Protective Mechanical Ventilation in Anesthesia Practice: Evidence Based Trends

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Objectives

- Describe various modes of ventilation and appropriate utilization techniques
- Examine the concepts of barotrauma, volutrauma, and atelectotrauma and their association with ventilation strategies
- Discuss prevention of ventilator associated lung injury (VALI) and postoperative respiratory failure in anesthetized patients
- Explore PEEP and PC ventilation strategies focusing on their appropriate implementation and benefits
- Understand appropriate use and untoward affects of hyperventilation (hypocapnia)
Basic Ventilator Concepts

- **3 Elements of Mechanical Breath:**
  - Trigger: ventilator or patient
    - With patient trigger, either pressure or flow change is sensed
  - Limits: pressure or volume
  - Cycle: what ends the mechanical breath delivery
    - Volume, time, or flow decrement

- **Example:** Pressure Support ventilation (PSV) is patient-triggered, pressure-limited, flow-cycled (stops breath when a decrease in patient inspiratory flow to 25% of peak inspiratory flow is noted).

Allain et al., 2010
Full vs Partial Ventilation

- Full/Mandatory ventilation: the ventilator provides all of the patient’s minute ventilation ($V_e$).
  - Appropriate after neuromuscular blockade or when patient efforts are undesirable (energy expenditure, surgical exposure)
  - VC, PC; no contribution from the patient
- Partial/Assisted ventilation: patient provides contribution to respiratory effort
  - PS ventilation or IMV with patient efforts
  - More synchrony in spontaneously ventilating patient
  - Less positive airway pressure required with patient efforts
CO2 clearance

- CO2 clearance is reliant on partial pressure gradient between blood and alveoli
- Need to exhale CO2 and bringing fresh gas with lower CO2 concentration in the lungs
- More CO2 can then diffuse for exhalation
- CO2 clearance is reliant on minute ventilation (there needs to be air moving in and out to achieve higher minute ventilation)
- CO2 is diffusion limited and hypercarbia will result if CO2 is not being cleared from lungs
CO₂ Diffusion Limitation

Nonoxygenated Blood

$P_{\text{O}_2} = 40 \text{ mm Hg}$
$P_{\text{CO}_2} = 46 \text{ mm Hg}$

Alveolus

$P_{\text{A}_2} = 100 \text{ mm Hg}$
$P_{\text{ACO}_2} = 40 \text{ mm Hg}$

Reoxygenated Blood

$P_{\text{A}_2} = 100 \text{ mm Hg}$
$P_{\text{ACO}_2} = 40 \text{ mm Hg}$

Blood Flow
Oxygenation

- Normally, O2 easily diffuses and is perfusion limited
  - Hemoglobin (Hb) is fully saturated and diffusion across alveolar capillary membrane is not a problem
  - When CO is very high (vigorous exercise), all O2 cannot get across fast enough to fully saturate Hb, O2 becomes diffusion limited

- O2 also becomes diffusion limited when Fick’s principles are not in favor (ARDS/ALI/Aspiration/Fibrosis)

- In diseased lung, O2 becomes reliant on increased surface area for diffusion, achieved by increased Mean Airway Pressure and PEEP
  - Increasing minute ventilation via RR or Vt may not achieve adequate oxygenation as it does CO2 clearance
**PIP & Mean Airway Pressure**

- **PIP** - peak inspiratory pressure: max pressure throughout the respiratory cycle
  - PEEP+ Pressure to deliver breath = PIP

- **Mean airway pressure**: average pressure in the airways throughout the respiratory cycle
  - Increasing MAP, either via PEEP or other ventilator settings will improve oxygenation and increase area for effective gas exchange (decrease dead space)
  - Adding PEEP is the simplest way to increase mean airway pressure and improve alveolar recruitment (reduce deadspace)
Limitation for O2 diffusion

