Key Concepts in Oxygenation

Swimmers never take a breath for granted!

Nurses never take a life for granted!
Pulmonary Physiology

Physiology of Pulmonary System

- Ventilation and Perfusion
- Diffusion
- Relationship of Oxygen to Hemoglobin
- Oxygen Delivery to the Tissues
- Cellular Respiration
Ventilation

- **Definition:** The movement of air between the atmosphere and alveoli and the distribution of air within the lungs to maintain appropriate concentrations of oxygen and carbon dioxide in the blood.

- Process of ventilation occurs through inspiration and expiration.
Ventilation

- Pressure difference between airway opening and alveoli
  - Contraction of inspiratory muscles
  - Lowers intrathoracic pressure
  - Creates a distending pressure
  - Alveoli expand
  - Alveolar pressure is lowered
  - Inspiration occurs
  - Result: Negative pressure breathing

Ventilation

- Minute ventilation ($V_F$) = Total volume of air expired in one minute
  - Respiratory rate x tidal volume ($V_T$) (tidal volume = amount of air per breath)
  - Normal minute ventilation = $12 \times 500 \text{ ml} = 6000\text{ml}$
  - Note: (hypoventilation can occur with normal or even high respiratory rate)
Alveolar Ventilation ($V_A$)

- $V_A = V_T -$ anatomical dead space
- $V_A = $ Approximately 350 ml per breath
  - This is the ventilation that participates in gas exchange

**Anatomical dead space:**
Walls are too thick for diffusion
Mixed venous blood not present

Approximately 1 ml per ideal pound of body weight (150 ml)

Respiratory Anatomy

Conducting Airways: Resistance
- Nose
- Pharynx
- Larynx
- Trachea
- Right and Left Bronchi
- Non-Respiratory Bronchi

Gas Exchange Airways
- Respiratory Bronchioles (transitional zone)
- Alveolar Ducts
- Alveoli

$V_A$: Alveolar ventilation
### Alveolar Cells

<table>
<thead>
<tr>
<th>Type I (make up 90% of alveolar surface area)</th>
<th>Type II</th>
</tr>
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<tbody>
<tr>
<td>• Squamous epithelium</td>
<td>• Can generate into Type 1 cells</td>
</tr>
<tr>
<td>• Adapted for gas exchange</td>
<td>• Produces surfactant (allows alveoli to remain inflated at low distending pressures by decreasing surface tension, decreases work of breathing, detoxifies inhaled gases)</td>
</tr>
<tr>
<td>• Prevents fluid from entering alveoli</td>
<td>• Lipoprotein (phospholipid)</td>
</tr>
<tr>
<td>• Easily injured</td>
<td>• Hypoxemia / hypoxia may lead to decreased production or increased destruction</td>
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</tbody>
</table>

- **Type II**
  - Produces surfactant
  - Lipoprotein (phospholipid)
  - Hypoxemia / hypoxia may lead to decreased production or increased destruction
  - Metabolically active

**Alveolar Macrophages**
- Phagocytosis
Lung Volumes

Measurements including RV are made by helium dilution or body plethysmography, not spirometry.
Ventilation

- Work of Breathing Affected by:
  - Compliance (elastic work of breathing)
    - Lungs distend most easily at low volumes
    - Compliance is opposite of elastic recoil
  - Airway Resistance (flow resistance / resistive work of breathing)
    - Total resistance is comprised of tissue (20%) and airway resistance (80%)
    - Directly proportional to viscosity and length of tube / indirectly proportional to radius
    - Small airway resistance offset by numerous small airways (greatest resistance normally in medium bronchi)

Resistive work of breathing greatest during forced expiration.

