1 Types and Causes of Injuries

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Exercise and physical activity are the most important determinants of health in developing and transitioning countries, and sedentary living is the fourth independent risk factor for morbidity and mortality from noncommunicable disease. Regular physical activity reduces the risk of early death in general, and of cardiovascular disease, high blood pressure, type 2 diabetes, and even some types of cancer. Indeed, physical *in*activity can present as great a risk to health as smoking, being overweight, high cholesterol, or high blood pressure. Furthermore, intense exercise is not necessarily more effective than other forms of exercise for prevention and treatment of chronic disease. Significant health benefits can be achieved through moderate physical activity; as a matter of fact, standing as opposed to sitting will also incur health benefits. This holds true even at an advanced age. The least fit people are the ones who can derive the greatest health benefit from regular physical activity.

Unfortunately, exercise and physical activity also have some unfortunate side effects. Injuries are a particular risk. Nevertheless, the net health effect is positive–the benefits of physical activity far exceed the problems caused by injuries.

Acute Injuries and Overuse Injuries

A sports injury may be defined as damage to the tissues of the body that occurs as a result of sport or exercise. In this book, the term applies to any damage that results from any form of physical activity. Physical activity can be defined as moving or using the body, and it includes numerous forms of activity such as working, fitness exercise, outdoor activity, playing, training, getting in shape, working out, and physical education.

Sport injuries can be divided into acute injuries and overuse injuries, depending on the injury mechanism and onset of symptoms. In most cases, it is easy to classify an injury as acute or overuse, but in some cases it may be difficult. Acute injuries occur suddenly and have a clearly defined cause or onset. Overuse injuries occur gradually. However, an important concept with overuse injuries is that they exist along a spectrum where the inciting events are below the threshold for clinical symptomatology, but if not rectified, they eventually produce sufficient tissue damage to result in clinical symptoms. This is important for physicians, therapists, and patients

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to understand, because it is not uncommon to "react" to "new" clinical symptoms the same way one reacts to acute injuries. Such a response may ignore the underlying clinical symptomatology and thus may interfere with effective treatment. For example, an athlete with a stress fracture (a fatigue fracture) in the foot will often state that the symptoms originated during a specific run, perhaps even from a specific step. The injury may accordingly be misclassified as an acute injury. However, the actual cause of the stress fracture is that the specific run was a precipitating event on top of the underlying spectrum of tissue damage on the skeleton from overuse over time. Therefore, these types of injuries should be classified as overuse injuries.

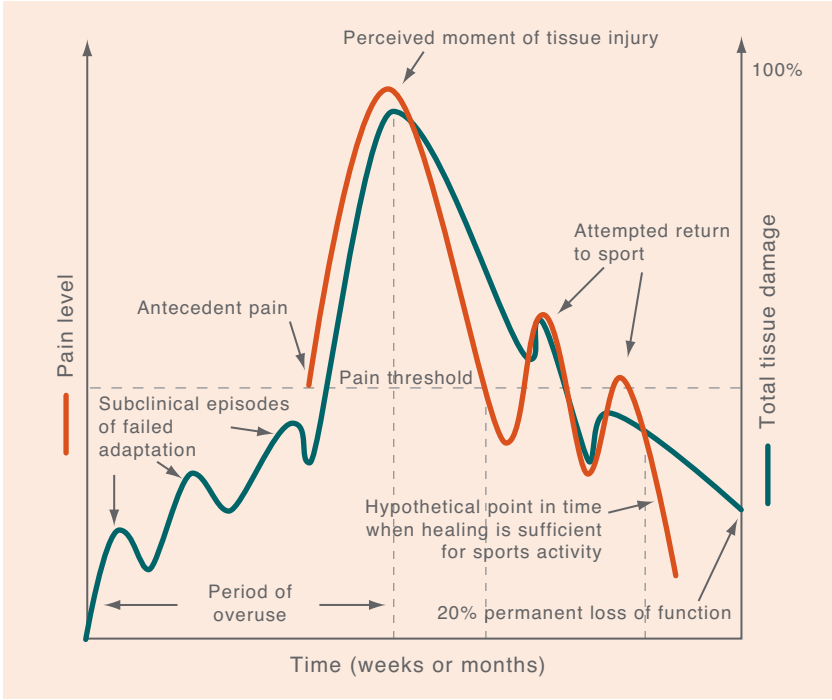

Figure 1.1 Hypothetical overview of pain and tissue injury in a typical overuse injury. (Reproduced with permission from the Norwegian Sports Medicine Association.)

As shown in Figure 1.1, the pathological process is often under way for a period of time before the athlete notices the symptoms. Repetitive low-grade forces that lead to microtrauma in the tissues cause overuse injuries. In most cases, the tissue will repair without demonstrable clinical symptoms. However, if this process continues, the ability of the tissue to repair can be exceeded, resulting in a clinical overuse injury with symptoms. It is vitally important that athletes as well as therapists and physicians understand this concept so that correct treatment can be initiated.

The difference between acute injuries and overuse injuries can also be described in biomechanical terms. Dynamic or static muscle action creates internal resistance in the loaded structures (stress) that counteracts deformation (strain) of the tissue. All tissue has a characteristic ability to tolerate deformation and stress, and injuries occur when the tolerance level is exceeded. An acute injury occurs when loading is sufficient to cause irreversible deformation of the tissue, whereas an overuse injury occurs as a result of repeated overloading either in the loading itself or through inadequate recovery time between loadings. Each incidence, alone, is not enough to cause irreversible deformation, but the repeated actions can result in an injury over time.