I:E Ratio

- The time spent delivering a breath compared to the time spent allowing for exhalation of that breath
- Inspiratory : Expiratory ratio
- Normal is 1:2
  - i.e. 2 sec in and 4 seconds out if RR 10 (6sec/breath)
- May need to decrease if air trapping to avoid auto/intrinsic PEEP (buildup of intrapulmonary pressure after inadequate exhalation)
  - COPD, Asthma, obstructive diseases (1:2.5 or 1:3)
- May increase with restrictive disease
  - ARDS/ALI/Sarcoidosis/Fibrotic lung diseases (1:1.5)
PEEP

- Positive End Expiratory Pressure
  - The pressure left in the airways at end expiration
- Promotes alveolar recruitment, increases FRC, and increases PaO2, among other benefits (later)
- 5cm H2O is a reasonable starting point and titrated to effect for oxygenation/maintenance of alveolar recruitment
- May reduce venous return to the heart by reduction in transmural pressure at higher levels (>10cmH2O) in patients with heart disease.
- Appropriate in patients with COPD (pursed lip breaths)
  - Low levels may help stent small airways open to allow better exhalation (reduce closing capacity)

Allain et al., 2010
Small airway closure during exhalation; PEEP may reduce this effect

Dynamic Airway Collapse

Forced expiration

Narrow cartilagenous bronchus (equal pressure point)

Elastic pull

Alveolus

$P_{al} = 1000 \text{ Pa}$

Elastic recoil

Expiratory effort --- Increased kinetic energy --- Reduced lateral pressure --- Dynamic Airway Collapse

Fig. 13-5

Note on PEEP

- PEEP is not the only culprit in reducing venous return to the heart
- Increased intrathoracic pressure and secondary HD effect is the result of elevated mean airway pressure
- If you need to use higher Vt or increased PIP to achieve adequate gas exchange, then the venous return to the heart is still limited (will also have a more pulsatile return and more respiratory variance in systemic BP)
- In addition, you have decreased alveolar recruitment without PEEP and increased risk for atelectotrauma, volutrauma, and hypoxic pulmonary vasoconstriction (coming up)
Detection of Air Trapping

- Up-sloping capnograph throughout exhalation rather than flat plateau
- Expiratory flow has not reached zero prior to next breath if displayed on ventilator
- More advanced ventilators can measure with an expiratory hold
- The patient can be disconnected from the vent and improved BP may be indicative of auto-PEEP (air trapping)

Hines & Marschall, 2012
Air trapping on capnogram
More on Obstructive Disease

- Reduced I:E ratio 1:2.5, 1:3
- Reduce Vt 6-8ml/kg IBW or less
- Reduce RR 8-12
- Permissive Hypercapnia
  - Usually not harmful, outweighs risk of air trapping
  - May assist in peripheral vasodilation
  - Unloading of O2 at tissue (Right shift in Hb curve)
  - Coronary vasodilation
  - Hypercapnia will avoid hypocapnic bronchoconstriction
- All will allow decreased auto-PEEP

Barash et al., 2013
• Increased I:E Ratio 1:1.5
• Increase PEEP to reduce FiO2
• Increase RR as needed to avoid high airway pressures (decreased stretch physiology)
• Consider modes that promote higher mean airway pressures (Inverse Ratio Ventilation, Bi-Level)-
discussion coming up
• High Frequency Oscillator Ventilation as a rescue mode
NPPV/CPAP

- CPAP: Continuous pressure delivered to the airways throughout the respiratory cycle in SV patient
- Can use BiPAP setting where there is inspiratory pressure over PEEP
- Beneficial alternative in cardiogenic pulmonary edema (CPE), COPD, and ALI in immunosuppressed patients (may reduce need for intubation, decrease VAP, reduce mortality)
- Increased mortality when used for respiratory failure post extubation, increased MI rates in CPE
- Consider in awake, cooperative, low aspiration risk, rapidly reversible cause, or obtunded due to high PaCO2

