Orthopaedic Physical Therapy Secrets-
Module 1: Basic Science

Course Description:
This course is derived from the textbook by Jeffrey Placzek, MD, PT and David Boyce, PT, EdD, OCS. This textbook is part of the popular Secrets series. It presents physical therapy concepts and then introduces different healing modalities, specialties and orthopedic procedures typically prescribed for common injuries such as shoulders and extremities. Common diseases are included as well as more innovative diagnostic tools for physical therapists such as radiology. The text features concise information that includes tips, and memory “secrets”. Bulleted lists, algorithms and illustrations provide a quick review of the specific topics discussed. The information is interesting, entirely evidence-based, outcome based and up-to-date.

Module 1: Basic Science covers chapters 1 through 5.

Chapter 1: Muscle Structure and Function
Chapter 2: Biomechanics
Chapter 3: Soft Tissue Injury and Repair
Chapter 4: Bone Injury and Repair
Chapter 5: Exercise Physiology

Methods of Instruction:
Online course available via internet

Target Audience:
Physical Therapists, Physical Therapist Assistants, Occupational Therapists, Occupational Therapist Assistants and Athletic Trainers.

Educational Level:
Intermediate

Prerequisites:
None

Course Goals and Objectives:
At the completion of this course, participants should be able to:
1. Identify fusiform muscles
2. Recognize muscular changes dependent upon position
3. Differentiate between force and strength
4. Identify factors that affect stability
5. Recognize how stress and motion affect connective tissue after repair
6. Identify the proper use of NSAIDs after muscular strain
7. Differentiate between types of fractures
8. Identify the advantages of a closed reduction
9. Recognize oxygen deficit and what effect it has on the body
10. Identify the ACSM guidelines for specific exercise programs

Criteria for Obtaining Continuing Education Credits:
A score of 70% or greater on the post-test
DIRECTIONS FOR COMPLETING
THE COURSE:

1. This course is offered in conjunction with and with written permission of Elsevier Science Publishing.
2. Review the goals and objectives for the module.
3. Review the course material.
4. We strongly suggest printing out a hard copy of the test. Mark your answers as you go along and then transfer them to the actual test. A printable test can be found when clicking on “View/Take Test” in your “My Account”.
5. After reading the course material, when you are ready to take the test, go back to your “My Account” and click on “View/Take Test”.
6. A grade of 70% or higher on the test is considered passing. If you have not scored 70% or higher, this indicates that the material was not fully comprehended. To obtain your completion certificate, please re-read the material and take the test again.
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8. You have up to one year to complete this course from the date of purchase.
9. If we can help in any way, please don’t hesitate to contact us utilizing our live chat, via email at info@advantageceus.com or by phone at 405-974-0164.
1. What is the organizational hierarchy of skeletal muscle, and how is it achieved?

   The hierarchy:
   - Muscle fascicles
   - Muscle fibers or cells
   - Myofibrils (arranged in parallel)
   - Sarcomeres (arranged in series)

   Achieved as follows: The connective tissue that surrounds an entire muscle is called the epimysium; the membrane that binds fibers into fascicles is called the perimysium. Two separate membranes surround individual muscle fibers. The outer membrane of fibers has three names that are interchangeable: basement membrane, endomysium, or basal lamina. An additional thin elastic membrane is found just beneath the basement membrane and is termed the plasma membrane or sarcolemma.

2. Describe the characteristics of the sarcomere.

   - In the middle of the sarcomere, the areas that appear dark are termed anisotropic. This portion of the sarcomere is known as the A band.
   - Areas at the outer ends of each sarcomere appear light and are known as I bands because they are isotropic with respect to their birefringent properties.
   - The H band is in the central region of the A band, where there is no myosin and actin filament overlap.
   - The H band is bisected by the M line, which consists of proteins that keep the sarcomere in proper spatial orientation as it lengthens and shortens.
   - At the ends of each sarcomere are the Z discs. The sarcomere length is the distance from one Z disc to the next.
   - Optimal sarcomere length in mammalian muscle is 2.4 to 2.5 μm. The length of a sarcomere relative to its optimal length is of fundamental importance to the capacity for force generation.

3. What are the contractile and regulatory proteins?

   The most prominent protein making up the myofibrillar fraction of skeletal muscle is myosin, which constitutes approximately one half of the total myofibrillar protein. The other contractile protein, actin, comprises about one fifth of the myofibrillar protein fraction. Other myofibrillar proteins include the regulatory proteins tropomyosin and the troponin complex.

4. Name the structural proteins in skeletal muscle.

   - C protein—part of the thick filament; involved in holding the tails of myosin in their correct spatial arrangement
   - Titin—links the end of the thick filament to the Z disc
   - M line protein—also known as myomesin; functions to keep the thick and thin filaments in their correct spatial arrangement
   - α-Actinin—attaches actin filaments together at the Z disc
   - Desmin—links Z discs of adjacent myofibrils together
   - Spectrin and dystrophin—have structural and perhaps functional roles as sarcolemmal membrane proteins

5. What are the characteristics of myosin?

   Myosin is of key importance for the development of muscular force and velocity of contraction. A myosin molecule is a relatively large protein (approximately 470–500 kD) composed of two identical
myosin heavy chains (MHCs) (approximately 200 kD each) and four myosin light chains (MLCs) (16–20 kD each). In different muscle fibers, MHCs and MLCs are found in slightly different forms, called isoforms. The isoforms have small differences in some aspects of their structure that markedly influence the velocity of muscle contraction.

6. Describe the components of myosin.
Light-meromyosin (LMM) is the tail or backbone portion of the molecule, which intertwines with the tails of other myosin molecules to form a thick filament. Heavy-meromyosin (HMM) consists of two subfragments: S-1 and S-2. The S-2 portion of HMM projects out at an angle from LMM, and the
S-1 portion is the globular head that can bind to actin. S-1 and S-2 together are also termed a myosin cross-bridge. There are approximately 300 molecules of myosin in one myofilament or thick filament. Approximately one half of the MHCs combine with their HMM at one end of the thick filament; the other half have their HMM toward the opposite end of the thick filament—a tail-to-tail arrangement. When molecules combine, they are rotated 60 degrees relative to the adjacent molecules and are offset slightly in the longitudinal plane. As a consequence of these three-dimensional structural factors, myosin has a characteristic bottlebrush appearance, with HMM projecting out along most of the filament.

7. Explain the role of the enzyme myosin adenosinetriphosphatase (ATPase).
A specialized portion of the MHC provides the primary molecular basis for the speed of muscular contraction. The enzyme myosin ATPase is located on the S-1 subfragment. In different fibers, the myosin ATPase can be one of several isoforms that range along a functional continuum from slow to fast. The predominant isoforms of MHC are the slow type I and the fast types IIa, IIx, and IIb.

8. What are the characteristics of actin?
Actin consists of approximately 350 monomers and 50 molecules of each of the regulatory proteins—tropomyosin and troponin. The actin monomers are termed G-actin because they are globular and have molecular weights of approximately 42 kD. G-actin normally is polymerized to F-actin (ie, filamentous actin), which is arranged in a double helix. The polymerization from G-actin to F-actin involves the hydrolysis of ATP and the binding of adenosine diphosphate (ADP) to actin; 90% of ADP in skeletal muscle is bound to actin. The actin protein has a binding site that, when exposed, attaches to the myosin cross-bridge. The subsequent cycling of cross-bridges causes the development of muscular force. The actin filaments also join together to form the boundary between two sarcomeres in the area of the A band. α-Actinin is the protein that holds the actin filaments in the appropriate three-dimensional array.

9. Explain the sliding filament theory of muscle contraction.
A muscle shortens or lengthens because the myosin and actin myofilaments slide past each other without the filaments themselves changing length. The myosin cross-bridge projects out from the myosin tail and attaches to an actin monomer in the thin filament. The cross-bridges then move as ratchets, forcing the thin filaments toward the M line and causing a small amount of sarcomere shortening. The major structural rearrangement during contraction occurs in the region of the I band, which decreases markedly in size.

10. What are the functions of muscle?
- Movement
- Support and protection
- Heat generation
- Energy storage

11. List the functions of myonuclei and satellite cells, and identify the number of nuclei found in the skeletal muscle fiber.
- Growth and development of muscle
- Adaptive capacity of skeletal muscle to various forms of training or disuse
- Recovery from exercise-induced or traumatic injury. Approximately 200 to 3000 nuclei per millimeter of fiber length

12. List the energy production systems in skeletal muscle.
- Creatine kinase reaction
- Adenylate kinase reaction
- Glycolysis
- Tricarboxylic acid (TCA) cycle and oxidative phosphorylation

13. What are the major steps of fatty acid metabolism in muscle that result in the release of energy?
- Fatty acid activation and transport into the mitochondria
- Beta-oxidation
- Tricarboxylic acid (TCA) cycle
- Oxidative phosphorylation

14. What is the range of muscle fiber lengths?
Muscle fiber lengths range from a few millimeters in the intraocular muscles of the eye to >45 cm in the sartorius muscle.
15. Discuss the role of satellite cells in the formation of a new muscle fiber.
Satellite cells are normally dormant, but under conditions of stress or injury, they are essential for the regenerative growth of new fibers. Satellite cells have chemotactic properties, meaning they migrate from one location to another of higher need within a muscle fiber and then participate in the normal process of developing a new muscle fiber. The process of new fiber formation begins with satellite cells entering a mitotic phase to produce additional satellite cells. These cells then migrate across the plasma membrane into the cytosol, where they recognize each other, align, and fuse into a myotube, an immature form of a muscle fiber. The multinucleated myotube then differentiates into a mature fiber.

16. Identify and define or describe muscle growth factors.
Muscle growth factors are proteins that either promote muscle growth and repair or inhibit muscle protein breakdown. Examples include insulin-like growth factor, fibroblast growth factor, hepatocyte growth factor, and transforming growth factor.

17. What are the characteristics of myofibrils?
Individual myofibrils are approximately 1 μm in diameter and comprise approximately 80% of the volume of a whole muscle. The variable number of myofibrils is regulated during the hypertrophy of muscle fibers that is associated with growth; for example, the number of myofibrils ranges from 50 per muscle fiber in the muscles of a fetus to approximately 2000 per fiber in the muscles of an untrained adult. The hypertrophy and atrophy of adult skeletal muscle are associated with certain types of training and disuse and result from the regulation of the number of myofibrils per fiber. Training and disuse have negligible effects on the number of fibers in mammals.

18. Describe the characteristics of individual muscle fibers.
The cross-sectional area of an individual muscle fiber ranges from approximately 2000 to 7500 μm², with the mean and median in the 3000 to 4000 μm² range. Muscle fiber and muscle lengths vary considerably. For example, the length of the medial gastrocnemius muscle is approximately 250 mm, with fiber lengths of 35 mm, whereas the sartorius muscle is approximately 500 mm, with fiber lengths of 450 mm. The numbers of fibers range from several hundred in small muscles to >1 million in large muscles, such as those involved in hip flexion and knee extension.

19. What are the factors that upregulate protein synthesis in skeletal muscle?
- Amino acids
- Insulin
- Anabolic hormones such as growth hormone and testosterone
- Resistance training/muscle contraction

20. What is a strap or fusiform muscle? List examples of fusiform muscles.
Muscles that have a parallel-fiber arrangement are strap or fusiform muscles. In a parallel-fiber muscle, the muscle fibers are arranged essentially in parallel with the longitudinal axis of the muscle itself. Muscles with a parallel-fiber arrangement generally produce a greater range of motion (ROM) and greater joint velocity than muscles with the same cross-sectional area but with a different fiber arrangement.
- Sartorius
- Biceps brachii
- Sternohyoid

21. What are the factors that upregulate protein degradation in skeletal muscle?
- Inflammation
- Oxidative stress
- Catabolic hormones such as cortisol
- Energy stress such as starvation

22. Explain the role of pennation in force production.
When muscles are designed with angles of pennation, which is the most common architecture, more sarcomeres can be packed in parallel between the origin and insertion of the muscle. By packing more sarcomeres in a muscle, more force can be developed. As the angle of pennation increases, an increasing portion of the force developed by sarcomeres is displaced away from the tendons. As long as the angle of pennation is <30 degrees, the force lost as a result of the angle of pennation is more than compensated for by the increased packing of sarcomeres in parallel, producing an overall benefit to the force-producing capacity of muscle.
23. Describe the differences among unipennate, bipennate, and multipennate muscles.
   - In unipennate muscles, such as the flexor pollicis longus, the obliquely set fasciculi fan out on only one side of a central muscle tendon.
   - In a bipennate muscle, such as the gastrocnemius, the fibers are obliquely set on both sides of a central tendon.
   - In a multipennate muscle, such as the deltoid, the fibers converge on several tendons.

24. Define the force-velocity relationship.
   The muscle shortens at different velocities depending on the load placed on the muscle. As the load increases, the velocity decreases. When the load exceeds the maximal force capable of being developed by the muscle, a lengthening contraction ensues. The force developed during a shortening contraction is less than the isometric force. The force developed during a lengthening contraction exceeds the isometric force by 50% to 100% because of the increased extension of the attached cross-bridges.

25. Describe additional factors influencing muscle strength.
   The myosin structural state, the ratio of strong binding and weak binding cross-bridges to actin, muscle innervation, motor unit recruitment, and synchronization are all factors influencing muscle strength.

26. What is active insufficiency at the sarcomere level?
   Active insufficiency is the diminished ability of a muscle to produce or maintain active tension when elongated to the point at which there is no overlap between myosin and actin. It may also refer to the muscle being excessively shortened.

