A PHYSIOLOGICAL APPROACH TO ONE-LUNG VENTILATION

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OUTLINE

• physiology of lateral decubitus

• goals of one-lung ventilation

• lung collapse

• mechanisms and treatment of hypoxemia

• avoiding lung injury
WELCOME TO...
LATERAL DECUBITUS
Before anaesthesia

After anaesthesia
GOAL OF ONE-LUNG VENTILATION
ONE-LUNG VENTILATION

- OPTIMIZE COLLAPSE
- AVOID HYPOXEMIA
- AVOID LUNG INJURY

PROTOCOLIZED APPROACH
OPTIMIZING LUNG COLLAPSE
OLV

**LUNG ISOLATION**

to avoid contamination with blood, pus, secretions, lavage

- DLT

**LUNG SEPARATION**

functional – to optimize surgical exposure

- DLT

LEFT

- lesion left main stem bronchus
- (large thoracic aortic aneurysm)

RIGHT

- bilateral intervention
- pneumectomy
- sleeve lobectomy
- lobectomy
- lung transplantation (SLTX – SSLTX)

LEFT

- ETT – SLT in situ
- unanticipated OLV required during procedure
- (tracheal bronchus)

BB

- segmentectomy
- mediastinal surgery
- esophagectomy
- cardiac surgery
- other non-pulmonary surgery requiring OLV
DEVICE: DLT VERSUS BB

bronchial blocker: deflation of both lungs before inflation of BB

GAS MIXTURE: DE-NITROGENATION

FiO₂ = 0.4

SUCTIONING

Effect of Suction (-20 cmH20) on Lung Collapse

Complete Collapse

10 Min. Lung Collapse Score

Nil

Arndt
(+ = with suction, - = without suction)

DLT

AVOIDING HYPOXEMIA
## INCIDENCE

<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>Incidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tarhan et al.</td>
<td>1973</td>
<td>25%</td>
</tr>
<tr>
<td>Kerr et al.</td>
<td>1974</td>
<td>24%</td>
</tr>
<tr>
<td>Slinger et al.</td>
<td>1993</td>
<td>8%</td>
</tr>
<tr>
<td>Hurford et al.</td>
<td>1993</td>
<td>9%</td>
</tr>
<tr>
<td>Schwarzkopf et al.</td>
<td>2001</td>
<td>4%</td>
</tr>
<tr>
<td>Brodsky et al.</td>
<td>2003</td>
<td>1%</td>
</tr>
<tr>
<td>Ehrenfeld et al.</td>
<td>2008</td>
<td>10%</td>
</tr>
</tbody>
</table>
SIGNIFICANCE: POCD

increased risk POCD when SctO2 < 65%

Number of patients (%) vs. Minimal absolute SctO2 value:
- <55: 7
- 55-59: 15
- 60-64: 31
- >64: 47
PREDICTION OF HYPOXEMIA

55 y.o. F, Emphysema
FEV1= 28%

60 y.o. M, Lung Ca.
Non-smoker, FEV1= 98
PREDICTION OF HYPOXEMIA

• right versus left thoracotomy/scopy

• elastic recoil
  o low FEV1: airtrapping
  o prevention of atelectasis in dependent lung
  o delayed collapse of non-dependent lung
  o conflicting evidence

• preoperative PaO2
PREDICTION OF HYPOXEMIA: DISTRIBUTION OF PERFUSION
PREDICTION OF HYPOXEMIA: DISTRIBUTION OF PERFUSION

V/Q to the surgical side
PREDICTION OF HYPOXEMIA: END-TIDAL CO2 DIFFERENCE
MECHANISM OF HYPOXEMIA
MECHANISM OF HYPOXEMIA

- right-to-left shunt

Both ventilated areas of the lung and right-to-left transpulmonary shunt are illustrated. The shunt equation can be derived from the information presented in this diagram. $V_A$, alveolar ventilation.
DETERMINANTS OF HYPOXEMIA