Barash et al., 2013
Oxygen titration

- Set FiO2 dependent on patient requirements
  - What is a reasonable SpO2/PaO2 for this patient?
- May reduce FiO2 by adding PEEP or other ventilator settings that promote increased mean airway pressure
  - ARDS/ALI/Aspiration
- May need to increased FiO2 if hypoxic pulmonary vasoconstriction is an issue (temporizing measure)
  - Need to find more definitive pharmacologic fix eventually
- Oxygen toxicity, especially in neonates
  - Retinopathy of Prematurity
  - Longer periods of high FiO2 in all patients will increase free radicals and may result in damage/inflammation
  - Debate as to high PEEP (>15cmH2O) vs higher FiO2 (>50%) if needed
    - Weigh benefit/risks of each for patient’s unique patho

Barash et al., 2013
Why Care about fancy modes in Anesthesia

- Does the patient need post-op mechanical ventilation? What mode is best then?
- What happens to the patient’s lungs with high Vt and no PEEP? This seems fine while in the OR?
- Is it necessary to keep my patient paralyzed and on full support or hyperventilated to avoid spontaneous breaths? What modes may allow smoother spontaneous breathing intra-op?
- Wait, I need to use the ICU ventilator because my machine doesn’t have settings that work for this ARDS/ALI/TRALI patient!?
Ventilator Modes Overview

- **Volume Control:**
  - Asynchronous
  - Set Vt
  - Set respiratory rate
  - Delivers breath until set volume is reached
  - Fluctuating inspiratory pressure
    - May need to increase RR or change mode if PIP of >30-40 cmH2O are consistently met to achieve adequate Vt.
    - Minute Ventilation will remain relatively constant.

Cereda, 2009; Allain et al., 2010
Ventilator Modes Overview

• Pressure Control:
  ○ Asynchronous
  ○ Set inspiratory pressure
  ○ Set respiratory rate
  ○ Delivers inspiratory pressure over inspiratory period (derived from I:E ratio)
  ○ Tidal volumes will fluctuate based on pulmonary/chest wall compliance
    ▫ Important to monitor Vt and CO2 clearance after pneumoperitoneum, position changes, changes in pulmonary compliance, periodically, etc...
    ▫ Minute Ventilation will fluctuate to some degree with compliance changes

Cereda, 2009; Allain et al., 2010
Ventilator Modes Overview

- **Assist Control Ventilation:**
  - Synchronized with patients efforts
    - Will ensure entire Vt or inspiratory pressure is delivered with or without patient initiation of breath
    - Maintains minimum set RR if patient is not initiating breaths
  - **Volume Limited** usually has slower inspiratory flows which may not match patients own effort, resulting in dysynchrony, excessive respiratory work, and fatigue
    - Patients inspiratory flow may be greater than speed vent can deliver breath
  - **Pressure Limited** may deliver inspiratory flows up to 180L/m or greater, eliminating the above mentioned problem, although
    - Vt will fluctuate and inspiratory pressure may need periodic adjustment

Cereda, 2009; Allain et al., 2010
PC vs VC

**Ventilator Modes Overview**

- **IMV/SIMV**
  - Intermittent mandatory set Vt breaths are delivered and patient is able to breath between breaths
    - Rate of mandatory breaths can be decreased as a weaning measure
  - SIMV-intermittent breaths will be supported with a set inspiratory pressure
  - Cannot guarantee consistent minute ventilation as intermittent breaths will cause fluctuations

*Cereda, 2009; Allain et al., 2010*
Inverse Ratio Ventilation

- I:E ratio is greater than or equal to 1:1 to promote longer inspiratory period and shorter expiratory period
- Mean airway pressure will be increased
- May cause significant air trapping in patients with obstructive disease
- Possible choice in patients with poor oxygenation on conventional settings

