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Textbook of Ventilation, Fluids Electrolytes and Blood Gases

Mohan Gurjar

Forewords
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CHAPTER 7

Mechanical Ventilation in Specific Clinical Scenarios

Afzal Azim

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INTRODUCTION

Respiratory failure is one of the most common indications for admission to the emergency department and specialist wards, and the need for respiratory system support represents the most common indication for admission to intensive care unit (ICU). Approach to patients with respiratory failure based on the principles of noninvasive and invasive respiratory support is essential for all healthcare professionals involved in patient management.

Clinical application of mechanical ventilation requires strategy modulation according to disease pathophysiology. Physiologically, hindrances to lung movement during respiration can be grouped into elastic resistance and nonelastic resistance.[1,2]

- Elastic resistance includes resistance due to lung tissue and chest wall. It also includes resistance from surface tension at the alveolar gas liquid interface. It is measured when no air flows in the respiratory system. Increased elastic resistance is seen in acute respiratory distress syndrome (ARDS) and restrictive lung diseases.
- Nonelastic resistance includes the influence of friction to gas flow through the airway, friction resistance from thoracic tissue deformation, and a small contribution due to inertia of gas and movement of tissues. It is measured when air is flowing through the respiratory system. Nonelastic resistance is increased in obstructive airway disease.

Work of breathing consists of overcoming these resistances. Increase in one or other forms of resistance leads to increased work of breathing.

Two classic examples of fundamentally different approaches of mechanical ventilation are chronic airflow obstruction and adult respiratory distress syndrome. The current chapter deals with managing mechanical ventilation in patient having obstructive airway disease, bronchopleural fistula, severe metabolic acidosis and ARDS.

OBSTRUCTIVE AIRWAY DISEASES

Airflow limitation is the hallmark feature of obstructive airway diseases, which mainly comprise of asthma and chronic obstructive pulmonary disease (COPD).[3] Traditionally asthma is characterized by a childhood or early adulthood onset disease with reversible airflow limitation while COPD frequently manifests at a later stage in life and has irreversible or partially reversible airflow limitation. Some patients have features of both COPD and asthma and are termed as overlap syndrome. Airway inflammation is neutrophil predominant in COPD, eosinophil predominant in asthma, and mixed in overlap syndrome.

BRONCHIAL ASTHMA[4,5]

Indications for intubation include:

- Progressive fatigue and exhaustion
- Depressed level of consciousness
- Respiratory arrest.

Physiological Concepts

Before discussing ventilator strategy, some basic physiological concepts are discussed below.

Concept of Time Constant

Time constant is the product of airway resistance and compliance. Expiratory time is a function of time constant. It takes approximately three times the time constants for complete exhalation of given volume of gas. At higher lung

volumes lung compliance decreases and shortens the time constant. Patients with increased airway resistance increase their lung volume and thus reduce their time constants and expiratory time.

Time Constant = Resistance × Compliance.

At higher lung volumes lung compliance decreases (you cannot inflate an already inflated balloon).

In patients with increased airway resistance, decrease in lung compliance finally results in decreased time constant and decreased expiratory time. Thus, a new steady state is achieved where end-expiratory lung volume (EELV) is higher than resting functional residual capacity (FRC), but permits expiration of the entire tidal volume.

Concept of Functional Residual Capacity or End Expiratory Lung Volume

During the embryonic development of the fetus, the chest wall grows faster than the lung. As a result of this differential growth, a negative pressure develops in the intrapleural space, which is responsible for the inward recoil of the lungs and the opposite recoil of the chest wall. FRC or EELV represents the point of respiratory system where inward recoil of lungs is equal to the outward recoil of chest wall with respiratory muscles at rest. This state is achieved at the end of expiration after normal tidal breathing.

Static and Dynamic Hyperinflation

Pulmonary hyperinflation is term used for increase in lung volume above its resting FRC.[6] Static hyperinflation is term used for increased lung volume due to loss of elastic recoil of the lung tissue as seen in patients of emphysema and old age. The resting point of the respiratory system is now shifted at higher FRC and the tendency of lung to recoil reduces. Dynamic hyperinflation (DH) is the term used when time between two inspiratory efforts is insufficient to completely decompress the lung to its resting state.

Pulmonary hyperinflation is the key deciding factor for initial ventilator settings in severe asthma.[7,8]

Mechanism of Hyperinflation

Patients with acute severe asthma breathe near their total lung capacity and with mechanical ventilation, lung volumes are likely to increase further. DH is triggered when there is insufficient time during expiration for complete exhalation of delivered tidal volume. This results in an increase in EELV leading to generation of auto-positive end-expiratory pressure (auto-PEEP). Subsequent breaths lead to progressive increase in lung volume which ultimately leads to improved expiratory gas flow due to increase in elastic recoil pressure and an increase in airways diameter, thereby permitting the entire tidal volume to be exhaled.

Although DH may be an adaptive process that enhances expiratory flow, it can result in severe hypotension and barotraumas. Several terms are used inconsistently as synonyms as listed below (Table 1):

- Dynamic hyperinflation
- Auto-PEEP (AP)
- Intrinsic PEEP (PEEPi)
- Gas trapping
- Occult PEEP
- Breath stacking.

Dynamic hyperinflation is not always the same as air trapping. DH can also occur without physically entrapping the air in certain conditions (Dependent gas trapping observed in obesity, recumbent position in ARDS patients). DH can also be better understood by the statements given below:

- Persistent inspiratory muscle activity during early part of expiration
- Beginning of inhalation before the respiratory system has returned to Vrel (resting lung volume).

Factors Contributing to Dynamic Hyperinflation or PEEPi

- Bronchospasm
- Mucosal edema
- Inspissated mucus plugs
- Tachypnea
- Anxiety leading to increased ventilatory demands.

