# Basic Athletic Training
# Course Pack A

# Therapeutic Interventions

SECTION III

CHAPTER

10

# Tissue Healing and Wound Care

## STUDENT OUTCOMES

1. Describe the major mechanical forces that produce injury to biological tissues; namely, compression, tension, shear, stress, strain, bending, and torsion.
2. Explain the effect of the material constituents and structural organization of the skin, tendons, ligaments, muscles, and bone on their ability to withstand the mechanical loads to which each is subjected.
3. List common injuries of the skin, muscles, tendons, joints, and bone.
4. Describe the processes by which tissue healing occurs in the skin, tendons, muscles, ligaments, and bone.

5. Explain wound care for both superficial and deep soft-tissue injuries.
6. Describe the appropriate immediate management of bone injuries.
7. Explain the mechanisms by which nerves are injured and the processes by which nerves can heal.
8. Describe the types of altered sensations that can result from a nerve injury.
9. Describe the appropriate immediate management of nerve injuries.
10. Explain the neurological basis of pain, including factors that mediate pain.

## INTRODUCTION

Human movement during sport and exercise typically is faster and produces greater force than activities of daily living. As a result, the potential for injury also is heightened. Understanding the different ways in which forces act on the body is necessary to comprehend techniques to prevent injuries. Likewise, knowing the material and structural properties of the skin, tendons, muscles, ligaments, bones, and nerves can lay a foundation for understanding the response of these tissues to applied forces can facilitate an individual's safe return to sport participation.

This chapter begins with a general discussion of injury mechanisms, including descriptions of force and torque as well as their effects. This is followed by sections on soft tissues, bones, and nerves, addressing the mechanical characteristics of these tissues, the classification of injuries, processes by which the specific tissues heal, and general wound care for these injuries. A more detailed explanation of wound care for specific injuries is discussed in individual chapters.

# INJURY MECHANISMS

? When the human body sustains force the potential exists to strengthen body tissues or to injure them. A previously sedentary middle-aged adult wants to initiate a running program as a way to improve his cardiovascular fitness. What advice should be provided to this individual for reducing the risk of sustaining a mechanical stress–related injury? Why?

Analyzing the mechanics of injuries to the human body is complicated by several factors. First, potentially injurious forces applied to the body act at different angles, over different surface areas, and over different periods of time. Second, the human body is composed of many different types of tissue, which respond differently to applied forces. Finally, injury to the human body is not an all-or-nothing phenomenon; that is, injuries range in severity. This section introduces the types of mechanical loading that can cause injury and describes the basic mechanical responses of biological tissues to these forms of loading.

## Force and Its Effects

Force may be thought of as a push or a pull acting on a body. A multitude of forces act on our bodies routinely during the day. The forces of gravity and friction enable us to move about in predictable ways when muscles produce internal forces. During participation in sports and physical activities, force is applied to sticks, balls, bats, racquets, clubs, and other objects. Force is absorbed from impact with the ground or floor, the object used, in contact sports, and even other participants.

When a force acts, two potential effects on the target object exist. The first is acceleration, or change in velocity, and the second is deformation, or change in shape. For example, when a racquetball is struck with a racquet, the ball is both accelerated (i.e., put in motion in the direction of the racquet swing) and deformed (i.e., flattened on the side that is struck). The greater the stiffness of

the material to which a force is applied, the greater the likelihood that the deformation will be too small to be easily seen. The more elastic the material to which a force is applied, the greater the likelihood that the deformation will be temporary, with the material springing back to regain its original shape.

When tissues sustain a force, two primary factors dictate whether injury occurs—namely, the size, or magnitude, of the force and the material properties of the involved tissues (Box 10.1). A load-deformation curve demonstrates the deformation of a structure in response to progressive loading or force application. If a load is relatively small, the response of the structure is elastic. As such, when the load is removed, the material will return to its original size and shape. Within the elastic region of the load-deformation curve, the greater the stiffness of the material, the steeper the slope of the line becomes. Therefore, greater stiffness translates to less deformation in response to a given load. If a load exceeds the material's **yield point**, or **elastic limit**, however, the response of the structure is plastic. In this situation, when the load is removed, some amount of deformation will remain. Loads exceeding the ultimate failure point on the load-deformation curve result in mechanical failure of the structure, which translates into the fracturing of bone or rupturing of soft tissues.

### BOX 10.1 Factors Affecting the Likelihood of Injury

- Size or magnitude of the force
- The force's moment arm, which determines the amount of torque generated
- Direction in which the force is applied (i.e., axial torque generated compression, tensile, or shear force)
- Material properties of the tissues affected and their ability to sustain strain
- Area over which the force is applied
- Magnitude of stress produced by the force

The direction of an applied force also has important implications for injury potential. Many tissues are **anisotropic**, meaning that the structure is stronger in resisting force from certain directions compared to others. The anatomical design of many of the joints of the human body also means they are more susceptible to injury from a given direction. For example, lateral ankle sprains are much more common than medial ankle sprains because of the bony configuration and ligamentous support on the medial side. Consequently, when discussing injury mechanisms, force commonly is categorized according to the direction from which the force acts on the affected structure.

Force acting along the long axis of a structure is termed **axial force**. In fencing, when an opponent is touched with the foil, the foil is loaded axially. When the human body is in an upright standing position, body weight creates axial loads on the femur and tibia.

Axial loading that produces a squeezing or crushing effect is termed **compressive force**, or compression (**Fig. 10.1A**). The weight of the human body constantly produces compression on the bones that support it. The 5th lumbar vertebra must support the weight of the head, trunk, and arms when the body is erect, producing compression on the intervertebral disk below it. When a football player is sandwiched between two opposing players, the force acting on that player is compressive. In the absence of sufficient padding, compressive forces often result in bruises or contusions.

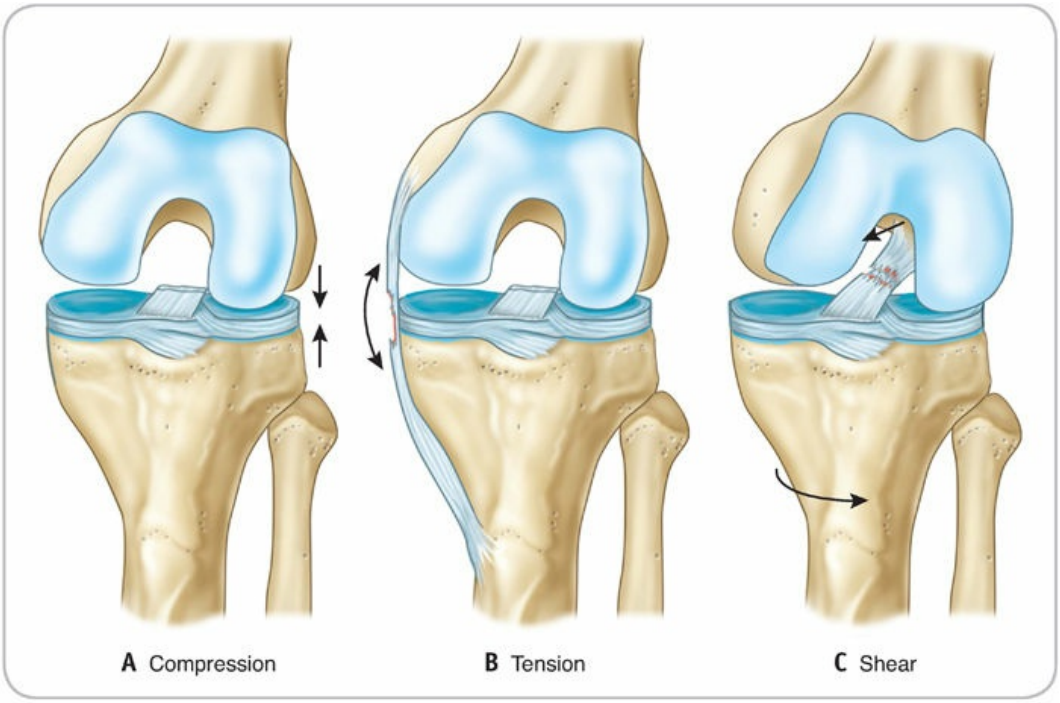



**Figure 10.1. Mechanisms of injury.** Compression **(A)** and tension **(B)** are directed along the longitudinal axis of a structure, whereas shear **(C)** acts parallel to a surface.

Axial loading in the direction opposite that of compression is called **tensile force**, or tension (Fig. 10.1B). Tension is a pulling force that tends to stretch the object to which it is applied. Muscle contraction produces tensile force on the attached bone, enabling movement of that bone. When the foot and ankle are inverted, the tensile forces applied to the lateral ligaments may result in an ankle sprain.

Whereas compressive and tensile forces are directed, respectively, toward and away from an object, a third category of force, termed **shear force**, acts parallel or tangent to a plane passing through the object (Fig. 10.1C). Shear force tends to cause one part of the object to slide or displace with respect to another part of the object. For example, shear forces acting on the spine can cause spondylolisthesis, a condition involving anterior slippage of a vertebra with respect to the vertebra below it.

When the human body sustains force, another important factor related to the likelihood of injury is the magnitude of the stress produced by that force. Mechanical **stress** is defined as force divided by the surface area over which the force is applied (Fig. 10.2). When a given force is distributed over a large

area, the resulting stress is less than if the force were distributed over a smaller area. Alternatively, if a force is concentrated over a small area, the mechanical stress is relatively high. A high magnitude of stress, rather than a high magnitude of force, is what tends to result in injury to biological tissues. One of the reasons that participants in contact sports wear pads is because a pad dissipates force across its entire area, thereby reducing the stress acting on the player.

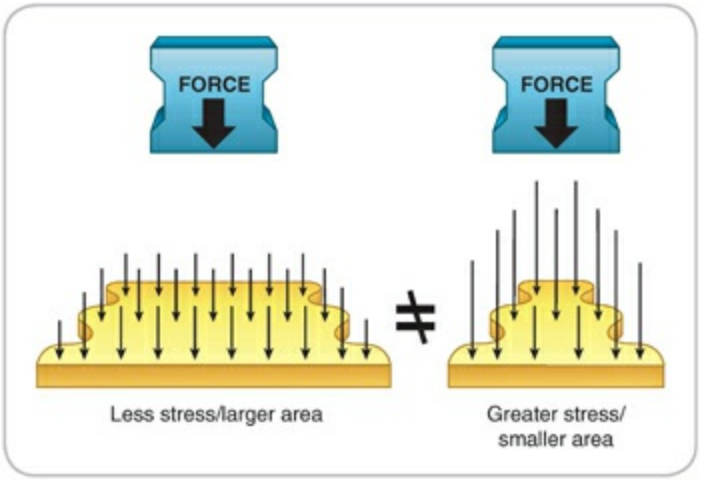



**Figure 10.2. Stress.** The stress produced by a force depends on the area over which the force is spread. For example, the stress sustained by the superficial tissues of the arm would be lower than that with the force distributed directly over a small, bony landmark. The risk of injury increases when force is sustained over smaller areas.

**Strain** may be thought of as the amount of deformation an object undergoes in response to an applied force. Application of compressive force to an object produces shortening and widening of the structure, whereas tensile force produces lengthening and narrowing of the structure. Shear results in internal changes in the structure on which the force is acting. The ultimate strength of biological tissues determines the amount of strain that a structure can withstand without fracturing or rupturing.

Injury to biological tissues can result from a single traumatic force of relatively large magnitude or from repeated forces of relatively smaller magnitude. When a single force produces an injury, the injury is called an **acute injury** and the causative force is termed a **macrotrauma**. An acute injury, such as a ruptured anterior cruciate ligament or a fractured humerus, is

characterized by a definitive moment of onset followed by a relatively predictable process of healing. When repeated or chronic loading over a period of time produces an injury, that injury is called a **chronic injury** or **stress injury**, and the causative mechanism is termed **microtrauma**. A chronic injury, such as glenohumeral bursitis or a metatarsal stress fracture, develops and worsens gradually over time, typically culminating in a threshold episode in which pain and inflammation become evident. Chronic injuries may persist for months or years.

Many tissues, including tendons, ligaments, muscles, and bones, tend to respond to gradually increased mechanical stress by becoming larger and stronger. According to the basic principle of overload, when stressed at tolerable levels, the tissues of the body will adapt and improve their function. Accordingly, in designing an exercise program, it is important to ensure a gradual increase in the intensity and duration of activities. For example, when a runner's training protocol incorporates progressively increasing mileage, it is important that this occur in a deliberate and carefully planned manner so that the body can adapt to the increased mechanical stress and, thereby, prevent a potential injury. Overuse syndromes and stress fractures result from the body's inability to adapt to an increased training regimen.

## Torque and Its Effects

When a swinging door is opened, a hand applies force to the door, causing it to rotate about its hinges. Two factors influence whether the door will swing in response to the force. One factor is the force's **magnitude**. Equally important, however, is the force's **moment arm**, which is the perpendicular distance from the force's line of action to the axis of rotation. The product of a force and its moment arm is called **torque**, or moment. Torque may be thought of as a rotary force. It is the amount of torque acting on an object that determines whether a rotating body, such as a door, will move.

In the human body, torque produces rotation of a body segment about a joint. When a muscle develops tension, it produces torque at the joint that it crosses. The amount of torque produced is the product of muscle force and the

muscle's moment arm with respect to the joint center (Fig. 10.3). For example, the torque produced by the biceps brachii is the product of the tension developed by the muscle and the distance between its attachment on the radius and the center of rotation at the elbow.

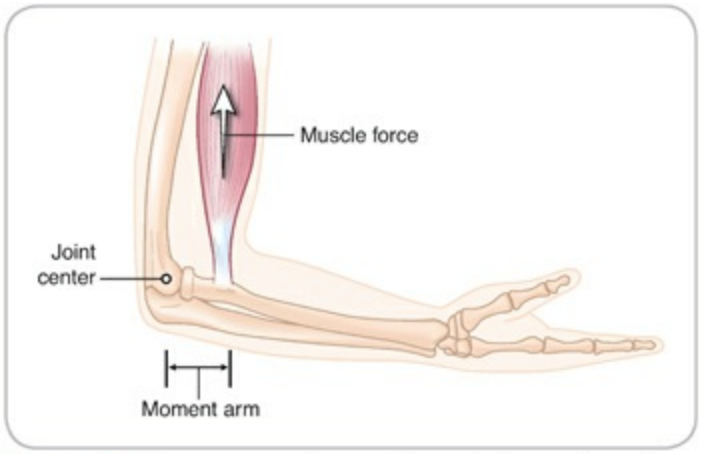



**Figure 10.3. Movement and torque.** Torque is the product of the magnitude of muscle force and the muscle's moment arm (perpendicular distance of the muscle's line of action to the axis of rotation at the joint center).

Excessive torque can produce injury. Such torque usually is generated by forces external to the body rather than by the muscles. The simultaneous application of forces from opposite directions at different points along a structure, such as a long bone, generates a torque known as a **bending moment**, which can cause bending and, ultimately, fracture of the bone. For example, if a football player's leg is anchored to the ground and the player is tackled on that leg from the front while being pushed into the tackle from behind, a bending moment is created on the leg. When bending is present, the structure is loaded in tension on one side and in compression on the opposite side (Fig. 10.4A). Because bone is stronger in resisting compression compared with tension, the side of the bone loaded in tension will fracture if the bending moment is sufficiently large.

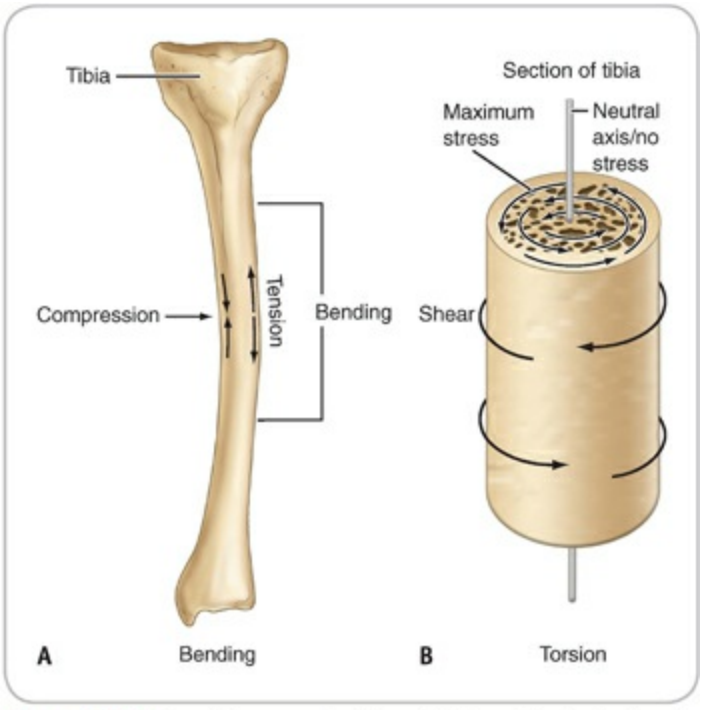



**Figure 10.4. Bone injury mechanisms. A,** Bones loaded in bending are subject to compression on one side and tension on the other. **B,** Bones loaded in torsion develop internal shear stress, with maximal stress at the periphery.