Acute injuries are most common in sports in which the speed is high and the risk of falling is great (e.g., downhill skiing) and in team sports where there is much contact between players (e.g., ice hockey and soccer). Overuse injuries make up the large portion of injuries in aerobic sports that require long training sessions with a monotonous routine (e.g., long-distance running, bicycling, or cross-country skiing). But a large number of overuse injuries also occur in technical sports, in which the same movement is repeated numerous times (e.g., tennis, javelin throwing, weightlifting, and high jumping).

Why Do Injuries Occur?

The basic principle for training is that the body reacts to a specific physical training load with specific predictable adaptation. Loading that exceeds what an athlete is

used to will cause the tissue that is being trained to attempt to adapt to the new loading. For example, training provides a stimulus that causes the muscles to increase the production of contractile proteins, the muscle fibers become larger (and more numerous), and the muscle fibers specifically adapt to whether the training requires primarily endurance or maximum strength. This principle applies to all types of tissue. The skeleton, tendons, ligaments, and cartilage adapt accordingly. The tissue becomes stronger and tolerates more (Figure 1.2).

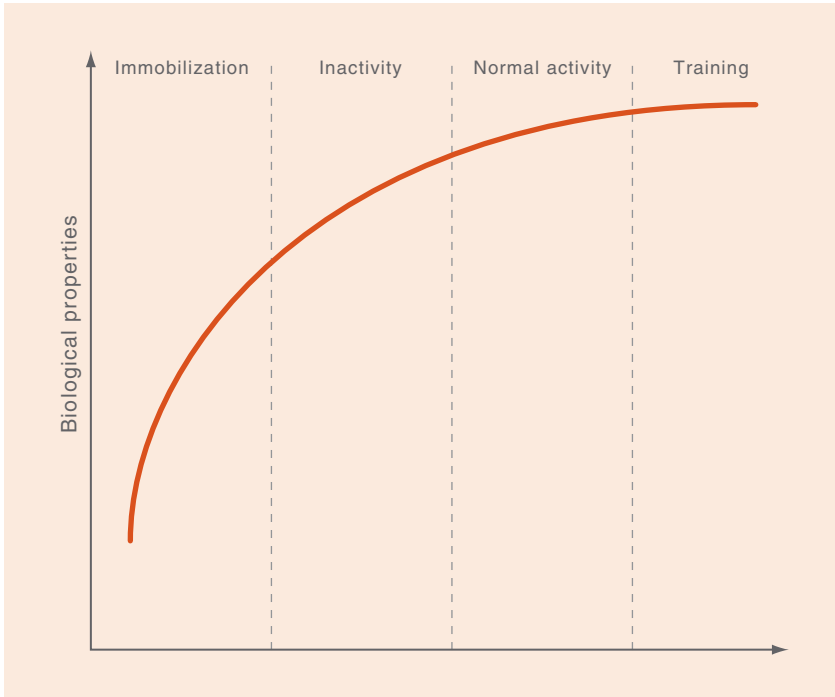

Figure 1.2 Adaption to training. Immobilization significantly weakens the biological properties of the tissue, whereas exercise improves function. (Reproduced with permission from the Norwegian Sports Medicine Association.)

However, if the training load exceeds the tissue's ability to adapt, injuries will occur. The risk of overuse injuries increases when training load increases. This could result from an increase in the duration of individual training sessions or an increase in training intensity or the frequency of training sessions. Often the duration, intensity, and frequency of training increase at the same time, such as at a training camp or at the beginning of the season. Therefore, it is common to say that overuse injuries are due to "too much, too often, too quickly, and with too little rest," which means that training load increases more quickly than the tissue is able to adapt.

Various Types of Injuries

Sport injuries can be divided into *soft-tissue injuries* (cartilage injuries, muscle injuries, tendon injuries, and ligament injuries) and *skeletal injuries* (fractures). The various types of tissue have distinctly different biomechanical properties and their ability to adapt to training also varies. This chapter examines the characteristics of the various types of tissue and the ways in which the skeleton, cartilage, muscles, tendons, and ligaments can be injured.

Ligaments

Structure and Function

Ligaments consist of collagen tissue that connects one bone to another. Their primary function is passive stabilization of the joints. In addition, the ligaments serve an important proprioceptive function.

Ligaments consist primarily of cells, collagen fibers, and proteoglycans. Fibroblasts are the most important cell type, and their main function is to produce collagen (primarily type I but several other types as well). The amount of proteoglycan is much lower than the amount found in cartilage. While the collagen fibers in tendons are organized in a parallel manner (in the longitudinal direction of the muscles), the orientation of the fibers in ligaments can be parallel, oblique, or even spiral (e.g., the anterior cruciate ligament). The organization of fiber direction is specific to the

function of each ligament. In addition, ligaments contain slightly more elastic fibers than tendons.

Ligaments may insert directly or indirectly into the bone: directly with a transition zone consisting of fibrocartilage first and mineralized fibrocartilage last (including specialized collagen fibers that go down into the bone vertically), or indirectly by growing into the surrounding periosteum.

Ligaments may be intra-articular (localized within a joint inside the joint capsule), capsular (where the ligament projects as a thickening of the joint capsule), or extracapsular (localized outside the joint capsule). The cruciate ligaments are intra-articular ligaments. The anterior talofibular ligament is a capsular ligament, where it may be difficult to distinguish between the ligament and the rest of the capsule, whereas the calcaneofibular ligament is an extracapsular ligament. The type of ligament is important for the healing potential after a total rupture. Following total rupture of an intra-articular ligament, such as the anterior cruciate ligament, healing will not take place, whereas the capsular ligaments have excellent healing potential. Blood supply to ligaments also differs. Capsular ligaments have a good blood supply, just as the surrounding joint capsule does, whereas the blood supply to intra-articular ligaments enters proximally or distally, typically resulting in a midzone of marginal vascularization. The blood supply is important for the healing potential after an injury.

