



Physiology

EFM for the Experienced Nurse

Fetal Physiology

- Why do we need to know fetal physiology?
 - *The fetal heart rate pattern exhibits certain characteristics under the influence of hypoxic and non-hypoxic events. The clinician should have a basic understanding of physiology of fetal respiratory exchange and fetal heart rate.*

• Freeman, Garit, Nagoette and Miller (2012)

Extrinsic Physiological Influences on Fetal Heart Rate and Oxygenation

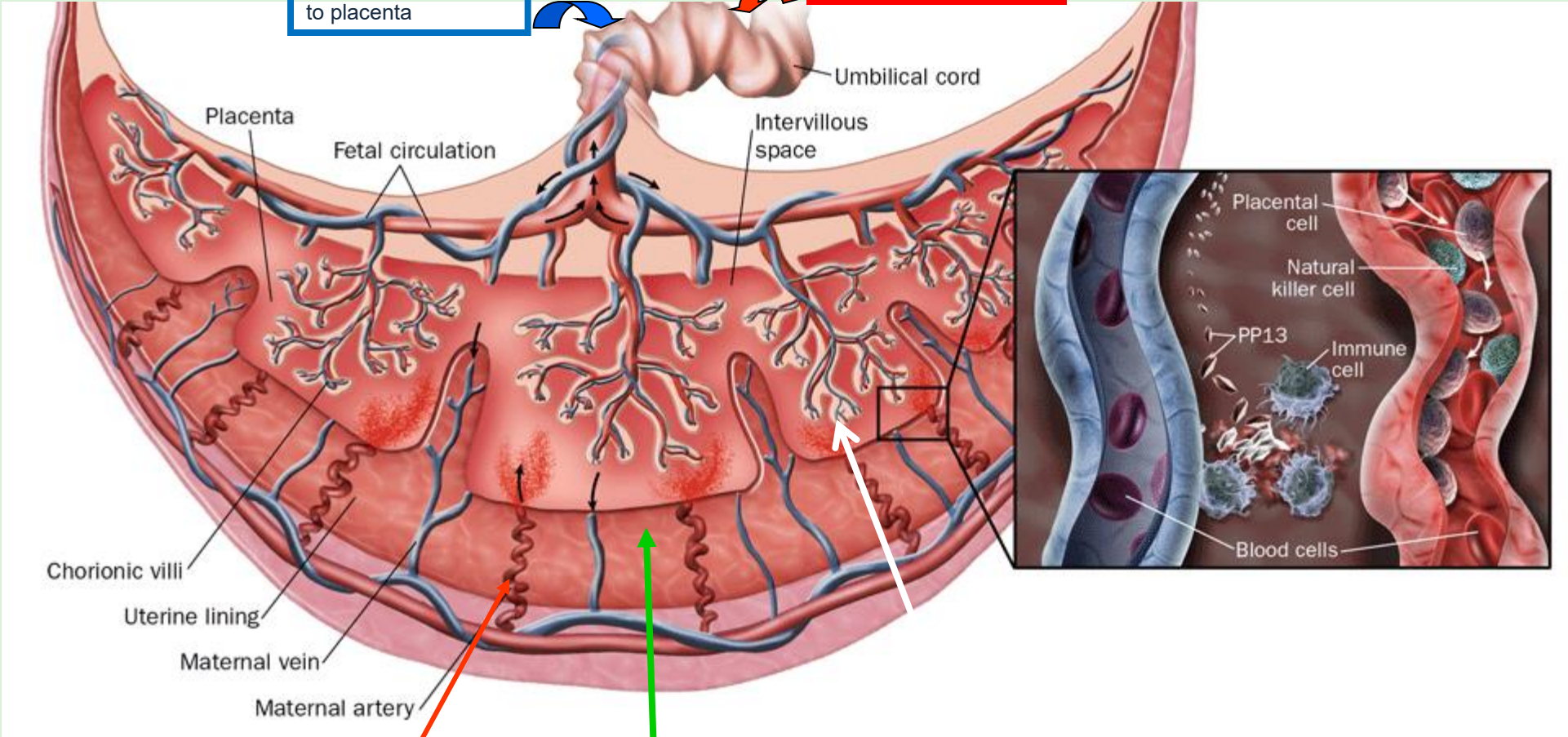
- Placental Structure and Function
- Uteroplacental and fetal-placental circulation
- Placental Transfer
- Maternal Oxygen Transport
- Uterine Blood Flow
- Umbilical Cord
- Amniotic Fluid and function

• AWHONN (2021)

Placental Structure and Function

2 umbilical arteries carry deoxygenated blood and waste products from fetus to placenta

One umbilical vein carries oxygenated blood and nutrients from placenta to fetus



Maternal PaO₂ leaving maternal vein is 40 mmHg

Spiral arteries

Average intervillous space PaO₂ is 45 mmHg

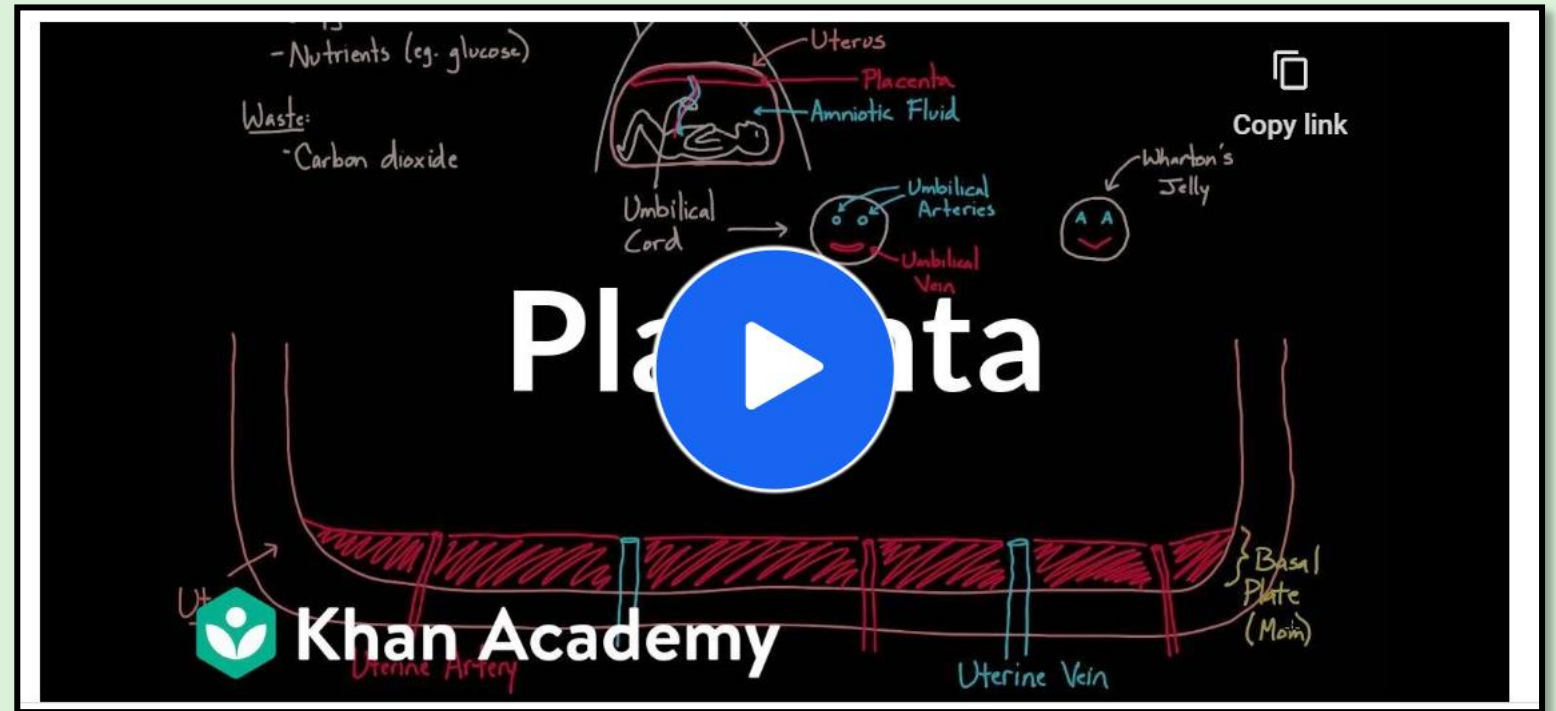
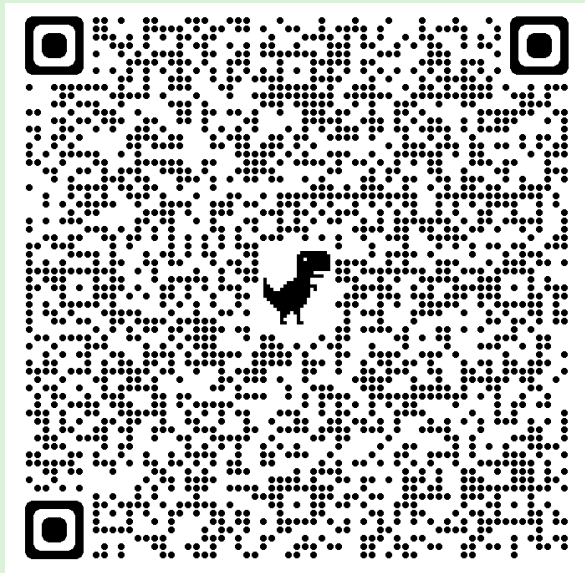
Oxygen diffuses across placental blood-blood barrier into fetal blood; paO₂ in fetal blood is about 30 mmHg