The application of torque about the long axis of a structure such as a long bone can cause **torsion**, or twisting of the structure (Fig. 10.4B). Torsion results in the creation of shear stress throughout the structure. This often is seen in skiing accidents in which one boot and ski are firmly planted as the skier rotates during a fall. The result is a torsion load that can cause a spiral fracture of the tibia.

According to the overload principle the body when stressed at tolerable limits will adapt and improve its function. As such the runner should be advised to incorporate gradual and progressive increases in mileage and time so that the body can adapt to the new training and thereby reduce the likelihood of injury.

## SOFT-TISSUE INJURIES

Two weeks into a new running program, a previously sedentary, middle-aged man reports a dull, diffuse pain along the distal third of the posteromedial border of the tibia of his lower left leg. He indicates that 5 days ago, he began experiencing pain while running, but it did not restrict his performance. He reports that for the past 2 days, however, the pain has increased with continued activity and as such has restricted his performance. Based on this information, what actions, if any, should be taken with regard to this patient? Why?

The skin, tendons, muscles, and ligaments are soft (nonbony) tissues that behave in characteristic ways when subjected to different forms of loading. The anatomical structure and material composition influence the mechanical behavior of each tissue.

## Anatomical Properties of Soft Tissue

The major building block of the skin, tendons, and ligaments is collagen, a protein that is strong in resisting tension. Collagen fibers have a wavy configuration in a tissue that is not under tension (Fig. 10.5). This enables collagenous tissues, which are inelastic, to stretch slightly under tensile loading as these fibers straighten. As such, collagen fibers provide strength and flexibility to tissues, but they are relatively inelastic. Elastin, another protein substance, provides added elasticity to some connective tissue structures.

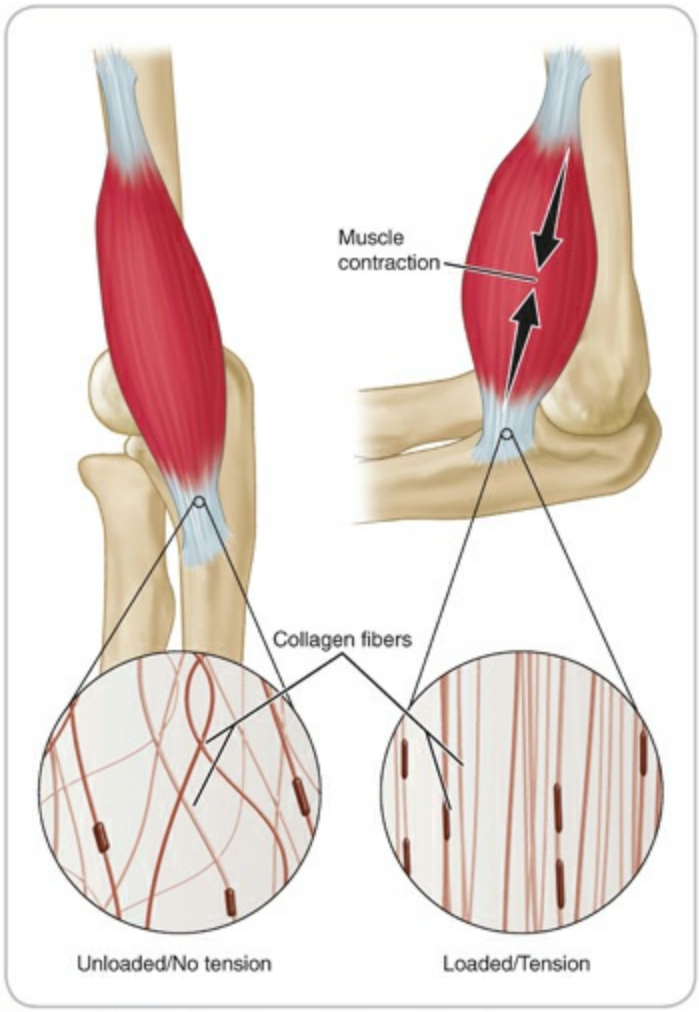



**Figure 10.5. Collagen fibers.** Collagen fibers have a wavy configuration when unloaded and a straightened configuration when loaded in tension.

## *The Skin*

The integumentary system comprises the skin, hair, nails, and glands of the skin and is the largest organ in the body. It provides protection and sensation, regulates fluid balance and temperature, and produces vitamins (e.g., vitamin D) and immune system components. The skin is composed of three regions. The outer region, known as the epidermis, has multiple layers containing the pigment melanin, along with the hair, nails, sebaceous glands, and sweat glands (Fig. 10.6). The outer surface, made up of dead epithelial cells, is replaced every 3 to 4 weeks by new cells pushed up from the dermis. The dermis is the largest portion of the skin and provides both strength and

structure. It contains blood vessels, nerve endings, hair follicles, sebaceous glands, and sweat glands and is beneath the epidermis. The dermis is composed of dense, irregular connective tissue, which is characterized by a loose, multidirectional arrangement of collagen fibers. This fiber arrangement enables resistance to multidirectional loads, including compression, tension, and shear. This type of tissue also forms fascia, which are fibrous sheets of connective tissue that surround muscles. Dense, irregular connective tissue also covers internal structures, such as the liver, lymph nodes, and testes, as well as bones, cartilage, and nerves. The innermost layer of skin is the subcutaneous or hypodermal layer. The primary tissue is adipose, which provides cushioning between the skin layers, muscles, and bones and is instrumental in regulating body temperature because of its insulating properties.

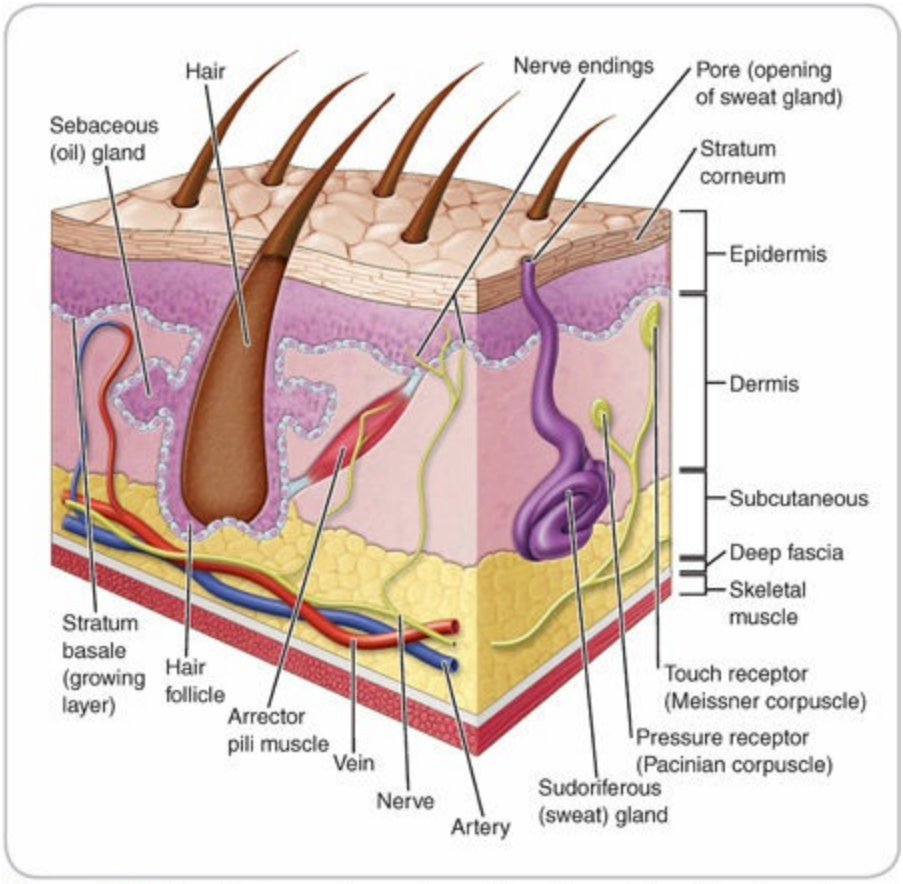



**Figure 10.6. Skin.** The two major regions of the skin are the epidermis and the dermis.

Elastic fibers and reticular fibers are other components of the skin. Elastic

fibers provide the skin with some elasticity. Reticular fibers are composed of a type of collagen known as reticulin. These fibers function like collagen fibers but are much thinner, and they provide support for internal structures, such as the lymph nodes, spleen, bone marrow, and liver.

## *Tendons, Aponeuroses, and Muscles*

Tendons connect muscles to bones. They are composed of dense, regular connective tissue that consists of tightly packed bundles of unidirectional collagen fibers (Fig. 10.7). The collagen fibers are arranged in a parallel pattern, enabling resistance to high, unidirectional tensile loads when the attached muscle contracts. By virtue of their collagenous composition, tendons are about twice as strong as the muscles to which they attach.

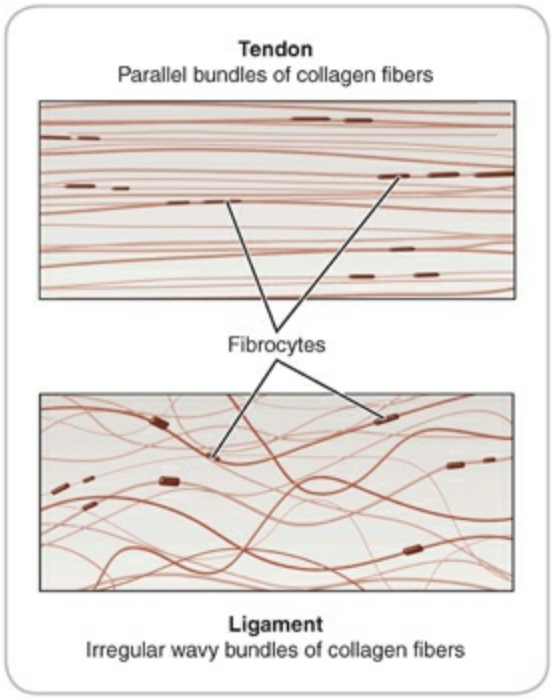



**Figure 10.7. Collagen arrangements in tendon and ligament tissue.** The arrangement of collagen in tendons and ligaments differs, producing differences in their ability to resist tensile loads.

The aponeuroses are another set of structures formed by dense, regular connective tissue. These are strong, flat, sheetlike tissues that attach muscles to other muscles or bones.

Muscle is a highly organized structure that can

- Be stretched or increased in length (extensibility)
- Return to normal length after lengthening or shortening takes place (elasticity)
- Respond to a stimulus (irritability)
- Develop tension

A sheath known as the endomysium surrounds each muscle cell, or fiber. Small numbers of fibers are bound up into fascicles by a dense connective tissue sheath called the perimysium. A muscle is composed of several fascicles surrounded by the epimysium (Fig. 10.8).

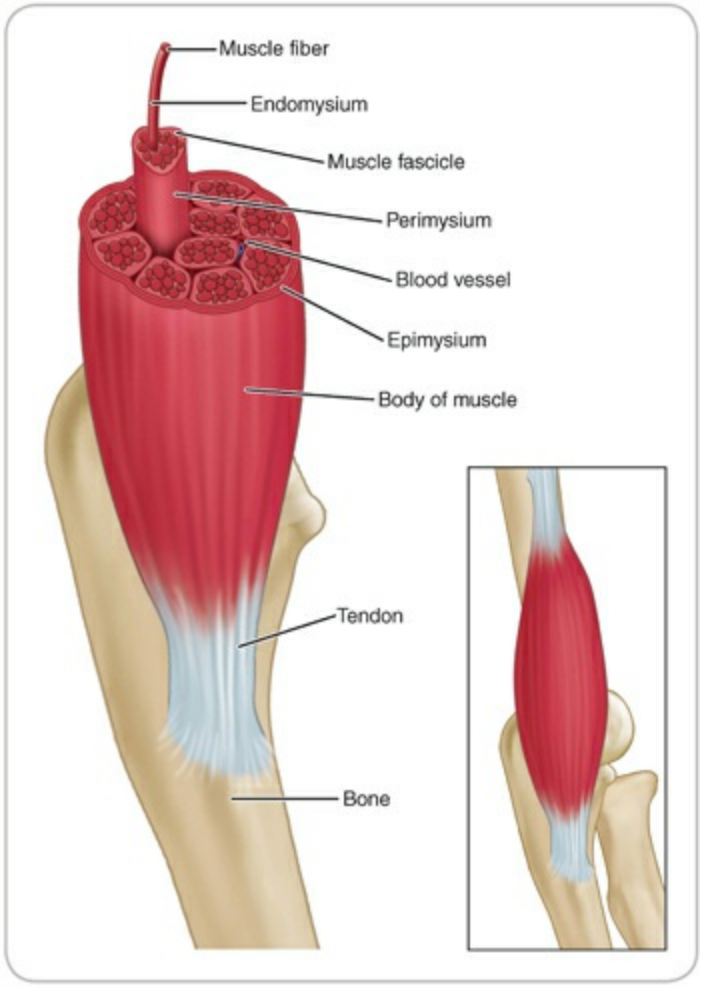



**Figure 10.8. Muscle tissue.** Skeletal muscle is composed of muscle cells, connective tissue, blood vessels, and nerves.

The structure and composition of muscles enable it to function in a

**viscoelastic** fashion, that is, with both elasticity and time-dependent extensibility. **Extensibility** is the ability to be stretched or increase in length, whereas **elasticity** is the ability to return to normal length after either lengthening or shortening has taken place. The viscoelastic aspect of muscle extensibility enables muscle to stretch to greater lengths over time in response to a sustained tensile force. This means that a static stretch maintained for 30 seconds is more effective than a series of short, ballistic stretches for increasing muscle length.

Another of muscle's characteristic properties, **irritability**, is the ability to respond to a stimulus. Stimuli affecting muscles can be either electrochemical, such as an action potential from the attaching nerve, or mechanical, such as an external blow to the muscle. If the stimulus is of sufficient magnitude, muscle responds by developing tension. The ability to develop **tension** is a property unique to muscle. Although some sources refer to this ability as contractility, a muscle may or may not shorten when tension is developed. For example, isometric "contraction" involves no joint movement and no change in muscle length, and eccentric "contraction" actually involves lengthening of the muscle developing tension. Only when a muscle develops tension concentrically does it also shorten. When a stimulated muscle develops tension, the amount of tension present is the same throughout the muscle and tendon and at the site of the tendon attachment to bone.

## *Joint Capsule and Ligaments*

A joint capsule is a membrane that encloses a joint. It functions to hold the bones firmly in place. The outer portion of the capsule is fibrous and composed primarily of collagen. The inner lining of the capsule consists of a synovial membrane, which secretes a clear, slightly yellow liquid, called synovial fluid that provides lubrication inside the articular capsule at synovial joints. (Joints are explained in more detail later in this chapter.)

Ligaments connect bone to bone. Similar to tendons, they are composed of dense, regular connective tissue that consists of tightly packed bundles of unidirectional collagen fibers. In ligaments, however, the parallel collagen fibers also are interwoven. This arrangement is well suited to ligament

function, not only by providing resistance to large tensile loads along the long axis of the ligament but also by providing resistance to smaller tensile loads from other directions.

Ligaments contain more elastin than tendons and, as such, are somewhat more elastic. This is critical from a functional standpoint, because ligaments are connected at both ends to bones, whereas tendons attach on one end to muscle, a tissue with some elasticity.

### *Bursa*

Bursae are membranous sacs that contain small amounts of synovial fluid and serve to reduce friction. Common sites for bursa are in the tissues where friction develops, including the area between tendons and bones, tendons and skin, and ligaments and bones.

## Anatomical Properties of Joints

A joint is the site at which two bones connect. The study of joints is called **arthrology**. Joints can be classified by structure, function, or the number of axes present that permit motion.

### *Classification of Joints*

The structural classification of joints focuses on the material binding the bones together. In this case, joints are classified as fibrous, cartilaginous, or synovial. Functionally, joints are classified as synarthrodial (i.e., immovable joints), amphiarthrodial (i.e., slightly movable joints), or diarthrodial (i.e., freely movable joints). Human movement often is described in three dimensions based on a system of planes and axes. Based on movement potential, joints are classified as nonaxial (i.e., slipping movements only because the joint has no axis around which movement can occur), uniaxial (i.e., movement in one plane), biaxial (i.e., movement in two planes), or multiaxial (i.e., movement in or around three planes).

