MULTIPLE CHOICE

1. Which type of cell adaptation occurs when normal columnar ciliated epithelial cells of the bronchial lining have been replaced by stratified squamous epithelial cells?
   a. Hyperplasia
   b. Metaplasia
   c. Dysplasia
   d. Anaplasia

   ANS: B
   Metaplasia is the reversible replacement of one mature cell by another, sometimes a less differentiated cell type. The best example of metaplasia is the replacement of normal columnar ciliated epithelial cells of the bronchial (airway) lining by stratified squamous epithelial cells. The other options do not accurately describe the event in the question.

   PTS: 1 REF: Page 54

2. The loss of the adenosine triphosphate (ATP) during ischemia causes cells to:
   a. Shrink because of the influx of calcium (Ca).
   b. Shrink because of the influx of potassium chloride (KCl).
   c. Swell because of the influx of sodium chloride (NaCl).
   d. Swell because of the influx of nitric oxide (NO).

   ANS: C
   A reduction in ATP levels causes the plasma membrane's sodium-potassium (Na^+–K^+) pump and sodium-calcium exchange to fail, which leads to an intracellular accumulation of sodium and calcium and diffusion of potassium out of the cell. (The Na^+–K^+ pump is discussed in Chapter 1.) Sodium and water can then freely enter the cell, and cellular swelling results. The other options do not accurately describe the result of ATP at the cellular level.

   PTS: 1 REF: Page 57

3. The mammary glands enlarge during pregnancy primarily as a consequence of hormonal:
   a. Atrophy
   b. Hyperplasia
   c. Anaplasia
   d. Dysplasia

   ANS: B
   Hormonal hyperplasia occurs chiefly in estrogen-dependent organs, such as the uterus and breast. The remaining options do not adequately describe the consequence of hormones on breast tissue during pregnancy.

   PTS: 1 REF: Page 53

4. Free radicals play a major role in the initiation and progression of which diseases?
   a. Cardiovascular diseases such as hypertension and ischemic heart disease
   b. Renal diseases such as acute tubular necrosis and glomerulonephritis
   c. Gastrointestinal diseases such as peptic ulcer disease and Crohn disease
   d. Muscular disease such as muscular dystrophy and fibromyalgia

   ANS: A

Emerging data indicate that reactive oxygen species play major roles in the initiation and progression of cardiovascular alterations associated with hyperlipidemia, diabetes mellitus, hypertension, ischemic heart disease, and chronic heart failure. No current research connects the disorders mentioned in the other options to the effects of free radicals.

PTS: 1 REF: Pages 59-60

5. Free radicals cause cell damage by:
   a. Stealing the cell's oxygen to stabilize the electron, thus causing hypoxia
   b. Stimulating the release of lysosomal enzymes that digest the cell membranes
   c. Transferring one of its charged, stabilized atoms to the cell membrane, which causes lysis
   d. Giving up an electron, which causes injury to the chemical bonds of the cell membrane

   ANS: D
   A free radical is an electrically uncharged atom or group of atoms having an unpaired electron. Having one unpaired electron makes the molecule unstable; thus to stabilize, the molecule gives up an electron to another molecule or steals one. Therefore it is capable of forming injurious chemical bonds with proteins, lipids, or carbohydrates—key molecules in membranes and nucleic acids. The remaining options do not accurately describe the role played by free radicals in cell damage.

   PTS: 1 REF: Page 60

6. What is a consequence of plasma membrane damage to the mitochondria?
   a. Enzymatic digestion halts DNA synthesis.
   b. Influx of calcium ions halts ATP production.
   c. Edema from an influx in sodium causes a reduction in ATP production.
   d. Potassium shifts out of the mitochondria, which destroys the infrastructure.

   ANS: B
   The most serious consequence of plasma membrane damage is, as in hypoxic injury, to the mitochondria. An influx of calcium ions from the extracellular compartment activates multiple enzyme systems, resulting in cytoskeleton disruption, membrane damage, activation of inflammation, and eventually DNA degradation. Calcium ion accumulation in the mitochondria causes the mitochondria to swell, which is an occurrence that is associated with irreversible cellular injury. The injured mitochondria can no longer generate ATP, but they do continue to accumulate calcium ions. The remaining options do not accurately describe the consequence of plasma membrane damage to the mitochondria.

