# **VENTILATION**

- Normal Physiology
- Ventilation
- Humidity and Humidification

#### INTRODUCTION

Normally, Alveolar Ventilation is unconsciously regulated to maintain constant arterial blood gas tensions, despite variable levels of oxygen consumption and CO2 production.

Many drugs and techniques used in anaesthesia interfere with control or mechanics of ventilation, and it is the Anaesthetists responsibility to ensure the adequacy of ventilation during the perioperative period.

Equipment related to ventilation is consequently of great importance to the Anaesthetist and Anaesthetic Technician. Correct use of the equipment relies on a good understanding of basic respiratory physiology as well as how the individual ventilator operates.

#### NORMAL PHYSIOLOGY

# **Basic Principles**

Venous blood always has a lower PaO2 (40 mmHg or 75% saturated or 15 ml O2/100ml blood) and higher PaCO2 (46 mmHg) than inspired gas (PiO2 150 mmHg, PiCO2 usually 0), so that there is normally a partial pressure gradient driving Oxygen in and CO2 out of the pulmonary capillary blood.

Ventilation of the lungs with inspired gases results in mixing of the inspired gases with alveolar gas.

If there is no ventilation at all, there will be no replenishment of oxygen and no removal of CO2, so the PAO2 will fall and PACO2 will rise towards the venous O2 and CO2 tensions.

If the ventilation is much greater than is needed, then the alveolar gas tensions will be much closer to inspired gas.

### **Definitions**

**Ventilation** is the process by which Oxygen and CO2 are transported to and from the Lungs.

**Tidal Volume** (VT) is the amount of gas expired per breath - typically 500ml at rest.

**Deadspace Volume** (VD) is the sum of the Anatomic Deadspace, due to the volume of the airways (typically 150ml), and Physiologic Deadspace, due to alveoli which are ventilated but not perfused (usually insignificant).

**Minute Volume** (VE) is the amount of gas expired per minute.

**Alveolar Ventilation** (VA) is the amount of gas which reaches functional respiratory units (ie, alveoli) per minute. VA = (Tidal Volume - Deadspace) x Respiratory rate

### **Lung Volumes**

- FRC (Functional Residual Capacity) 2.21.(supine)
- TLC (Total Lung Capacity) 6.21.
- Maximum Inspiratory Volume 4.0l. above FRC.
- ERV (Expiratory Reserve Volume) 1.0l. below FRC.
- RV (Residual Volume) 1.21.
- MVV (Maximal Voluntary Ventilation) 150 l/m.

# **Lung Mechanics**

#### **Inspiration**

An active process requiring musular effort; 75% diaphragmatic at rest; intercostals used on exertion.

Inspiratory effort causes:

- Fall in intrapleural pressure
- Fall in Alveolar pressure
- Pressure gradient from mouth to alveoli
- Gas flow down pressure gradient

Maximum inspiratory force sometimes used as an index of resp. effort; if < 20 cmH2O most patients have difficulty

### **Expiration**

Usually a passive process due to lung recoil:

- Relaxation of inspiratory muscles causes:
- Intrapleural pressure becomes less negative

- Alveolar pressure rises
- Pressure gradient from alveoli to mouth
- Gas flow down pressure gradient

### **Airway Resistance**

- Limits gas flow down airways
- Due mostly to airway/ETT diameter (fourth power of radius)
- Normal response to increased resistance is increased effort
- GA's increase resistance and decrease response, causing hypoventilation

# **Intrapleural Pressure**

- Normally -10 cmH2O, due to elastic recoil of lung opposed by chest wall.
- Becomes more negative on inspiration.
- Less at the dependent regions of the lung, reducing alveolar size.

# **Compliance**

"Static" Compliance is a measure of the "stiffness" of lung and chest wall, typically 50 ml/cmH2O in adults and proportionally less in kids. It is usually due equally to lung and chest wall compliances (100 ml/cmH2O each).

Surfactant improves lung compliance, especially at low lung volumes; its absence as in ARDS, results in stiff lungs and a tendency for the alveoli to collapse and fill with fliud.

"Dynamic" compliance includes the extra pressure needed to overcome resistance to airflow, inertia of chest wall, and viscoelasticity of tissues.

Total compliance varies from person to person and from time to time. A ventilator with pressure limited inspiration will deliver varying tidal volumes during an anaesthetic and from patient to patient. Most modern anaesthesia ventilators are of the "Volume Preset" type to minimise this problem.

### **Work of Breathing**

 $Work = Pressure \times Volume$ 

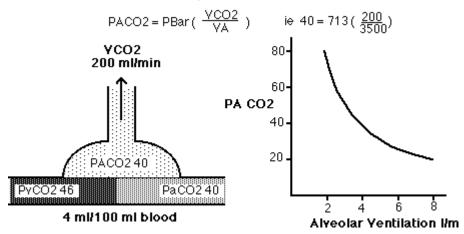
Respiratory work at rest or during exercise is seldom responsible for more than 5% of the total body work. Most of this is used to overcome the lung and chest wall stiffness during inspiration. Work to overcome airway resistance is usually very small, except during exercise or in athsmatics.