- **Fibrous Joints**

The fibrous joints of the bones are held together by fibrous tissue. The amount of movement at a fibrous joint (**synarthrosis**) depends on the length of the fibers uniting the bones. A fibrous joint can absorb shock, but it permits little or no movement of the articulating bones. **Sutures**, which are seen only in the skull, involve irregularly grooved, articulating bone sheets tightly bound by fibers that are continuous with the periosteum. **Syndesmoses** are joints that are joined by dense fibrous tissue that permit extremely limited motion. The tissue may be a ligament or a fibrous membrane (e.g., the inferior tibiofibular joint or the interosseous membrane between the interosseous borders of the radius and ulna). A unique joint, the **gomphosis** joint, is found between a tooth and the bone in its alveolus (socket) where the fibrous tissue of the periodontal ligament firmly anchors the tooth.

## Cartilaginous Joints

The cartilaginous joints (amphiarthroses) unite bones by either hyaline cartilage or fibrocartilage. The sternocostal joints and the epiphyseal plates before ossification are examples of a primary cartilaginous joint, or a **synchondrosis**, whereby the articulating bones are held together by a thin layer of hyaline cartilage. In a secondary cartilaginous joint, the articular surfaces of the bones are covered with hyaline cartilage, which in turn is fused to an intervening pad, or plate, of fibrous tissue or fibrocartilage. These are strong, slightly movable joints designed for strength and shock absorption. Examples of secondary cartilaginous joints are the pubic symphysis (**symphyses**), intervertebral joints, and the manubriosternal joint (between the manubrium and the body of the sternum).

## Synovial Joints

From a functional perspective, synovial joints (**diarthroses**) are the most common and most important type of joint. They normally provide free movement between the articulating bone surfaces. Freely movable joints predominantly are seen in the limbs, whereas immovable and slightly movable joints largely are restricted to the axial skeleton. Diarthrodial joints are classified according to their shape, which dictates the type and range of motion

permitted. The classifications are as follows:

- **Plane.** The articulating surfaces are nearly flat, and the only movement permitted is nonaxial gliding or short slipping movement. Examples include the intermetatarsal, intercarpal, and facet joints of the vertebrae.
- **Hinge.** One articulating bone surface is concave, and the other is convex. Strong collateral ligaments restrict motion to a single plane (uniaxial). Hinge joints permit flexion and extension only and can be seen at the elbow and the interphalangeal joints.
- **Pivot.** A rounded or conical end of one bone rotates within a sleeve or ring composed of bone (and, possibly, ligaments), allowing the uniaxial rotation of one bone around its own long axis or against another. The atlantoaxial joint and both the proximal and distal radioulnar joints are examples of this type of joint.
- **Condyloid.** The oval (ellipsoidal) articular surface of one bone fits into a reciprocal concavity of another. These biaxial joints permit all angular motions, namely, flexion, extension, abduction, adduction, and circumduction. The key characteristic of these joints is that both articulating surfaces are oval. The radiocarpal (wrist) joint and the metacarpophalangeal (knuckle) joints are typical condyloid joints.
- **Saddle.** These biaxial joints resemble a condyloid joint; however, saddle joints allow greater freedom of movement. Each articular surface has both concave and convex areas; that is, it is shaped like a saddle. The carpometacarpal joint of the thumb is an example of this type of joint.
- **Ball-and-socket.** The spherical or hemispherical head of one bone articulates with the cuplike socket of another. These joints are multiaxial and the most freely moving synovial joints in that they permit universal movement in all axes and planes, including rotation. Examples include the shoulder and hip joints.

## *General Structure of Synovial Joints*

Diarthrodial joints are distinguished by five features (Fig. 10.9):

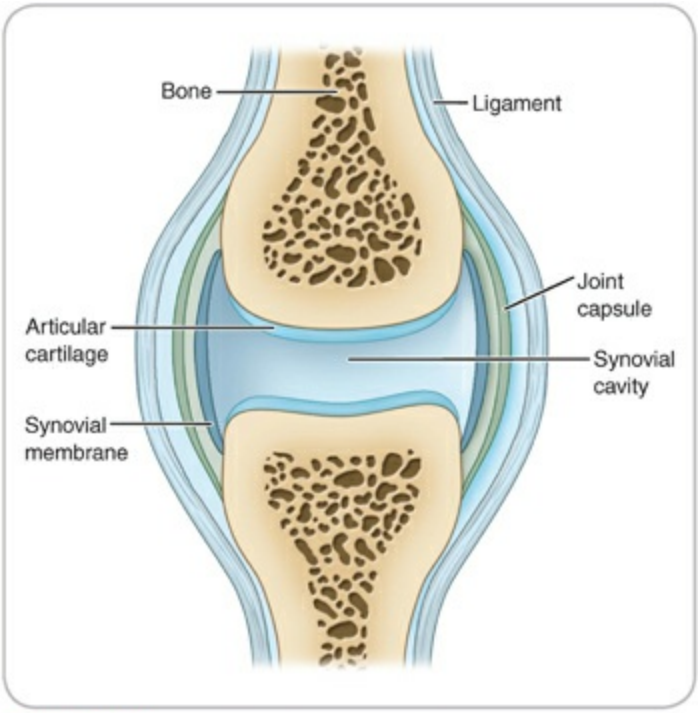



**Figure 10.9. Joint capsule.** Synovial joints have articular cartilage, a joint (synovial) cavity, an articular capsule, a synovial membrane, synovial fluid, and reinforcing ligaments.

1. **Articular cartilage.** Glassy-smooth hyaline cartilage covers the ends of the bony surfaces. These cushions absorb compression placed on the joint and thereby protect the bone ends from being crushed. The cartilage has no nerves or blood vessels; it is nourished by the synovial fluid covering its free surface. The nutrients in the synovial fluid come from the capillaries in the synovial membrane.
2. **Joint (synovial) cavity.** Unique to synovial joints, the joint cavity is filled with synovial fluid.
3. **Articular capsule.** The joint cavity is enclosed by a double-layered capsule. The external layer is a tough, flexible, fibrous capsule that is continuous with the periosteum of the articulating bones. The capsule functions to help hold the bones of the joint in place. The inner layer is a synovial membrane composed of loose connective tissue, which covers all internal joint surfaces that are not hyaline cartilage. The synovial

membrane produces synovial fluid that lubricates the joint.

4. **Synovial fluid.** A small amount of synovial fluid occupies all free spaces within the joint capsule. This fluid is derived largely by filtration from blood flowing through the capillaries in the synovial membrane. Synovial fluid has a viscous, egg white consistency because of its content of hyaluronic acid secreted by cells in the synovial membrane, but it thins and becomes less viscous as it warms during joint activity. Synovial fluid also is found within the articular cartilages, providing a slippery, weight-bearing film that reduces friction between the cartilages. In a weeping action, the fluid is forced from the cartilages when a joint is compressed. As pressure on the joint is relieved, the synovial fluid seeps back into the articular cartilages like water into a sponge. Synovial fluid also contains phagocytic cells that clear the joint cavity of microbes and cellular debris.
5. **Reinforcing ligaments.** Synovial joints are reinforced by a number of ligaments. More commonly, the ligaments are intrinsic, or capsular; that is, they are thickened parts of the fibrous capsule. In some cases, ligaments may remain distinct and are found outside the capsule (extracapsular) or deep to it (intracapsular). Because intracapsular ligaments are covered with synovial membrane, they do not actually lie within the joint cavity (extrasynovial).

Some synovial joints have other distinguishing features besides those listed previously. **Articular disks** are present in some synovial joints where the articulating surfaces are incongruous (e.g., the articular disk of the wrist joint). These fibrocartilaginous pads help to protect and hold the bones together. In some joints, they are attached to only one of the bones (e.g., the menisci in the knee). Articular disks have no nerves except at the attached margins. Some synovial joints have a fibrocartilaginous ring, called a **labrum**, which deepens the articular surface of one of the bones (e.g., the acetabular labrum in the hip joint). In other synovial joints, a tendon passes within the capsule of the joint (e.g., the long head of the biceps brachii muscle runs within the shoulder joint capsule).

## Classification of Skin Injuries

Forces applied to the body in different ways and from different directions result in different types of injury. Because the skin is the body's first layer of defense against injury, it is the most frequently injured body tissue.

Abrasions are common, such as minor skin injuries caused by shear when the skin is scraped with sufficient force, usually in one direction, against a rough surface. The greater the applied force, the more layers of skin are scraped away.

Blisters are minor skin injuries caused by repeated application of shear in one or more directions, as happens when a shoe rubs back and forth against the foot. The result is the formation of a pocket of fluid between the epidermis and dermis as fluid migrates to the site of injury.

Skin bruises are injuries resulting from compression sustained during a blow. Damage to the underlying capillaries causes the accumulation of blood within the skin.

Incisions, lacerations, avulsions, and punctures are breaks in the skin resulting from injury. An incision is a clean cut produced by the application of a tensile force to the skin as it is stretched along a sharp edge. A laceration is an irregular tear in the skin that typically results from a combination of tension and shear. An avulsion is a severe laceration that results in the complete separation of the skin from the underlying tissues. A puncture wound results when a sharp, cylindrical object penetrates the skin and underlying tissues with tensile loading.

## Classification of Muscle/Tendon Injuries

**Muscle contusions** result from a direct compressive force sustained from a heavy external blow, such as an opponent's knee. These acute direct muscle injuries may not lead to any structural damage to the muscle itself and vary in severity in accordance with the contact force, the contraction state of the affected muscle at the moment of injury, and the area and depth over which blood vessels are ruptured (Box 10.2). **Ecchymosis**, or tissue discoloration, may be present if the hemorrhage is superficial. As blood and lymph flow into

the damaged area in a diffuse or circumscribed (**hematoma**) manner, swelling occurs, which can compress muscle fibers causing pain and loss of motion. The most frequently injured muscles are the rectus femoris and vastus intermedius, which lie next to the femur and have limited space for movement when exposed to a direct blow. Complications of contusions may involve acute compartment syndrome, active bleeding, or large hematomas.

### BOX 10.2 Signs and Symptoms of Contusions

- Mechanism is an acute direct compressive force from a heavy external blow.
- Pain is localized over the injury site.
- Ecchymosis may be present if the hemorrhage is superficial.
- Bleeding and lymph flow may be diffuse or circumscribed (hematoma) which can limit range of motion.
- Swelling may compress nerves, leading to pain and temporary paralysis.

Muscle contusions are rated in accordance with the extent to which associated joint range of motion is impaired (Table 10.1). A first-degree contusion causes little or no restriction in the range of movement, a second-degree contusion causes a noticeable reduction in range of motion, and a third-degree contusion causes severe restriction of motion. With a third-degree contusion, the fascia surrounding the muscle may be ruptured, causing swollen muscle tissues to protrude.

**TABLE 10.1 Classifications of Contusions**

| | FIRST DEGREE | SECOND DEGREE | THIRD DEGREE |
|---|---|---|---|
| Damage to tissue | Superficial tissues are crushed. | Superficial and some deep tissues are crushed. | Deeper tissues are crushed (fascia surrounding muscle may rupture, allowing swollen tissues to protrude). |
| Weakness | None | Mild to moderate | Moderate to severe |
| Muscle spasm | None | None | Possible |
| Loss of function | Mild | Moderate | Severe |
| Ecchymosis | Mild | Moderate | Severe |
| Swelling | Mild | Moderate | Severe |
| Range of motion | No restriction | Decreased | Significantly decreased because of swelling |

Traumatic injury to muscles and tendons, termed **strains**, are indirect injuries, that is, stretch induced caused by sudden forced lengthening over the viscoelastic limits of muscles during a powerful contraction. The likelihood of strains depends on the magnitude of the force and the structure's cross-sectional area. The greater the cross-sectional area of a muscle, the greater its strength, meaning it can produce more force and translate that force to the attached tendon. In a similar manner, the larger the cross-sectional area of the tendon, the greater the force it can withstand. The increased cross-sectional area translates to reduced stress. Because tendons are stronger than their attached muscle, the muscle portion of the musculotendinous unit almost always ruptures first. This area is associated with a biomechanically weak point because the muscle cross-sectional area is smallest. The injury can produce rupturing of tissue and subsequent hemorrhage and swelling (Box 10.3). A tendon begins to develop tears when it is stretched approximately 8% to 10% beyond its normal length.[1] The highly vascular paratenon is susceptible to inflammation, more commonly at the tendon's bony attachment.

## BOX 10.3 Signs and Symptoms of Strains and Sprains

### Strains

- History of acute onset is present.
- Mechanism of injury results from overstretch or overload.
- Pain is localized over the injury site, which tends to be at or near a musculotendinous junction.

- Discoloration, in severe cases, is caused by blood pooling distal to the site of trauma.
- If moderate, muscle weakness is evident.

**Sprains**

- History of acute onset is present.
- Mechanism of injury may result from overstretch or overload.
- Pain is localized over the injury site.
- Joint instability is detectable, if assessed before joint effusion.
- If severe, injury may result in subluxation or dislocation of the joint.

The terminology and classification of muscle injuries in sport is in flux. In 2013, the Munich Consensus Statement was published reclassifying traditional grading of muscle injuries based on functional or structural disorders.[2] However, the recommendations have not been universally accepted. For athletic trainers, the traditional grading system is currently being followed. Strains are graded by the extent of anatomical damage as first, second, and third degree (**Table 10.2**). First-degree strains involve only microtearing of the collagen fibers anywhere along the muscle-tendon-bone unit, although most injuries are located at the muscle–tendon junction. These partial tears have a maximum diameter of less than a muscle fascicle/bundle tear and are characterized by mild pain and local tenderness but may present with no readily observable symptoms and no loss of function. Second-degree or moderate injuries involve muscle tears greater than a muscle fascicle/bundle and are characterized by moderate pain, muscle weakness, and some loss of function. A simultaneous injury to the external perimysium, which may serve as an intramuscular barrier function in case of bleeding, may also be used to differentiate a moderate from a minor partial muscle tear. The majority of partial muscle tears heal without scar formation, whereas greater muscle tears can result in a fibrous scar.[2]

**TABLE 10.2 Classifications of Strains**

| | FIRST DEGREE | SECOND DEGREE | THIRD DEGREE |
|---|---|---|---|
| Tears to muscle | < A muscle fascicle/ bundle | > A muscle fascicle/ bundle | Most or all muscle fibers are torn (rupture) or a tendinous avulsion is seen. |
| Weakness | Mild | Moderate to severe (reflex inhibition) | Moderate to severe |
| Muscle spasm | Mild | Moderate to severe | Moderate to severe |
| Loss of function | Mild | Moderate to severe | Severe (reflex inhibition) |
| Swelling | Mild | Moderate to severe | Moderate to severe |
| Palpable defect | No | No | Yes (if early) |
| Pain on contraction | Mild | Moderate to severe | None to mild |
| Pain with stretching | Yes | Yes | No |
| Range of motion | Decreased | Decreased on swelling | May increase or decrease depending on swelling |

Third-degree injuries produce a major loss of tissue continuity that results in a significant loss of function or movement. Total muscle tears where the continuity of the whole muscle is disrupted are rare. More frequently, a *subtotal* muscle tear or tendinous avulsion is seen. Clinical experience shows that injuries involving more than 50% of the muscle diameter (subtotal tears) usually have a similar healing time compared with complete tears.[2] Tendinous avulsions are included here because they mean biomechanically that a total tear of the proximal or distal attachment of the muscle has occurred. The most frequently involved locations are the proximal rectus femoris, the proximal hamstrings, the proximal adductor longus, and the distal semitendinosus.[2] In third-degree injuries, tearing of muscle tissue can damage small blood vessels, which may present as swelling and ecchymosis, particularly if the damage is superficial rather than deep. Severe pain may be followed by decreased pain attributed to nerve separation.