Conditions Altering Ventilation

- Non Pulmonary Conditions
  - Drug overdose
  - Spinal cord injury
  - Brain injury

- Pulmonary Conditions
  - Decreased Compliance
  - Increased Resistance
Pulmonary Conditions Altering Ventilation

\textbf{\textit{Lung or Chest Wall Compliance}}

- Restrictive disorders (fibrosis, interstitial lung disease)
- Decreased surfactant production
- Atelectasis
- Pulmonary vascular engorgement
- Air, blood or excess fluid in pleural space
- Obesity / musculoskeletal disorders (chest wall compliance)

\textbf{\textit{Airway Resistance}}

- Obstructive Disorders
  - Asthma
  - Emphysema
  - Bronchitis
  - Foreign body causes a fixed obstruction
  - Sleep apnea can be obstructive
- Narrowing of airways (secretions / bronchospasm)

Improving Resistance and Compliance

\textbf{\textit{Airway Resistance}}

- Effective coughing
- Bronchodilators (albuterol) or steroids for bronchospasm
- Repositioning and suctioning to mobilize and aspirate secretions
- Decrease endotracheal tube resistance.
  - > 8 mm
  - Short tubes

\textbf{\textit{Lung / Chest Compliance}}

- Deep breath and hold
- Incentive spirometry (10 breaths per hour)
- Prevent abdominal distention / positioning
- Thoracentesis or chest tube for pleural effusion
- Diuretics for pulmonary edema
- CPAP
- PEEP (positive expiratory pressure)
Assessment of Ventilation

- Rate and depth of respirations
- Work of breathing
- Efficiency and effectiveness of ventilation is measured by PaCO$_2$ (inversely related to $V_A$)

  - PCO$_2$ > 45 mm Hg indicates alveolar hypoventilation *
  - PCO$_2$ < 35 mm Hg indicates alveolar hyperventilation

Note: Only one physiologic reason for increased PaCO$_2$.

Treatment of Ventilation Problems

VENTILATION PROBLEMS ARE TREATED BY RATE AND $V_T$

Options: Reverse sedation or underlying cause, ambu bag, BiPAP, or intubation and mechanical ventilation
More on Ventilation

- Normal ventilation on room air results in an alveoli with a partial pressure of oxygen of approximately 100 mmHg.

Untreated Alveolar Hypoventilation

Untreated alveolar hypoventilation will lead to hypoxemia. The hypoxemia is secondary to uncorrected alveolar hypoventilation.

In acute respiratory failure a blood gas is necessary to assess the PaCO\(_2\) to determine if inadequate ventilation contributed to the hypoxemia.
Perfusion

- Definition: The movement of blood through the pulmonary capillaries
Perfusion

Blood supply to lung

- **Pulmonary blood flow**
  - Entire output of right ventricle
  - Mixed venous blood
  - Gas exchange with alveolar air into pulmonary capillaries

- Bronchial blood flow
  - Left ventricle
  - Part of tracheal bronchial tree
  - Systemic arterial blood

Perfusion Fun Facts

- Pulmonary capillaries are slightly smaller than average erythrocyte
- Gas exchange actually starts in smaller pulmonary arterial vessels that are not true capillaries (functional pulmonary capillaries)
- 280 billion capillaries supply 300 million alveoli
- Potential surface area for gas exchange is 50-100 m²
- Alveoli are completely enveloped in pulmonary capillaries
- At rest each red blood cell spends only about 0.75 seconds in the pulmonary capillary. Less time during exercise.
Zones of Perfusion

- **Zone 1**: May be no blood flow. (alveolar deadspace – no zone 1 in normal breathing)
- **Zone 2**: Flow during systole.
- **Zone 3**: Flow during entire cardiac cycle.

Note: Zones are not static.

Zone 1 increased in positive pressure ventilation and PEEP.

Pulmonary Vascular Resistance

- **Comparison with systemic vascular resistance**
  - 1/10 of systemic vascular resistance
  - Pulmonary vascular resistance is evenly distributed between the pulmonary arteries, the pulmonary capillaries, and the pulmonary veins.

- **Relationship to pulmonary artery pressures and cardiac output**
  - Increase in cardiac output = Increase in PAP = Increased capillary recruitment = Decrease in PVR
  - Increased pulmonary artery pressure may lead to pulmonary edema

- **Relationship to lung volumes**
  - High lung volumes pull pulmonary vessels open. Results in a decrease PVR.
Pulmonary Vascular Resistance

- During positive pressure mechanical ventilation, both the alveolar and extra-alveolar vessels are compressed during lung inflation and PVR is increased.

- PEEP increases PVR further.

