Applied respiratory physiology

1. Pregnancy

The following anatomical changes are associated with pregnancy

- Capillary engorgement of the nasophaynx, larynx, trachea and bronchi
- Diaphragm raised 4 cm
- Ribcage displaced upwards with 2 cm↑ in diameter ↑ 6 cm increases in circumference
- Lowered abdominal muscle tone
- Resultant increase in MV by ~25-40% at full term
- Alveolar and arterial PCO$_2$ values are reduced (~30 mmHg) given that MV is increased beyond requirements for CO$_2$ removal/O$_2$ uptake
- Alveolar and arterial PO$_2$ are increased by ~7.5 mmHg
- This state of hyperventilation is attributed to progesterone levels and increased sensitisation of the central chemoreceptors

The following alterations occurring with lung volumes and flows:

- Increased dead space
- TV ↑s by 35-50%
- TLC ↓s by 5%
- FRC, RV and ERV volume ↓ by 20% (and thus a significant decrease in O$_2$ stores)
- Alveolar and arterial PCO$_2$ values are reduced (~30 mmHg) given that MV is increased beyond requirements for CO$_2$ removal/O$_2$ uptake
- Alveolar and arterial PO$_2$ are increased by ~7.5 mmHg
- This state of hyperventilation is attributed to progesterone levels and increased sensitisation of the central chemoreceptors
- VC, FEV$_1$ and MBC remain unchanged

The following changes to ventilatory control occur with pregnancy:

- Unchanged respiratory rate but increased tidal volume
- Resultant increase in MV by ~25-40% at full term
- Alveolar and arterial PCO$_2$ values are reduced (~30 mmHg) given that MV is increased beyond requirements for CO$_2$ removal/O$_2$ uptake
- Alveolar and arterial PO$_2$ are increased by ~7.5 mmHg
- This state of hyperventilation is attributed to progesterone levels and increased sensitisation of the central chemoreceptors
- The slope of the PCO$_2$ response curve increases three-fold
- The hypoxic ventilatory response increases two fold, and this change precedes the increased demand placed on the maternal respiratory system during pregnancy
- The sense of dyspnoea (experienced in ~50% of pregnancies) is associated with an increased minute ventilation for a given progesterone level
- Oxygen consumption rises from ~194 mLmin$^{-1}$ to ~250 mLmin$^{-1}$

2. Exercise
The response varies with the level of exercise:

- **Moderate**: below the anaerobic threshold. No rise in lactate. Oxygen transport adequate. Steady state
- **Heavy**: above the anaerobic threshold. Blood lactate rises and remains constant. Steady state
- **Severe**: far above anaerobic threshold with lactate levels rising continuously. Unsteady state

Exercise intensity often described in terms of metabolic equivalents (METs):

- \( O_2 \) consumption at the basal metabolic rate is 200-250 mL/min
- \( O_2 \) consumption increases by \( \sim 12 \text{mL/min}^1 \text{W}^{-1} \)
- METs measure the number of multiples of resting \( O_2 \) consumption
- Brisk walking on a flat surface \( \sim 4 \) METs
- Running at 12 km/h is \( \sim 12 \) METs

Maximal \( O_2 \) uptake (\( V\text{O}_{2\text{max}} \)) is the oxygen consumption of a subject reached during exercise whereat a plateau occurs, beyond which increased work rate is not associated with an increased oxygen consumption. This definition is problematic for elite athletes where respiratory quotient and lactate levels must also be taken into account. Work rates occurring beyond this point can only be sustained by anaerobic glycolysis. \( V\text{O}_{2\text{max}} \) is normally 3L/min in a fit 70 kg adult and decreases to \( \sim 2 \text{L/min}^{-1} \) by age 70. Higher levels are achieved in athletes (5 L/min) with rowers noted to have the highest (6 L/min).

The respiratory quotient changes and approaches or exceeds 1 during heavy/severe exercise, reflecting the increased reliance upon carbohydrate over fat as a fuel source. \( R \) values higher than 1 reflect the unsteady state during this form of exercise wherein lactate is produced as a result of anaerobic glycolysis and additional \( CO_2 \) is eliminated from bicarbonate.