27. Define the all-or-none principle of muscle contraction.
   When a motor neuron is activated, all the muscle fibers innervated by that motor neuron contract maximally.

28. What is active insufficiency at the muscle level?
   This type of insufficiency is most commonly encountered when the full ROM is attempted simultaneously at all joints crossed by a two-joint or multi-joint muscle. During active shortening, a two-joint muscle becomes actively insufficient at a point before the end of a joint range, when full ROM at all joints occurs simultaneously. Active insufficiency also may occur in one-joint muscles, but this is not common.

29. Define excitation-contraction coupling.
   Excitation-contraction coupling is the physiologic mechanism whereby an electric discharge at the muscle initiates the chemical events that lead to contraction.

30. Summarize how excitation-contraction coupling occurs in skeletal muscle.
   1. Action potentials in the alpha motor neuron propagate down the axon to the axon terminals.
   2. Acetylcholine, the neurotransmitter at the neuromuscular junction, is released from the axon terminals.
   3. Acetylcholine diffuses across the neuromuscular junction and binds with acetylcholine receptors on the sarcolemma of the muscle.
   4. A muscle action potential is generated at the motor end plate.
   5. The muscle action potential travels along the sarcolemma and into the depths of the transverse tubules, which are continuous with the sarcolemma.
   6. The action potential (voltage change) is sensed by the dihydropyridine receptors in the transverse tubules.
   7. The dihydropyridine receptors communicate with the ryanodine receptors of the sarcoplasmic reticulum, a mechanism poorly understood.
   8. Calcium is released from the sarcoplasmic reticulum through the ryanodine receptors.
   9. Calcium binds to the regulatory protein, troponin C, and the interaction between actin and myosin can occur.
   10. Myosin cross-bridges, previously activated by the hydrolysis of ATP, attach to actin.
   11. The myosin cross-bridges move into a strong binding state, and force production occurs.
31. What are the characteristics of the different skeletal muscle fiber types?

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<thead>
<tr>
<th>PROPERTY</th>
<th>I (S) (SO)</th>
<th>IIA (FR) (FOG)</th>
<th>IIB (FF) (FG)</th>
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<tbody>
<tr>
<td>Contraction speed</td>
<td>Slow</td>
<td>Fast</td>
<td>Fast</td>
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<tr>
<td>Force production</td>
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<td>Intermediate</td>
<td>Large</td>
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<td>Fatigue resistance</td>
<td>High</td>
<td>High (intermediate)</td>
<td>Low</td>
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<tr>
<td>Fiber diameter</td>
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<td>Red color</td>
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<td>Myoglobin</td>
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<td>Low</td>
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<td>Poor</td>
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<td>Anaerobic</td>
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<td>Mitochondria</td>
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<td>Few</td>
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<td>Z line thickness</td>
<td>Intermediate (wide)</td>
<td>Wide (intermediate)</td>
<td>Narrow</td>
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<td>Glycogen content</td>
<td>Low</td>
<td>High (intermediate)</td>
<td>High</td>
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<td>Acid ATPase</td>
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<td>Moderate</td>
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<td>Medium-high</td>
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<tr>
<td>Glycolytic ability</td>
<td>Low</td>
<td>High</td>
<td>High</td>
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</table>

32. Define the type IIx myosin heavy chain in human fibers.
   The type IIx myosin heavy chain was first described in animals (rat, mouse). Type IIx myofibers have maximal shortening velocity and maximal isometric tension, which are intermediate between types IIa and IIb. The fiber type IIb in animals is not found in humans. Rather, the type IIb that is described in humans has a myosin composition very similar to that of type IIx.

33. Define the function of muscle spindles, and describe their appearance.
   Muscle spindles provide sensory information concerning changes in the length and tension of muscle fibers. Their main function is to respond to the stretch of a muscle and, through reflex action, to produce a stronger contraction to reduce the stretch.

   The spindle is fusiform in shape and is attached in parallel to the regular or extrafusal fibers of the muscle. Consequently, when the muscle is stretched, so is the spindle. There are more spindles in muscles that perform complex movements. There are two specialized cells within the spindle, called intrafusal fibers. There are two sensory afferents and one motor efferent innervating the intrafusal fibers. The gamma efferent innervates the contractile portion—the striated ends of the spindle. These fibers, activated by higher cortex levels, provide the mechanism for maintaining the spindle at peak operation at all muscle lengths.

34. What is the size principle of motor unit recruitment?
   When skeletal muscles contract voluntarily against a load, motor units are recruited from smallest to largest.

35. Discuss the function of the Golgi tendon organs.
   Connected in series to 25 extrafusal fibers, these sensory receptors also are located in the ligaments of joints and are primarily responsible for detecting differences in muscle tension. The Golgi tendon organs respond as a feedback monitor to discharge impulses under one of two conditions: (1) in response to tension created in the muscle when it shortens and (2) in response to tension when the muscle is
passively stretched. Excessive tension or stretch on a muscle activates the tendon’s Golgi receptors. This causes a reflex inhibition in the muscles they supply. The Golgi tendon organ functions as a protective sensory mechanism to detect and inhibit subsequently undue strain within the muscle-tendon structure.

36. Describe the adaptations in muscle structure that occur with progressive resistance exercises.
The major adaptation is an increase in the cross-sectional area of muscle, which is termed hypertrophy. The number of muscle fibers is minimally affected. Progressive resistive exercise involves 10 repetitions a day at 60% to 90% of maximal capacity; this results in an increase in strength by 0.5% to 1.0% per day over a period of several weeks. The fast-twitch type II fibers are more responsive to progressive resistance exercise than slow-twitch type I fibers. There are increases in the amounts of transverse tubular and sarcoplasmic reticulum membranes as well. Furthermore, neural adaptations result in an increased ability to recruit high-threshold motor units. The functional significance of this morphologic change is primarily a greater capacity for strength and power development.

37. List the effects of progressive resistance exercise.
- Increased mass and strength
- Increased cross-sectional area of muscle (increased number of myofibrils, leading to hypertrophy)
- Increased type I and type II fiber area
- Decreased mitochondrial density per fiber and oxidative capacity
- Increased intracellular lipids and capacity to use lipids as fuel
- Increased intracellular glycogen and glycolytic capacity
- Increased intramuscular high-energy phosphate pool and improved phosphagen metabolism

38. Describe the adaptations in muscle structure that occur with endurance exercises.
Endurance exercise has minimal impact on the cross-sectional area of muscle and muscle fibers. The smaller cross-sectional area allows better diffusion of metabolites and nutrients between the contractile filaments and the cytoplasm and between the cytoplasm and the interstitial fluid. There is a decrease in fatigability. The number of capillaries increases around each fiber, and there is an increase in mitochondria, especially in the type I fibers. The increased mitochondria can provide a good supply of ATP during exercise. The more extensive capillary bed improves the delivery of oxygen and circulating energy sources to the fibers, whereas the products of muscle activity are removed more efficiently. The functional significance of these changes is observed during sustained exercise, in which there is a delay in the onset of fatigue.

39. List the effects of endurance exercise.
- Improved ability to obtain ATP from oxidative phosphorylation
- Increased size and number of mitochondria
- Less lactic acid produced per given amount of exercise
- Increased myoglobin content
- Increased intramuscular triglyceride content
- Increased lipoprotein lipase (enzyme needed to use lipids from blood)
- Increased proportion of energy derived from fat; less from carbohydrates
- Lower rate of glycogen depletion during exercise
- Improved efficiency in extracting oxygen from blood
- Decreased number of type Iib fibers; increased number of type Ila fibers

40. What are the consequences of muscle disuse?
- The most striking consequence is atrophy—a reduction in muscle and muscle fiber cross-sectional area.
- The slow type I fibers show greater atrophy with disuse than the fast type II fibers.
- A few fibers undergo necrosis, and there is an increase in the endomysial and perimysial connective tissue.
- The muscles develop smaller twitch and tetanic tensions, beyond those expected on the basis of fiber atrophy.
- There is an increase in fatigability.
- There is a tendency for slow-twitch fibers to be transformed into fast-twitch fibers, with changes in the isoforms of the myofibrillar proteins.
In the sarcolemma, there is a spread of acetylcholine receptors beyond the neuromuscular junction, and the resting membrane potential is diminished.
The motor nerve terminals are abnormal in showing signs of degeneration in some places and evidence of sprouting in others.
There is a loss of motor drive, such that the motor units cannot be recruited fully.

41. What adaptations occur if muscles are immobilized in a shortened position?
- Decreased number of sarcomeres
- Increased amount of perimysium
- Thickening of endomysium
- Increased ratio of collagen concentration
- Increased ratio of connective tissue to muscle fiber tissue
- Atrophy
- Altered strength
- Increased stiffness to passive stretch
- Increased fatigability

42. Define the term sarcopenia.
Sarcopenia is the term used to describe age-related loss of skeletal muscle mass and strength.

43. What occurs as a result of lengthening the muscles?
Sarcomeres are added.

44. What are the changes in skeletal muscles that occur with aging?
- Decreased size of muscle cells
- Decreased number of muscle cells
- Preferential loss of type 2 muscle fibers

45. Define disease-associated muscle atrophy, such as cachexia.
Disease-associated muscle atrophy occurs as a result of accelerated proteolysis. This form of skeletal muscle atrophy is systemic and associated with metabolic and/or inflammatory factors.

46. Differentiate apoptosis from necrosis as applied to skeletal muscle.
Apoptosis, or programmed cell death, is a regulated physiologic process critical to cellular homeostasis, which can become dysregulated, leading to disease states including muscle disease or dysfunction. Apoptosis results in cell shrinkage, DNA fragmentation, membrane blebbing, and disassembly into apoptotic bodies (membrane-bound cell fragments). Necrosis is a pathologic process caused by the progressive degradative action of enzymes that is generally associated with severe cellular trauma in muscles, leading to cell death.

47. What are the hallmarks of muscles undergoing degeneration-regeneration?
- Central nuclei
- Increased variation of fiber sizes

BIBLIOGRAPHY
1. Does kinematic similarity ensure kinetic similarity?
No. Kinematics is the description of motion without reference to the cause of motion. Kinetics refers to the causes of motion (forces). Although two movements may appear similar (kinematics), the underlying forces causing those movements (kinetics) may be very different. This fact should be appreciated when using readily available motion analysis tools (such as recording movements on smartphones or tablets). For example, some patients who have undergone ACL reconstructive surgery and subsequent rehabilitation have gait patterns that look normal compared with healthy controls but are produced by altered joint kinetics (larger contributions from the hip and decreased contributions from the knee). These differences have been found to persist even a year after surgery.

2. Explain how impulse can be manipulated to prevent injury.
Impulse is the area under the force-time curve and accounts not only for the magnitude of the force but also for the duration over which the force is applied. Impulse determines the change in a body’s momentum, which is the product of mass and velocity. Applying a smaller force over a longer period of time will have the same impulse (and effect on a body’s momentum) as applying a larger force over a shorter period of time. Increasing the time of the impact, which can be accomplished by cushioned shoes and/or bending the knees when making contact with the ground, can attenuate the magnitude of an impact force and may decrease the risk of injury.

3. What are some considerations to keep in mind when using elastic resistance?
Elastic materials, such as bands and tubes, are often used as a form of resistance and follow Hooke’s law (the force is proportional to the stiffness and elongation). The stiffness is determined by the manufacturer (which uses different colors for different levels of stiffness) and will decrease with time as the material fatigues. It is also important to keep in mind that the elongation is related to the resting length of the band or tube and not just the elongation during the exercise. For example, consider two scenarios. In the first, an exercise starts with the band at its resting length and is elongated by a certain amount, “x.” In the second scenario, the exercise starts slightly elongated by a certain amount “a” but is still elongated by the amount “x.” In the first case, the amount of elongation is “x,” and in the second case the elongation is “a+x.” The band is providing greater resistance in the second scenario, even though the elongation during exercise is the same. This highlights the need to ensure patients are using the same starting length each time they perform an exercise.

4. Define commonly used biomechanical terms and equations.

<table>
<thead>
<tr>
<th>Common Biomechanical Terms and Equations</th>
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<tr>
<td><strong>TERM (LINEAR; ANGULAR)</strong></td>
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<tr>
<td>Displacement</td>
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<td>Velocity</td>
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</table>
5. **What is the relation between the linear motion at the joint surface and the angular motion of a bone around the joint axis?**

A theoretical construct, developed to describe this relation and advocated by Kaltenborn, is known as the convex-concave rule. In brief, if the convex surface of one bone is moving on the fixed concave surface of another bone, rotation and translation will occur in opposite directions. Additionally, if the concave surface of one bone is moving on the fixed convex surface of another bone, rotation and translation occur in the same direction. This rule should be appreciated when joint mobilizations are performed. It is proposed that in order to restore rotational motion at a joint, a linear mobilization is performed in relation to the treatment plane, which is parallel to the concave joint surface. Mobilization can be performed on either segment in accordance with the convex-concave rule.

6. **Has the convex-concave rule been experimentally verified?**

No, at least not for all joints. For example, it has been demonstrated that the glenohumeral joint contradicts the convex-concave rule during external rotation when the humerus is abducted to 90 degrees, and there is no clear consensus that the femur translates anteriorly when the knee is flexing in a weight-bearing position. However, these findings may not violate the convex-concave rule if the amount of translation in the direction of rolling is less than what the curvature of the convex segment would predict. The amount of rolling in one direction may be greater than the sliding in the opposite direction.
This “rule” pertains to the shape of the articular surfaces but does not account for the regional characteristics of the para-articular tissues, which may present as directional restrictions. Therefore the application of the convex-concave rule to treatment may need to be further informed by the direct method of assessing a restriction of joint gliding.

