Anatomy and Physiology II
Exam #3 Review

1. Mix and match
2. What two major factors affect blood pressure?

Blood pressure(BP) = cardiac output(CO) x peripheral resistance(PR)

3. What is cardiac output and what two variables affect cardiac output?

Cardiac Output(CO) – The amount of the blood the heart pumps out of the right or left ventricles each minute.
CO = SV x HR
Stroke Volume(SV) – The amount of blood ejected from the right or left ventricle in each contraction
Heart Rate(HR) – The number of heart beats per minute

4. What is peripheral resistance? What 5 factors affect peripheral resistance and how do they affect peripheral resistance?

Peripheral resistance – The resistance to blood flow through the peripheral part of the circulatory system.
Factors that increase PR:
A. Increased blood volume
B. Increased blood viscosity (thickness)
C. Reduced vessel diameter
D. Increased vessel wall roughness
E. Increased vessel length

5. What is cardiac reserve? Compare the cardiac reserve of a well trained athlete with the cardiac reserve of a person with severe heart disease.

Cardiac Reserve = CO Max / CO at rest
Well trained athletes have a cardiac reserve that is 7-8 times their CO at rest. Severe heart disease can reduce a person's cardiac reserve to the point where the person can't do any exercise.

6. Define the end systolic volume and end diastolic volume and explain how they are used to calculate the stroke volume

End systolic volume (ESV) – The amount of blood still in the ventricles after the contraction is complete.
End diastolic volume (EDV) – The amount of blood in the ventricles after they have filled but prior to the beginning of the contraction.
Stroke Volume (SV) = EDV – ESV

7. What three factors influence the stroke volume.

A. Preload – As preload increases stroke volume increases.
B. Contractility – As contractility increases the stroke volume increases.

C. Afterload – As afterload increases stroke volume decreases.

8. What is preload and how does it affect to stroke volume? What is the Frank starling law?

Preload – The stretching of the ventricle when it is filled.
Frank Starling Law – The greater the stretching of the heart during filling of the chambers (i.e. preload), the greater the force of contraction.

9. What is contractility and how does it affect stroke volume? What affect do positive and negative inotropic agents have on contractility. Name three positive inotropic agents and three negative inotropic agents.

Contractility – The force of contraction of the individual muscle fibers in the heart.
Positive inotropic agents – Agents which increase contractility:
A. Sympathetic nerve signals to the heart
B. Epinephrine and norepinephrine – hormones which open calcium gates.
C. Increased plasma calcium levels
D. The drug digitalis
Negative inotropic agents – Agents which decrease contractility:
A. Inhibition of sympathetic nerves
B. Anoxia (i.e. low blood oxygen levels)
C. Acidosis (i.e. blood pH below 7.35)
D. Increased plasma potassium levels
E. Some anesthetics like halothane

10. What is afterload and how does it affect stroke volume? Name two factors that affect afterload.

Afterload – The pressure that must be exceeded before ejection of the blood from the ventricles (i.e.the pressure required to opent he semilunar valves).
A. High blood pressure – Makes it harder to force blood through the semilunar valves
B. Reduced vessel diameter (vasoconstriction)

11. List four neural or sensory systems that trigger the cardiovascular center of the medulla oblongata to increase or decrease the heart rate.

A. The limbic system of the cerebrum may increase the heart rate before exercise starts.
B. Proprioceptors trigger heart rate increase when you exercise.
C. Chemoreceptors sense high blood CO_2, low blood O_2, or low blood pH (H^+).
D. Baroreceptors sense blood pressure in the aortic arch and carotid sinus and increase the heart rate if the blood pressure drops.

12. Name four other factors that directly affect the heart rate by affecting the heart muscle.

A. Blood loss

B. Heart damage that reduces stroke volume
C. Exercise or increased tissue demand for oxygen
D. Low blood pressure

13. How do extracellular concentrations of K^+, Na^+, and Ca^{++} affect the heart rate.

High K^+ - decreases the heart rate by reducing membrane potential
High Na^+ - decreases the heart rate because it competes with calcium for the calcium gates.
High Ca^{++} - increases the heart rate as it increases the concentration gradient so the Ca^{++} diffuses into the cell faster.

