MAKING THE CASE:

Reinforcing A&P Concepts with Simple Case Studies

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**MAKING THE CASE:**
Reinforcing A&P Concepts with Simple Case Studies

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Introduction

Advantages of Using Case Studies in A&P

- To illustrate real-world relevance of the A&P content taught in class
- To integrate content from multiple body systems into a single activity
- To require students to engage in critical thinking
- To get students actively involved in the learning process
- To make students look at different viewpoints and scenarios
- To provide a good opportunity for assigned group work

Ways to Use Case Studies

- Homework assignment
- Group activity in class
- Group activity outside of class
- Problem-based learning
- Multi-part “interrupted” activity
- Drama with role-playing
- Preparation to answer exam questions

Where to Get Cases

- Write your own cases
- Use cases written by others - some sources include:
  - National Center for Case Study Teaching in Science (NCCSTS): http://sciencecases.lib.buffalo.edu/cs/
  - Problem-based Learning (PBL) at University of Delaware http://www1.udel.edu/inst/
  - Hematology Case Studies at University of Delaware http://udel.edu/~rmaser/hematology/case-studies/
INSTRUCTIONS
This case study is designed to help students review the anatomy of the nervous system, primarily the brain, and apply their knowledge to one of the brain pathologies. This case study will be implemented during a regular class time and will include the following steps:

1. The students will form groups of 4-5 students per group, each group will receive the case study scenario, one sheet of self-stick easel paper (to be hung on a wall or whiteboard) and 2 markers.

2. Using their notes and figures in the textbook, the students will answer all questions listed under “QUESTIONS ABOUT CASE STUDY.”

3. Classroom discussion will start after about 30 minutes; the groups will take turns presenting their answers to 2-4 case study questions (since all questions will be addressed, the number of questions per group will depend on the number of groups formed by all students in the class).

CASE STUDY
Mrs. Sweetheart (74 years old) has lived alone for the past several years. Her daughter, Tara, who lives in another city, calls Mrs. Sweetheart each week although she has not seen her mother for about 6 months.

During the last phone call, Tara became concerned. Her mother seemed distracted, frequently interrupted the conversation and repeatedly said that she was "so worried." When asked what worried her, Mrs. Sweetheart said, "I just don't know."

When Tara arrived, she was shocked to see how thin her mother had become. Mrs. Sweetheart's skin turgor (rigidity of cells or tissues, typically due to the absorption of fluid) was sluggish.

Mrs. Sweetheart said the coffeemaker and the TV did not work. The daughter used both and found them to be working. Mrs. Sweetheart often started tasks but did not finish them, she seemingly forgot what she was doing. She often could not think of simple words, such as the word "dresser" for the piece of furniture in her bedroom. As evening approached Mrs. Sweetheart became more agitated and was unable to sleep. She said she had to "see about the children."

The next day during the doctor's examination, Mrs. Sweetheart was unable to focus on the nurse's questions and instructions. She knew her own identity, was unsure of her exact location and did not know the current date. She became visibly agitated with the questions and said she didn't want to answer or says "I don't know, well I do know but I am not going to answer." (This was probably because she was unable to answer). Mrs. Sweetheart thought the physician was the son of one of her friends from home and asked him several times about his mother. She complained of fatigue and epigastric tenderness. She was 20 pounds under her ideal body weight and she was pale. Lab tests revealed iron deficiency anemia, low albumin, and dehydration.

Mrs. Sweetheart is showing moderate signs of confusion.
1. She is oriented to person (she knows her daughter), but not to place and time.
2. Her judgment is poor as reflected in not making good decisions about eating.
3. She is showing signs of "sundowning," which means she is more agitated in the evening and is unable to sleep. This is a symptom consistent with stage II Alzheimer's disease.
4. She repeatedly asks the same question (amnesia: loss of ability to retain new information or recall old information, which often appears as forgetfulness).

5. Other signs are
   a. apraxia: inability to perform purposeful movements, like making coffee or controlling the TV
   b. agnosia: inability to recognize common objects
   c. aphasia: inability to speak or to understand spoken or written language
* Note that amnesia, apraxia, agnosia and aphasia are the “4 A’s of Alzheimer's.”

QUESTIONS ABOUT CASE STUDY

Part 1: Microscopic and macroscopic changes in the brain

1. Increased number of active neuroglia
   o List the neuroglia located in the CNS and name the function of each.

2. Reduced level of acetylcholine
   o Define acetylcholine and acetylcholinesterase, and list the function of each in the brain.

3. Narrowed gyri and widened sulci of the brain in the:
   o frontal lobe (prefrontal cortex)
     ✓ Locate this area and name its functions.
   o parietal lobe (superior gyri)
     ✓ Locate this area and name its functions.
   o temporal lobe (inferior gyri)
     ✓ Locate this area and name its functions.

4. Areas not affected and fairly well preserved
   o precentral gyrus
     ✓ Locate this area and name its functions.
   o postcentral gyrus
     ✓ Locate this area and name its functions.
   o occipital gyrus
     ✓ Locate this area and name its functions.

5. Ventricular enlargement
   o List the brain ventricles and name their functions.

Part 2: Linking brain changes with behavioral changes

The ten warning signs of Alzheimer's disease are listed as:
1. Recent memory loss that affects job skills
2. Difficulty performing familiar tasks
3. Problems with language
4. Disorientation to time and place
5. Poor or decreased judgment
6. Misplacing items
7. Changes in mood or behavior
8. Changes in personality
9. Loss of initiative
10. Problems with abstract thinking

Based on your answers and discussion from Part 1, explain the relationship between the microscopic and macroscopic changes seen in the brain and the ten warning signs associated with Alzheimer’s disease.

This case study was adapted from http://www.austincc.edu/adnlev1/rnsg1341online/neurosensory/celia.html
1. **Increased number of active neuroglia**: List the neuroglia located in the CNS and name the function of each.

   **Answer:**
   - **Astrocytes** – assist in formation of blood-brain barrier, facilitate transport of nutrients and gases between blood vessels and neurons; regulate extracellular environment of brain (regulate ions & gases); may influence synaptic signaling by secreting/removing neurotransmitters (essential for learning & memory)
   - **Oligodendrocytes** – have radiating processes with flattened sacs that wrap around axons of nearby neurons to form myelin; single oligodendrocyte may form myelin sheath around portions of several axons
   - **Microglia** – activated by injury within the brain; become wandering phagocytic cells that ingest disease-causing microorganisms, dead neurons, and cellular debris
   - **Ependymal cells** – epithelial ciliated cells lining brain ventricles and central canal of spinal cord; form choroid plexus (ependymal cells + blood vessels) that functions to manufacture and circulate cerebrospinal fluid (CSF)

2. **Reduced level of acetylcholine**: Define acetylcholine and acetylcholinesterase, and list the function of each in the brain.