7. Is the axis of rotation for a joint fixed?
   In a nonpathologic joint, the AOR is generally fixed within the convex joint member and may stay in the same location (fixed AOR). A degenerated joint may lose its integrity, and the AOR may change its location throughout the range of motion. To reflect that change, the axis (or center) of rotation is called the instantaneous axis (or center) of rotation. Cartilage degeneration often accompanies the presence of a nonfixed axis of rotation.

8. What is the difference between an absolute and relative joint angle?
   An absolute angle is the angle that the distal point of a segment (eg, foot, shank, thigh) makes with respect to some reference line (such as the horizontal for sagittal plane movements). A relative angle is the joint angle made by two segments (eg, the knee angle is the angle between the shank and thigh). Relative angles can be stated as either internal (included) or external (anatomic) angles. An internal angle is the angle between the longitudinal axes of the two segments comprising a joint, while the external angle is the angular displacement from the anatomic position. For example, in the anatomic position, the internal knee angle is 180 degrees, while the external angle is 0 degrees. If this angle were decreased by 30 degrees, the internal angle would be 150 degrees and the external angle would be 30 degrees.

   ![A graphic depiction of the three types of angles: (A) absolute angle from the horizontal; (B) relative, internal angle; and (C) relative, external angle.](image)

9. Are the terms valgus and varus used unambiguously?
   Surprisingly, no. In accordance with their Latin derivations, in early writings varum referred to “knock-knees” and valgus referred to “bow-legged.” Taber’s Cyclopedic Medical Dictionary (21st ed.) still uses these definitions, which are the exact opposite of how they are generally used clinically. Because of the confusion this discrepancy can cause, it is prudent to understand how these terms are being used by various authors and not to assume that there are unambiguous and universally accepted definitions.
10. Provide examples of the concept of moment.

The moment of a force ("moment" for short), or torque, is the turning effect of a force. A force will have a tendency to rotate a body according to its magnitude, its direction, and the perpendicular distance between its line of application and the axis of rotation (this perpendicular distance is known as the moment arm). Knowing that the moment is the product of the force and the moment arm, the length of the moment arm can be manipulated to increase or decrease the force required to complete a task. For example, low back injury prevention strategies are based on the premise of decreasing the moment about the low back during lifting by keeping the load as close to the spine as possible, thus reducing the moment arm of the external resistance. Similarly, flexing the elbows during abduction will decrease the moment arm about the shoulder, thus making the movement easier to perform. On the other hand, during manual muscle testing, the therapist can increase the demand on a muscle by applying the resistance as far from the axis of rotation as possible.

11. What is the effect of a muscle’s force on a joint system?

Just as forces can be combined together to determine a resultant, they can also be broken into components. The components are useful in identifying the different effects of a force on a joint. For example, a muscle force can be divided into the component that is perpendicular to the bone (causing it to rotate and create a shear force across a joint) and the component that is parallel to the bone (usually increasing the compressive force across a joint). Therefore in addition to causing movement at a joint, all muscle forces will affect the amount of compression at a joint. During rehabilitation of certain joint pathologies, it may be necessary to identify which therapeutic exercises will increase the force of a muscle (to strengthen it) without applying excessive compressive forces across the joint. For example, performing unilateral (as opposed to bilateral) exercises for the lumbar extensors will decrease compressive forces on the spine while increasing the demand on those muscles.

12. Explain how torque-producing capabilities of a muscle vary over a joint’s range of motion.

Over the range of motion of a joint, the magnitudes of moment arms and forces may vary. The amount of force a muscle can produce is influenced by several properties, including the length-tension relationship, which states that when a muscle is too stretched or too shortened it cannot actively produce as high amounts of force as it can when the muscle is at its optimal length. For example, when using the deltoid to abduct the shoulder from 0 to 180 degrees, the moment arm of the deltoid increases and the force-producing capabilities also increase to a position of optimal length and then decrease. These variations result in an initial increase of torque production, until the optimal position is reached, and then a relatively constant amount of torque production from the deltoids ensues for the remainder range of motion. Based on this, it is important to keep the same manual muscle testing position, especially if the therapist wants to compare among patients or examine the effect of training over a period of time.

13. Can a muscle’s action at a joint change?

Yes. A muscle’s action at a joint is determined by the magnitude of the force and the direction of the force vector (a line roughly extending from the effective origin to the effective insertion). Tendon-transfer surgeries will often make use of this fact when a certain muscle group is paralyzed. Additionally, with flat feet (pes planus) the tibialis anterior’s role can change from that of a subtalar invertor to an evertor. Even if the absence of pathology, this can occur most notably at the hip. For example, the piriformis is an external rotator when the hip is in a neutral position but becomes an internal rotator when the hip is flexed beyond 90 degrees.

14. When a study refers to a net joint moment, what does that mean and what are the assumptions behind it?

One of the greatest limitations in biomechanics is that we cannot, with current technology, measure muscle forces in a noninvasive way. However, we can measure the acceleration of the limbs and forces between the body and the ground to calculate the net joint moment (NJM), which is the moment required to accelerate a limb in accordance with Newton’s second law. Despite the fact that muscles and other
soft tissue structures contribute to the NJM, and cocontractions of the antagonists can make the
actual moment much greater than the NJM, we usually equate high NJMs with high muscle forces
needed to produce that moment. So when a research study suggests that exercise A has a greater
extensor NJM at the knee than exercise B, it assumes that there is no cocontraction of the hamstrings
during both exercises, and exercise A has a higher demand on the quadriceps. Studies will normally
report internal moments (as described previously) or external moments (which are as a result of external
forces and inertia). Internal moments are equal in magnitude and opposite in direction to the external
moment.

15. What is joint instability, and how does it differ from hypermobility?
Joint stability is the ability of a joint to maintain a posture or trajectory similar to an undisturbed behavior in
the presence of a perturbation. Although joint instability would represent the lack of this ability, the
definition used by investigators and clinicians within studies is inconsistent, with three main definitions:
(1) excessive and occasionally uncontrolled range of motion resulting in frank joint dislocation; (2) small,
abnormal movement in an otherwise normal range of motion that may result in pain because of
“impingement” at the joint; and (3) a small amount of force necessary to move a joint through its range of
motion (or low stiffness). Joint hypermobility describes a laxity of the joint, where there is increased
flexibility and range of motion, and is often used interchangeably with instability by clinicians. However, a
hypermobile joint may still be stable because of muscular influence and motor coordination, whereas an
unstable joint may not be lax because of neurologic and muscular control.

16. How are force and strength related?
Force is a push or pull of one object on another. Force is a vector quantity, having both a magnitude and a
direction. Strength may be thought of as the ability to produce or absorb force. Measures of strength
typically determine the maximum force a muscle or muscle group can produce.

17. Does the amplitude of the electromyography (EMG) signal quantify a muscle’s
force-producing (absorbing) capability?
No. A muscle’s force-producing (absorbing) capability is primarily determined by the:
• Type of muscle action (concentric, eccentric, isometric)
• Length of muscle (force-velocity relation)
• Physiologic cross-sectional area of the muscle
• Number of motor units within a muscle that are activated (intramuscular coordination)
• Rate of motor unit activation (rate-coding)
• Intrinsic force-generating capability of the muscle (specific tension)
• Contractile history of the muscle (e.g., prestretch)

The EMG signal quantifies the number of motor units and their rate of activation within the
electrode field. In addition, because electrode placement can affect the number of motor units within
the field, it is important to compare relative values (usually normalized to a maximum voluntary
isometric contraction) rather than absolute values when comparing differences in EMG signals.

18. What are the benefits of having three different types of muscle actions?
Skeletal muscles are required to produce force, reduce (or absorb) force, or stabilize against a force. There
is a different type of muscle action to fulfill each of these roles. A concentric muscle action produces
force—the muscle moment is greater than the moment of an external force, and movement occurs in the
direction of the muscle moment. An eccentric muscle action reduces force—the muscle moment is less
than the moment of an external force, and movement occurs in the direction opposite of the muscle
moment. The eccentric muscle action reduces the external force, and consequently decreases the
acceleration caused by it. An isometric muscle action stabilizes against a force—the muscle moment is
equal and opposite to the moment created by an external force, and no movement occurs.

19. What information can be obtained from studying the force-velocity curve?
Examining this relationship reveals that greater force can be produced isometrically (when the velocity
is zero) than can be produced concentrically, and greater force can be produced eccentrically than
can be produced isometrically.
Peak eccentric force is estimated to be between 120% and 140% of peak concentric force. Additionally, there is a negative relationship between force and velocity in the concentric range and a positive one between force and velocity in the eccentric range.

20. **Is there a mechanical variable that can identify the types of muscle actions?**

Yes; mechanical power is the product of the net joint moment and the angular velocity. If the NJM and the angular velocity are in the same direction, the power is positive and a concentric muscle action is controlling the velocity. If the NJM and angular velocity are in opposite directions, the work is negative and an eccentric muscle action is controlling the velocity. If there is an NJM but no angular velocity, the power is zero because there is no angular velocity, but the presence of an NJM indicates an isometric muscle action is preventing a velocity.

21. **Why is eccentric strength important in the prevention of injury?**

Although energy can be absorbed by all of the tissues of the body (e.g., bone, ligament, muscle-tendon), the muscle-tendon complex has the greatest potential to safely absorb or distribute energy within the body. Eccentric muscle actions are the primary means by which energy is safely absorbed by the body. If the muscles are not strong enough, then other tissues must absorb this energy. Because the other tissues are not as capable of absorbing or distributing energy, energy levels can quickly exceed the tissues’ limits, resulting in injury.

22. **Explain the length-tension relationship of muscle.**

The amount of force or tension that a muscle can produce varies with the length of the muscle at the time of contraction. Maximum force is produced when the muscle is approximately at its resting length. When the fibers shorten beyond resting length, the force production decreases slowly at first and then rapidly. There is a progressive decline as the fibers are lengthened beyond resting length. This relationship can be used to help explain why surgically lengthened muscles are weak postoperatively (see figure). Although muscles typically do not operate over the entire length, this relationship helps explain the positions used for manual muscle tests, particularly for biarticular muscles. Biarticular muscles are
typically tested with one end of the muscle lengthened and the other end shortened, to place the muscle in the middle of its operating range. For example, when testing the hamstrings’ action at the hip, the hip is usually extending (muscle shortening) while the knee is extended (muscle in a lengthened position).

23. What is the stretch-shortening cycle?
The stretch-shortening cycle (SSC) involves 1) a well-timed preactivation of the muscle before an eccentric muscle action; 2) a short, rapid eccentric action; and 3) an immediate transition from an eccentric muscle action to a concentric muscle action. The subsequent concentric action is more forceful than it typically is because it was preceded by the rapid eccentric action. The SSC is involved in many movements, from gait to jumping and throwing. Plyometric exercises are usually used to improve utilization of the stretch-shortening cycle.

24. Is excessive force the cause of pain and injury?
Not directly. A better measure would be stress (force per unit area), which gives an indication of how that force is distributed. Although the term stress is used for reference to internal forces and pressure is used for external forces, clinically they can be used synonymously without much difficulty. Although a certain amount of stress is desirable, too much is believed to be the cause of injury and pain. Patellofemoral pain syndrome is believed to be the result of too much force (from the quadriceps) over too little area (patellofemoral contact area). The smaller contact area seems to have a stronger relationship to symptoms than does the increased amount of force. The insensate and poorly vascularized foot, in association with connective tissue changes, is vulnerable to increases in pressure and consequently the development of pressure sores. If the body weight transmitted to the foot can be dispersed over a larger surface area of the foot, the magnitude of pressure is decreased as is the chance for ulceration. The same factors apply to a person confined to prolonged bed rest; pressure sores may develop on areas where bony prominences contact the bed.

25. What is the tissue response to a force (stress), and how is it measured?
The tissue response to a force (or load) is deformation, which is a change in the size or shape of the tissue. Deformation is usually expressed as the quotient of the change in tissue length divided by the tissue’s original length, or strain. Laboratory experiments usually apply a given force (N) to a tissue of known cross-sectional area (mm$^2$) and specified length (mm), in which the resulting deformation (mm) is measured. Simple calculations will produce the applied stress and resulting strain. In vivo, force, either exerted by subject (active) or caused by an apparatus (passive), is measured using a dynamometer and the deformation (here displacement) is measured using an imaging technique (ie, ultrasound).

Not all tissues can be measured in this way; musculotendinous units are accessible to testing in vivo, but cartilage is not.
26. What information can be ascertained from studying force-deformation curves?
Plotting force on the vertical axis and the corresponding deformation on the horizontal axis produces a force-deformation curve, which graphically represents the relationship between the two (see figure).

Several important tissue qualities can be determined from this curve, including:
- **Ultimate strength**—the point on the curve where the tissue fails
- **Yield point**—the point at which a permanent deformation occurs
- **Elastic region**—the portion of the curve preceding the yield point
- **Plastic region**—the portion of the curve following the yield point
- **Stiffness**—the slope of the curve in the elastic range
- **Energy**—the area under the curve

When force is normalized to the area over which it is distributed and elongation is normalized to the resting length, we will have a stress on the vertical axis and strain on the horizontal axis. This curve provides insight into the material properties of the studied tissue, and its slope is the Young’s modulus.