Maternal blood in the intervillous space becomes depleted and exits the intervillous space through the uterine veins. paO₂ leaving uterine veins is about 40 mmHg

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Meet the Placenta

Approximately 12 ½ minutes in length



PLACENTA TRANSFER

- *“facilitates the exchange of gases, nutrients, wastes, and other molecules (antibodies, hormones, medications) between maternal blood in the intervillous space and fetal blood in the villous capillaries.”*
 - Miller, Miller & Cypher (2027) pgs. 7-10)
- The placenta serves as the fetal lungs, kidneys, gastrointestinal tract, skin and as a barrier to certain dangerous substances
 - The placenta is an endocrine organ that produces
 - Steroids (estrogen and progesterone)
 - Protein (HCG, human placental lactogen)
 - Freeman et. al. (2012)



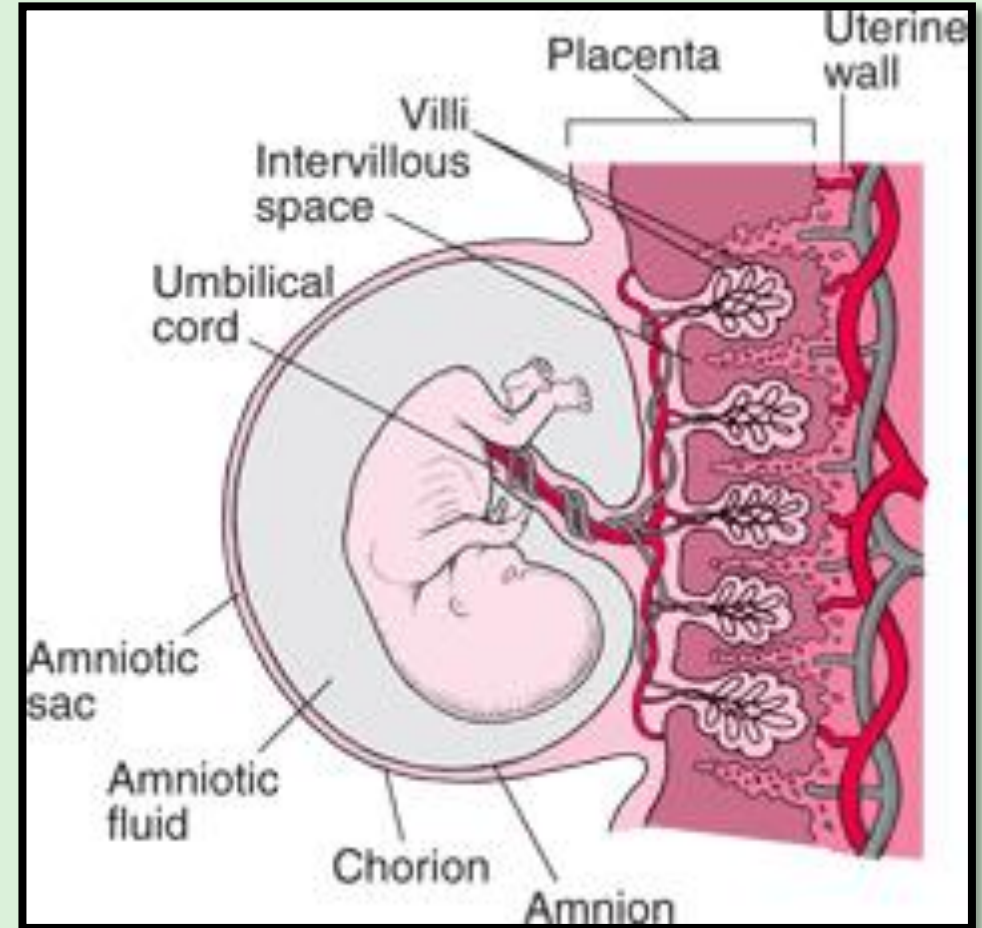
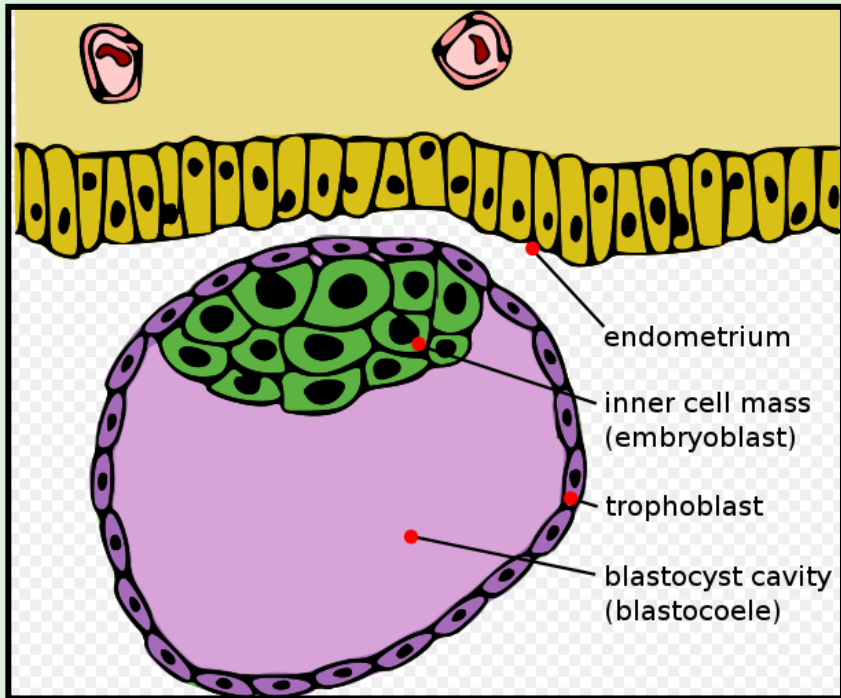
Transfer Mechanisms Between Fetal and Maternal Blood

Transport Mechanisms	Examples
Simple (passive) Diffusion Transfer down concentration gradient (higher to lower concentration); no energy required	Water, oxygen, carbon dioxide, some electrolytes, urea, creatinine, fatty acid, steroids, fat-soluble vitamins, narcotics, barbiturates, anesthetic gases, antibiotics
Facilitated Diffusion Transfer down concentration gradient involving protein transporters or carrier molecules	Glucose, lactate, possibly some oxygen
Active Transport Transfer against concentration gradient; carrier molecules and energy required	Amino acids, water –soluble vitamins, calcium, potassium, phosphate, iron, iodine
Pinocytosis Engulfing, and then suspending particles within small vesicles to transfer through cell (form endocytosis)	Immunoglobulin G (IgG), phospholipids, lipoproteins, some viruses
Bulk Flow Transfer resulting from changes in hydrostatic or osmotic gradient	Water, electrolytes
Capillary breaks or leaks Transfer of substances through microscopic breaks in placental membrane	Intact blood cells
Independent Movement Transfer of cells under their own power	Maternal leukocytes, microorganisms such as Treponema pallidum (syphilis), some viruses

PLACENTA TRANSFER

- O₂, Co₂, waste products and nutrients all must cross two layers of fetal trophoblast, the fetal connective tissue within the villus and the fetal capillary wall
- Uterine blood flow is supplied from the uterine arteries
- Spiral arteries must transverse thru the thickness of the myometrium to reach the intervillous space

PLACENTA Cont.



MATERNAL OXYGEN TRANSPORT

- Fetus relies on a constant supply of oxygenated maternal blood to maintain aerobic metabolism
- 4 Components of oxygen transport physiology:
 1. Oxygen content
 2. Oxygen affinity
 3. Oxygen delivery
 4. Oxygen consumption

• AWHONN (2021) chapter 2

Oxygen Content

- Oxygen content = total sum of oxygen in the patient's arterial blood
- Oxygen is transported to the tissues in two ways:
 1. Dissolved under pressure in the plasma
 - Is expressed as the partial pressure of oxygen or PaO₂ and is 1-2% of total oxygen.
 - PaO₂ plays a role in Hgb affinity
 2. Bound to hemoglobin molecules in RBCs
 - Is expressed as SaO₂ – the amount of Oxygen carried on the hemoglobin
 - Accounts for 98-99% of total oxygen
 - Unless compromised Hgb can carry four molecules of O₂ on each RBC

Oxygen Content Continued

- PaO₂ is typically the highest in the lungs and as blood circulates to other body tissues the PaO₂ is lower and the hemoglobin releases oxygen
- The oxygen-carrying capacity is determined by the amount of hemoglobin present in the blood.
 - ***Consider maternal conditions where the patient does not have adequate hemoglobin to carry oxygenated blood to her fetus..***

