

Ventilatory strategy in various diseases

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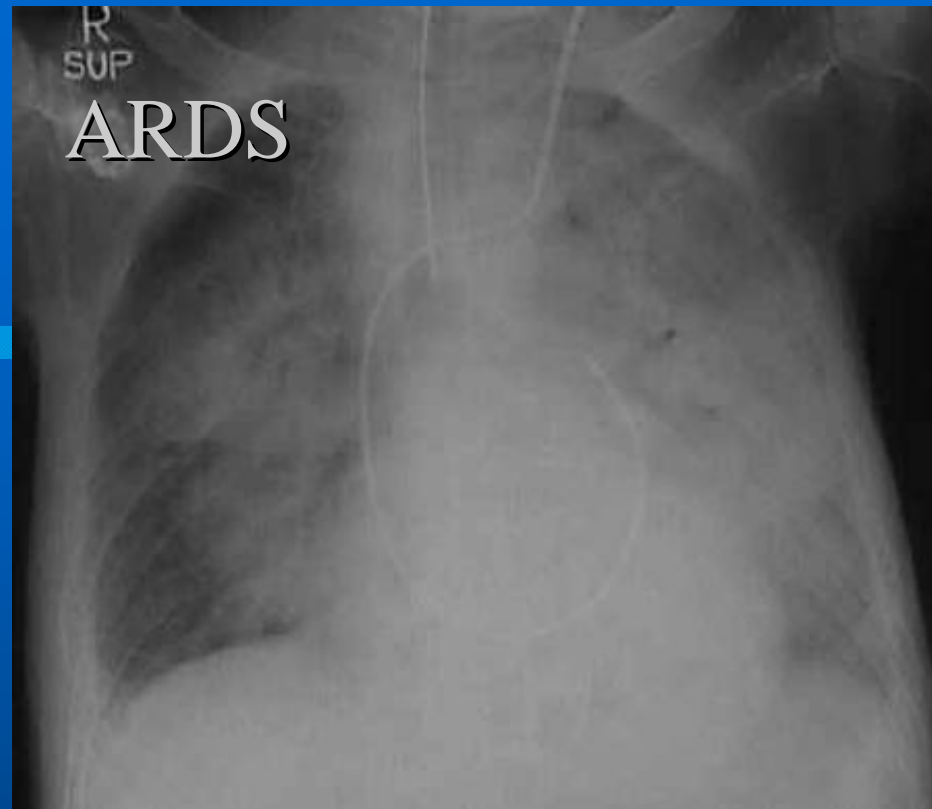
General principle:

To direct strategy towards pathophysiology

- **Low compliance**
 - Acute respiratory distress syndrome
 - Acute pulmonary oedema (APO)
 - Pneumonia
- **High resistance**
 - Asthma
 - COPD
 - Emphysema (high compliance)
- **Neuromuscular (low chest wall compliance + loss of control)**
 - Motor neuron disease
 - Myasthenia crisis
 - Gullain-Barre syndrome
- **Mixture of the above**
 - E.g. patient with COPD and APO given muscle relaxant

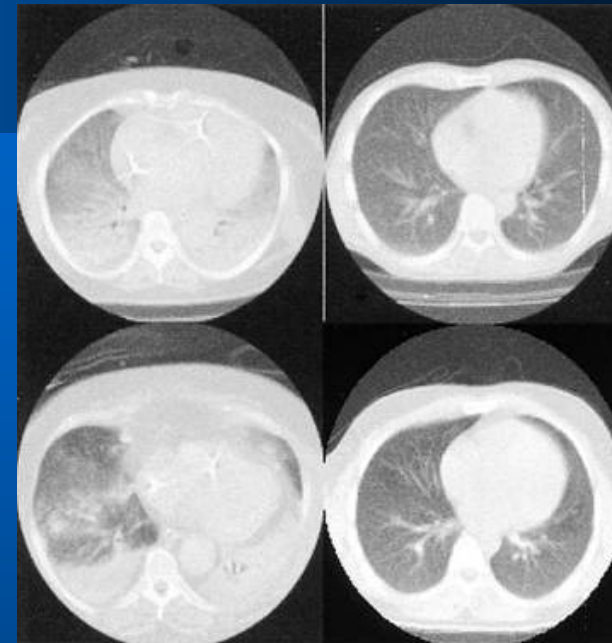
Low compliance lungs

ARDS, APO, Pneumonia

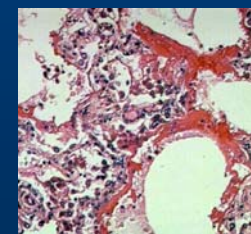
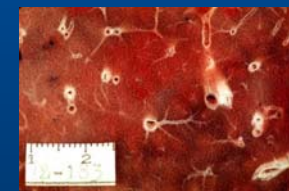
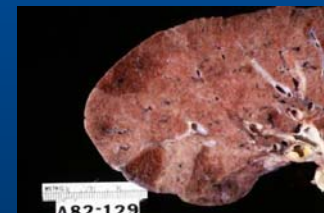
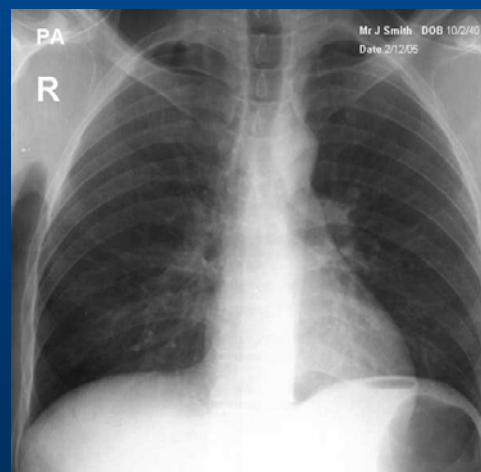