   **Answers:**
   - **Acetylcholine** (ACh) – a small molecule neurotransmitter which binds to cholinergic receptors in postsynaptic neurons; usually excites postsynaptic neurons
   - **Acetylcholinesterase** (AChE) – an enzyme that degrades ACh in synaptic cleft; by-products of reaction are taken back into presynaptic neuron for recycling and reuse

3. **Narrowed gyri and widened sulci of the brain in the:**

   - **frontal lobe** (prefrontal cortex): Locate this area and name its functions.
     **Answer** – most anterior lobe of the cerebrum; contains the prefrontal cortex which is responsible for complex mental functions such as behavior, conscience, and personality
   - **parietal lobe** (superior gyri): Locate this area and name its functions.
     **Answer** – posterior to frontal lobe and central sulcus; contains postcentral gyrus; responsible for processing and integrating sensory information and functions in attention
   - **temporal lobes** (inferior gyri): Locate this area and name its functions.
     **Answer** – form lateral surfaces of each cerebral hemisphere; separated from parietal and frontal lobes by lateral fissure; involved in hearing, language, memory, and emotions
4. Areas not affected and fairly well preserved

- **precentral gyrus**: Locate this area and name its functions.
  
  **Answer** – located in the frontal lobe, immediately posterior to the central sulcus; contains the primary motor cortex; involved in conscious planning of movement

- **postcentral gyrus**: Locate this area and name its functions.
  
  **Answer** – located in the parietal lobe; makes the primary somatosensory area that receives input from receptors in skin, skeletal muscles, and joints

- **occipital gyri**: Locate this area and name its functions.
  
  **Answer** – posterior gyri of the occipital lobe; responsible for vision

5. Ventricular enlargement: List the brain ventricles and name their functions.

  **Answer** – four brain ventricles are linked cavities that are continuous with central canal of spinal cord; they are lined with ependymal cells and filled with cerebrospinal fluid
  
  - Right and left lateral ventricles (first and second ventricles); within their respective cerebral hemispheres
  - Third ventricle – narrow cavity found between two lobes of diencephalon; connected to lateral ventricles by interventricular foramen
  - Fourth ventricle – between pons and cerebellum; connected to third ventricle by cerebral aqueduct (in midbrain)
    - Continuous with central canal of spinal cord
    - Contains several posterior openings that allow CSF in ventricles to flow into subarachnoid space

Based on your answers and discussion from Part 1, explain the relationship between the microscopic and macroscopic changes seen in the brain and the ten warning signs associated with Alzheimer’s disease.

**Answer** – Most of the warning signs of Alzheimer’s disease are associated with changes in the anatomy and functions of the frontal, parietal and temporal lobes.

Specifically:
1. Recent memory loss that affects job skills – frontal lobes (prefrontal cortex) and temporal lobes (amygdala and hippocampus)
2. Difficulty performing familiar tasks – frontal lobes (prefrontal cortex)
3. Problems with language – frontal lobes (Broca’s area) and temporal lobes (Wernicke’s area)
4. Disorientation to time and place – frontal lobes, parietal lobes (spatial navigation) and temporal lobes (the hippocampus)
5. Poor or decreased judgment – frontal lobes
6. Misplacing items – frontal lobes (prefrontal cortex; working memory)
7. Changes in mood or behavior – frontal lobes, temporal lobes (amygdala and hippocampus) and hypothalamus
8. Changes in personality – frontal lobes (prefrontal cortex)
9. Loss of initiative – frontal lobes (prefrontal cortex)
10. Problems with abstract thinking – frontal lobes (prefrontal cortex)
INSTRUCTIONS
You may work with classmates to discuss the case study, but you must write your own answers. You should not get help from faculty in the tutoring center for answers to the homework. Identical answers will count as cheating. Your answers have to be typed or word-processed, proofread, double-spaced & contain proper grammar and spelling. The assignment can only be submitted through Canvas. Hand written assignments will not be accepted. This assignment can be submitted any time before the due date, but no later than the due date/time.

One bonus point
Due Date __________

CASE STUDY
Margaret Wilson, a 10-year-old African American female, went to see her primary care physician. She complained of having shortness of breath, fatigue, and pain in her chest and joints. Upon taking a family history, the physician realized that Margaret’s mother experiences similar symptoms, while her father shows none of these symptoms. The physician ordered a CBC and a blood smear. The most significant test results were:

- Hematocrit 25%
- Hemoglobin 8 g/dL
- Sickled erythrocytes

From the clinical symptoms and the blood tests, the doctor made the diagnosis of sickle cell anemia. Sickle cell anemia is an autosomal recessive disorder caused by a defect in the gene that codes for hemoglobin. The normal gene for hemoglobin is designated as \text{Hb}^A, while the defective gene is designated as \text{Hb}^S. Margaret’s mother is homozygous recessive for the condition and her father is heterozygous.

QUESTIONS ABOUT CASE STUDY
For questions on genetics you can refer to chapter 29 (Development & Inheritance) of your textbook. Use \text{Hb}^A or \text{Hb}^S to indicate appropriate genes.

1. a. What does the term “homozygous recessive” mean?
   b. What does the term “heterozygous” mean?

2. a. What is the genotype for Margaret?
   b. What is the genotype for Margaret’s mother?
   c. What is the genotype for Margaret’s father?

3. Make a Punnett square using Margaret’s parents to show inheritance of sickle cell anemia.

4. Use your Punnett square to explain the percent chances for Margaret’s parents to have:
   a. a normal child (not affected and not a carrier) and then give the genotype of that child.
   b. a child who is a carrier for sickle cell anemia and then give the genotype of that child.
   c. a child who is affected by sickle cell anemia) and then give the genotype of that child.
5. a. What does “CBC” stand for?
   b. What specific tests are included in a CBC? List at least five different tests.
   c. What is the normal range for hematocrit? (Look it up in your textbook or applications manual. If you use another source please give a reference.)
   d. Is Margaret’s hematocrit high or low as compared to the normal range?
   e. Explain specifically what is happening in sickle cell anemia that would result in this change in the hematocrit.