Oxygen Affinity

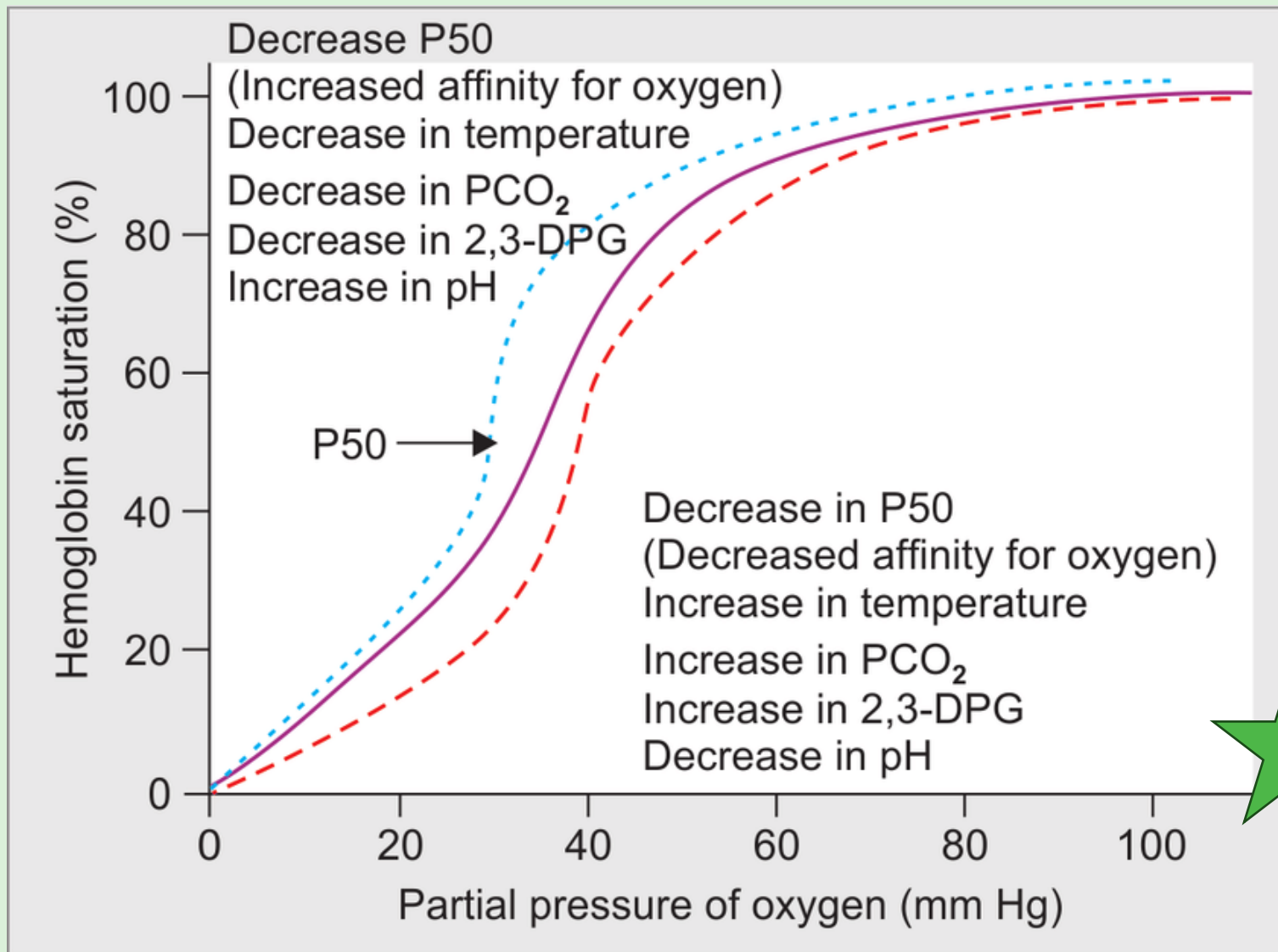
- Affinity = Is the ability of hemoglobin to acquire and release oxygen molecules
- During pregnancy, the oxyhemoglobin dissociation curve typically shifts to the right, enhancing the transfer of oxygen from the mother to the fetus
 - The fetus' hemoglobin has a greater affinity for oxygen than maternal hemoglobin
- Diseases and conditions can affect oxygen affinity and cause the oxyhemoglobin dissociation curve to shift to the left- easier for hemoglobin to pick up oxygen but harder to release it to the tissues (hypothermia, alkalosis and carbon monoxide)

OXYHEMOGLOBIN DISSOCIATION CURB

Left shift Higher O₂ Affinity – **Lower** O₂ release to the tissues

Maternal Conditions:

- Hypothermia
- Hypothyroidism
- Alkalosis
- Hyperventilation leading to Hypocapnia



Right shift Lower O₂ Affinity – **Higher** Release O₂ to the tissues

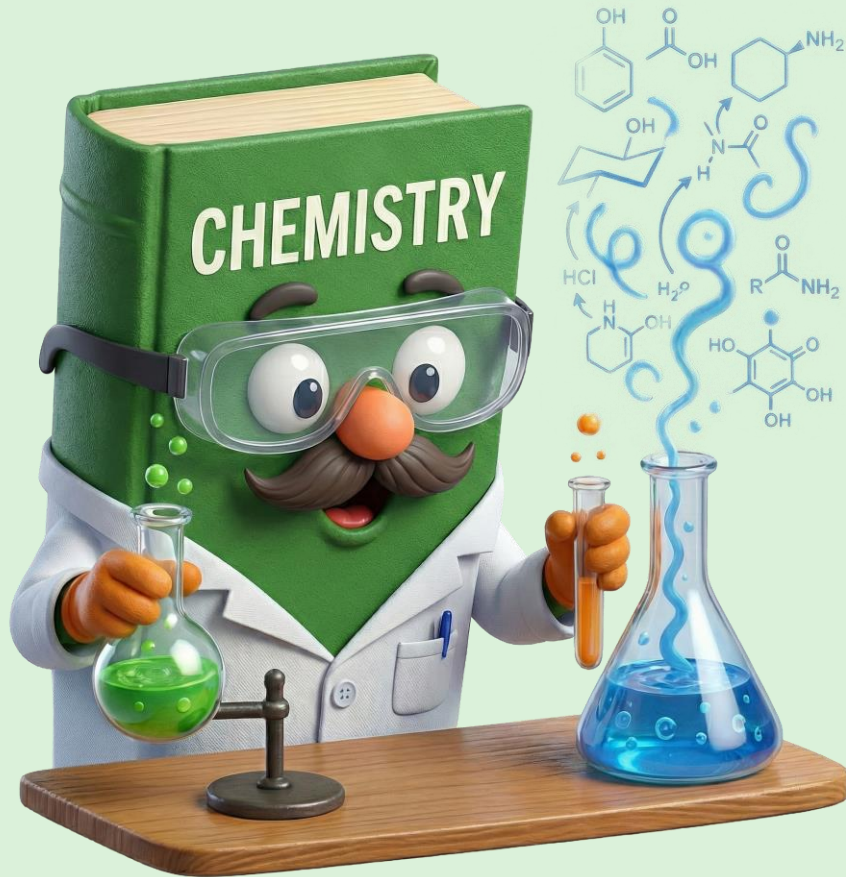
Maternal Conditions:

- Hyperthermia
- Hyperthyroidism
- Acidosis
- Hypoventilation leading to Hypercapnia
- DKA

In pregnancy there is a normal shift to the right to facilitate transfer of oxygen from the host to the fetus

Just a Bit of Chemistry

Aerobic Metabolism – With Oxygen



- Common metabolism
 - Oxygen with glucose result in:
 - ATP
 - Carbon Dioxide
 - Water
 - $O_2 + \text{Glucose} = \text{ATP (energy)} + \text{CO}_2 + \text{H}_2\text{O}$

Anaerobic Metabolism – Without Oxygen



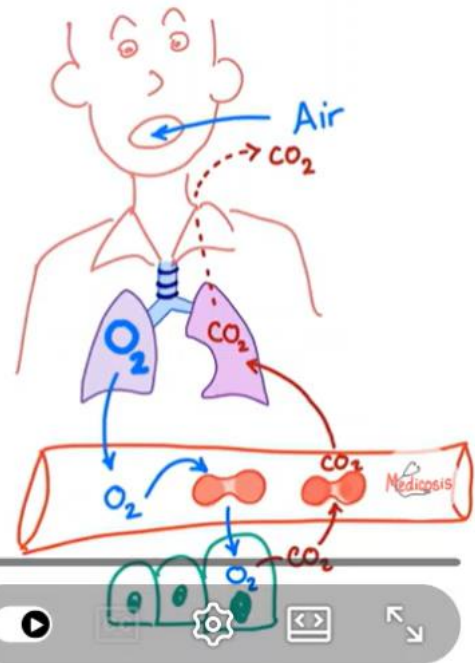
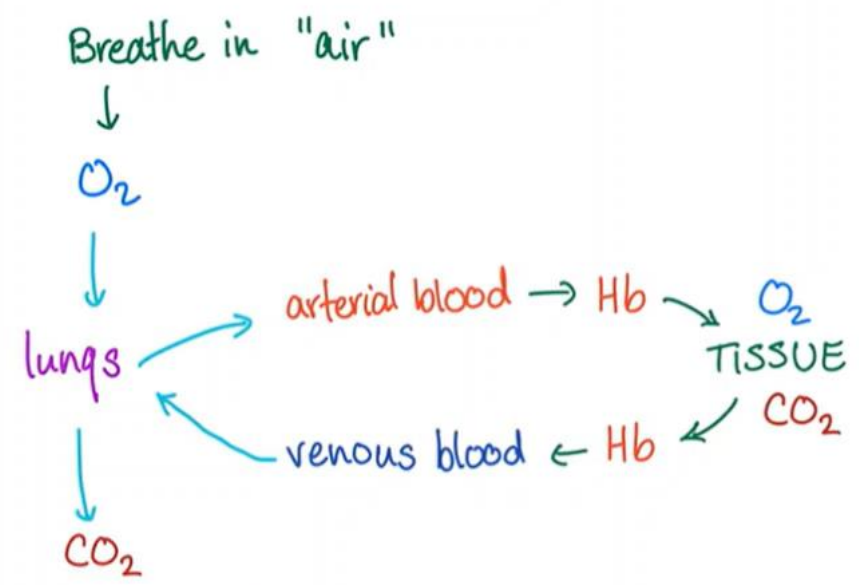
- Uses only glucose
- The results of this is:
 - ATP (very little energy), carbon dioxide, water, and LACTIC ACID
 - Glucose = ATP (energy) + CO₂ + H₂O + **LACTIC ACID**