14. Why does constriction of the veins increase the blood volume in circulation?

Typically 60% of the blood volume is in the veins (at rest), contriction of the veins reduces the volume of blood they can contain causing the blood pushed out of the veins to go to the heart.

15. What hormones affect blood volume and what do they do?

Aldosterone – from the adrenal cortex is released when blood plasma levels drop and is triggered through the renin-angiotensin pathway. Causes Na^+ to be reabsorbed from the urine to the blood (Na^+/K^+ pump). This increases blood plasma levels because it creates an osmotic gradient, increasing the reabsorption of water from the urine to the blood plasma by osmosis.

Antidiuretic Hormone (ADH) – from the pituitary is released in response to low blood pressure or low blood volume and increases the permeability of the distal convoluted tubule to water. This increases the blood volume because more water to be reabsorbed from the urine to the blood plasma by osmosis.

Atrial natriuretic peptide (ANP) – from the atria of the heart reduces blood volume by increasing the loss of water in the kidneys. It does this by increasing the glomerular filtration rate and causing vasodilation. It also inhibits the release of aldosterone and antidiuretic hormone. It is released from the atria when the right atria is stretched during filling.

16. What factors affect blood viscosity?

A. Increased movement of plasma into the tissues due to capillary dilation (blood becomes thicker, decreased viscosity). Histamine causes this.
B. Infections can trigger the immune system to increase blood proteins such as gamma globulins (i.e. antibodies) and white blood cells.
C. Dehydration reduces plasma in the blood.

17. Explain how tissue temperatures affect vasoconstriction or dialation?

High body temps – Causes vasodialation and more blood to the extremities and closer to the skin to reduce temps.
Low body temps – Causes vasoconstriction and forces the blood to the core.

18. Explain how low oxygen levels in the lungs and in the tissues affect vasodilation or constriction.
Systemic vessels: vasodialation increased blood to tissue
Pulmonary vessels: vasoconstriction to the areas of the lungs with low oxygen levels to improve gas exchange.

19. Which hormones cause vasodilation and which hormones or nerves cause vasoconstriction?
Hormones causing vasodialation:
Atrial natriuretic peptide: From the atria of the heart.
Epinephrine: Adrenal medulla – acting on adrenergic beta receptors. Norepinephrine has litte effect.
Calcitonin: The reduction of blood calcium levels reduces vasodialation.
Hormones casuing vasoconstriction:
Epinephrine & norepinephrine: Adrenal medulla – acting on adrenergic alpha receptors.
Renin-angiotensin: increases aldosterone increasing blood volume.
Antidiuretic hormone (ADH): pituitary – (vasopressin)
Calcitriol – The active form of vitamin D

20. How does vessel stretching affect vasoconstriction?
As vessels are stretched, the smooth muscle in them contracts, reducing vessel diameter and increasing peripheral resistance.

21. What increases the roughness of vessel walls?
Arteriosclerosis is the main factor increasing vessel wall roughness.

22. How does vessel length affect resistance to blood flow and what would causes vessel length to increase?
As vessel length increases, more blood/vessel wall contact thus increased peripheral resistance.

23. How does blood pressure change as blood travels from the left ventricle through the body to the right atrium?
Blood pressure decreases from the left ventricle to right atrium of the heart in the systemic circulation.

24. What happens if mean blood pressure drops below 60 mm Hg. What is shock? Explain three hormonal and neural responses to low blood pressure.
Shock – Inadequate cardiac output, occurs if the average BP is below 60 mm Hg.
A. Sympathetic nerves increase the heart rate and vasoconstriction.
B. Epinephrine and norepinephrine are released by the adrenal

medulla to increase the heart rate and vasoconstriction.
C. Renin-angiotensin system is activated which increases vasoconstriction and causes the release of aldosterone from the adrenal cortex. Aldosterone increases the reabsorption of sodium and water from the urine to the blood increasing blood volume.
D. Antidiuretic hormone (ADH) is released from the pituitary and increases blood volume.