   PTS: 1 REF: Page 63

7. What is a consequence of leakage of lysosomal enzymes during chemical injury?
   a. Enzymatic digestion of the nucleus and nucleolus occurs, halting DNA synthesis.
   b. Influx of potassium ions into the mitochondria occurs, halting the ATP production.
   c. Edema of the Golgi body occurs, preventing the transport of proteins out of the cell.
   d. Shift of calcium out of the plasma membrane occurs, destroying the cytoskeleton.

   ANS: A

Enzymatic digestion of cellular organelles, including the nucleus and nucleolus, ensues, halting the synthesis of DNA and ribonucleic acid (RNA). The remaining options do not accurately describe the consequence of lysosomal enzyme leakage during chemical injury.

PTS: 1 REF: Page 63

8. Lead causes damage within the cell by interfering with the action of:
   a. Sodium and chloride
   b. Potassium
   c. Calcium
   d. ATP

   ANS: C
   Lead affects many different biologic activities at the cellular and molecular levels, many of which may be related to its ability to interfere with the functions of calcium. Lead does not appear to cause damage by interfering with the action of the other options.

   PTS: 1 REF: Page 66

9. Which statement is a description of the characteristics of apoptosis?
   a. Programmed cell death of scattered, single cells
   b. Characterized by swelling of the nucleus and cytoplasm
   c. Unpredictable patterns of cell death
   d. Results in benign malignancies

   ANS: A
   Apoptosis is an active process of cellular self-destruction, also known as *programmed cell death, which* is implicated in normal and pathologic tissue changes. The remaining options do not accurately describe the characteristics of apoptosis.

   PTS: 1 REF: Page 91

10. Lead poisoning affects the nervous system by:
    a. Interfering with the function of neurotransmitters
    b. Inhibiting the production of myelin around nerves
    c. Increasing the resting membrane potential
    d. Altering the transport of potassium into the nerves

    ANS: A
    Alterations in calcium may play a crucial role in the interference with neurotransmitters, which may cause hyperactive behavior and the proliferation of capillaries of the white matter and intercerebral arteries. The remaining options do not accurately describe the effects of lead poisoning of the nervous system.

    PTS: 1 REF: Page 66

11. Carbon monoxide causes tissue damage by:
    a. Competing with carbon dioxide so that it cannot be excreted
    b. Binding to hemoglobin so that it cannot carry oxygen
    c. Destroying the chemical bonds of hemoglobin so it cannot carry oxygen
    d. Removing iron from hemoglobin so it cannot carry oxygen

    ANS: B

Because carbon monoxide's affinity for hemoglobin is 200 times greater than that of oxygen, it quickly binds with the hemoglobin, preventing oxygen molecules from doing so. The remaining options do not accurately describe the means by which carbon monoxide damages tissue.

PTS: 1 REF: Page 67

12. Acute alcoholism mainly affects which body system?
    a. Hepatic
    b. Gastrointestinal
    c. Renal
    d. Central nervous

ANS: D
Acute alcoholism mainly affects the central nervous system but may induce reversible hepatic and gastric changes. Other systems may evidentially be affected by chronic alcoholism.

PTS: 1 REF: Page 68

13. During cell injury caused by hypoxia, an increase in the osmotic pressure occurs within the cell because:
    a. Plasma proteins enter the cell.
    b. The adenosine triphosphatase (ATPase)–driven pump is stronger during hypoxia.
    c. Sodium chloride enters the cell.
    d. An influx of glucose occurs through the injured cell membranes.

ANS: C
In hypoxic injury, movement of fluid and ions into the cell is associated with acute failure of metabolism and a loss of ATP production. Normally, the pump that transports sodium ions out of the cell is maintained by the presence of ATP and ATPase, the active-transport enzyme. In metabolic failure caused by hypoxia, reduced ATP and ATPase levels permit sodium to accumulate in the cell, whereas potassium diffuses outward. The increase of intracellular sodium increases osmotic pressure, which draws more water into the cell. (Transport mechanisms are described in Chapter 1.) The remaining options do not accurately describe the cell injury that results in increased osmotic pressure caused by hypoxia.

PTS: 1 REF: Page 84

14. Which statement is *true* regarding the difference between subdural hematoma and epidural hematoma?
    a. No difference exists, and these terms may be correctly used interchangeably.
    b. A subdural hematoma occurs above the dura, whereas an epidural hematoma occurs under the dura.
    c. A subdural hematoma is often the result of shaken baby syndrome, whereas an epidural hematoma rapidly forms as a result of a skull fracture.
    d. A subdural hematoma usually forms from bleeding within the skull, such as an aneurysm eruption, whereas an epidural hematoma occurs from trauma outside the skull, such as a blunt force trauma.