Good News Is:

- Remember, in pregnancy, the host has a normal shift to the right on the oxyhemoglobin dissociation curve which will allow more oxygen to the fetus
- In addition, the fetus' hemoglobin has a greater affinity for oxygen



Oxyhemoglobin Dissociation Curve



0:00 / 5:03

Video player controls: play, volume, settings, full screen

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O₂ delivery & Oxygen-Hemoglobin dissociation curve
11:03

Oxygen-Hemoglobin Dissociation Curve - Oxygen ...
Medicosis Perfectionalis ✓
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OXYGEN-HEMOGLOBIN DISSOCIATION CURVE
16:20

The Oxygen Hemoglobin Dissociation Curve EXPLAINED!
ICU Advantage ✓
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Effect of 2,3-BPG on Hemoglobin
15:12

Effect of 2,3-BPG on Hemoglobin
Andrey K
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Oxygen Hemoglobin Dissociation Curve (with a mnemonic)

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Oxygen Delivery

- Oxygen delivery = Is the amount of oxygen delivered to the tissues per unit of time
- Cardiac Output and the content of oxygen in the arterial blood affect the delivery of oxygen from the lungs to the tissues.
- ***Consider factors which increase maternal cardiac output.....***
- Transfusions of RBCs increase total arterial oxygen content

Oxygen Delivery Continued

- Oxygen content is decreased as a result of dilutional anemia of pregnancy
- However, oxygen delivery is maintained at or above normal because cardiac output increases 50% by the 3rd Trimester
- The pregnant person is more dependent than the nonpregnant person on cardiac output for delivery of oxygen
- ***If oxygen delivery decreases, especially during labor both the host and fetus can be compromised!***

Oxygen Consumption

- Oxygen Consumption = Reflects the rate which oxygen is consumed by the tissues each minute
 - Normal adult = 20%
 - In pregnancy ↑ by 20% in single pregnancy and additional ↑ 10% for twin gestations
 - During labor, oxygen consumption ↑ as much as 40-60%
 - *Normal healthy patients in labor can compensate, again, if oxygen delivery decreases during labor may result in maternal or fetal compromise*

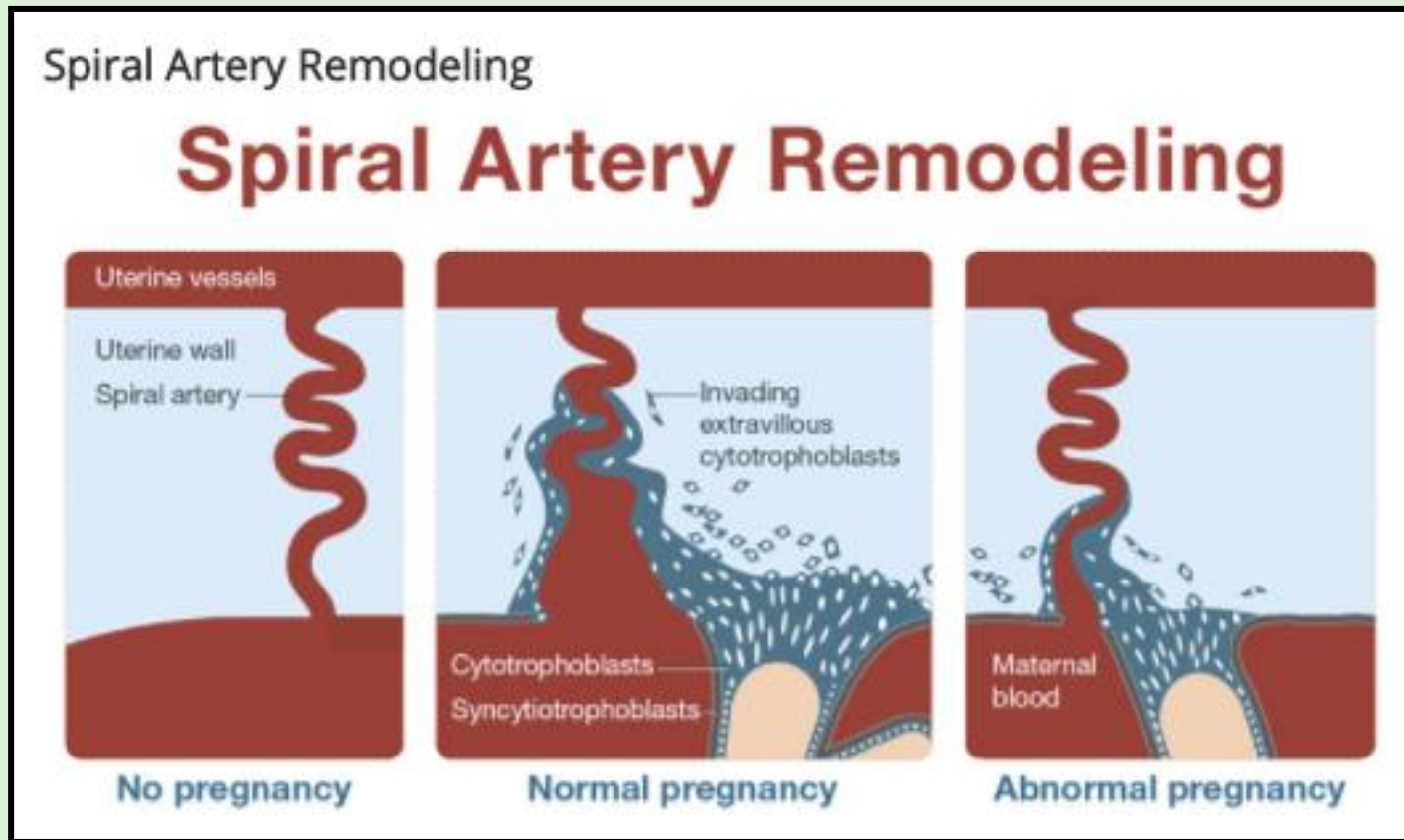
Uterine Blood Flow

- Uterine perfusion accounts for 10 to 15% of maternal cardiac output
- 700 to 800 cc/min
- Approximately 70-90% of total uterine blood flow supplies the placental (intervillous) circulation
- Remainder supplies the myometrium and endometrium

Uterine Blood Flow

- In pregnancy the arterials lose their ability to autoregulate (the vessels don't constrict to allow for increased blood flow if the pressure in the vessels drop)
- What this means is the volume of the blood delivered to the intervillous space is dependent on adequate blood flow to the uterus
 - No agents (drugs etc.) can increase vasodilatation
 - However, increase blood flow to the uterus can occur through maternal position changes, maintaining maternal blood pressure and with tocolytics in the presence of tachysystole

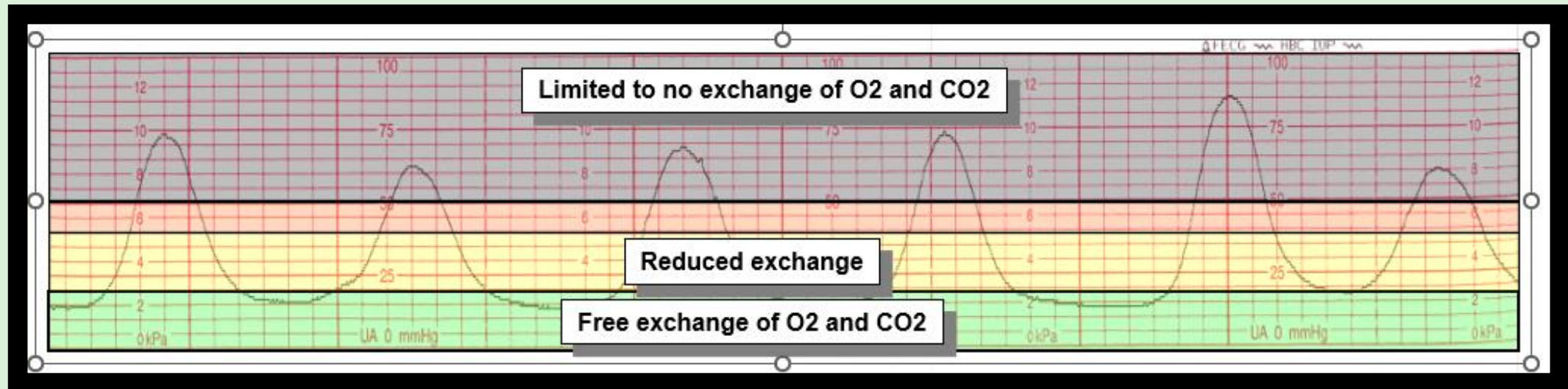
In Normal Pregnancy - Spiral Artery Remodeling