• shunt equation
  ○ $Q_s/Q_t = \frac{(C_cO_2 - C_aO_2)}{(C_cO_2 - C_vO_2)}$
  ○ $C_aO_2 = C_cO_2 - (C_cO_2 - C_vO_2) \cdot (Q_s/Q_t)$

• mixed venous oxygen content
  ○ $C_vO_2 = C_aO_2 - (VO_2/Q_t)$

• $C_aO_2 = C_cO_2 - (VO_2/Q_t) \cdot \frac{(Q_s/Q_t)}{(10(1 - Q_s/Q_t))}$
DETERMINANTS OF HYPOXEMIA

• $\text{VO}_2$ : oxygen consumption (mixed venous saturation)

• $\text{Qs}/\text{Qt}$: shunt fraction

• $\text{Qt}$: cardiac output

• $\text{CcO}_2$: haemoglobin content – alveolar ventilation
SHUNT FRACTION

Two-Lung Ventilation vs One-Lung Ventilation

Nondependent Lung:
- Fractional Blood Flow: 40%
- \( P_{aO_2} = 400 \text{ mm Hg} \)
- \( Qs/Qt = 10\% \)

Dependent Lung:
- Fractional Blood Flow: 60%
- \( P_{aO_2} = 150 \text{ mm Hg} \)
- \( Qs/Qt = 27.5\% \)

non-dependent lung

dependent lung
The slopes of the plots are determined by the oxygen content differences between pulmonary end-capillary and the mixed-venous blood (CcO₂ - CvO₂). Note that, for any shunt fraction (Qs/Qt), the CaO₂ is less if CvO₂ (represented in the presence of a constant Hb by venous saturation) is decreased. Values used to plot these relationships are: hemoglobin concentration 15 g/dL, inspired oxygen fraction 0.5, and arterial carbon dioxide partial pressure 40 mmHg.
SHUNT: APPROACH TO NON-DEPENDENT LUNG: HPV

- optimizes V/Q (reduction 40%)
- contraction smooth muscle
- $\text{PAO}_2$ 40-100 mmHg
- determinants: $\text{PAO}_2$ and $\text{PvO}_2$
- early response 15 min
- maximal response 4 h
SHUNT: APPROACH TO NON-DEPENDENT LUNG: HPV and ANESTHETIC TECHNIQUE

• Halothane/ Enflurane

• Isoflurane/ Desflurane/ Sevoflurane

• Total Intravenous Anesthesia (TIVA)

• Combined TEA plus General Anesthesia?
SHUNT: APPROACH TO NON-DEPENDENT LUNG

- insufflation
- CPAP
- IPAP (intermittent positive airway pressure)
- modified CPAP
- HFJV
Limited use during thoracoscopic procedures due to decreased visualisation.
SHUNT: APPROACH TO NON-DEPENDENT LUNG: IPAP

- 6 aliquots of 70 ml
- $2\text{I}O_2$ – 2 sec – 8 sec
SHUNT: APPROACH TO NON-DEPENDENT LUNG: MODIFIED CPAP

Ku et al. JCVA 2009; 23: 850-852
SHUNT: APPROACH TO NON-DEPENDENT LUNG: HFJV

• one-lung ventilation
  o air-trapping
  o improved RV function

• as alternative for CPAP
  o optimal exposure

• to avoid one-lung ventilation (2-lung HFJV)
  o lower peak pressures

• Settings:
  o frequency +/- 180
  o pressure 1.8 – 2.2 bar
  o higher PCO2 levels
SHUNT: DEPENDENT LUNG

- HIGH TIDAL VOLUMES WITHOUT PEEP
- LOW TIDAL VOLUMES WITH PEEP
- RECRUITMENT BEFORE OLV

- PEEP
  - effect not predictable
  - evaluate oxygenation/compliance
SHUNT: APPROACH TO DEPENDENT LUNG: PEEP

SHUNT: APPROACH TO DEPENDENT LUNG: PEEP

Lower Inflection Point

Auto-PEEP
SHUNT: APPROACH TO DEPENDENT LUNG: PEEP

- Total PEEP increases less
- Dynamic hyperinflation
  - Shunt
  - Hemodynamic collapse
- Response not predictable