Ligaments contain a number of different nerve endings that supply the nervous system with information about body position, movement, and pain. This information is key in controlling the muscles that surround a joint such as the knee. Even if the main function of ligaments is passive stabilization of the joint, much evidence indicates that the proprioceptive function of ligaments is more important than previously thought. Ligament injuries may reduce the ability to register the position and movements of the joint, even when the injury does not result in significant mechanical instability. This may increase the risk of recurrent injuries.

Figure 1.3 shows how ligaments react to stretching. At first, the wavy pattern of the microscopic collagen fibers straightens out and minimal force is required to cause a significant change in length. As force increases further, the collagen fibers will be stretched, and the relationship between load and deformation is linear. This means that the ligament serves as an ideal spring in the elastic zone, as long as the change in length does not exceed about 4%. If a force causes a change in length in excess of this, the collagen fibers will rupture–first single fibers and then all of the fibers will fail (a total rupture). The strength and stiffness of a ligament depends on the longitudinal and cross-sectional area. The greater the cross-sectional area, the stronger and stiffer the ligament. A longer ligament is less stiff, but the maximum tensile strength

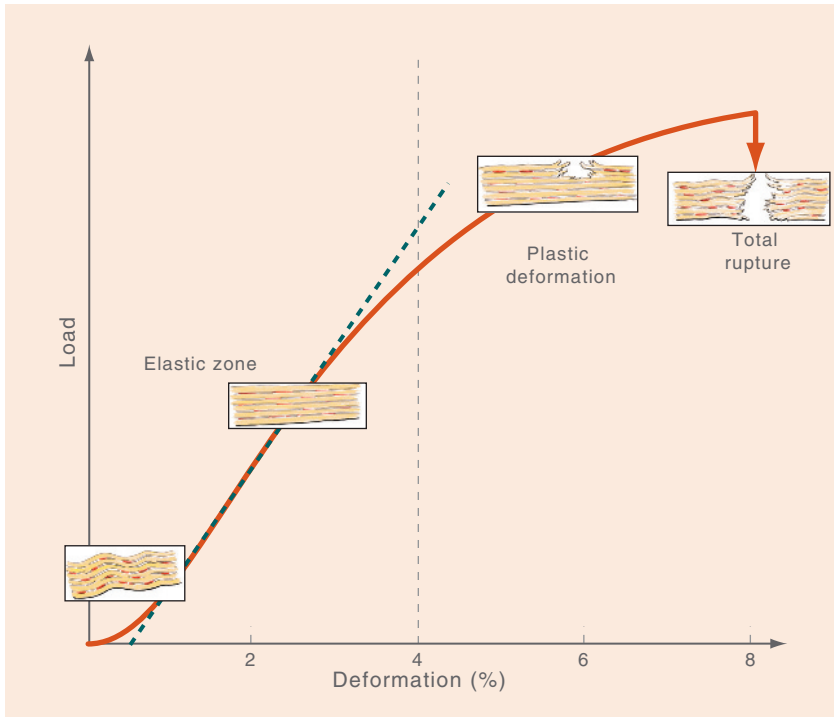

Figure 1.3 Acute stress–deformation curve for ligaments. (© Medical Illustrator Tommy Bolic, Sweden.)

does not change if the cross-sectioned area is the same.

Adaption to Training

Connective tissue adapts slowly to increased loading but weakens rapidly as a result of immobilization (Figure 1.4). The ligaments adapt to training by increasing the cross-sectional area, as well as by changing the material properties so that they become stronger per unit area. Normal everyday activity (without specific training) is apparently sufficient to maintain 80–90% of the ligament's mechanical properties. Systematic training increases ligament strength by 10–20%. In contrast, the negative effect of immobilization sets in quickly. After a few weeks, strength is reduced to about half. Systematic training causes strength in the ligament substance to return after several weeks, but the tensile strength in the ligament–bone junction will remain at a reduced level for several months despite systematic retraining.

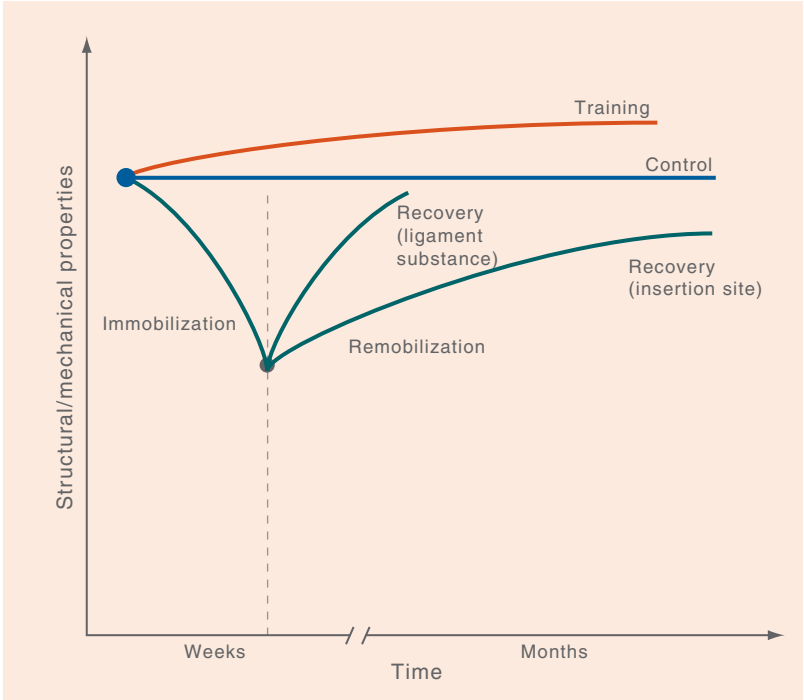

Figure 1.4 Schematic representation of the relationship between training, immobilization, and remobilization on the structural and mechanical properties of the ligaments. (Reproduced with permission from the Norwegian Sports Medicine Association.)

