



Postintubation Hypotension

- The time immediately after intubation can be difficult for the patient with severe airflow obstruction.
- Immediate concerns are hypotension and pneumothorax. Hypotension has been reported in 25% to 35% of patients after intubation. It occurs from a loss of vascular tone due to sedation, hypovolemia, tension pneumothorax, or overzealous ventilation. The latter results in dangerous levels of DHI when adequate time is not provided for exhalation.
- Clues to DHI include excessive effort during manual inflation, decreased breath sounds, hypotension, and tachycardia. A trial of hypopnea (2 to 3 breaths/min) or apnea in a preoxygenated patient is both diagnostic and therapeutic for DHI.
- Irrespective of clinical improvement, tension pneumothorax should be considered. Close inspection of the chest radiograph is mandatory because DHI may limit lung collapse. Because it causes preferential ventilation to the contralateral lung, unilateral pneumothorax increases the risk of bilateral pneumothoraces.

Ventilator settings:

- During mechanical ventilation, the expiratory time, tidal volume, and severity of airway obstruction determine the level of DHI.
- Because treatment of airway obstruction has been maximized in most intubated patients, expiratory time and tidal volume become important variables during ventilator management.
- Minute ventilation and inspiratory flow rates determine exhalation time. At a set inspiratory flow, a drop in minute ventilation prolongs expiratory time and decreases DHI.
- To avoid dangerous levels of DHI, initial minute ventilation should be less than 115 mL/kg/min or approximately 8 L/min in a 70-kg patient. This can be achieved with a respiratory rate between 12 and 14 breaths/min combined and a tidal volume between 7 and 8 mL/kg.
- Shortening the inspiratory time by use of a high inspiratory flow rate also prolongs expiratory time. Use an inspiratory flow rate of 80 L/min, using a square or constant flow regimen. High inspiratory flow rates increase peak airway pressure by elevating airway resistive pressure, but peak airway pressure does not correlate with morbidity or mortality. Rather it is the state of lung hyperinflation that predicts outcome, and any ventilator strategy that lowers peak airway pressure shortens expiratory time and worsens DHI.

Assessing Lung Inflation

- Determination of the severity of DHI is central to risk management and adjustment of ventilator settings. Numerous methods have been proposed to measure DHI.
- The volume at end-inspiration, termed VEI, is determined by collecting expired gas from total lung capacity to functional residual capacity during 40 to 60 seconds of apnea. A VEI greater than 20 mL/kg has been correlated with barotrauma.
- Alternate measures of DHI include the single-breath plateau pressure (Pplat) and auto-PEEP.
- Pplat is an estimate of average end-inspiratory alveolar pressures that is determined by stopping flow at end-inspiration.
- Auto-PEEP is the lowest average alveolar pressure achieved during the respiratory cycle. It is obtained by measuring airway-opening pressure during an end-expiratory hold maneuver.
- In the presence of auto-PEEP airway-opening pressure increases by the amount of auto-PEEP present. Persistence of expiratory gas flow at the beginning of inspiration (which can be detected by auscultation or flow tracings) also demonstrates auto-PEEP.
- experience suggests that when Pplat is less than 30 cm H_2O the outcome is generally good.
- In most cases, however, auto-PEEP less than 15 cm H_2O is acceptable.

epidemiology

- According to consensus guidelines, a severe episode is believed to exist when one or more of the following features are present:
 - accessory muscle activity,
 - a paradoxical pulse exceeding 25 mm Hg,
 - a heart rate greater than 110 beats/minute,
 - a respiratory rate greater than 25-30 breaths/minute,
 - a limited ability to speak,
 - a peak expiratory flow rate (PEFR) or FEV1 <50% of predicted, and
 - an arterial saturation less than 91-92%
- Altered mental status, paradoxical respiration, bradycardia, a quiet chest, and absence of pulsus paradoxus from respiratory muscle fatigue are features of imminent respiratory arrest.

pathophysiology

- the classic gross anatomic features of those who die from asthma are airway narrowing, extensive plugging of the airways with mucus and inflammatory infiltrates, hyperinflation, and atelectasis
- increased airway resistance and dynamic hyperinflation leads to:
 - increased work of breathing
 - ventilation-perfusion mismatch as a result of airway narrowing & closure
 - adverse cardiopulmonary interactions
- spontaneously breathing patients with acute severe asthma can generate inspiratory pressures as low as -35cm H_2O which are sufficient to cause increased left ventricular afterload and promotes egress of fluid into alveolar airspace
- right ventricular afterload is increased by hypoxic pulmonary vasoconstriction, acidosis & increased lung volume
- patients with severe asthma exacerbation have critical airflow obstruction limiting exhalation.
- in severe cases, expiratory flow may not cease for as long as 60 seconds. Because expiratory time is shorter (1 to 5 seconds) during spontaneous or assisted breathing, there is incomplete emptying of gas and dynamic lung hyperinflation (DHI).
- Fortunately, DHI is self-limiting because as lung volume increases so do lung elastic recoil pressure and airway diameter-factors that favor expiratory flow.
- However, DHI places the diaphragm in a mechanically disadvantageous position

differential diagnosis

- Most patients suffering from acute asthma request therapy with a constellation of complaints consisting of **dyspnea, cough, and wheezing**.
- The physical signs that are encountered are tachypnea, tachycardia, wheeze, hyperinflation, accessory muscle use, pulsus paradoxus, diaphoresis, cyanosis, and obtundation
- Sweating, the use of accessory muscles, a paradoxical pulse, and the inability to communicate in full sentences are all associated with the presence of substantial airway narrowing
- In patients who die from acute severe asthma two patterns may be seen at autopsy. One in which there is significant inflammation with prominent bronchial mucus and one in which airway obstruction is predominantly due to intense smooth muscle contraction
- this is mirrored in the clinical presentations of episodes requiring intensive care:
 - hyperacute fulminating asthma
 - develops rapidly often preceding to intubation within a few hours of symptoms & tends to respond rapidly to bronchodilator therapy (occurs in younger patients with normal lung function but high bronchial reactivity)
 - acute severe asthma:
- progresses slowly over hours or days and responds more slowly to treatment
- differential diagnosis includes:
 - left ventricular failure
 - aspiration
 - upper airway obstruction
 - inhaled foreign body
 - pulmonary embolism

risk factors for fatal or near fatal asthma

Frequent emergency department visits
Frequent hospitalizations
Intensive care unit admission
Prior intubation
Hypertension
Eczema
Psychiatric illness
Medical noncompliance
Illicit drug use
Low socioeconomic status
Inadequate access to medical care
Use of more than two consecutive months of inhaled beta agonist
Poor perception of airflow obstruction
Comorbidities such as coronary artery disease
Sensitivity to allergenic species

prognosis

causes of death in 99 adults with acute severe asthma admitted to ICU

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Cerebral ischaemia/hypoxia	40
Hypotension	15
Pneumonia or sepsis with hypotension	10
Tension pneumothorax	6
Technical complications with ventilation	6
Arrhythmia	4
Respiratory complication (aspiration, tracheostomy, sputum plug)	3
Gastrointestinal complication	3
Suspected pulmonary embolus	3
Arrest post extubation	3
Arrest post ICU	2
Treatment withdrawn (endstage)	2
Unknown	3
TOTAL	99

therapy

- Established therapies include:
 - oxygen
 - beta agonists
 - anticholinergics
 - corticosteroids
 - aminophylline
- Non-established treatments:
 - adrenaline
 - magnesium sulphate
 - heliox
 - ketamine
 - bronchoalveolar lavage
 - hydration