Allain et al., 2010
Ventilator Modes Overview

- **Airway Pressure Release Ventilation (APRV)**
  - Patient breaths spontaneously at a high airway pressure with intermittent releases to low or no PEEP
    - Maintains alveolar recruitment and oxygenation without alveolar over distention
    - $\text{CO}_2$ exchange is achieved by patient’s own breaths and intermittent release of pressure
      - Better $\text{O}_2$ diffusion with higher mean airway pressure
      - Must avoid in patients with COPD/Asthma due to air trapping
    - Used in patients with severe hypoxemia (ARDS/ALI)
    - Ability to exhale $\text{CO}_2$ may be limited without patients own spontaneous breaths over ventilator pressure changes

Cereda, 2009; Allain et al., 2010
APRV waveform
Ventilator Mode Overview

- **Bi-Level (Bi-Vent; Biphasic Positive Airway Pressure)**
  - Similar to APRV, but longer expiratory periods
  - Patient spontaneously breathes during both the high airway pressure and low airway pressure periods

- **Pressure Support Ventilation (PSV)**
  - Patient initiates all breaths which are then supported by a set inspiratory pressure
  - Backup mode required if patient does not have enough effort to maintain adequate ventilation
  - Great as a weaning mode, gaining popularity for this
  - Consider in SV patients intraoperatively, even with LMA

Cereda, 2009; Allain et al., 2010
PSV (note patient efforts on Paw graph-negative deflections)

http://www.respiratoryupdate.com/members/PSV_Pressure_Support_Ventilation.cfm
**High Frequency Oscillatory Ventilation**
- Delivers very high RR at small Vt (3-15Hz; 180-900 breaths/min)
- Mean airway pressure determines oxygenation
- Continuous gas flow determines CO2 clearance
- Can adjust amplitude of oscillations, gas flow, and mean airway pressure to achieve ventilation
- Used as a rescue mode in patients with severe pulmonary disease with refractory hypoxemia
  - Able to maintain a high mean airway pressure with minimal alveolar distention and reduced risk of trauma in patients with severe restrictive disease

Cereda, 2009
Which Mode is Best?

- Little evidence suggests one mode contributes significantly to major outcomes, BUT....
- There are select cases that may benefit from the PC ventilation vs VC ventilation
- More advanced modes in patients having difficulty with oxygenation/gas exchange on standard PC/VC/PS
- PEEP is beneficial to nearly all patients by reducing atelectotrauma and promoting/maintaining alveolar recruitment
- Lower Vt (6-8ml/kg PBW) may limit volutrauma/barotrauma and are beneficial for a number of specific cases

Barah et al., 2013; Cereda, 2009; Allain, 2010
Concerns with Mechanical Ventilation

- **HD instability secondary to increased IT pressure**
  - Decreased venous return, reduced CO, reduced BP
  - RV distension due to increased Pulmonary VR and thus septal shift to the left decreasing diastolic compliance of LV

- **Decreased IT pressure after discontinuation of MV**
  - Increased venous return and LV transmural pressure
  - In hypovolemic patients, CO may increase after d/c of MV
  - Patients with poor LV function may not tolerate surge of preload, resulting in pulmonary edema and myocardial ischemia
  - Abrupt decrease in high levels of PEEP may also cause this

- **Overall, not an issue in normovolemic patients without heart disease and lower levels of PEEP**

- **Patients with ventricular dysfunction will likely tolerate low levels of PEEP without adverse effects**

Allain et al., 2010
Transmural Pressure-Difference in pressure along two sides of a wall (think of alveoli here as the RV of the heart and outside being ITP)

http://chestpmk.wordpress.com/page/16/
Optimization of PEEP

- Opening of hypoventilated Alveoli, decreases hypoxic pulmonary vasoconstriction and improves PBF
- Decreased LV afterload with reduction of transmural pressure at optimal levels
- Optimal PEEP augments LV contractility and may allow greater elastic recoil for LV passive filling
- Higher levels of PEEP (12cmH2O) resulted in lower LVEDV in patients coming off bypass
  - Concluded that Echocardiographic measurements should be assessed at the same PEEP for diastolic assessment
  - MAP 83.5 at 0PEEP; 81.8 at 6PEEP; 75.9 12 PEEP
  - Trendelenberg negated the effects of PEEP on HDs