ANS: C

A subdural hematoma is a collection of blood between the inner surface of the dura mater and the surface of the brain, resulting from the shearing of small veins that bridge the subdural space. Subdural hematomas can be the result of blows, falls, or sudden acceleration-deceleration of the head, which occurs in the *shaken baby syndrome*. An epidural hematoma is a collection of blood between the inner surface of the skull and the dura and is almost always associated with a skull fracture. The other options do not accurately describe the differences between the two hematomas.

PTS: 1 REF: Page 72 | Table 2-6

15. What physiologic change occurs during heat exhaustion?
    a. Hemoconcentration occurs because of the loss of salt and water.
    b. Cramping of voluntary muscles occurs as a result of salt loss.
    c. Thermoregulation fails because of high core temperatures.
    d. Subcutaneous layers are damaged because of high core temperatures.

    ANS: A
    Heat exhaustion occurs when sufficient salt and water loss results in hemoconcentration. The other options do not accurately describe the physiologic changes that occur during heat exhaustion.

    PTS: 1 REF: Page 77

16. In hypoxic injury, sodium enters the cell and causes swelling because:
    a. The cell membrane permeability increases for sodium during periods of hypoxia.
    b. ATP is insufficient to maintain the pump that keeps sodium out of the cell.
    c. The lactic acid produced by the hypoxia binds with sodium in the cell.
    d. Sodium cannot be transported to the cell membrane during hypoxia.

    ANS: B
    In hypoxic injury, movement of fluid and ions into the cell is associated with acute failure of metabolism and a loss of ATP production. Normally, the presence of ATP and ATPase, the active-transport enzyme, maintains the pump that transports sodium ions out of the cell. In metabolic failure caused by hypoxia, reduced ATP and ATPase levels permit sodium to accumulate in the cell, whereas potassium diffuses outward. The other options do not accurately describe the cause of the swelling caused by hypoxia.

    PTS: 1 REF: Page 84

17. What is the most common site of lipid accumulation?
    a. Coronary arteries
    b. Kidneys
    c. Liver
    d. Subcutaneous tissue

    ANS: C
    Although lipids sometimes accumulate in heart and kidney cells, the most common site of intracellular lipid accumulation, or fatty change**,** is liver cells. Subcutaneous tissue is not a common site of lipid accumulation.

    PTS: 1 REF: Pages 84-85

18. What mechanisms occur in the liver cells as a result of lipid accumulation?
    a. Accumulation of lipids that obstruct the common bile duct, preventing flow of bile

from the liver to the gallbladder
b. Increased synthesis of triglycerides from fatty acids and decreased synthesis of apoproteins
c. Increased binding of lipids with apoproteins to form lipoproteins
d. Increased conversion of fatty acids to phospholipids

ANS: B
Lipid accumulation in liver cells occurs after cellular injury sets the following mechanisms in motion: increased synthesis of triglycerides from fatty acids (increases in the enzyme, α-glycerophosphatase, which can accelerate triglyceride synthesis) and decreased synthesis of apoproteins (lipid-acceptor proteins). The other options do not accurately describe this event.

PTS: 1 REF: Pages 84-85

19. Hemoprotein accumulations are a result of the excessive storage of:
a. Iron, which is transferred from the cells to the bloodstream
b. Hemoglobin, which is transferred from the bloodstream to the cells
c. Albumin, which is transferred from the cells to the bloodstream
d. Amino acids, which are transferred from the cells to the bloodstream

ANS: A
Excessive storage of iron, which is transferred to the cells from the bloodstream, causes hemoprotein accumulations in cells. Hemoglobin, albumin, or amino acids will not cause hemoprotein accumulations.

PTS: 1 REF: Page 86

20. Hemosiderosis is a condition that results in the excess of what substance being stored as hemosiderin in cells of many organs and tissues?
a. Hemoglobin
b. Ferritin
c. Iron
d. Transferrin

ANS: C
Hemosiderosis is a condition that occurs only when excess iron is stored as hemosiderin in the cells of many organs and tissues.