25. What is normal blood pressure? What is primary hypertension. What is the difference between primary hypertension and secondary hypertension.
Normal BP: below 80/120, womens typically is lower then mens.
Primary hypertension – Cause is unknown
Secondary hypertension – Cause is known.

26. Explain three causes of secondary hypertension.
Aldosteronism - High levels of aldosterone
Kidney disease – Kidney blockage causing excessive rennin secretion which leads to the release of aldosterone
Pheochromocytoma – Tumors of the adrenal gland which cause the release of excessive epinephrine and norepinephrine. Can also be caused by stress.
Arteriosclerosis – Narrowing of the arteries due to plaque accumulatin.

27. What diseases problems are caused by hypertension?
Strokes, heart attacks, congestive heart failure, arteriosclerosis, kidney failure (due to tears in the glomeruli of the kidneys which are replaced with scar tissue), and detached retinas

28. How can hypertension be treated? List 5 things people can do to reduce high blood pressure.
A. Loose weight
B. Reduce alcohol intake
C. Exercise
D. Reduce sodium intake
E. Maintain proper dietary levels of potassium, calcium, and magnesium
F. Don’t smoke
G. Manage stress

29. What causes congestive heart failure. Explain how congestive heart failure occurs.
CHF is caused by coronary artery disease, long term high blood pressure and congenital heart defects.
Typically, CHF occurs on one side of the heart, due to blockage or low blood flow to that area of the heart. This causes that chamber to be less effective, reducing the overall flow of blood from the heart and thus reducing the oxygenation of the rest of the heart muscle. If the oxygen decrease is sufficient it will cause more muscle to die and spiral down to death.

30. List 5 factors that increase a person's risk of heart disease?

A. High blood cholesterol
B. High blood pressure
C. Cigarette smoking
D. Obesity
E. Lack of exercise
F. Diabetes mellitus
G. Genetic predisposition (family history of heart disease)
H. Male gender (after 70 risk becomes equal for men vs. women)

31. What are three things the lymphatic system does? How is lymph fluid reabsorbed back into the lymphatic system?

A. Collects fluid from the tissue and returns it to the cardiovascular system.
B. Returns proteins to the blood.
C. Carries bacteria and viruses from the tissues to the blood and helps initiate the immune response.
D. Moves lipids from the small intestine to the blood, including the fat soluble vitamins A, D, E and K.

Fluid moves into the lymph capillaries through intercellular clefts in the walls of the lymph capillaries. The vessels are held open by connective tissue fibers and the intercellular clefts act as a one way valve so the fluid from the tissue can move in through them, but not out. These openings also allow the bacteria and viruses to move from the tissue to the lymphatic system.

32. How are lymph nodes and the spleen alike and how are they different?

The spleen has chambers that resemble a lymph node, but the spleen has more blood circulating through it then a lymph does.

33. Explain the difference between specific and non-specific immunity with respect to activation, specificity of attack, and how well each is at removing an antigen.

Non-specific – Does not require activation, recognizes any foreign materials and attacks. Not very effective.
Specific – Produces cells that once activated attack a single specific antigen. Very effective at attacking that antigen.

34. List the parts of the non-specific or specific immune system and the function of the parts.

Non-specific:
Natural killer cells: Lymphocytes found in the spleen, liver, lymph nodes, and bone marrow that bond to foreign cells causing them to lyse. Can also lyse some tumor cells.
Neutrophils: Circulating white blood cells that phagocytize foreign cells.
Reticuloendothelial system (R.E. System): Has two parts. 1.