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**Graphical Representation:**

- **Lower Inflection Point**
- **Total PEEP**
- **Auto-PEEP**

**Axes:**
- **Volume (L.)**
- **Pressure (cm H2O)**

**Graph Notes:**
- The graph illustrates the relationship between pressure and volume, highlighting the points at which different PEEP levels are applied.
SHUNT: APPROACH TO DEPENDENT LUNG: PEEP

- Auto-PEEP
- Lower Inflection Point
SHUNT: APPROACH TO DEPENDENT LUNG: PEEP

- Total PEEP increases more
- Improved oxygenation
SHUNT: APPROACH TO DEPENDENT LUNG: PEEP

PEEP-responders (>20%)  
PEEP-NON-responders

not studied if PO2<60mmHg  
prediction not possible  
no recruitment maneuvers

The effect of PEEP* on several cardiovascular performance parameters. Part A summarizes values from the entire patient population, while part B summarizes values from the subpopulation of 20 patients in whom the FloTrac™/Vigileo™ monitor was utilized. Values are presented as mean ± SEp (pooled standard error).

*Positive end expiratory pressure; †Heart rate; ‡Mean arterial blood pressure; §Cardiac stroke volume; ‖Cardiac index; PEEP - Positive end-expiratory pressure.
SHUNT: APPROACH TO DEPENDENT LUNG: PEEP

• PEEP improves oxygenation

• mandatory when using lower volumes

• effect not predictable
  
  o observe clinical effect on oxygenation
  o observe improvement in compliance
SHUNT: APPROACH TO NON-DEPENDENT LUNG: RECRUITMENT

BEFORE OLV (2-LUNGS)

RECRUITMENT

20 – 40 cmH2O
- followed by PEEP
- improves oxygenation
- reduces inflammation
- transient decrease in CO – significance?
- repeated recruitment?

NON-DEPENDENT LUNG (OLV)

DEPENDENT LUNG (OLV)

Unzueta et al BJA 2012
Park et al EJA 2011; 28: 298-302
TV 10 ml/kg
TV 5 ml/kg PEEP 5
TV 5 ml/kg PEEP 0

NO RECRUITMENT MANEUVERS!
### SHUNT: DEPENDENT LUNG – VASODILATORS (NO)

<table>
<thead>
<tr>
<th>Study</th>
<th>Concentration</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wilson et al 1997</td>
<td>40 PPM</td>
<td>no decrease in PVR no improvement oxygenation</td>
</tr>
<tr>
<td>Fradj et al 1999</td>
<td>20 PPM</td>
<td>no decrease in PVR no improvement oxygenation</td>
</tr>
<tr>
<td>Rich et al 1994</td>
<td>20 PPM</td>
<td>decrease in PVR when light PH (25-35 mmHg)</td>
</tr>
<tr>
<td>Moutafis et al 1997</td>
<td>20 PPM + Almitrine (16y)</td>
<td>improved oxygenation</td>
</tr>
</tbody>
</table>

*no effect on PAP and oxygenation in absence of hypoxia or PH excessive vasodilation by isoflurane or thoracic epidural?*
EFFECT OF CARDIAC OUTPUT AND APPROPRIATE MANAGEMENT
The values used to plot these relationships are a FiO₂ of 0.5 and PaCO₂ 40 mmHg. Plot A: Hb 15 g/dl, VO₂ 150 ml/min, Qs/Qt 0.2; Plot B: Hb 15 g/dl, VO₂ 150 ml/min, Qs/Qt 0.4; Plot C: Hb 15 g/dl, VO₂ 75 ml/min, Qs/Qt 0.4; Plot D: Hb 10 g/dl, VO₂ 150 ml/min, Qs/Qt 0.2.