PTS: 1 REF: Page 86

21. What is the cause of free calcium in the cytosol that damages cell membranes by uncontrolled enzyme activation?
a. Activation of endonuclease interferes with the binding of calcium to protein.
b. Activation of phospholipases, to which calcium normally binds, degrades the proteins.
c. An influx of phosphate ions competes with calcium for binding to proteins.
d. Depletion of ATP normally pumps calcium from the cell.

ANS: D
If abnormal direct damage occurs to membranes or ATP is depleted, then calcium increases in the cytosol. The other options do not accurately describe the cause of free calcium in cytosol to damage cell membranes.

PTS: 1 REF: Pages 37-38 | Page 87 | Figure 2-24

22. What two types of hearing loss are associated with noise?
    a. Acoustic trauma and noise-induced
    b. High frequency and low frequency
    c. High frequency and acoustic trauma
    d. Noise-induced and low frequency

    ANS: A
    Two types of hearing loss are associated with noise: (1) acoustic trauma or instantaneous damage caused by a single sharply rising wave of sound (e.g., gunfire), and (2) noise-induced hearing loss, the more common type, which is the result of prolonged exposure to intense sound (e.g., noise associated with the workplace and leisure-time activities). The remaining options are not related to noise but rather to the amplitude of the sound.

    PTS: 1 REF: Page 83

23. What type of necrosis results from ischemia of neurons and glial cells?
    a. Coagulative
    b. Liquefactive
    c. Caseous
    d. Gangrene

    ANS: B
    Liquefactive necrosis commonly results from ischemic injury to neurons and glial cells in the brain. The other types of necrosis are not related to ischemic injuries in the brain.

    PTS: 1 REF: Page 90

24. What type of necrosis is often associated with pulmonary tuberculosis?
    a. Bacteriologic
    b. Caseous
    c. Liquefactive
    d. Gangrenous

    ANS: B
    Caseous necrosis**,** which commonly results from tuberculous pulmonary infection, particularly *Mycobacterium tuberculosis,* is a combination of coagulative and liquefactive necrosis. The other types of necrosis are not observed in pulmonary tuberculosis.

    PTS: 1 REF: Page 90

25. What type of necrosis is associated with wet gangrene?
    a. Coagulative
    b. Liquefactive
    c. Caseous
    d. Gangrene

    ANS: B
    Wet gangrene develops only when neutrophils invade the site, causing liquefactive necrosis.

    PTS: 1 REF: Page 91

26. Current research supports the believe that, after heart muscle injury, the damage:
    a. Remains indefinitely because cardiac cells do not reproduce.
    b. Is repaired by newly matured cardiomyocytes.
    c. Gradually decreases in size as mitotic cell division occurs.
    d. Is replaced by hypertrophy of remaining cells.

ANS: B
The recent discovery that cardiac stem cells exist in the heart and differentiate into various cardiac cell lineages has profoundly changed the understanding of myocardial biology; it is now believed that bone marrow–derived cardiac stem cells or progenitor cells that have the ability to mature into cardiomyocytes may populate the heart after injury. The other options do not accurately describe the process that is believed to occur to address cardiac muscle damage.

PTS: 1 REF: Page 52 | What's New box

27. After ovulation, the uterine endometrial cells divide under the influence of estrogen. This process is an example of hormonal:
a. Hyperplasia
b. Dysplasia
c. Hypertrophy
d. Anaplasia

ANS: A
Hormonal hyperplasia chiefly occurs in estrogen-dependent organs, such as the uterus and breast. After ovulation, for example, estrogen stimulates the endometrium to grow and thicken for reception of the fertilized ovum. The other options do not accurately describe the process identified in the question.

PTS: 1 REF: Pages 51-53

28. The abnormal proliferation of cells in response to excessive hormonal stimulation is called:
a. Dysplasia
b. Pathologic dysplasia
c. Hyperplasia
d. Pathologic hyperplasia

ANS: D
Pathologic hyperplasia is the abnormal proliferation of normal cells and can occur as a response to excessive hormonal stimulation or the effects of growth factors on target cells (see Figure 2-4). The other options do not accurately identify the term for the results of excessive hormonal stimulation on cells.

PTS: 1 REF: Page 53

29. Removal of part of the liver leads to the remaining liver cells undergoing compensatory:
a. Atrophy
b. Metaplasia
c. Hyperplasia
d. Dysplasia

ANS: C
Compensatory hyperplasia is an adaptive mechanism that enables certain organs to regenerate. For example, the removal of part of the liver leads to hyperplasia of the remaining liver cells (hepatocytes) to compensate for the loss. The other options do not accurately identify the compensatory process described in the question.

