### Objectives:

1. Review respiratory anatomy.
2. Understand mechanics of breathing, gas pressure vocabulary, and the principles of surface tension, compliance, and recoil.
3. Respiratory disorders and spirometry
4. How gas exchange occurs between the alveoli & pulmonary vessels, and between capillaries & tissue.
5. Regulation of breathing (voluntary vs involuntary)
6. Hemoglobin & hemoglobin disorders

### 1. Respiratory Anatomy - REVIEW

#### 2 Zones of Respiratory System:
1) Conduction zone, 2) Respiratory zone

1) **Conduction Zone** = from oral/nasal cavities to,
- Pharynx
- Larynx
- Trachea
- 1° bronchi
- 2° bronchi
- 3° bronchi
- Terminal bronchioles
Functions of Conducting Zone

- Transports air to the lung.
- Warms, humidifies, filters, and cleans the air.
  - Mucus traps small particles, and cilia move it away from the lungs. Expectoration = coughing up the mucus & debris.
- Voice production in the larynx as air passes over the vocal folds

**Structures in the larynx**

- **Glottis** – opening between vocal cords
- **Epiglottis** – closes upon swallowing to prevent food from entering airway
- **False vocal cords** – muscles fibers that assist the epiglottis
- **True vocal cords** – muscles folds that vibrate when air passes by them
2) Respiratory zone

Respiratory bronchioles = smallest bronchioles, branch from tertiary bronchioles.

Alveolar sacs = honeycomb shaped, 1-cell thick sacs for gas exchange.

[~300 mill in lungs! ~760 sq ft area!]

How gases are exchanged with blood

Surrounded by arterial & venous capillaries ("capillary plexus") for gas exchange between alveoli & blood.
Gas exchange occurs at the Alveoli

- thin cellular walls covered with capillary networks
- **300 million sacs**! - very large surface area
  - surfactant keeps the alveoli inflated

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2 Types Alveolar Cells:

**Type 1 Alveolar Cells** = Make up wall of alveolus. 97% of total lung surface area where most gas exchange occurs.

**Type 2 Alveolar Cells** - secrete **surfactant**

**Surfactant** = \( \sqrt{r} \) surface tension (due to water layer) so that alveolar sacs won't collapse during breathing.
Why is surfactant important?

Non-obstructive Atelectasis =
not enough surfactant
↑ alveolar surface tension
lead to alveolar collapse
collapsed lung.

Not to be confused with
Obstructive Atelectasis =
foreign body in airway leading to
alveolar collapse & collapsed lung.

Surfactants ↓ intra-alveolar pressure & prevent collapse

Infant Respiratory Distress Syndrome (IRDS)
• Surfactant is produced > 28 weeks (7-8 months)
• Babies are born < 28 wks - not enough surfactant. High surface tension inside alveoli, results in collapsed alveoli, which collapses lung (non-obstructive atelectasis)

Adult
Acute Respiratory Distress Syndrome (ARDS)
• Due to inflammation from infection (septic shock)
• Results in protein (serum) secretion in lungs.
• Fluid dilutes surfactant, ↑ surface tension, alveoli collapse,
• could cause lung collapse (non-obstructive atelectasis)
Thoracic cavity: REVIEW!

Membranes of the lungs:
- Visceral pleura = membrane covering lungs
- Parietal pleura = membrane lining plural cavity
  - Parietal pleura held tight against thoracic wall by surface tension of water layer.
  - As thoracic cage changes volume (w/ breathing) so do the lungs.

Intrapleural space = potential space in between membranes
- The 2 pleura pressed together w/serous fluid between them.

2. Mechanics of Respiration

1) Air moves from high to low pressure
   - Atmospheric air pressure = constant (760 mmHg)
   - Lung air pressure depends on volume of thoracic cavity

2) Air pressure in lungs (closed chamber) changes with volume of chamber
   “Boyle’s Law” = as volume of closed chamber ↑, air pressure within ↓
   as volume of closed chamber ↓, air pressure within ↑

Translates to lung volume & air pressure within lungs (“intrapulmonary pressure”):
- As thoracic volume ↑, lung volume ↑, & intrapulmonary pressure ↓
- As thoracic volume ↓, lung volume ↓, & intrapulmonary pressure ↑
Boyle's Law