![Force-deformation curve](image)

27. Do human tissues respond to all stresses in the same way?
No. Depending on the tissue and its role, tissues respond quite differently, and this difference in response is called anisotropism. For example, a tendon responds well to tension, but not as well to shear, and not at all to compression. Cartilage, on the other hand, responds well to compression. Human bone can handle compressive force best (such as pushing both ends of the bone toward each other), followed by tension (such as pulling both ends of the bone away from each other), and then shear force (such as pushing the top of the bone to the right and the bottom of the bone to the left). A bending force basically subjects one side of the bone to compression, while the other side experiences tension; therefore the side subjected to tension usually fails first (immature bone may fail in compression first). For torsional loading (such as twisting the top part of the bone, while holding the bottom of the bone in a fixed position), fracture patterns typically show that the bone fails as a result of shear forces and then tension.

28. When the force is applied to the tissue externally, does the tissue return to its original state after the force is removed?
It depends on the amount of force applied. At lower levels of force the tissue returns to its original form, and therefore this stage is called the elastic region. It is in the elastic region that the characteristics of the tissue are stable and therefore are used to describe the tissue’s stiffness. If the force continues to increase, it reaches a transitional point—the yield point. The yield point is where the material changes from the elastic range to the plastic range. Beyond this yield point, permanent deformation will occur even after the load is removed.
29. **Give an example of the clinical implications of the force-deformation curve.**
   The force-deformation curve can be appreciated clinically most easily during ligamentous testing. If the injurious force did not exceed the yield point, the ligament would return to its original length with no detectable changes in joint laxity. This injury would be classified as a first-degree sprain. If the injurious force exceeded the yield point but did not reach the ultimate strength of the ligament, the ligament would experience a permanent deformation that would be manifested as an increase in joint laxity. This injury would be classified as a second-degree sprain. If the injurious force exceeded the ultimate strength of the ligament, the ligament would catastrophically fail, and the subsequent force applied during ligamentous testing would be met with no resistance. This injury would be classified as a third-degree sprain.

30. **Discuss some factors that affect the biomechanical properties of tendons and ligaments.**

### The Most Commonly Cited Factors Affecting the Biomechanical Properties of Tendons and Ligaments

<table>
<thead>
<tr>
<th>FACTOR</th>
<th>PHYSIOLOGIC EFFECT ON COLLAGEN</th>
<th>MECHANICAL EFFECT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Physical activity</td>
<td>↑Glycosaminoglycan content</td>
<td>Strengthens</td>
</tr>
<tr>
<td></td>
<td>↑Cross-linking</td>
<td></td>
</tr>
<tr>
<td></td>
<td>↑Alignment of fibers</td>
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</tbody>
</table>
31. Is cartilage the same in all joints?
No. There are morphologic, biomechanical, metabolic, and histologic differences between types of cartilage in the joints of the lower extremities. Those differences, in part, are the reason why osteoarthritis is more prominent in the knee and hip joints than in the ankle joint.

32. Do all tissues adapt to change at the same rate?
No. An obvious example would be the difference in change in volume response to resistive exercise by a muscle and a tendon. A tendon adapts to change more slowly than muscle because it has fewer cells (in this case, tenocytes) that are capable of facilitating adaptation. Bone adapts more slowly than muscle. Evidence on the rate of adaptation of ligaments, cartilage, and intervertebral discs is scarce, but it is believed that they develop more slowly than muscle. It is important to realize, during rehabilitation, that a muscle will regain its strength before the other tissues of the musculoskeletal system, and therefore muscle strength alone is not a good indicator of the rehabilitation process.

33. What does it mean that a tendon is more compliant?
A more compliant tendon, typically accompanying degeneration, is a tendon where more displacement (m) occurs as a result of the same amount of force (N) produced by the muscle on the contralateral extremity. Compliance is the opposite of stiffness, which is the rate of change in force over displacement (N/m). Mechanical stiffness and patient-reported perceived sensation of stiffness are not related.

34. Are tissue responses to a submaximal stress time dependent?
Yes; tissue responses do change with time of application (or loading rate). Even if the amount of load is in the elastic range, but applied for a longer time, it will continue to cause a deformation. This type of deformation is reversible and is called creep. Creep is caused by the exudation of interstitial fluid. The fluid exits most rapidly at first and diminishes gradually over time. Human cartilage takes 4 to 16 hours to reach creep equilibrium, and this is why humans become slightly shorter as the day passes. Creep can also be associated with injury. Prolonged flexion of the lumbar spine results in a creep of the posterior ligaments, which decreases joint stiffness and may predispose the low back to injury. It is prudent to advise patients to allow this flexion-creep to reverse itself before performing activities that require lumbar stability.

35. What is hysteresis?
When viscoelastic tissue is loaded and then subsequently unloaded, the amount of stress is lower for a given amount of strain. This phenomenon is a consequence of the tissue’s viscosity and is called hysteresis. The area between the loading and unloading curves (shaded area, see figure) is a measure of hysteresis and represents the energy absorbed by the tissue, which is usually lost in the form of heat (although it could cause tissue damage).

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The Most Commonly Cited Factors Affecting the Biomechanical Properties of Tendons and Ligaments (Continued)

<table>
<thead>
<tr>
<th>FACTOR</th>
<th>PHYSIOLOGIC EFFECT ON COLLAGEN</th>
<th>MECHANICAL EFFECT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Disuse/immobilization</td>
<td>↑Turnover</td>
<td>Weaken</td>
</tr>
<tr>
<td></td>
<td>↑Reducible cross-linking</td>
<td></td>
</tr>
<tr>
<td></td>
<td>↑Nonuniform orientation</td>
<td></td>
</tr>
<tr>
<td></td>
<td>↑Glycosaminoglycan and water content</td>
<td></td>
</tr>
<tr>
<td>Aging</td>
<td>↓In number and quality of cross-links</td>
<td>Weaken</td>
</tr>
<tr>
<td></td>
<td>↓In fibril diameter</td>
<td></td>
</tr>
<tr>
<td>Corticosteroid use</td>
<td>Collagen synthesis</td>
<td>Weaken</td>
</tr>
<tr>
<td></td>
<td>Stiffness</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Ultimate stress</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Energy to failure</td>
<td></td>
</tr>
<tr>
<td>Pregnancy-induced hormones</td>
<td>Collagen degradation</td>
<td>Increases laxity</td>
</tr>
<tr>
<td>NSAIDs</td>
<td>Variable, depending on specific drug</td>
<td></td>
</tr>
</tbody>
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Repeated loadings, as well as acute and chronic stretching, increase a tendon’s compliance and decrease the amount of hysteresis. These changes increase the energy returned during the stretch-shortening cycle (improving performance) and can decrease the risk of injury. These changes show that stretching has beneficial effects other than just improving the range of motion of a joint.

36. What is the role of cartilage in joint lubrication, and how might pathology affect it?
There are three different types of joint lubrication processes: hydrodynamic (fluid film), elastohydrodynamic, and boundary. With fluid film lubrication, the fluid between two surfaces separates the contact surfaces and distributes the loading between them. Synovial fluid is attracted to the area of contact between the joint surfaces, resulting in the maintenance of a fluid film. Increased fluid pressure deforms the articular surface (cartilage), creating greater contact area and resulting in the elastohydrodynamic process. With boundary lubrication, the fluid is absorbed on the joint surface, preventing direct contact between two surfaces and decreasing friction.

Effective sealing of fluid within the joint is important for maintaining joint lubrication. For example, it has been found that a hip acetabular tear undermines the fluid sealing and joint lubrication processes, thereby leading to an elevation in the hip joint friction. This can ultimately cause cartilage degeneration and arthritis.

37. What is friction, and is it good or bad?
Friction is a force, parallel to the contact surface, that opposes motion between two objects. The interlocking of irregularities in the contact surfaces causes friction. The magnitude of the friction force will depend on the material characteristics of the two contacting surfaces and will be lower if there is relative motion between the two surfaces.

Friction may be good or bad, depending on the situation. A certain amount of friction between the ground and our shoes is necessary for efficient movement and to prevent slipping, but it also wears
38. **List biomechanical factors that affect a joint implant**
- Initial stability—based mainly on the surgery technique used and the implant design
- Late stability—determined by bone growth and remodeling of the bone around the implant (biologic fixation); if cement is used, late stability is determined by the bone-cement and cement-implant interfaces
- Stress shielding—affects the bone around the implant as the load typically goes through the stronger implant and not the bone surrounding it
- Wear of the implant—cobalt-chrome implants are typically used for the femoral head to decrease frictional wear on the acetabular component; ceramic acetabular cups and femoral heads are used because of their low coefficient of friction, but their implant strength has been questioned in some studies (they break and you never get all the fine ceramic debris out of the joint); highly cross-linked plastic has been used for acetabular components, but microscopic wear over time has proven to create an environment of aseptic loosening at the bone/cement/implant interfaces
- Wear debris—polyethylene wear can cause osteolysis and potential aseptic loosening
- Changing the anatomic alignments—by the manner in which the implant is installed or the correction of any preoperative deformity (hip dysplasia, knee varum or valgum)

39. **List factors that affect the stability of an external fixator.**
- Pin diameter—bending stiffness increasing by an order of the fourth power as the diameter increases
- Number of pins used
- Distance from the surface to the bone
- Stiffness of the frame
- Number of fixation planes
- Bone integrity into which the pins are placed (infection, avascular necrosis/poor vascularity)

40. **What happens to the strength of an intramedullary rod when its diameter is increased?**
Strength increases as the rod size increases by an order of the third power.

41. **What happens biomechanically with improper fixation size?**
In the case of total knee arthroplasty (TKA), if the plastic tibial insert is too big, then the space is “over-stuffed” and the knee will not have full flexion or extension capability. If the insert is too small, then the joint is unstable and the ligamentous and capsular structures are too lax; the muscular length tension characteristics are also compromised and both scenarios are often accompanied by pain. In the case of total hip arthroplasty (THA), if the acetabular component is too big, then the femoral implant “fulcrums” against the lip of the cup with flexion and can cause dislocation. If the stem is too long, then leg length discrepancy may occur. If the femoral head is too big, then range of motion is decreased in all planes. If any of the components is too small, then risk of joint dislocation increases. Furthermore, the muscular length tension characteristics are disrupted, and the supporting capsule and ligaments are lax. The patients often feel weak and walk with a limp.

42. **How do holes in the bone (ie, missing screw or following removal of plate) affect its strength?**
- Decreases the cross-sectional area of the bone; less bone at the hole and strength is decreased
- Decreases strength by causing a stress concentration point, determined by the geometry of the hole and bone
- A hole of 20% of the bone diameter decreases strength by 50%
43. How long does it take for strength to return to normal levels after the removal of a screw?
   It takes between 4 months and 1 year for strength to return to normal (provided normal bone physiology).

44. List the types of metals that are closest biomechanically to bone.

<table>
<thead>
<tr>
<th>With Regard to Modulus:</th>
<th>With Regard to Biocompatibility:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aluminum</td>
<td>Titanium (and titanium alloys)</td>
</tr>
<tr>
<td>Titanium (and titanium alloys)</td>
<td>Cobalt-chromium</td>
</tr>
<tr>
<td>Stainless steel</td>
<td>Stainless steel</td>
</tr>
<tr>
<td>Cobalt-chromium</td>
<td>Aluminum</td>
</tr>
</tbody>
</table>

45. How much strength does a well-placed lag screw add to fracture fixation?
   One should be able to assume that the strength of the fixation is determined by the pull-out strength of the lag screw, or approximately a 40% increase in strength over plating alone.

46. Do movement screens have diagnostic value?
   It is doubtful. Although a movement screen may alert you that something could be wrong with the way a person is moving, it cannot tell you the cause of a dysfunction. Additionally, some dysfunctions may not even be picked up by visual inspection as a result of compensations. The concept of a support moment highlights the need to be cautious when evaluating multijoint movement. Collapse of the lower extremity requires flexion at all three joints (ankle, knee, and hip). A sufficiently large NJM at the ankle or hip can prevent flexion at the knee, and vice versa. Summing all three NJMs is called a support moment, and the support moment has been shown to be less variable during repeated gait trials than any of the individual NJMs. The support moment demonstrates how a decrease in one NJM can be compensated for by an increase in another.

**BIBLIOGRAPHY**


1. What is the body’s initial response to soft tissue injury? How is it identified?
   The inflammatory response is characterized by a chemically mediated amplification cascade that represents the body’s initial reaction to injury, whether caused by trauma, surgery, or metabolic or infectious disease. The principal signs of the inflammatory response are erythema (rubor), swelling (tumor), elevated tissue temperature (calor), and pain (dolor). Local vasodilation, fluid leakage into the extracellular and extravascular spaces, and impaired lymphatic drainage are responsible for the erythema, swelling, and increased tissue temperature. The fourth cardinal sign of inflammation—pain—is the result of mechanical distention and pressure of the soft tissues and chemical irritation of pain-sensitive nerve receptors.

2. Describe the phases of soft tissue healing.
   The acute inflammatory phase begins immediately after injury and lasts 24 to 48 hours, although some aspects may continue for up to 3 weeks. The proliferative phase may begin early in the inflammatory phase but is thought to be most extensive approximately 21 days after injury. The matrix formation/remodeling phase begins 3 weeks after injury and may last for up to 2 years, although in many cases the majority of remodeling has occurred by 2 months. Because the time frames for these three phases overlap considerably, the accepted delineations should be used as general guidelines only.

