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.

- 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
  - Increased airway resistance and dynamic hyperinflation
    - Spontaneously breathing patients with acute severe asthma can generate inspiratory pressures as low as -35 cmH2O 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 expiratory time, which may be 1 to 5 seconds during spontaneous or assisted breathing.

- Increased airway resistance results in dangerous levels of DHI when adequate time is not provided for exhalation.

- 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 eosinophilic exudate in the airways
  - 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:
  - Hypotension, tachycardia, wheeze, hyperinflation, accessory muscle use, pulsus paradoxus, diaphoresis, cyanosis, and obtundation

- The physical signs that are encountered are tachycardia, tachypnea, wheeze, hyperinflation, accessory muscle use, pulse paradoxus, diaphragm, cyanosis, and obtundation.

- The pathophysiology of asthma exacerbation includes:
  - 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
    - Increased airway resistance and dynamic hyperinflation
      - Spontaneously breathing patients with acute severe asthma can generate inspiratory pressures as low as -35 cmH2O 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.

- 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.

- In most patients suffering from acute asthma request therapy with a constellation of complaints consisting of dyspnea, cough, and wheezing.

- The differential diagnosis includes:
  - Left ventricular failure
  - Pulmonary embolism
  - Upper airway obstruction
  - Aspiration
  - Inhaled foreign body
  - Diaphoresis, cyanosis, and obtundation

- 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 VIE, is determined by collecting expired gas from total lung capacity to functional residual capacity during 40 to 60 seconds of apnea. A VIE greater than 20 mL/kg has been correlated with barotrauma.
  - 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 H2O the outcome is generally good.
  - Established therapies include:
    - Oxygen
    - Beta agonists
    - Anticholinergics
    - Corticosteroids
    - Aminophylline

- Non-established therapies include:
  - Adrenaline
  - Magnesium sulphate
  - Sodium bicarbonate
  - Aldosterone
  - Hydration

- Causes of death in 59 adults with acute severe asthma admitted to ICU.