Hypoxic Pulmonary Vasoconstriction

- Diverts blood away from poorly ventilated alveoli
- Also occurs in response to more global hypoxia
  - Increases pulmonary artery pressure and recruits pulmonary capillaries to improve ventilation and perfusion matching
- Has limitations because of small amount of vascular smooth muscle in the pulmonary arteries
  - Hypoxic vasoconstriction greatly increases the workload of the right ventricle
- Increased pulmonary artery pressure may lead to pulmonary edema.
Conditions that Alter Pulmonary Perfusion

- #1 = pulmonary embolism (blocks perfusion distal to clot)
- Any decrease in cardiac output from right ventricle: shock
- Also remember:
- An increase in PVR for any reason can lead to right heart failure
- Increased pulmonary artery pressures can lead to pulmonary edema

Diffusion
Prior to Diffusion

- Ventilation and Perfusion Occur Simultaneously

Diffusion

- Movement of gases between the alveoli, plasma, and red blood cells
- Net movement of molecules from an area where the particular gas exerts a high partial pressure to an area where it exerts a lower partial pressure
- Different gases each move according to their own partial pressure gradients

*Diffusion of oxygen from alveoli to capillary determines the patient's oxygenation status*
Determinants of Diffusion

- **Surface Area:** negatively affected by any type of pulmonary resection; tumor, emphysema, pneumothorax
- **Driving pressure:** negatively affected by low inspired fraction of O2 (smoke inhalation) or by low barometric pressure (high altitudes)
  - Barometric pressure is the sum of the pressures of all the gases it contains
- **Thickness of alveolar capillary membrane (< 1 RBC):** negatively affected by pulmonary edema, pneumonia, or fibrosis
Assessment of Diffusion

- **PaO\textsubscript{2} and oxygen saturation (SaO\textsubscript{2})**
  - However, a simple diffusion problem rarely results in hypoxemia at rest.

- **Clinical Application:** CO\textsubscript{2} is 20 times more diffusible than O\textsubscript{2} - so a diffusion problem causing hypoxemia does not result in the same problem with CO\textsubscript{2} retention (hypercapnia)

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Increased FIO\textsubscript{2} and increased pressure (CPAP / PEEP) will increase driving pressure of oxygen.
Ventilation versus Diffusion
Assessment and Treatment

- **Ventilation problems**
  - Assessed by:
  - Corrected with?

- **Diffusion problems**
  - Assessed by:
  - Corrected with?

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Ventilation and Perfusion Ratios

Alveoli in upper regions have greater volume and are less compliant. Alveoli in lower parts of lung have a greater change in volume during inspiration and are considered better ventilated.
Ventilation / Perfusion Ratio (V/Q)

- **Ventilation (V)**
  - Alveolar minute ventilation = 4 to 6 L

- **Perfusion (Q)**
  - Normal cardiac output = 5 L

Normal ventilation / perfusion ratio (V/Q ratio) = 0.8 to 1.2

Ventilation and perfusion must be matched at the alveolar capillary level
Decreased V/Q Ratio: Intrapulmonary Shunting

- **Intrapulmonary shunt** occurs when there is significant alveolar hypoventilation in relation to normal perfusion (Example: Poorly ventilated alveoli in ARDS)
- V/Q ratio < 0.8

**Result**
- Poorly oxygenated blood returns to left side of heart resulting in low PaO2 and SaO2 (oxygenation problem)
Increased V/Q Ratio (Dead Space)

In increased ventilation perfusion ratio:
- Alveolar $O_2$ will rise
- Alveolar $CO_2$ will fall

Increased V/Q Ratio: Alveolar Dead Space

- Alveolar dead space: When ventilation is greater than perfusion
- V/Q ratio > 0.8
- Classic example: Pulmonary Embolus
Causes of V/Q Mismatching

- Non uniform ventilation
  - Uneven resistance
    - Collapsed airways (Emphysema)
    - Bronchoconstriction (Asthma)
    - Inflammation (Bronchitis)
  - Uneven compliance
    - Fibrosis
    - Pulmonary vascular congestion
    - Atelectasis

- Non uniform perfusion:
  - Pulmonary Emboli
  - Compression of pulmonary capillaries (high alveolar pressures)
  - Tumors
  - Shock (pulmonary vascular hypotension)

Assessing Oxygenation

- Cannot assess PaO₂ (arterial) without considering alveolar oxygenation content (PAO₂)
  - Increase in FIO₂ will increase PAO₂
  - Increase in PACO₂ will decrease PAO₂

Note: With normal diffusion the majority of oxygen in the alveoli should diffuse across the alveolar capillary membrane.
ALVEOLAR O\(_2\)

Cannot directly measure the amount of oxygen in the alveoli. It is a calculated value.