Two muscle conditions related to overexertion include *fatigue-induced muscle disorders* and *delayed-onset muscle soreness (DOMS)*. Muscle fatigue is known to predispose an individual to injury. Fatigued muscles absorb less energy in the early stages of stretch as compared to nonfatigued muscles and have increased stiffness, which can also predispose an individual to subsequent injury.[3] DOMS occurs several hours after unaccustomed deceleration movement (eccentric contractions), whereas fatigue-induced muscle disorders occur during activity. DOMS has characteristic acute inflammatory pain with stiff, weak muscles and pain at rest and usually

resolves within 1 week. Fatigue-induced muscle disorders are characterized by an aching, circumscribed firmness, dull ache to stabbing pain that increases with activity. If unrecognized, the pain can persist for a longer period of time and lead to structural injuries such as partial tears.[2]

Although typically not associated with injury, muscle **cramps** and **spasms** are painful involuntary muscle contractions common in sport. A cramp is a painful, involuntary contraction that may be **clonic**, with alternating contraction and relaxation, or **tonic**, with continued contraction over a period of time. Cramps appear to be brought on by a biochemical imbalance, sometimes associated with muscle fatigue. Exercise-associated muscle cramps (EAMCs) are a common condition experienced by recreational and competitive athletes. Despite their commonality and prevalence, their cause remains unknown. Theories for the cause of EAMC are primarily based on anecdotal and observational studies rather than sound experimental evidence. Without a clear cause, treatments and prevention strategies for EAMC are often unsuccessful.[4] A muscle spasm is an involuntary contraction of short duration caused by a reflex action that can be biochemically derived or initiated by a mechanical blow to a nerve or muscle.

**Myositis** and **fasciitis** refer, respectively, to inflammation of a muscle's connective tissues and inflammation of the sheaths of fascia surrounding portions of muscle. These are chronic conditions that develop over time as the result of repeated body movements that irritate these tissues.

**Tendinopathy** refers to any tendon pathology. Because tendons lack a good blood supply, many tendons lack a direct inflammatory response (**tendinitis**). Instead, degenerative changes result (**tendinosis**). Although both conditions may be present simultaneously, tendinosis is far more common than tendinitis. Because neither of these two conditions can be verified without histopathological examination, the term tendinopathy is preferred.[1] Tendinopathy is characterized by pain and swelling with tendon movement (Box 10.4).

## BOX 10.4 Signs and Symptoms of Tendinopathy

- History of chronic onset is present.
- Mechanism of injury is caused by overuse or by repetitive overstretching or overload.
- Pain exists throughout the length of the tendon and increases during palpation.
- Swelling may be minor to major, and thickening of the tendon may be present.
- Crepitus may be present.
- Pain occurs at the extremes of motion during passive and active ranges of motion.
- Pain increases during stretching and resisted range of motion; strength decreases with pain.

**Tenosynovitis** denotes inflammation of the synovial sheath surrounding a tendon and is common in the hands and feet. Tenosynovitis may be acute or chronic. Acute tenosynovitis is characterized by a grating sound (crepitus) with movement, inflammation, and local swelling. Chronic tenosynovitis has the additional symptom of nodule formation in the tendon sheath. Not all tendons are encased in a synovial sheath. Some tendons have a peritendinous layer of thick tissue around the tendon. Inflammation of these tendons is called **peritendinitis**. Long-term tendinopathy can lead to the accumulation of mineral deposits resembling bone in the affected tissues, a process known as **ectopic calcification**. Accumulation of mineral deposits in muscle is known as myositis ossificans. A common site for this condition is the quadriceps region. The muscle typically is very tender, and as the ossificans develops, a hardened mass can be palpated within the muscle mass. In tendons, the condition is called **calcific tendinopathy**.

Overuse injuries may result from intrinsic factors (e.g., a malalignment of limbs, muscular imbalances, other anatomical factors) or extrinsic factors (e.g., training errors, faulty technique, incorrect surfaces and equipment, poor environmental conditions). In general, overuse injuries are classified in four

stages based on pain and dysfunction:

**Stage 1:** pain after activity only

**Stage 2:** pain during activity that does not restrict performance

**Stage 3:** pain during activity that restricts performance

**Stage 4:** chronic, unremitting pain even at rest

## Joint Injury Classifications

Sprains are acute traumatic injury to ligaments. Abnormally high tensile forces produce a stretching or tearing of tissues that compromise the ability of the ligament to stabilize the joint. The tissue tearing also results in the flow of blood and lymph into the damaged area, producing swelling and restricting range of motion.

Sprains are categorized as first, second, and third degree (Table 10.3). First-degree sprains involve only microtearing of the collagen fibers. Signs and symptoms include mild discomfort, mild point tenderness, minimal or no swelling, and minimal or no loss of function. Second-degree injuries involve tearing of nearly half the ligament fibers, which results in a moderate loss of function and detectable joint instability. They are characterized by moderate pain, moderate swelling, and ecchymosis. Third-degree sprains produce a major loss of tissue continuity that results in a significant loss of function, severe instability, and severe pain.

**TABLE 10.3 Classifications of Sprains**

| | FIRST DEGREE | SECOND DEGREE | THIRD DEGREE |
|---|---|---|---|
| Damage to ligament | Few fibers of ligament are torn. | Nearly half of fibers are torn. | All ligament fibers are torn (rupture). |
| Distraction with | <5-mm distraction | 5–10-mm distraction | >10-mm distraction stress tests |
| Weakness | Mild | Mild to moderate | Mild to moderate |
| Muscle spasm | None | None to minor | None to minor |
| Loss of function | Mild | Moderate to severe | Severe (instability) |
| Swelling | Mild | Moderate | Moderate to severe |
| Pain on contraction | None | None | None |
| Pain with stretching | Yes | Yes | No |
| Range of motion | Decreased | Decreased on swelling; dislocation or subluxation possible | May increase or decrease depending on swelling; dislocation or subluxation possible |

A dislocation is a traumatic injury that occurs when the bones that comprise a joint are forced beyond their normal position, resulting in the displacement of one joint surface on another. A partial or incomplete dislocation is called a **subluxation**. The resultant damage includes rupturing of the joint capsule and ligaments as well as potential tearing of surrounding muscle–tendon units. In addition, many acute dislocations have an associated fracture or nerve injury. Signs and symptoms associated with a dislocation include pain, swelling, point tenderness, deformity, and loss of limb function.

Because of extensive stretching of the connective tissues surrounding a joint associated with a traumatic dislocation, susceptibility to chronic or recurrent dislocations is increased. Less force is required to sustain a recurrent dislocation. Whereas recurrent dislocations may be less painful, the subsequent damage to joint structures can be extensive and may lead to chronic joint problems. The most common sites for dislocations are the fingers and the glenohumeral joint of the shoulder.

Osteoarthritis is a type of arthritis attributed to the degeneration of the articular cartilage in a joint. Individuals with osteoarthritis experience pain and limited movement at the involved joint. Osteoarthritis has no definitive cause; rather, it is attributed to a combination of factors, including stresses sustained during certain types of physical activity, joint trauma, and the aging process. It is one of the leading causes of disability among American adults. It should be noted, however, that physical activity is being promoted as a potential strategy for managing arthritis.[5]

**Bursitis** involves irritation of one or more bursae. It may be acute or chronic, depending on whether it is brought on by a single traumatic compression or by repeated compressions associated with overuse of the joint. Local swelling of a bursa can be very pronounced, particularly at the olecranon bursa of the elbow and the prepatellar bursa of the knee. An inflamed bursa is typically swollen, point tender, and can be warm to the touch.

The runner’s symptoms suggest a stage 3 overuse injury. He should be advised to stop running until a complete assessment can be performed.

Because the pain is only present on the left leg the assessment should include attention to both intrinsic and extrinsic factors that could contribute to injury.

## SOFT-TISSUE HEALING

An assessment of the middle-aged male runner suggests a possible lower leg strain. Clearly identification of the type of injury is advantageous in making decisions concerning management but identification is only part of the process. Why is an understanding of the healing of soft tissue essential in determining the management of injury?

The reparative process for injured soft tissues involves a complex series of interrelated physical and chemical activities. Because the normal healing process takes place in a regular and predictable fashion, knowledge of the various signs and symptoms exhibited at the injury site is essential for monitoring the progress of healing. Ultimately, this information is critical to decisions regarding appropriate rehabilitation and return to play decisions.

Healing of soft tissues is a three-phase process involving inflammation, proliferation, and maturation. Although it is useful to discuss the healing phenomenon in terms of these different stages, it should be recognized that these processes usually overlap, both spatially and temporally, within injured tissues.

### Inflammatory Phase (Days 0 to 6)

The familiar symptoms of inflammation have long been recognized and, in fact, were documented by early Greek and Roman physicians as rubor (redness), calor (local heat), tumor (swelling), dolor (pain), and, in severe cases, functio laesa (loss of function). Although inflammation can be produced by an adverse response to chemical, thermal, and infectious agents, the focus of this explanation is the characteristic course of the inflammatory response following

injury.

Depending on the nature of the causative forces, inflammation can be acute or chronic. Acute inflammatory response is of relatively brief duration and involves a characteristic hemodynamic activity that generates **exudate**, a plasmalike fluid that exudes out of tissue or its capillaries and is composed of protein and granular leukocytes (white blood cells). Alternatively, chronic inflammatory response is of prolonged duration and is characterized by the presence of nongranular leukocytes and the production of scar tissue.

The beginning of the acute inflammatory phase involves the activation of three mechanisms that act to stop blood loss from the wound. The first mechanism involves local vasoconstriction that lasts from a few seconds to as long as 10 minutes. Larger blood vessels constrict in response to signals from neurotransmitters, and the capillaries and smaller arterioles and venules constrict because of the influence of serotonin and catecholamines released from the platelets and serum during injury. The resulting reduction in the volume of blood flow in the region promotes increased blood viscosity or resistance to the flow, which further reduces blood loss at the injury site.

A second response to the loss of blood is the platelet reaction. The platelet reaction provokes clotting as individual cells irreversibly combine with each other and with fibrin to form a mechanical plug that occludes the end of a ruptured blood vessel. As the fibrin forms the blood clot or scab, this covering mechanism on the skin surface protects the underlying tissues from harmful bacterial invasion. The platelets also produce an array of chemical mediators that play significant roles in the inflammatory and proliferation phases of healing. These mediators include serotonin, adrenaline, noradrenaline, and histamine, all of which are primary agents in the inflammatory response. The enzyme adenosine triphosphatase, which is central in supplying the energy needed for healing, also is found in platelets.

The third response is the activation of the coagulation cascade. A cascade is a heightened physiological response consisting of several different, interrelated processes. Fibrinogen molecules are converted into fibrin for clot formation through two different pathways. The extrinsic pathway is activated by thromboplastin, which is released from damaged tissue. The intrinsic

pathway, inside the blood vessels, is enabled by the interaction between platelets and the Hageman factor. Both paths result in the formation of prothrombin activator, which converts prothrombin into thrombin.

Following vasoconstriction, vasodilation is brought on by a local axon reflex and the complement and kinin cascades. In the complement cascade, approximately 20 proteins that normally circulate in the blood in the inactive form become active to promote a variety of activities essential for healing. One process activated is the attraction of neutrophils and macrophages to rid the injury site of debris and infectious agents through **phagocytosis**. As the flow of blood to the injured area slows, these cells are redistributed to the periphery, where they begin to adhere to the endothelial lining. The movement of a neutrophil from the circulation into tissue is called **diapedesis**. Mast cells and basophils also are stimulated to release histamine, further promoting vasodilation. The kinin cascade provokes the conversion of the inactive enzyme kallikrein to the activated bradykinin in both blood and tissue. Bradykinin promotes vasodilation and increases permeability of the blood vessel wall, contributing to the formation of tissue exudate.

In addition, increased blood flow to the region causes swelling. Blood from the broken vessels and damaged tissues forms a hematoma, which in combination with necrotic tissue, forms the **zone of primary injury**.

Approximately 1 hour postinjury, swelling, or **edema**, occurs as the vascular walls become more permeable, and increased pressure within the vessels forces a plasma exudate out into the interstitial tissues (**Fig. 10.10**). This increased permeability or porosity of the blood vessel walls typically exists for only a few minutes in cases of mild trauma, with a return to normal permeability in 20 to 30 minutes. More severe trauma can result in a prolonged state of increased permeability and, sometimes, in delayed onset of increased permeability, with swelling not becoming apparent until sometime after the original injury. The tissue exudate provides a critically important part of the body's defense, both by diluting toxins present in the wound and by enabling delivery of the cells that remove damaged tissue and enable reconstruction.

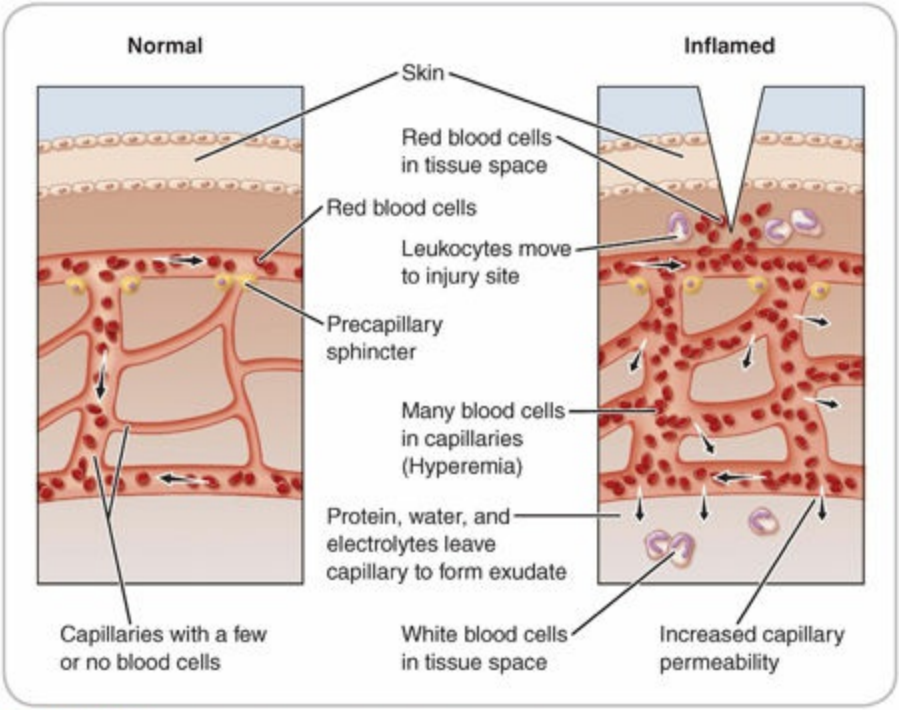



**Figure 10.10. Acute inflammatory process.** Edema forms when histochemical agents open the pores in the vascular walls, allowing plasma to migrate into the interstitial space.

The complement and kinin cascades act to speed the arrival of reparative cells in the exudate. **Mast cells** are connective tissue cells that carry heparin, which prolongs clotting, and histamine. Platelets and basophil leukocytes also transport histamine, which serves as a vasodilator and increases blood vessel permeability. The leukocytes release enzymes that interact with phospholipids in the cell membranes to produce arachidonic acid. Arachidonic acid activates further inflammation of the affected cells through the production of chemical mediators, including prostaglandins and leukotrienes. Bradykinin, a major plasma protease present during inflammation, increases vessel permeability and stimulates nerve endings to cause pain. This chain of chemical activity produces the **zone of secondary injury**, which includes all the tissues affected by inflammation, edema, and hypoxia. After the debris and waste products from the damaged tissues are ingested through phagocytosis, the leukocytes reenter the bloodstream, and the acute inflammatory reaction subsides.

## The Proliferative Phase (Days 3 to 21)

The proliferative phase involves repair and regeneration of the injured tissue and takes place from approximately 3 days following the injury through the next 3 to 6 weeks, overlapping the later part of the inflammatory phase. The proliferative processes include the development of new blood vessels (angiogenesis), fibrous tissue formation (fibroplasia), generation of new epithelial tissue (reepithelialization), and wound contraction.

This stage begins when the size of the hematoma is sufficiently diminished to allow room for the growth of new tissue. Although the skin has the ability to regenerate new skin tissue, the other soft tissues replace damaged cells with scar tissue.

Healing through scar formation begins with the accumulation of exuded fluid containing a large concentration of protein and damaged cellular tissues. This accumulation forms the foundation for a highly vascularized mass of immature connective tissues that include fibroblasts, which are capable of generating collagen. The fibroblasts begin to produce immature collagen through the process known as fibroplasia.

Fibroplasia and angiogenesis are interdependent processes, with the deposition of the new connective tissue matrix fueled by nutrients from the blood supply and the newly forming blood vessels reliant on mechanical support and protection from the matrix. The developing connective tissue at the wound site is primarily types I and III collagen, cells, blood vessels, and a matrix containing glycoproteins and proteoglycans. Type III collagen is particularly useful at this stage because of its ability to rapidly form cross-links that contribute to stabilization at the wound site. Fibroblasts are chemically drawn to the region that secretes the collagen. The enzymatic reactions involved in collagen production are dependent on specific concentrations of oxygen, ascorbate, ferrous ions, and lactate within the microenvironment of the wound.