3. Describe the basic vascular and cellular activities associated with the inflammatory reaction and the primary function of each activity.
   Blood vessels at the site of injury initially undergo vasoconstriction, which is mediated by norepinephrine and usually lasts from a few seconds to a few minutes. If serotonin is released by mast cells in the area of injury, a secondary prolonged vasoconstriction occurs to slow blood loss in the affected region. Additional cellular activities after soft tissue injury include margination of leukocytes, which adhere to the vessel wall, and chemotaxis (movement of white blood cells through the extravascular space toward the site of injury), which begins the process of phagocytosis and removes the cellular debris caused by the injury.

4. Identify the key chemical mediators of the inflammatory response.
   Both histamine and serotonin (5-HT) are released from granules of mast cells in the area of the injury. Histamine results in elevated vascular permeability, whereas serotonin is a potent vasconstrictor. Kinins, notably bradykinin, also cause a marked increase in vascular permeability, much as histamine does. It is now recognized that numerous cytokines and growth factors are involved in the cellular response to inflammation and injury. Proinflammatory prostaglandins are believed to sensitize pain receptors, attract leukocytes to the inflamed area, and increase vascular permeability by antagonizing vasconstriction. The primary mode of action of aspirin, nonsteroidal antiinflammatory drugs (NSAIDs), and steroids is to inhibit prostaglandin synthesis by deactivation of a key enzyme (cyclooxygenase).

5. Which cell type is especially prominent in the proliferative and matrix formation phases of connective tissue healing?
   The fibroblast is the most common connective tissue cell. It is responsible for synthesizing and secreting most of the fibers and ground substance of connective tissue. Soft tissue injury signals the fibroblast to multiply rapidly and mobilizes free connective tissue cells to the injured area. Tissue bleeding, in the case of trauma-induced inflammation, will result in deposition of fibrin and fibronectin in the tissues. These substances form a substratum that enhances the adhesion of various cells during later stages of repair.

6. Describe the elements that comprise the connective tissue matrix.
   The connective tissue matrix is comprised of fibrous elements (such as collagen, elastin, and reticulin) and ground substance, which consists principally of water, salts, and glycosaminoglycans (GAGs). The matrix provides the strength and support of the soft tissue and also serves as the means for diffusion of tissue fluid and nutrients between capillaries and cells.
7. What general factors affect connective tissue repair after tissue injury?
Healing after soft tissue injury is affected by the availability of a number of factors, including blood supply, proteins, minerals, and amino acids. Enzymes and hormones also play a role in tissue healing, as do mechanical stress and infection. Steroids suppress the mitotic activity of fibroblasts, which results in diminished deposition of collagen fibers and reduction in tensile strength. Antibiotic medicines inhibit protein synthesis and may adversely affect wound healing and scar formation. Disease processes such as diabetes mellitus significantly retard wound healing because small-vessel disease inhibits normal collagen synthesis.

8. What is the association of antibiotic medicines and acute tendinopathy and tendon ruptures?
Fluoroquinolones (FQs) (Cipro) are a popular class of antibiotics with broad-spectrum coverage for a number of gram-negative pathogens. Beginning first in 1983 there have been many anecdotal and case-controlled studies reporting the incidence of tendon ruptures associated with FQs, with 90% occurring in the Achilles tendon. Tendon rupture is nearly always preceded by spontaneous onset of pain within 2 to 3 cm of the insertion point, thought to be closely correlated with reduced vascularization at this site. Other tendons reported to be affected include the biceps brachii, supraspinatus, and extensor pollicis longus.

9. What risk factors are associated with FQ antibiotic-induced tendon rupture?
Identified risk factors associated with FQ (eg, Cipro)-induced tendon rupture or tendinopathy include age over 60, previous corticosteroid use, renal failure, diabetes mellitus, and a history of musculoskeletal disorders such as other tendon ruptures. The latency period between the administration of FQ antibiotic treatment and occurrence of tendinopathy has a median onset of 6 days with half of tendon ruptures occurring within 1 week of taking the medicine. FQs are known to cause direct toxicity to type 1 collagen synthesis and promote collagen degradation.

10. What influence does nutrition play in the soft tissue repair process?
Collagen biosynthesis is especially sensitive to the availability of proper nutrients. Lack of vitamins C and A impedes the process of collagen synthesis. Glucosamine, found within collagen type II, is the critical component in connective tissue repair and production. Glucosamine is the precursor for compounds important to connective tissue health, such as chondroitin sulfate and hyaluronic acid, and increases proteoglycan production. Whether dietary supplements such as glucosamine have a significant and lasting effect on joint disease has not been well established in controlled clinical trials, though mounting evidence suggests that such supplements are beneficial. Recent studies indicate that glucosamine may limit the advancement of joint space narrowing associated with osteoarthritis, resulting in improved functional scores. Minerals such as zinc contribute to the normal rate of cell proliferation and ultimate wound strength.

11. What role does aging play in altering the soft tissue injury healing process?
Age-related effects on wound healing include attenuated metabolic activity, decreased vascular supply, diminished cellular biosynthesis, delayed collagen remodeling, and decreased wound strength. Despite these differences, many of which have been confirmed in animal studies, clinical experience indicates that older patients often undergo surgical treatment with no adverse healing responses related to aging.

12. How does tendinitis differ from tendinosis?
Historically, painful tendon conditions were referred to as “tendinitis” and were assumed to be an inflammatory condition. Over time, however, further analysis revealed that the condition was not attended by inflammatory cells but was a degenerative change in the tendon—more accurately referred to as “overuse tendinosis.” Tendinosis (paratendinitis) is a rather rare condition that may occur occasionally in the Achilles tendon but almost always in conjunction with a primary tendinosis. The paratenon, a double-layered sheath of loose areolar tissue, is attached to the outer connective tissue surface of tendons that do not have a synovial lining. Paratendinitis refers to inflammation and thickening of the paratenon sheath. The pathology of tendinosis is characterized by a loss of collagen continuity and an increase in ground substance, vascularity, and cellularity. Research indicates that substance P, a neuropeptide known to contribute to tendon pain, is upregulated with tendon overuse. The cellularity increase associated with tendinosis results from fibroblasts and myofibroblasts, but inflammatory cells are absent. Because of this new understanding, it has been recommended that the term tendinopathy replace the term tendinitis for describing tendon pathology.
13. What is the appropriate treatment for tendinosis?
Treatment efforts to reduce pain and tenderness include ice application, oral NSAID administration, iontophoresis, rest, and cortisone injection. However, because tendinosis is by definition a chronic condition, treatment usually focuses on a controlled eccentric training program, often lengthy (10 to 12 weeks or more in some cases) in duration.

For Achilles tendinopathy, the most widely adopted approach is the Alfredson protocol of eccentric heel-drop exercises. This high-volume regimen has been shown to be particularly effective in athletic patients, though less so in nonathletic and older patients. Evidence clearly points toward exercise and mechanical loading as the best documented strategy for treating patients with tendinosis.

14. What tissue changes occur in response to a period of immobilization after soft tissue injury?
Immobility after soft tissue injury alters the rate of the biological process of remodeling. Changes that result in this alteration include an increased density of cells (usually fibroblasts), the presence of myofibroblasts, a reduction in hyaluronic acid and chondroitin sulfate levels in the periaricular connective tissue, and a 4% to 6% reduction in water content of the same tissues after only 9 weeks of immobilization. Further changes include a shift in the balance between collagen synthesis and degradation, which results in a reduction in total collagen.

15. What is the effect of immobilization on stiffness and strength of injured soft tissue?
Experimental evidence in rabbits indicates that 9 weeks of immobilization results in a 50% reduction in the normal breaking strength of the medial collateral ligament. At the same time a significant increase in the intermolecular cross-links of collagen leads to contracture formation. Therefore the remodeled connective tissue after immobilization is both thicker (tendency toward contracture) and weaker, possibly because of the random alignment of collagen fibers.

16. How do stress and motion affect connective tissue repair after injury?
Stress and motion have a profound effect on the quality of soft tissue repair after injury or surgery. Many studies have documented that scar tissue forms earlier in mobilized tendons, is well oriented, and is not attended by adhesions, in contrast to scar tissue that develops without physiologic stresses. Exposure of scar tissue to physiologic tensile forces during the healing process results in a more mature and stronger union of tendon and ligament. Healing of articular cartilage involves a greater amount of collagen and glycosaminoglycans, less cellularity, and fewer scar tissue adhesions when accompanied by modest joint movements. Some experimental evidence indicates that ultrasound application to tenotomized Achilles tendons improves tensile strength of the tissue if administered during postoperative days 2 to 4. This response appears to be time-dependent and may be related to limiting the inflammatory response and encouraging fibroplasia and fibrillogenesis. In a similar manner, high-voltage electrical stimulation appears to augment protein synthesis and the ultimate strength of the tendon if applied during the early stages of healing.

17. After ligament and tendon repair or reconstruction, when is the soft tissue the strongest and when is it the weakest?
Much of the information related to this question has been derived from studies using animal models (primates and others) and should be interpreted with caution. General data indicate that the strength of the patellar tendon autograft used in anterior cruciate ligament reconstruction cases is strongest on the day that it is surgically implanted. As the tissue heals in its new location, its strength diminishes to significantly <50% during the first 4 to 8 weeks postoperatively. In the ensuing 3 to 6 months, there is a slow transformation of collagen type and revascularization of the graft tissue. Stiffness and load to failure continue to increase for many months, and at 1 year the tissue is reported to have achieved 82% of its original strength. The clinical implications are fairly straightforward: protect the graft in the early stages of rehabilitation, encourage closed-chain axial loading activity to minimize shear forces (joint translation), and emphasize maximal motor unit activation throughout the rehabilitation process.

18. What is the response of articular cartilage to chondroplasty (microfracture technique, abrasion, and drilling) of the undersurface of the patella?
The microfracture technique is used to stimulate tissue repair of full-thickness articular cartilage defects. A drill is used to make multiple perforations in the subchondral bone in the area of the cartilage defect in an effort to produce a “super clot.” Over a period of 8 weeks or more the super clot heals with a hybrid mixture of fibrocartilage and type II (hyaline-like) collagen. This hybrid repair tissue may be functionally better than fibrocartilage alone; early animal and human studies suggest that it is durable enough to function like articular cartilage.
19. Describe the scientific evidence supporting articular cartilage repair.
Reproduced chondrocyte cells harvested from the patient are injected under a periosteal flap covering the articular defect. Two-year follow-up studies of patients with femoral condyle transplants indicate excellent results; most patients developed hyaline-like cartilage in the defect site. Patellar lesions have not done as well, possibly because of shear forces or noncorrection of underlying malalignment abnormalities. Research is encouraging for focal chondral defects but not for generalized osteoarthritis of the joint. In addition, there is evidence that articular cartilage exposed to electric and electromagnetic fields can lead to a sustained upregulation of growth factors, enhancing its viability. The degradative enzymes in the synovial fluid of osteoarthritic joints are not conducive to cell transfer with cartilage transplant experimental procedures.

20. What growth factors are involved with soft tissue healing?
- Chemotactic factors—prostaglandins, complement, platelet-derived growth factor (PDGF), and angiokines
- Competence factors—activate quiescent cells, PDGF, and prostaglandins
- Progression factors—stimulate cell growth, such as IL-1 and somatomedins
- Enhancing factors—fibronectin and osteonectin

21. What is the effect of NSAIDs on muscle recovery?
Short-term use (<1 week) of NSAIDs after muscular strain may improve recovery. However, long-term use (>1 month) may result in decreased recovery.

22. What factors affect allograft strength?
Freeze-drying reduces the immunogenic response but also decreases strength. Greater than 3-megarad irradiation will also decrease strength. Less radiation (2 megard) in combination with ethylene oxide will decrease graft strength. Allografts have a slower, less predictable recovery than autografts.

23. What growth factors may aid in soft tissue repair?
Platelet-rich plasma (PRP) has been shown to improve soft tissue healing in horses with improved collagen abundance and organization. Macrophage-secreted myogenic factors may someday play a role in inducing muscle repair. Specific chondrocyte growth factors and bone morphogenetic protein (BMP) have shown promise in improving cartilage repair.

BIBLIOGRAPHY
1. What are the components that make up bone?
   - Cells
   - Ground substance
   - Fibrous tissue network

   The cellular component consists of osteoblasts, which produce and initiate mineralization of new bone and cartilage, and osteoclasts, which are essential for the removal of the callus for lamellar bone to be laid down. A third cell type found in mature adult bone is the osteocyte.

   The ground substance component of bone contains mostly calcium phosphate, glycosaminoglycans, and hyaluronic acid. Calcium phosphate helps to add rigidity and hardness to the bone.

   The fibrous component consists of collagen fibers, which help resist tensile stress, and elastin fibers, which add a resilient aspect to the bone.

2. Describe the effects of aging on bone structure.

   The most commonly known age-related change is a calcium-related loss of mass and density. This loss ultimately causes the pathologic condition of osteoporosis. Osteoporosis is a major bone mineral disorder in older adults that decreases the bone mineral content; as a result, bone mass and strength decline with age. In geriatric patients, the hormonal system regulating calcium metabolism is less efficient and responds poorly to the challenge of a calcium-incorporating process, such as callous formation. Aging influences tissues (ie, the kidneys, gastrointestinal tract, and endocrine system) of the body that affect calcium metabolism and bone physiology. Thus the process of fracture healing in the geriatric patient is altered to some extent. Calcitonin, a hormone associated with decreasing serum calcium levels and possibly the remodeling of bone, has a decreased responsiveness to a calcium challenge with age. This decrease in calcitonin response may account, in part, for the slow bone healing in geriatric patients. Bones of older adults can withstand about half the strain of the bones of younger adults. Bones of older adults are less pliable and less able to store energy.