- If the remodeling process is impeded during placental growth, the spiral arteries remained tightly coiled

Uterine Blood Flow

- Anything that affects maternal cardiac output will affect the flow of blood through the spiral arteries
- As the uterus contracts, the intramyometrial pressure may exceed the intraarterial pressure
 - Causes an occlusion of these vessels and cessation of blood flow



THE POWER OF THE PLACENTA

- When the placental transfer capacity is near **100%**, fetus is provided with approximately 2 X the resources it needs to grow and be well oxygenated = ***FETAL RESERVE***
- **75%** - limited growth
- **50%** - both oxygen and carbon dioxide transfer decreased, and fetal compromise can result

Potential Clinical Causes of Decreased Uteroplacental Blood Flow and Maternal-Fetal Exchange

- Maternal conditions
 - Chronic or gestational hypertension; preeclampsia or cardiac disease
- Maternal hypotension
 - Supine position, regional anesthesia/analgesia (sympathetic blockade), Hemorrhage/hypovolemic shock
- Placental changes
 - Degenerative (maternal hypertension, diabetes, nicotine use, prolonged pregnancy)
 - Infection
 - Edema
 - Decreased surface area – small placenta, abruptio placenta, infarcts

Potential Clinical Causes of Decreased Uteroplacental Blood Flow and Maternal-Fetal Exchange Continued

- Excessive uterine activity
 - Tachysystole
 - Hypertonus
 - Medications that cause contractions (pitocin and prostaglandins)
 - Abruptio placenta
 - Cocaine
- Vaso constriction
 - Endogenous – catecholamines
 - Exogenous – most sympathomimetics(dobutamine, dopamine, norepinephrine, epinephrine, and isoproterenol), except ephedrine; cocaine, amphetamines

TABLE 1-2. POSSIBLE ETIOLOGIES OF DECREASED UTERINE BLOOD FLOW

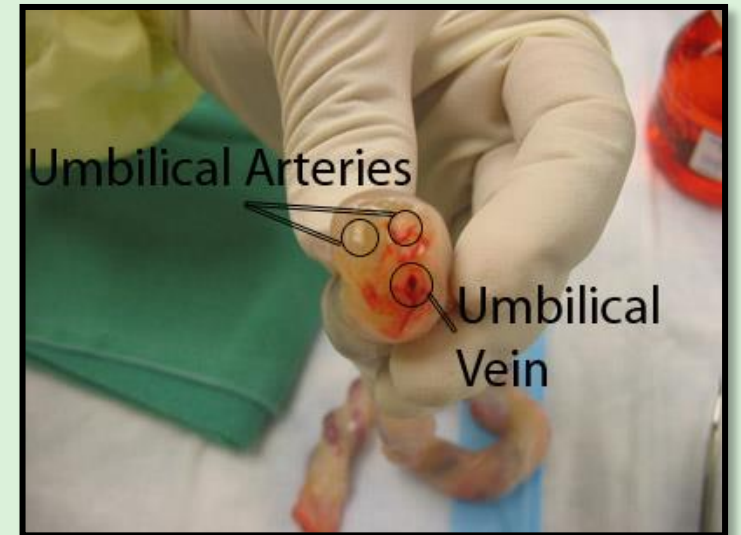
Etiology	Physiologic Effect
Iatrogenic	
Maternal supine position	Inferior vena cava compression, possible aortic compression, decreased venous return, decreased uteroplacental blood flow
Tachysystole (induced)	Decreased uteroplacental blood flow
Regional anesthesia	Maternal hypotension and decreased uteroplacental blood flow
Valsalva maneuver (closed-glottis "pushing")	Decreased fetal oxygenation ^a
Noniatrogenic	
Placental abruption	Alteration of placental vasculature, chronic placental change, decreased uteroplacental blood flow
Pregnancy-induced hypertension	
Chronic hypertension	Maternal vasoconstriction, decreased uteroplacental blood flow
Tachysystole (spontaneous)	
Smoking	
Substance abuse	
Malnutrition	
Pregnancy-induced hypertension	
Chronic hypertension	
Smoking	
Substance abuse	
Collagen vascular disease	
Placental abruption	Decreased functional placenta surface area due to calcifications, infarctions
Type 1 diabetes	
Postdate pregnancy	
Smoking	

- Menihan & Kopel-Puretz (2019 pg. 5)

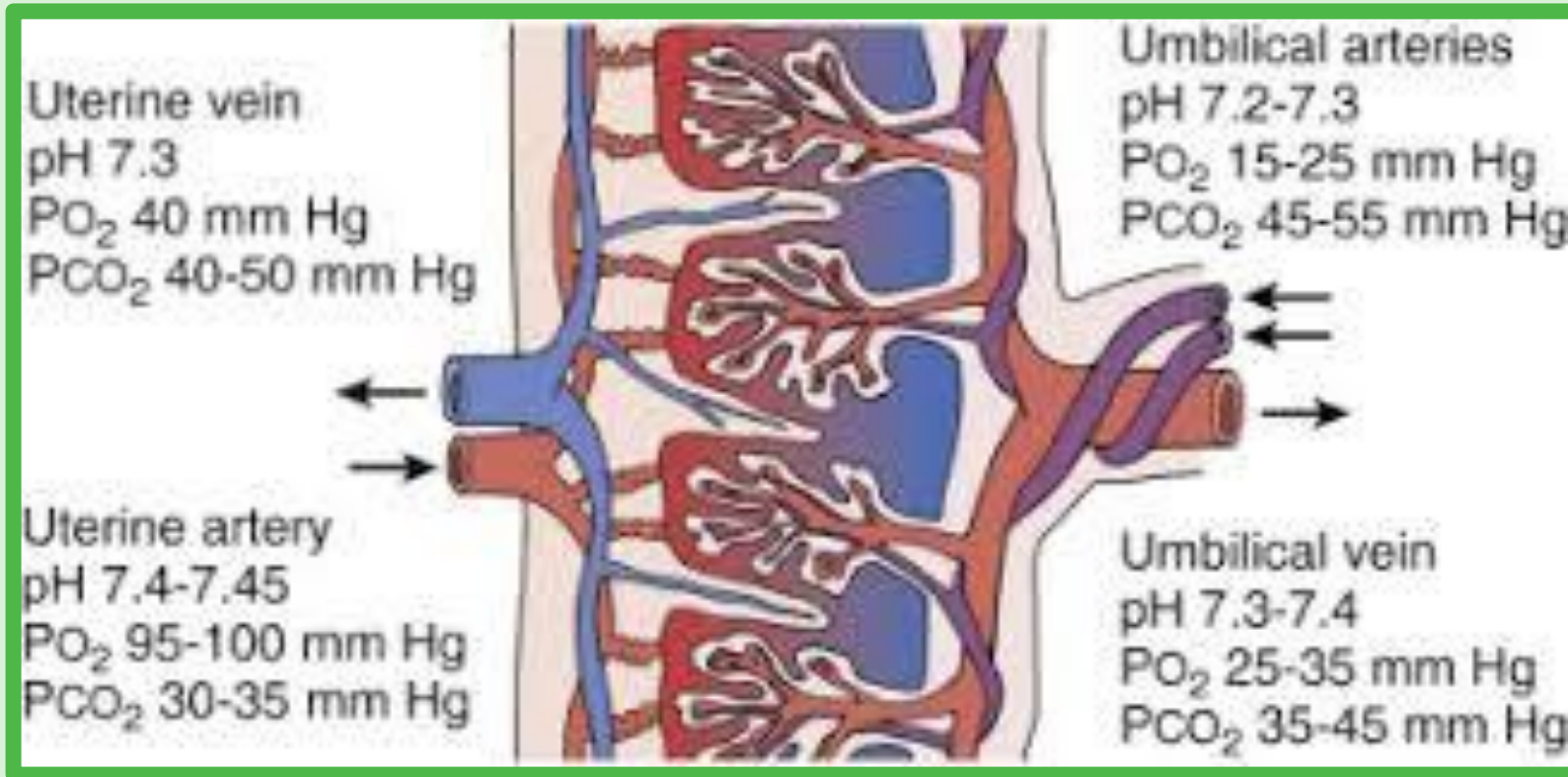
Umbilical Cord

A-V-A

- One vein
 - Carries oxygenated blood and nutrients from placenta to fetus
 - Thin-walled
 - More easily compressed
- Two arteries
 - Carry deoxygenated blood and waste products from fetus back to placenta