Fibroblasts also produce attachment factors that promote angiogenesis by helping the growing vessels attach to basement membrane collagen. The primary driving force for angiogenesis, however, comes from the platelet response and the hypoxic wound environment. New vessel formation begins with the activation of enzymes by a potent growth factor, which acts on the

existing vessels to dissolve their basement membranes and liberate endothelial cells. These cells are then chemically drawn to hypoxic sites within a wound, where they fuse and form new vessels.

Other characteristics of the proliferation stage include an increase in the number of blood vessels present, increased water content in the injury zone, and reepithelialization at the surface caused by epithelial cells migrating from the periphery and toward the center of the wound.

## Maturation Phase (Up to 1+ Year)

The final phase of soft-tissue wound repair is known as the maturation, or remodeling, phase. This period involves maturation of the newly formed tissue into scar tissue. The associated processes include decreased fibroblast activity, increased organization of the extracellular matrix, decreased tissue water content, reduced vascularity, and a return to normal histochemical activity. In soft tissue, these processes begin approximately 3 weeks postinjury, overlapping the proliferative phase. Types I and III collagen continue to increase, replacing immature collagen and resulting in contraction of the wound.

Although the epithelium typically has regenerated completely by 3 to 4 weeks postinjury, the tensile strength of the wound at this time is only approximately 25% of normal.[6] After several more months, strength may still be as much as 30% below the preinjury level.[7] This is partly because of the orientation of the collagen fibers, which tends to be more vertical during this period of time than in normal tissue, where their orientation usually is horizontal. The collagen turnover rate in a newly healed scar also is very high, so failure to provide appropriate support for the wound site can result in a larger scar. Excessive scar tissue is called keloid tissue.

Because scar tissue is fibrous, inelastic, and nonvascular, it is less strong and less functional than the original tissues. In addition, the development of the scar typically causes the wound to shrink, resulting in decreased flexibility of the affected tissues following the injury. This explains why someone, such as a person with paraplegia who has a healed pressure sore from a wheelchair seat

in poor condition, can easily redevelop breakdown in the same area if the pressure stresses reoccur in that same area.[8]

Remodeling continues for a year or more as collagen fibers become oriented along the lines of mechanical stress to which the tissue usually is subjected. The tensile strength of scar tissue may continue to increase for as long as 2 years postinjury. **Box 10.5** summarizes the three stages of the healing process. **Figure 10.11** demonstrates the injury healing timeline schematic.

## BOX 10.5 Phases of Soft-Tissue Wound Healing

### Inflammation (0–6 Days)

- Vasoconstriction promotes increased blood viscosity (thickness), reducing blood loss through bleeding.
- The platelet reaction initiates clotting and releases growth factors that attract reparative cells to the site.
- The coagulation cascade affects clot formation.
- The complement and kinin cascades provoke vasodilation and increase blood vessel wall permeability, facilitating the migration of neutrophils and macrophages in plasma exudate to cleanse the site through phagocytosis.

### Proliferation (3–21 Days)

- Fibroblasts produce a supportive network of types I and III collagen.
- The platelet response and hypoxic wound environment stimulate angiogenesis.
- Epithelial cells migrate from the periphery toward the center of the wound to enact reepithelialization.

### Maturation (Up to 1+ Years)

- Fibroblast activity decreases and habitual loading produces increased organization of the extracellular matrix.
- A return to normal histochemical activity allows for reduced vascularity

and water content.

- Types I and III collagen continue to proliferate, replacing immature collagen precursors and resulting in contracture of the wound.
- Scar tissue formation results in decreased size and flexibility of the involved tissues.
- Remodeling causes collagen fiber alignment along lines of habitual stress, with tensile strength increasing for up to 2 years postinjury.

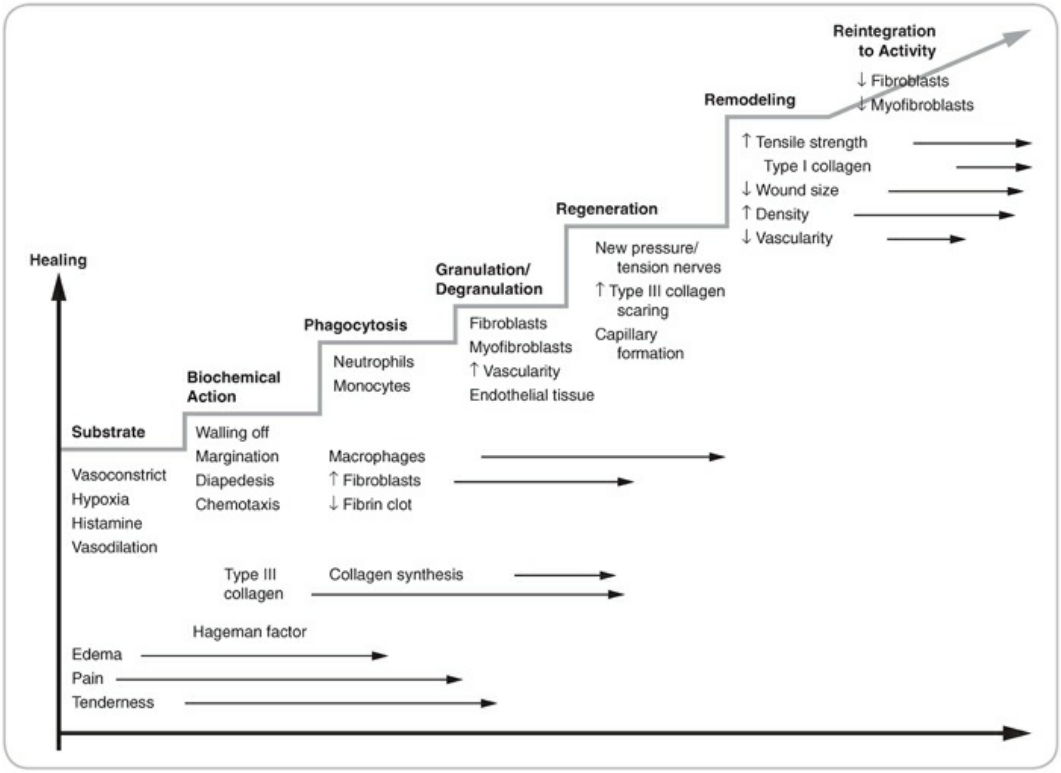



**Figure 10.11.** Injury healing timeline schematic.

Muscle fibers are permanent cells that do not reproduce or proliferate in response to either injury or training. Reserve cells in the basement membrane of each muscle fiber, however, are able to regenerate muscle fiber following injury. Severe muscle injury can result in scarring or the formation of **adhesions** within the muscle, which inhibits the potential for fiber regeneration from the reserve cells. Consequently, following severe injury, muscle may regain only approximately 70% of its preinjury strength.[9]

Because tendons and ligaments have few reparative cells, healing of these

structures is a slow process that can take more than a year. Regeneration is enhanced by proximity to other soft tissues that can assist with supplying the chemical mediators and building blocks required. For this reason, isolated ligaments, such as the anterior cruciate, have poor chances for healing.[10] If tendons and ligaments undergo abnormally high tensile stress before scar formation is complete, the newly forming tissues can be elongated. If this occurs in ligaments, joint instability may result.

Because tendons, ligaments, and muscles **hypertrophy** and **atrophy** in response to levels of mechanical stress, complete immobilization of the injury leads to atrophy, loss of strength, and a decreased rate of healing in these tissues. The amount of atrophy generally is proportional to the time of immobilization. As such, although immobilization may be necessary to protect the injured tissues during the early stages of recovery, strengthening exercises should be implemented as soon as appropriate during rehabilitation of the injury. Increased risk for reinjury exists as long as the strength of affected tissues is less than the preinjury level.

## The Role of Growth Factors

Growth factors are proteins that play crucial roles during all three phases of the healing process. Their functions include attracting cells to the wound, stimulating their proliferation, and directing the deposition of the extracellular matrix.

First discovered in the granules of platelets, platelet-derived growth factor (PDGF) also is released from macrophages, endothelial cells, vascular smooth cells, and fibroblasts. The presence of PDGF is one of the most important factors in the success of the first two stages of healing. PDGF acts as a chemical attractant for fibroblasts, neutrophils, and macrophages and also promotes the replication of fibroblasts and vascular smooth muscle cells. It also activates macrophages and stimulates fibroblasts to secrete types I and III collagen.

Another important growth factor, transforming growth factor–β, is actually a group of several different proteins with similar structural and chemical

properties. Transforming growth factor–β is produced by platelets, macrophages, bone cells, monocytes, and lymphocytes and is capable of both stimulating and inhibiting fibroblasts. Transforming growth factor–β also stimulates angiogenesis and accelerates collagen deposition.

Another group of proteins with a common high affinity for heparin is called basic fibroblast growth factor. The platelet-derived enzyme heparinase chemically separates this factor from heparin. Basic fibroblast growth factor stimulates proliferation of endothelial cells and is distributed in endothelial cells, macrophages, and fibroblasts. It is critically important in angiogenesis, releasing the basement membrane–degrading enzymes that separate endothelial cells before new vessel formation.

Two closely related growth factors are epidermal growth factor and transforming growth factor–α. Both originate from transmembrane proteins, and both act on the epidermal growth factor receptor. Whereas both of these growth factors facilitate the development of granulation tissue, transforming growth factor–α also regulates angiogenesis and promotes epidermal regrowth.[11]

Fortunately the normal healing process takes place in a regular and predictable fashion. Knowledge of the various signs and symptoms exhibited at the injury site from the time of injury and its subsequent progression is essential for monitoring the progress of healing and for making appropriate decisions regarding management of the injury. If the runner's condition is not managed properly it could result in both delayed and less-than-optimal healing exacerbation of the condition and development of additional injuries.

## SOFT-TISSUE WOUND CARE MANAGEMENT

The initial management of the runner's condition involves reducing pain and inflammation. Describe a strategy for accomplishing this goal.

Soft-tissue injuries may involve open wounds (e.g., abrasions, blisters, lacerations, and puncture wounds) or closed wounds (e.g., contusion, muscle tears, sprains, and bursitis). This section explains the immediate care of both broad categories of soft-tissue injuries.

## Care of Open Soft-Tissue Injuries

In providing wound care for open soft-tissue injuries, it is critical to follow universal precautions and infection control standards (**Box 10.6**). Wound care includes the use of effective cleansers to control bacteria that may lead to infection. Discussion must also include whether these cleaners are toxic to healthy cells. One study comparing the bactericidal effectiveness and cytotoxicity to human fibroblast cells of four common cleansers (i.e., Cinder Suds, Nitrotan, hydrogen peroxide, and Betadine) at various dilutions determined that only Betadine was effective against bacteria and not harmful to human fibroblast cells at a 1:10 dilution of the commercially purchased solution.[12] In addition to using safety equipment, disinfecting equipment and materials used during wound care, and properly discarding contaminated materials, several steps in general wound care should be followed:

- If possible, wash hands before beginning treatment.
- Apply gloves.
- Apply direct pressure to the wound with sterile gauze or a nonstick material.
- Cleanse the wound and the area around the wound (at least twice the size of the wound) with normal saline or potable tap water.[13]
- Dress and bandage the wound site securely for continued activity.
- Creams or ointments may or may not be used with occlusive dressings. If used, dressings should be covered beyond their borders with underwrap and elastic adhesive tape where possible.
- Change dressings as necessary and look for signs of infection (i.e., local heat, swelling, redness, pain, pus, and elevated body temperature).

- Maintain a moist wound environment for optimal healing.[13]

## BOX 10.6 General Guidelines for Preventing the Spread of Bloodborne Pathogens

- Latex gloves always should be worn. Other protective equipment that should be worn when blood and/or other bodily fluids could be splashed, spurted, or sprayed include the following:

  Eyewear/face guards

  Masks and/or protective guards

  Gowns or aprons
- Following any exposure to potentially infectious material, immediately wash and disinfect hands and other skin surfaces.
- Clean large spills of bodily fluids and bloodborne pathogens by flooding the contaminated area with disinfectant prior to removing the spill. Following removal, the area should be disinfected again and thoroughly scrubbed.
- Disinfect all horizontal surfaces (i.e., treatment tables, taping tables, work space, and floors) regularly, after each use, and immediately after spills or soiling occurs. Use a scrubbing process.
- Disinfect with a cleaning solution of 1:10 to 1:100 solution (bleach to water). Caution should be used when using this solution near therapeutic modalities or skin because of the caustic and corrosive properties of bleach. The solution must be mixed daily to be effective.
- Soiled linens and towels should be separated from regular laundry, handled with gloves, and placed in a leak-proof bag that is visibly designated for biohazard items.
- All items should be washed with detergent and water for 25 minutes at a minimum of 71°C (160°F). Disinfectant solution such as chlorine bleach can provide an extra margin of safety in low-temperature washing and transporting laundry inside and outside health care facilities.

- All disposable contaminated products (e.g., gauze, paper towels, cotton) should be handled with gloves and placed in leak-proof biohazard bags.
- Sharps containers should be readily available, leak-proof, puncture-resistant, red in color, and visibly designated with a biohazard sign. Reusable sharps, such as pointed scissors or tweezers, should be sterilized after each use.
- Disposal of contaminated items and sharps containers should be in compliance with OSHA standards.

*OSHA*, Occupational Safety and Health Administration.

See **Box 10.6**: **General Guidelines for Preventing the Spread of Bloodborne Pathogens** on the companion Web site at thePoint.

Wounds must be covered with an appropriate dressing. Proper dressings have several essential functions in that they (1) should absorb the wound exudate, (2) should not stick to the wound surface, (3) should not create a new injury to the patient (e.g., through allergy or macerations of the surrounding skin), and (4) should support the injured part.[14] The decision about what type of dressing to use depends on the location, size, type/depth of wound, amount of drainage/exudate expected, presence of infection, need for debridement, and expected frequency and difficulty in dressing change, costs, and patient comfort.[8]

Common gauze dressings are relatively inexpensive, but they must be changed frequently, which increases its cost factor. Dry gauze prevents contamination, but it can lock in exudate and does not keep the wound moist. Wet gauze strips are used to pack wounds with tunnels or fistulas to aid in drainage. Nonadherent gauze, such as Telfa and Xeroform, absorbs some drainage or allows drainage through it but prevents any outer gauze dressing from adhering to the wound or sutures/staples and, thereby, prevents further injury when the dressing is removed.[8]

Several choices exist beyond the traditional gauze dressing, including

hydrocolloids, hydrogels, transparent films, collagen, alginates, foams, and antimicrobials. Hydrocolloids are indicated for use on partial- and full-thickness wounds with or without necrotic tissue and are useful on areas that require contouring, such as the heels or ears. Hydrogel dressings, made commonly with a glycoprotein base, hydrate to contribute to bulk absorbency and act as a semipermeable membrane to water vapor and for gas exchange. A hydrogel membrane, Xenaderm, which was developed and manufactured in New Zealand, is absorbent to 7 times its own weight in plasma, is semipermeable to allow water vapor loss and gas exchange, and is translucent so that the progress of the wound can be observed.[14] Hydrogel dressings may be used on wounds with necrotic tissue, such as minor burns or tissue damaged by radiation. Transparent films are used on partial-thickness wounds with little or no exudate, on wounds with necrosis, or as a primary or secondary dressing over lacerations, abrasions, and second-degree burns. Collagen dressings typically are indicated for pressure ulcers, venous ulcers, surgical wounds, diabetic ulcers, abrasions, second-degree burns, and traumatic wounds, but they usually require a secondary dressing. Alginates are seaweed derivatives similar in nature to hydrogels but often manufactured in sheet or fiber forms that hydrate and act as semipermeable barriers to water vapor.[14] Alginates are indicated for wounds with moderate-to-heavy exudate, such as overinfected wounds, diabetic ulcers, and pressure ulcers, and also require a secondary dressing. Foam dressings have small, open cells capable of holding fluids. The area of the dressing directly over the wound is nonadherent (for easy removal) and is available with an adhesive border that can act as a bacterial barrier. Antimicrobial dressings are indicated for wounds that would benefit from topical antimicrobial agents. These dressings reduce the risk of infection in wounds, sites with percutaneous lines, and surgical incisions.[15] In all cases, regardless of the dressing selected, a moist—not wet—environment is best to ensure adequate healing.[16]

Individuals who have diabetes, wound contamination with a foreign material, a wound length greater than 2 in (5 cm), and wounds on the lower extremity increase the risk factors for wound infection.[17] Sutures may be necessary if the laceration exposes the full dermis. The so-called “golden

period" of laceration care that necessitated suture application within 6 to 10 hours from the time of the initial injury to reduce the risk of infection appears to not be as important as previously thought. Improvements in irrigation and decontamination of open wounds have led to successful healing in lacerations older than 12 hours on a variety of sites.[17]

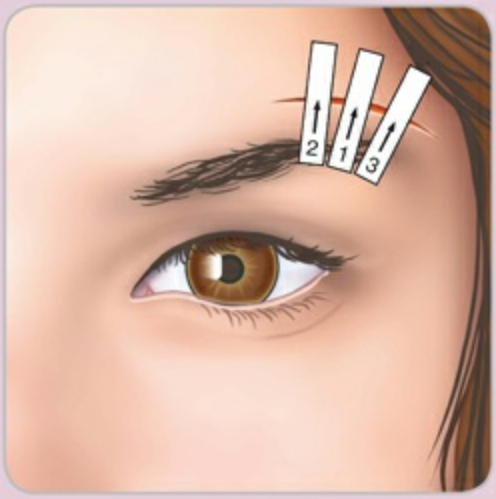
Application Strategy 10.1 explains the basic care for common skin injuries. It is assumed that the clinician is already gloved. These techniques can be adapted for use with other open wounds as well.