Patients with most respiratory diseases have increased respiratory workloads, which may be due to high respiratory rates, stiff lungs, or high airway resistances. When the

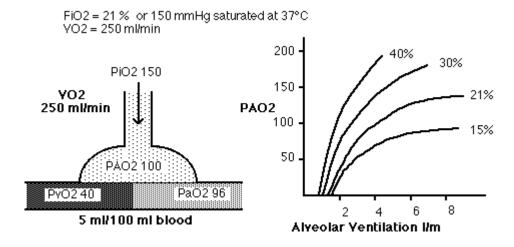
patient becomes so exhausted that they can no longer keep up the workload, respiratory failure ensues. Anaesthetic machine tubing, one-way valves, and ETTs all increase total resistance and respiratory work, while drugs will diminish respiratory effort, so that the patient with poor respiratory function usually requires ventilating both during and after the operation.

## **CO2** Elimination





# **Oxygen Transport**



### **Effect of Shunts**

Some venous blood passes through the lungs without equilibration with Alveolar gas. This "Venous Admixture" or "Shunt" subsequently mixes with oxygenated blood in the pulmonary veins, and has the effect of reducing PaO2 and elevating PaCO2.

While the slight rise in PaCO2 can be overcome easily by increasing the ventilation to normal alveoli, the same is not true for PaO2. For example, a 50% shunt needs 100%

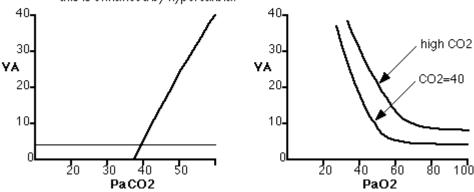
inspired oxygen to get a PaO2 of about 60 mmHg, but only a doubling of ventilation for normocarbia.

This is because the normal alveoli can blow off lots more CO2 than normal, but can never saturate the Hb any more than 100%.

## **Control of Ventilation**

Normal control of ventilation is by the arterial CO2 tension, and there is a steep slope on the VE/PaCO2 line (2 l/min per mmHg CO2).

Hypoxic ventilatory drive is minimal until the PaO2 is < 60, although this is enhanced by hypercarbia.



### Effects of Anaesthesia

- Impaired control of ventilation; volatile agents almost totally abolish hypoxic responses, narcotics, sedatives, anaesthetics impair CO2 responses
- Increased Deadspace (equipment and physiological)
- Increased work of ventilation due to:
- Increased circuit and airway resistance
- Decreased lung compliance
- Increased Shunt, leading to hypoxia, due to:
- Atelectasis of dependent parts of the lung
- Impaired sputum clearance (cilia, atropine, sedation, pain)
- Decreased FRC

#### VENTILATION

## Classification

- Mouth-to-Mouth/mask/ett etc
- IPPV "Conventional" Mechanical Ventilation
- PCV Pressure Control Ventialtion
- IMV Intermittent Mandatory (Volume) Ventilation
- MMV Mandatory Minute Ventilation
- SIMV -- Synchronised IMV ("Assisted")

- PRVC Pressure Regulated Volume Controlled (volume preset pressure ventilation; machine alters pressure on a breath by breath basis to generate the tidal volume set by the user)
- BiPAP Two-level CPAP (pt can breathe during inspiration and expiration)
- J et Ventilation (Sanders Injector)
- HFV High-Frequency Ventilation
- HFO High-Frequency Oscilation
- HFJV High-Frequency Jet Ventilation
- PEEP Positive End-Expiratory Pressure
- CPAP Constant Positive Airway Pressure (pt can breathe duting expiration)
- NPV Negative Pressure Ventilation
- TRIO Tracheal Insufflation of Oxygen
- Apnoeic oxygenation

### **Effects of IPPV/PEEP**

# Respiratory:

- Decreased PaCO2 due to increased Alveolar Ventilation
- Improved PaO2 (see previous graphs)
- Intrapleural Pressure less negative
- Work of breathing reduced
- Decreased lung water
- Optimum PEEP increases alveolar size, FRC, compliance, etc
- Hazards associated with intubation, paralysis or sedation, equipment failure.

#### Cardiovascular:

- Pressure gradient for venous return decreased whenever intrathoracic pressure rises
- CVP and peripheral venous pressure rise
- Reduced RV filling and increased RV afterload; opposite effects on LV
- May cause fall in Cardiac Output, particularly in hypovolaemic patients, causing reflex increase in contractility, heart rate, MVO2, vasoconstriction to augment venous pressure, reduced mixed venous oxygen tension, which may worsen aterial PO2

#### Renal

- Decreased renal function due to fall in Cardiac Output & Renal perfusion
- Increased ADH due to decreased central venous wall tension

#### Effects of CPAP/IMV or BiPAP

- Patient can breathe spontaneously; paralysis not always required
- Optimum CPAP and a low-resistance circuit reduces work of breathing

• Intrapleural pressure not as high as for IPPV so less depression of C.O.

