Interpretation of Arterial Blood Gases

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Before interpretation of ABG

- Make/Take note of
  - Correct puncture and transport
  - $F_1O_2$ : room air (0.21) or under $O_2$
  - Body posture : sitting or supine
  - At rest or during exercise
Arterial Blood Gases

- Measurement of oxygenation
  - $P_{a}O_{2}$
  - $S_{a}O_{2}$
  - $P_{A-a}O_{2}$

- Measurement of alveolar ventilation and acid-base status
  - $P_{a}CO_{2}$
  - pH
  - $[HCO_{3}^{-}]$
  - Base Excess (BE)
Normal Arterial Blood Gases

- Measurement of oxygenation
  - $P_{a}O_2$ 100 mmHg*
  - $S_{a}O_2$ 97 %
  - $P_{A-a}O_2$ <10 mmHg

* $P_{a}O_2 = 105 - (\text{age}/3)$
Normal Arterial Blood Gases

- Measurement of alveolar ventilation and acid-base status
  - $P_a\text{CO}_2$ 40 mmHg
  - pH 7.4
  - $[\text{HCO}_3^-]$ 24 mEq/L
  - Base Excess (BE) 0 mEq/L
Abnormal Arterial Blood Gases

- **Hyperoxemia**: high $P_aO_2$
  - No pathophysiological substrate
    - (except mild hyperoxemia in extreme alveolar hyperventilation)
  - Usually indicates
    - sampling error (air in blood sample) or
    - high $F_1O_2$ (oxygen administration)
Abnormal Arterial Blood Gases

- Hypoxemia: low $P_aO_2$
  - Desaturation: low $S_aO_2$
OXYHEMOGLOBIN DISSOCIATION CURVE

SO₂ (%)

CO₂ (vol %)

pH = 7.40
T = 37°C
PCO₂ = 40 mmHg
Hb = 15 gm/dl

PO₂ (mmHg)
Hypoxemia: causes?

Alveolo-arterial $O_2$ gradient

- $P_{A-a}O_2 = P_AO_2 - P_aO_2$
- $P_aO_2 = P_AO_2 - P_{A-a}O_2$
Hypoxemia: causes?

Alveolar gas equation

- \( P_{A\text{O}_2} = P_{I\text{O}_2} - \left[ P_{A\text{CO}_2} / R \right] \)
- \( P_{A\text{O}_2} = \left[ (P_b - P_{\text{H}_2\text{O}}) \times F_{I\text{O}_2} \right] - \left[ P_{A\text{CO}_2} / R \right] \)

- \( P_{A\text{O}_2} = \) partial pressure of oxygen in alveolar air
- \( P_{I\text{O}_2} = \) partial pressure of oxygen in inspired air
- \( P_{A\text{CO}_2} = \) partial pressure of carbon dioxide in alveolar air
- \( R = \) respiratory exchange ratio = \( V_{CO_2}/V_{O_2} \)
- \( P_b = \) barometric pressure
- \( P_{H_2O} = \) partial pressure of water vapour in inspired air
- \( F_{I\text{O}_2} = \) fractional concentration of oxygen in inspired air
Hypoxemia: causes?

Alveolo-arterial O₂ gradient

- \( P_{A-a}O₂ \) (mmHg) = \( P_AO₂ - P_aO₂ \)
- \( P_AO₂ \) is calculated using the alveolar gas equation
  - \( P_AO₂ = P_{I}O₂ - [P_{A}CO₂ / R] \)
  - \( P_AO₂ = [(P_b - P_{H2O}) \times F_{I}O₂] - [P_{a}CO₂ / 0.8] \)
  - \( P_AO₂ = [(760 - 47) \times 0.21] - [P_{a}CO₂ \times 1.25] \)
  - \( P_AO₂ = 149 - [P_{a}CO₂ \times 1.25] \)
- \( P_aO₂ \) and \( P_aCO₂ \) are measured (ABG)
- Normal \( P_{A-a}O₂ \)
  - < 5 – 10 mmHg (up to 20 mmHg in elderly)
Hypoxemia: 3 main causes