6. Explain why sickle cell anemia would cause shortness of breath.

7. Explain why sickle cell anemia would cause pain in joints.

8. Explain why sickle cell anemia would cause fatigue.
Case #2
ANSWER KEY
Genetics & Blood
Presenter – Javanika Mody

1. a. What does the term “homozygous recessive” mean?
   Answer - a genotype having same recessive allele at the same locus on both members of a pair of homologous chromosomes

   b. What does the term “heterozygous” mean?
   Answer - a genotype consisting of two different alleles of a gene for a particular trait

2. a. What is the genotype for Margaret?
   Answer - Hb\textsuperscript{S} Hb\textsuperscript{S}

   b. What is the genotype for Margaret’s mother?
   Answer - Hb\textsuperscript{S} Hb\textsuperscript{S}

   c. What is the genotype for Margaret’s father?
   Answer - Hb\textsuperscript{A} Hb\textsuperscript{S}

3. Make a Punnett square using Margaret’s parents to show inheritance of sickle cell anemia.
   Answer:
   \[\begin{array}{ccc}
   & Hb^A & Hb^S \\
   Hb^S & Hb^A Hb^S & Hb^S Hb^S \\
   Hb^S & Hb^A Hb^S & Hb^S Hb^S \\
   \end{array}\]

4. Use your Punnett square to explain the percent chances for Margaret’s parents to have:
   a. a normal child (not affected and not a carrier) and then give the genotype of that child.
   Answer - 0 %, Hb\textsuperscript{A}Hb\textsuperscript{A}

   b. a child who is a carrier for sickle cell anemia and then give the genotype of that child.
   Answer - 50%, Hb\textsuperscript{A}Hb\textsuperscript{S}

   c. a child who is affected by sickle cell anemia) and then give the genotype of that child
   Answer - 50%, Hb\textsuperscript{S}Hb\textsuperscript{S}

5. a. What does “CBC” stand for?
   Answer - Complete Blood Count
b. What specific tests are included in a CBC? List at least five different tests.

Possible Answers include: total WBC count, total RBC count, total platelet count, differential count, hematocrit, hemoglobin measurement, reticulocyte count

c. What is the normal range for hematocrit? (Look it up in your textbook or applications manual. If you use another source please give a reference.)

Answer - male range is between 40 to 54%; female range is between 37 to 47%

d. Is Margaret’s hematocrit high or low as compared to the normal range?

Answer - low

e. Explain specifically what is happening in sickle cell anemia that would result in this change in the hematocrit.

Answer - Sickled RBCs are fragile and can hemolyze. Sickled RBCs that get stuck in capillaries will ultimately be phagocytized. Both of these processes reduce the number of red blood cells in circulating blood.

6. Explain why sickle cell anemia would cause shortness of breath.

Answer - Normal RBCs are flexible and biconcave in shape. They can easily pass through the blood vessels. RBCs of a person with sickle cell anemia become sticky, rigid and fragile. These irregularly-shaped cells can get stuck in capillaries, resulting in slow or blocked blood flow (micro-occlusion). Sickle-shaped RBCs also have reduced oxygen-carrying capacity, resulting in reduced oxygen supply to the parts of the body.

7. Explain why sickle cell anemia would cause pain in joints.

Answer - The most common cause of pain is blockage of capillaries in the joints causing low oxygen levels or ischemia.

8. Explain why sickle cell anemia would cause fatigue.

Answer - Sickle-shaped RBCs have reduced oxygen-carrying capacity, resulting in reduced oxygen supply to parts of the body. This can result in fatigue.
INSTRUCTIONS

You may work with classmates to discuss the case study, but you must write your own answers. You should not get help from faculty in the tutoring center for answers to the homework. Identical answers will count as cheating. Your answers have to be typed or word-processed, proofread, double-spaced & contain proper grammar and spelling. The assignment can only be submitted through Canvas. Handwritten assignments will not be accepted. This assignment can be submitted any time before the due date, but no later than the due date/time.

CASE STUDY

John Smith, a 60 year old male, was eating dinner when he suddenly started drooling from the right side of his mouth and the knife fell from his right hand. When asked by his wife what was going on, he could not talk. When he tried to reply, his face pulled to the left side. His wife asked him to lie down, but when he got up his right leg was dragging. Twenty minutes later he could talk normally but complained about numbness on the right side of his body including his face. After ten more minutes everything was back to normal.

His wife called the nurse practitioner, who was covering for the family physician. She insisted that Mr. Smith be taken to the emergency room. The following data were collected in the ER:

- Blood pressure 170/90 mmHg
- Bruit (murmur) in left internal carotid artery
- Cholesterol 300 mg/dL
- LDL 210 mg/dL
- His father had a myocardial infarction (MI) at age 54
- Mr. Smith smokes 1 pack of cigarettes each day
- CT scan of brain is normal

The ER physician prescribed 325 mg/day of aspirin. Mr. Smith wondered why aspirin would be a preferred treatment for his condition. Upon research he found the following information on aspirin:

- Aspirin is an analgesic, non-steroidal anti-inflammatory, antipyretic and anti-platelet agent. Aspirin in a dose of 80 to 325 mg/day is used prophylactically to reduce the likelihood of transient ischemic attacks and thromboembolic diseases of the cerebral vessels for individuals with coronary artery disease. Aspirin causes prolongation of bleeding time. The same anti-platelet effects can also result in intestinal bleeding, peptic ulcers, stomach pain, heartburn, nausea and vomiting.
QUESTIONS ABOUT CASE STUDY

The Nervous system
1. What is the most likely diagnosis for Mr. Smith?
2. The ER assessments detected a problem with Mr. Smith’s left internal carotid artery, yet all of Mr. Smith’s symptoms were on the right side of the body. Provide an anatomical explanation for why this was observed.
3. Explain why Mr. Smith’s face pulled to the left side when he tried to talk?
4. Damage to what specific part of Mr. Smith’s brain caused his speech difficulty? Provide the specific name of this part. Indicate if this would be on the right or left side of the brain.
5. Damage to what specific part of Mr. Smith’s brain resulted in dragging his right leg? Indicate if this would be on the right or left side of the brain.