## APPLICATION STRATEGY 10.1

### Care of Open Wounds

**Abrasions**

1. Clean and remove visible contaminants with a fluid flush using water and sweeps of gauze.
2. Clean the wound site and area around the wound with normal saline or potable tap water.
3. Dress and bandage the wound securely for continued play.
4. For dirty abrasions, or when it has been at least 5 years since a tetanus booster, refer for medical care.




**Blisters**

1. Clean both the wound site and the area around the wound with normal saline or potable tap water.
2. If a blister isn't too painful, try to keep it intact. Unbroken skin over a blister provides a natural barrier to bacteria and decreases the risk of infection.
3. Cover the area with a topical triple antibiotic and a dry, sterile dressing. Do *not* aspirate a blood-filled blister unless it will open during activity possibly leading to infection.
4. If the blister is large and subject to continued compression, use a small, sterile needle to aspirate the clear fluid, or incise the skin of clear blisters according to physician protocol.
5. Once the fluid is removed, cleanse the area again with normal saline or potable tap water.
6. Pad the nontender skin around the blister with an adhesive, soft-foam material (donut pad), New-Skin, or 2nd Skin.
7. Dress and bandage the wound site securely for continued play.

**Incisions and Lacerations**

1. Clean both the wound site and the area around the wound with normal saline or potable tap water.
2. Spray tape adherent on a cotton-tipped applicator and apply above and below the wound.
3. Beginning in the middle of the wound, bring the edges together, and secure the Steri-Strips below the wound. Lift up and secure above the wound. Make sure the edges of the wound are well approximated.
4. Apply a second Steri-Strip in a similar manner immediately adjacent to one side of the original strip. Apply a third strip on the other side. Alternate sides until the entire wound is covered.
5. Dress the wound with an occlusive or nonstick sterile dressing.
6. Individuals who have diabetes, wound contamination with a foreign material, a wound length greater than 2 in (5 cm), and wounds on the lower extremity increase the risk factors for wound infection.[18]
7. Sutures may be desirable for any depth of laceration. At least one study

has shown that in a large number of lacerations older than 12 hours, DERMABOND may be used on facial lacerations. Any wound open to the full thickness of the dermis should be sutured as soon as possible.

8. Refer for medical care if it has been more than 5 years since a tetanus booster or if signs of infection appear.

## Care of Closed Soft-Tissue Injuries

Closed wound care focuses on immediately reducing inflammation, pain, and secondary hypoxia. Several initial steps should be followed in providing general acute care:

- Apply crushed ice packs for 30 minutes directly to the skin as quickly as possible following the injury (40 minutes for a large muscle mass, such as the quadriceps). Do not place a towel or elastic wrap (dry or wet) between the crushed ice pack and skin, because this will reduce the effectiveness of the treatment.
- Apply the crushed ice cold pack for 30 minutes. Elevate the body part at least 10 to 12 in above the level of the heart.
- Following the initial ice treatment, remove the ice pack, apply a compression wrap, and continue elevation.
- Reapply the crushed ice pack every 2 hours (every hour if the individual is active between applications, such as walking on crutches or showering) until bedtime.
- Instruct the individual to wear the compression wrap throughout the night.

See **Application Strategy 10.2: Care of Closed Wounds** on the companion Web site at thePoint for information regarding the basic care of closed soft-tissue injuries after the acute protocol has been followed. Because the severity of injuries can range from mild to severe these guidelines are provided as a general guide. Each injury must be assessed and treated on an individual basis.

**Application Strategy 10.2** provides greater detail in caring for a variety

of closed wounds.

APPLICATION STRATEGY 10.2

## Care of Closed Wounds

### Contusions

For superficial contusions:

1. Pad with soft (open-cell) material next to the skin and denser (closed-cell) material as an external covering.
2. Cut a hole that matches the contused area in the soft material, or keep it solid.
3. Secure the pad with elastic athletic tape or an elastic wrap.
4. Following play, and periodically during the next few days, apply ice with the limb in a lengthened position, compression, and elevation to the site.
5. Repeat until pain and swelling are gone.

For deep contusions, determine disability after temporary paralysis subsides:

1. In moderate and severe cases, follow acute protocol for at least 24 hours with the muscle in a stretched position.
2. If an antalgic gait is present, fit for crutches and instruct the athlete to use a partial or non–weight-bearing gait.
3. Seek medical advice for complications.

### Muscle Injuries

1. Rest the affected area and avoid any activity that may cause pain or lead to further degeneration or tearing of the tendon.
2. Ice with a compression wrap to the muscle belly or tendon to help decrease inflammation.
3. Maintain soft-tissue and joint integrity and mobility through passive movements within pain limits, specific to the muscle involved to

prevent joint stiffness.

4. Do gentle isomeric muscle contractions performed intermittently and at a low intensity to avoid pain. Muscle setting may be done in a shortened position to maintain mobility of the actin–myosin filaments without stressing the injured tissue.
5. Gradually resume activity at a lower intensity than you maintained before your symptoms began. Substitute a different activity from the type of activity that caused the initial injury.
6. Use nonsteroidal anti-inflammatory drugs (as directed) and encourage regeneration of the injured muscle tendon through progressive strengthening exercises, monitoring any flare-up of the initial symptoms.
7. Resume low-intensity functional activities that do not exacerbate the condition.

## Ligament Sprains

### Mild Injury

1. Use protective device (brace) or athletic tape to limit joint laxity and motion at end range.
2. If ice is applied immediately before return, use a mild warm-up before allowing return to play.
3. Heat may be applied when swelling has subsided, and palpable pain upon movement is minimal.

**Moderate-to-severe cases are treated by acute care protocol and referred for medical care:**

1. In a neutral stable position, follow general guidelines for ice application and immobilize, if necessary.
2. Modify daily activity, sport activity level, or equipment.

### Dislocations

1. Splint using standard first aid procedures and refer for medical care.
2. Assess the distal pulse, sensation, and movement; treat for shock and activate emergency action plan (EAP).

## Bursitis

### Mild Cases

1. Pad (donut pad) the area surrounding the bursitis.
2. Apply heat after 72 hours; continue to pad until pain-free.

### Moderate-to-Severe Cases

3. Limit movement by placing a compression pad over the bursa using a compression wrap.
4. Immobilize, if necessary, and modify daily activities.
5. Apply heat when pain and inflammation are under control.

### Infected Bursa

1. Immobilize the joint and apply hot packs.
2. Refer to a physician for medical care. Antibiotics may be prescribed.

An accepted strategy for managing the runner's pain and inflammation is the application of cold to the area. Cold ideally in the form of a crushed ice pack should be applied directly to the site and surrounding area for 30 minutes and then reapplied every 2 hours throughout the day.

# BONE INJURIES



Following a period of rehabilitation, including abstaining from running for a period of 3 weeks, the runner is symptom-free. He is given clearance from his physician to resume his running program with some modifications (i.e., a more gradual increase in time and intensity of running sessions). Within 4 weeks, however, he returns complaining of localized pain in the same area (i.e., posteromedial tibia approximately 3 in proximal to the ankle joint). The pain has been present for 1 week

and is particularly noticeable during weight-bearing activities. What injury should be suspected? What are the implications for the individual's continued training?

In keeping with its material constituents and structural organization, bone behaves predictably in response to stress. The composition and structure of bone make it strong for its relatively light weight.

## Anatomical Properties of Bone

The primary constituents of bone are calcium carbonate, calcium phosphate, collagen, and water. The minerals, making up 60% to 70% of bone weight, provide stiffness and strength in resisting compression. Collagen provides bone with some degree of flexibility and strength in resisting tension. Aging causes a progressive loss of collagen and an increase in bone brittleness. As such, the bones of children are more pliable than those of adults.

Longitudinal bone growth continues only as long as the bone's epiphyseal plates, or growth plates, continue to exist (**Fig. 10.12**). Epiphyseal plates are cartilaginous disks near the ends of the long bones. Longitudinal bone growth takes place on the diaphysis (central) side of the plates. During or shortly after adolescence, the plate disappears and the bone fuses, terminating longitudinal growth. Most epiphyses close by age 18 years, but some may be present until approximately age 25 years.

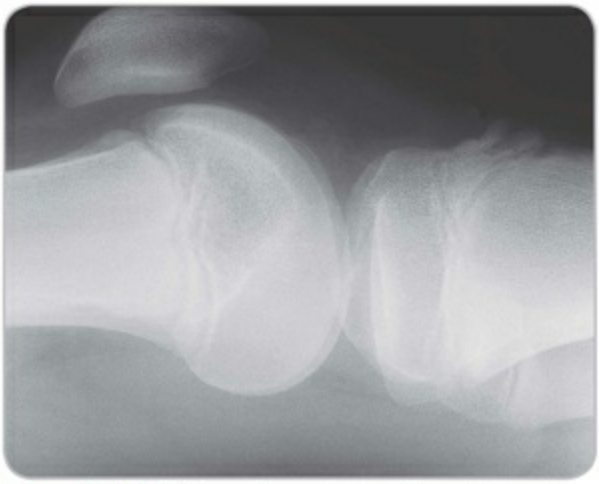


**Figure 10.12. Epiphyseal growth plate.** The epiphyseal plates of the femur and tibia are visible in this radiograph. Note that the individual has a mild irregularity of the tibial tubercle characteristic of Osgood-Schlatter disease.

Although the most rapid bone growth occurs before adulthood, bones continue to grow in diameter throughout most of a person's lifespan. The internal layer of the periosteum builds new, concentric layers of bone tissue on top of the existing ones. At the same time, bone is resorbed or eliminated around the sides of the medullary cavity so that the diameter of the cavity is continually enlarged. The bone cells that form new bone tissue are called **osteoblasts**, and those that resorb bone are known as **osteoclasts**. In healthy adult bone, the activity of osteoblasts and osteoclasts, referred to as bone turnover, is largely balanced. The total amount of bone remains approximately constant until women reach 40 years and men reach 60 years, when a gradual decline in bone mass begins. As such, physically active individuals past these ages may be at increased risk for bone fractures; however, regular participation in weight-bearing exercise has been shown to be effective in reducing age-related bone loss.

Regardless of age, some bones are more susceptible to fracture as a result of their internal composition. Bone tissue is categorized as either **cortical**, if the porosity is low (with 5% to 30% nonmineralized tissue), or **cancellous**, if the porosity is high (with 30% to >90% of nonmineralized tissue) (Fig. 10.13). Most human bones have outer shells of cortical bone, with cancellous bone underneath. Cortical bone is stiffer, which means that it can withstand greater stress but less strain than cancellous bone; however, cancellous bone has the

advantage of being spongier than cortical bone, which means that it can undergo more strain before fracturing. The mineralization of cancellous bone varies with the individual's age and with the location of the bone in the body. Both cortical and cancellous bone is anisotropic. As such, they exhibit different strengths and stiffness in response to forces applied from different directions. Bone is strongest in resisting compressive stress and weakest in resisting shear stress.

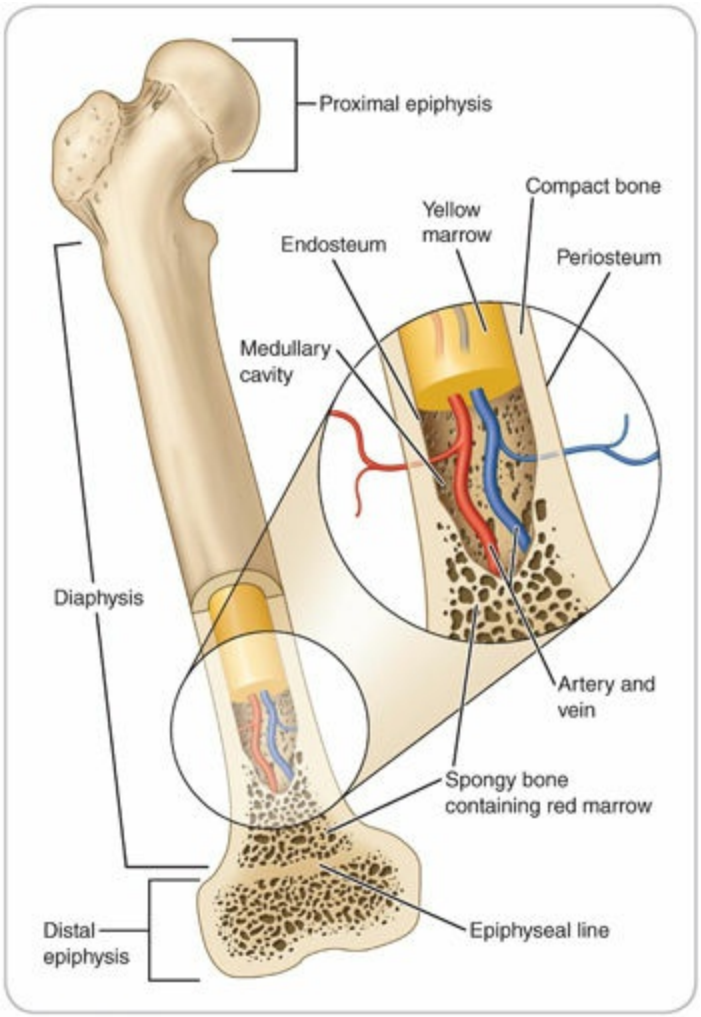



**Figure 10.13. Bone macrostructure.** Epiphyseal growth lines are found at both ends of the bone. Cortical bone surrounds the cancellous bone and the medullary cavity. Cancellous bone is more porous than cortical bone.

Bone size and shape also influence the likelihood of fracture. The direction and magnitude of the forces to which they are habitually subjected largely determine the shape and size of the bone. The direction in which new bone

tissue forms is in response to the adaptation required in resisting encountered loads, particularly in regions of high stress, such as the femoral neck. The mineralization and girth of bone increases in response to increased levels of stress. For example, the bones of the dominant arm of tennis players and professional baseball players have been found to be larger and stronger than the bones of their nondominant arms.[18–20]

## Classification of Bone Injuries

A **fracture** is a disruption in the continuity of a bone (Fig. 10.14). Signs of fracture include swelling and bruising (discoloration), deformity or shortening of the limb, point tenderness, grating or crepitus, guarding or disability, and exposed bone ends.