- \[ P_{A\,O_2} = [(P_b - P_{H2O}) \times F_{I\,O_2}] - [P_{A\,CO_2} / R] \]
- \[ P_{A-a\,O_2} = P_{A\,O_2} - P_{a\,O_2} \]
- \[ P_{a\,O_2} = P_{A\,O_2} - P_{A-a\,O_2} \]

- (1) Reduced \( P_{A\,O_2} \)
- (2) Increased \( P_{A-a\,O_2} \)
- (3) Reduced \( P_{v\,O_2} \)
Hypoxemia: causes (1)

- \( P_{A}O_{2} = [(P_{b} - P_{H2O}) \times F_{I}O_{2}] - [P_{A}CO_{2} / R] \)
- \( P_{a}O_{2} = P_{A}O_{2} - P_{A-a}O_{2} \)

- **Reduced \( P_{A}O_{2} \) (and normal \( P_{A-a}O_{2} \))**
  - Reduced \( P_{b} \): high altitude
  - Reduced \( F_{I}O_{2} \): inhalation of hypoxic gas mixtures
  - Increased \( P_{A}CO_{2} > \) hypercapnia
Hypoxemia: causes (2)

- $P_{A\ O_2} = [(P_b - P_{H_2O}) \times F_\text{I}O_2] - [P_{A\ CO_2} / R]$
- $P_{a\ O_2} = P_{A\ O_2} - P_{A-a\ O_2}$

- **Increased** $P_{A-a\ O_2}$
  - Failure of the lung as a gas exchanger
  - Oxygenation failure or Type I Respiratory Failure
  - Intrapulmonary mechanism/cause of hypoxemia

- Ventilation/perfusion mismatch
- Diffusion disturbance
- Right>Left shunt
Hypoxemia: causes (2)
Oxygenation Failure

- Ventilation/Perfusion mismatch
  - Obstructive lung diseases (COPD, asthma, ...)
  - Parenchymal lung disease (pneumonia, atelectasis, ILD, ...)
  - Vascular lung disease
Non-uniform ventilation normal perfusion (i.e. bronchial obstruction)

UNCOMPENSATED

M.V. = 6.0 liters

4.0 liters

NON-UNIFORM VENTILATION

UNIFORM BLOOD FLOW

mixed venous blood (A + B)

A

B

arterial blood (A - B)
Uniform ventilation non-uniform perfusion (i.e. pulmonary embolus)

UNCOMPENSATED

M.V. = 6.0 liters

V. = 4.0 liters

UNIFORM VENTILATION

NON-UNIFORM BLOOD FLOW

mixed venous blood (A + B)

arterial blood (A + B)
Hypoxemia: causes (2)

Oxygenation Failure

- Ventilation/Perfusion mismatch
- Diffusion limitation (on exercise)
  - Interstitial lung diseases
  - Emphysema
Thickened capillary alveolar interface (i.e. interstitial fibrosis)

M.V. 10.8 liters
T.V. 600 ml.
Freq. 18/min.

Sarcoidosis

W.M.

Dead space 110 ml. (assumed)

O. sat. 91%
Pco. 36 mm. Hg.
Hypoxemia: causes (2)

Oxygenation Failure

- Ventilation/Perfusion mismatch
- Diffusion limitation
- Anatomic R>L shunt
  - Intracardiac (ASD, VSD, ...)
  - Intrapulmonary (A-V malformations, fistulas)
Hypoxemia: causes (3)

- Decreased $P_vO_2$
  - Reduced cardiac output ($Q_T$)
    - Increased (tissue) oxygen extraction
  - Increased (tissue) oxygen consumption ($VO_2$)
Hypoxemia: consequences

- Desaturation → central cyanosis
- Chemoreceptor stimulation
  - ↑ Central respiratory drive
  - ↑ Output respiratory muscles
  - ↑ $V_E$ and ↑ WOB: dyspnea
  - ↑ $V_A$ and hypocapnia & respiratory alkalosis
Hypoxemia: consequences

- Tissue hypoxia
  - organ dysfunction: CNS, CV, kidneys
  - anaerobic metabolism $\rightarrow$ lactic acidosis
- Pulmonary vasoconstriction
  - Pulmonary hypertension
  - Cor pulmonale & right heart failure
- Increased renal erythropoietin production
  - Secondary polycythemia
Abnormal Arterial Blood Gases