The Cardiovascular system
1. a. Name the specific blood vessel which precipitated Mr. Smith’s signs and symptoms. 
   b. Where is this blood vessel located in the human body?
2. Trace a drop of blood from right atrium to this blood vessel.
3. Look up the meaning of the following terminologies and explain them in your own words: 
   analgesic
   anti-inflammatory
   antipyretic
   anti-platelet agent
   thromboembolic diseases
4. Even though his symptoms disappeared after 30 minutes why did the doctor advise Mr. Smith to take aspirin every day?
5. According to his research, aspirin is used prophylactically to prevent transient ischemic attacks and thromboembolic diseases of cerebral vessels. How exactly will the medication be useful to him? In other words what is the mechanism of action for aspirin in preventing these conditions?
6. a. Using your textbook find the normal values for:
      Blood pressure
      Cholesterol
      LDL
   b. Are Mr. Smith’s values for blood pressure within normal limits? If not, which values are too high or too low?
   c. Is his value for cholesterol within normal limits? If not, is it too high or too low?
   d. What does LDL stand for?
   e. Is his value for LDL within normal limits? If not, is it too high or too low?
7. Based on the data collected in the ER, list all of the risk factors that Mr. Smith has which probably led to his transient episode.
Case #3
ANSWER KEY
Nervous & Cardiovascular Systems
Presenter – Javanika Mody

The Nervous system

1. What is the most likely diagnosis for Mr. Smith? 
   **Answer** - transient ischemic attack (TIA); I do not accept cerebrovascular accident (CVA) for the answer

2. The ER assessments detected a problem with Mr. Smith’s left internal carotid artery, yet all of Mr. Smith’s symptoms were on the right side of the body. Provide an **anatomical** explanation for why this was observed.
   **Answer** - Decussation of sensory as well as motor fibers, the symptoms are felt on the opposite side of the body

3. Explain why Mr. Smith’s face pulled to the left side when he tried to talk?
   **Answer** - Motor neurons in the primary motor cortex on the left side of the body temporarily ceased to function due to lack of oxygen. Thus nerve impulses could not be sent to facial muscles on the right side of the face. However, there was no damage to the right primary motor cortex, so the muscles on the left side of the face were still stimulated to contract and pull.

4. Damage to what specific part of Mr. Smith’s brain caused his speech difficulty? **Provide the specific name of this part.** Indicate if this would be on the right or left side of the brain.
   **Answer** - Broca’s Area or Motor Speech Area, located on the left side

5. Damage to what specific part of the Mr. Smith’s brain resulted in dragging his right leg? Indicate if this would be on the right or left side of the brain.
   **Answer** - Primary motor cortex in frontal lobe of cerebrum on the left side

The Cardiovascular system

1. a. Name the specific blood vessel which precipitated Mr. Smith’s signs and symptoms.
   **Answer** - Left internal carotid artery

   b. Where is this blood vessel located in the human body?
   **Answer** - From the neck it enters the skull through the left carotid foramen

2. Trace a drop of blood from right atrium to this blood vessel.
   **Answer** - R atrium, tricuspid valve, R ventricle, pulmonary valve, pulmonary trunk, pulmonary artery, lungs, pulmonary vein, L atrium, bicuspid valve, L ventricle, aortic valve, ascending aorta, arch of aorta, L common carotid artery, L internal carotid artery
3. Look up the meaning of the following terminologies and explain them in your own words:

**Answers:**
- *analgesic:* medication that reduces pain
- *anti-inflammatory:* medication that reduces the cardinal signs and symptoms of inflammation
- *antipyretic:* medication that reduces fever
- *anti-platelet agent:* medication that prevents action of platelets to aggregate and initiate a blood clot
- *thromboembolic diseases:* class of disorders that involve formation of a thrombus (stationary clot) or an embolus (mobile clot)

4. Even though his symptoms disappeared after 30 minutes why did the doctor advise Mr. Smith to take aspirin every day?

**Answer:** He has several risk factors, including a murmur in his internal carotid artery, indicating the presence of plaque which causes turbulent blood flow. Thus he is at a higher risk of developing thromboembolic diseases. He is advised to take aspirin because it works to decrease platelet aggregation and will make new blood clots less likely to develop.

5. According to his research, aspirin is used prophylactically to prevent transient ischemic attacks and thromboembolic diseases of cerebral vessels. How exactly will the medication be useful to him? In other words what is the mechanism of action for aspirin in preventing these conditions?

**Answer:** Aspirin prevents platelets from sticking to each other. This prevents platelet plug and blood clot formation.

6. a. Using your textbook find the normal values for:

**Answers:**
- *Blood pressure:* 120/80 mmHg
- *Total Cholesterol:* lower than 200mg/dL
- *LDL:* lower than 100 mg/dL

b. Are Mr. Smith’s values for blood pressure within normal limits? If not, which values are too high or too low?

**Answer** - Both the systolic & diastolic pressures are too high

c. Is his value for cholesterol within normal limits? If not, is it too high or too low?

**Answer** - No, it is very high

d. What does LDL stand for?

**Answer** - Low Density Lipoprotein

e. Is his value for LDL within normal limits? If not, is it too high or too low?

**Answer** - No, it is high

7. Based on the data collected in the ER, list all the risk factors that Mr. Smith has that probably led to his transient episode.

**Answer** - High blood pressure, high cholesterol, high LDL, smoking, and genetics (father having an MI at age 54).
Case #4
Myasthenia Gravis
Presenter - Carol Veil

INSTRUCTIONS
1. To do this assignment, you should use your class notes, the handout on skeletal muscle physiology, your lab manual, and the textbook. You do not need to do any outside research, other than to look up one definition for question 4a.

2. Remember the AACC Academic Integrity Policy: This is to be your own work. Make sure that your answers (other than the definition in 4a and any clinical terminology) are written in your own words - NOT copied from the handout or from the textbook, and NOT identical to the answers of another student in the class. In addition, do NOT discuss this assignment with tutors in the Science Tutoring Center. If you have any questions about the assignment, you should speak with your professor.

CASE STUDY
Joe, a 57-year-old male, made an appointment to talk to his family physician about some unusual symptoms he had begun to experience. He thought his eyelids looked a little funny and he was having double vision. The doctor did an eye examination and asked Joe some questions. He made notes in Joe’s chart, including the words “ptosis” and “diplopia”. He could not determine the cause of the problem and referred Joe to a neurologist. However, since the symptoms seemed to come and go during the day, Joe thought he could just live with it and never made the appointment.