Juhl-Olsen et al., 2013
Optimization of PEEP

- Applying PEEP is a balance between maintaining alveolar recruitment and increasing oxygenation without impeding CV function.
- Some PEEP may actually improve CV function as previous slide indicates.
- High ITP, whether achieved by PEEP, high inspiratory pressure, or high mean airway pressure all result in HD effects.
- 5-10cmH₂O may improve PBF, alveolar recruitment, oxygenation, and maintain HD stability.
- PEEP is most effective if implemented after a recruitment maneuver (signs or sustained inspiratory pressure at 30cmH₂O for 30seconds).
Ventilator Associated Lung Injury (VALI)

- Historically 12-15ml/kg Vt, PEEP up to 25-30cm H2O were considered safe
  - ARDS mortality rate was 90% at this time
  - Many still use 10-12ml/kg (often not adjusted for PBW). Is this too much?

- What are the mechanisms of VALI?
  - Barotrauma
  - Volutrauma
  - Biotrauma
  - Atelectotrauma

Gattinoni et al., 2010
Barotrauma- pressure related injury
- Pneumothorax
- Pneumomediastinum
- Gas Emboli
  - All considered gross barotrauma

The transpulmonary pressure is the factor determining injury, not airway pressure

1980 Dreyfuss et al:
- Rats ventilated with extremely high airway pressure and straps to chest; other group with lower pressures and no straps
- Second group sustained lesions on lungs, strap group no injurious marking. VOLUTRAUMA comes into picture
- Group without straps had higher transpulmonary pressure compared to group with straps

Gattinoni et al., 2010
- $P_{AW(airways)} - P_{PL(pleura)} = P_{L(TP)}$; $ERS(resp\ system) = E_L + E_{CW(chest\ wall)}$
- If airway pressure is increased, pleural pressure must go up to maintain the same transpulmonary pressure ($P_L$) i.e. Diver at 10m needs 2 ATM of pressure in tank to inflate lungs without damage, but at surface, 2 ATM could cause significant damage
- Same concept with elastance of lungs. As elastance of lung goes up, increased airway pressure needed to generate same transpulmonary pressure against greater elastic resistance (i.e. restrictive disease)

Gattinoni et al., 2010
Transpulmonary Pressure

However: elastance of lung in ALI/ARDS is nearly the same as in healthy subjects, so higher airway pressures may still result in trauma.

Limiting pressures to 30-40cmH2O is appropriate in most cases.

In light of baro/volutrauma it is the over distention of lung tissue secondary to increased transpulmonary pressure that results in tissue damage and inflammation.

Higher pressures can result in gross barotrauma.

Gattinoni et al., 2010
• Biotrauma: unphysiological stress/strain results in release of cytokines, WBC recruitment, and inflammatory response in lungs secondary to mechanical forces

• Atelectotrauma: cyclic opening and closing of alveoli during mechanical ventilation may result in biotrauma
  ○ PEEP can be beneficial at reducing this cyclic airway closure, as long as it is able to stent the airways/alveoli open at end expiration (may benefit from recruitment maneuver prior to initiating PEEP)

Gattinoni et al., 2010
VALI

- Occurs when taking lungs closer to TLC and end expiration/FRC (high Vt, no PEEP)
  - Cyclic opening and closing and unphysiologic stretch
- FRC norm 3L; 1.7 L under anesthesia
  - Utility of PEEP to maintain norm
- At PIP 30cmH2O, 140cmH2O can occur between open and closed regions
  - PEEP/Recruitment maneuvers may reduce this shear pressure force by maintaining patent small airways/alveoli