- Hypercapnia: high $P_aCO_2$
- Respiratory acidosis: low pH
Abnormal Arterial Blood Gases

- Hypercapnia: high $P_aCO_2$
  - Respiratory acidosis: low pH
- Hypocapnia: low $P_aCO_2$
  - Respiratory alkalosis: high pH
Henderson-Hasselbalch equation

\[ \text{pH} = \text{pK} + \log \left[ \text{HCO}_3^- \right] / 0.03 \times \text{PCO}_2 \]

- Hypercapnia leads to decreased pH, i.e. Respiratory acidosis
- Hypocapnia leads to increased pH, i.e. Respiratory alkalosis
Hypercapnia: 3 main causes

\[ P_{a}CO_2 = \frac{VCO_2}{V_A} \]
- \( VCO_2 = \) CO\(_2\) production
- \( V_A = \) alveolar ventilation

1. Increased \( VCO_2 \)
2. Reduced \( V_A \)
3. Severe V/Q mismatching
Hypercapnia: causes (1)

- \( P_a \text{CO}_2 = \frac{V\text{CO}_2}{V_A} \)
  - \( V\text{CO}_2 = \text{CO}_2 \) production
  - \( V_A = \text{alveolar ventilation} \)

- **Increased** \( V\text{CO}_2 \)
  - Exercise with extreme effort
  - Fever and other hypermetabolic states
Hypercapnia : causes (2)

- $P_aCO_2 = \frac{VCO_2}{V_A}$
  - $VCO_2 = CO_2$ production
  - $V_A = alveolar ventilation$

- Reduced $V_A$ : alveolar hypoventilation
  - Failure of the respiratory system as an air pump
  - Ventilatory Failure or Type II Respiratory failure
  - Any hypoxemia is secondary to hypercapnia, i.e., the $P_A-aO_2$ is normal
  - Extrapulmonary mechanism/cause of hypoxemia
Hypercapnia: causes (2)

Ventilatory Failure

- \( P_a CO_2 = \frac{VCO_2}{V_A} \)
  - \( VCO_2 = CO_2 \) production
  - \( V_A = \) alveolar ventilation

- Reduced \( V_A \): alveolar hypoventilation
  - Reduced respiratory drive (central controller)
  - Dysfunction of respiratory neuromuscular apparatus
  - Chest wall disorders including severe obesity
  - Severe Upper Airway Obstruction
Hypercapnia: causes (2)

Ventilatory Failure

- Reduced respiratory drive (central controller)
  - CNS disorders
  - Drug (illicit or not) overdose
  - Metabolic disorders
  - Obesity-hypoventilation syndrome (Pickwick syndrome)
  - Central alveolar hypoventilation
Hypercapnia: causes (2)

Ventilatory Failure

- Dysfunction of respiratory neuromuscular apparatus
  - Motor neurons (ALS, Poliomyelitis, ...)
  - Peripheral nerves (Guillain-Barré, Phrenic neuropathy, ...)
  - Myoneural junction (Myastenia, drugs, ...)
  - Muscle (Myopathy, metabolic disorders, malnutrition, ...)
- Chest wall disorders including severe obesity
- Severe asphyxiating Upper Airway Obstruction
Hypercapnia: causes (3)

- Severe V/Q mismatch
  - Severely reduced efficiency of the lung as a gas exchanger
  - $\rightarrow \downarrow O_2$ uptake $\rightarrow$ hypoxemia
  - $\rightarrow \downarrow CO_2$ elimination $\rightarrow$ hypercapnia
  - $\rightarrow$ stimulation of chemoreceptors
    - $\uparrow$ Central respiratory drive and $\uparrow V_A$
      - $\rightarrow \uparrow$ WOB: dyspnea
      - $\rightarrow P_aCO_2$ decreases towards but does not reach normal
      - $\rightarrow$ less complete correction of $P_aO_2$ (poorly ventilated
        'shunt-like' regions keep bypassing venous blood)
Hypercapnia: consequences