Chamber volume larger
BUT air pressure lower

Chamber volume smaller
BUT air pressure higher

Table 16.1 | Pressure Changes in Normal, Quiet Breathing

<table>
<thead>
<tr>
<th></th>
<th>Inspiration</th>
<th>Expiration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intrapulmonary pressure (mmHg)</td>
<td>-3</td>
<td>+3</td>
</tr>
<tr>
<td>Intrapleural pressure (mmHg)</td>
<td>-6</td>
<td>-3</td>
</tr>
<tr>
<td>Transpulmonary pressure (mmHg)</td>
<td>+3</td>
<td>+6</td>
</tr>
</tbody>
</table>

Note: Pressures indicate mmHg below or above atmospheric pressure.
Mechanism of Breathing

**Inspiration/inhalation** = When the diaphragm and intercostal muscles contract, the thoracic cage volume ↑, & the lung volume ↑.

Rate of breathing is controlled by the medulla oblongata, which sends signals to the diaphragm and intercostal muscles.

Mechanism of Breathing

**Expiration/exhalation** = When the diaphragm and intercostal muscles relax, the thoracic cage volume ↓, & the lung volume ↓.
**Muscles involved in inspiration**

Diaphragm – contracts (moves downward) to expand thorax.

**external intercostal muscles** – contraction lifts ribs & sternum.

**scalenes, pectoralis minor, & sternocleidomastoid** – contract to pull thoracic cage up and outward.

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**Muscles involved in expiration**

Diaphragm – relaxes (diaphragm moves upward) to compress thorax.

**internal intercostal muscles** – contraction pulls ribs and sternum in.

**abdominal wall muscles** – contract to push diaphragm higher.
Gas Pressure Vocabulary:

- **Intrapulmonary pressure** = pressure inside lungs
  - *During inhalation* – is lower than atmospheric pressure (-3 mmHg)
  - *During exhalation* – is above atmospheric pressure (+3 mmHg)

- **Intrapeural pressure** = pressure between the pleural membranes due to elastic recoil (parietal pleura sticks to wall)
  - *During inhalation* – is lower than atmospheric (-6 mmHg)
  - *During exhalation* – is still lower atmospheric (-3 mmHg)

*** intrapeural pressure should ALWAYS be negative. If air enters this space, the lung can detach from thoracic wall, trapped air puts pressure on lung, & lung can collapse. ***

- **Transpulmonary pressure** = difference between intrapulmonary & intrapeural pressure (is ALWAYS above atmospheric pressure).

Causes of a collapsed lung

"Pneumothorax" = air trapped within intrapeural space.

Pocket of air creates pressure that collapses the lung. (Can be traumatic or tension)

- Air trapped between the two pleural membranes removes the pressure gradient.

Result = can’t expand lungs to get air to enter! Lung collapses.
**Clinical Applications**

If air enters the intrapleural space and thereby raises the intrapleural pressure, the difference in pressure between the inside of the lungs (intrapulmonary pressure) and the outside of the lungs (intrapleural pressure) is abolished. As a result, the lung is no longer stuck to the thoracic wall; this is like releasing a stretched rubber band, and the lung’s elastic recoil causes it to collapse. The condition of air entering the intrapleural space and causing the collapse of a lung is known as a **pneumothorax**. Fortunately, a pneumothorax usually causes only one lung to collapse, because each lung is contained in a separate pleural compartment.

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**Important properties of the lungs:**

**A) Surface tension** = pressure resulting from thin film of water lining alveoli that resists their expansion. Makes alveoli want to collapse with exhalation.

**B) Compliance** = lungs expand when stretched (when thoracic volume ↑).
- more lung compliance = greater capacity for “stretchiness”
- less lung compliance = less capacity for “stretchiness”

**C) Elasticity/Recoil** = tendency of lungs to return to normal shape after stretching.
(When thoracic volume ↓, lungs volume also ↓ parietal pleura keeps lungs “stuck” to thoracic wall).
A) Surface Tension & Law of La Place

**Law of LaPlace & surface tension in alveoli:**

Small alveoli have ↑ surface tension within, & ↑ air pressure within (intra-alveolar pressure), ↓ gas exchange.

Greater:
- Air pressure
- Surface tension

Large alveoli have ↓ surface tension, & ↓ air pressure within, ↑ gas exchange.