   Although there are physiologic changes that occur during the aging process that can affect bone health, the more sedentary lifestyle of many older individuals also may account for many of the age-associated changes in bone health.

3. How does Wolff’s law apply to bone healing?

   The ability of bone to adapt by changing size, shape, and structure depends on the mechanical stress on the bone. When optimal stress is placed on bone, there is greater bone deposition than bone reabsorption. This results in hypertrophy of periosteal bone and increased bone density. When bone is subjected to less than optimal stress, reabsorption of periosteal bone can occur, resulting in a decrease in strength and stiffness. Optimal stress within an appropriate range is essential for bone strength.

4. List the different types of bone fractures.

   - Compound (open)—occurs when sharp ends of the broken bone protrude through the victim’s skin or when some projectile penetrates the skin into the fracture site
   - Closed—skin remains intact
   - Perforating (eg, gunshot-bullet penetration)—may involve loss of bone from the effect of high-level energy at the fracture site
   - Depressed or fissured—occurs when a sharply localized blow depresses a segment of cortical bone below the level of the surrounding bone (eg, a skull fracture)
   - Greenstick—occurs on one side of the bone but does not tear the periosteum of the opposite side (seen in children)
   - Spiral—caused by opposite rotatory forces pulling on the bone (twisting)
   - Oblique—oriented at an angle of $\geq 30$ degrees to the axis of the bone
   - Transverse—oriented at a right angle to the axis of the bone
Avulsion—may be produced by a sudden muscle contraction, with the muscle pulling off the portion of the bone to which it is attached; also may result from traction on a ligamentous or capsular attachment
Comminuted—involves multiple fracture fragments
Stress—results from stresses repeated with excessive frequency to a bone
Pathologic—arises in abnormal or diseased bones; pathologic conditions that can lead to fractures include carcinomas, infection, and osteoporosis

5. What is a bone bruise and how does it relate to bone fractures?
Bone bruise (bone marrow contusion) is now considered to be one of four types of bone injuries that fall under the general heading of fracture—the others being stress fractures, osteochondral fractures and frank fractures (described in the list earlier in #3). The distribution of bone marrow edema has been likened to a footprint left behind by the musculoskeletal injury and is produced by compression and traction forces impacting adjacent bones. Further analysis indicates that there are actually three types of bone bruises:

a. Subperiosteal hematoma—a concentrated accumulation of blood underneath the periosteum after high-force trauma and most often seen in the lower extremities
b. Interosseous bone bruise—occurs most often with repetitive, high-compression forces that damage the blood supply in the bone marrow; usually occurs in the knee and ankle of professional athletes (eg, football and basketball players and elite runners)
c. Subchondral lesion—occurs beneath the cartilage layer of a joint and is usually caused by extreme compression or shear forces. Often there is microscopic separation of the cartilage and the underlying bone and is, again, most often seen in football and basketball players.

6. How are bone bruises identified?
Bone bruises are not visible with plain film radiographs, though this imaging modality may confirm that a frank fracture has not occurred. Most often bone bruises are visualized by means of T1- or T2-weighted fat-suppressed MRI. Patients with bone bruises tend to have a prolonged clinical recovery time with antalgic gait, slower recovery of motion, and persistent effusion compared with those with similar joint injuries who do not also have this complication.

7. Discuss the stages of bone healing.
The first stage is referred to as the inflammatory phase, or the granulation stage, fracture stage, or clot stage. During this phase surviving cells are sensitized to chemical messengers that are involved in the healing process. This initial aspect of the first stage is probably completed within 7 days. A second feature of the initial stage is the development of a clot around the fracture site (not seen in stress fracture healing). After the formation of the clot, granulation tissue forms in the space between the fracture fragments. This granulation tissue activates macrophages, whose function is to remove the clot. This second aspect of the initial stage lasts about 2 weeks.

The second stage is known as the reparative phase or callous stage and can be divided further into soft callous and hard callous stages. Osteoblasts and chondrocytes within the granulation tissue begin to synthesize cartilage and weave bone matrices (soft callus). Approximately 1 week later, the newly formed soft callus begins to mineralize. This mineralization concludes several weeks later with the formation of a fracture (hard) callus. The hard callus is detectable on radiographs because of the calcium it contains. The creation and mineralization of the callus can require 4 to 16 weeks to complete.

The third stage is called the remodeling or consolidation phase and involves several processes. First the callus is replaced by woven bone, which, in turn, is replaced with packets of new lamellar bone. The callus plugging the marrow cavity is removed, restoring the cavity. It has been estimated that the complete replacement of the callus with functionally competent lamellar bone can take 1 to 4 years.

8. Name some conditions that have a negative effect on the bone healing process.

<table>
<thead>
<tr>
<th>Technical Factors*</th>
<th>Biological Failures†</th>
<th>Miscellaneous Conditions</th>
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<tbody>
<tr>
<td>Infection</td>
<td>Vascular injury</td>
<td>Poor nutrition</td>
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<td>Poor reduction</td>
<td>Failure to make or mineralize callus (because of metabolic abnormalities)</td>
<td>Alcohol abuse</td>
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<tr>
<td>Distraction</td>
<td>Formation of scar and fat tissue instead of callus</td>
<td>Smoking</td>
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</tbody>
</table>
Repeated gross motion of fracture fragments
Inability to replace woven bone with lamellar bone (eg, children with osteogenesis imperfecta)
Loss of local blood supply because of injury and/or surgical procedure

*In these situations, the potential for normal healing is present, but problems during the treatment have prevented the healing process from proceeding, resulting in delayed union or nonunion.
†Biological failures refer to abnormalities in the biology of the healing process that delay or prevent union even with proper treatment.

9. Discuss the effect that smoking has on the bone healing process.
In studies in which animals were administered nicotine, a significant decrease in callous formation and an increase in the prevalence of nonunions were documented. Nicotine-exposed bones have been shown to be significantly weaker in a three-point bending test compared with controls. Smoking and nicotine have been shown to delay the revascularization and incorporation of bone grafts and to increase the pseudarthrosis rate in spinal fusion patients. One study found that patients with fractured tibias who smoked took 62% longer to heal than nonsmokers. Nicotine has been shown to have a direct inhibitory effect on bone cellular proliferation and function. These changes, taken together with the vascular effects, result in a decrease in the quantity and maturity of the fracture callus. It has been estimated that the risk of fractures is two to six times higher in patients who smoke because of reduced bone density in these patients. Somewhat unexpectedly, current and previous smokers have been shown to be significantly more likely to develop infections (including osteomyelitis) after fractures. Damaged soft tissue and impaired nerve function (neurogenic inflammation) can impede fracture healing by increasing the metabolic demand of the tissue repair system and limiting the benefit of supportive muscle function around the fracture site. Such failures usually require downward revision of the rehabilitation timetable and ultimate recovery potential for the patient.

10. What steps may be taken by a patient to promote accelerated fracture healing?
   a. Traumatic fractures of the long bones and patients with multiple fractures require as much as three times the caloric intake compared with normal nutritional demands.
   b. Specifically, increasing protein intake enhances growth factors, such as insulin-like growth factor-1, which exerts a beneficial effect on skeletal integrity and bone renewal in particular.
   c. Vitamins C, D, and K, along with mineral intake and antiinflammatory nutrients, should be increased. Antinflammatory nutrients (antioxidants) repair oxidative damage that would otherwise suppress fracture healing. Such antioxidants include vitamins E and C, lycopene, and alpha-lipoic acid.

11. Discuss the effect calcium nutrition has on bone healing.
Calcium plays an important role in helping attain peak bone mass during bone development and in preventing fractures later in life. The daily recommended allowance of calcium for nonpregnant, nonlactating women is 800 mg/day. This level increases to 1500 mg/day in postmenopausal, estrogen-depleted women. It is estimated that 75% of all women ingest less than the recommended daily allowance. Men tend to meet their calcium needs more successfully by consuming twice as much calcium at the same age. Multiple factors can affect the bioactivity of calcium. High-fat or high-fiber diets can interfere with or decrease the activity of calcium. Large doses of zinc supplementation or megadoses of vitamin A can lower calcium bioactivity. High-protein diets can decrease calcium reserves by increasing urinary excretion of calcium.

12. What other factors affect calcium absorption?
Alcohol consumption can decrease the absorption of calcium by a direct cytotoxic effect on the intestinal mucosa. Various medications, such as glucocorticoids, heparin, and anticonvulsants, can affect calcium activity. Vitamin D increases serum calcium levels by enhancing intestinal absorption of calcium and enhancing parathyroid hormone–stimulating reabsorption of bone. A low level of vitamin D impairs the ability of the body to adapt to low levels of calcium intake and may contribute to the pathogenesis of osteoporosis. Intake of vitamin D alone has never been shown to improve fracture healing.
   - Closed reduction—use of casting or traction
   - Open reduction—surgical intervention using plates, screws, or other internal fixation devices
   - Rigid external fixation—combination of closed and open reduction using percutaneous pins and external stabilizing bars

14. What are the advantages of closed reduction?
   Avoidance of surgery, reduction of the fracture, and usually (except in the case of traction) a shorter hospital stay are all advantages of closed reduction. Usually the patient can safely begin gentle range of motion exercises several weeks before the fractured limb is strong enough to return to normal weight-bearing function or to withstand resistance at the fracture site. In later stages of fracture healing, splints can be worn to protect the fractured limb, which is to be removed at intervals to permit joint mobilization or bathing.

15. List advantages and disadvantages of open reduction.
   **Advantages:**
   - Precise bone reduction
   - Early mobilization of joints
   - Immediate stability, allowing earlier return to full function
   **Disadvantages:**
   - Increased possibility of infection
   - Increased hospital stay
   - Metal devices may require subsequent removal

16. How does rigid fixation affect bone healing?
   When rigid fixation is used, there is no stimulus for the production of the external callus from the periosteum or the internal callus from the endosteum (secondary bone healing). Instead the fracture healing occurs directly between the cortex of one fracture fragment and the cortex of the other fracture fragment (primary bone healing). Primary bone healing involves a direct repair of the bone lesion by new bridging osteons that become oriented through haversian remodeling to the long axis of the bone.

17. What effects can internal fixation have on bone healing?
   - Improper placement or tightening of plates, screws, nuts, or bolts in bone surgery may cause bone reabsorption because of local stress concentration or decreased vascular perfusion.
   - Plates that are too rigid may cause bone atrophy secondary to preventing the bone from perceiving intermittent compressive stress.
   - If the hardware needs to be removed, a secondary inflammatory response occurs that leads to weakening of the bone; the bone needs to be protected until it regains strength.
   - If the plates are left in place, problems with stress along the plate-bone interface can occur.

18. List some advantages of weight-bearing activities after sustaining a fracture.
   - Enhanced rehabilitation (eg, improved range of motion)
   - Shorter hospital stay
   - Less overall postfracture morbidity

   Fat-pad signs constitute radiologic evidence of an effusion in the elbow joint and appear as areas of translucency on the lateral radiograph of the elbow flexed to a right angle. The fat-pad sign has an overall high negative predictive value (87%). The absence of the fat-pad sign can exclude a fracture and is a reliable indicator of the absence of a fracture. The presence of a fat-pad sign should only raise the suspicion of a fracture being present, however, because there may be a positive fat-pad sign with no fracture.

20. What is the most commonly overlooked fracture in adults at the time of injury?
   Carpal scaphoid fractures are easily overlooked. Because fractures of the scaphoid may result in loss of blood supply to the bone and consequent avascular necrosis, most physicians elect to treat wrist injuries as a fracture (immobilization) until properly interpreted radiographs indicate otherwise.

21. Discuss the role of ultrasound in the treatment of acute fractures.
   Low-intensity pulsed ultrasound (LIPUS) stimulation can accelerate the normal repair process in a fresh fracture and may help stimulate the healing process of nonunions. In animal models low-intensity
pulsed ultrasound at 0.1 to 0.5 W/cm² accelerated fracture healing. Pulsed ultrasound at higher doses (1.0–2.0 W/cm²) significantly inhibited the synthesis of collagen and noncollagenous protein, however. In clinical double-blind studies, ultrasound has been shown to decrease significantly the time for overall healing of grade I open tibial fractures and distal radial fractures. Ultrasound has been shown to reduce significantly the prevalence of delayed union in nonsmokers and smokers. In animal studies ultrasound increased bone mineral content and density, increased peak torque, and accelerated the overall endochondral ossification process. Ultrasound stimulation may increase the mechanical properties of the healing fracture callus by stimulating earlier synthesis of extracellular matrix proteins in cartilage. Recent systematic reviews of the literature regarding LIPUS clinical studies have questioned the quality of the supporting research to date. However, the current evidence indicates that LIPUS may be useful for comminuted and/or open fractures that involve patients with associated risk comorbidities such as older age, smoking history, those with diabetes, and malnourished individuals. Simple fractures in otherwise healthy people should not be the target of LIPUS therapy according to the most recent evidence.

22. **What effect does bioelectric stimulation have on fracture healing?**
Implantable electric stimulation and pulsed electromagnetic field (surface application) have been used for healing nonunion tibial fractures with some success. Electric stimulation generally is thought to convert fibrous connective tissue to bone, possibly by simulating mechanical stress in the bone. The best results with implantable electrodes in animal studies have been associated with the cathode located in the fracture gap and the anode in adjacent bone or in the soft tissue. Ionic migration in response to external direct current is believed to be one probable explanation for the apparent efficacy of electric stimulation on bone healing.