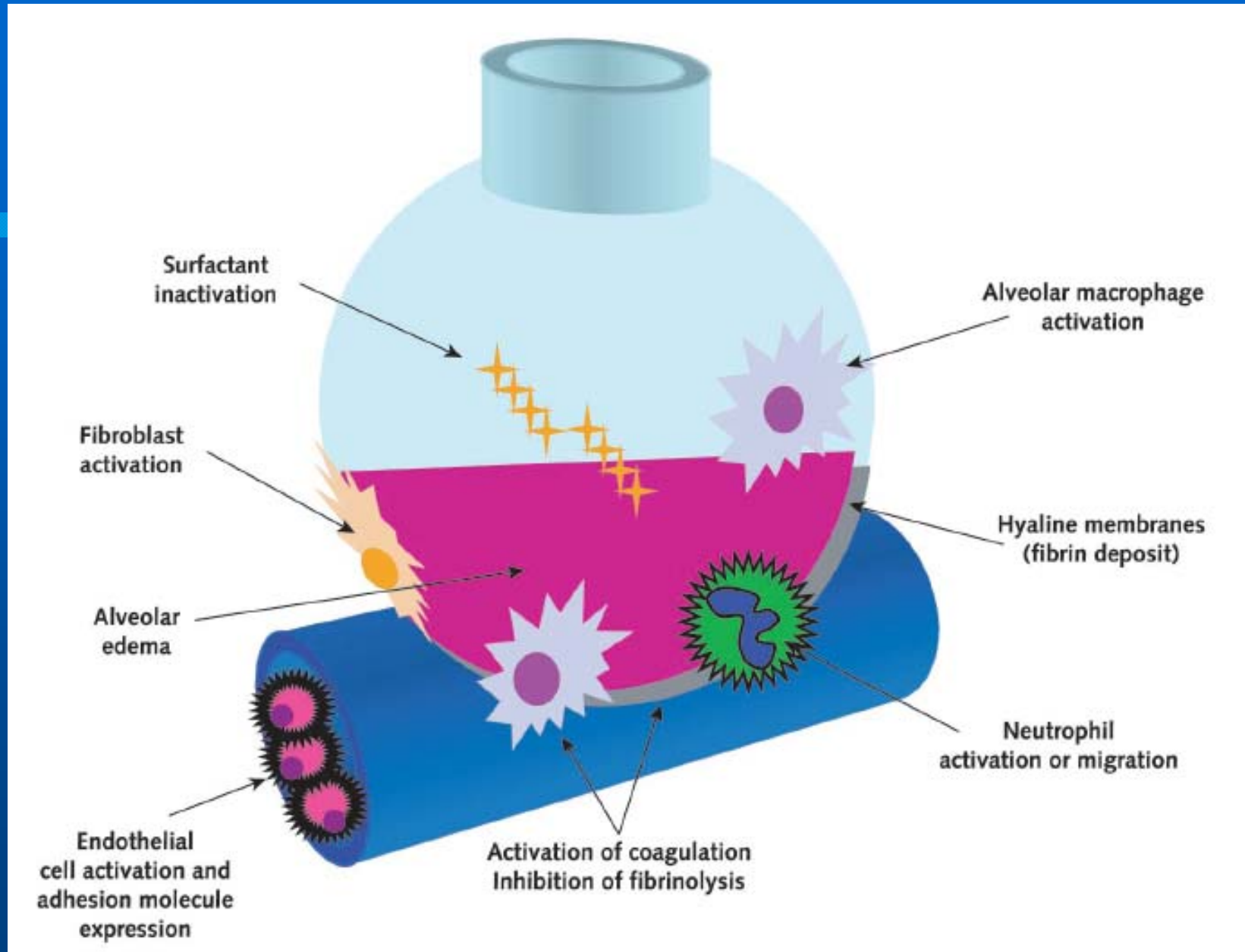
ARDS

Normal



Normal





Ventilator mode

- **Invasive MV:**
 - PCV or VC
 - Decelerating flow
- **Noninvasive**
 - Evidence points to harm

Noninvasive ventilation for acute lung injury or ARDS

- 123 patient RCT of CPAP vs standard oxygen therapy
 - 55% pneumonia
 - 17% cardiogenic APO
- NIV group
 - Inc 1 hr oxygenation
 - Same intubation rate
 - Same hospital mortality
 - Same ICU LOS
 - Inc adverse events
- 4 pts had cardiac arrest (3 at time of intubation)
- 4 pts had stress ulcers

Invasive ventilation

“Lung protective strategy”

- TV: start at 6-7 ml/kg predicted BW (to maintain plateau pressure <30-40 cm H₂O)
- RR: up to 35/min, watch out for dynamic hyperinflation
- N.B. Minute volume = RR x TV