In a few months, Joe started to experience some additional symptoms. He had muscle weakness in flexing his right hip as he started to walk or sit. Joe finally made the appointment with the neurologist, complaining of this new symptom as well as the continuing eye problems. The neurologist ran a series of tests and concluded that Joe suffered from an autoimmune disease called myasthenia gravis. In this disease, protein molecules called antibodies are produced that attack the motor end plates of certain neuromuscular junctions. As a result, the motor end plates lose their normal folded shape. The antibodies can block, alter, or destroy receptors for acetylcholine.

QUESTIONS ABOUT CASE STUDY
1. a. Name three different muscles that contract to cause flexion at the hip joint.
   b. What specific type of muscle tissue makes up these muscles?
   c. What specific neurotransmitter is used to stimulate muscle contraction in these muscles? (Write out the complete name of the neurotransmitter.)

2. a. At a neuromuscular junction, is the motor end plate part of the nerve cell or part of the muscle cell?
   b. Why is it significant for proper muscle function that a normal motor end plate has a folded shape rather than being smooth?

3. At each level indicated below, explain what is actually happening in the body to account for the weak muscle contractions seen with myasthenia gravis.
   (Note: Do not just repeat your answer to 2b above. Give new information here.)
   a. What is happening at the neuromuscular junction?
   b. What is happening at the level of individual muscle cells?
   c. What is happening at the level of the whole muscle?
4. a. Look up the term “ptosis” and give a definition.
   b. Explain how ptosis would be caused by myasthenia gravis.

5. A number of different treatments have been used for myasthenia gravis. One treatment is the use of cholinesterase inhibitors.
   a. Describe the role of cholinesterase (a.k.a. acetylcholinesterase) in the healthy body.
   b. Describe the effect that a cholinesterase inhibitor would have on skeletal muscle function in a healthy individual.
   c. Explain how a cholinesterase inhibitor would work to increase the strength of muscle contraction in a patient with myasthenia gravis.
1. a. Name three different muscles that contract to cause flexion at the hip joint.
   
   **Possible answers:**
   - Rectus femoris
   - Tensor fasciae latae
   - Pectineus
   - Psoas major
   - Adductor magnus
   - Sartorius
   - Adductor longus
   - Iliacus

   b. What specific type of muscle tissue makes up these muscles?
   
   **Answer** - Skeletal muscle

   c. What specific neurotransmitter is used to stimulate muscle contraction in these muscles? (Write out the complete name of the neurotransmitter.)
   
   **Answer** - Acetylcholine

2. a. At a neuromuscular junction, is the motor end plate part of the nerve cell or part of the muscle cell?
   
   **Answer** - Muscle cell

   b. Why is it significant for proper muscle function that a normal motor end plate has a folded shape rather than being smooth?
   
   **Answer** - It creates increased surface area for acetylcholine receptors

3. At each level indicated below, explain what is actually happening in the body to account for the weak muscle contractions seen with myasthenia gravis.
   (NOTE: Do not just repeat your answer to 2b above. Give new information here.)

   a. What is happening at the neuromuscular junction?

   **Possible Answers:**
   - Too few receptors are present for acetylcholine
   - There is not enough binding of acetylcholine to receptors
   - The cell does not receive adequate stimulus.

   b. What is happening at the level of individual muscle cells?

   **Possible Answers:**
   - Fewer gated Na\(^+\) channels open and less Na\(^+\) enters the cell.
   - The cell membrane cannot depolarize.
   - Calcium ions will not be released from the terminal cisternae into the sarcoplasm.
   - The contraction phase will not be initiated.
c. What is happening at the level of the whole muscle?

**Possible Answers:**

- Fewer total muscle cells/fibers contract.
- OR
- Fewer motor units are involved in the contraction.

4. a. Look up the term “ptosis” and give a definition.

**Answer** - Abnormal lowering or drooping of an organ or body part, especially the eyelid.

b. Explain how ptosis would be caused by myasthenia gravis.

**Possible Answers:**

- The muscle of eyelid cannot contract normally.
- OR
- The muscle of eyelid is too weak to contract.
- OR
- The muscle of eyelid is not stimulated to contract.

5. A number of different treatments have been used for myasthenia gravis. One treatment is the use of cholinesterase inhibitors.

a. Describe the role of cholinesterase (a.k.a. acetylcholinesterase) in the healthy body.

**Answer** - It destroys (OR breaks down OR hydrolyzes) acetylcholine.

b. Describe the effect that a cholinesterase inhibitor would have on skeletal muscle function in a healthy individual.

**Possible Answers:**

- Muscles would stay contracted.
- OR
- Muscles would not relax.

**Also acceptable:**

- Muscles would eventually fatigue.

c. Explain how a cholinesterase inhibitor would work to increase the strength of muscle contraction in a patient with myasthenia gravis.

**Answer:**

**FIRST:** More acetylcholine would be available in synaptic cleft.
- OR
- Acetylcholine would be available longer in the synaptic cleft.

**AS A RESULT:** Acetylcholine is more likely to bind to receptors.
- OR
- More muscle cells would be stimulated to contract.
- OR
- Muscle cells would stay contracted longer.
Case #5  
Carry’s Kidney Crisis  
Presenter - Carol Veil  

INSTRUCTIONS

1. To do this assignment, you should use your class notes, relevant handouts from class, your lab manual, and the textbook. You do not need to do any outside research, other than to look up a clinical term for question 3a and a clinical blood value for question 4a.

2. Remember the AACC Academic Integrity Policy: This is to be your own work. Make sure that your answers (other than the requested information in 3a and 4a and any clinical terminology) are written in your own words - NOT copied from the handouts or the textbook, and NOT identical to the answers of another student in the class. In addition, do NOT discuss this assignment with tutors in the Science Tutoring Center. If you have any questions about the assignment, you should speak with your professor.

CASE STUDY

Carry Smith, a 13-year-old girl, was vacationing in Mexico with her family. Shortly after breakfast one morning, Carry suddenly started vomiting and also developed diarrhea. Her parents took her to a local doctor’s office. Her radial pulse was 80 and her blood pressure was 116/70 mmHg. The doctor told the parents to give Carry fluids such as Pedialyte when the vomiting subsided and to keep an eye on her. However, as the day went on Carry’s condition did not improve. She was unable to keep fluid down and she had hardly urinated all day. By evening, when Carry became dizzy and uncoordinated, her family took her to the emergency room at a nearby hospital. When the ER physician took her vital signs, her radial pulse was 110 and her blood pressure was 84/52 mmHg. Carry was admitted to the hospital. A blood test performed on her included the following result: serum BUN (blood urea nitrogen) 44 mg/dL. Carry’s mother overheard the nurse talking to the doctor about Carry’s condition. The nurse used some terms that Carry’s mother had never heard before, including “tachycardia” and “oliguria”.