23. **What is the effect of NSAIDs on bone healing?**
Although there is still no well-defined answer, prostaglandins are known to participate in the inflammatory response and to stimulate osteoclasts as well as increase osteoblastic activity and subsequent new bone formation. Long-term excessive use of these medications may reduce normal bone healing.

24. **What are stress fractures, and how do they occur?**
Fatigue or stress fractures occur in otherwise healthy individuals usually in response to a sudden increase in physical activity of several weeks’ duration. First described in military training as “march fractures,” they are now fairly common in young individuals engaged in athletic activities and almost always represent a form of training error. In weight-bearing bones the overactivity causes microscopic fractures (debonding of osteons) that do not totally heal from day to day, eventually resulting in macroscopic bone failure and severe pain during ambulation or running. Though more common in the lower extremities, they can also occur in the medial epicondyle of the elbow with excessive throwing. Standard treatment involves early identification and rest of the involved extremity with avoidance of high-impact activities until healing has occurred. Signs of healing include resolution of bone tenderness with palpation and radiographic indication of healing—bone sclerosis.

25. **What is the best imaging method for detecting stress fractures?**
In spite of severe pain experienced by the patient, initial plain film radiographs of individuals suspected of a stress fracture are usually normal (up to 3 to 4 weeks after the initial onset of symptoms). Consequently, MRI and technetium bone scans are considered the best imaging studies for identifying stress fractures. Bone scans, in particular, may show signs of bone uptake as early as 72 hours after the onset of symptoms. However, radionuclide (bone) scans have a disadvantage, compared with MRI, of exposing the patient to ionizing radiation. The American College of Radiology also recommends computerized tomography without contrast for early detection of a stress fracture, if MRI is contraindicated.

26. **What is bone transplantation (replacement), and why is it used?**
Bone transplantation (replacement) is an aggressive surgical technique whereby an entire diseased bone is excised and a cadaveric allograft replacement is transplanted in its place. This is usually necessitated by malignant bone tumors—primary or metastatic—and most of the descriptions in the current literature are of cases of femur transplantation. The alternative is typically an above-knee amputation or a hip disarticulation. Allograft replacement of the femur is prone to a number of complications, such as refracture, infection, nonunion, and resorption of the graft.

27. **What treatments are available for nonunions?**
- Autogenous bone grafting and appropriate stable fixation
- Vascularized bone grafting
28. How do Salter-Harris fractures influence the pediatric population?
The growth plate appears on a radiograph as a lucent line near the joint, and a fracture through that line can be missed easily unless there is some disturbance in the alignment of the bone. When there is an injury to the growth plate, growth disturbances may occur in that bone. The younger the patient, the greater the growth potential remaining; however, there is also the danger of significant growth disturbance.

29. What are the roles of various growth factors on bone healing?
- **BMP**—Bone morphogenic protein induces metaplasia of undifferentiated perivascular mesenchymal cells into osteoblasts.
- **PDGF**—Platelet-derived growth factor is chemotactic for inflammatory cells at the fracture site.
- **TGF-β**—Transforming growth factor-β stimulates the production of type II collagen and proteoglycans at the fracture callus.
- **IGF-II**—Insulin-like growth factor II stimulates type I collagen production and cellular proliferation.

**BIBLIOGRAPHY**


1. What factor is considered to be the best indicator of an individual’s level of aerobic capacity?
   Maximum oxygen uptake (VO$_{2\text{max}}$) is the best indicator of aerobic capacity.

2. How is VO$_{2\text{max}}$ determined?
   VO$_{2\text{max}}$ is the product of cardiac output (heart rate $\times$ stroke volume) and arteriovenous oxygen difference ($A - V\text{O}_2\text{ diff}$).

3. How is VO$_{2\text{max}}$ measured?
   VO$_{2\text{max}}$ is measured via various methodologies, including, for example:
   a. Indirect calorimetry using a metabolic chart to estimate oxygen consumption via Haldane transformation.
   b. Field-based tests (eg, Cooper 12-minute test)
   c. Nonexercise algorithm taking into consideration the patient’s self-reported physical activity, age, gender, and body mass index

4. Why is VO$_{2\text{max}}$ considered the best indicator of aerobic fitness?
   It is dependent on several factors:
   - Cardiac output
   - Ventilatory capacity
   - Circulation
   - Ability of the tissues to remove oxygen from the blood

5. What are limiting factors in determining VO$_{2\text{max}}$?
   - In healthy individuals, maximal cardiac output
   - In individuals with asthma, chronic bronchitis, or emphysema, ventilatory compromise
   - In individuals with emphysema, abnormalities in the ventilation-perfusion ratio of the lungs
   - In individuals with peripheral vascular disease, decreased tissue perfusion

6. Are the VO$_{2\text{max}}$ values the same in an individual performing various exercises (eg, treadmill, cycling, arm ergometry)?
   No; the VO$_{2\text{max}}$ value is different for each exercise. Differences are thought to be a result of the amount of muscle mass involved in the exercise. If similar muscle mass is involved, the VO$_{2\text{max}}$ value is highest when the individual is performing the specific exercise for which he or she has trained.

7. Why is the cardiac output and arteriovenous oxygen difference larger in individuals who engage in regular physical activity?
   Chronic, sustained physical activity increases stroke volume, mitochondrial size and density, and CA pillarization.

8. How does the VO$_{2\text{max}}$ of a well-trained man compare with the VO$_{2\text{max}}$ of a well-trained woman?
   When VO$_{2\text{max}}$ is expressed per kilogram of body weight, the VO$_{2\text{max}}$ of a well-trained man is approximately 20% higher than that of a well-trained woman. If VO$_{2\text{max}}$ is expressed relative to lean body mass, it is only about 9% higher in men. The cause of the difference is not known, but it may be as a result of a greater oxygen-carrying capacity in men caused by a higher hemoglobin content, larger blood volume, and higher cardiac output.

9. Define other common indicators of physical fitness.
   - Blood lactate threshold—the intensity of exercise when there is a sudden increase in the amount of lactate in the blood
   - Ventilatory threshold—the intensity of exercise when there is an increase in ventilation corresponding to the development of metabolic acidosis during exercise
10. Differentiate between physical activity, exercise, and physical fitness.

- Physical activity is defined as any movement produced by a person’s skeletal muscles that results in the expenditure of energy
- Exercise is defined as a subset of physical activity that is planned, structured, and repetitive with the goal of improvement or maintenance of a person’s physical fitness
- Physical fitness is a set of attributes that can be either health- or skill-related and the degree of which can be measured with specific performance tests

11. What are the five components of physical fitness?

1. Cardiovascular fitness—also known as cardiorespiratory fitness, is the ability of the heart, lungs, and vascular system to deliver oxygen-rich blood to working muscles during sustained physical activity
2. Muscular strength—the amount of force a muscle or muscle group can exert against a resistance
3. Muscular endurance—the ability of a muscle or muscle group to repeat a movement many times or to hold a particular position for an extended period of time
4. Flexibility—the ability of a joint to move through its full range of motion, from a flexed to an extended position
5. Body composition—the amount of fat in the body compared with the amount of lean mass

12. What is the effect of regular exercise on cardiometabolic parameters?

Exercise has the potential to reduce levels of C-reactive protein (CRP), homocysteine, total cholesterol, low-density lipoprotein (LDL)-cholesterol, triglycerides, fasting glucose and insulin, hemoglobin A1c, blood pressure, and also increase high-density lipoprotein (HDL)-cholesterol

13. What is the effect of regular exercise on neurologic parameters?

Exercise is associated with a reduced risk of Parkinson’s disease, Alzheimer’s disease, and cognitive function, with possible mechanisms occurring from exercise-induced changes in the cerebral blood flow and metabolism, decreases in cortical accumulation of amyloid-β peptides, and increases in brain-derived neurotrophic factors.

14. What is oxygen deficit?

Oxygen deficit is the difference between the amount of oxygen that is consumed and the amount of oxygen that is required to perform an exercise.

15. What effect does warming up have on the oxygen deficit?

It decreases it. Warming up increases blood flow, muscle temperature, and mitochondrial respiration, and these factors enable oxygen to be delivered to and used by the tissues more rapidly. There is less time for a deficit to develop, and this results in a smaller deficit.

16. How do the resting stroke volume, heart rate, and cardiac output of a well-trained athlete compare with those of a sedentary individual?

The resting stroke volume of an athlete is greater than that of a sedentary individual because of hypertrophy of the cardiac muscle in the athlete, which results in an increase in contractility and an increase in venous tone that lead to more blood being returned to the heart. Both the increased contractility and increased venous tone cause an increase in the strength of contraction of cardiac muscle and in the stroke volume.

The resting heart rate of an athlete is lower than that of a sedentary individual (athlete, 40–60 beats/min; sedentary individual, 70–75 beats/min).

The higher stroke volume of an athlete is canceled out by the lower heart rate, resulting in the resting cardiac output of an athlete being similar to that of a sedentary individual.

17. How does the stroke volume response to exercise in the upright position differ between individuals who are physically fit and those who are not?

In a trained individual, stroke volume continues to increase until VO2max is reached; in an untrained individual, stroke volume increases as exercise intensity increases up to about 50% of VO2max and then remains steady. Maximal stroke volume is higher in fit individuals, and the stroke volume for any submaximal exercise intensity is higher in a fit individual.
18. How do heart rate, stroke volume, mean total peripheral resistance, mean arterial blood pressure, and respiratory rate change when exercise is performed using the upper extremities compared with a similar amount of exercise using the lower extremities?

These changes occur mainly because vasodilation occurs in exercising muscles, and vasoconstriction occurs in nonexercising muscles. Upper extremity exercise involves smaller muscles than lower extremity exercise. During upper extremity exercise, more vasoconstriction is occurring than vasodilation. This causes an increase in total peripheral resistance, and changes in the other variables occur as a result of this.

<table>
<thead>
<tr>
<th>Higher</th>
<th>Slightly Lower</th>
<th>Much Lower</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate</td>
<td>Cardiac output</td>
<td>Stroke volume</td>
</tr>
<tr>
<td>Mean arterial blood pressure</td>
<td></td>
<td></td>
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<tr>
<td>Respiratory rate</td>
<td></td>
<td></td>
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<tr>
<td>Total peripheral resistance</td>
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</tbody>
</table>

19. What is the acute response of systolic and diastolic blood pressure to aerobic exercise?

During acute incremental exercise, systolic blood pressure increases while diastolic blood pressure remains steady or slightly decreases. Post acute exercise, both systolic and diastolic pressure can lower in both hypertensive and normotensive individuals, often referred to as postexercise hypotension.

20. Describe the normal interaction of inotropes and chronotropes during exercise.

During exercise the initial chronotropes and inotropes are the sympathetic nerves that directly innervate the heart. A slightly delayed chronotrope and inotrope come from the adrenal medulla. When sympathetic nerves innervating the adrenal medulla are stimulated, epinephrine and norepinephrine are released into the blood. These hormones travel to the heart and perpetuate the response that was initiated by the sympathetic nerves.

21. What effect does a low partial pressure of oxygen (Po2) have on blood vessel diameter in the lung and in the systemic circulation?

Vessels in the lung constrict when exposed to a low Po2, whereas vessels in the systemic circulation dilate. The constriction of vessels in the lung shunts blood to the areas of the lung that are better ventilated. This results in better ventilation-perfusion matching, which causes more effective oxygenation of blood. Dilation of systemic vessels enables more blood to be delivered to the area. This results in better oxygenation of the localized tissues.

22. Discuss the effect long-term endurance training has on the heart and on blood volume.

Increases in plasma volume occur shortly after the initiation of intense endurance training. This appears to be caused by an increase in plasma albumin levels, which osmotically draws fluid into the vasculature. Higher plasma volumes cause an increase in venous return, left ventricular end-diastolic volume, and stroke volume. These changes can occur within 1 week of the initiation of endurance training. Hypertrophy of myocardial muscle also occurs with endurance training, but this is a slower process.

23. Describe the contributions of stored adenosine triphosphate (ATP), creatine phosphate, glycolysis, and aerobic metabolism toward providing ATP during intense exercise over time.

- Stored ATP is used primarily for maximal intensity exercise, causing fatigue after about 4 seconds.
- If the intensity of exercise is such that fatigue occurs after about 10 seconds, creatine phosphate is used to supply the energy to replenish the ATP stores during the last 6 seconds of exercise.
- Intense exercise lasting between 10 seconds and 2 minutes depends on anaerobic glycolysis for ATP production. The maximal intensity of exercise is not as great as it was when creatine phosphate was being used.
- For intense exercise lasting longer than 2 minutes, aerobic metabolism provides most of the ATP, and the maximal intensity of the exercise that can be sustained is only about half of what it was during anaerobic glycolysis.
24. What can be done to improve the systems for providing ATP during intense exercise?
To improve the ability of creatine phosphate to provide energy, several bouts of intense exercise should be performed for 5 to 10 seconds with a 30- to 60-second rest between bouts. To improve anaerobic capacity, several bouts of intense exercise should be performed for at most 1 minute in duration with 3 to 5 minutes of recovery between bouts.