Dynamic hyperinflation decreases the effectiveness of the respiratory system predisposing to respiratory fatigue and exhaustion which is attributed to the following points:

- Increased inspiratory effort to generate negative pressure equal in magnitude to PEEPi (resulting in loss of 20–30% of inspiratory muscle effort)

Table 1: Various terminologies related to PEEP (Positive end-expiratory pressure).

PEEP	Refers to extrinsic PEEP, which is preselected
Intrinsic PEEP	Refers to total PEEP (PEEPtot)
Intrinsic PEEP/total PEEP	Extrinsic PEEP + Auto-PEEP
Auto-PEEP (AP)	Intrinsic PEEP – Extrinsic PEEP
Auto-PEEP	Total PEEP – Extrinsic PEEP
Auto-PEEP	Equals to intrinsic PEEP when extrinsic PEEP is 0
PEEPi	Abbreviation for both AP and intrinsic PEEP

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- It decreases length tension relationship of muscles characterized by:
  - Decreased zone of apposition of diaphragm
  - Decreasing perfusion
- Increases end inspiratory lung volume reducing compliance and cause alveolar overdistension.

Hemodynamic Consequences of Dynamic Hyperinflation

This should always be kept in mind. These include:

- Reduced preload to right ventricular
- Increased right ventricular afterload
- Increased right ventricular end-diastolic volume (EDV)
- Shift of interventricular septum toward left
- Increased left ventricular afterload as a result of increased negative intrapleural pressure during inspiration.

Identification of Air Trapping and Auto-Positive End-expiratory Pressure in Flow-Time Scalar and Flow-Volume Loop

See Figures 1 and 2.

Initial Ventilator Settings

In severe asthma, the most important factors that determine the degree to which VEE (lung volume at end-expiration) increases during mechanical ventilation are:

- Expiratory resistance
- Tidal volume
- Expiratory time.

The ventilator settings that influence the severity of hyperinflation are tidal volume, respiratory rate, and inspiratory flow rate. In initial management, these variables must have optimal settings (Table 2); while subsequent ventilator adjustment should be based on pH and/or P_{plat} levels (Table 3).

The following therapies have to be used in conjunction with the ventilator therapy:

- Sedation with or without paralysis
- Therapies for airflow obstruction which include bronchodilator therapy and steroids as a part of bronchial hygiene therapy.

Monitoring during Ventilation

Pulmonary hyperinflation is the key deciding factor for ventilator settings in severe asthma: The degree of hyperinflation varies from patient to patient. Monitoring trends of hyperinflation is essential to bring changes in ventilator settings.[9,10]

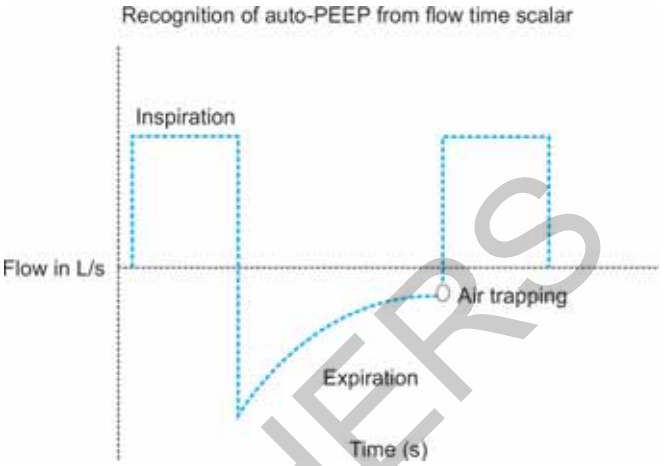

Fig. 1: The diagram represents the flow time scalar of patient on volume control mode and square wave pattern of flow. The graph does not touch the baseline at the end of expiration, thus representing air tapping.

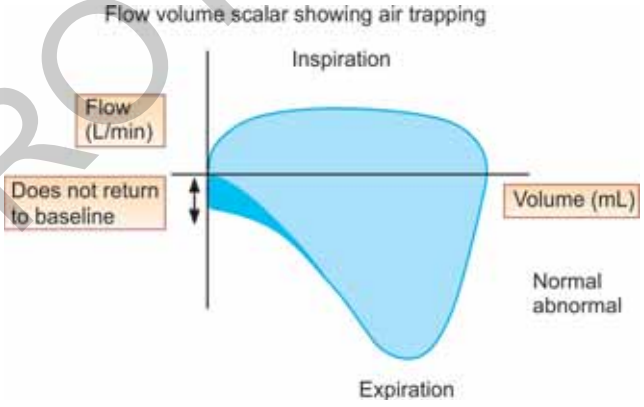

Fig. 2: Flow volume loop showing air trapping.

Table 2: Initial ventilator settings in severe asthma.

Initial ventilator settings	
Mode	Assist control
Tidal volume	6–8 mL/kg
Respiratory rate	12–14 breaths/min
Inspiratory flow	60–70 liters per minute
Waveform	Decelerating or constant
PEEP	<5 cmH_2O
FiO_2	Target a saturation of >90%

PEEP: positive end-expiratory pressure

Peak Airway Pressures (P_{peak})

Monitoring peak airway pressures are routinely done and easy to monitor. However, we need to understand its limitations with such patients.

- Peak airway pressures depend upon the resistive properties of the airway to inspiratory flow and may not be true representative of actual hyperinflation.

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Table 3: Ventilatory adjustments based on pH and/or P_{plat}.

Subsequent ventilatory targets	
Parameters	Ventilator adjustment
P_{plat} > 30 cmH_2O	Decrease minute ventilation (decrease rate)
If blood pH < 7.2 and P_{plat} < 25 cmH_2O	Increase minute ventilation (increase rate)
If blood pH < 7.2 and P_{plat} 25–30 cmH_2O	Consider to add buffers to counter effects of acidosis

- There is very limited evidence to suggest a direct relationship between peak airway pressures and barotrauma.
- Initial peak airway pressure in asthmatic patients may be ≥50 cmH_2O. A factor which may be contributing to high-peak airway pressures is the high inspiratory flow which is used in these patients.
- Moreover, decrement in P_{peak} may not even represent the decrease in DH with prolonged expiratory time due to increased airway resistance and decreased lung volume.