Ligament Injuries

Unlike the tendons, where both acute and overuse injuries can occur, the ligaments are typically injured because of acute trauma. The injury mechanism is sudden overloading, where the ligament is stretched with the joint in an extreme position. For example, inversion trauma in the ankle may cause the lateral ligaments–primarily the anterior talofibular ligament–to rupture.

Ruptures may occur in the midsubstance of the ligament or at the ligament–bone junction (Figure 1.5). Sometimes avulsion fractures also occur, which means that the ligament pulls a piece of the bone with it, usually with an eggshell shape. Several factors determine the location of the rupture, including the age of the patient. Children often sustain avulsion fractures, while midsubstance ruptures commonly occur in adolescents and adults. The ligament–bone junction can be the weak point in middle-aged patients, and avulsion fractures are most common in the elderly, particularly if the skeleton is osteoporotic.

Overuse injuries in the ligaments are rare, and symptomatic inflammatory conditions hardly ever occur. Nevertheless, overuse injuries may occur as the ligament is gradually stretched out, probably because of repetitive microtrauma. One example is the shoulder joint, where throwers (e.g., javelin throwers and baseball, handball, and volleyball players) may stretch out their anterior ligaments. This may reduce stability in the joint and predispose the athlete to pain because of entrapment of the subacromial structures. However, one must be aware that the primary ligament injury (stretching) is usually asymptomatic. The symptoms only appear if the instability causes muscular dysfunction and/or results in injury to other structures (e.g., the rotator cuff in the shoulder).

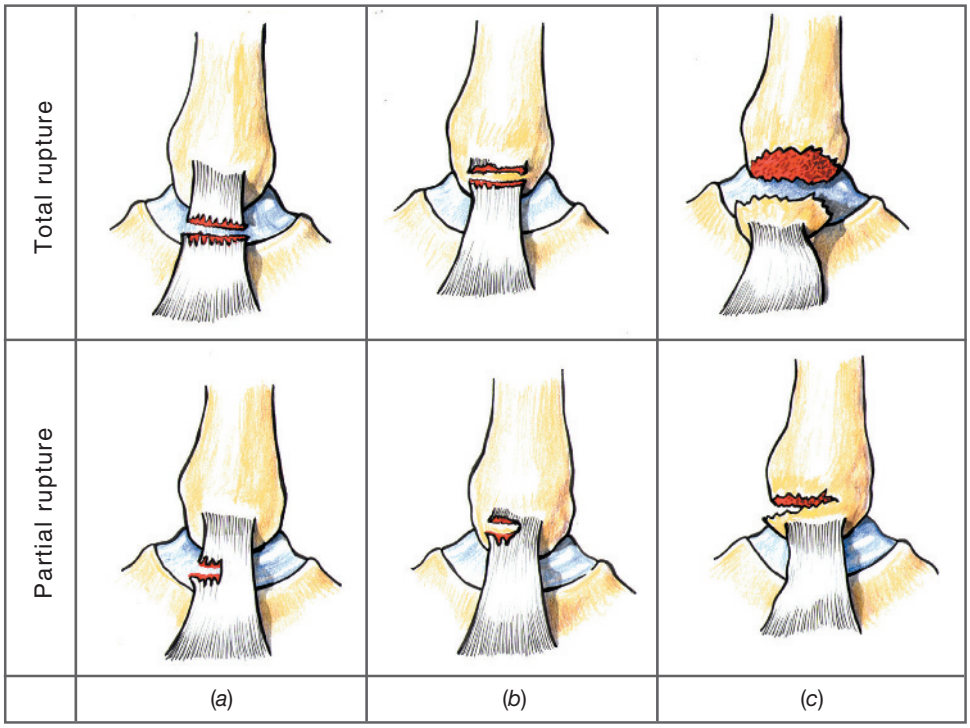

Figure 1.5 Various types of ligament injuries. Total and partial ruptures (*a*) in the midsubstance, (*b*) in the ligament–bone junction, and (*c*) avulsion fractures. (© Medical Illustrator Tommy Bolic, Sweden.)

Internationally, ligament injuries are usually classified as mild (grade 1), moderate (grade 2), or severe (grade 3). Mild injuries only cause structural damage on the microscopic level, with slight local tenderness and no instability. Moderate injuries cause a partial rupture with visible swelling and notable tenderness, but usually with little to no change in stability. Severe injuries cause a complete rupture with significant swelling and instability. Nevertheless, because the relationship between the degree of structural damage, tenderness, and instability is highly variable, this general classification of ligament injuries is very limited for clinical purposes. The use of classification systems developed for the individual ligaments and joints, for which specific tests have been developed, is recommended to grade the degree of the injury. These types of tests and classification systems are described in the discussion of the various regions of the body in Chapters 4–15.

An acute ligament rupture sets off a series of events–the inflammation process–which can be divided into three stages: the inflammatory phase (phase 1), the proliferative phase (phase 2), and the maturation phase (phase 3).

The Inflammation Process

Inflammation ("inflammare" [Lat.]; to set on fire, inflame) is a local tissue response in any vascularized tissue subjected to loading, which results in cell damage. Inflammation consists of a characteristic chain of vascular, chemical, and cellular events that may result in repair, regeneration, or formation of scar tissue. The five cardinal signs of inflammation are *rubor* (redness), *tumor* (swelling), *calor* (heat, increased temperature), *dolor* (pain), and *functio laesa* (loss of function). Among the cardinal signs, pain is generally the most prominent in sport injuries, both as a symptom that the patient experiences subjectively and as a finding, tenderness to palpation. However, it should be noted that painful conditions are not always related to inflammation, as will be described later in the section on tendon injuries. Under normal conditions,

erythrocytes, leukocytes, and plasma components are isolated intravascularly. An injury to the vascular endothelium results in leakage of plasma components, erythrocytes, and leukocytes. The inflammation process is activated by a series of different mediators that primarily result in increased vascular permeability, activation of leukocytes, blood platelets, and the coagulation system (Figure 1.6). Vasoactive mediators bind to specific receptors on endothelial cells and smooth muscle cells. This results in vasoconstriction or dilatation. Neutrophils, granulocytes, monocytes, and lymphocytes are attracted to the injury site by chemotactic factors that are released from the activated platelets and the injured cells. These cells release a series of inflammation mediators. Key among these are growth factors, cytokines, chemokines, prostaglandins, and leukotrienes.