**anything that functionally ↓ alveolar size:**
- ↑ surface tension & intra-alveolar pressure
- ↓ gas exchange & respiratory capacity

Fig 16.10

**anything that functionally ↓ alveolar size:** like fluid buildup
- dilute surfactant
- ↑ surface tension & intra-alveolar pressure
- ↓ gas exchange & respiratory capacity
B) Lung compliance

Factors that increase compliance:
- pulmonary “surfactants”

Factors that decrease compliance:
- many, many things!
- Anything that causes chronic inflammation can lead to ↓ compliance
  - Chronic inflammation of the airways (bronchitis) can lead to
  - scar tissue in lungs (pulmonary fibrosis),
  - and narrowing of airways (bronchoconstriction)

For Ex. - Lung damage from smoking causes chronic bronchitis and formation of scar tissue (fibrosis)
Review

• The respiratory system
  – The conduction & respiration zones
• Airway, lung, and thoracic cavity anatomy
• Alveoli (gas exchange, surfactant, factors that affect intra-alveolar surface tension and pressure)
• Mechanics of breathing (Boyle’s law and respiratory muscles), muscles of respiration.
• Gas pressure vocabulary, and pneumothorax,
• Important properties of the lungs
  – Surface tension, compliance, & elasticity.
  – Factors that affect these properties

3. Respiratory Disorders, & Diagnosing Them

Respiratory Disorders

A Restrictive Disorder = Lung tissue is damaged. Lungs are stiff, or respiratory muscles are weak.
  Examples: Pulmonary fibrosis

Obstructive Disorder = Lung tissue is normal, but resistance is increased (airways are narrowed)
  Examples: Asthma
  COPD (which includes emphysema & chronic bronchitis)
  Cystic fibrosis
Respiratory Disorders – **Pulmonary Fibrosis**

- **Pulmonary fibrosis** = buildup of fibrous tissue in lungs stiffens them (restrictive disorder)

  $\text{= scar tissue}$
  
  $\text{lungs damaged!}$

**MANY CAUSES**

> Breathing in small particles that accumulate in & irritate the lungs:  
  
  Ex: **Silicosis** = (inhalation of fine glass, rock, or sand particles)

  Ex: **Anthracosis** (black lung disease) = inhalation of coal dust

  Ex. **Mesothelioma** – breathing in asbestos.

- Breathing in chemicals that irritate the lungs:  
  Ex. **smoking**

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Respiratory Illnesses - **Asthma**

Muscles around the bronchioles are hyper-excitable to allergens.

- **Obstructive disorder** due to inflammation, mucous secretion, & narrowing of airways (bronchoconstriction).

**Treatment?** **Albuterol** (B2-agonist)
Respiratory Illnesses – Chronic Obstructive Pulmonary Disease (COPD)

Chronic inflammation of Airways Alveolar Tissue
- Narrows airways & destroys alveolar walls
- Proliferation of mucus-secreting goblet cells
- Development of scar (fibrous) tissue = pulmonary fibrosis
- Obstructive disorder – due to mucus buildup and narrowed airways.

**Physiology in Health and Disease**

People with pulmonary disorders frequently complain of dyspnea, which is a feeling of “shortness of breath.” The dyspnea, wheezing, and other symptoms of asthma are produced by increased resistance to airflow through the bronchioles (asthma is an obstructive pulmonary disorder, as discussed previously). The increased resistance to air flow is caused by bronchoconstriction and inflammation that may be provoked by allergic reactions (chapter 11).

Asthma may be treated on a sustained basis with glucocorticoid drugs (related to cortisone) that inhibit inflammation, thereby preventing or reducing the severity of “attacks.” New drugs (such as Singulair) that block the action of leukotrienes, a type of regulatory fatty acid (related to prostaglandins) that promote asthma, are now also available for this purpose. Acute asthma attacks are commonly treated with inhaled drugs (such as Albuterol) that stimulate the β₂-adrenergic receptors (a type of receptor for epinephrine and norepinephrine; see chapter 6) that promote dilation of the bronchioles.

Alveolar tissue is destroyed in emphysema, resulting in fewer but larger alveoli (see fig. 12.8). The loss of alveoli reduces the ability of the bronchioles to remain open during expiration, causing air trapping during expiration when the bronchioles collapse. The most common cause of emphysema is cigarette smoking, which indirectly causes different protein-digesting enzymes to destroy the lung tissue. The loss of alveoli and air trapping reduces gas exchange, so that people with emphysema have difficulty in both oxygenating the blood and eliminating carbon dioxide. Because of this, people with emphysema must often breathe from an oxygen tank.