**Alveolar Gas Equation:**
\[ \text{PAO}_2 = \text{FIO}_2 \times (\text{PB} - 47) - \text{PaCO}_2 / .8 \]

- \(\text{PAO}_2\) = partial pressure of alveolar oxygen
- \(\text{FIO}_2\) = fraction of inhaled oxygen
- \(\text{PB}\) = barometric pressure
- \(47\): PH\(_20\) = water vapor pressure
- \(\text{PaCO}_2\) = partial pressure of arterial carbon dioxide
- \(.8\) = respiratory quotient

Importance of FIO\(_2\)

Normal arterial oxygen content of 80-100 mm Hg is only normal when the FIO\(_2\) is 21% (.21)

To calculate the expected \(\text{PaO}_2\) for any given FIO\(_2\), use this formula:
\[ (\text{FIO}_2 \% \text{ (do not use decimal)} \times 6) - \text{PaCO}_2 \]

Example:
FIO\(_2\) of 100% with PaCO\(_2\) 40 mm Hg
\[ (100 \times 6) - 40 = 560 \text{ mm Hg} \]
**PaO₂ and FIO₂ Ratio**

- An assessment and trending tool
- PaO₂/ FIO₂ ratio:
  - Normal well above 300
  - Acute lung injury < 300
  - ARDS < or = 200

**PaO₂ of 60 mmHg with an FIO₂ of 0.5 (50%)
represents a PaO₂/FIO₂ ratio of
60 / 0.5 = 120.**

This is a clinically significant intrapulmonary shunt.

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**Linking Knowledge to Practice with PaO₂ / FIO₂ Ratios**

<table>
<thead>
<tr>
<th>PaO₂</th>
<th>FIO₂</th>
<th>Ratio</th>
<th>Treatment / Notes</th>
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A (Alveolar) – a (arterial) Gradient (Difference)

- Provides an index regarding diffusion.
- The majority of what is in the “A” should end up in the “a”.
- A large A-a gradient generally indicates that the lung is the site of dysfunction.
- Normal A-a Gradient is small = 5 to 15 mm Hg

Hypoxemia

- Causes
  - Untreated alveolar hypoventilation
  - Diffusion abnormality
  - Ventilation and perfusion mismatching

- Assessment Clues
  - PaCO₂ – Ventilation
  - PaO₂ / SaO₂
  - PaO₂ / FIO₂ ratio
  - A-a gradient
Hypoxia and Hypoxemia

- **Hypoxemia**
  - Insufficient oxygenation of the blood
    - Mild: PaO2 < 80 mm Hg or SaO2 95%
    - Moderate: PaO2 < 60 or mmHg or SaO2 90%
    - Severe: PaO2 < 40 mmHg or SaO2 75%

- **Hypoxia**
  - Insufficient oxygenation of tissues
    - Determined by cardiac index, Hgb, SaO2, cellular demand, patency of vessels

Oxygen Transportation

- Oxygen is transported both physically dissolved in blood and chemically combined to the hemoglobin in the erythrocytes

- **Hemoglobin**: 97% of oxygen is combined with hemoglobin
  - Represented by the SaO2

- **Plasma**: 3% of oxygen is dissolved in plasma
  - Represented by the PaO2 (measurement of O2 tension in plasma)
Oxygen Delivery to Tissues

Transport of Gases in the Blood

- **Definition:** movement of oxygen and carbon dioxide through the circulatory system; oxygen being moved from the alveolus to the tissues for utilization and carbon dioxide being moved from the tissues back to the alveolus for exhalation.
Oxygen Delivery To Tissues

- Oxygen delivery measured as $DO_2$: Volume of oxygen delivered to tissues each minute

- $DO_2 = \text{cardiac output} \times \text{arterial oxygen content} \ (\text{hemoglobin} \times \text{arterial oxygen saturation})$