QUESTIONS ABOUT CASE STUDY

1. Carry’s family noted that Carry “had hardly urinated all day”.
   a. Name one of the hormones that would have caused Carry’s reduced urine production?
   b. What gland releases that hormone?
   c. Give a good description of how that hormone actually works on the kidneys to reduce urinary output.

2. a. Was Carry’s blood pressure high, low or within the normal range when she visited the doctor’s office in the morning?
   b. Was Carry’s blood pressure high, low or within the normal range when she was admitted to the hospital in the evening?
   c. What is the most likely cause of the fall in Carry’s blood pressure? (Think back to what you have learned about blood pressure earlier this semester.)
   d. What effect will the fall in Carry’s blood pressure have on her GFR? Explain why.

3. a. Explain to Carry’s mother what the nurse meant by the term “oliguria”. (Look it up and cite your reference.)
   b. Explain how Carry’s altered GFR results in oliguria.
4. a. What is the normal range for serum BUN? (Look it up and cite your reference.)  
   b. Is Carry’s serum BUN value high, low or within the normal range?  
   c. Explain how the altered GFR accounts for Carry’s serum BUN value?

5. a. Explain to Carry’s mother what the nurse meant by the term “tachycardia”.  
   (Look it up in your lab manual if you do not remember from the ECG lab.)  
   b. In light of Carry’s other symptoms, what is the most logical explanation for her 
   tachycardia? (Recall what you have learned about cardiac physiology earlier this 
   semester.)
Case #5
ANSWER KEY
Carry’s Kidney Crisis
Presenter - Carol Veil

1. Carry’s family noted that Carry “had hardly urinated all day”.
   a. Name one of the hormones that would have caused Carry’s reduced urine production?
      **Possible Answers:**
      Antidiuretic hormone (a.k.a. Vasopressin) OR Aldosterone
      (NOTE: ADH is the best answer, but aldosterone is OK with good explanation)
   
   b. What gland releases that hormone?
      **Possible Answers:**
      For ADH - pituitary gland OR For aldosterone - adrenal gland
   
   c. Give a good description of how that hormone actually works on the kidneys to reduce urinary output.
      **Possible Answers:**
      ADH causes aquaporins to be inserted into the membranes of the epithelial cells lining the distal convoluted tubule and the collecting duct, so that they become more permeable to water. Water passes through the aquaporins, following the osmotic gradient set up by the countercurrent multiplier mechanism, and is reabsorbed from the filtrate rather than leaving the body as urine.
      OR
      Aldosterone causes increased reabsorption of sodium ions in the distal convoluted tubule and collecting duct. Water follows the sodium by osmosis and is also reabsorbed.

2. a. Was Carry’s blood pressure high, low or within the normal range when she visited the doctor’s office in the morning?
   **Answer** - Normal

   b. Was Carry’s blood pressure high, low or within the normal range when she was admitted to the hospital in the evening?
   **Answer** - Low

   c. What is the most likely cause of the fall in Carry’s blood pressure?
      (Think back to what you learned about blood pressure earlier this semester.)
      **Answer** - Low blood volume (due to dehydration)

   d. What effect will the fall in Carry’s blood pressure have on her GFR and why?
      **Answer** - Decrease GFR
      A drop in systemic blood pressure will decrease blood flow to the glomerulus, which will decrease glomerular blood hydrostatic pressure.
3. a. Explain to Carry’s mother what the nurse meant by the term “oliguria”.
   (Look it up and cite your reference.)
   **Answer** - Reduced urine production OR Urine production less than 20-100 ml/day
   Must give appropriate citation.

   b. Explain how Carry’s altered GFR results in oliguria.
   **Answer** - Since filtrate is the starting material that is modified (by reabsorption and secretion) to make urine, a decrease in filtrate production means less starting volume to make urine from.

4. a. What is the normal range for serum BUN? (Look it up and cite your reference.)
   **Answer** - Different sources give different ranges (e.g., 5-25 mg/dL or 7-20 mg/dL)
   Must give appropriate citation

   b. Is Carry’s serum BUN value high, low or within the normal range?
   **Answer** - High

   c. Explain how the altered GFR accounts for Carry’s serum BUN value?
   **Answer** - There is decreased filtration of urea due to decreased GFR. Since urea is not secreted in the kidney, filtration is the only way to remove it from the blood.

5. a. Explain to Carry's mother what the nurse meant by the term “tachycardia”.
   (Look it up in your lab manual if you forget from the ECG lab.)
   **Answer** - Faster than normal heart rate OR Heart rate faster than 100 bpm at rest

   b. In light of Carry's other symptoms, what is the most logical explanation for her tachycardia? (Recall what you learned about cardiac physiology earlier this semester.)
   **Answer** - Due to dehydration, Carry has low blood volume and low blood pressure. She is therefore in circulatory shock, with elevated heart rate being one of the typical signs of shock. The elevated heart rate is the body’s attempt to maintain Mean Arterial Pressure (MAP), when stroke volume and cardiac output are decreased. (Recall: CO = SV x HR)
INSTRUCTIONS FOR ALL 5 MINI-CASE STUDIES

For each patient described below, make a complete arterial blood gas (ABG) diagnosis, i.e., determine if the patient is experiencing respiratory acidosis, respiratory alkalosis, metabolic acidosis, metabolic alkalosis, or if the patient is “normal”, i.e., within the “normal” range for all 3 of the arterial blood gas variables: pH, P\textsubscript{co\textsubscript{2}}, and [HCO\textsubscript{3}⁻] (bicarbonate ion concentration). If the patient is in acidosis or alkalosis, then determine the degree of compensation as either fully compensated, partially compensated, or uncompensated. Lastly, please write grammatically correct sentences to completely answer the follow-up questions/explanations for each case.

For all of these cases use the following “normal” ranges for arterial blood: pH = 7.35-7.45, P\textsubscript{co\textsubscript{2}} = 35-45 mmHg, and [HCO\textsubscript{3}⁻] = 22-26 mEq/L.

*Although all 5 case studies described below attempt to be realistic and coherent, they are fictional and in real life may not require the drawing of arterial blood samples. However, the arterial blood gas values given for each patient are consistent with the modified Henderson-Hasselbalch equation for dissociation of H\textsubscript{2}CO\textsubscript{3} to H\textsuperscript{+} + HCO\textsubscript{3}⁻ in arterial blood.