Gattinoni et al., 2010
Regional Lung Stress

- Most strain noted in the dependent portion of lung (N=5)
- No difference in PC vs VC in supine position
- In the prone position VC ventilation reduced strain in dependent and increased strain in non-dependent regions (reducing strain gradient)
- Prone positioning in general reduced strain gradient between dependent and non-dependent regions
  - May explain benefits of prone patients in ICU
  - VC seemed to reduce strain gradient in this study while prone

Perchiazzi et al., 2011
Benefits of PC ventilation in Prone Spine Surgery

- PC ventilation during prone spine surgery reduced the PIP required to deliver same Vt compared to VC
  - 40 patients, 20 in each group
  - May have better delivery of breath via improved inspiratory flows during PCV compared to VCV
  - More effective recruitment throughout the breath with PCV?
- Higher pressures required with VC in this scenario may result in more regional strain for similar Vt
  - May increase risk for gross barotrauma if requiring higher pressures
  - Patients requiring higher PIPs may benefit from PC

Jo et al., 2012
Voluprotective Strategy for Intermediate to High Risk Abd Surgery Patients

- Control: 10-12ml/kg PBW, no PEEP, no recruitment maneuvers
- Voluprotective: 6-8ml/kg PBW, 6-8cmH2O PEEP, Recruitment 30cmH2O for 30 sec. every 30 min
- N=400 patients included
- 10.5% (exp) vs 27.5% (control) had major pulmonary complications
- 5% vs 17% required NPPV within 7d postoperatively
- Average hospital stay 2.45 days less in exp group

Futier et al., 2013
High Vt and no PEEP

- Mice subject to low Vt with PEEP; high Vt with PEEP; high Vt with no PEEP
- 4hr duration
- N=36
- Mice in the high Vt no PEEP group showed significant VALI; other groups did not
- Consider a synergistic mechanism of volutrauma and atelectotrauma during high Vt, no PEEP ventilation
  - This is common in anesthesia practice today
  - Do we know what’s happening to our patient’s lung down the road?

Seah et al., 2010
PEEP Matching for Intraabdominal HTN

- Porcine subjects with baseline, grade II IAH (18mmHg-24.5cmH₂O), grade III IAH(22mmHg-29.9cmH₂O)
- 7 subjects; all chemically induced ALI
- PEEP of 5cmH₂O, 0.5 x IAP, or 1 x IAP
- Control group tested with 5 and 15 cmH₂O
- Experimental PEEP: 5,12, 25 in grade II IAH; 5,15, 30cmH₂O in grade III IAH

Regli et al., 2012
PEEP in IAH and ALI continued...

- PEEP matching led to increased lung volumes and oxygenation
- Decreased shunt and dead space fraction
- High PEEP reduced CO
- Authors suggest applying moderate levels of PEEP to match IAP (0.5 x IAP) may be beneficial

Regli et al., 2012
• Grade II and Grade III IVH
• Low, Moderate, and High PEEP
• MAP 85, 75, 56 96, 87, 72
• HR 139, 139, 138 117, 112, 134
• CVP 6, 11, 17 5, 13, 18
• PAP 39, 38, 37 41, 41, 43

Regli et al., 2012
Laparoscopic Procedures Recommendations

- Increase $V_\text{E}$ 20-30% by increasing RR
- PC 6-8ml/kg PBW and PEEP 5-10cmH2O
- PEEP improved PaO2 during prolonged pneumoperitonium
- Prevent hyperventilation and hypocapnia (alkalosis)
- Mild hypercapnia ($\text{ETCO}_2 < 40$) can improve tissue oxygenation (Hb dissociation), increase CO, decrease SVR (peripheral vessel response to increased CO2-metabolic effect), and increase tissue perfusion