Formula for Oxygen Delivery

- $DO_2$ formula = $CO \times Hgb \times SaO2 \times 13.4 \ (\text{constant})$

- Normal $DO_2 = 900 - 1100 \ \text{ml/min}$ (1000)

- Normal $DO_2I = 550 - 650 \ \text{ml/min}$
Inadequate Delivery: Hypoxia

- Circulatory hypoxia
  - Inadequate cardiac output

- Anemic hypoxia
  - Inadequate hemoglobin

- Respiratory hypoxia
  - Inadequate $\text{SaO}_2$

Improving Oxygen Delivery

- Oxygen delivery can be improved by increasing cardiac output, hemoglobin or $\text{SaO}_2$

Some interventions more effective in clinical practice; interventions can be performed simultaneously.
Oxygen Consumption

- Measured as \( \text{VO}_2 \)

- Volume of oxygen consumed by the tissues each minute

- Determined by comparing oxygen content in arterial blood to the oxygen content in mixed venous blood
  - Normal \( \text{CaO}_2 \) is 20 ml/dl and normal \( \text{CVO}_2 \) is 15 ml/dl

- Normal \( \text{VO}_2 \): 200 – 300 ml / min (250 ml / min)

Causes of Increased \( \text{VO}_2 \)

- Fever per 1 degree C : 10%
- Shivering : 50-100%
- Suctioning : 7-70%
- Sepsis : 5-10%
- Non Family Visitor : 22%
- Position Change : 31%
- Sling Scale Weight : 36%
- Bath : 23%
- CXR : 25%
- Multi Organ Failure : 20-80%
Oxygen Reserve in Venous Blood

- Measured by mixed venous oxygen saturation (SVO$_2$)
- Normal 60-80% (75%)

- Tissues were delivered 1000 ml / min (DO$_2$)
- Tissues uses 250 ml / min (VO$_2$)
- This leaves a 75% reserve in venous blood
- Oxygen Extraction Ratio (O$_2$ER) = 25%

Oxygen Consumption and Oxygen Delivery

- Oxygen delivery and oxygen consumption are independent until a critical point of oxygen delivery is reached

- Tissues will extract the amount of oxygen needed independent of delivery because delivery exceeds need
Relationship of Delivery to Consumption

<table>
<thead>
<tr>
<th>DO₂</th>
<th>VO₂ (extraction is independent of delivery)</th>
<th>SVO₂ (SV0₂ will improve when you increase delivery)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1000 cc</td>
<td>250 cc (25%)</td>
<td>75%</td>
</tr>
<tr>
<td>750 cc</td>
<td>250 cc (33%)</td>
<td>67%</td>
</tr>
<tr>
<td>500 cc</td>
<td>250 cc (50%)</td>
<td>50%</td>
</tr>
</tbody>
</table>

Relation of Delivery to Consumption

- When oxygen delivery reaches a critical level then consumption will depend on delivery
- SVO₂ will not increase with increased delivery while you are in this dependent state
- Anaerobic metabolism occurs here because you have an oxygen deficit
SVO₂ Monitoring

- Global indicator between oxygen supply and demand
- Influenced by oxygen delivery and oxygen extraction
- Reflects mixing of venous blood from superior vena cava, inferior vena cava and coronary sinus
- Measured using a pulmonary artery fiberoptic catheter

Significant Changes In SVO₂

- SVO₂ < 60%
  - Decreased delivery
  - Increased consumption
- SVO₂ > 80%
  - Increased delivery
  - Decreased demand
  - Sepsis (tissues cannot extract)
  - Wedged catheter
- Clinically significant change is ± or – 5 to 10% over 3 to 5 minutes

- SVO₂ < 40% represents limits of compensation and lactic acidosis will occur (oxygen demand is greater than oxygen delivery and reserve can be depleted = oxygen debt)
ScVO2

- ScVO2 reflects oxygen saturation of blood returning to right atrium via the superior vena cava.
  - Can be obtained without a pulmonary artery catheter, using a modified central venous catheter with fiberoptic technology.
  - Normal value is > 70%.
  - ScVO2 trends higher than SVO2 but trends with SVO2.