Chronic obstructive pulmonary disease (COPD) is characterized by chronic inflammation with narrowing of the Airways and destruction of the alveolar walls. Included in the COPD category is emphysema and chronic obstructive bronchiolitis, which refers to fibrosis and obstruction of the bronchioles. The condition results in a faster age-related decline in the FEV₁ (discussed previously). COPD differs from asthma in that, unlike asthma, COPD is not reversible with the use of a bronchodilator such as Albuterol. Also unlike asthma, COPD is not helped much by inhaled glucocorticoids (drugs related to hydrocortisone). The vast majority of people with COPD are smokers, and stopping smoking once COPD has begun does not seem to stop its progression. In addition to the pulmonary problems directly caused by COPD, this condition increases the risk of pneumonia, pulmonary emboli (traveling blood clots), and heart failure. Patients with COPD may develop cor pulmonale—pulmonary hypertension with eventual failure of the right ventricle. COPD is now the fifth leading cause of death in the United States, and scientists have estimated that by 2020 it will become the third leading cause of death worldwide.
**Respiratory Illnesses - Emphysema**

Chronic destruction of alveolar tissue (walls between alveoli lost)
- reduces area for gas exchange
- alveoli expand easily, but can’t empty easily (air-trapping disorder)
- **obstructive disorder**

![Image of emphysema](http://www.nlm.nih.gov/medlineplus/ency/images/ency/fullsize/17055.jpg)

**Respiratory Illnesses - Cystic Fibrosis**

- Genetic disorder affecting Cl- channels on alveoli membrane. **Obstructive disorder**

Results in buildup of mucus within alveoli causing:
- Dilutes surfactant
- ↓ decreased functional alveolar size
- ↑ surface tension & intra-alveolar pressure (harder for alveoli to expand)
- ↓ with gas exchange
- Warmth & moisture (mucus) aids bacterial growth. (Vulnerable to **pneumonia**)

![Image of cystic fibrosis](http://www.nlm.nih.gov/medlineplus/ency/images/ency/fullsize/17055.jpg)
**Pulmonary Function Tests**

- **Spirometry**: air movement during respiration recorded on a spirogram.
  - Measures lung volumes and capacities
  - Can diagnose restrictive and obstructive lung disorders

3. **Lung Volumes & Respiratory Vocabulary**

**Spirometry** = clinical evaluation of pulmonary (respiratory) function, which allows diagnosis of lung disorders.
### Additional Respiratory Vocabulary:

- **Apnea** = absence of breathing
- **Dyspnea** = labored or difficult breathing
- **Eupnea** = normal breathing at rest
- **Hyperventilation** = excessively rapid ventilation (will decrease alveolar CO2)
- **Hypoventilation** = low ventilation (will increase alveolar CO2)
- **Pneumothorax** = presence of gas in intrapleural space causing lung collapse

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#### TABLE 12.1 Terms Used to Describe Lung Volumes and Capacities

<table>
<thead>
<tr>
<th>Term</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lung Volumes</td>
<td>The four nonoverlapping components of the total lung capacity</td>
</tr>
<tr>
<td>Tidal volume</td>
<td>The volume of gas inspired or expired in an unforced respiratory cycle</td>
</tr>
<tr>
<td>Inspiratory reserve volume</td>
<td>The maximum volume of gas that can be inspired during forced breathing in addition to tidal volume</td>
</tr>
<tr>
<td>Expiratory reserve volume</td>
<td>The maximum volume of gas that can be expired during forced breathing in addition to tidal volume</td>
</tr>
<tr>
<td>Residual volume</td>
<td>The volume of gas remaining in the lungs after a maximum expiration</td>
</tr>
<tr>
<td>Lung Capacities</td>
<td>Measurements that are the sum of two or more lung volumes</td>
</tr>
<tr>
<td>Total lung capacity</td>
<td>The total amount of gas in the lungs after a maximum inspiration</td>
</tr>
<tr>
<td>Vital capacity</td>
<td>The maximum amount of gas that can be expired after a maximum inspiration</td>
</tr>
<tr>
<td>Inspiratory capacity</td>
<td>The maximum amount of gas that can be inspired after a normal tidal expiration</td>
</tr>
<tr>
<td>Functional residual capacity</td>
<td>The amount of gas remaining in the lungs after a normal tidal expiration</td>
</tr>
</tbody>
</table>
Review