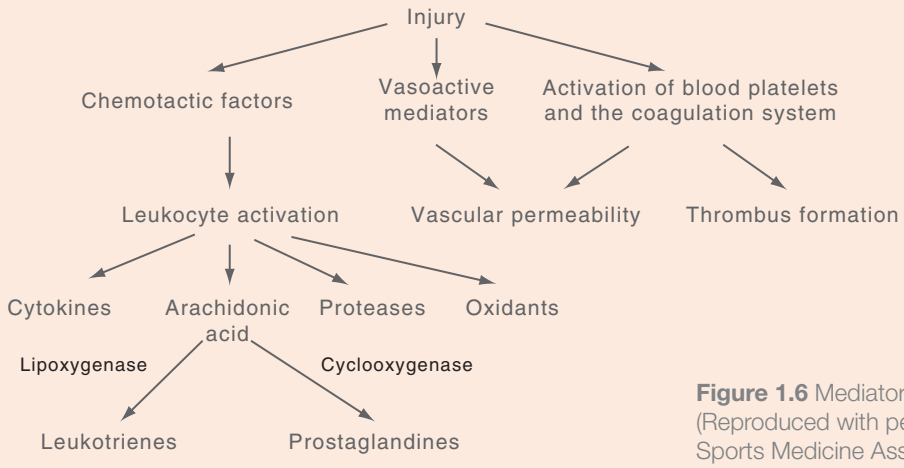

Figure 1.6 Mediators of inflammation. (Reproduced with permission from the Norwegian Sports Medicine Association.)

The Inflammatory Phase (Phase 1)

The inflammatory phase begins with bleeding and the exudation of plasma. Activation of the coagulation cascade causes clotting with a network of fibrin, fibronectin, and collagen blood cells. This network provides some initial strength to the clot. Blood platelets are activated and release a large number of growth factors from their granules. These growth factors function as chemotactic factors recruiting inflammatory cells to the site of the injury. Neutrophil granulocytes release a series of proteolytic enzymes that dissolve the damaged extracellular matrix. Blood platelets and monocytes are recruited into the injured area, invade the tissue and differentiate into macrophages that are actively engaged in the phagocytosis of cell debris and release growth factors that attract pericytes, endothelial cells, and fibroblasts and stimulate cells to the injured area. The inflammatory phase lasts a few days.

The Proliferative Phase (Phase 2)

The proliferative phase is characterized by the accumulation of large numbers of endothelial cells, macrophages, myofibroblasts, and fibroblasts to the site of the injury. Ingrowth of new capillaries (i.e., angiogenesis) begins at the edge of the injury site, and within a few days a rich capillary network supplying oxygen and nutrients is established. The myofibroblasts and fibroblasts organize themselves perpendicular to the direction of capillary ingrowth and an immature granulation tissue is formed. These cells produce an extracellular network that initially consists of fibronectin, type III collagen, and proteoglycans. After a week, the production of type I collagen increases greatly. Some of the fibroblasts transdifferentiate into the contraction-capable cells called myofibroblasts, which are responsible for the scar formation. At the same time, there is continuous breakdown of the initial clot and the injured extracellular early loose connective tissue, and the formation of mechanically stronger newly formed matrix. The macrophages accomplish this by “eating” the superfluous cell components. In addition to that, most of the macrophages transform from inflammatory to anti-inflammatory cells and direct the repair process by secreting growth factors needed for the repair. The continuous deposition and removal of extracellular matrix (with the balance toward deposition) results in remodeling of the injury and increased tensile strength. The proliferation stage lasts a few weeks.

The Maturation Phase (Phase 3)

The final tissue structure is established during the maturation and remodeling stage through continuous remodeling of the scar tissue. The numbers of macrophages and fibroblasts are significantly reduced and the few remaining fibroblasts transform to myofibroblasts, and blood supply is finally established by removal of the capillaries with lowered blood flow and most of the capillaries disappear. The granulation tissue is converted (contracted) by myofibroblasts into a small scar. Thicker collagen fibers are formed in the direction of tension in the tissue from external load, and a network of lateral, cross-bridges providing mechanical strength is established between them. Therefore, the form and function of the scar tissue depend on the degree to which the tissue is subjected to loading during this stage. This stage may last several months, which has important implications for return to sport.

Tendons

Structure and Function

Tendons consist of connective tissue that attaches muscle to bone. Their most important function is to transfer force from the muscles into the skeletal system, thereby contributing to stabilizing the joints. Further, the elasticity of tendon allows for short loading energy stored in the tendon to be released in, for example, jumping activity. Apart from water, the main element in tendons is type I collagen, which makes up 80–90% of the tendinous matrix content. To a large extent, the structure of tendons resembles the structure of ligaments. The collagen is arranged in parallel fibers and the tendons are constructed of increasingly large structures, the tropocollagen, microfibrils, subfibrils, fibrils, and fascicles (Figure 1.7). The strict organization into parallel bundles of various sizes is the main difference between tendons and ligaments. The organization of the ligaments is more variable and dependent on function.