Plateau Airway Pressures (P_{plat})

Monitoring plateau pressures may be better than peak airway pressures. The reasons are explained below:

- Patients with severe asthma have near-normal respiratory system compliance and an increase in P_{plat} may represent DH. Hence, if the patient is being ventilated at constant tidal volume, changes in the amount of DH in response to bronchodilator therapy or manipulation of I:E ratio can be due to changes in P_{plat}.
- No safe limit of P_{plat} has been described in literature but the usual practice is to keep it between 25 cmH_2O and 30 cmH_2O (Lung protective ventilation).
- One should be aware that P_{plat} measured on the ventilator is representative of average of all alveolar pressures. It is possible that some alveolar units may have higher alveolar pressures than the average.
- Measuring auto-PEEP in a mechanically ventilated patient may provide just an estimate of end-expiratory alveolar pressures, thereby representing plateau pressures.

Measurement of Auto-Positive End-expiratory Alveolar Pressure[11,12]

There is no ideal technique to measure auto-PEEP in a ventilated patient. The method of estimation of auto-PEEP varies from an actively breathing patient to a passively ventilated patient. PEEPi depends on factors other than respiratory mechanics of the patient which includes respiratory rate, tidal volume, position of the patient, and it keeps on changing in response to the adjunct therapies and also during the course of the disease. Still no consensus exists on the optimal method for its measurement. We will be discussing the common techniques practiced for its measurement.

Measurement of Static Auto-Positive End-expiratory Alveolar Pressure

End-expiratory occlusion technique (EEOT): This technique requires the occlusion of the airways at the end of exhalation. The difference between end-of-occlusion Paw and pre-occlusion Paw is known as static PEEP (Fig. 3) and represents the average auto-PEEP of a nonhomogeneous lung with different time constants of different lung units.

Timing of occlusion is important and it should begin just before the next inspiratory flow of the ventilator is about to begin. If it is applied too early then there is a chance of overestimation of auto-PEEP. Since occlusion at exhalation port compresses the gas within the ventilator tubings and the humidifier it can give falsely low values of auto-PEEP. As a rule shorter tubings should be used and the humidifier should be removed during measurement of auto-PEEP with this technique.

Measurement of Dynamic Auto-Positive End-expiratory Alveolar Pressure

It is measured as the increase in Paw from end-exhalation to the point corresponding to the beginning of inspiratory flow. The concept is that rise in Paw preceding inspiratory flow is equal the pressure required to counter the elastic recoil of the respiratory system. Theoretically this measurement should represent the lung units with the shortest time constants, meaning thereby the lung units which empty at the earliest. *Hence, there is a possibility that this technique can underestimate auto-PEEP.*

Measurement of both static and dynamic auto-PEEP can give an estimate about the severity of time constant inequalities existing in the respiratory system of the patient.

Measurement of Auto-Positive End-expiratory Alveolar Pressure by Esophageal Pressure (Pes) Change Predicting the Inspiratory Flow (Also Known as Counter-Balance Method)

This technique requires inserting an esophageal balloon catheter system and it estimates the changes in pleural pressure. It is invasive technique with limitations and

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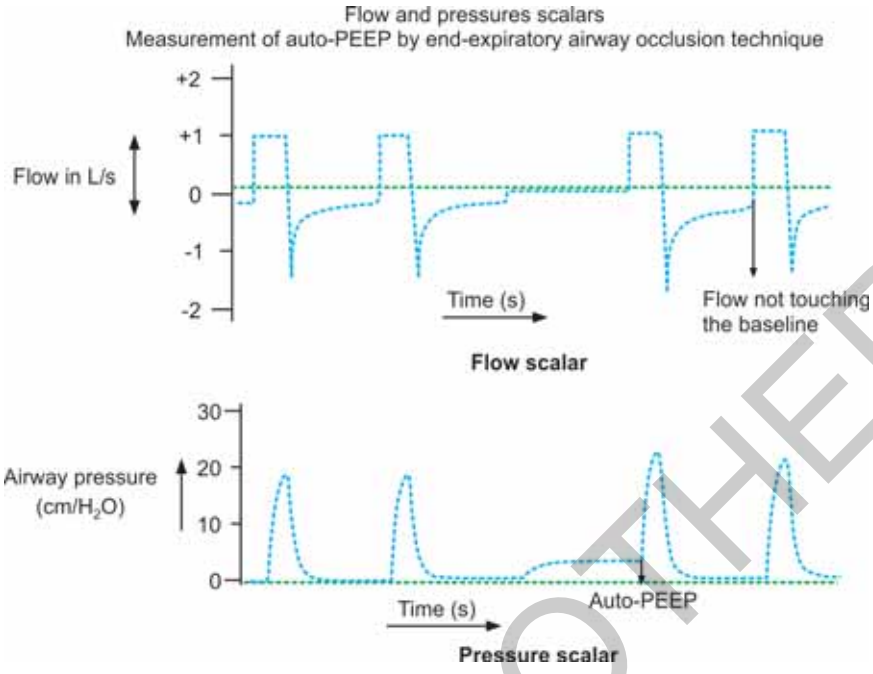

Fig. 3: Flow and pressures scalars. Measurement of auto-PEEP by end-expiratory airway occlusion technique.