Amato et al NEJM 1998

The ARDSnet mechanical ventilation study NEJM 2000 (N = 861)

“Open lung approach”: Positive end-expiratory pressure (PEEP)

- **Increase by 2-5 cm until the "best/optimal PEEP" is obtained:**
 - highest static compliance
 - lowest airway plateau pressure (keep below 30 cmH₂O)
 - Keep PEEP above lower inflection point on static P-V curve
- **PEEP over 20 cm is rarely beneficial**
- **Optimal PEEP**
 - improves gas exchange by maintaining alveolar patency
 - reduces the FiO₂ required (FiO₂ > 0.50 for >24 hrs is toxic to the lung)
- **Too high PEEP will cause lung injury and impairs cardiac output**

Inverse ratio ventilation (IRV)

- **I:E ratio > 1 can improve oxygenation in patients who remain hypoxic despite PEEP**
 - not all patients benefit from this strategy
 - muscle relaxants often required when I:E ratios of $> 2:1$ are used
 - small risk of causing haemodynamic compromise

Targets of ventilation

- **PCO₂: permissive hypercapnia**
 - allow PCO₂ to rise slowly (i.e. giving kidneys time to compensate for respiratory acidosis), aim to keep pH > 7.25 (instead of aiming for a target PCO₂, but advisable to not to allow Pco₂ to rise above 20 kPa)
- **PaO₂/SpO₂:**
 - Keep FiO₂ to < 0.50 for < 24 hrs
 - Keep PaO₂/SpO₂ low normal
- **Aimed to achieve plateau pressure <=30cmH₂O, moderate hypercapnia (aim PaCO₂<=90mmHg / 12 KPa) and acidosis (aim pH >=7.2)**

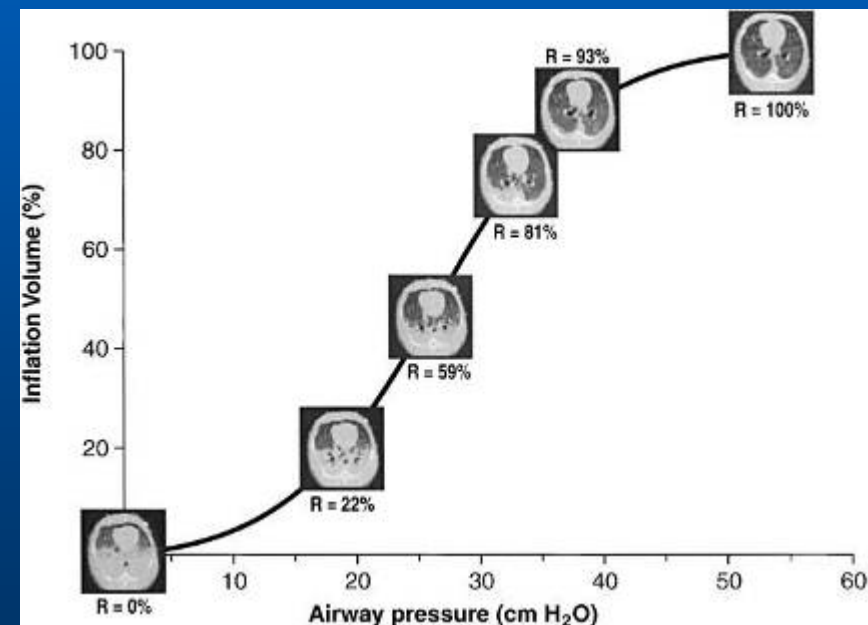
Curr Opin Pulm Med 1998, 4:4–8
Crit Care Clin 1998, 14:685–705

Adjunctive and experimental strategies

- Lung recruitment manoeuvre
- Prone positioning
- High frequency oscillation
- Airway pressure release ventilation
- Extracorporeal membrane oxygenation
- Tracheal gas insufflation
- Partial liquid ventilation
- Aspiration of gas from the dead space (ASPIDS)