Approximate maternal and fetal blood gas values



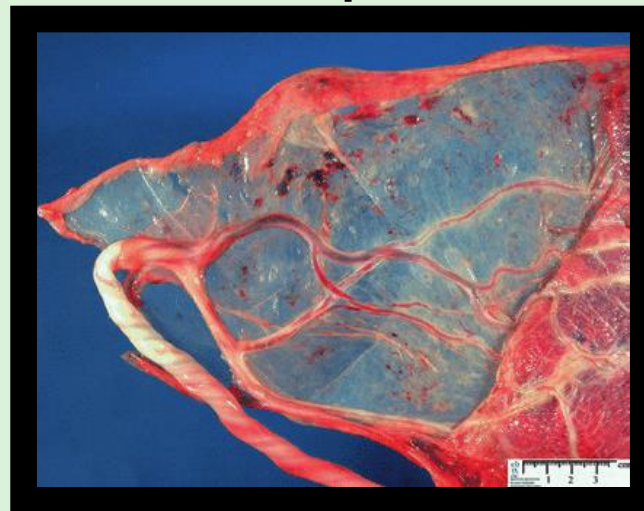
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Umbilical Cord Continued

- Length is usually 60 cm and is influenced by genetics, fetal movement and intrauterine space
- 5 seconds after birth the umbilical arteries begin to constrict and completely at 45 seconds
- Umbilical vein begins to constrict within 15 seconds and is functionally closed by 3-4 minutes
- Umbilical cord occlusion is the physiologic basis for FHR changes described as variable decelerations

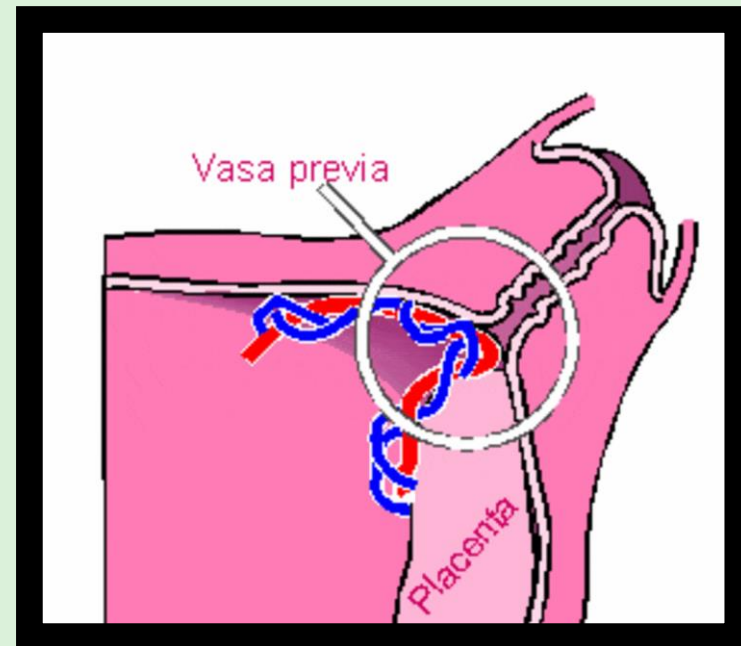
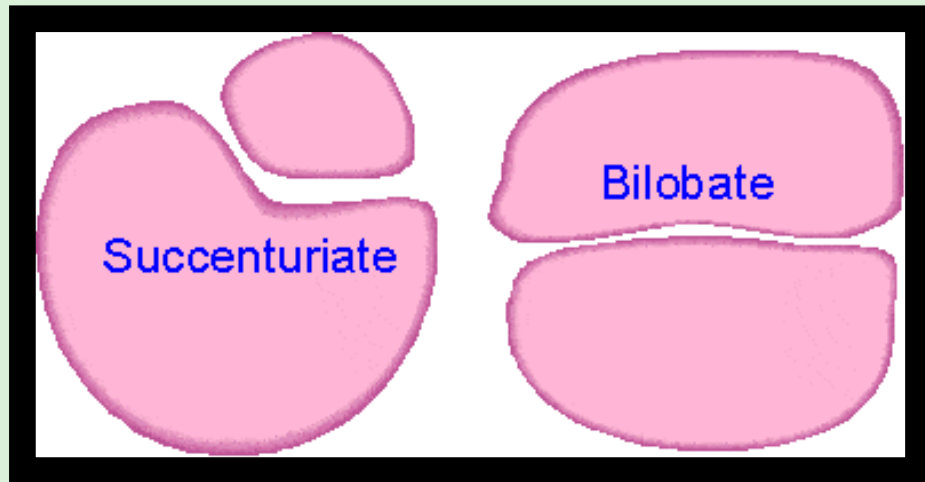
Umbilical Cord Continued

- Insertion – usually in the middle of the placenta
- Marginal or border insertion = Battledore placenta
- Velamentous cord insertion = placental end of the cord consists of opposing umbilical vessels that run between the amnion and chorion without the protection of Wharton's jelly



Umbilical Cord Continued

- If the fetal blood vessels are present in the membranes that cover the cervical os, this results in a Vasa Previa
 - Vasa Previas are associated with a velamentous cord insertion, bilobed or succenturiate placentas



Amniotic Fluid

- Critical for fetal development
- Starts at 10 ml at 8 weeks gestation and ↑ 770 ml by 28 weeks gestation and may remain the same until 36-38 weeks gestation, ↓ progressively
- 41 weeks gestation – 515 ml

Amniotic Fluid Continued

- Maintained by:
- Fetal Fluid Production – ***Fetal Urine*** and ***Fetal Lung Secretions***

AND

- Fluid Reabsorption – ***Fetal Swallowing*** and
Intramembranous Absorption (movement of fluid from the amniotic fluid into the fetal blood perfusing the amnion and chorion)

Amniotic Fluid Continued

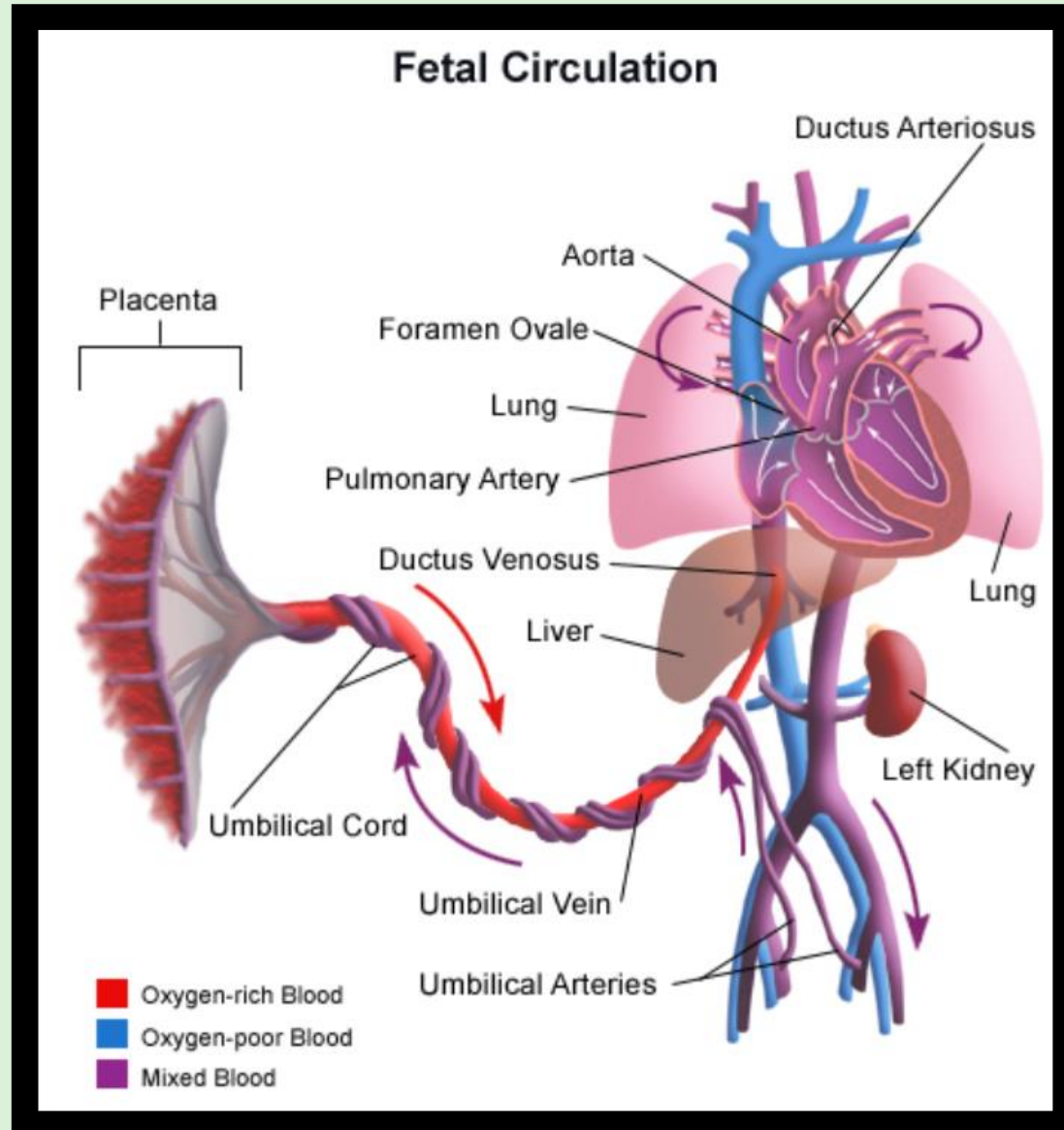
- Normal Values – Greater than 2 cm in a single deepest vertical pocket
- Oligohydramnios – low amniotic fluid
- Polyhydramnios – excessive amniotic fluid