Fascicles are surrounded by a loose connective tissue, endotenon, which makes it easy for them to move in relation to each other. Endotenon also contains veins,

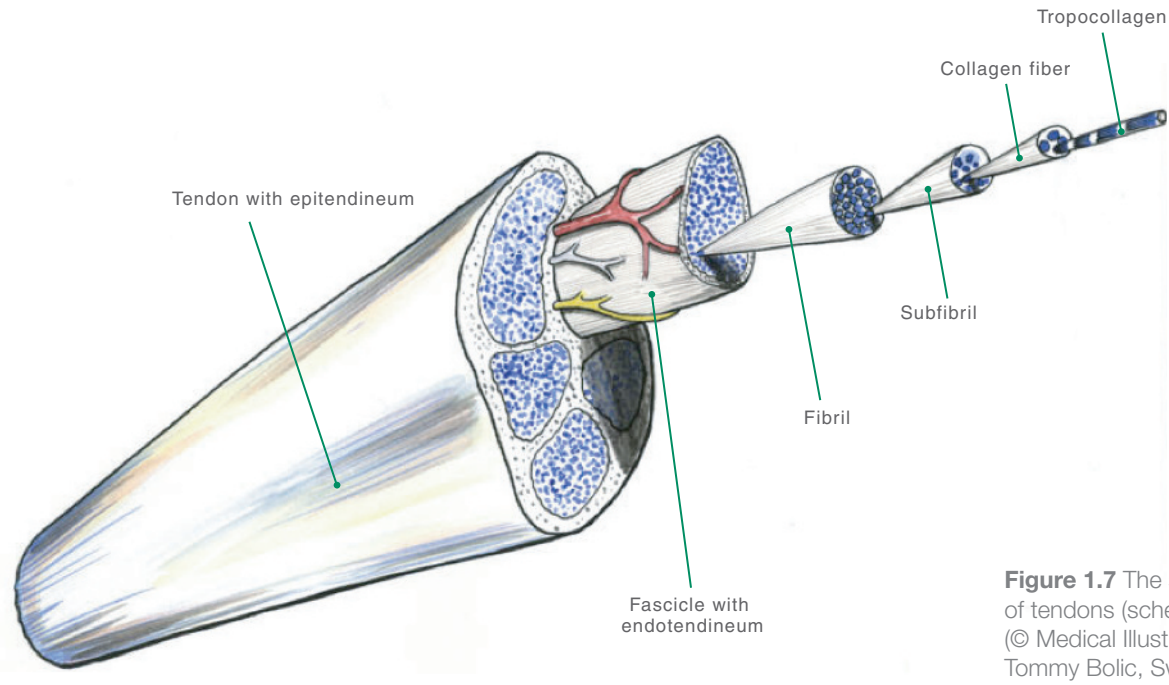

Figure 1.7 The structure of tendons (schematic). (© Medical Illustrator Tommy Bolic, Sweden.)

nerve fibers, and lymph vessels. The surface of the tendon is surrounded by a white synovial-like membrane, the epitenon, a loose connective tissue that also supports blood vessels, lymphatics, and nerves. Some tendons are covered by a loose areolar connective tissue, the paratenon, enveloping the tendon. The envelope of tendon is dominated by type IV collagen and acts as an epithelium hindering the tendon to adhere to surrounding tissues.

The muscle cell ends in a number of microscopic membranous infoldings that stick out like small fingers into the myotendinous junction. The collagen fibers creep into the folds that form between the fingers and attach to the basal membrane of the muscle. At the other end, the tendons attach to bone via fibrocartilage and mineralized fibrocartilage. Collagen fibers penetrate the mineralized fibrocartilage into the subchondral bone, contributing to better attachment.

The relationship between stress and deformation of tendons is the same as for the ligaments (Figure 1.3). Initially, the collagen fibers are easily stretched from their normal wavy appearance, in the elastic zone the tendon behaves like an ideal spring, whereas ruptures occur in the deformation zones: first single fibers, then total ruptures.

Adaptation to Training

The tendons adjust to training in the same manner as the ligaments–by increasing the tendon strength through collagen synthesis, cross-link formation and training improved material properties of the tendinous tissue, and if trained sufficiently some increase in tendon cross-sectional area can be seen. Acute exercise results in an increase in collagen synthesis within and around the tendon tissue. Collagen synthesis remains increased for 2–3 days, indicating that training every second or third day is most likely a sufficient stimulus for tendon protein generation. In addition, the relative load intensity required is less than in muscle, which means that also moderate exercise, either concentric or eccentric, will result in elevated formation of new collagen in tendon. Changes in physical activity levels, either increased training or detraining/immobilization, quickly (within 1–3 weeks) alter mechanical properties, most likely through increased or decreased cross-link formation, respectively. In contrast, changes in collagen-rich fibril structures require several months to years to occur.

Tendon Injuries

Tendons can be injured in several different ways, both as acute injuries and as overuse injuries. Because tendons are usually superficial, they can be severed by a penetrating stab or a cut, such as one caused by the edge of a skate. Acute tendon ruptures occur if force is generated in excess of the tendon's ability to tolerate it. These types of tendon ruptures usually occur in connection with eccentric force generation, such as in the Achilles tendon when pushing off at the start of a sprint run. Tendon ruptures may be partial or total, and they usually occur in the midtendon substance but may also occur in the bone–tendon junction or as avulsion fractures. Acute tendon injuries are most common in athletes and recreational exercisers between 30 and 50 years of age in explosive sports, often without previous symptoms or warning. Some studies reveal that structural and degenerative changes can be seen in the tendon prior to exercise.

Tendons are the type of tissue that is most often affected by overuse injuries. Several different terms are habitually used to describe these overuse injuries: tendinitis (tendon inflammation), tenosynovitis (tendon sheath inflammation), tenoperiostitis

(inflammation of tendon insertions and origins), periostitis (periosteal inflammation), and bursitis/hemobursitis (bursal inflammation, possibly with bleeding). All these terms describe the parts of the tendon or the surrounding tissue that is affected, and all have the ending "itis," indicating the pathophysiological condition of inflammation.