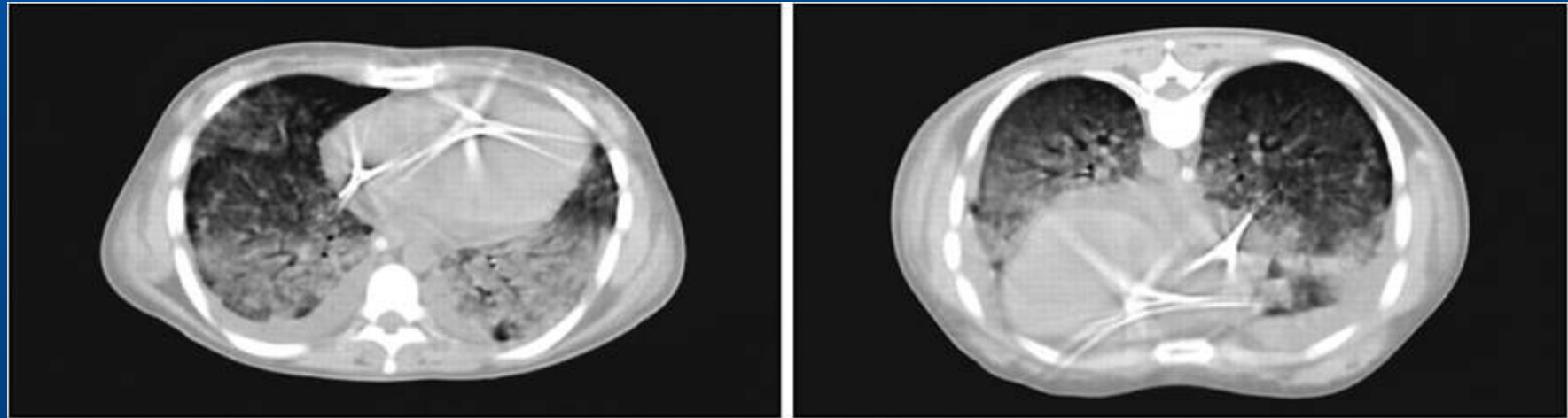
Recruitment maneuvers: methods

1. incremental levels of PEEP with constant peak airway pressure inspiratory pressure, or
2. prolonged increase in pressure (eg 40-50 cmH₂O) either as single maneuver (lasting 40-50 secs) or with stepwise increase in pressure with return to baseline between each increase, or
3. pressure control ventilation at high inspiratory pressures either with stepwise increases with return to baseline between each increase or single maneuver lasting 120 secs



Prone positioning

- No beneficial outcomes and some safety concerns (pressure sores, selective intubation, ETT obstruction) associated with prone positioning (applied as early as possible for at least 8 hr/day). For patients with hypoxemic ARF, prone position placement may lower the incidence of VAP – Guerin C et al, JAMA 2004
- Routine application not advisable
- Avoid in haemodynamically unstable cases

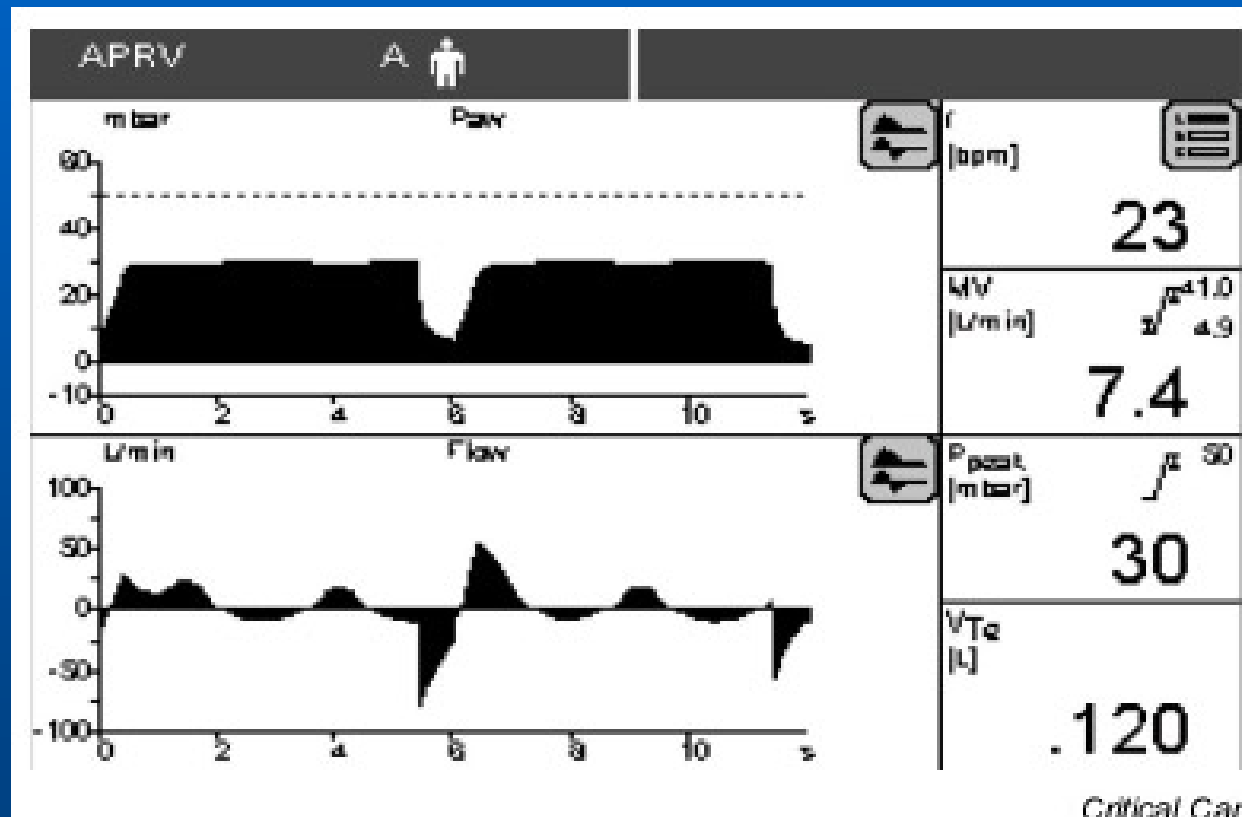


High frequency oscillatory ventilation

1. High Frequency Oscillatory Ventilation (HFOV)
2. High Frequency Jet Ventilation (HFJV)
3. High Frequency Flow Interruption (HFFI)
4. High Frequency Positive Pressure Ventilation (HFPPV)