But most IMV/CPAP systems do not maintain CPAP well and finding "Optimal" CPAP is difficult.

### **Ventilators**

#### Classification

- TYPE OF VENTILATION
- Positive/Negative
- OTHER CAPABILITIES
- PEEP/CPAP/IMV/MMV/HFV/HFO/HFJV etc
- CYCLING (reason inspiration commences)
- a) Automatic
- Time (Campbell, Bird)
- Pressure
- Other
- b) Manual c) Patient-Triggered
- INSPIRATION LIMIT
- Automatic
- Volume +/- Pressure limit (Bird with Bellows)
- Time (Campbell)
- Pressure (Bird)
- Flow
- Manual
- INSPIRATORY FLOW PATTERN
- Constant (Bird with Air-Mix control closed)
- Decelerating (Venturi-type, ie Campbell)
- Programmable
- Sinusoidal (Piston driven)
- CONTROL MECHANISM
- Pneumatic +/- Magnetic (Bird)
- Electronic (Servo)
- Fluidic Logic (Campbell)
- PATIENT CIRCUIT
- Single or Dual
- POWER REQUIREMENTS CONTROLS

#### Use in Anaesthesia

- Aim for normocarbia or slight hypocarbia
- Usually Volume preset IPPV devices
- Tidal volume and rate adjusted to suit patient (CO2 analysers useful)

#### With CO2 absorber ON:

- All inspired gas is free of CO2
- Effective ventilation depends only on Ventilator settings
- Very low Fresh Gas Flows may be used in the circle circuit

#### With the CO2 Absorber off:

- Provided that the ventilator settings deliver normal alveolar ventilation, the effective ventilation depends on Fresh Gas Flow.
- CO2 rebreathing occurs

#### Hazards

- Disconnection from circuit
- Failure to deliver ventilation
- Barotrauma

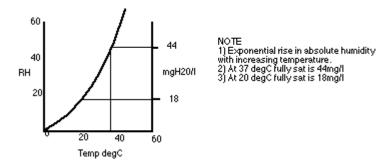
# **Monitoring Ventilation**

- Colour of the Patient
- Watching the chest move
- Precordial/Oesophageal Stethoscope +/- telemetry
- Listening to sound of ventilator
- Measurement of Circuit Parameters, such as pressure or tidal volume
- Measurement of Patient Parameters, such as ETCO2, SpO2, chest wall impedance,etc

# **HUMIDIFICATION**

# **Physics**

- Vapour Gas Phase of a liquid below boiling point
- Aerosol/Mist suspension of fine droplets of a liquid in a gas
- Absolute Humidity amount of water vapour per unit of gas (mg/l)
- Relative Humidity Absolute humidity of the sample as a % of the absolute humidity of fully saturated gas at the same temperature



# **Measurement of Humidity**

- Dew Point Hygrometer
- Hair Hygrometer
- Wet/Dry thermometer
- Humidity Sensors
- Measurement of water used by humidifier

# **Physiology**

The nose is a very efficient humidifier:

- 60% RH at the post-nasal space
- 5% RH in the mouth
- 100% at 37 degC in the bronchi

Mouth-breathing is less efficient (60% RH in the upper trachea)

Heat and water loss through the nose is minimised by cooling on inspiration and warming on expiration.

Humidification is required to maintain of ciliary activity, prevent squamous epithelial changes (Mucosal changes in 2-3 hours), prevent dehydration and thickening of secretions and possible ETT obstrucion, minimise atelectasis and tracheitis, and to decrease heatloss

#### **Methods**

### ANAESTHETIC CIRCUIT CONSIDERATIONS

- Cylinder gas is completely dry, and tracheal intubation bypasses the nose
- Waters CO2 absorber heats and humidifies gas very effectively
- Circle CO2 absorbers are of slight benefit only
- Bain circuit allows some warming but very little humidification

#### **HEAT AND MOISTURE EXCHANGERS**

- Relatively cheap
- 70%-80% effective humidification
- Increased deadspace, resistance, risk of disconnection

#### **HUMIDIFIERS**

Up to 100% humidification, essential for longterm respiratory care. Modern types heat both the water bath and patient hose to prevent rainout. Disadvantages:

- Cost
- Potential for leaks, disconnection
- Drowning if tipped
- Source of infection
- Unreliable
- Airway burns
- Increased airway resistance

# **EQUIPMENT**

### FISHER & PAYKEL

• Water heated to 37 degC, servo controlled hose heaters in newer units

# **GRANT - NICHOLAS**

• Water heated to 45 degC, hose servo to 37 degC. Inefficient at >101/min flows

#### **BOURNS**

• Basic Kettle type

# **NEBULISERS**

- Produce aerosols with humidity depending on temperature.
- Air is usually cooled by the droplets ->cold wet air
- Most useful for drug delivery

Comments? please use this form.