The values used to plot these relationships are at FiO₂ of 0.5 and PaCO₂ 40 mmHg. The curves have similar conditions to those specified in Fig. 4. Construction of this relationship used a lookup table in Excel relating CaO₂ to saturation for a particular Hb and then using an oxygen dissociation curve to relate saturation to PaO₂.
CARDIAC OUTPUT

PaO2

Qs/Qt

SvO2

100

Cardiac Output %
CARDIAC OUTPUT

• excessive increase in cardiac output
  
  o $\uparrow$MvSO$_2$ $\rightarrow$ $\downarrow$HPV
  
  o $\uparrow$PAP $\rightarrow$ $\uparrow$ perfusion of non-ventilated areas $\rightarrow$ $\downarrow$HPV
  
  o inotropes $\rightarrow$ $\downarrow$HPV
EFFECT OF OXYGEN CONSUMPTION AND APPROPRIATE MANAGEMENT
OXYGEN CONSUMPTION

- lower oxygen consumption: higher \( \text{MvSO}_2 \)

- high dose anesthetics beneficial
  - \(<>\) decrease in cardiac output
  - high opioids – low anesthetic

- inotropes
  - increase oxygen consumption
  - \(<>\) effect on cardiac output more pronounced
EFFECT OF CcO2 AND APPROPRIATE MANAGEMENT
HEMOGLOBIN CONCENTRATION

- effect on CcO$_2$

The values used to plot these relationships are a FiO$_2$ of 0.5 and PaCO$_2$ 40 mmHg. Plot A: Hb 15 g/dl, VO$_2$ 150 ml/min, Qs/Qt 0.2; Plot B: Hb 15 g/dl, VO$_2$ 150 ml/min, Qs/Qt 0.4; Plot C: Hb 15 g/dl, VO$_2$ 75 ml/min, Qs/Qt 0.4; Plot D: Hb 10 g/dl, VO$_2$ 150 ml/min, Qs/Qt 0.2.

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HEMOGLOBIN CONCENTRATION

- effect on $\text{CcO}_2$

- hemodilution
  - decreases $\text{PO}_2$ in COPD patients
  - effect on shunt - HPV?

- higher hematocrit (45%)
  - predicts low PO2
  - polycythemia due to higher shunt?
ALVEOLAR VENTILATION AND FiO2

- effect on CcO₂
- \[ PAO₂ = PiO₂ - (PaCO₂/RQ) \]

- increasing FiO₂
- (increasing alveolar ventilation)
- <= lower cardiac output
- <= increased shunting
- <= protective ventilation
EFFECT OF TEA

• decrease in CO (sympathicolysis)
• decrease in HPV
• effect still unknown
• less influence when low dose LA and maintenance of CO
• ropivacaine 0.75%: ↓PO2
• dexmedethomidine: ↑PO2
AVOIDING LUNG INJURY
EVIDENCE OF LUNG INJURY

- epithelial lining fluid /broncho-alveolar lavage/systemic
EVIDENCE OF LUNG INJURY

- one-lung ventilation induces
  - systemic inflammation
  - pulmonary inflammation dependent lung
  - pulmonary inflammation non-dependent lung
  - indication for neutrophilic injury
  - duration of one-lung ventilation
    - 100 – 300 min.
  - Peak AwP > 35 CmH2O
MECHANISM OF LUNG INJURY

Atelectrauma (open lung concept)
- Repetitive opening and closure of atelectatic zones
- Recruitment and PEEP

Overdistention (baby lung)
- Volutrauma in functional reduced lung volume
- Reduction in tidal volume

Zone of over-distension
Zone of atelectasis
LUNG INJURY IS MULTIFACTORIAL

- VENTILATION
- EXCESS FLUID ADMINISTRATION
- ANATOMICAL RESECTION
- INFLAMMATION
- CAPILLARY LEAK INTERSTITIAL FLUID
- REDUCED PULMONARY VASCULAR BED
- RIGHT HEART FAILURE
- REDUCED LYMPH FLOW
- ALI/ARDS
- NODAL DISSECTION
MECHANISM OF LUNG INJURY OLV

VENTILATED LUNG

- hyperoxia
  - reactive oxygen species
  - oxygen toxicity
- hyperperfusion
  - endothelial damage
  - vasculare pressure
- ventilatory stress
  - volutrauma
  - atelectrauma
  - barotrauma