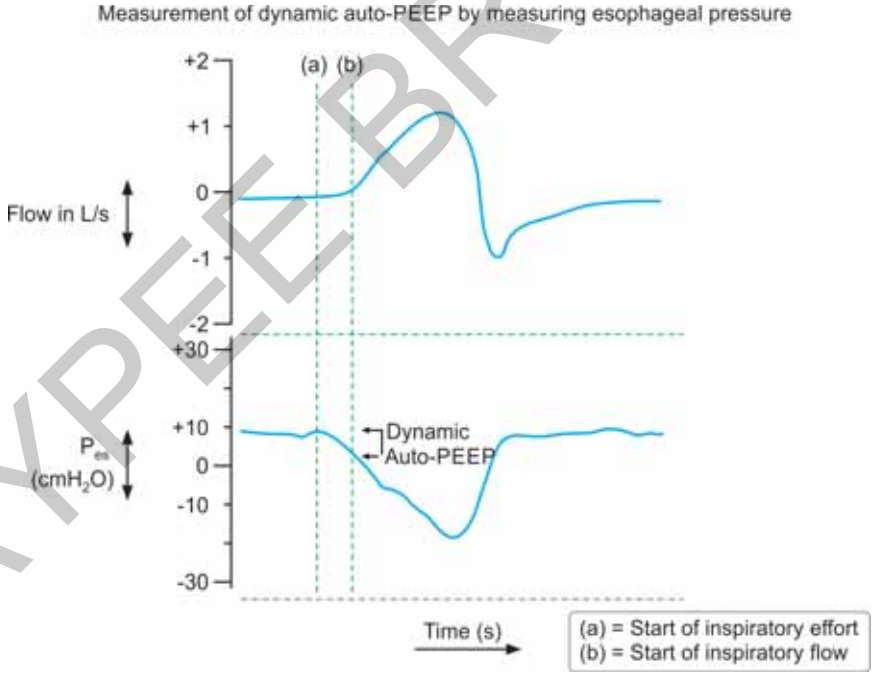

Fig. 4: Method of measuring dynamic auto-PEEP (difference between start of inspiratory effort as measured by esophageal pressure, an indirect measure of pleural pressure and start of inspiratory flow).

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also not readily available in most of the ICUs and has not become the standard practice for measuring auto-PEEP.

It measures the dynamic auto-PEEP as change in esophageal pressure from the initiation of patient's inspiratory effort to the beginning of inspiratory flow (Fig. 4). It measures the inspiratory muscle pressure which is required to offset the end-expiratory recoil of the respiratory system. This technique is based on certain *assumptions* which add to the limitations of this technique.

- End-expiratory alveolar pressure represents the elastic recoil pressure when the respiratory system is relaxed.

- Changes in esophageal pressure from the beginning of inspiratory effort to start of inspiratory flow reflects the inspiratory muscle pressure which is required to counter the elastic recoil of respiratory system at the end of exhalation.
- Time constant inequalities do exist.
- It means that PEEPi represents the pressure to start inspiratory flow in alveolar units with short time constants.
- Exhalation is passive.
- It assumes that at the end of exhalation there is absent expiratory muscle contraction.

Selection of Positive End-expiratory Pressure

The optimal PEEP should be the PEEP that does not harm the patient and at the same time it is able to balance the auto-PEEP. The waterfall theory explains that increasing the pressure away from the small airway closure or collapse should not cause any decrement in expiratory flow until the downstream water (external positive end-expiratory pressure) reaches the critical pressure (Fig. 5). Hence, it is advisable that the external PEEP should be kept below 75–85% of auto-PEEP. It would lessen DH and avoid circulatory compromise.

CHRONIC OBSTRUCTIVE PULMONARY DISEASE

The pathophysiology of COPD patients is similar to asthmatic patients with some extra considerations which are described below.[13,14]

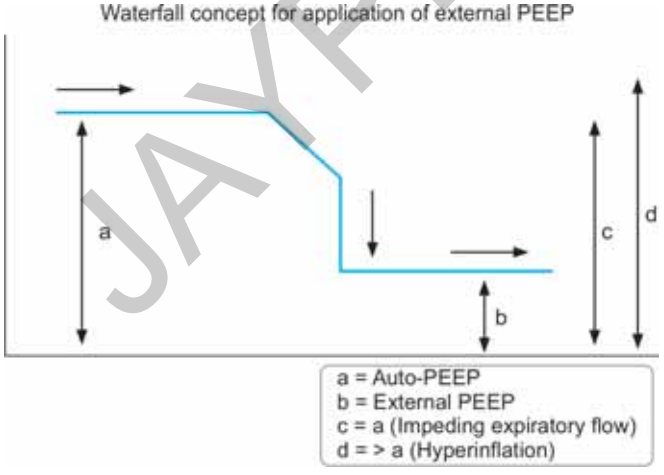

Fig. 5: Represents the water fall concept for determination of PEEP requirement during mechanical ventilation.

Some features associated with respiratory mechanics with these patients include:

- Increased inspiratory airway resistance
- Increased expiratory airway resistance
- Respiratory muscle weakness
- Respiratory muscle fatigue.

Chronic obstructive pulmonary disease patients have greatly increased expiratory resistance. The mechanisms are listed below:

- Resistance is more at low lung volume due to dynamic narrowing of small airways during exhalation
- "Wave speed limitation"—tracheobronchial tree cannot adjust to airflow resulting in expiratory flow limitation (EFL)
- Small airway collapse at critical closing pressures
- EFL exists in as many as 60% of stable patients even during resting breathing
- The consequences of EFL include
- EFL → Air trapping → Activation of expiratory muscles → Increases alveolar pressure
- Expiratory flow limitation seen in these patients is a condition that occurs because gas flow cannot be increased any more by:
  - Raising alveolar pressures
  - Reducing the airway opening pressure.

Consequences of Expiratory Flow Limitation[15,16]

- Expiratory muscle contraction—do not achieve storage of elastic energy at end of exhalation
- Relaxed expiratory muscles at the onset of inhalation do not assist inspiratory muscles in expanding the respiratory system during inhalation
- Entire burden of breathing is borne by inspiratory muscles
- Expiratory muscle recruitment dissipates precious energy substrate.