Even though the concept of inflammation has been used traditionally, the pathogenesis for overuse injuries in tendons is uncertain. Although tendon loading does not normally cause more than a 4% change in length (i.e., within the physiological elastic zone), some sports require repetitive loading in excess of this (4–8% change in length), which may cause collagen fibrils to rupture. Therefore, a potential explanation of what is called tendinitis is that repetitive microtrauma causes injuries that are greater than the fibroblasts are able to repair, resulting in inflammation. It is also possible that cumulative microtrauma can affect collagen cross-bridges, other matrix proteins, or microvascular elements in the tendon. Also, loading that extends the tendon less than 4% can lead to overuse symptoms, and it is likely caused by inadequate time to adapt to each training load.

One problem with explaining tendon overuse as inflammation is that the histological findings do not match those seen with inflammation–surgical specimens are devoid of inflammatory cells. However, degenerative changes, changed fibril organization, reduced cell count, vascular ingrowth, and, occasionally, local necrosis with or without calcification are seen. The concept of tendinosis was introduced to describe these types of focal degenerative changes. Because the relationship between degenerative changes and symptoms is unclear, the terms "tendinosis" or "tendinopathy" are now commonly used to describe chronic tendon pain. Table 1.1 provides an overview of old and new terminology for tendon disorders and injuries. The new terminology emphasizes the need for the terminology to correspond to the histological findings.

New	Old	Definition	Histologic findings
Paratenonitis	Tenosynovitis Tenovaginitis Peritendinitis	An inflammation of only the paratenon, either lined by synovium or not	Inflammatory cells in paratenon or peritendinous areolar tissue
Paratenonitis with tendinosis	Tendinitis	Paratenon inflammation associated with intratendinous degeneration	Same as above, with loss of tendon collagen, fiber disorientation, scattered vascular ingrowth, but no prominent intratendinous inflammation
Tendinosis	Tendinitis	Intratendinous degeneration due to atrophy (aging, microtrauma, vascular compromise, etc.)	Noninflammatory intratendinous collagen degeneration with fiber disorientation, hypocellularity, scattered vascular ingrowth, occasional local necrosis, and/or calcification
Tendinitis	Tendon strain or tear (a) acute (less than 2 weeks) (b) subacute (4–6 weeks) (c) chronic (over 6 weeks)	Symptomatic degeneration of the tendon with vascular disruption and inflammatory repair response	Three recognized subgroups. Each displays variable histology from pure inflammation with hemorrhage and tear, to inflammation superimposed upon preexisting degeneration, to calcification and tendinosis changes in chronic conditions. In chronic stage there may be: interstial microinjury central tendon necrosis frank partial rupture acute complete rupture

Table 1.1 Terminology for tendon disorders and tendon injuries.

Bone

Structure and Function

The skeleton consists of bone, a special type of connective tissue that remodels continuously as a response to a complex interplay between mechanical loading, systemic hormones, and the calcium level in the blood. Bone may be classified as cortical (compact) or trabecular (spongy), and the two types of bone have different functions and properties. The long bones consist primarily of cortical bone, whereas the vertebrae in the spinal column consist of trabecular bone. Bone has many important functions, such as protecting the underlying organs, serving as the body's major calcium store, and providing the environment for hematopoiesis in marrow. However, in relation to injuries, the skeleton's most important function is as a lever in the locomotor apparatus.

Like other connective tissue, bone consists of cells, collagen fibers, and extracellular matrix. Bone cells develop from stem cells in the bone marrow, primarily as osteocytes, osteoblasts, or osteoclasts. The osteoblasts and osteoclasts are responsible for remodeling bone. Located on bone surfaces, osteoblasts are bone-forming cells. When an osteoblast has formed enough bone to be completely surrounded by a mineralized matrix, it is called an osteocyte. Osteoclasts are also found on the surface of bone–their job is to absorb bone. Osteocytes communicate with each other and with osteoblasts and osteoclasts on the surface through channels in the extracellular matrix, and this is an important signaling path from mechanical loading to remodeling. A recommended daily intake of minerals (calcium and magnesium) and vitamin D is necessary for optimum remodeling of bone.

The extracellular bone matrix consists of both organic and inorganic components. The inorganic component constitutes more than half the bone mass and consists primarily of calcium and phosphate as crystals of hydroxyapatite. The inorganic components contribute greatly to the characteristic hardness and strength of bone. Strength increases with increasing bone mineral density, but skeletal architecture is also very important. The main organic component is collagen, which contributes to bone's elastic properties.

The skeletal surface is covered by a thick layer of fibrous connective tissue, called periosteum. Periosteum has a rich supply of nerves and blood. For this reason, direct trauma that causes bleeding in or underneath the periosteum can be very painful. Periosteum is particularly well attached to bone in areas where muscles, tendons, and ligaments attach to the skeleton. In these areas, collagen bundles (Sharpey's fibers) go down from the periosteum and into the underlying osseous tissue.