COLLAPSED LUNG

OLV

- ischemia/reperfusion
- reexpansion
- cytokine release
- altered redox state

Surgery

- manipulation trauma
- lymphatic disruption

SYSTEMIC

cytokine release
- reactive oxygen species
- overhydration
- chemotherapy/radiation

ARDS/ALI
MECHANISM OF LUNG INJURY OLV: RISK FACTORS

• **PATIENT**
  - poor postoperative predicted lung function
  - preexisting lung injury
    - trauma
    - infection
    - chemotherapy
  - ethanol abuse
  - female gender

• **PROCEDURE**
  - lung transplantation
  - major resection (pneumonectomy > lobectomy)
  - esophagectomy – fluid administration
  - transfusion
  - prolonged OLV (>100 min) Peak pressure > 35-40 cmH2O
  - plateau pressure > 25 cmH2O
MECHANISM LUNG RE-EXPANSION

- Low FiO2!
- Gradual opening!
MECHANISM OF LUNG INJURY: HISTORICAL FACTS AND PITFALLS

• **high tidal volumes** 10 – 15 ml/kg
  o oxygenation
  o “end-inspiratory alveolar recruitment”

• **PPE** (postpneumonectomy pulmonary edema)

• low tidal volume in **ARDS** is beneficial

• **outcome**? surrogate markers

• effect of protective lung ventilation on **healthy lungs**
<table>
<thead>
<tr>
<th>protective OLV</th>
<th>conventional OLV</th>
<th>OUTCOME</th>
</tr>
</thead>
<tbody>
<tr>
<td>TV (ml/kg) – FiO2 – PEEP (cmH₂O)</td>
<td>TV (ml/kg) – FiO2 – PEEP (cmH₂O)</td>
<td></td>
</tr>
<tr>
<td>Ahn et al. Anaesth Intensive Care 2012</td>
<td>6 – 0.5 - 5</td>
<td>10 – 1 - 0</td>
</tr>
<tr>
<td>Yang Chest 2011</td>
<td>6 – 0.5 – 5 VCV</td>
<td>10 – 1 – 0 PCV</td>
</tr>
<tr>
<td>Licker Crit Care 2009</td>
<td>5 – 0.6 - 6</td>
<td>7 – 0.6 - 3</td>
</tr>
<tr>
<td>Schilling A&amp;A 2005</td>
<td>5 - 0.8 - 0</td>
<td>10 – 0.8 - 0</td>
</tr>
</tbody>
</table>
CONFLICT BETWEEN OXYGENATION AND PROTECTIVE VENTILATION?

OLV

$V_T = 8 \text{ ml kg}^{-1}$
PEEP = 5 cm H$_2$O

15 min

T0 (baseline)

Randomization

Group A

High volume
$V_T = 8 \text{ ml kg}^{-1}$
PEEP = 5 cm H$_2$O

Low volume
$V_T = 5 \text{ ml kg}^{-1}$
PEEP to keep the same $P_{\text{plat}}$

Group B

Low volume
$V_T = 5 \text{ ml kg}^{-1}$
PEEP to keep the same $P_{\text{plat}}$

10 min

T1

10 min

T2

Lower PO2

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Rozé BJA 2012
PROTECTIVE LUNG STRATEGY

- low tidal volume
  - 4-6 ml/kg

- PEEP
  - 5-10 cmH2O

- PROTECTIVE
  - lower shunt fraction
  - improved oxygenation
  - less atelectasis
  - lower cytokine release

Yang et al Chest 2011; 139: 530-537
Lung recruitment maneuver before OLV and ventilation with a $V_T$ of 5 ml/kg during OLV is associated with a more homogeneous distribution of lung tissue in the dependent ventilated lung.
MODE OF VENTILATION

• up to date no clear benefit for PCV or VCV

• more homogeneous distribution with PCV?