Breathing at low respiratory rates is recommended for hyperinflated lungs to improve their gas exchange. Hence, it is recommended that for these patients the most effective combination of respiratory rate and the ratio of inspiratory time divided by the total respiratory cycle (IT/TRC) should be used.

The ability of the respiratory muscles to sustain an increased inspiratory load in these patients depends on two ratios:

1. Respiratory duty cycle, which is equal to inspiratory time divided by the time of a total respiratory cycle (IT/TRC) and

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2. Mean transdiaphragmatic pressure per breath divided by the maximum static transdiaphragmatic pressure (MTDP/Mx STDP).

When we combine the above two ratios we get tension time index (TTI) which give us an idea regarding mechanical load, utilization of energy during the process of breathing, blood flow, and susceptibility of muscles to get fatigued.

IT/TRC × MTDP/Mx STDP = tension–time index (TTI)
TTI > 0.15 = Task failure (Healthy subjects TTI = 0.02)

Stable COPD patients have TTI = 0.05 which extrapolates to more than 2 times the phrenic motor neuron discharge and almost 5 times greater diaphragmatic recruitment at resting breathing.

The main goal of mechanical ventilation in patients with obstructive airway disease is to facilitate pulmonary gas exchange and allowing sufficient rest to compromised respiratory muscles. Ventilating a COPD patient is often difficult because the disease may not have a reversible component. However, adequate planning of ventilation strategy with modifications based on response to adjunct therapies can influence the overall outcome of these patients.

BRONCHOPLEURAL FISTULA

Bronchopleural fistula (BPF) in patients who have not undergone thoracic surgery is an infrequently encountered complication in intensive care units.[17,18] Ventilatory management of such patients is a challenge for the intensivist. The reported mortality in patients with BPF can be anywhere between 18% and 50%, with tension pneumothorax and aspiration pneumonia as leading causes of death. A patient of BPF developing ARDS becomes all the more difficult case to manage on ventilator.

Definition and Classification

Bronchopleural fistula is defined as leakage of inspired air from the airways into the pleural cavity persisting for more than 24 hours. Classification according to timing

- Early (1–7 days)
- Intermediate (8–30 days)
- Late fistulas (more than 30 days)
- PAL (persistent air leak)—if ≥5–7 days.

Pathophysiology

"Patients do not die because of BPF/they die with BPF."

Anatomically BPF is a sinus tract between the main bronchi, lobar bronchi, or segmental bronchus, and the pleural space. Acute BPF can be life-threatening due to clinical manifestations of tension pneumothorax or asphyxiation from pulmonary flooding of secretions. Clinical features may include sudden onset of dyspnea, hypotension, subcutaneous emphysema, expectoration of purulent fluid, tracheal or mediastinal shift, persistent air leak, and a reduction or disappearance of pleural effusion on the chest radiograph. The mechanisms for BPF in nonsurgical patients are enumerated below (Table 4).

In routine clinical practice, the severity of the air leak can be classified into three categories (from least to most severe):

- Air bubbling present during inspiration only
- Air bubbling present during both inspiration and expiration
- Air bubbling present during both inspiration and expiration with a measurable difference in the inspired and expired tidal volumes.

Diagnosis

Diagnosis of BPF can be based on:

- Clinical (pneumothorax with chest tube)
- Fiberoptic bronchoscopy
- Selective bronchography
- CT-scan to diagnose the site/size/etiology of BPF
- Methylene blue test
- Ventilation scintigraphy (postsurgical cases).

The harmful effects of BPF should be first understood which aid in optimizing the ventilatory strategy for these patients (Table 5).

Table 4: Etiologies of bronchopleural fistula (BPF) in ICU settings.

Mechanisms for BPF in ICU settings	
Spontaneous rupture	Direct injury
Volutrauma	Chest trauma
High PEEP therapy/High plateau pressures	Difficult airway with multiple intubation attempts
Severe ARDS	Central line placement
Necrotizing pneumonia	Thoracocentesis
Right main bronchus intubation	Chest tube placement
Pneumonectomy	Transbronchial biopsy
Manual ventilation	

(ARDS: acute respiratory distress syndrome; PEEP: positive end-expiratory pressure)

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Table 5: Harmful effects of bronchopleural fistula.

Effect	*Problem*
Incomplete lung expansion	Atelectasis and V/Q mismatch
Loss of tidal volume	Decreased minute ventilation/ respiratory acidosis
Loss of PEEP	Hypoxemia
Secretions seep into pleural space	Pleural space infections
Factitious triggering	Asynchrony/inadequate ventilation

(PEEP: positive end-expiratory pressure)

General Management Principles

In general, management of the patient with a bronchopleural fistula is the same as if the fistula were not there.

- Use the lowest number of mechanical breaths (it helps to decrease both mean airway pressure and number of high pressure breaths).
- Partial ventilatory support is preferred to total ventilatory support [pressure support ventilation/assist control mode of ventilation/synchronized intermittent mandatory ventilation-pressure support (PSV/ACV/SIMV-PS)].
- Limit the effective tidal volume based on permissive CO_2 levels.
- Minimize the inspiratory time by keeping low inspiration to expiration ratio (1:2).
- Use high inspiratory flow.
- Avoid pause at the end of inspiration.
- Avoid inverse ratio ventilation.
- Use low compressible-volume ventilator circuit to minimize the delivered tidal volume.
- Minimize PEEP (Best PEEP for acceptable oxygenation).
- Monitor auto-PEEP (Take measures to reduce auto-PEEP).
- Explore positional differences in BPF leak. Use the position with least leak.
- Treat other causes of expiratory flow obstruction.

Specific Strategies for Management of Bronchopleural Fistula

Bronchopleural fistula can usually be managed by conventional ventilation. The need for special ventilation techniques is uncommon.