The anaerobic threshold for exercise is the m

With high minute volumes seen with exercise, the oxygen consumption of the respiratory muscles becomes significant: 5% with moderate exercise and 10% at \( V\text{O}_{2\text{max}} \)

The following changes facilitate the 10 to 20-fold increase in \( O_2 \) consumption:

- Increased \( O_2 \) delivery (the product of \( CO \) and \( C_aO_2 \)). Cardiac output increases to 5 x maximum (\( \sim 25 \text{L/min}^{-1} \)) at most
- Increased \( O_2 \) extraction: \( S_{mv}O_2 \) normally \( \sim 70\% \) and this decrease to \( \sim 20\% \) with heavy exercise. This fall in saturation occurs on the steep part of the Hb-O2 dissociation curve and thus occurs without a large fall in \( PO_2 \) (37.5 - 15 mmHg). Increased lactate levels shift the Hb-O2 dissociation curve to the right (\( \therefore \) improved extraction) within the capillary bed.
- If delivery and extraction do not meet the metabolic demand then anaerobic metabolism commences

Maximal breathing capacity:

- The maximum minute volume of ventilation sustainable for 15 seconds
Normally 15-20 times the resting minute ventilation
- 50-60$
- $O_2$ consumption increases by $\sim 12\text{mLmin}^{-1}\text{W}^{-1}$
- METs measure the number of multiples of resting $O_2$ consumption
- Brisk walking on a flat surface $\sim 4$ METs
- Running at $12\text{kmh}^{-1}$ is $\sim 12$ METs

The ventilatory response to exercise

Ventilatory control:

- Imagining exercise causes increased ventilation
- Ventilation normally increases prior to any other change aside from $\uparrow CO$ suggesting that neural factors play a significant role
- The $PO_2$ response curve is steeper during exercise, inhibition/resection of the carotid bodies reduces the ventilatory response to exercise. pH, $PO_2$ and $PCO_2$ values are typically unchanged during exercise. $PCO_2$ may decrease during heavy of severe exercise beyond Owles point (where lactate is thought to stimulate an increase in MV beyond that predicated by linear extrapolation)
- Metabolic acidosis associated with heavy and severe exercise increases ventilation (Owles point on the MV vs $O_2$ consumption curve)
- Hyperthermia may stimulate ventilation
- Passive limb movement may stimulate ventilation through afferent input arising from joint and muscle reflexes

Pulmonary vascular and blood flow changes:

- $\uparrow CO$ is associated with $\uparrow$ pulmonary arterial and pulmonary venous pressure
- Pulmonary vascular resistance decreases (following increased recruitment of vessels)

Volumes, V/Q, diffusion and dissociation:

- Tidal volume $\uparrow$s to 50% of VC
- V/Q mismatch decreases in normal people with little effect overall. In elite athletes exercising at high levels, V/Q mismatch (thought due to interstitial fluid accumulation in the context of raised pulmonary vascular pressures) occurs
- Diffusing capacity at altitude may be a limiting factor at altitude
- In elite athletes hypoxia may arise due to a combination of diffusion limitation, V/Q mismatch and airflow limitation
- The Hb-O2 dissociation curve shifts to the right within exercising muscles due to increases in $[H^+]$, $PCO_2$ and temperature, favouring peripheral $O_2$ unloading. At the pulmonary level there may a temperature mediated leftward shift in the dissociation curve.
- Additional capillaries open during exercise and the number of capillaries and mitochondriae increase over the longer term within skeletal muscle (the former decreasing the length of distance required for peripheral diffusion)

3. Age related respiratory changes

Control:
- Hypoxic and hypercapnoeic ventilatory responses are diminished

Volumes and anatomical changes:

- Between 20- and 80 years of age ~30% of alveolar tissue is lost
- There is an associated loss of lung elastic recoil and parenchymal traction
- The consequences include: increased residual volume, increased closing volume and increased FRC, decreased VC and decreased FEV$_1$
- Chest wall compliance decreases
- Respiratory muscle mass decreases
- Airway protective reflexes are diminished

Ventilation-perfusion matching is increasingly mismatched with PaO$_2$ decreasing with age

Physiologic dead space increases and diffusing capacity decreases