- Hypoxemia with normal $P_{A-a}O_2$
- Respiratory acidosis: reduced pH
- If persistent:
  - $HCO_3^-$ retention by the kidneys
  - Increased $[HCO_3^-]$ and positive BE
  - Low pH will increase to almost normal (but not above 7.4)

“Compensatory metabolic alkalosis”

- Acutely, $\Delta HCO_3^- = 0.1 \times \Delta P_aCO_2$
- Chronically, $\Delta HCO_3^- = 0.35 \times \Delta P_aCO_2$
Clinical signs of hypercapnia

- CNS: cerebral vasodilatation
  - Increased cerebral Q
  - Intracranial hypertension
  - Papilledema, headache
  - Lethargy, confusion progressing to coma → ‘carbonarcosis’

- Peripheral vasodilatation
  - Full bounding pulses with
  - Warm, cherry-red skin
Abnormal Arterial Blood Gases

- Hypocapnia: low $P_a\text{CO}_2$
  - Respiratory alkalosis: high pH
Hypocapnia: 1 main mechanism

- $P_aCO_2 = \frac{VCO_2}{V_A}$
  - $VCO_2 = CO_2$ production
  - $V_A = \text{alveolar ventilation}$

- (1) Increased $V_A$
Hypocapnia ~ alveolar hyperventilation: causes

- Stimulation of chemoreceptors
  - Hypoxemia
  - Metabolic acidosis
- Pulmonary J-receptor stimulation
- Tissue hypoxia
  - Anemia, CO-, Sulf-, or MetHb
  - Hypotension, shock, sepsis
- Fever, thyrotoxicosis, strenuous exercise
- Psychogenic: hyperventilation syndrome
Hypocapnia: consequences

- Respiratory alkalosis: increased pH
- If persistent:
  - \( \text{HCO}_3^- \) excretion by the kidneys
  - reduced \([\text{HCO}_3^-]\) and negative BE (= base deficit)
  - Elevated pH will decrease to almost normal (but not below 7.4)

“Compensatory metabolic acidosis”

- Acutely, \( \Delta \text{HCO}_3^- = 0.2 \times \Delta P_a\text{CO}_2 \)
- Chronically, \( \Delta \text{HCO}_3^- = 0.5 \times \Delta P_a\text{CO}_2 \)
Abnormal Arterial Blood Gases

- **Acidosis**: low pH
  - Respiratory
  - Metabolic

- **Alkalosis**: high pH
  - Respiratory
  - Metabolic
Metabolic acidosis

- Reduced $\text{HCO}_3^-$, hence reduced pH and negative BE (base deficit), due to
  - Acid [$\text{H}^+$] accumulation (and buffering by $\text{HCO}_3^-$)
    - Renal failure
    - Diabetes mellitus: ketoacidosis
    - Tissue hypoxia/hypoperfusion (shock): lactic acidosis
    - Intoxications: ASA, antifreeze, methanol, paraldehyde
  - Loss of $\text{HCO}_3^-$
    - diarrhea
Metabolic acidosis

- If persistent, the increase in \([\text{H}^+]\) leads to:
  - Chemoreceptor stimulation
  - Increased central respiratory drive
  - Increased \(V_E\) and \(V_A\) : hyperventilation
  - Hypocapnia
  - The low pH rises towards normal (not exceeding 7.4)
    - “compensatory respiratory alkalosis”
    - \(\Delta P_a\text{CO}_2 = 1 \text{ à } 1.3 \times \Delta \text{HCO}_3^-\)
Metabolic alkalosis

- Increased $\text{HCO}_3^-$, hence increased pH and positive BE, due to
  - $\text{HCO}_3^-$ accumulation
    - Excessive ingestion of alkali
    - Drug intake: diuretics, corticosteroids
    - Cushing syndrome
    - Hypokalemia
  - Loss of acid
    - Loss of gastric acid: prolonged vomiting, nasogastric suction
    - Via kidneys
Metabolic alkalosis

- If persistent, the reduction in $[H^+]$ leads to:
  - Reduced chemoreceptor stimulation
  - Reduced central respiratory drive
  - Reduced $V_E$ and $V_A$: hypoventilation
  - Hypercapnia
  - The increased pH will decrease towards normal (but not below 7.4)
    - “compensatory respiratory acidosis”
    - $\Delta P_{aCO_2} = 0.6 \times \Delta HCO_3^-$
Arterial Blood Gases: main use