Barash et al., 2013
Obesity Recommendations

- Vt higher than 13ml/kg PBW have no benefit
- Plateau pressures may be difficult to maintain <30cmH2O (greater adipose tissue and weight on chest wall reduces compliance)
- Higher pressure may be tolerated due to decreased compliance (higher PIP is needed to achieve the same transpulmonary pressure)
  - Lungs resistant to over distention at higher PIP
- PC may correlate with better oxygenation

Barash et al., 2013
Obesity

- Moderate PEEP (10cmH2O), especially after recruitment maneuvers (3 short up to 40-55 cmH2O for 6 seconds) can improve V/Q matching and oxygenation
- Higher pressure with recruitment maneuvers needed to compensate for decreased chest wall compliance and achieve adequate transpulmonary pressure for alveolar recruitment
- 50% reduction in FRC compared to 20% in healthy adult in supine position after anesthesia

Barash et al., 2013
Which position requires less PIP?
One Lung Ventilation

- Common to use 10-12ml/kg PBW
- Higher FiO2 may cause absorptive atelectasis but considered appropriate (100%)
- 80/20% O2/N2O have been used
- 15ml/kg may shunt blood to unventilated lung, but may be used as intermittent recruitment maneuver
- Higher Vt may be associated with increased post-op respiratory failure (10-12ml/kg PBW)

Barash et al., 2013
OLV

- 6-7ml/kg PBW, PEEP (10cmH2O), frequent recruitment recommended for the dependent lung
- CPAP 5-10cmH2O to non-dependent lung
- These measures will improve V/Q matching
  - shunting open the non-dependent lung with some intrapulmonary pressure
  - improving oxygenation of the dependent lung, thus decreasing hypoxic/hypercapnic pulmonary vasoconstriction
- 18% develop ARF post pneumonectomy in general so important to consider
Hypocapnia

- Frequently instituted for the treatment of increased ICP
- CBF decreases 3% per mmHg decrease in PaCO2 in the range of 60-20mmHg CO2
- In as little as 6 hrs, buffering of CSF brings pH in CSF back to normal at the given PaCO2
- When patient’s CO2 is brought back to normal, significant rebound intracranial hypertension can occur
  - Especially dangerous if allowing patient to hypoventilate during emergence

Curley et al., 2010
Hypocapnia

- May cause cerebral hypoxia in the injured brain
  - “Inverse Steal”: injured areas may have increased CO₂ responsiveness, and vasoconstriction will divert blood away from ischemic regions
  - Increased CMRO₂ due to increased neuronal excitability (hypocalcemia-more protein binding of calcium)
  - Increased seizure activity in TBI
  - Bronchoconstriction
  - Attenuated hypoxic pulmonary vasoconstriction (more shunt)
  - Left shift of oxy-Hb dissociation curve
  - More anaerobic glycolysis
Hypocapnia

- **Acute Lung Injury from low CO2**
  - Increased volumes to hyperventilate
  - Increased lung capillary permeability and edema
  - Inhibition of surfactant
  - Potentiation of inflammatory response
Hypocapnia

- Effects on Heart
  - Lowers myocardial O2 deliver (Hb shift, vasoconstriction of myocardial vessels)
  - Increased coronary spasm (hypocalcemia and decreased vasodilation with lower CO2)
    - Classic variant angina that occurs when patients hyperventilate
  - Increased platelet levels and aggregation
  - Dysrhythmias
  - Potentiate digoxin toxicity (hypokalemia)
  - Hypokalemia, Hypocalcemia, Hypomagnesemia, Hypophosphotemia
  - Systemic hypotension (hypocalcemia, less SNS stimulation)
  - Arteriolar constriction and increased SVR (less CO2 induced dilation)
  - Increased capillary permeability

Curley et al., 2010; Barash et al., 2013
Hypocapnia

- Overall: save it until you need it
- PCO2 of 28 is a relatively safe level for temporary relief of increased ICP
- PCO2 of 23 results in decreased cerebral autoregulation
- PCO2 of 27-32 can produce critical reduction in blood flow to injured areas of brain tissue, consider risks and befits with TBI/Stroke