*Note: Except where otherwise noted, arterial blood gases were measured with the patient breathing room air. If the patient was breathing oxygen-supplemented air, this is noted in the case description.

MINI-CASE STUDY #1

Astrid Metabolki, a 17 year old uncontrolled type I diabetic, was brought to the emergency room exhibiting the rapid, deep, gasping breathing known as Kussmaul respirations. One of the nurses commented that her breath smelled sweet, like acetone. (Acetone, acetoacetic acid and β-hydroxybutyric acid are the ketone bodies produced when the body’s cells rapidly break down fat to produce ATP because glucose entry into cells is severely limited by the lack of insulin.) The nurse went on to suspect that Astrid might be in ketoacidosis because of the high level of acidic ketone bodies in her blood. Her arterial blood gas results were sobering: pH = 7.02, P\textsubscript{co\textsubscript{2}} = 24 mmHg, and [HCO\textsubscript{3}⁻] = 6 mEq/L.

QUESTIONS ABOUT MINI-CASE STUDY #1

1. What is the ABG diagnosis?

2. Describe the individual cause and effect steps leading from uncontrolled type I diabetes to the correct ABG diagnosis. If you wish, instead of sentences you may use arrow diagrams where a down arrow (↓) means “decreased”; an up arrow (↑) means “increased”; and a horizontal arrow pointing toward the right (→) means “causes” or “results in”. A complete explanation will require multiple steps between uncontrolled type I diabetes and the ABG diagnosis.

3. Name the correct compensating factor and explain how one knows that it has to be the compensating factor.

4. For this case describe the logical steps in the process that one must go through to eliminate 2 of the 3 possible compensating statuses so as to arrive at the correct one.
MINI-CASE STUDY #2

Yesterday twenty-three year old Al K. Losis took an early morning flight from Miami, FL, to Denver, CO, and drove directly to Rocky Mountain National Park where he met some friends who had been camping and hiking there for the past week. Their campsite was about 10,000 feet above sea level. As he set up his tent and moved around the campsite, Al noticed that he was breathing heavily and had a mild headache, but that was to be expected at this altitude. Even so the mountain air felt cool and dry and wonderful. Al and his friends decided it would be nice to hike up to 11,500 feet to watch the sun go down. This hike did Al in. He was hyperventilating big time. He complained that his headache was much worse and that he felt dizzy, nauseated and too tired to continue. A friend agreed to hike with him back to camp. Since Al’s symptoms were worsening, his friend drove him to the Estes Park emergency room where Al was diagnosed with acute mountain sickness. Al’s arterial blood gas results were: pH = 7.52, Pco₂ = 28 mmHg, and [HCO₃⁻] = 22 mEq/L. Note: As far as I know in cases of acute mountain sickness, oxygen is usually given to the patient, and arterial blood is not routinely drawn.

QUESTIONS ABOUT MINI-CASE STUDY #2

1. What is the ABG diagnosis?
2. Describe the individual cause-and-effect steps leading from hyperventilation to the correct ABG diagnosis. If you wish, you may use arrow diagrams instead of sentences. A complete explanation will require several steps between hyperventilation and the ABG diagnosis.
3. Name the correct compensating factor and explain how one knows that it has to be the compensating factor.
4. For this case describe the logical steps in the process that one must go through to eliminate 2 of the 3 possible compensating statuses so as to arrive at the correct one.

MINI-CASE STUDY #3

Mr. Un Metabolki (Astrid’s uncle) was admitted to the emergency room dehydrated and complaining of persistent vomiting over the past 2 days. He stated that during this time he had only eaten a few crackers and sipped a sports drink – both of which usually came right back up. His respirations appeared to be slightly depressed. His arterial blood gas results were: pH = 7.52, Pco₂ = 48 mmHg, and [HCO₃⁻] = 38 mEq/L.

QUESTIONS ABOUT MINI-CASE STUDY #3

1. What is the ABG diagnosis?
2. Describe the individual cause-and-effect steps leading from prolonged persistent vomiting to the correct ABG diagnosis. For this question you may use arrow diagrams instead of sentences. Again a complete explanation will require several steps between prolonged vomiting and the ABG diagnosis.
MINI-CASE STUDY #4

Several years ago Normallo R. Spiratori survived a severe heart attack that damaged primarily his left ventricle. Consequently, his left ventricle cannot keep up with his right ventricle, i.e., his right ventricle pumps more blood into the pulmonary circuit than his left can pump out into the systemic circuit. This condition is known as left congestive heart failure. Normallo was put on supplemental oxygen therapy. During this year’s annual examination, his cardiologist detected fluid in Normallo’s lungs, a condition known as pulmonary edema. To assess the severity of this condition he sent Normallo to the respiratory physiologist for an arterial blood gas analysis. Normallo’s oxygen-supplemented arterial blood gas results were: pH = 7.39, Pco₂ = 68 mmHg, and [HCO₃⁻] = 40 mEq/L.

QUESTIONS ABOUT MINI-CASE STUDY #4

1. What is the ABG diagnosis?

2. Describe the individual cause-and-effect steps leading from pulmonary edema to the correct ABG diagnosis. If you wish, you may use arrow diagrams instead of sentences. A complete explanation will require multiple steps between pulmonary edema and the ABG diagnosis.

3. For this case name the compensating factor and then describe the logical steps in the process that one must go through to eliminate 2 of the 3 possible compensating statuses so as to arrive at the correct one.

4. Lastly, as a review of capillary filtration/reabsorption physiology, please describe the cause-and-effect steps leading from left ventricular failure to pulmonary edema. If you wish, you may substitute arrow diagrams for sentences. A complete explanation will require multiple steps between left ventricular failure and pulmonary edema.

MINI-CASE STUDY #5

Ms. G.I. Donno’s patient history was temporarily separated from her arterial blood gas results which were: pH = 7.43, Pco₂ = 39 mmHg, and [HCO₃⁻] = 25 mEq/L. As part of your clinical training the attending physician asked for your opinion as to what the ABG diagnosis was likely to be.

QUESTION ABOUT MINI-CASE STUDY #5

1. What is the ABG diagnosis?

Note: There are no further questions for mini-case study #5. You’ve worked hard enough. You’re done. Your best effort has been appreciated. Thank you.
MINI-CASE STUDY #1

1. What is the ABG diagnosis?

   Answer – metabolic acidosis, partially compensated

2. Describe the individual cause and effect steps leading from uncontrolled type I diabetes to the correct ABG diagnosis. If you wish, instead of sentences you may use arrow diagrams where a down arrow (↓) means “decreased”; an up arrow (↑) means “increased”; and a horizontal arrow pointing toward the right (→) means “causes” or “results in”. A complete explanation will require multiple steps between uncontrolled type I diabetes and the ABG diagnosis.