- Chest tube manipulation (Theoretical with few anecdotal reports)
  - Intermittent inspiratory chest tube occlusion
  - Application of intrapleural pressure at expiration
- One lung ventilation
- High frequency ventilation
- Extracorporeal oxygenation.

Chest Tube Management

A short wide-bore chest tube should be used to achieve the best results when a large air leak is present.

- Small negative pressures can be applied (up to 10 cmH_2O) but we should be aware that it
  - Can increase fistula
  - Can lead to autotriggering
- A specialized pleural drainage unit (PDU) should be used to provide suction and a water seal to prevent backflow of air into the pleural cavity
  - The traditional PDUs regulate suction pressures by the height of the water column (keep a watch on Ht. of column)
- Newer-generation "dry" suction units are also available now.

Preventive Strategies for Bronchopleural Fistula

- Use lung protective ventilation (low tidal volume strategy)
- Monitor for auto-PEEP
- Monitor the static respiratory system compliance (tidal volume/plateau airway pressure-PEEP)
  - Decrease applied PEEP if the static compliance decreases when the applied PEEP is increased.
- Separate the patient from mechanical ventilation as soon as possible
- Extreme care when inserting a subclavian or internal jugular central venous catheter, or performing thoracentesis.

SEVERE METABOLIC ACIDOSIS

Critically ill patients are prone to encounter disturbances of acid–base equilibrium. Acidosis can be either respiratory due to increases in arterial partial carbon dioxide tension (PCO_2: respiratory acidosis) or metabolic due to accumulation of a variety of organic or inorganic, fixed acids. Understanding the pathophysiology is crucial for proper management. Though metabolic acidosis is not uncommon in patients admitted in intensive care unit, the precise incidence and prevalence of metabolic acidosis has not been established. Often acid–base disorders are diagnostic markers of some underlying pathology. The true cause–effect relationship between acidosis and adverse clinical outcomes remains uncertain for critically ill patients. Clinical manifestations of severe

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acidemia can include cerebral edema, seizures, diaphragm dysfunction, poor cardiac function, increased pulmonary vasoconstriction, and decreased systemic vascular resistance manifesting as hypotension.

Metabolic acidosis is defined as an arterial blood pH of less than 7.35 with plasma bicarbonate levels of less than 22 mmol/L. Respiratory compensation is the first to occur and normally starts immediately. Compensation can be partial (very early in time course) or full compensation bringing back the pH to normal. Winter's formula can be helpful to calculate the CO_2 compensation for the existing metabolic acidosis—(the formula allows calculation of the expected compensating pCO_2). If the measured pCO_2 is more than what is expected for that metabolic acidosis then coexisting respiratory acidosis may also be present in the same patient. Clinically the patients experience dyspnea due to stimulation of the respiratory center in an attempt to "blow off" CO_2 and thereby increasing the blood pH. Sometimes they may not have significant tachypnea but instead they hyperventilate (large tidal volumes) to compensate for their pH (Kussmaul's breathing).

Physiological Effects of Hyperventilation may Influence Ventilator Settings

- Hemodynamic disturbances
- Asynchrony
- Dyssynchrony
- Increased work of breathing (WOB)
- Predisposes to barotrauma
- Interferes with respiratory mechanics
- Dynamic hyperinflation.

Intubation and ventilation can lead to decompensation of physiological compensations and hence caution is advised at initiation of mechanical ventilation.[19] Patients with a metabolic acidosis rely on their hyperventilation to maintain their pH. Hence, it is advised to *maintain hyperventilation in such patients* till we provide therapy or the etiology of acidosis.

If ventilation is set to some standard value and the pCO_2 is allowed to rise toward 40 mm Hg, then this represents further worsening of acute respiratory acidosis and pH can fall rapidly. Anesthetic agents and initial ventilator settings to a normal respiratory rate may lead to further decompensation. The target of mechanical ventilation is to keep the pH in the normal range (7.35–7.45). Initiation of mechanical ventilation in these patients should be with adequate precautions and knowledge about heart–lung interactions which can occur after initiation of positive pressure ventilation.

Factors that may Worsen the Hemodynamic Effects of MV in Acidotic Patients

- Decrease in venous return (in situations of hypovolemia, venodilation)
- Increase in mean intrathoracic pressure (e.g. the use of large tidal volume or use of high PEEP)
- Blunting of compensatory sympathetic reflexes (e.g. sedatives).

ACUTE RESPIRATORY DISTRESS SYNDROME

Acute respiratory distress syndrome is a disease in which the lungs suffer severe heterogeneous injury, interfering with their ability to take up oxygen. It is the final common pathway of lung damage caused of a variety of insults ranging from direct causes like aspiration pneumonia, inhalational injury, etc. to indirect causes like cardiopulmonary bypass and disseminated infection of extrapulmonary origin.

Definition

Acute respiratory distress syndrome was first defined in 1994 during the American-European Consensus Conference (AECC) as:[20]

- A condition with acute respiratory failure
- Bilateral infiltrates on chest radiology
- PaO_2/FiO_2 ratio ≤200 mm Hg
- No evidence of left atrial hypertension or a pulmonary capillary pressure <18 mm Hg (if measured).

Acute lung injury (ALI) was less severe form of ARDS with $PaO_2/FiO_2 \leq 300$ mm Hg.[19] During the year 2011, a revised definition of ARDS was given by European Society of Intensive Care Medicine with endorsement from the American Thoracic Society (ATS) and the Society of Critical Care Medicine (SCCM) to address the limitations of the above definition.[21] The term acute lung injury was dropped and ARDS was classified on the basis of PaO_2/FiO_2 as mild (200 mm Hg to 300 mm Hg), moderate (100 mm Hg to 200 mm Hg), and severe ($PaO_2/FIO_2 \leq 100$ mm Hg). Other salient features of Berlin definition included acute onset respiratory distress as new worsening within 1 week, bilateral chest infiltrates NOT fully explained by lung pathologies like effusion, nodule or collapse and pulmonary edema, fluid overload or heart failure.

