**Obesity**

= essentially all related to reduction in FRC

**Uncomplicated Obesity**

- Even in the absence of specific pulmonary pathology there is restrictive pulmonary physiology
  - ↓ Lung volumes (TLC, VC, TV, FRC, ERV)
  - ↓ Compliance

- Due to
  - Weight of adipose tissue on chest wall
  - Cephalad displacement of abdominal contents
  - ↑ Intrathoracic blood volume

1) **Lung Mechanics**

- ↓ FRC
  - ↓ 1% for every unit that BMI > 30kg/m²
  - FRC 1L if BMI > 40, ie absolute reduction in FRC compared to non-obese, not just per kg
  - Atelectasis and shunting if FRC < CC
  - Accentuated by anaesthesia and supine position
  - Secretion retention
  - Rapid desaturation

- ↓ Lung compliance
  - Operating on lower, flatter part of the compliance curve due to ↓ lung volumes

- ↑ pulmonary blood volume → stiffer lungs

- ↓ Chest wall compliance
  - Due to weight of adipose tissue surrounding thoracic cage

- ↑ Airway resistance
  - Due to ↓ lung volumes
  - Normal when corrected for lung volume

- ↑ Work of breathing
2) **Ventilation**

- **↓ Tidal volume**
  - Normalised to TBW or LBW, TV is reduced by 50% and 20% respectively
- **↑ Respiratory rate**
  - Resting RR in morbidly obese is 40% higher than normal weight individuals

3) **Gas Exchange**

- **↑ O2 consumption, CO2 production and minute ventilation**
  - Due to ↑ metabolic requirements of greater LBM and excess adipose tissue
- **Impaired gas exchange**
  - ↑ V/Q mismatch - dependent part of lungs relatively over-perfused and under-ventilated
- **↓ FRC →atelectasis and shunting**
  - The above causes ↑ venous admixture →↑ A-a gradient

4) **Measurement**

- **↓ FEV1 and FVC**
- FEV1/FVC remains normal

5) **Obesity Related Pathology**

- **Obstructive sleep apnoea**
- > 5 apnoea episodes per hour (> 10 sec duration) due to pharyngeal collapse
  - Snoring
  - Daytime somnolence
- **Pathophysiological changes**
  - chronic hypoxaemia
  - secondary polycythaemia
  - chronic hypercapnia →↓ sensitivity to hypercapnic respiratory drive
  - systemic vasoconstriction
  - pulmonary vasoconstriction →right heart failure
- Sensitivity to effects of CNS depressant drugs
- Respiratory depression
- Risk of airway obstruction

- **Obesity hypoventilation**
- The presence of awake alveolar hypoventilation in an obese individual which cannot be attributed to other conditions associated with alveolar hypoventilation
  - 90% of patients with OHS also have OSA but they are discrete entities

- **Pathophysiological changes**
  - CO2 sensitivity and respiratory drive is partly under control of leptin
  - Relative insensitivity to leptin in obesity $\rightarrow$ ventilatory response to CO2
  - Hypoventilation is accentuated by depressant drugs e.g. anaesthetic agents, opioids

**Clinical Implications**

- Time to desaturation during apnoea
- Hypoventilation during supine, spontaneous breathing
- Low threshold for RSI, intubation, IPPV with PEEP