Curley et al., 2010
Effects of Hypercapnia (hypoventilation)

- **CNS**
  - Ventilatory stimulation
  - Unconsciousness at high levels
  - Cerebral vasodilation, increased ICP
  - Increased SNS tone, decreased PNS tone
  - Increased adrenal medullary and cortical output

Kregenow & Swenson, 2002; Hines & Marschall, 2012
Hypercapnia

- CV (opposing battle between direct and indirect)
  - Direct effects:
    - Impaired contractility of cardiac and smooth muscle
    - Reduced afterload and systemic vasodilation (decreased SVR)
    - Increased PVR
  - SNS effects:
    - Increased HR
    - Increased contractility
    - Increased venous tone and return
    - Increased CO
- Overall: at mild hypercapnia, SNS dominates, but CV collapse will occur at higher levels

Kregenow & Swenson, 2002; Hines & Marschall, 2012
Hypercapnia

- **Metabolism/Electrolytes/Blood**
  - Hyperkalemia
  - Insulin Resistance
  - Inhibition of anaerobic glycolysis
  - Right shift of oxy-Hb dissociation curve
  - Renal vasoconstriction at high levels
  - HCO3 reabsorption and H+ secretion
  - Suppressed erythropoietin release

- Hypercapnia at lower levels may be more beneficial than Hypocapnia by improving blood flow to vital organs, reduced afterload, offloading of O2 at tissue, decreased inflammatory response, less bronchoconstriction, and maintaining electrolyte balance

- **PATIENT SPECIFIC** as to which direction is better (i.e. pulmonary HTN, acidosis/alkalosis preexisting, ICP, etc...)

Kregenow & Swenson, 2002; Hines & Marschall, 2012
Readiness to Extubation

- Full reversal of NMB
- Following Commands (if appropriate)
- RR<30, $V_E < 10$LPM (norm 5-6LPM), $V_t > 300$ml
- RSBI (RR/$V_t$ in L)< 105LPM
- Max negative inspiratory pressure 30cmH20
- Vital Capacity of 10ml/kg (700-800ml)
- Clearing of underlying pathology requiring MV
- HD and pulmonary stability (acute/chronic problems)
- Neurological status that ensures protected airway
- *Consider criteria based on specific patient and scenario

Allain et al., 2010; Barash et al., 2013
Weaning and Extubation; Spontaneous Breathing Trials

- **PSV wean: progressively decreased support**
  - Proportional Assist Ventilation (newer ventilators have complex synchrony mechanism to deliver a continually adjusting breath to patient)
  - PS 15/5 to 10/5 to 5/5

- **T piece trials: Patient placed on a T piece with humidified oxygen and no support**
  - Can alternatively leave on vent with 0 support or PEEP (as is done in OR to assess readiness for extubation)
  - Concerns for atelectasis
  - Decreases the risk of premature extubation AND faster separation from mechanical ventilation is possible

Allain et al., 2010; Barash et al., 2013
30-120 minutes without support (or PS 5/5) indicates readiness for extubation

Subjective indicators of failure during SBTs: Dyspnea, fatigue, chest discomfort, anxiety, confusion, and restlessness

Objective evaluation: ventilators patterns (as prior), hemodynamics, and ABG may be useful

Assessment parameters should be guided based on situation (extubation of healthy patient in OR vs patient on vent x 4 days postop for ARF)

Allain et al., 2010; Barash et al., 2013
Conclusion

- Ventilator Mode not as important as maintaining Vt 6-8ml/kg PBW and PEEP
- Transpulmonary pressure and overdistention of the lungs (volutrauma) and cyclic opening and closing of alveoli (atelectotrauma) are the mechanisms of VALI HD effects at higher levels of PEEP (>10cmH2O)
- Obesity, Laparoscopy, OLV
- Hypocapnia is not benign, use when appropriate and temporarily
References