Intrinsic Physiological Influences on Fetal Heart Rate and Oxygenation

- Fetal Circulation
- Oxygenation of Fetal Tissues
- Factors Controlling FHR Regulation

• AWHONN (2021)

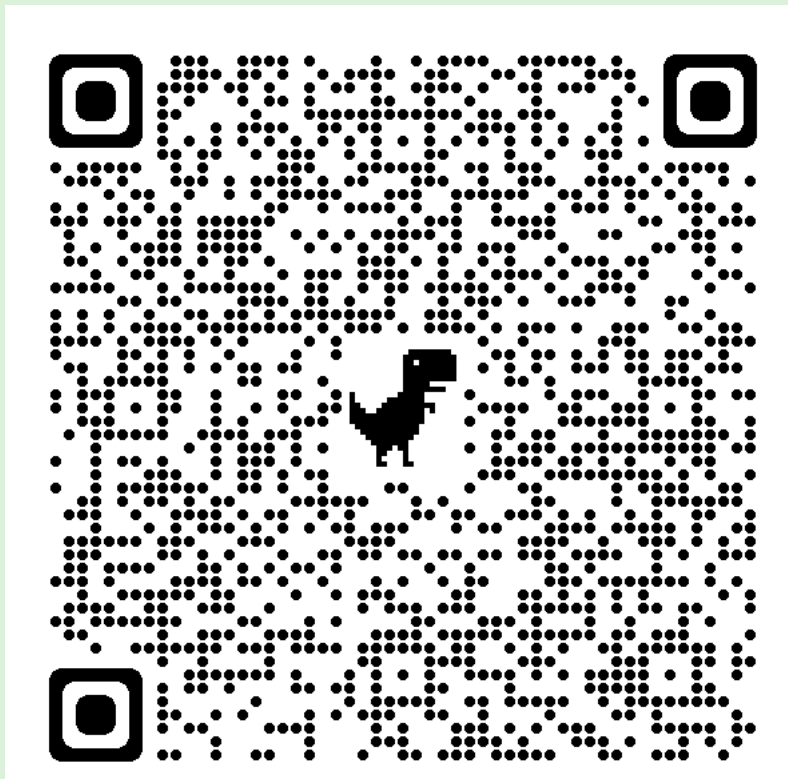
Fetal Circulation



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Fetal Circulation right before birth

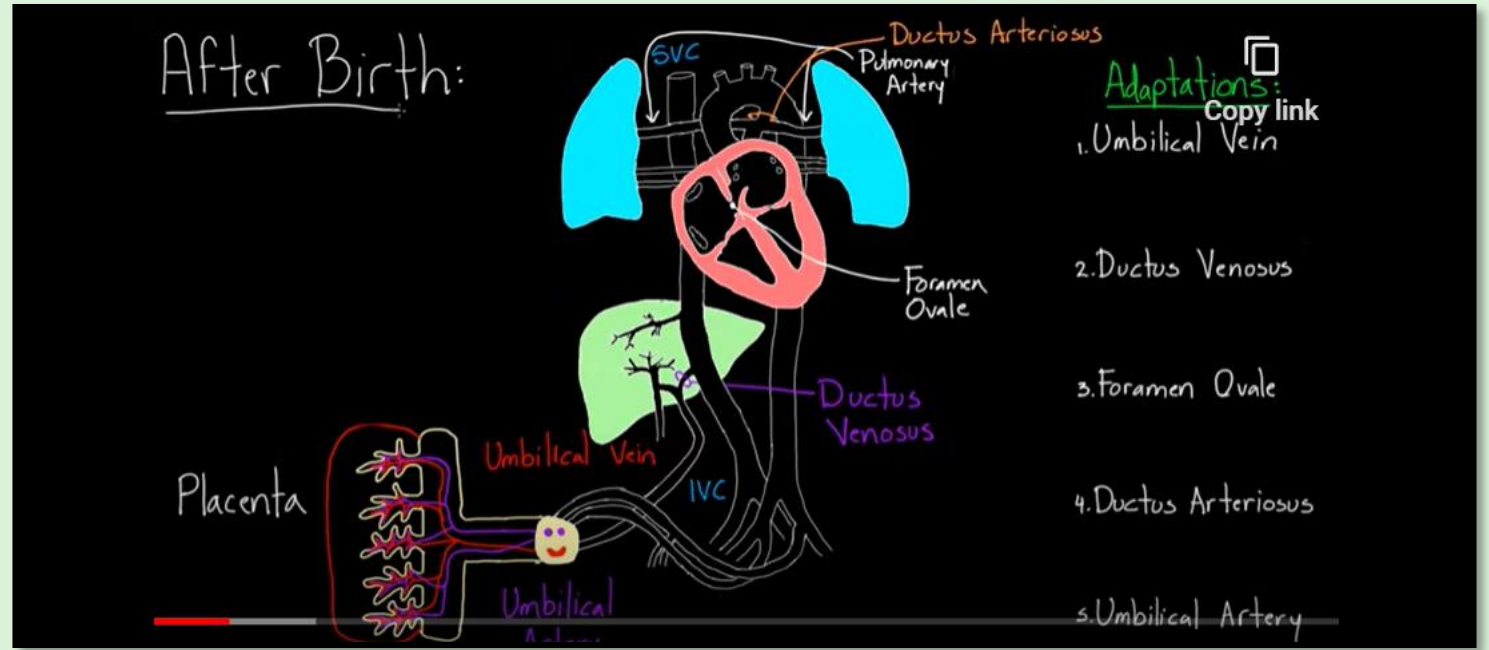
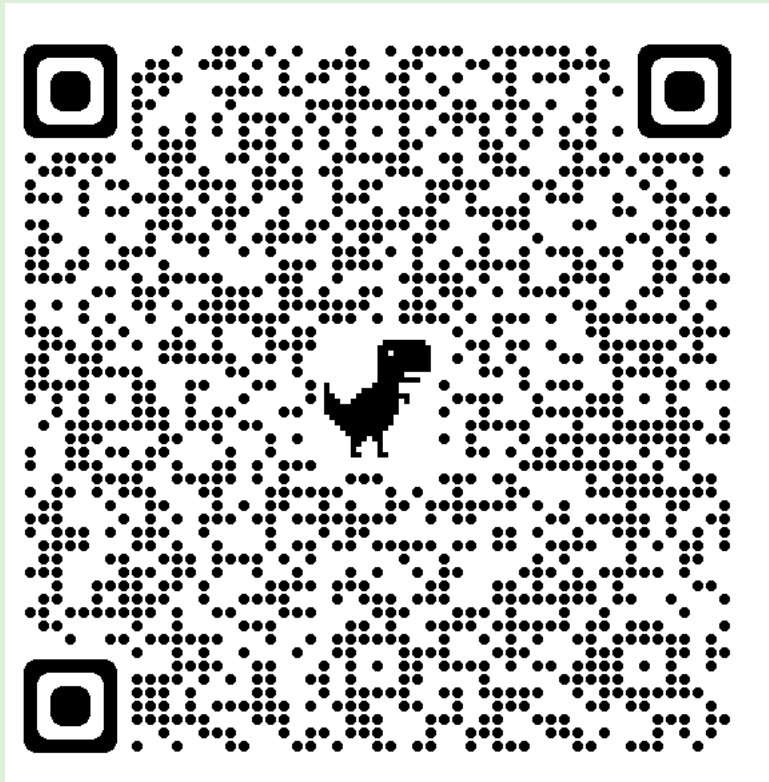
Approximately 12 minutes in length



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Fetal Circulation after birth

Approximately 13 ½ minutes in length



Oxygen Transport to the Fetus

- Despite a low PO_2 , the fetal blood transports large amounts of oxygen from the placenta to the fetal tissues
- Several fetal “adaptations” are responsible for this

Fetal Physiological Adaptations...