Fixed macrophage found in spleen, liver, lymph nodes, and the bone marrow. 2. Circulating macrophages called monocytes. Both phagocytize foreign cells.

Specific:

T-Cells (Cell mediated immunity) – lymphocytes that attack antigens directly.

B-Cells (Humoral immunity) – lymphocytes that produce antibodies

35. What is the major histocompatibility complex? Explain how these proteins function in cells. Explain the difference between in function and location of MHC-I and MHC-II proteins.

Histocompatibilty Complex – Antigens found on the surface of cells that mark cells as from the body or foreign.

MHC-I – Proteins found in the membranes of all cells except red blood cells. MHC-I can only present endogenous antigens (antigens from pathogens living within the cell).

MHC-II – Proteins found on special cells called antigen presenting cells. These are found on: macrophages/monocytes, B-Cells, Kupffer cells in the liver, microglia cells of the CNS, Langerhans cells of the skin, dendritic cells of the lymph nodes and spleen. Can present antigens from either endogenous or exogenous pathogens. Exogenous pathogens only when phagocytosis is being performed.

36. Explain in detail how B-cells or T-cells are activated. What is costimulation?

T-Cell Activation:

1. A tumor cell or foreign cell with a MHC-I protein on it's cell wall is recognized.
2. An immature T-Cell (T8 cell or CD8 cell) bonds to the MHC-I foreign cell complex.
3. Costimualation occurs – A signal is received by the T-Cell from a T-4 helper cell.

Costimulation of T-Cells:

1. A macrophage digests an antigen, part of it bonds to MHC-II proteins int eh cell sand this complex moves to the cell membrane.
2. The T-Cell receptor site on a T-4 helper cell bonds to the MHC-II/foreign protein complex on the macrophage membrane, starting activation of the T-4 helper cell. The macrophage also releases interleukin-1 (IL-1) to the T-4 helper cell causing it to divide rapidly.
3. The T-4 helper cells produce interleukin-2 (IL-2) which activates the T-cells that have already bonded with a MHC-I/foreign protein complex. The IL-2 causes the T-cells to form cytotoxic T-Cells (activated T-Cells) and causes the T-cells to also divide rapidly. NOTE: The IL-2 also causes B-Cells, T-4 helper cells, and natural killer cells to divide rapidly.
4. An activated T-Cell is called a cytotoxic T-cell or a T-killer cell.

B-Cell Activation:

1. The B-cells have antibodies on their membranes that act as antigen receptor sites. These antibodies bind the same antigens as the macrophages use for costimulation of the B-Cells.
2. Costimulation occurs.

Costimulation of B-Cells:

1. A macrophage digests an antigen, part of it bonds to MHC-II proteins in the cell sand this complex moves to the cell membrane.
2. The T-Cell receptor site on a T-4 helper cell bonds to the MHC-II/foreign protein complex on the macrophage membrane, starting activation of the T-4 helper cell. The macrophage also releases interleukin-1 (IL-1) to the T-4 helper cell causing it to divide rapidly.
3.The T-4 helper cell picks up the pieces of the antigen from the macrophage and carries it to the B-Cell. The antigen bonds to MHC-II/foreign protein complex on the B-Cell. The T-4 helper cell also releases interleukin-2 (IL-2) to the B-Cell. It is unclear if the bonding of the antigen or the IL-2 activates the B-Cell, but it does become active forming a plasma cell (B-Cell that secretes antibodies).

NOTE: The T-4 helper cell also releases interleukin-4 which causes the B-Cells to release IgE and interleukin-5 which causes the B-Cells to release IgA antibodies.

4. Activated B-Cells (plasma cells) divide rapidly and produce proteins called antibodies that attack a specific antigen. Antibodies cause antigens to be bonded together which increases the destruction by macrophages and other immune cells.