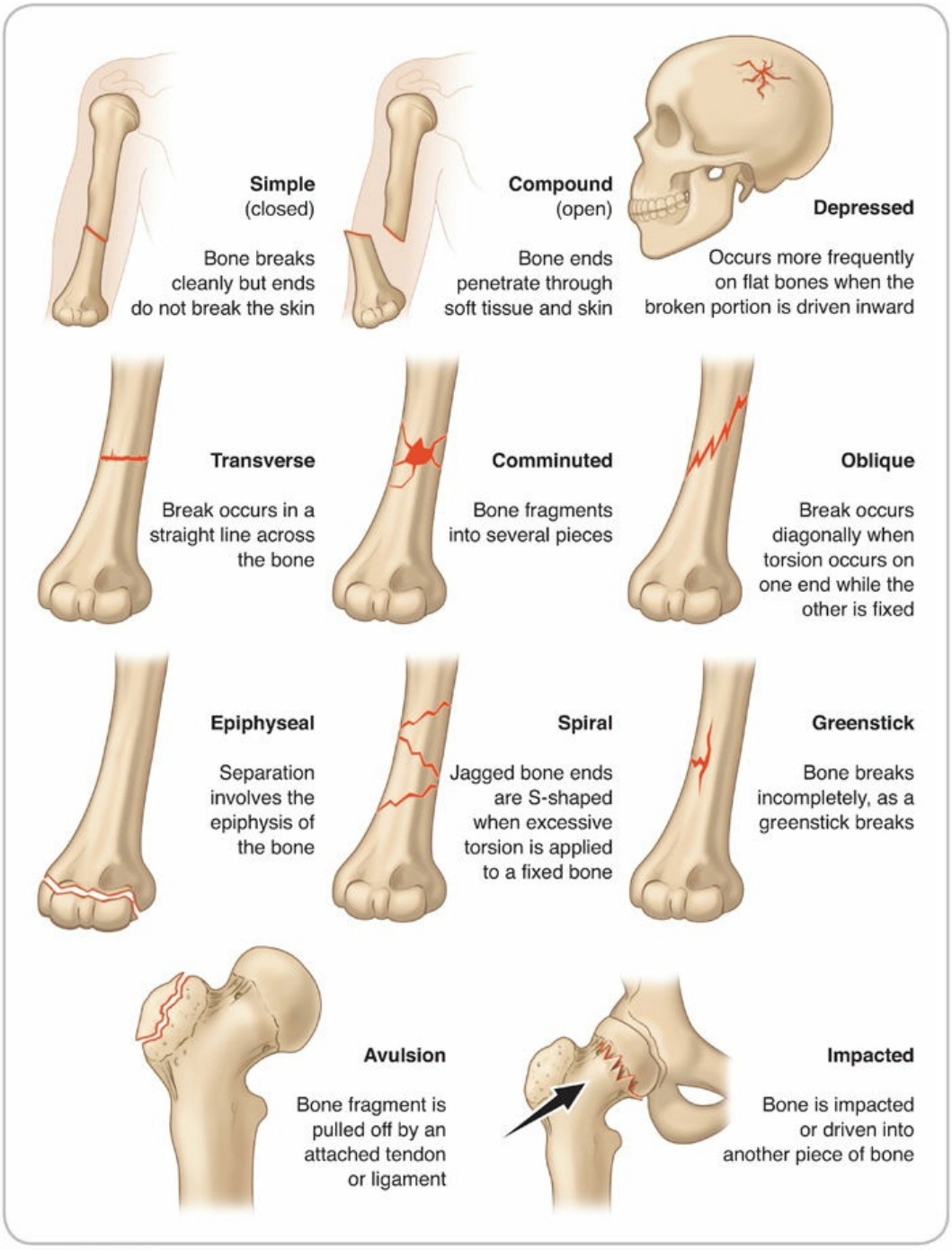



**Figure 10.14. Types of fractures.** The type of fracture sustained is dependent on the type of mechanical load that causes the injury.

The type of fracture that is sustained depends on the type of mechanical loading that caused it as well as on the health and maturity of the bone at the time of injury. Fractures are considered to be closed when the bone ends remain intact within the surrounding soft tissues and to be open, or compound,

when one or both bone ends protrude from the skin.

Excessive torsional and bending loads, as exemplified by tibial fractures resulting from skiing accidents, often produce spiral fractures of the long bones. Such fractures are the result of a combined loading pattern of shear and tension, producing failure at an oblique angle to the long axis of the bone.

Because bone is stronger in resisting compression than in resisting tension and shear, acute compression fractures of bone are rare. Under combined loading, however, a fracture resulting from a torsional load may be affected by the presence of a compressive load. An **impacted** fracture is one in which the opposite sides of the fracture are compressed together. Fractures that result in depression of bone fragments into the underlying tissues are termed **depressed**.

Because the bones of children contain relatively larger amounts of collagen compared with adult bones, they are more flexible and more resistant to fracture under day-to-day loading than adult bones are. Consequently, **greenstick** fractures, or incomplete fractures, are more common in children than in adults. A greenstick fracture is an incomplete fracture typically caused by bending or torsional loads.

**Avulsions** are another type of fracture caused by tensile loading that involve a tendon or ligament pulling a small chip of bone away from the rest of the bone. Explosive throwing and jumping movements may result in avulsion fractures. When loading is very rapid, a fracture is more likely to be **comminuted**, meaning that it contains multiple fragments.

**Stress fractures** result from repeated, low-magnitude forces. Stress fractures differ from acute fractures in that they can worsen over time, beginning as a small disruption in the continuity of the outer layers of cortical bone and ending as a complete cortical fracture, with possible displacement of the bone ends. Stress fractures of the metatarsals, femoral neck, and pubis have been reported among runners when bone growth is exceeded by bone breakdown from repetitive stress. Stress fractures of the pars interarticularis region of the lumbar vertebrae occur with higher-than-normal frequencies among football linemen and female gymnasts.

**Osteopenia**, a condition of reduced bone mineral density, predisposes an individual to all types of fractures but particularly to stress fractures. The

condition is found primarily among adolescent female athletes, especially distance runners, who are amenorrheic. Although **amenorrhea** among this group is not well understood, it appears to be related to a low percentage of body fat and/or high training mileage. The link between the cessation of menses and osteopenia also is not well understood. Possible contributing factors include hyperactivity of osteoclasts, hypoactivity of osteoblasts, hormonal factors, and insufficiencies of dietary calcium or other minerals or low caloric intake.

## Classification of Epiphyseal Injuries

The bones of children and adolescents are vulnerable to epiphyseal injuries, including injuries to the cartilaginous epiphyseal plate, articular cartilage, and apophysis. The apophyses are sites of tendon attachments to bone, where bone shape is influenced by the tensile loads to which these sites are subjected. Both acute and repetitive loading can injure the growth plate, potentially resulting in premature closure of the epiphyseal junction and termination of bone growth. Little League elbow, for example, is a stress injury to the medial epicondylar epiphysis of the humerus. Salter[21] has categorized acute epiphyseal injuries into five distinct types (**Fig. 10.15**):

**Type I:** a complete separation of the epiphysis from the metaphysis with no fracture to the bone

**Type II:** a separation of the epiphysis and a small portion of the metaphysis

**Type III:** a fracture of the epiphysis

**Type IV:** a fracture of a part of the epiphysis and metaphysis

**Type V:** a compression of the epiphysis without fracture, resulting in compromised epiphyseal function

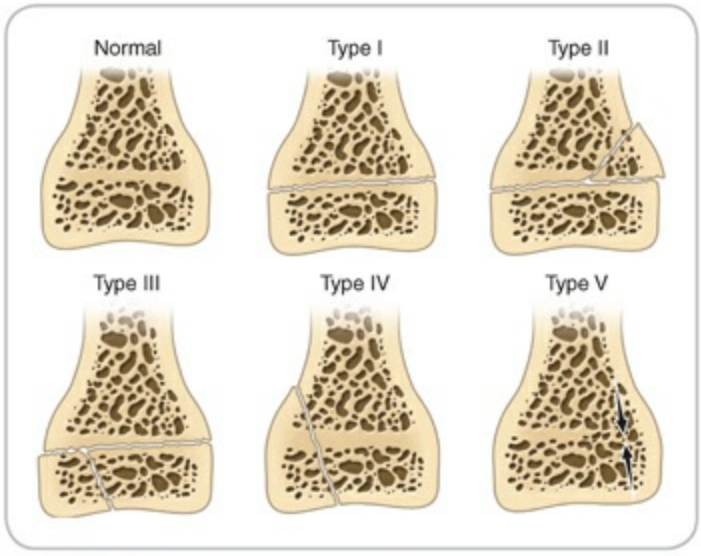



**Figure 10.15. Epiphyseal injuries.** Five distinct types of injuries involve the epiphysis.

Another category of epiphyseal injuries is referred to collectively as osteochondrosis. **Osteochondrosis** results from the disruption of blood supply to an epiphysis, with associated tissue necrosis and potential deformation of the epiphysis. Because the cause of the condition is poorly understood, it typically is termed idiopathic osteochondrosis. Osteochondroses most commonly occur between the ages of 3 and 10 years and are more prevalent among boys than girls.[20] Specific disease names have been given to sites where osteochondrosis is common, such as Legg-Calvé-Perthes disease, which is osteochondrosis of the femoral head.

The apophyses also are subject to osteochondrosis, particularly among children and adolescents. These conditions, referred to as **apophysitis**, may be idiopathic; however, they can be associated with traumatic avulsion-type fractures. Common sites for apophysitis are the calcaneus (i.e., Sever disease) and the tibial tubercle at the site of the patellar tendon attachment (i.e., Osgood-Schlatter disease).

## Bony Tissue Healing

Healing of acute bone fractures is a three-phase process, as is soft-tissue healing. The acute inflammatory phase lasts approximately 4 days. The formation of a hematoma in the medullary canal and surrounding tissues causes

damage to the periosteum and the surrounding soft tissues. The ensuing inflammatory response involves vasodilation, edema formation, and histochemical changes associated with soft-tissue inflammation.

During repair and regeneration, osteoclasts resorb damaged bone tissues, whereas osteoblasts build new bone. Between the fractured bone ends, a fibrous, vascularized tissue, known as a **callus**, is formed (**Fig. 10.16**). The callus contains weak, immature bone tissue that strengthens with time through bone remodeling. The process of callus formation is known as endochondral bone healing. An alternative process, known as direct bone healing, can occur when the fractured bone ends are immobilized in direct contact with one another. This enables new, interwoven bone tissue to be deposited without the formation of a callus. Unless a fracture is fixed by metal plates, screens, or rods, healing normally takes place through the endochondral process. Because noninvasive treatment generally is preferred, a fixation device is only implanted when it appears unlikely that the fracture will heal acceptably without one.

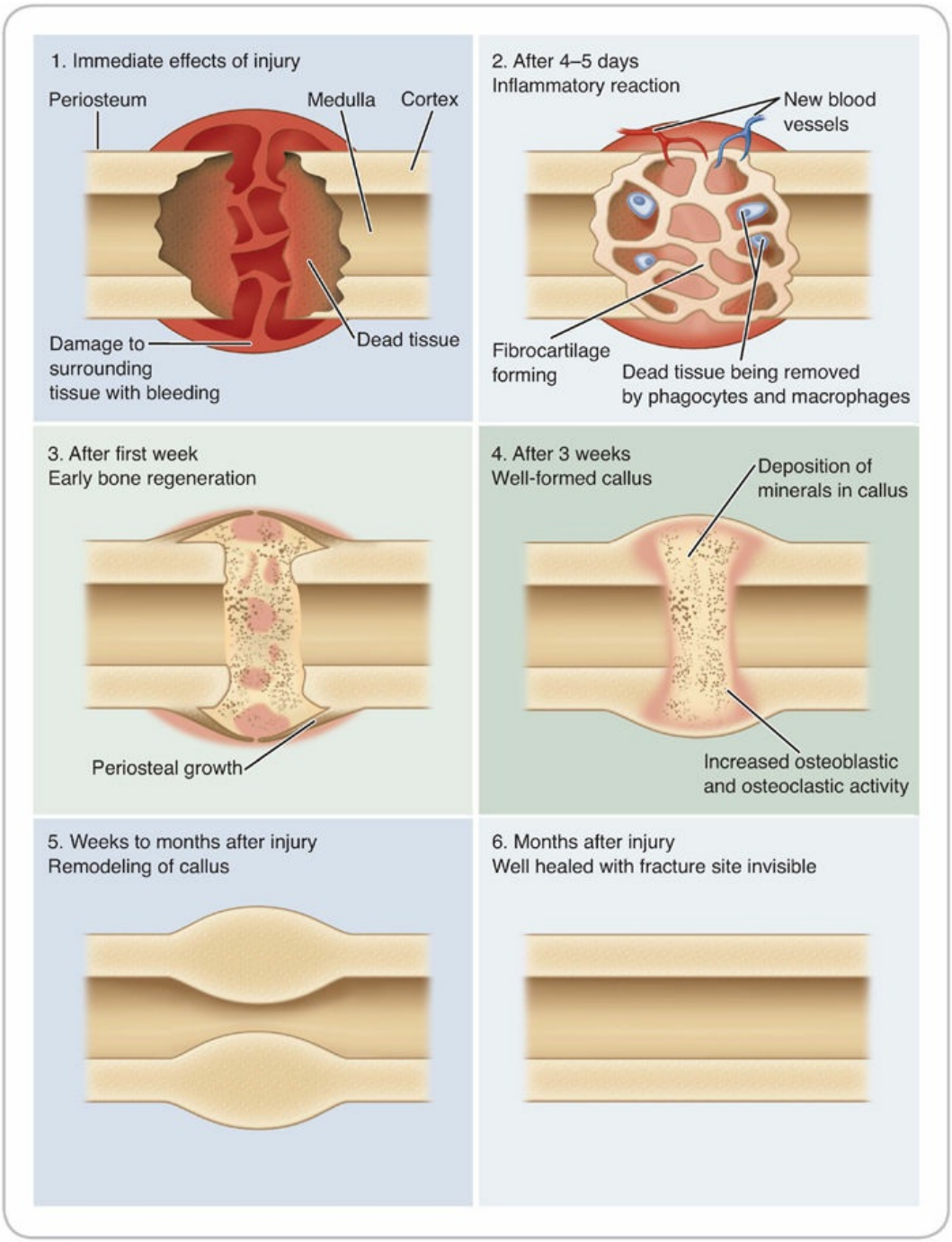



**Figure 10.16. Bone healing.** The process of endochondral bone healing involves callus formation.

Maturation and remodeling of bone tissue involves osteoblast activity on the concave side of the fracture, which is loaded in compression, and osteoclast activity on the convex side of the fracture, which is loaded in tension. The process continues until normal shape is restored and bone strength is commensurate with the loads to which the bone is routinely subjected.

Because stress fractures continue to worsen as long as the site is overloaded, it is important to recognize these injuries as early as possible. Elimination or reduction of the repetitive mechanical stress causing the fracture is the primary factor necessary for healing. This allows for a gradual restoration of the proper balance of osteoblast and osteoclast activity in the bone.

## Management of Bone Injuries

Possible fractures can be detected with palpation, radiographs and magnetic resonance imaging, percussion, compression, and distraction (see Fig. 6.2). Palpation can detect deformity, crepitus, swelling, or increased pain at the fracture site. Compression is performed by gently compressing the distal end of the bone toward the proximal end or by encircling the body part (e.g., a foot or a hand) and gently squeezing, thereby compressing the heads of the bones together. Again, if a fracture is present, pain increases at the fracture site. Distraction employs a tensile force, whereby the application of traction to both ends of the fractured bone helps to relieve pain.

A suspected fracture should be splinted before the individual is moved to avoid damage to surrounding ligaments, tendons, blood vessels, and nerves. Application Strategy 10.3 explains the immediate management of fractures.

**APPLICATION STRATEGY 10.3**

### Management Algorithm for Bone Injuries

1. Remove clothing and jewelry from around the injury site. (Cut clothing away with scissors to avoid moving the injured area.)
2. Check distal pulse and sensation. If either is abnormal, activate EAP.
3. Cover all wounds, including open fractures, with sterile dressings, and secure them.
4. Do not attempt to push bone ends back underneath the skin.
5. Pad the splint to prevent local pressure.
6. Immobilize the joints above and below the fracture site.

7. Splint in the position found if
   - Pain increases with gentle traction or the limb resists positioning.
   - The fracture is severely angulated.
   - Do not straighten unless it is absolutely necessary to incorporate the limb into the splint; move as little as possible.
   - Splint firmly but do not impair circulation.
   - Recheck distal pulse and sensation after applying splint.
   - Check vital signs, treat for shock, and transport to medical facility.

Pain localized over a bone that is particularly painful during weight-bearing activities is a classic symptom of a stress injury. The runner should be referred to a physician for further evaluation. In addition he should be advised to stop running and to reduce weight-bearing activities through cross-training.

## NERVE INJURIES

The runner is diagnosed with a tibial stress fracture. If the runner had continued his running program rather than seeking medical attention would he have been susceptible to a nerve injury?

The nervous system is divided into the central nervous system, consisting of the brain and spinal cord, and the peripheral nervous system, which includes 12 pairs of cranial nerves and 31 pairs of spinal nerves, along with their branches (Fig. 10.17). The human body has 8 pairs of cervical spinal nerves (C1 through C8), 12 pairs of thoracic nerves (T1 through T12), 5 pairs of lumbar nerves (L1 through L5), 5 pairs of sacral nerves (S1 through S5), and 1 pair of tiny coccygeal nerves (designated C0). Injuries to any of these nerves can be devastating to the individual, potentially resulting in temporary or even permanent disability.