• Respiratory disorders
  – Restrictive vs Obstructive
  – Pulmonary fibrosis (and its causes)
  – Asthma
  – COPD
  – Emphysema
  – Cystic fibrosis

• Testing for respiratory disorders
  – Spirometry
  – Spirometry vocabulary (IRV, ERV, TV, VC, RV, and TLC, minute ventilation)
  – Additional respiratory vocabulary (eupnea, dyspnea, apnea, hyperventilation, hypoventilation)

4. Basics of Gas Exchange at Lungs and at Body Tissues

Gas exchange between 2 structures is dependent on pressure gradient of dissolved O2 & CO2

* Gas moves from side with higher pressure (from dissolved gases) to side with lower pressure & visa versa

*Gas wants to move “downhill” from high to low pressure!
Gas exchange between lung alveoli & pulmonary vessels:

> Alveolar PO2 = 105 mmHg, higher than that in pulmonary arteries (40 mmHg)

> Alveolar PCO2 = 40 mmHg, lower than that in pulmonary arteries (46 mmHg)

Gas exchange between systemic capillaries & tissues:

> Tissue PO2 (<100 mmHg) = lower than O2-rich arterial blood (100 mmHg)

> Tissue PCO2 (>40 mmHg) = higher than that in arterial blood (40 mmHg)
Review

- Pulmonary function tests (spirometry)
- Alveolar PO$_2$ lower than atmospheric
- Gas exchange at tissues & at alveoli of lungs
  Depends on differences in partial pressures of O$_2$ and CO$_2$

5. Regulation of Respiration – regulation of blood O$_2$ & CO$_2$

Motor neurons from 3 brain areas control breathing muscles:

1) **Voluntary Breathing**
   = primary motor cortex of frontal cerebral lobe.

2) **Involuntary Breathing** =
   - Medulla – respiratory center regulates respiratory rate.
   - Pons – apneustic center (stimulate inhalation)
     - pneumotaxic center (inhibit inhalation)
What happens to minute ventilation after:
• Hypoventilation?

• Hyperventilation?

Autonomic motor control breathing involves:

Chemoreceptors:

» Aorta & carotid artery chemoreceptors (called peripheral chemoreceptors)
  - sense blood O2 and CO2 levels

» Medulla chemoreceptors (called central chemoreceptors)
  - sense CSF O2 and CO2 levels
### Negative Feedback regulation of blood pH (by blood CO2 content)

**Stimulus** = ↓ blood pH (acidosis) blood O2 is too low (CO2 is high)  
**Sensors** = arterial chemoreceptors detect high CO2 in blood, Medulla senses high CO2 in CSF.  
**Integrating center** = Medulla’s respiratory center  
**Effectors** = respiratory muscles  
   - ↑ respiratory depth & rate (↑ minute ventilation) Get rid of excess CO2  
**Effect** = ↑ blood pH to normal

**Stimulus** = ↑ blood pH (alkalosis) blood O2 is too high (CO2 is too low)  
**Sensors** = arterial chemoreceptors detect low CO2 in blood, Medulla senses low CO2 in CSF.  
**Integrating center** = Medulla’s respiratory center  
**Effectors** = respiratory muscles  
   - ↓ respiratory depth & rate (↓ minute ventilation) Retain a little CO2  
**Effect** = ↓ blood pH to normal

### The role of chemoreceptors in respiration

The most important chemical in influencing breathing rate is **carbon dioxide**.

\[ \text{CO}_2 + \text{H}_2\text{O} \rightarrow \text{H}_2\text{CO}_3 \rightarrow \text{H}^+ + \text{HCO}_3^- \]

Oxygen-sensitive chemoreceptors only alter breathing rate when blood oxygen levels fall critically low (or CO2 is too high)
Blood pH (Acid/Base balance) based primarily on blood CO\textsubscript{2} content and metabolic activities in body:

1) Respiratory component = where CO\textsubscript{2} (a volatile acid) in blood eliminated by lungs (exhalation).
   - Increased respiratory rate ↑ blood pH (respiratory alkalosis)
   - Decreased respiratory rate ↓ blood pH (respiratory acidosis)

2) Metabolic component = non-volatile acids in blood (i.e. lactic acid, fatty acids, ketones) eliminated by liver, kidneys, or other organs.

