional residual capacity. PVR increases when lung volume is below or above the functional residual capacity (Fig. 7.7). With atelectasis, there is local hypoxic pulmonary vascular resistance and kinking of vessels which leads to an increase in PVR. Hyperinflation of the lungs results in alveolar vessel compression and a rise in PVR. During spontaneous inspiration, right ventricular preload increases. The increase in right ventricular end-diastolic volume leads to a decrease in left ventricular end-diastolic volume and compliance and reduced left ventricular filling. The effects on afterload are different in right and left ventricle. Changes in intrathoracic pressure are shared equally by the right ventricle and pulmonary circulation, but while these changes are experienced by the left ventricle, a large part of systemic circulation is outside the thorax and not subjected to them. The left ventricular afterload is thus increased during inspiration while breathing spontaneously as it must generate greater cavity tension to overcome systemic vascular resistance. The decreased preload and increased afterload during inspiration is thought to be the mechanism for pulsus paradoxus during obstructed breathing such as with croup and asthma.

Practical Applications of Heart–Lung Interactions

The clinician has to consider several practical applications of heart–lung interactions when managing patients with mechanical ventilation. Many factors may influence decision making during invasive ventilation, the weaning process and thereafter.

Oxygen Cost of Breathing

Under resting conditions, oxygen cost of breathing is minimal, about 5% or less of the total oxygen consumption. A patient with normal cardiovascular function has a large reserve capacity to increase the cost of breathing such as with exercise. But with heart failure, the oxygen cost of breathing increases, and around 15% or greater, patient becomes dyspneic. When the demand of the respiratory muscles

outstrips the cardiovascular system's ability to supply O_2, the respiratory muscles are prone to fatigue resulting in respiratory pump failure. Mechanical ventilation reduces the work of breathing and the oxygen cost of breathing, thereby reducing the fraction of blood flow needed for the respiratory muscles. During weaning, spontaneous breathing increases and may unmask cardiac dysfunction by a reduction in central/mixed venous oxygen saturation and an increase in lactate production.

Preload Responsiveness of the Heart

With a normal heart, which is preload-dependent, there is a cyclic variation in systolic blood pressure during mechanical ventilation. The normal difference between the peak increase and peak decrease is about 5–10 mmHg. This magnitude is increased with hypovolemia or a compromise in venous return such as with application of PEEP. The greater the magnitude, greater is the response to a fluid bolus. Thus, in a patient with circulatory shock, systolic pressure variation during mechanical ventilation can be used to determine whether a patient will respond to a fluid bolus. On the other hand, in heart failure where the heart is preload-independent, systolic blood pressure variation may be minimal or absent.

Improvement in Cardiovascular Performance in Heart Failure

In patients with heart failure, mechanical ventilation may improve cardiovascular performance by several mechanism: (1) increasing stroke volume by reducing the left ventricular afterload, (2) decreasing PVR by normalizing FRC, (3) reducing the demand on the heart by reducing the work of breathing, and (4) decreasing lactate production by respiratory muscles, and (5) improvement in gas exchange by decreasing alveolar edema and recruitment of alveoli.

Hemodynamic stability in Functionally Univentricular Lesions

Following a Fontan procedure for tricuspid atresia where pulmonary blood flow is passive, increase in intrathoracic pressure will not only decrease venous return but also increase the PVR. The end result is a decrease in the driving pressure for pulmonary blood flow and a decrease in cardiac output. Early extubation and spontaneous breathing are to be encouraged. Some patients may develop atelectasis and may need lung inflation or a distending pressure to maintain adequate lung volumes. Negative pressure ventilation offers an attractive alternative to these patients by creating a negative intrathoracic pressure which will recruit the lungs and increase the gradient to pulmonary blood flow.

In patients with univentricular physiology where the pulmonary and systemic blood flows are dependent on a single pumping chamber such as after Norwood procedure, a switch from positive pressure ventilation to spontaneous respiration may pose a formidable challenge after transitioning from positive pressure ventilation to spontaneous respirations after surgical repair. Increased afterload for the systemic circulation compared to that for the pulmonary circulation, may direct the cardiac output from the single pumping chamber preferentially to the pulmonary circulation resulting in potentially life-threatening systemic hypoperfusion

syndrome and lactic acidosis. Such patients may benefit from reinstitution of either invasive or noninvasive positive pressure ventilation.

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