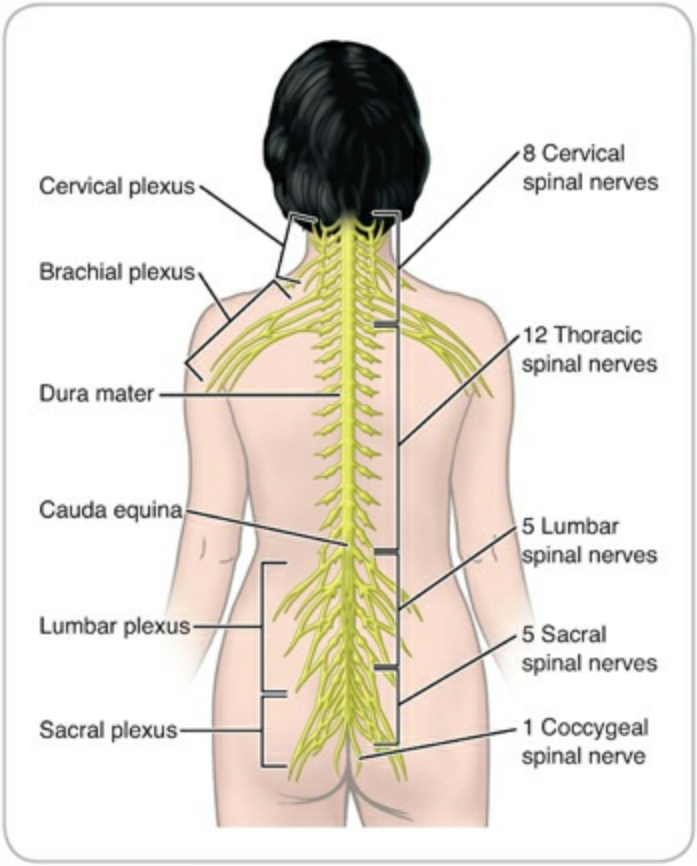



**Figure 10.17. Spinal nerves.** Each spinal nerve is formed from anterior and posterior roots on the spinal cord. The posterior branches are afferent nerves; the anterior branches are efferent nerves.

## Anatomical Properties of Nerves

Each spinal nerve is formed from anterior and posterior roots on the spinal cord that unite at the intervertebral foramen. The posterior branches are the **afferent (sensory)** nerves that transmit information from sensory receptors in the skin, tendons, ligaments, and muscles to the central nervous system. The anterior branches are the **efferent (motor) nerves** that transmit control signals to the muscles. The nerve fibers are heavily vascularized and are encased in a multilayered, segmental protective sheath called the myelin sheath. Myelin protects and electrically insulates fibers from one another, and it increases the speed of transmission of nerve impulses. Myelinated fibers (axons bearing a myelin sheath) conduct nerve impulses rapidly, whereas unmyelinated fibers tend to conduct impulses quite slowly.

## Classification of Nerve Injuries

Tensile or compressive forces most commonly injure nerves. Tensile injuries are more likely to occur during severe, high-speed accidents, such as automobile accidents or impact collisions in contact sports. When a nerve is loaded in tension, the nerve fibers tend to rupture before the surrounding connective tissue sheath. Because the nerve roots on the spinal cord are not protected by connective tissue, they are particularly susceptible to tensile injury, especially in response to stretching of the brachial plexus or cervical nerve roots.

Nerve injuries caused by tensile forces typically are graded in three levels. Grade I injuries represent **neurapraxia**, the mildest lesion. A neurapraxia is a localized conduction block that causes temporary loss of sensation and/or motor function from selective demyelination of the axon sheath without true axonal disruption. Recovery usually occurs within days to a few weeks. Grade II injuries are called **axonotmesis injuries**, which produce significant motor and mild sensory deficits that last at least 2 weeks. Axonotmesis disrupts the axon and myelin sheath but leaves the epineurium intact. The epineurium is the connective tissue that encapsulates the nerve trunk and binds the fascicles together. Axonal regrowth occurs at a rate of 1 to 2 mm per day; full or normal function usually is restored. Grade III injuries represent **neurotmesis injuries**, which disrupt the endoneurium. These severe injuries have a poor prognosis, with motor and sensory deficits persisting for up to 1 year. Surgical intervention often is necessary to avoid poor or imperfect regeneration.

Compressive injuries of nerves are more complex because their severity depends on the magnitude and duration of loading and on whether the applied pressure is direct or indirect. Because nerve function is highly dependent on oxygen provided by the associated blood vessels, damage to the blood supply caused by a compressive injury results in damage to the nerve.

Nerve injuries can result in a range of afferent symptoms, from severe pain to a complete loss of sensation. Terms used to describe altered sensations include **hypoesthesia** (a reduction in sensation), **hyperesthesia** (heightened sensation), and **paresthesia** (a sense of numbness, prickling, or tingling).

Pinching of a nerve can result in a sharp wave of pain that is transmitted through a body segment. Irritation or inflammation of a nerve can result in chronic pain along the nerve's course, known as **neuralgia**.

## Nerve Healing

When a nerve is completely severed, healing does not occur, and loss of function typically is permanent. Unless such injuries are repaired surgically, random regrowth of the nerve occurs, resulting in the formation of a **neuroma**, or nerve tumor.

When nerve fibers are ruptured in a tensile injury but the surrounding myelin sheath remains intact, it sometimes is possible for a nerve to regenerate along the pathway provided by the sheath. Such regeneration is relatively slow, however, proceeding at a rate of less than 1 mm per day, or approximately 2.5 cm per month.

## Management of Nerve Injuries

If nerves are injured, cutaneous sensation or muscle movement may become impaired. Neurological testing is explained and demonstrated in Chapter 6. Sensation is assessed by touching the person with a cotton ball, paper clip, pads of the fingers, fingernails, or a neurological hammer to determine if the individual can differentiate between sharp and dull. It also is important to determine if the sensation feels the same on the injured body segment as it does on the uninjured body segment.

The motor component can be tested with manual muscle testing. In testing a myotome, a normal response is a strong muscle contraction. A weakened muscle contraction may indicate partial paralysis of the muscles innervated by the nerve root being tested. A peripheral nerve injury results in complete paralysis of the muscles supplied by that nerve. If a muscle tear is present, a weakened muscle contraction is accompanied by pain.

It is critical that a possible nerve injury be identified and the individual referred immediately to a physician for advanced evaluation and care. For this reason, **Application Strategy 10.4** explains only basic principles used in the

management of nerve injuries.

See **Application Strategy 10.4: Management of Nerve Injuries** on the companion Web site at thePoint for basic principles used in the management of nerve injuries.

## APPLICATION STRATEGY 10.4

### Management of Nerve Injuries

**Mild Cases: Rest the Extremity**

1. Ice, transcutaneous electrical nerve stimulation, ultrasound, and nonsteroidal anti-inflammatory drugs may be used to address persistent pain and tenderness.
2. Use protective padding or bracing to decrease repetitive compression or excessive tension forces.
3. Athlete may participate when motor and sensory nerve function returns to normal.

**Moderate-to-severe cases need to be referred to a physician. After appropriate acute care protocol:**

1. Perform neural flossing of neural tissues, beginning away from the site of the lesion and applying only gentle movement or tensile loads across the injured nerve.
2. Include strengthening exercises of the injured region and carefully monitor proper technique.
3. Instruct the athlete on appropriate posture, muscle tension, and joint stability.
4. Correct any biomechanical factors that may have contributed to the injury.
5. Do not allow a return to activity until the athlete is asymptomatic.

Nerve injuries typically are the result of tension or compression forces. As such the runner was not likely to sustain a nerve injury. If

left untreated however a stress fracture can become an acute fracture. The fractured ends of the bones and the swelling associated with an acute fracture pose a possible danger to nerves in the involved area.

# PAIN

? An observation of two individuals involved in rehabilitation programs for similar lower leg injuries reveals different patient responses. One patient avoids exercises because of excessive pain; the other patient performs workouts without complaint. What variables may explain the different responses?

Pain is a negative sensory and emotional experience associated with actual or potential tissue damage. It also is a universal symptom common to most injuries. An individual's perception of pain is influenced by various physical, chemical, social, and psychological factors.

## The Neurological Basis of Pain

Pain can originate from somatic, visceral, and psychogenic sources. Somatic pain originates in the skin as well as internal structures of the musculoskeletal system. Visceral pain, which often is diffuse or referred rather than localized to the problem site, originates from the internal organs. Psychogenic pain involves no apparent physical cause of the pain, although the sensation of pain is felt.

The stimulation of specialized afferent nerve endings, called **nociceptors**, produces the pain sensation. The name *nociceptor* is derived from the word noxious, meaning physically harmful or destructive. Nociceptors are prevalent in the skin, meninges, periosteum, teeth, and some internal organs.

In most acute injuries, pain is initiated by **mechanosensitive** nociceptors responding to the traumatic force that caused the injury. In chronic injuries and during the early stages of healing of acute injuries, pain persists because of the

activation of **chemosensitive** nociceptors. Bradykinin, serotonin, histamines, and prostaglandins are all chemicals transported to the injury site during inflammation, which activate the chemosensitive nociceptors. Thermal extremes also can stimulate other specialized nociceptors to produce pain.

Two types of afferent nerves transmit the sensation of pain to the spinal cord. Small diameter, slow transmission, unmyelinated C fibers transmit low-level pain that might be described as dull or aching. Sharp, piercing types of pain are transmitted by larger, faster, and more thinly myelinated A fibers. Pain can be transmitted along both types of afferent nerves from somatic and visceral sources. Activity involving A and C fibers from the visceral organs also can provoke autonomic responses, such as changes in blood pressure, heart rate, and respiration.

Afferent nerves carrying pain impulses, along with those transporting sensations such as touch, temperature, and proprioception, articulate with the spinal cord through the substantia gelatinosa of the cord's dorsal horn. Specialized T cells then transmit impulses from all the afferent fibers up the spinal cord to the brain, with each T cell carrying a single impulse. Within the brain, the pain impulses are transmitted to the thalamus (primarily to its ventral posterior lateral nucleus) as well as to the somatosensory cortex, where pain is perceived.

According to the gate control theory of pain proposed by Melzack and Wall,[22] the substantia gelatinosa acts as a gatekeeper by allowing either a pain response or one of the other afferent sensations to be transported by each T cell. This theory is substantiated by the observation that increased sensory input can reduce the sensation of pain. For example, extreme cold often can numb pain. Because hundreds or thousands of "gates" are in operation, however, added sensory input more commonly reduces rather than eliminates the feeling of pain, because pain impulses get through to some of the T cells.

## Factors That Mediate Pain

Some brain cells have the ability to produce narcotic-like, pain-killing compounds known as opioid peptides, which include β-endorphin and

methionine enkephalin. Both compounds work by blocking neural receptor sites that transmit pain. Several different sites in the brain produce endorphins. Stressors such as physical exercise, mental stress, and electrical stimulation provoke the release of endorphins into the cerebrospinal fluid. A phenomenon called runner's high, which is a feeling of euphoria that occurs among long-distance runners, has been attributed to endorphin release. The brainstem and the pituitary gland produce enkephalins. Enkephalins block pain neurotransmitters in the dorsal horn of the spinal cord.

The central nervous system also imposes a set of **cognitive** (i.e., the quality of knowing or perceiving) and **affective** (i.e., pertaining to feelings or a mental state) filters on both the perception of pain and the subsequent expression of perceived pain. Social and cultural factors can powerfully influence the level of pain tolerance. For example, in American society, it is much more acceptable for females than males to express feelings of pain. Individual personality and a state of mental preoccupation also can be significant modifiers of pain.

## Referred and Radiating Pain

Referred pain is perceived at a location that is remote from the site of the tissues actually causing the pain. A proposed explanation for referred pain begins with the fact that neurons carrying pain impulses split into several branches within the spinal cord. Although some of these branches connect with other pain-transmitting fibers, some also connect with afferent nerve pathways from the skin. This cross-branching can cause the brain to misinterpret the true location of the pain. In some instances, referred pain behaves in a logical and predictable fashion. Pain from internal organs typically is projected outward to corresponding **dermatomes** of the skin. For example, heart attacks can produce a sensation of pain in the superior thoracic wall and medial aspect of the left arm. In most cases, the affected internal organ and corresponding dermatome receive innervation from the same spinal nerve roots.

Referred pain should not be confused with radiating pain, which is pain that is felt both at its source and along a nerve. Pinching of the sciatic nerve at

its root may cause pain that radiates along the nerve's course down the posterior aspect of the leg.

Differences in pain perception and tolerance can be caused by differences in chemical social and psychological influences as well as by differences in the severity of the original injury and the progression of the healing process.

## SUMMARY

1. When a force acts, two effects occur on the target tissue: acceleration, or change in velocity, and deformation, or change in shape.
2. Two factors determine if injury occurs to a tissue: the magnitude of the force, and the material properties of the tissues involved.
3. Biological tissues are strongest in resisting the form of loading to which they most commonly are subjected.
4. Force exceeding a structure's yield point causes rupture or fracture.
5. Most common mechanisms of injury include compressive force from axial loading, which compresses or crushes an object; tensile force from tension or traction on an object; and shearing force, which acts parallel or tangent to a plane passing through the object.
6. In tendons, the collagen fibers are arranged in a parallel pattern, enabling resistance to high, unidirectional tensile loads when the attached muscle contracts.
7. In ligaments, the collagen fibers are largely parallel but also interwoven, providing resistance to large tensile loads along the long axis of the ligament and to smaller tensile loads from other directions.
8. The viscoelastic aspect of muscle extensibility enables muscles to stretch to greater lengths over time in response to a sustained tensile force.

9. Injury to soft tissue and joints can be acute or chronic. Muscle tears and sprains are common acute injuries associated with participation in sports and physical activities. Tendinitis and osteoarthritis are common chronic or overuse injuries.
10. Wound healing entails three overlapping phases: inflammation, proliferation, and maturation (remodeling). During the inflammatory phase, blood loss is curtailed, clotting takes place, and histochemical cascades promote coagulation, vasodilation, and attraction of specialized cells to rid the wound site of foreign or infectious agents. The proliferative phase includes angiogenesis, fibroplasia, reepithelialization, and wound contraction. The maturation phase involves remodeling of the newly formed tissue.
11. Growth factors are proteins that attract cells to the wound, stimulate their proliferation, and direct the deposition of the extracellular matrix.
12. The mineralization and girth of bone increases in response to increased levels of stress.
13. Because bone is stronger in resisting compressive forces than in resisting both tension and shear forces, acute compression fractures are rare. Most fractures occur on the side of the bone placed in tension.
14. Maturation of bone tissue involves osteoblast activity on the concave side of the fracture, which is loaded in compression, and osteoclast activity on the convex side of the fracture, which is loaded in tension.
15. Nerve injuries caused by tensile forces are graded in three levels: neurapraxia injury, with temporary loss of sensation or motor function; axonotmesis injury, with significant motor and sensory deficits that last at least 2 weeks; and neurotmesis injury, with significant motor and sensory deficits persisting for up to 1 year.
16. Pain associated with injury is transmitted by specialized afferent nerve endings called nociceptors. Mechanosensitive nociceptors respond to traumatic forces that cause the injury. Chemosensitive nociceptors respond

to chronic injuries and are activated during the early stages of healing in acute injuries.

17. Pain is transmitted along two types of afferent nerves: small diameter, slow transmission, unmyelinated C fibers, which transmit low-level pain, and larger, faster, and finely myelinated A fibers, which transmit sharp, piercing types of pain.
18. Afferent nerves carry nerve impulses to the spinal cord through the substantia gelatinosa of the cord's dorsal horn up to the thalamus as well as to the somatosensory cortex, where pain is perceived.
19. Stressors, such as physical exercise, mental stress, and electrical stimulation, provoke the release of endorphins into the cerebrospinal fluid and can mediate the perception of pain.

## APPLICATION QUESTIONS

1. An individual falls on an outstretched arm and sustains an elbow injury. How might you distinguish between a sprain and a muscular injury?
2. A 13-year-old female gymnast complains of foot pain that has been present for the past 2 weeks and continues to get worse, even after rest. The primary pain is the distal area of the 2nd metatarsal. The gymnast does not recall a specific acute mechanism of injury. What injury might you suspect? How could you assess this injury to confirm your course of action? What type of mechanical loading would most likely be associated with this injury?
3. A sedentary 60-year-old woman wants to begin a weight-bearing exercise program. What precautions should be taken to prevent any injury? Why?
4. A middle school athlete has groin pain but does not recall an injury. He is unable to balance on one leg. What type of pain might the athlete be experiencing? What would your course of action entail?
5. A middle-aged runner training for a first marathon overhead a statement

concerning a surge of endorphin release resulting in a "runner's high." How would you respond?

6. A 16-year-old male sustains a mild ankle sprain during practice. Explain your initial acute management of the condition. What would you suggest that the athlete do during the evening and weekend hours to prevent further inflammation and pain?

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