37. What is the difference in how cytotoxic T-cells and B-cells attack antigens?

38. What do B-cells attack and what do T-cells attack?

B-Cells primarily attack:

Bacteria

T-Cells primarily attack:

Transplanted tissue
Cancer cells
Viral infections
Fungal infections
Parasites

39. How are the five classes of antibody different? Which are most common and least common in the blood? Which class crosses mucus membranes? Which class crosses the placenta? Which classes act as receptor sites on B-cells? Which classes are involved in allergic reactions?

IgG – 75%, Blood, Lymph & Intestine
- Protects against: Bacteria & Viruses
- Only class that cross placenta

IgA – 15%, sweat, tears, saliva, mother’s milk and gastrointestinal secretions

- Protects against: Bacterial and viral infections of mucus membranes
- Crosses mucus membranes in nose, lungs and gut
- Often involved in allergic reactions including food allergies

IgM – 5-10%, Blood and Lymph
- First antibody to be secreted after initial contact with an antigen
- Activates complement and causes agglutination and lysis of microbes.
- Acts as antigen receptor on B-Cells
- Involved in food allergies.

IgD – 1%, Blood, Lymph and on surface of B-Cells (receptor sites)
- Acts as antigen receptor sites on B-Cells

IgE - < 0.1%, Mast cells, Basophiles
- Triggers histamine release
- Involved in allergic reactions
- Protects against parasitic worms.

40. Why does immunization work? Be able to explain the primary and secondary immune responses.

Immunization involves the process of inducing a small amount of a foreign substance into the body so that the B-Cells and T-Cells can become activated against this particular antigen. Once activated some of the T-Cells and B-Cells produce memory cells which remain and are already active to fight this specific antigen. Therefore, when the antigen appears again, these cells will begin the fight much faster before the foreign cells have become established. This second and faster response is called the secondary or anamnestic response.

Also, in the primary (first) response to an antigen, the body produces equal amounts of IgG and IgM antibodies. In the secondary response the body produces 4-5 times as many IgG antibodies as it does IgM.

41. What are gamma globulin shots and what do they do?

Gamma globulin injections are usually given in an attempt to provide a temporary boost to a patients immunity against a disease. Typically used for those exposed to Hepatitis A or measles.

Gamma globulin shots contain fused cells made up of a plasma cell (activated B-Cell) and a tumor cell. These cells grow rapidly and produce large quantities of a single type of antibody (monoclonal antibodies).

42. What causes type I hypersensitivity allergic reactions and how can they be treated? What happens in anaphylactic shock and how can it be treated?

Type I hypersensitivity = anaphylaxis/immediate hypersensitivity.
- Reactions occur within minutes
- Repeated exposure to an antigen causes sensitivity to it and IgE antibodies attach to the surface of mast cells and basophiles in mucus membranes and cutaneous membranes.

- when the antigens enter the body, they bond to the antibodies on the mast cells and basophiles causing them to release histamine, prostaglandins, leukotrienes and kinin. These compounds cause inflammation, vasodilation, tissue swelling, increased mucus production and contraction of smooth muscle in the lungs.
- Responsible for allergies, can be as minimal as eyes watering and itching to life threatening (anaphylactic shock)
- Anaphylactic shock = hives, vasodialation to the extent that blood pressure drops dangerously low, bronchioles/throat may constrict blocking the airway. Tx: epinephrine to increase blood pressure, antihistamines or albuterol to open up the airway.

43. What occurs in type II hypersensitivity reactions.
Type II = Cytotoxic reactions or antibody dependent
Typically take 1-3 hours to occur.
Reactions to foreign blood in the body, involve IgG, IgM antibodies and complement
Cause blood rejection when the wrong blood type is given.