Pathophysiology of ARDS

The affected areas in ARDS are the alveoli (air sacs). The thin wall between the blood and air (alveolar-capillary membrane—less than 0.5 micrometers in width) is at its

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thinnest segment where the gas exchange takes place. ARDS pathology involves both the capillary and alveolar cells are injured. This injury results in fluid spillage into the alveolar units, thus hindering or preventing gas exchange.[22] Regardless of the triggering event, an inflammatory chain reaction is set off. Molecules released by infected or injured cells attract white cells from the blood to enter the affected area. The incoming white blood cells combine with resident lung cells to produce more chemicals (called cytokines and chemokines), thereby exaggerating the inflammatory process. The inflammatory exudates decrease the surfactant function leading to collapse/consolidation of distal airspaces subsequently leading to loss of gas exchange. This would normally be compensated for by hypoxic pulmonary vasoconstriction, if the inflammatory process did not also effectively paralyze the vascular tone thereby allowing deoxygenated blood to flow through unventilated lung units. The combination of these two processes initially causes hypoxemia but if allowed to persist eventually leads to type 2 respiratory failure with CO_2 retention.

From mechanical ventilation point of view, ARDS comprises of heterogeneous lung with different ventilatory requirements in different zones of lungs. These patients have some normal alveoli, some consolidated while some alveoli recruitable with ventilatory strategies.

Ventilatory Management of ARDS

Management of these patients consists of appropriate treatment of the underlying or causative illness, supportive care, and prevention of complications associated with mechanical ventilation. Appropriate treatment of an underlying infection consists of identifying the causative organism as best as possible and treated with appropriate/sensitive antibiotics. Mechanical ventilation is a key component of holistic supportive care and in this chapter we highlight the important principles of mechanical ventilation in patients with ARDS.

Last few decades have highlighted the injurious effects of mechanical ventilation. In the 1970s, ventilator settings were titrated to normalize blood gas values. Clinicians used tidal volume of 12–15 mL/kg of body weight. Barotrauma in the form of pneumothorax, pneumomediastinum, and pneumoperitoneum were common, and mortality in severe ARDS was as high as 90%. In order to reduce mortality associated with ARDS and design optimal management strategy, the National Heart, Lung, and Blood Institute (NHLBI), National Institutes of Health (NIH), conducted multiple multicenter clinical trials from 1994 to 2014. Over this period of two decades almost 10 randomized controlled trials and one observational study were conducted. Out of these 11 studies showed that lung protective ventilation strategy was associated with improved outcome and decreased mortality.

Lung Protective Mechanical Ventilation

Work by Amato and colleagues and later the landmark ARDSNet (Acute Respiratory Distress Syndrome Network) trial in 2000 showed improved outcomes with low tidal volume (TV) ventilation strategies [4–6 mL/kg of ideal body weight (IBW)] versus higher traditional TV (10–12 mL/kg IBW).[23,24] A better understanding of ventilator-induced lung injury (VILI) in recent years has created a renewed interest on practicing lung protective ventilation strategy. Recent data favor low tidal volume ventilation not only in ARDS but in patients without ARDS also.

It incorporates two major components:

1. Low tidal volume ventilation (4–6 mL/kg of predicted body weight)
2. Limiting the plateau pressures to less than 30 cmH_2O.

The recommendation to use lower tidal volume (less than or equal to 6 mL/kg predicted body weight) ventilation with a plateau pressure less than or equal to 30 cmH_2O is strong despite moderate quality of evidence for hospital mortality and barotrauma, and low quality of evidence for 60 day mortality. Additional studies and meta-analysis have showed large tidal volume ventilation and high pressures are harmful.[25-27] However, lack of adverse effects, strong mechanistic and physiologic rationale for its use and supportive data from ARDS prevention studies have resulted in *its universal acceptance as a gold standard of care*.

There is no safe plateau pressure in ARDS. However, the recommendations are to keep them less than 30 cmH_2O. Some experts believe it should be kept as low as 20 cmH_2O. It has been chosen as a surrogate for transpulmonary pressures because measuring transpulmonary pressures is not clinically possible. Methods to reduce plateau pressures are to work on low tidal volumes but one should be cautious regarding $PaCO_2$ and pH (try keeping these parameters in acceptable range). This is one more reason to choose high respiratory rates in ARDS patients to maintain minute ventilation.

PEEP Selection in ARDS

The treatment for hypoxemia in ARDS is to recruit collapsed lung alveolar units by recruiting them through use of external PEEP. The optimal PEEP settings, or even the best method to go about choosing PEEP, are controversial.

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The ARDSnet trial also provides algorithms for bedside selection of PEEP based on FiO_2 but still it is best left to the treating clinician to choose the right PEEP for each individual patient.

The use of relatively higher PEEP in patients with moderate or severe ARDS did not get support from high-quality evidences, as there is inconsistency among studies for strategy to set the level of PEEP. The evidences favoring use of higher PEEP in patients with at least moderate ARDS is based on subgroup and individual patient data, rather than the evidence based on a randomized controlled trial investigating higher PEEP in this group of patients. Also, the risk of barotrauma with the use of higher PEEP in moderate or severe ARDS is not quantified very well in studies. The quality of evidence to quantify risks' of high PEEP is also limited by inconsistency as the meta-analysis has included trials of high PEEP with different tidal volume strategies. There is no universal consensus as of now regarding the methodology used to optimize PEEP or to manage the respiratory acidosis that can be frequent accompaniment of lung protective ventilation. However, the suggestions for clinical practice are to use of high PEEP strategies for patients with moderate/severe ARDS.[28]

Default PEEP setting in ARDS can be kept at 10 cmH_2O. Patients likely to benefit from PEEP above 10 cm are the ones with reduced chest wall compliance and those with airway and alveolar edema that can be easily redistributed. It should be noted that edema fluid can be more easily redistributed only in early phase of inflammation as in latter stages it gets converted to gel state. With further progression of ARDS there is remodeling and fibrosis.