- Fetal blood has a higher hemoglobin concentration than adult blood. This allows for greater oxygen carrying capacity
- Fetal hemoglobin has a **higher affinity** for oxygen compared with maternal blood
- Fetus has a higher heart rate and cardiac output relative to its body size compared to the adults. This allows for more rapid circulation of oxygenated blood.
 - These adaptations support aerobic metabolism which can be maintained until the available oxygen in the intervillous space decreases to 50% of normal levels

RevisitingAerobic Metabolism

- Normal homeostasis requires an adequate supply of oxygen and fuel to generate the energy required for cellular function
- When aerobic metabolism occurs efficiently it generates energy in the form of adenosine triphosphate (ATP)
 - The by products of aerobic metabolism include carbon dioxide and water

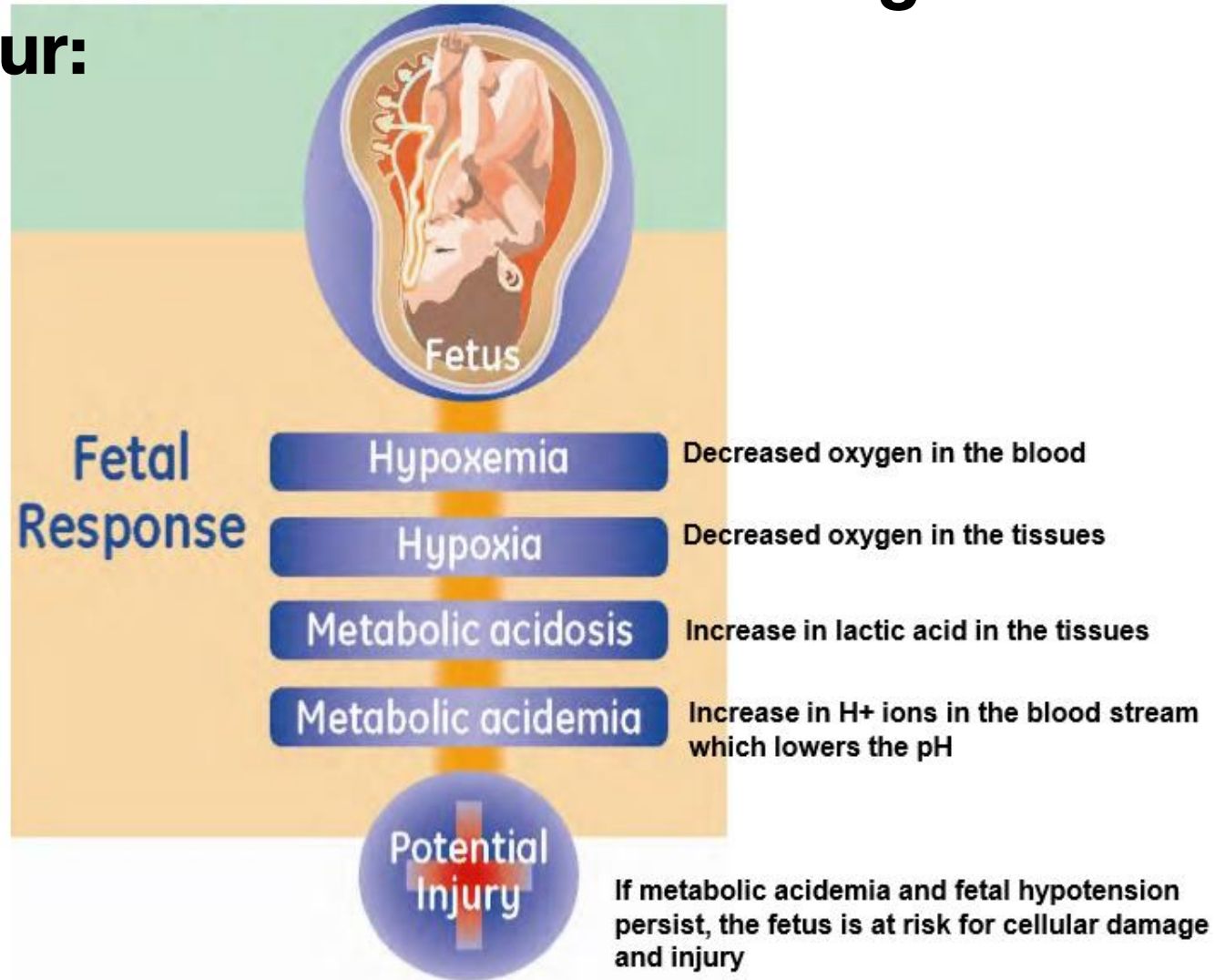
Revisiting Anerobic Metabolism

- When oxygen is in short supply, tissues may be forced to convert from Aerobic to Anerobic metabolism
- Energy is less effective and byproducts of anerobic metabolism is lactic acid
- The maternal placenta through the different transfer/transport methods, rids the fetus of carbon dioxide and lactic acid
 - However, removal of lactic acid requires more energy – Active Transport versus Passive Transport

With Transient Fetal Hypoxemia...

- The normal oxygenated fetus compensates by:
 - Shunting blood to the 3 vital organs
 - Brain, Heart and Adrenals
 - Decreases in total oxygen consumption – the fetal heart slows in response to less oxygen ; the myocardium consumes less oxygen and fetal oxygen consumption drops to 50% of normal levels

If the situation is not corrected the following cascade may occur:

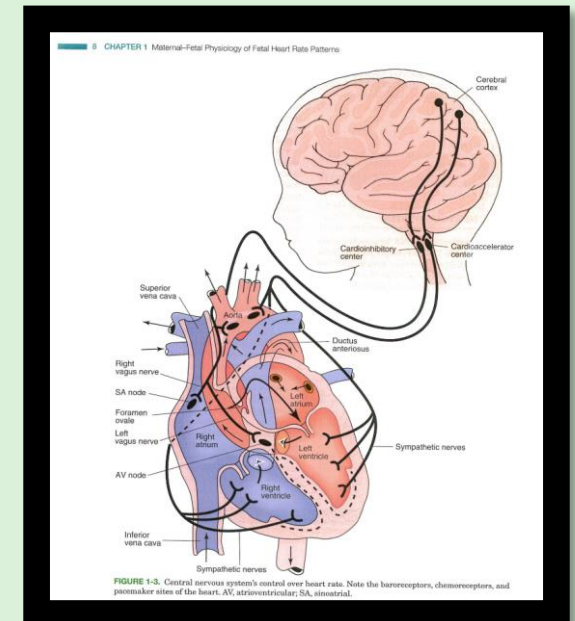


Adapted with permission from Dr. David Miller's presentation *Safer and Easier Establishing a Shared Mental Model in EFM*.(2013).

14th Annual National Conference on Fetal Monitoring Maternal-Fetal Assessment and Interventions

Factors Controlling Fetal Heart Rate

- Structure of the fetal heart is similar to the adult – the SA node functions as the intrinsic pacemaker
- Cardioregulatory Center (CRC) is a collection of neurons in the ventral and lateral surfaces of the medullar oblongata
- CRC receives impulses from a variety of sources:
 - Autonomic nervous system (parasympathetic & sympathetic)
 - Baroreceptors & Chemoreceptors
 - Fetal Hormones
 - Sleep wake cycles
 - Breathing movements
 - Painful stimuli
 - Sound and vibrations
 - Temperature



**Please Refer to the Handout Entitled:
Influences on Fetal Heart Rate Control**



WAIT, THERE IS MORE!
Just a Few More Points of Interest....



NORMAL PHYSIOLOGIC CHANGES OF PREGNANCY

↑ work on the ♥

- Heart rate ↑ 15-20 beats/minute
- Stroke volume ↑ 25-30%
- Cardiac output ↑ 30-50%

Dilutional Anemia

- Blood volume ↑ 40-60%
 - Greater ↑ plasma vs. RBCs
 - Results in hemodilution
 - ↓ in Hematocrit

Makes sepsis screening more difficult in pregnancy

- WBC ↑ to 15,000

Gestational age is important

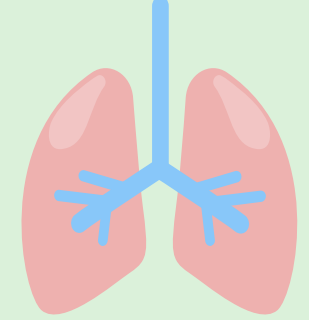
- Blood pressure is ↓ the second trimester


Pregnancy is a hypercoagulable state

↑ risk of VTE

- ↑ procoagulant activity and ↓ fibrinolytic activity

RESPIRATORY STRUCTURAL CHANGES



- Ph \uparrow 7.40-7.45
 - **Compensated respiratory alkalosis** 
 - This allows the transfer CO₂ from the fetus to the maternal circulation
 - Elevated levels of progesterone cause an increase in minute ventilation
 - Due to an increase in ventilation, pregnant patients typically experience mild respiratory alkalosis
- Expiratory Reserve Vol
 - \downarrow 15-20%
- \uparrow in Oxygen consumption
 - Shortness of breath is a common complaint
 - \uparrow in progesterone in pregnancy – Respiratory stimulant – may be the reason women complain of shortness of breath and hyperventilate during labor
- Hyperemia of the upper airway results from elevated estrogen levels and increased plasma volume can cause the tissues to become more friable and prone to increased bleeding.
- O₂ saturation should be at a minimum of **95%** to perfuse the fetus!

For Additional Information or Clarification

- AWHONN's Fetal Heart Monitoring Principles and Practices (2021) – Chapter Two
- Miller, Miller and Cypher (2022) – Chapter Two or Miller, Miller and Cypher (2027) – Chapter One
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QUESTIONS?



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- Fetal Circulation Right Before Birth
 - <https://youtu.be/-IRkisEtzsk>
- Fetal Circulation Right After Birth
 - <https://youtu.be/jFn0dyU5wUw>
- Fetal Heartrate Regulation
 - <https://149752527.v2.pressablecdn.com/wp-content/uploads/2018/05/Electronic-Fetal-Monitoring-Physiology-An-update-FINAL-20180509.pdf>