Airway pressure release ventilation (APRV)



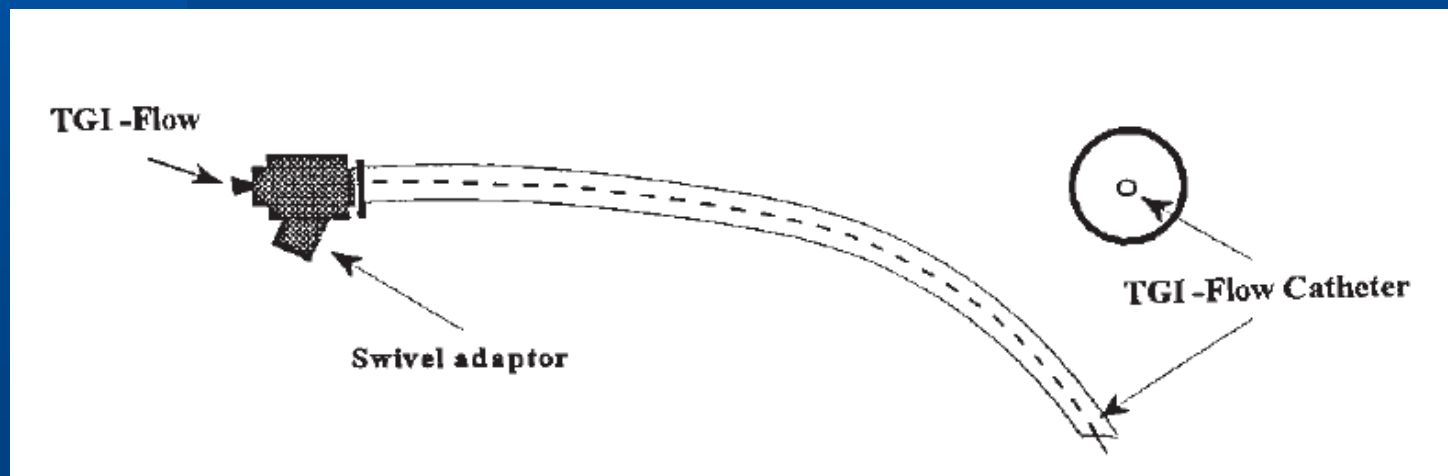
Extracorporeal membrane oxygenation (ECMO)

- very expensive not associated with improved survival



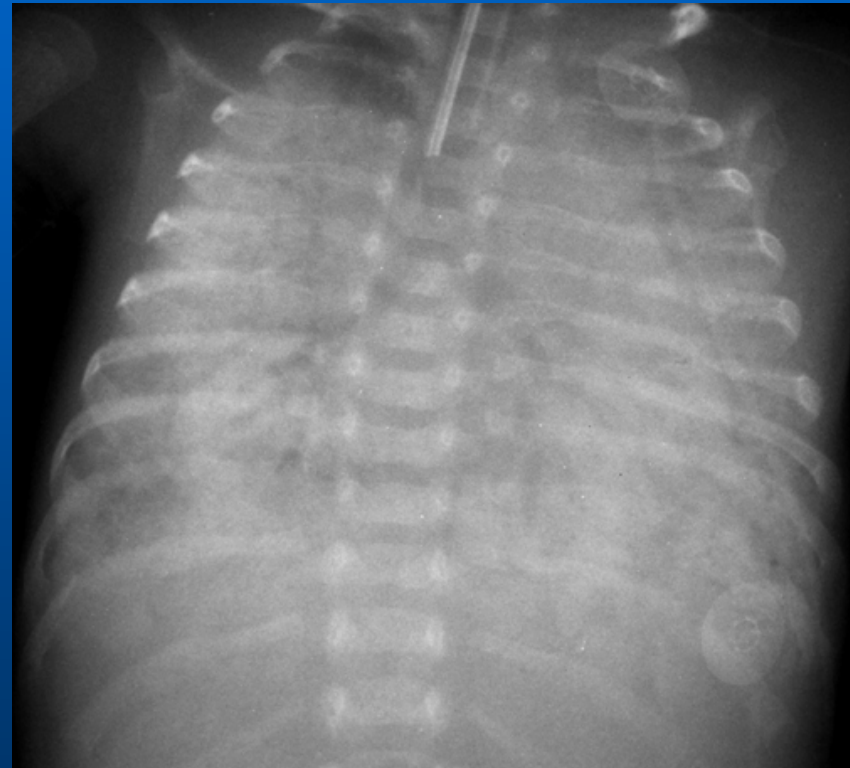
Tracheal gas insufflation

- enhance CO₂ elimination efficiency
- Method
 - During expiration, fresh gas insufflated through the TGI catheter washes out the CO₂ that remains in the dead space proximal to the catheter tip