Normal Blood pH = 7.35 – 7.45
Blood pH maintained by buffering CO\textsubscript{2} with HCO\textsubscript{3}\textsuperscript{-}

Blood with high CO\textsubscript{2} or H+ content = acidic (acidosis)
Blood w/lower CO\textsubscript{2} or high HCO\textsubscript{3}\textsuperscript{-} content = alkaline (alkalosis)

Acidosis = increased acids in blood (pH below 7.35)
Alkalosis = decreased acids in blood (pH above 7.45)

Respiratory acidosis = ↓ blood pH due to ↓ respiratory rate (hypoventilation) – not enough CO\textsubscript{2} waste exhaled by lungs.

Respiratory alkalosis = ↑ blood pH due to ↑ respiratory rate (hyperventilation) – too much CO\textsubscript{2} exhaled by lungs.

Metabolic acidosis = excess metabolic production of acids (i.e. ketosis) OR loss of bases (i.e. bicarbonate) from chronic diarrhea or kidney problems (excrete too much HCO\textsubscript{3}\textsuperscript{-})

Metabolic alkalosis = too much bicarbonate (not enough excreted by kidneys) OR loss of metabolic acids such as with chronic vomiting (lose HCL).
Review

- Regulation of breathing (voluntary vs involuntary)
  - Primary motor cortex (voluntary)
  - Medulla & Pons (involuntary)

- Acid / Base imbalance
  - Metabolic Acidosis & Alkalosis versus
  - Respiratory Acidosis & alkalosis

6. Hemoglobin & Hemoglobin Disorders

Hemoglobin =

- 4 protein chains w/4 iron-containing heme (pigments)
- Each heme group binds with 1 O2 molecule
- Each RBC has ~280 million hemoglobin molecules (each RBC can carry ~billion O2 molecules! (4 X 280 million)
- Hemoglobin bound to O2 = “oxyhemoglobin” (Arterial blood 97% saturated w/oxyhemoglobin = bright red)
- Hemoglobin lacking O2 = “deoxyhemoglobin” (venous blood dull red or maroon)
**Hemoglobin Disorders:**

**Carbon Monoxide** = odorless, color-less gas that binds w/hemoglobin to create **carboxyhemoglobin** in RBCs.

Carboxyhemoglobin has lower affinity for O2.

**Result:**
> Hypoxia (called carboxyhemoglobinemia)
> Death
**Hemoglobin Disorders contin...**

**Methemoglobinemia** = disorder in which hemoglobin’s iron (a component of heme) is “ferric” rather than “ferrous”.
> this hemoglobin called **methemoglobin** (pronounce as “met-hemoglobin”)
> Methemoglobin has ↓ ability to release (unload) O2 at tissues.
> Tissues chronically O2-starved.
> Patients are hypoxic & BLUE!

“**Blue baby syndrome**” = babies turn blue (hypoxia) from drinking milk made w/nitrate contaminated water. Nitrate causes formation of methemoglobin.

---

**Hemoglobin Disorders contin...**

**Neonatal jaundice** At birth switch from hemoglobin-F (fetal) to hemoglobin-A (adult)
- Body removes RBCs with hemoglobin f.
- Liver removes biliruben from destroyed hemoglobin f.
- Liver sometimes not mature enough to remove biliruben.
- Biliruben builds up.
- Baby turns yellow. (happens in up to 50% newborns)

**Treatment:**
“blue light exposure” – breaks biliruben down to water-soluble form excreted by kidneys.
Sickle Cell Anemia = homozygous recessive condition in which body produces RBCs with hemoglobin-S rather than hemoglobin-A.
- Hemoglobin-S turns RBCs into sickle-shape.
- Sickled RBCs carry less O2 (cause hypoxia)
- Sickled RBCs tend to form clots (thrombus)
- Patients more prone to embolism.
- More prone to ischemic events.
Review

• Regulation of breathing
  – Medulla & pons
• Chemoreceptors
  – central, peripheral
• Hemoglobin O₂ transport:
  – Oxyhemoglobin & deoxyhemoglobin
  – Abnormal hemoglobin (carboxyhemoglobin, methemoglobin)
  – Neonatal jaundice
  – Sickle cell