Plateau airway pressure is useful only when it is able to recruit more lung units otherwise it can cause harmful effects like decreased venous return, increased intrathoracic pressure, decreased cardiac output, and ventilation perfusion mismatch.

Prone Ventilation

Prone ventilation is one of the strategies to improve oxygenation in ARDS patients. Edema fluid redistribution and recruitment of alveoli are most easily achievable in early phase of the disease and that is why prone ventilation works best in initial phase.

Current evidence regarding prone ventilation favors survival outcome when combined with lung-protective ventilation and when delivered for minimum 12 hours to patients with moderate/severe ARDS.[29,30] The most recent and well-conducted randomized study (PROSEVA trial) focused on severe ARDS, and involved a multimodal intervention comprising lung-protective ventilation with prolonged-duration prone positioning resulting in significant reduction in short as well as long-term mortality.[31] This study provides the strong rationale for practicing prone ventilation.

The possibility for substantial patient benefit must be considered despite possibilities of prone position-related adverse events (facial edema, potential dislodgement of invasive lines, pressure sores, and ocular complications); as prevalence of these adverse events are either low or very low. However, it should be ensured that sufficient skilled personnel should deliver this intervention.

Recent data by the PROSEVA group examined prone positioning in severe ARDS (PaO_2/FiO_2 ratio < 150 mm Hg) and found an overall reduction in 28-day mortality (16.0% vs 32.8%) without any increase in complications in the prone group.[31] Though the investigators who participated in this trial had significant experience with prone positioning that could have minimized complications (airway and central line dislodgment, pressure ulcers).

The recommendations for clinical practice are to use prone positioning for at least 12 hours per day in patients with moderate/severe ARDS.

Recruitment Maneuvers

Increased lung weight due to interstitial and alveolar edema can cause collapse of dependent alveolar units.[32,33] Recruitment maneuvers (RMs) are used to achieve recruitment of collapsed alveolar units by transiently increasing applied airway pressure and driving pressure (Plateau airway pressure—PEEP). Various RMs have been described in literature including progressive incremental PEEP at constant driving pressure, continuous positive airway pressure (30–40 cmH_2O for 40 seconds), etc. RMs cause transient improvement in oxygenation and lung compliance in patients with recruitable alveolar units. These maneuvers are potentially dangerous in patients with shock and fluid deficit as they can worsen hemodynamic compromise. Current guidelines suggest use of RMs in ARDS patients. There are no recommendations for the best RM in ARDS. The clinicians can practice the maneuver they are best versed with.

Extracorporeal Membrane Oxygenation

Extracorporeal membrane oxygenation (ECMO) is a modality of oxygenating the blood via an extracorporeal circuit. Blood is drawn from the patient via large bore catheter placed in artery or vein, passed through an oxygenator, and returned back into a large central vein.

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Table 6: Outline for management of ARDS.

Mild ARDS	*Moderate ARDS*	*Severe ARDS*
200 mm Hg < PaO_2/FIO_2 < 300 mm Hg PEEP or CPAP ≥ 5 cmH_2O	100 mm Hg < PaO_2/FIO_2 < 200 mm PEEP ≥ 5 cmH_2O	PaO_2/FIO_2 < 100 mm Hg PEEP ≥ 5 cmH_2O
Conservative fluid strategy for all grades of ARDS		
Low tidal volume ventilation with Plateau pressures less than 30 cmH_2O for all grades of ARDS		
	Prone ventilation for >12 hours/day	
	PEEP optimization/recruitment (Preferred high PEEP ≥ 10 cmH_2O)	
		Referral to ECMO center

(ARDS: acute respiratory distress syndrome; ECMO: extracorporeal membrane oxygenation; PEEP: positive end-expiratory pressure)

Extracorporeal support techniques were extensively used in 2009 H1N1 pandemic with promising results.[34] However, current evidence in this field is limited. More robust data is needed before strong recommendations can be made regarding ECMO use in ARDS. Current recommendation for ECMO is that patients with severe ARDS to be referred to ECMO center with trained staff (also see chapter on ECMO).

Extracorporeal Carbon Dioxide Removal

It is still at research level and should be the subject of a suitably powered multicenter randomized controlled trial (RCT) with long-term follow up and economic analysis, that focuses on both potential benefits and harms (also see chapter on $ECCO_2R$).

High Frequency Oscillatory Ventilation

The use of HFOV for the management of ARDS has a poor recommendation based on moderate quality evidence. Current evidence from multiple RCTs demonstrated no benefit from HFOV and one RCT even demonstrated an increase in mortality with HFOV (also see Chapter on HFOV).

Our knowledge regarding mechanical ventilation for ARDS has evolved significantly over last few decades. Lung protective ventilation, prone ventilation, restrictive fluid strategy, optimal PEEP and RM are the cornerstone of ventilating ARDS patients (Table 6).

SALIENT POINTS

- Lung protective ventilation is the key in any form of ventilator strategy, following the principle of "do no harm".
- Low tidal volume ventilation (VT: 6–8 mL/kg) targeting P_{plat} < 30 cmH_2O should be the target in all patients.
- Full support ventilation is initially provided targeting adequate minute ventilation and avoiding hyperoxia (Target: SpO_2: 88–92%; PaO_2: 70–100 mm Hg).
- Analgosedation is to be provided to maintain patient's synchrony with the ventilator. Daily sedation vacation should be practiced after initial stabilization.
- Neuromuscular blockade should be considered in select cases but should be withdrawn early.
- After initial management, subsequent changes in mechanical ventilation settings should be based on clinical condition and regular monitoring of arterial blood gases.

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- Useful for trainees as well as consultants of various clinical specialties who manage critically ill adult patients, including critical care medicine, emergency medicine, pulmonary medicine, and anesthesiology.

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