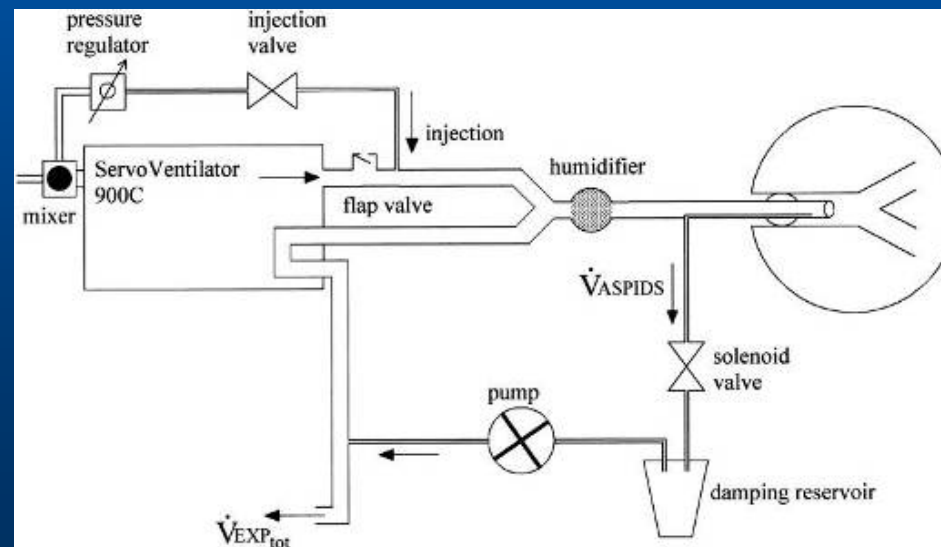
Partial liquid ventilation

- filling the lungs with a fluid (perfluorocarbon, also called Liquivent or Perflubron) which has
 - very low surface tension, similar to surfactant
 - high density, oxygen readily diffuses through it
 - may have some anti-inflammatory properties
- Liquid will help the transport of oxygen to parts of the lung that are flooded and filled with debris, help remove this debris and open up more alveoli improving lung function.



Aspiration of gas from the dead space (ASPIDS)

- gas rich in CO₂ during late expiration is aspirated through a channel ending at the distal end of the tracheal tube.
- Simultaneously, fresh gas injected into the inspiratory line fills the airway down to the same site.



Conclusion

- Lung protective strategies
 - Low tidal volume (6 ml/kg PBW)
 - High PEEP (similar outcome for lower PEEP if small TV is used)
 - Low plateau pressure (<30 cmH₂O)
 - Permissive hypercapnia
 - Inverse ratio ventilation if necessary
- Adjunctive or experimental
 - Recruitment manoeuvres
 - Prone positioning
 - ECMO
 - Partial liquid ventilation
 - High frequency ventilation
 - Tracheal gas insufflation
 - ASPIDS

	ARDS	APO	Pneumonia
Mechanism	Leakage from damage to the integrity of the lung's alveolar-capillary barrier	Hydrostatic pressure from heart failure	Infection with neutrophil activation
Alveolar content	Protein-rich	Fluid-rich	Infected
Symmetry	Y	Y	Y/N
Compliance	Very low	Low	Low in consolidative area
Progress	Exudative Fibro-proliferative Organising >> recovery, or with permanent fibrosis	Depends on heart condition	Depends on drainage, antibiotics Rx and host defence
Ventilatory strategy	Lung protective strategy, Open lung approach, Permissive hypercapnia	Simpler, very responsive to just high PEEP	Variable

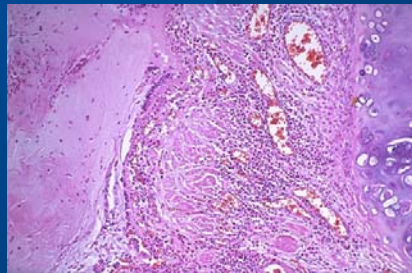
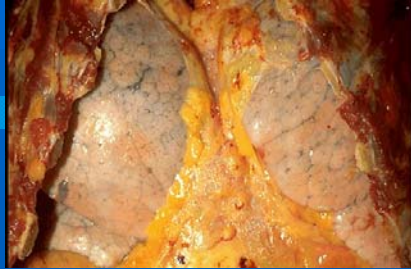
High resistance lungs

Asthma: status asthmaticus

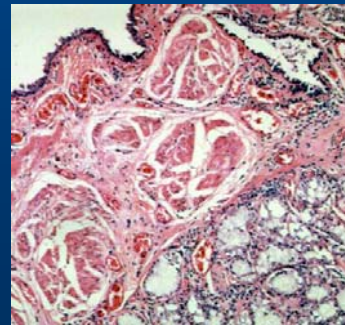
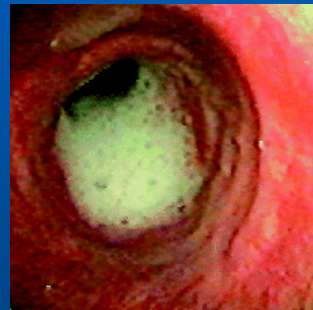
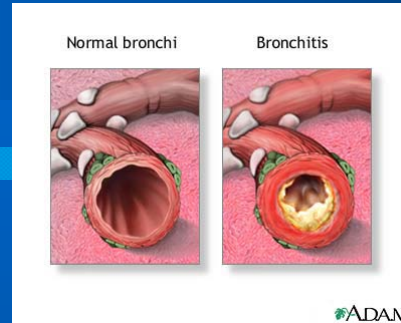
Chronic bronchitis