Cellular Respiration

- Definition: Utilization of oxygen by the cell
- Estimated by the amount of carbon dioxide produced and amount of oxygen consumed
- Oxygen is used by the mitochondria in the production of cellular energy – prolonged oxygen deficit can result in lethal cell injury
Acid–Base Balance

ABG Analysis: Parameters

- pH
  - **Normal 7.35-7.45**
  - < 7.35 Acidosis
  - >7.45 Alkalosis

- PaCO2
  - **Normal 35-45 mm Hg**
  - < 35 alkalosis or respiratory compensation for metabolic acidosis
  - >45 acidosis or respiratory compensation for metabolic alkalosis
## ABG Analysis: Parameters

### HCO₃⁻
- **Normal 22-26 mEq/L**
- < 22 metabolic acidosis or metabolic compensation for respiratory alkalosis
- > 26 metabolic alkalosis or metabolic compensation for respiratory acidosis

### Base Excess (BE)
- **Normal +2 to −2**
- < -2 (base deficit) metabolic acidosis or metabolic compensation for respiratory alkalosis
- > +2 metabolic alkalosis or metabolic compensation for respiratory acidosis

### Pao₂
- **Normal 80-100 mm Hg**
- >100 hyperoxemia
- < 80 mild hypoxemia
- < 60 moderate hypoxemia
- < 40 severe hypoxemia

### Sao₂
- **Normal 95% or >**
- < 95% mild desaturation of HGB
- < 90% moderate desaturation of HGB
- < 75% severe desaturation of HGB
Compensation

An acidosis or alkolosis for which there has been compensation causes the pH to return to the normal range while **leaning toward the initial disorder**.

The body never overcompensates. A non leaning pH with two abnormal indicators suggests a mixed disorder (one alkalotic and one acidotic process).

Anion Gap

- The anion gap is used to help determine the cause of the patient’s metabolic acidosis.
- **Anion Gap = Na+ - [Cl- +HCO3-]**
- A normal anion gap is 12 + or – 4 mEq/L.
- An increased anion gap typically indicates an increased concentration of anions other than Cl- and HCO3-.
  - Lactic acidosis
  - Ketoacidosis
  - Renal retention of anions
More on Anion Gap

- Most common etiology of normal anion gap acidosis: Diarrhea.
- Second most common: Renal tubular acidosis.
- Both result in a loss of bicarbonate ions.
- To compensate there is an increase in plasma chloride.
- Normal ion gap acidosis is often referred to as hyperchloremic acidosis.

Common Causes of Respiratory Acidosis

- Depression of respiratory control centers
- Neuromuscular disorders
- Chest wall restriction
- Lung restriction
- Airway obstruction
- Pulmonary parenchymal disease
Common Causes of Respiratory Alkalosis
- Central nervous system disorders
- Drugs
- Hormones
- Bacteremia
- High altitude
- Over mechanical ventilation
- Acute asthma
- Pulmonary embolism

Common Causes of Metabolic Acidosis
- Ingested toxic substances
- Loss of bicarbonate ions
- Lactic acidosis
- Ketoacidosis
- Renal failure
Practice ABGs

ABG Analysis Practice

- pH           7.30
- PaCO₂        54 mmHg
- HCO₃         26 mEq/L
- PaO₂         64 mmHg
ABG Analysis Practice

- pH 7.48
- PaCO2 30 mmHg
- HCO3 24 mEq/L
- PaO2 96 mmHg

ABG Analysis Practice

- pH 7.30
- PaCO2 40 mmHg
- HCO3 18 mEq/L
- PaO2 85 mmHg
ABG Analysis Practice

- pH 7.50
- PaCO2 40 mmHg
- HCO3 33 mEq/L
- PaO2 92 mmHg

ABG Analysis Practice

- pH 7.35
- PaCO2 54 mmHg
- HCO3 30 mEq/L
- PaO2 55 mmHg
ABG Analysis Practice

- pH: 7.21
- PaCO2: 60 mmHg
- HCO3: 20 mEq/L
- PaO2: 48 mmHg

ABG Analysis Practice

- pH: 7.54
- PaCO2: 25 mmHg
- HCO3: 30 mEq/L
- PaO2: 95 mmHg
Reflection