- Note sampling conditions
- Careful sampling and handling

- Detection of Respiratory Failure
- Detection of Acid-Base disturbances
Interpretation of ABG

- **Oxygenation**
  - Look at $P_aO_2$ and $S_aO_2$
  - Calculate $P_{A-a}O_2$

- **Alveolar ventilation**
  - Look at $P_aCO_2$

- **Acid-Base status**
  - Look at pH, $HCO_3^-$ and BE
### Classification of Respiratory Failure

<table>
<thead>
<tr>
<th>Acute</th>
<th>Type I</th>
<th>Type II</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypoxemia</td>
<td>Hypoxemia with increased $P_{A-a}O_2$</td>
<td>Hypoxemia with normal $P_{A-a}O_2$</td>
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<tr>
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<td>Respiratory alkalosis</td>
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<tr>
<td>Chronic</td>
<td>Hypoxemia</td>
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## Classification of Respiratory Failure

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<td></td>
</tr>
<tr>
<td>$P_aO_2$</td>
<td>Low</td>
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</tr>
<tr>
<td>$P_{A-a}O_2$</td>
<td>High</td>
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<tr>
<td>pH</td>
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<tr>
<td>BE</td>
<td>negative</td>
<td>positive</td>
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</table>
Thank you for listening
Hope you enjoyed it, or at least learned something

Prof. Dr. W. Vincken
Head Respiratory Division
AZ VUB
Interpretation of ABG

- Oxygenation
  - reduced $P_aO_2$ and $S_aO_2$  60/88%
  - = hypoxemia and arterial desaturation
Interpretation of ABG

- Alveolar ventilation
  - Increased $P_a\text{CO}_2 = \text{hypercapnia}$ 60
- Acid-Base status
  - Look at pH, $\text{HCO}_3^-$ and BE
Interpretation of ABG

- **Alveolar ventilation**
  - Increased $P_a$CO$_2$ = hypercapnia 60

- **Acid-Base status**
  - Reduced pH = acidosis 7.20
  - Normal HCO$_3^-$ and BE 24/0
  - = acute respiratory acidosis
Interpretation of ABG

- **Alveolar ventilation**
  - Increased $P_aCO_2$ = hypercapnia 60

- **Acid-Base status**
  - Minimally reduced pH = acidosis 7.36
  - Increased $HCO_3^-$ and BE 32/+8

  = chronic respiratory acidosis with compensatory metabolic alkalosis
Interpretation of ABG

- Alveolar ventilation
  - Reduced $P_aCO_2 = \text{hypocapnia}$

- Acid-Base status
  - Look at pH, $HCO_3^-$ and BE
Interpretation of ABG

- Alveolar ventilation
  - Reduced $P_aCO_2 = $ hypocapnia 30

- Acid-Base status
  - Increased pH = alkalosis 7.50
  - Normal $HCO_3^-$ and BE 24/0

= acute respiratory alkalosis
Interpretation of ABG

- Alveolar ventilation
  - Reduced $P_a\text{CO}_2 = \text{hypocapnia}$ 30

- Acid-Base status
  - Minimally increased pH = alkalosis 7.42
  - Reduced $\text{HCO}_3^-$ and BE 20/-4

= acute respiratory alkalosis with compensatory metabolic acidosis
Interpretation of ABG

- Alveolar ventilation
  - Normal $P_a\text{CO}_2 = \text{normocapnia}$ 40
Interpretation of ABG

- Alveolar ventilation
  - Normal $P_aCO_2 = \text{normocapnia}$
- Acid-Base status
  - Look at pH, $HCO_3^-$ and BE
Interpretation of ABG

- **Alveolar ventilation**
  - Normal $P_a CO_2 = \text{normocapnia}$ 40

- **Acid-Base status**
  - Reduced pH = acidosis 7.27
  - Look at $HCO_3^-$ and BE
Interpretation of ABG

- Alveolar ventilation
  - Normal $P_a CO_2 = $ normocapnia 40

- Acid-Base status
  - Reduced pH = acidosis 7.27
  - Reduced $HCO_3^-$ and BE 16/-7
= acute metabolic acidosis
Interpretation of ABG