Emphysema

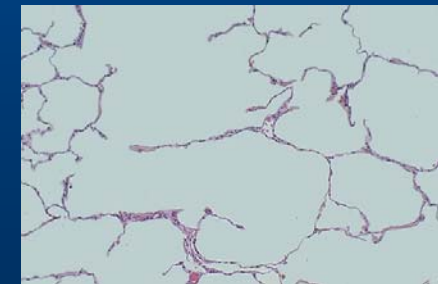
Asthma



Chronic bronchitis



Emphysema



Asthma

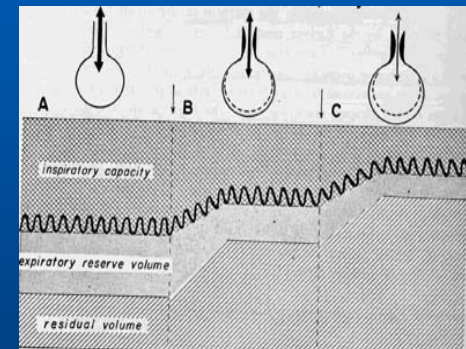


Modes of ventilation

- **Invasive: for acute severe asthma or COPD**
- **Noninvasive:**
 - evidence strong for COPD (dec intubation and mortality)
 - Not strong for asthma

Goals of mechanical ventilation

- To reduce dynamic hyperinflation (DHI)
 - limited minute ventilation (MV), using low tidal volume (V_t) and respiratory rate
 - extended expiratory time (TE)
- Targets:
 - Permissive hypercapnia
 - Aim at the lowest positive inspiratory pressure (PIP) and the lowest expiratory lung volume, avoiding intrinsic positive end expiratory pressure (PEEP)



Ventilator modes

- **volume controlled mode: guarantees reproducible VT despite variations of airways resistance but then, the consequent rise of pressure must be limited**
- **pressure controlled mode: variations of resistance induce important variations of VT. A continuous monitoring of ventilation is always indicated (inspiratory and expiratory parameters of the ventilator, saturation, capnography, transcutaneous pCO₂, ABG)**
- **Assisted and spontaneous mode are not indicated**

Neuromuscular blockade

- Indicated in patients who, in spite of sedation, continue to breathe in a desynchronized manner
- E.g. rocuronium

Initial ventilator settings in status asthmaticus

Setting	Recommendation
Mode	Pressure-controlled ventilation
Respiratory rate	10–15 breaths/min
Tidal volume	6–10 ml/kg
Minute ventilation	8–10 L/min
PEEP	0 cmH ₂ O
Inspiratory/Expiratory ratio	≥1:3
Inspiratory flow	≥100 L/min
F _I O ₂	Maintain SaO ₂ >90%
Pplat	<35 cm H ₂ O
V _{EI}	<1.4 L

F_IO₂, fraction of inspired oxygen; PEEP, positive end-expiratory pressure; Pplat, end-inspiratory plateau pressure; SaO₂, oxygen saturation; V_{EI}, end-inspired volume above apneic functional residual capacity.

Further ways to reduce hyperinflation

- transitory use of a high PEEP (> 10 cm H₂O)
- with 100 % oxygen, ventilation with a rate of 2 to 3 breaths per min for several minutes
- acceleration of expiratory flow by manually compressing the rib cage, the patient being disconnected from ventilator

Evaluation of DHI

- **Elevated intrinsic PEEP**
 - failure of expiratory flow curve to return to the baseline before next inspiration
- **Elevated plateau pressure (Pplat)**
 - Pplat above 30 cm H₂O in adults has been correlated with complications
- **Elevated end inspiration volume (VEI)**
 - measured by disconnecting sedated and relaxed patient from the ventilator
 - apnea interval 20 to 30 seconds so that expiratory flow ceases
 - Total expired volume is the VEI. The difference between V_t and VEI is the volume of gas trapped in the lungs (VDHI)
 - VEI must be < 20 ml/kg/min to reduce volu- and barotrauma risks

Complications

- **Baro- or volu-trauma: Pneumothoraces, pneumomediastinum, pneumoperitoneum, pulmonary interstitial emphysema**
- **hemodynamic**
 - after intubation hypotension is linked to hyperinflation, hypovolemia and sedation, decreased venous return, tissue hypoxia, arrhythmias, oedema
- **mucous plugs: atelectasis**
- **nosocomial infection (ventilator-associated pneumonia, catheter-associated blood stream infection, sinusitis, etc ...)**

Alternative technique in difficult patients

- **Volatile inhalational agents (isoflurane, halothane): at 0.5-2% induce bronchodilation and sedative effect decreased bronchospasm. All are cases reports only and no RCT a/v**
 - Crit Care Med 1986;14: 832-833
 - Intensive Care Med 1990; 16:104-107
 - Pediatr Crit Care Med. 2000 Jul;1(1):55-9
 - Arerugi. 2005 Jan;54(1):18-23. Japanese
- **Ketamine**
 - **cause bronchodilatation by both sympathomimetic potentiation and direct effect on airway smooth muscle**
 - Pediatr Emerg Care 1996; 12: 294-7
 - **Some case reports showed beneficial effects**
 - Pharmacotherapy. 2001 Sep;21(9):1100
 - J Asthma. 2001 Dec;38(8):657-64