   Answer – Uncontrolled type I diabetes → ↓ insulin secretion → ↓ plasma insulin → ↓ glucose entry into cells → ↓ cellular ATP production from glucose → ↑ cellular ATP production from fat → ↑ cellular fat breakdown → ↑ release of acidic ketone bodies into plasma → ↑ plasma H+ → ↓ plasma HCO3− (as it absorbs H+ released from acidic ketone bodies) → metabolic acidosis.

3. Name the correct compensating factor and explain how one knows that it has to be the compensating factor.

   Answer – Compensating factor is CO2. Compensating factor has to be either CO2 or HCO3−, and it has to be the factor that is NOT “causing” or not associated with the cause of the acidosis/alkalosis. In this case decreased HCO3− is associated with the cause of the acidosis. Therefore, Pco2 or more accurately CO2 has to be the compensating factor.

4. For this case describe the logical steps in the process that one must go through to eliminate 2 of the 3 possible compensating statuses so as to arrive at the correct one.

   Note: Three compensating statuses exist:
   - Fully compensated - pH normal range & compensating factor out of normal range
   - Partially compensated - both pH & compensating factor out of normal range
   - Uncompensated - pH out of normal range & compensating factor in normal range

   Answer – Astrid’s pH of 7.02 is well out of the normal range of 7.35 – 7.45. So fully compensated is eliminated. Pco2, the compensating factor, is out of its normal range of 35–45 mmHg. So uncompensated is eliminated. That leaves partially compensated as the only choice left. But let’s double check. Is Astrid’s arterial blood pH out of its normal range? Answer: yes! Is the compensating factor also out of its normal range? Answer: yes! Therefore, partially compensated is correct.

   Note: Astrid’s arterial blood pH is very close to the lethal limit, which is generally a pH below 7.00. If her respiratory system had not been able to partially compensate with Kussmaul breathing, she might have died before getting to the emergency room.
MINI-CASE STUDY #2

1. What is the ABG diagnosis?
   **Answer** – respiratory alkalosis, uncompensated

2. Describe the individual cause-and-effect steps leading from hyperventilation to the correct ABG diagnosis. If you wish, you may use arrow diagrams instead of sentences. A complete explanation will require several steps between hyperventilation and the ABG diagnosis.
   **Answer** – Hyperventilation → ↓ CO₂ (Pco₂) in blood → ↓ H₂CO₃ in blood → ↓ H⁺ in blood → ↑ blood pH → respiratory alkalosis.

3. Name the correct compensating factor and explain how one knows that it has to be the compensating factor.
   **Answer** – HCO₃⁻ is the compensating factor because it is not causing the alkalosis. Low CO₂ is causing the alkalosis, and the causative factor cannot also be the compensating factor.

4. For this case describe the logical steps in the process that one must go through to eliminate 2 of the 3 possible compensating statuses so as to arrive at the correct one.
   Note: If necessary, please refer to the description of the three compensating statuses given in the note for answer #4 of mini-case study #1.
   **Answer** – Because HCO₃⁻ is within its normal range, both fully and partially compensated are eliminated. That leaves uncompensated. But let’s double check. Is Al’s arterial blood pH out of the normal range? **Answer**: yes! Is the compensating factor within its normal range? **Answer**: yes! Therefore, uncompensated has to be correct.

MINI-CASE STUDY #3

1. What is the ABG diagnosis?
   **Answer** – metabolic alkalosis, partially compensated

2. Describe the individual cause-and-effect steps leading from prolonged persistent vomiting to the correct ABG diagnosis. For this question you may use arrow diagrams instead of sentences. Again a complete explanation will require several steps between prolonged vomiting and the ABG diagnosis.
   **Answer** – Persistent vomiting → ↑ H⁺ expelled from stomach, body and blood → ↓ H⁺ in blood → ↑ HCO₃⁻ in blood (because it absorbs fewer H⁺) → metabolic alkalosis.
MINI-CASE STUDY #4

1. What is the ABG diagnosis?
   Answer – respiratory acidosis, fully compensated

2. Describe the individual cause-and-effect steps leading from pulmonary edema to the correct ABG diagnosis. If you wish, you may use arrow diagrams instead of sentences. A complete explanation will require multiple steps between pulmonary edema and the ABG diagnosis.
   Answer – Fluid filling alveoli during pulmonary edema → ↓ CO₂ diffusion from pulmonary blood into alveolar air → ↑ CO₂ (Pco₂) in blood → ↑ H₂CO₃ in blood → ↑ H⁺ in blood → ↓ blood pH → respiratory acidosis.

3. For this case name the compensating factor and then describe the logical steps in the process that one must go through to eliminate 2 of the 3 possible compensating statuses so as to arrive at the correct one.
   Note: Again, if necessary, refer to the description of the three compensating statuses given in the note for answer #4 of mini-case study #1.
   Answer – HCO₃⁻ is the compensating factor. Because pH is within the normal range, both partially compensated and uncompensated are eliminated. That leaves fully compensated. But let’s double check. Is Normallo’s arterial blood pH within the normal range? Answer: yes! Is the compensating factor out of its normal range? Answer: yes! Therefore, fully compensated has to be correct.

4. Lastly, as a review of capillary filtration/reabsorption physiology, please describe the cause-and-effect steps leading from left ventricular failure to pulmonary edema. If you wish, you may substitute arrow diagrams for sentences. A complete explanation will require multiple steps between left ventricular failure and pulmonary edema.
   Answer – Left ventricular failure (left ventricle cannot pump out all of the blood flowing into it from pulmonary circuit) → ↑ blood pooling in left atrium and pulmonary veins → ↑ pulmonary venous blood volume → ↑ pulmonary venous blood pressure → ↑ pulmonary capillary hydrostatic (blood) pressure → ↑ filtration across pulmonary capillaries → ↓ reabsorption capillaries → ↑ pulmonary interstitial fluid → pulmonary edema → ↑ alveolar fluid

MINI-CASE STUDY #5

1. What is the ABG diagnosis?
   Answer – “Normal” because all 3 arterial blood variables are within their “normal” ranges.