

Respiratory / ventilatory failure

- There are 2 principle functions of the respiratory system, uptake of O₂ and elimination of CO₂.
- Although these functions are closely linked, respiratory failure can result in hypoxaemia, hypercapnia or both.
- Arterial oxygen tension is principally determined by the fraction of inspired oxygen (FiO₂), ventilation / perfusion matching and the blood's oxygen carrying capacity (essentially haemoglobin concentration).
- Assuming a fixed rate of CO₂ production, arterial CO₂ tension is primarily determined by alveolar minute ventilation i.e. (tidal volume anatomical & physiological dead space) x the respiratory rate.

IPPV and other acronyms

- The terminology surrounding IPPV has become unnecessarily complex, especially ventilatory modes and their acronyms.
- The value of regular patient review, including respiratory examination, cannot be overstated.
- There has been a huge philosophical shift in the last few years away from trying to normalise gas exchange and towards minimising ventilator induced lung injury (VILI), which should perhaps more appropriately be termed physician induced lung injury.
- Regional lung ventilation and perfusion are not homogenous and this heterogeneity increases in disease. Positive pressure ventilation can cause lung injury via overdistension (volutrauma), excessive pressure (barotrauma) and cyclical recruitment and derecruitment (alectratrauma or biotrauma).
- Be aware of heart lung interactions and the effects on these of IPPV. In particular, be aware of the detrimental effects of sustained high airway pressures on right heart function and the risks of inducing right heart failure.

Goals of IPPV and how to achieve them

PaO₂ > 7 kPa and pH > 7.20 whilst minimising VILI and cardiovascular compromise.

To treat hypoxaemia

- Consider performing a recruitment manoeuvre
- Increase the CPAP / PEEP.
- Consider increasing the I:E ratio.
- Consider changing the patient's position
- Increase the FiO₂

To fix hypercapnia

- DON'T if the pH is normal or within acceptable limits (permissive hypercapnia)
- Increase respiratory rate and / or tidal volume watch the effect on peak pressures, inspiratory and expiratory flows. Remember that the volume of dead space ventilation is fixed hence increasing the rate at the expense of tidal volume will result in a reduction in alveolar minute ventilation and a rise in PaCO₂.

Things to set & things to measure

FiO₂

- Start high and reduce gradually.
- Minimise to achieve PaO_2 8-10 kPa / SpO2 88-92%.

Mode "PCV+" = pressure control SIMV

- Set PEAK airway pressure "Pinsp" note that the inspiratory pressure delivered = "Pinsp" – PEEP
- Increase / to achieve MEASURED expiratory Vt 6-8ml/Kg (IBW) and aim for <30cmH₂O MEASURED peak airway pressure
- Set inspiratory pressure support "ΔPsupp" for spontaneous breaths (breaths in excess of set rate) "ΔPsupp" = Pinsp – PEEP
- Set breath rate and Tinsp MEASURE minute ventilation, I:E ratio and look for ZERO end expiratory flow
- Set PEEP to recruit and retain units "higher FiO₂" should prompt "higher PEEP" – usual range 5-10cmH₂O

Things to set & things to measure

FiO₂

- Start high and reduce gradually.
- Minimise to achieve PaO₂ 8-10 kPa / SpO2 88-92%.

Mode CPAP + pressure support

- Set inspiratory pressure support "ΔPsupp"
- Increase / to achieve MEASURED expiratory Vt 6-8ml/Kg (IBW) and aim for <30cmH₂O MEASURED peak airway pressure

Things to remember

Alarms

- Respond to them!
- Set them appropriately

Patient position

• Sit up / head up

And . . .

- Spontaneous breathing IS BETTER than mechanical breaths
- Weaning from support is a continual process and linked to CNS start as soon as possible
- So called "clever" stuff APRV HFOV etc ... another day!