- **Alveolar ventilation**
  - Reduced $P_aCO_2$ = hypocapnia 30

- **Acid-Base status**
  - Minimally reduced pH = acidosis 7.37
  - Reduced $HCO_3^-$ and BE 16/-7
  = chronic metabolic acidosis with compensatory respiratory alkalosis
Interpretation of ABG

- Alveolar ventilation
  - Normal $P_a CO_2 = \text{normocapnia}$ 40
Interpretation of ABG

- Alveolar ventilation
  - Normal $P_aCO_2 = \text{normocapnia}$ 40

- Acid-Base status
  - Look at pH, $HCO_3^-$ and BE
Interpretation of ABG

- Alveolar ventilation
  - Normal $P_a\text{CO}_2 = \text{normocapnia}$ 40

- Acid-Base status
  - Increased pH = alkalosis 7.50
  - Look at $\text{HCO}_3^-$ and BE
Interpretation of ABG

- **Alveolar ventilation**
  - Normal $P_aCO_2 = \text{normocapnia}$ 40

- **Acid-Base status**
  - Increased pH = alkalosis 7.50
  - Increased $HCO_3^-$ and BE 32/+8

= acute metabolic alkalosis
Interpretation of ABG

- **Alveolar ventilation**
  - Increased $P_aCO_2$ = hypercapnia
  - $P_aCO_2 = 50$

- **Acid-Base status**
  - Minimally increased pH = alkalosis
  - $pH = 7.43$
  - Increased $HCO_3^-$ and BE
  - $HCO_3^- = 32$ and $BE = +8$

= chronic metabolic alkalosis with compensatory respiratory acidosis
Classification of Respiratory Failure According to type of onset/duration

- Acute Respiratory Failure
- Chronic Respiratory Failure
- Acute on Chronic Respiratory Failure
Definition of Respiratory Failure

- Abnormal Arterial Blood Gases
  - Hypoxemia with $P_aO_2 < 60$ mmHg without or with
  - Hypercapnia with $P_aCO_2 > 50$ mmHg
- Without ABGs No Diagnosis of RF
Classification of Respiratory Failure According to type of ABG abnormality

- Respiratory Failure Type I
  - Hypoxemia without hypercapnia
- Respiratory Failure Type II
  - Hypoxemia with hypercapnia
Classification of Respiratory Failure According to type of ABG abnormality

- **Type I RF ~ Oxygenation Failure**
  - Failure of the lung as a gas exchanger ($O_2$ and $CO_2$)

- **Type II RF ~ Ventilatory Failure**
  - Failure of the respiratory system as an air pump

- **Mixed Failure**
Oxygenation Failure

- Reduced $P_aO_2$ (hypoxemia)
- Increased $P_{A-a}O_2$
- Reduced $P_aCO_2$ (hypocapnia)
- Increased pH (respiratory alkalosis)
- If persistent: compensatory metabolic acidosis (renal bicarbonate excretion)
Oxygenation Failure

- Abnormal Chest X-Ray
  - Diffuse pulmonary diseases
  - Localised pulmonary diseases
- Normal Chest X-Ray
  - Anatomic R>L shunts
  - Asthma (except for hyperinflation)
  - Pulmonary embolism/vascular disease
Ventilatory Failure

- Increased $P_a CO_2$ (hypercapnia)
- Reduced pH (respiratory acidosis)
- Reduced $P_a O_2$ (pro rata hypoxemia)
- Normal $P_{A-a}O_2$

If persistent: compensatory metabolic alkalosis (renal bicarbonate retention)
  - Acutely, $\Delta HCO_3^- = 0.1 \times \Delta P_a CO_2$
  - Chronically, $\Delta HCO_3^- = 0.35 \times \Delta P_a CO_2$
Uniform ventilation and perfusion

IDEAL

V = 4.0 liters

M.V. = 6.0 liters

UNIFORM VENTILATION

UNIFORM BLOOD FLOW

mixed venous blood
(A + B)

arterial blood
(A + B)

A

B
OXYGEN SATURATION (%)

\[ P_{50} = 26.6 \text{ torr} \]