44. Explain what happens in type III hypersensitivity reactions and why they can cause autoimmune diseases.
Type III = Immune complex disorders (autoimmune diseases)
Typically take 1-3 hours to occur.
Involve IgA, IgM antibodies and complement
Occur when certain ratios of antigen to antibody occur in the body, the antigen/antibody complexes are small and become trapped under the basement membrane of the endothelium of blood vessels, causing inflammation and constriction of the vessels and thus greatly reducing the blood flow through them.
Causes several autoimmune diseases: lupus, glomerulonephritis and rheumatoid arthritis.
Lupus: also damages tissue in the body when the antigen/antibody complex is attacked by other parts of the immune system.
Glomerulonephritis: causes inflammation in the kidneys leading to damaged kidney tissue and scar tissue in the kidneys, thus reduced function of kidneys.
Rheumatoid arthritis: Attack of the antigen/antibody tissue causes damage to the synovial membranes, which grow thicker as they repair the damage.

45. What are haptens and explain how they are involved in type IV hypersensitivity allergic reactions?
Type IV = delayed hypersensitivity
Typically take 12-72 hours
Can be transferred to a person during a blood transfusion.
Involve activated T-Cells (also T-4 helper cells and macrophages)
Important for protecting the body from parasite, viral and fungal infections, also involved in transplant tissue rejection.
Molecules from antigens called **haptens** move across the mucus membrane or cutaneous membranes and bond with proteins. The antigen/antibody complexes are picked up by antigen presenting

cells such as Langerhans cells in the skin and migrate to the lymph nodes, where they present their antigens to T-Cells. The activated T-Cells release interferon which activates the macrophages to release tumor necrosis factor which causes inflammation and rashes of the skin (dermatitis).
Common haptens: poison oak, poison ivy, cosmetics, deodorants, and heavy metals.

46. List and explain 5 things in the body other than the immune system that help prevent infection and disease.
Epidermis of the skin: forms a barrier to parasite and microbe infection
Mucus membranes: Help prevent microbes from moving into the body
High acidity of stomach: Many parasites cannot pass through without being in a protected egg.
IgA antibodies: The only antibodies that can cross the mucus membrane.
Human Milk: Carries substances that kill Entamoeba, histolytica, and Giardia lamblia.
Inflammation: Tissue swells so lymphocytes can enter the area and attack foreign cells.
Fibroblasts: Produce connective tissue and build walls around parasites.
Fevers: Higher temperatures during an infection increases the effectiveness of interferon and phagocytic activities of macrophages while hindering the replication of some pathagens.

47. How does the HIV virus affect the immune system?
HIV/AIDS knocks out the T-4 helper cells preventing activation of the specific immune systems.

48. What causes general adaptation syndrome and what does it cause to happen? Explain how it affects susceptibility to infection and disease
GAS is caused by stress.
Effects of alarm stage (activation of fight or flight response):
- Increase: Heart rate, breathing rate, vasoconstriction, blood flow to skeletal muscles, blood glucose levels
- Decrease: Digestion
Effects of resistance stage (release of hormones):
- Hypothalamus CRT (Corticotropin releasing hormone) -> Pituitary ACTH (Adrenocorticotropic hormone)-> Adrenal cortex Cortisol
-- Increases: Gluconeogenesis from lipids, protein catabolism, vasoconstriction
-- Decreases: Inflammation, connective tissue formation
- Hypothalamus GHRH (Growth hormone releasing hormone) -> Pituitary hGH (Human growth hormone)
-- Increases: Blood pressure
- Hypothalamus TRH (Thyrotropin releasing hormone) -> Pituitary TSH (Thyrotropin stimulating hormone) -> Thyroid T3/T4

(Thyroxine and triiodothyronine)
-- Increases: Metabolism
- Adrenal cortex – Aldosterone
-- Increases: Sodium reabsorbtion in the kidneys, water reabsorbtion in the kidneys, increased blood pressure, lower blood pH.

How effects immune system:

- fevers are reduced
- inflammation is reduced
- connective tissue formation is reduced
- neutrophil production drops
- lymphocyte production drops
- gamma globulin formation drops
- interferon production is reduced
- break down of immune system proteins such as complement is increased

Overall: The likelihood of infection and disease is increased when a person is stressed due to the GAS effects on the immune system.