On intrapulmonary shunt

In stupid terms:
Shunt is the percentage of blood passing through the lungs which doesn't get oxygenated.

Normally its no more than 4%.

PEEP: Positive End Expiratory Pressure and its Consequences

PEEP is airway pressure artificially kept above atmospheric pressure. In the stupidest form of saying it, **PEEP IMPROVES OXYGENATION and REDUCES WORK OF BREATHING.**

**HOW PEEP IMPROVES OXYGENATION:**
- Increasing lung volume by recruiting collapsed alveoli (thereby reducing the intrapulmonary shunt)
- Pushes alveolar edema fluid out of the alveoli and into the interstitium

**HOW PEEP REDUCES THE WORK OF BREATHING:**
- Supplies the pressure required to overcome airway obstruction
- Supplies the pressure required to overcome Intrinsic PEEP

**EFFECTS OF PEEP ON PRELOAD:**

- Increased intrathoracic pressure, thus
  - Decreased venous return,
  - Thus reduced left ventricular stroke volume
  - Thus reduced left ventricular contractility
  - Thus reduced left ventricular oxygen demand
  - If the left ventricle is decompensating because it is overfilled and overstretched ("congestive" heart failure) the decreased preload will push it back into the more efficient area of the Frank-Starling curve.

**On Pressure**

For some reason, we measure gas partial pressure in mmHg, and ventilator gas pressures in cmH₂O.

1 mmHg = 1.36 cm of H₂O

Measured in cmH₂O, sea level pressure is 1033.

Thus, we ventilate our patients with pressure differences which are minute in comparison to the total pressures in play in the environment around them, or even to the pressures in their own cardiovascular system.

**Pressure**

**Flow**

**Volume**

From "Goodman & Gilman’s The Pharmacological Basis of Therapeutics" 11th ed by Brunton et al, and "Basic & Clinical Pharmacology" 11th ed. By Katzung et al
Effects of PEEP on Right Ventricular Afterload

- Increased intrathoracic pressure = increased pulmonary artery pressure, thus
  - Increased right ventricular afterload
  - Thus, increased right ventricular work and thus oxygen demand
  - With a crappy right ventricle, this could really impair the left ventricular function - the left ventricle depends on the right for filling.

Effects of PEEP on Left Ventricular Afterload

- Left ventricular afterload = sum of systemic arterial resistance and left ventricular transmural pressure.
- Increased intrathoracic pressure = increased transmural pressure
  
i.e. the pressure generated by the left ventricle =
  = (pressure generated by LV muscle) + (pressure added to it by the PEEP)

- In normal people, that contribution is tiny.
  - Remember, PEEP of, say, 10cmH2O is equal to 7.35 mmHg
  - In normal people, the left ventricle generates a systolic ejection pressure of about 80mmHg - and that’s just to open the aortic valve.

  - If your left ventricle is diseased, it can’t pump effectively against the afterload, and pulmonary oedema ensues.
    - If pulmonary oedema ensues, the lung volume and lung compliance drop.
    - If the lung volume and compliance drop, the respiratory effort must generate lower pressures to suck more air in
    - The lower pressures decrease the transmural pressure, and thus increase the relative afterload.
    - The increased afterload causes more pulmonary oedema.

  - Thus, by reducing the afterload-increasing effects of increased respiratory effort, PEEP it can’t pump effectively against the afterload, and pulmonary oedema ensues.

PEEP-induced lung injury

- If you over-PEEP, you end up distending the lungs and cause barotrauma
  - This results in broken capillaries and increased alveolar-capillary permeability
  - The alveoli flood with fluid, and inflammatory mediators are released by the dying cells, resulting in more oedema, and, stupidly, increased PEEP requirements.

- If you under-PEEP, the atelectatic alveoli will open and close every breath cycle
  - The constant opening and closing of intermittently atelectatic alveoli will cause shear stress, and this also cause the release of inflammatory mediators.

PEEP-related organ effects

- Reduced urine output due to decreased cardiac output, if already volume depleted
- Decreased splanchnic blood flow due to decreased cardiac output, etc etc
- Increased hepatic venous congestion due to decreased venous return to the heart
- Increased intracranial pressure due to decreased venous return to the heart
- Decreased peribronchial lymphatic flow could actually decrease the rate of oedema removal, not to mention clearance of necrotic debris in pneumonia.

From “Goodman & Gilman’s The Pharmacological Basis of Therapeutics” 11th ed by Brunton et al. and “Basic & Clinical Pharmacology” 11th ed. By Katzung et al