Alternative technique in difficult patients

- **Heliox: inert gas, 1/4 the density of air and mixtures of helium and oxygen (60% / 40%) decrease the work of breathing by decreasing inspiratory and expiratory resistance**
Crit Care Med 2000, 28:2721–2728
- **The Cochrane Database of Systematic Reviews 2003, Issue 2. Art. No.: CD002884**
 - 6 RCTs, 369 asthmatics
 - Heliox use did not improve pulmonary functions compared to standard care

Alternative techniques in difficult patients

- **MgSO₄: block Ca channels thereby mediating smooth muscle relaxation and bronchodilation**

JAMA 1989; 262: 1210-3

- **The Cochrane Database of Systematic Reviews 2000, Issue 1. Art. No.: CD001490**
 - 7 RCTs, 665 patients
 - Non-significant improvement of PFR
 - Admission rate decreased in severe subgroup but not in overall asthmatics

Comparison with COPD

	Asthma	COPD
Clinical	AFO can be very severe and episodic AFO may completely resolved	AFO is not episodic AFO not completely resolved
General strategy	Reduce DHI	Reduce DHI Reduce secretion Rehabilitation
NIV	N	Y
Adjunctive therapy	Muscle relaxant, Isoflurane, Ketamine, Heliox, MgSO ₄	Muscle relaxant, longer term care with tracheostomy, pulmonary rehabilitation

Neuromuscular disease



Motor neuron disease
Myasthenia gravis with crisis
Guillain-Barre syndrome
Spinal cord injury



Table 4 Neuromuscular Diseases That May Be Associated with Need for Mechanical Ventilation

Disease	Comments
Myasthenia gravis only	Acute occurrence, temporary ventilation is usually required, rarely requires tracheostomy, may have bulbar involvement.
Guillain-Barré syndrome	If progressing and doing poorly despite therapy will typically require long-term ventilation averaging 37 days with high complication rate.
Muscular dystrophy	Respiratory failure primarily seen late in disease process and with Duchenne's and Becker's dystrophy. Decision for initiation of mechanical ventilator support should be discussed thoroughly with the patient owing to poor progress of underlying disease.
Amyotrophic lateral sclerosis (ALS)	Usually not ambulatory when respiratory failure occurs, usually with bulbar involvement, same issues for intubation decision as above.
Multiple sclerosis	Late occurrence as with ALS, similar ethical issues as with muscular dystrophy and ALS.
Spinal cord injury (C1-2, C2-3)	Long-term support invariable.
Spinal cord injury (C3-4, C4-5)	Variable needs for chronic support.
Spinal cord injury (C5-6)	Ventilatory support usually not required unless underlying lung injury.

Problems

- Removal of secretions
- Ventilatory pump failure
- Progressive atelectasis
- Increasing oxygen requirement
- Decreasing MIP and VC

Table 5 Guidelines for Intubation and Initiation of Mechanical Ventilation in Acute Respiratory Failure Due to Neuromuscular Disease

Severe dyspnea

Paradoxical diaphragm excursion with inspiration

$P_{I\max}$ of -20 cmH₂O or more positive—effort dependent

$P_{I\max}$ of -20 to -35 and rapidly decreasing serial measurements—effort dependent

Vital capacity <10 ml/kg

Acute respiratory acidosis (late finding)

Progressive atelectasis

Noninvasive positive-pressure ventilation may be tried if patient is not in extremis, is free of cardiovascular instability, does not have excessive secretions, is alert and cooperative, and has ability to protect airway.

Choice of ventilatory support

- **Noninvasive positive-pressure ventilation**
 - If reversibility is expected over hours to days, e.g. mild LRTI in chronic neuromuscular disease as polymyositis or MG
 - Problem: secretion retention
- **Intermittent positive pressure ventilation via endotracheal tube**

Mode of ventilation

- Assist control or high-level pressure-support
- Decelerating ramp
- High flow early in inspiration
- Larger tidal volumes (12 – 14 ml) may be better tolerated and maximize stimulation of surfactant production
- PEEP: use physiological PEEP (3- 5 cm H₂O)
- MV adjusted for desired pH
- Flow triggering
- Tracheostomy for failure to wean within 3 weeks

<i>Disease condition</i>	<i>Acute Resp distress syndrome (ARDS)</i>	<i>Acute pulmonary oedema</i>	<i>Obstructive lung disease (COPD/ Asthma)</i>	<i>Restrictive lung disease</i>
<i>Chief disease mechanism</i>	<i>Very low lung compliance</i>	<i>Low lung compliance</i>	<i>Airflow obstruction</i>	<i>Low lung &/ or chest wall compliance</i>
Tidal volume (ml/kg predicted BW)	6	8 – 10	6 – 8	12 – 14
Frequency (breath/min)	Permissive hypercapnia (keep pH just above 7.25 as “lung protective strategy”)	Assisted control/ SIMV/ pressure support (PS) mode to achieve patient comfort	10 – 14 Ensure long enough expiratory time to avoid air-trapping	To achieve desired pH and ABG
Positive end-expiratory pressure/ PEEP (cmH ₂ O)	May need > 10 (open lung approach)	High (5 – 10) initially, can be rapidly tailed down	0 – 3 max	3 – 5
Adjunctive measures	Neuromuscular blockers (NMB)	Cardiac intervention	NMB Manually assisted expiration (esp asthma)	Tracheostomy

Thank you