PTS: 1 REF: Pages 52-53

30. What is the single most common cause of cellular injury?
a. Hypoxic injury
b. Chemical injury
c. Infectious injury
d. Genetic injury

ANS: A
Hypoxia, or lack of sufficient oxygen, is the single most common cause of cellular injury (see Figure 2-8). The other options are not a commonly observed as is the correct option.

PTS: 1 REF: Page 56

31. During cell injury caused by hypoxia, sodium and water move into the cell because:
a. Potassium moves out of the cell, and potassium and sodium are inversely related.
b. The pump that transports sodium out of the cell cannot function because of a decrease in ATP levels.
c. The osmotic pressure is increased, which pulls additional sodium across the cell membrane.
d. Oxygen is not available to bind with sodium to maintain it outside of the cell.

ANS: B
A reduction in ATP levels causes the plasma membrane's sodium-potassium (Na^+–K^+) pump and sodium-calcium exchange to fail, which leads to an intracellular accumulation of sodium and calcium and diffusion of potassium out of the cell. (The Na^+–K^+ pump is discussed in Chapter 1.)

PTS: 1 REF: Page 57

32. In decompression sickness, emboli are formed by bubbles of:
a. Oxygen
b. Nitrogen
c. Carbon monoxide
d. Hydrogen

ANS: B
If water pressure is too rapidly reduced, the gases dissolved in blood bubble out of the solution, forming emboli. Oxygen is quickly redissolved, but nitrogen bubbles may persist and obstruct blood vessels. Ischemia, resulting from gas emboli, causes cellular hypoxia, particularly in the muscles, joints, and tendons, which are especially susceptible to changes in oxygen supply. The remaining options are not involved in the formation of decompression sickness emboli.

PTS: 1 REF: Page 77

33. Which is an effect of ionizing radiation exposure?
a. Respiratory distress
b. Sun intolerance
c. DNA aberrations
d. Death

ANS: C
The effects of ionizing radiation may be acute or delayed. Acute effects of high doses, such as skin redness, skin damage, or chromosomal aberrations, occur within hours, days, or months. The delayed effects of low doses may not be evident for years. The other options are not commonly considered effects of radiation exposure.

PTS: 1 REF: Pages 78-79

34. What is an example of compensatory hyperplasia?
a. Hepatic cells increase cell division after part of the liver is excised.
b. Skeletal muscle cells atrophy as a result of paralysis.
c. The heart muscle enlarges as a result of hypertension.

d. The size of the uterus increases during pregnancy.

ANS: A
Compensatory hyperplasia is an adaptive mechanism that enables certain organs to regenerate. For example, the removal of part of the liver leads to hyperplasia of the remaining liver cells (hepatocytes) to compensate for the loss. The other options do not accurately describe the term compensatory hyperplasia.

PTS: 1 REF: Pages 52-53

35. It is *true* that nondividing cells are:
    a. Found in gastrointestinal lining
    b. Affected by hyperplasia
    c. Incapable of synthesizing DNA
    d. Affected by only hypertrophy

ANS: A
Gastrointestinal lining is made up of rapidly dividing cells. Hyperplasia and hypertrophy take place if the cells are capable of synthesizing DNA; however, only hypertrophy occurs in *nondividing cells*.

PTS: 1 REF: Pages 51-53

36. Dysplasia refers to a(n):
    a. Abnormal increase in the number of a specific cell type
    b. True adaptive process at the cellular level
    c. Modification in the shape of a specific cell type
    d. Lack of oxygen at the cellular level

ANS: C
Dysplasia refers only to abnormal changes in the size, shape, and organization of mature cells.

PTS: 1 REF: Pages 53-54

37. Current research has determined that chemical-induced cellular injury:
    a. Affects the permeability of the plasma membrane.
    b. Is often the result of the damage caused by reactive free radicals.
    c. Is rarely influenced by lipid peroxidation.
    d. Seldom involves the cell's organelles.

ANS: B
Not all the mechanisms causing chemical-induced membrane destruction are known; however, the only two general mechanisms currently accepted include: (1) direct toxicity by combining with a molecular component of the cell membrane or organelles, and (2) reactive free radicals and lipid peroxidation.

PTS: 1 REF: Pages 62-63