25. What are the main muscle fiber types and their characteristics?

<table>
<thead>
<tr>
<th>Properties</th>
<th>Type I</th>
<th>Type IIa</th>
<th>Type IIb</th>
</tr>
</thead>
<tbody>
<tr>
<td>Motor unit type</td>
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<td>Fast oxidative glycolytic</td>
<td>Fast glycolytic</td>
</tr>
<tr>
<td>Motor unit type</td>
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<td>Fast</td>
<td>Fast</td>
</tr>
<tr>
<td>Diameter</td>
<td>Small</td>
<td>Medium</td>
<td>Large</td>
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<tr>
<td>Twitch velocity</td>
<td>Low</td>
<td>Intermediate</td>
<td>High</td>
</tr>
<tr>
<td>Twitch force</td>
<td>Small</td>
<td>Medium</td>
<td>Large</td>
</tr>
<tr>
<td>Resistance to fatigue</td>
<td>High</td>
<td>Intermediate</td>
<td>Low</td>
</tr>
<tr>
<td>Glycogen content</td>
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<td>High</td>
<td>High</td>
</tr>
<tr>
<td>Capillary density</td>
<td>Rich</td>
<td>Rich</td>
<td>Poor</td>
</tr>
<tr>
<td>Myoglobin content</td>
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<td>Dark red</td>
<td>Pale</td>
</tr>
<tr>
<td>Mitochondrial density</td>
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<td>High</td>
<td>Low</td>
</tr>
<tr>
<td>Oxidative capacity</td>
<td>High</td>
<td>Moderate-high</td>
<td>Low</td>
</tr>
</tbody>
</table>

26. Which type of muscle fiber is activated during moderate-intensity, long-duration exercise, such as jogging?
Slow-twitch type 1 fibers are primarily activated.

27. Which type of muscle fiber is activated during high-intensity, short-term exercise, such as sprinting?
Slow-twitch type 1 and fast-twitch type 2 fibers are activated.

28. Why are specific muscle fiber types activated during different kinds of exercise?
The activation of a particular motor unit depends on the size of the α-motor neuron that innervates it. Type 1 fibers are innervated by small α-motor neurons, which have a lower threshold of stimulation than type 2 fibers; type 1 fibers always are stimulated first. Type 2 fibers are stimulated only if the intensity of the exercise requires it.

29. Explain why movements become less precise and refined as low-intensity exercise is continued for a prolonged period of time.
Initially, low-intensity exercise uses motor units consisting of slow-twitch muscle fibers. These motor units have fewer muscle fibers than motor units with fast-twitch fibers, and this accounts for better control during low-intensity exercises compared with high-intensity exercises. If low-intensity exercise is prolonged to the point that glycogen is depleted, the fast-twitch motor units are recruited. These motor units have more muscle fibers and result in less control of movements.

30. Can the three muscle fiber types be changed as a result of exercise?
Type 1 fibers cannot be converted to type 2 fibers, but type 1 fibers can improve their ability to use anaerobic metabolism, and type 2 fibers can improve their ability to use aerobic metabolism. Type 2b fibers can be converted to type 2a fibers with endurance training or strength training.

31. What changes occur in muscle with endurance training?
Endurance training results in improvements in oxygen delivery and use. This is caused by an increase in capillary and mitochondria content and aerobic oxidative enzyme activity. Type 2b muscle fibers are converted to type 2a. The cross-sectional area of the muscle decreases, resulting in shorter diffusion distances for oxygen and carbon dioxide.
32. **What changes occur in muscle with resistance training, and how long does it take for those changes to occur?**

Resistance training causes synthesis of proteins in thick and thin filaments, resulting in an increase in the cross-sectional area. The ratio of mitochondrial volume to contractile protein volume decreases. The aerobic capacity of the muscle decreases, which hinders performance in endurance activities. Type 2b muscle fibers are converted to type 2a. It takes about 6 to 8 weeks for the addition of protein filaments, but conversion of type 2b to type 2a fibers begins after about 2 weeks.

33. **What causes improvements in strength with resistance training?**

In the first 2 weeks, 90% of the improvements are attributed to neural changes, including improvements in the recruitment pattern of motor units, increases in CNS activation, more synchronization of motor units, and less neural inhibition. After about 6 weeks of training, 80% of the improvements are from an increase in contractile proteins.

34. **What is the cause of athletic amenorrhea?**

Women who train heavily have higher levels of catecholamines, cortisol, and β-endorphins. These hormones inhibit the release of luteinizing hormone and follicle-stimulating hormone, which results in decreased levels of estradiol. This contributes to the cause of athletic amenorrhea. Studies have shown that physical and emotional stress, diet, and the presence of menstrual irregularity before training also contribute. The exact mechanism is not known.

35. **Is it true that pregnant women who are physically fit deliver more easily?**

There is some evidence to suggest this, but there is also evidence stating otherwise. However, the perception of pain may be less in physically fit women.

36. **Summarize some physiologic changes that occur during pregnancy that affect exercise.**

The American College of Obstetrics and Gynecology (ACOG) recognizes the following:

- a. After the first trimester, the supine position results in relative obstruction of venous return by the enlarging uterus and a significant decrease in cardiac output.
- b. Stroke volume and cardiac output during steady-state exercise are increased significantly.
- c. Exercise during pregnancy induces a greater degree of hemoconcentration than does exercise in the nonpregnant state.
- d. There is a 10% to 20% increase in baseline oxygen consumption during pregnancy.
- e. Because of the increased resting oxygen requirements and the increased work of breathing brought about by physical effects of the enlarged uterus on the diaphragm, decreased oxygen is available for the performance of aerobic exercise during pregnancy.
- f. There is a shift in the physical center of gravity that may affect balance.
- g. Basal metabolic rate and heat production increase during pregnancy.
- h. Approximately 300 extra kilocalories per day are required to meet the metabolic needs of pregnancy; this caloric requirement is increased further in pregnant women who exercise regularly.
- i. Pregnant women use carbohydrates during exercise at a greater rate than do nonpregnant women; adequate carbohydrate intake for exercising pregnant patients is essential.

37. **What are the American College of Sports Medicine (ACSM) guidelines for physical activity?**

ACSM’s physical activity guidelines for healthy adults recommends participating in at least 30 minutes of moderate-intensity physical activity 5 days per week or 150 minutes total of moderate-intensity exercise. For vigorous intensity physical activity, 20 minutes for 3 days per week is recommended.

38. **What are the American College of Sports Medicine (ACSM) guidelines for muscular fitness?**

The American College of Sports Medicine (ACSM) recommends that a strength training program should be performed a minimum of two nonconsecutive days each week, with one set of 8 to 12 repetitions for healthy adults or 10 to 15 repetitions for older and frail individuals. Eight to 10 exercises should be performed that target the major muscle groups.
39. List the general American College of Sports Medicine (ACSM) guidelines for an exercise program to decrease body weight.
   a. The most successful program to decrease body weight is one that combines exercise with dieting. Such a program decreases weight, decreases fat mass, and maintains or increases fat-free mass. If one diets without exercising, one may lose more weight than by combining diet and exercise, but fat-free mass is lost in addition to fat mass.
   b. An aerobic exercise program is most effective.
   c. Exercise should be performed at least 3 days per week at an intensity and duration to expend 250 to 300 kilocalories per exercise session for a 75-kg person. This usually requires a duration of at least 30 to 45 minutes for a person in average physical condition.

40. What are the American College of Sports Medicine (ACSM) guidelines for sustaining weight loss?
   For sustaining weight loss, the ACSM recommendation is to engage in >250 minutes/week of moderate-intensity physical activity.

41. Describe the “fit-but-fat” paradigm.
   • An individual could have adequate cardiorespiratory capacity but still be of an undesirable bodyweight.
   • Evidence suggests that overweight (and possibly obese) adults who are physically active may be just as healthy, or even healthier, than inactive normal weight adults.

42. What are the American College of Sports Medicine (ACSM) guidelines for an exercise program to preserve bone health?
   a. Type of exercise should include weight-bearing endurance activities such as tennis, stair climbing, and jogging intermittently during walking; jumping activities such as volleyball and basketball; and resistance exercises that involve all major muscle groups, such as weight lifting.
   b. Intensity should be moderate to high, in terms of bone-loading forces.
   c. The frequency of weight-bearing endurance activities should be 3 to 5 times per week, resistance exercise 2 to 3 times per week.
   d. Duration should be 30 to 60 minutes per day.
   e. The older adult should also perform activities to improve balance for the prevention of falls.

43. How do exercise and training affect the endocrine system and the resting levels of hormones?
   Most hormone levels increase during submaximal, short-term exercise with the exception of insulin, which decreases, and thyroid hormones, which do not change. Resting levels of ACTH, cortisol, catecholamines, insulin, and glucagons decrease with training. This may be related to greater energy stores or a decreased perception of stress.

44. Discuss prolonged, moderate-intensity exercise training and blood glucose levels in individuals with type 1 and type 2 diabetes.
   Blood glucose levels do not seem to change with a prolonged exercise program in individuals with type 1 diabetes, but they decrease in individuals with type 2 diabetes. Exercise causes the cells of type 2 diabetic patients to be less resistant to insulin. This seems to be most effective if exercise is performed at an intensity of 60% to 75% of VO2max. Most type 2 diabetic patients are overweight. Exercise may help to reduce body fat percentage, which results in an increase in the number of insulin receptors, an increase in their sensitivity, or both. Exercise reduces the cholesterol level in type 2 diabetic patients. This, along with the accompanying weight loss, decreases the cardiovascular risk factors of these individuals, which is the most significant benefit of performing exercise. Although exercise has not been shown to improve blood glucose levels in individuals with type 1 diabetes, it is still recommended for the same reasons that exercise is recommended for individuals without diabetes.

45. Does exercise affect the prevalence of upper respiratory tract infections (URTI)?
   Few studies have addressed the effect of moderate-intensity exercise on URTI. Preliminary results indicate a decrease in URTI with moderate exercise. More evidence indicates an increased prevalence of URTI during heavy endurance training and 1 to 2 weeks after a marathon-type event.
46. Should patients with chronic obstructive pulmonary disease (COPD) be encouraged to exercise?
Ambulation distance and feeling of well-being can increase significantly with an exercise program in individuals with mild or moderate COPD. There is controversy regarding the benefits of exercise for individuals with severe COPD. Some studies have shown improvements in endurance, whereas others have found no change. Only patients with stable COPD are encouraged to participate in an exercise program in a nonmedical setting.

47. How does the heart rate response to exercise differ between normal individuals and individuals who have had heart transplants?
In normal individuals, heart rate increases rapidly with moderate exercise as a result of a decrease in parasympathetic nerve activity and an increase in sympathetic nerve activity. Transplanted hearts are denervated. Any change in heart rate must be caused by changes in circulating levels of catecholamines, which takes more time than altering nerve activity. It takes longer for the heart rate to increase when exercise is initiated, and it takes longer for it to return to resting levels after exercise.

48. How does resting heart rate differ between normal individuals and individuals who have had heart transplants?
Resting heart rate is higher in individuals who have had a heart transplant because they no longer have the normal parasympathetic tone to slow the intrinsic rate of depolarization of the sinoatrial node.

49. Why are individuals with thoracic-level spinal cord injuries at risk for fainting after exercising in the upright position with the upper extremities?
There is no sympathetic innervation to the lower limb vasculature, and there may not be any innervation to the adrenal glands (depending on how high the injury is). This results in a lack of vasoconstriction of the vessels of the lower extremities, venous pooling occurs, and syncope follows.

50. What is the most common problem associated with exercising in cold environments?
When people know they are going to be exercising in cold environments, they usually overdress, resulting in hyperthermia.

51. List strategies to avoid hypothermia and hyperthermia when exercising in a cold environment.
- Dress in layers that can be removed as the exercise progresses.
- Stay dry; heat is lost much more rapidly when you are wet than when you are dry.

52. Describe the physiologic changes that occur with exercising in the cold.
Compared with a thermoneutral environment, exercising in the cold results in less lipid metabolism and free fatty acid use but greater lactate production and higher ventilation, oxygen consumption, respiratory heat loss, and peripheral heat loss.

53. List possible causes for decreased maximal muscle strength and power with hypothermia.
- Increased viscosity of skeletal muscle
- Increased resistance to blood flow
- Decreased maximal nerve conduction velocity

54. What are the two most common problems associated with exercising in hot environments?
Dehydration and hyperthermia are the two most common problems in this situation.

55. How can dehydration and hyperthermia be avoided?
These problems cannot be avoided completely, but they can be limited by ingesting fluid while exercising. There appears to be a similar benefit between ingestion of pure water compared with carbohydrate and electrolyte drinks as far as controlling core temperature and cardiovascular changes.

56. Describe the physiologic changes that occur with exercising in the heat.
The principal physiologic responses of exercise in the heat include skin and muscle vasodilation, nonactive tissue vasoconstriction, maintenance of blood pressure, and sweating. The hypothalamus plays a crucial role in thermoregulatory integration.
57. Does living at high altitude improve exercise tolerance at high altitude?
Yes. The exercise response of subjects at a high altitude who live at moderate altitudes compared with subjects who live at sea level shows that individuals who live at a moderate altitude have less of a decrease in VO$_{2\text{max}}$ and blood lactate accumulation. They also have larger maximal ventilation during maximal exercise. Hematocrit levels increase after about 25 days of exposure to high altitude, which should increase performance. Some studies indicate that pulmonary function, cardiac output, muscle enzyme capacity, and lean body mass decrease at high altitudes. World-class athletes performing endurance exercises consistently seem to perform better if they train at a moderate altitude.

BIBLIOGRAPHY