• historical impact of limited AwP
MODE OF VENTILATION

MODE OF VENTILATION

$P_{\text{peak}}$ (cm H$_2$O)

VCV
PCV

$P<0.0001$

Respirator
Bronchial

HYPERCAPNIA?

- consequence of protective ventilation
- reduces inflammation
  - subset analysis of ARDS Network
  - ↓alveolar-systemic cytokine release
  - ↓neutrophil accumulation
  - ↓radical injury
- improves tissue oxygenation
- improves SjO2 (50 mmHg)
- hypcapnia: induces acute parenchymal lung injury
EFFECT OF ANESTHETICS ON INFLAMMATION

• VOLATILE ANESTHETICS REDUCE INFLAMMATION
  o sevoflurane vs propofol
    • ↓plasma IL-6
    • Lee JCTVA 2012
    • ↓BAL IL-6
    • Sugasawa J Anesth 2012
    • ↓BAL IL-6, TNF-a, IL-8
    • De Conno Anesthesiology 2009
  o isoflurane vs propofol
    • ↓plasma and BAL IL-8 and TNF-a
    • Mahmoud Anesthesiol Res Pract 2011
EFFECT ON CLINICAL OUTCOME?
TAILORED - PROTOCOLIZED APPROACH
ONE SIZE DOES NOT FIT ALL
<table>
<thead>
<tr>
<th>parameter</th>
<th>target</th>
<th>remark</th>
</tr>
</thead>
</table>
| FiO2          | 0.9 - reduce to 0.5 if possible (after onset of HPV) | • adjust 5 min prior to OLV  
• less inflammation with lower FiO2  
• re-inflation of non-dependent lung with air + recruitment |
| Tidal Volume  | 4-6 ml/kg                                   | • reduce stretch                                                       |
| Respir Rate   | increase to maintain MV                     | • cave: increased Vd: higher RR necessary to maintain Va  
• cave: airtrapping if inadequate E-time: decrease RR  
• obstructive: I:E = 1:3 / restrictive: I:E = 1:1 |
| Pplat AwP     | limit to 25-30 cmH20                        | • allow hypercapnia if necessary  
• air leak with BB when higher than 25 cmH20                           |
| PEEP          | 5-10 cmH20                                  | • titrate to oxygenation (LIP)  
• reduces atelectasis – shear-stress  
• consider auto-PEEP  
• consider recruitment                                                   |
| PCO2          | 40 – 60 mmHg                                | • permissive hypercapnia is protective  
• permissive hypercapnia in case of airtrapping or high Pplat AwP      |
| ventilatory mode | PCV - VCV                                   | • until now, no evidence for beneficial effect of a specific mode. allow PpeakAwP to be higher during VCV  
• cave: pressure in circuit is higher than alveolar pressure.            |
HYPOXIA

1. increase FiO2 to 1.0
2. check position DLT/BB
3. optimize cardiac output
   • preload: 250-500 CC colloids
   • contractility
   • arrhythmias
4. recruitment dependent lung
   • AwP 20-30 cmH2O during 30 sec
   • followed by PEEP
   • cave: reduction in C.I.
5. optimize PEEP dependent lung towards LIP (↑ or ↓)
6. CPAP to non-dependent lung
   • recruitment first
   • 5-10 cmH2O
   • NOT during VATS (surgical exposure)
7. intermittent inflate non-dependent lung (communicate with surgeon)
8. partial ventilation of non-ventilated lung
   • lobar re-inflation
   • selective lobar collapse (BB)
   • oxygen insufflation (consider insufflation in surgical field, cave combustion)
   • (high frequency ventilation)
9. reduce blood flow to non-ventilated lung
   • clamping of pulmonary artery (cave increased afterload to right ventricle)
   • (inhaled NO) (lowering IV or inhalational anesthesia)
10. maintain oxygen carrying capacity
11. (ECMO as rescue)

MILD/GRADUAL (90%)

SEVERE (<85%)

1. resume 2-lung ventilation
2. (communicate with surgeon)
3. increase FiO2 to 1.0
4. check position DLT/BB

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THANK YOU