The longitudinal growth of the skeleton takes place in the growth zones (the physes) (Figure 1.8). The growth zones are subject to injuries: 15% of all acute fractures

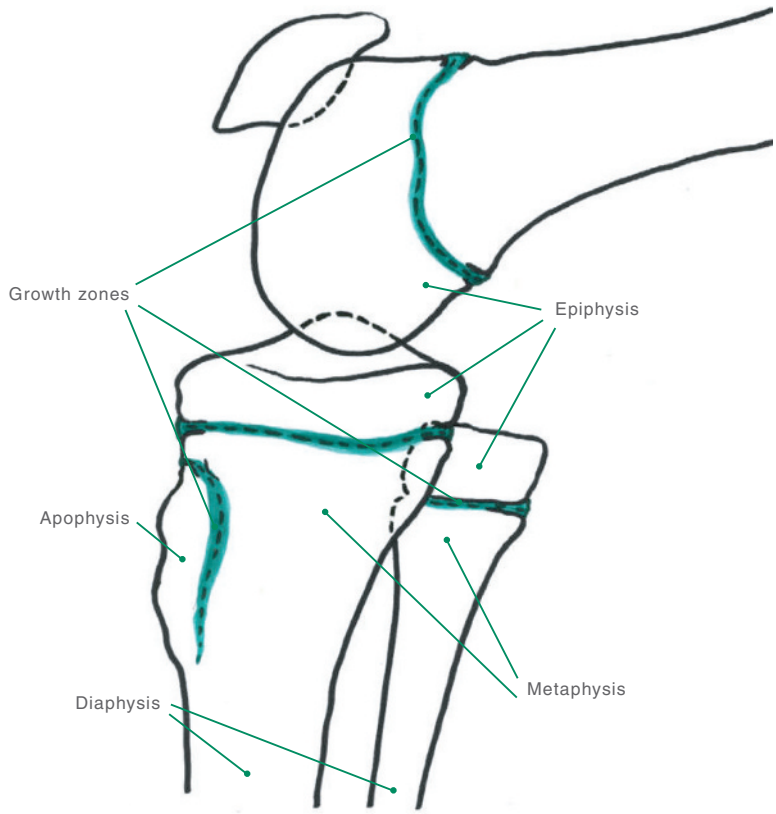

Figure 1.8 Growth zones in the tubular bones, example from the tibia, fibula, and femur. The physes are vulnerable to injuries during the growth spurt. (© Medical Illustrator Tommy Bolic, Sweden.)

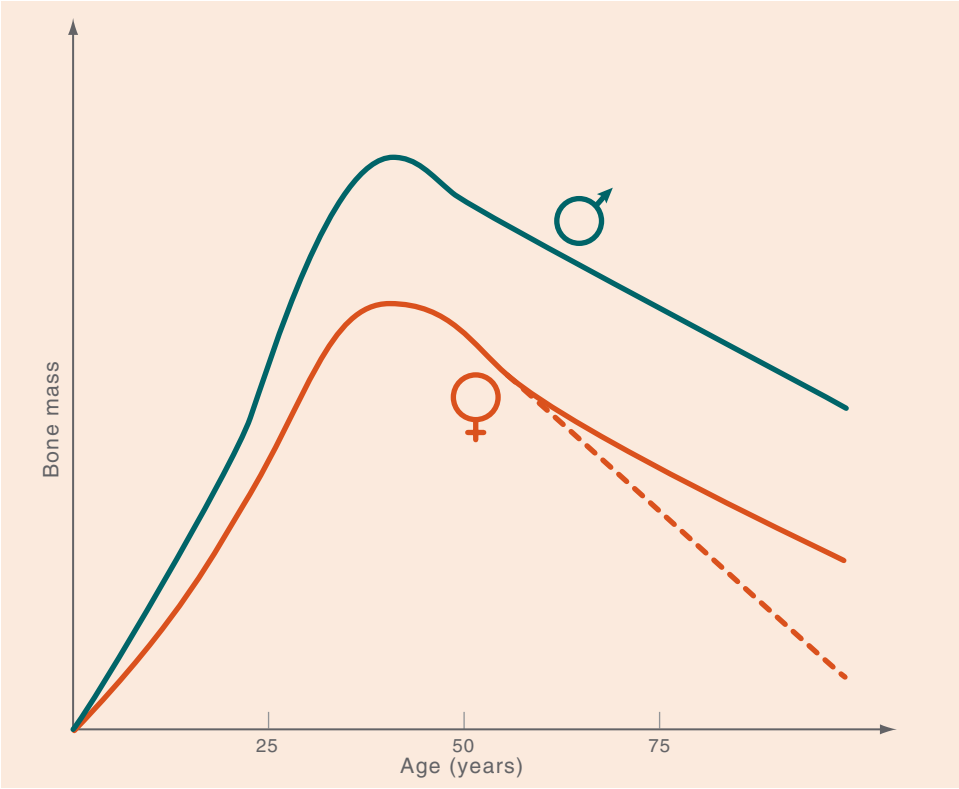

Figure 1.9 The development of bone mass as a function of age and sex. The dotted line shows the potential development in osteoporotic women after menopause. (Reproduced with permission from the Norwegian Sports Medicine Association.)

in children involve the physes. In addition, the apophyses are subject to overuse injuries during the growth spurt. The combination of rapid development of muscle strength and a large amount of training leads to physeal overuse injury (e.g., the quadriceps muscle, as in Osgood–Schlatter disease, and the triceps surae muscle, as in Sever disease insertions). Bone mass also increases during the growth period and peaks when the athlete is in her third decade (Figure 1.9). After a period where bone mineral remains stable at best, density decreases quite rapidly (1% per year or more) in most women after menopause.

Bone has characteristic stress–deformation curves (Figure 1.10). Initially, in the elastic zone, there is a linear relationship between load and deformation. If the load increases into the plastic deformation zone, even small changes in force will cause greater and greater deformation. Greater loading in the deformation zone results in a complete fracture.

Adaptation to Training

When considering the effect of physical training on bone, it is important to consider both the material property of bone ("bone mass") as well as the geometric properties (bone shape and size). Bone is a structure (like a building or a bridge) and its strength depends both on the material it is made of (bone mass in this case) and the shape in which the material is arranged (geometry). That's why there is an emphasis on estimating the effect of physical training on bone strength–the load that a bone can withstand before fracturing. Bone strength includes aspects of both bone mass and bone geometry.

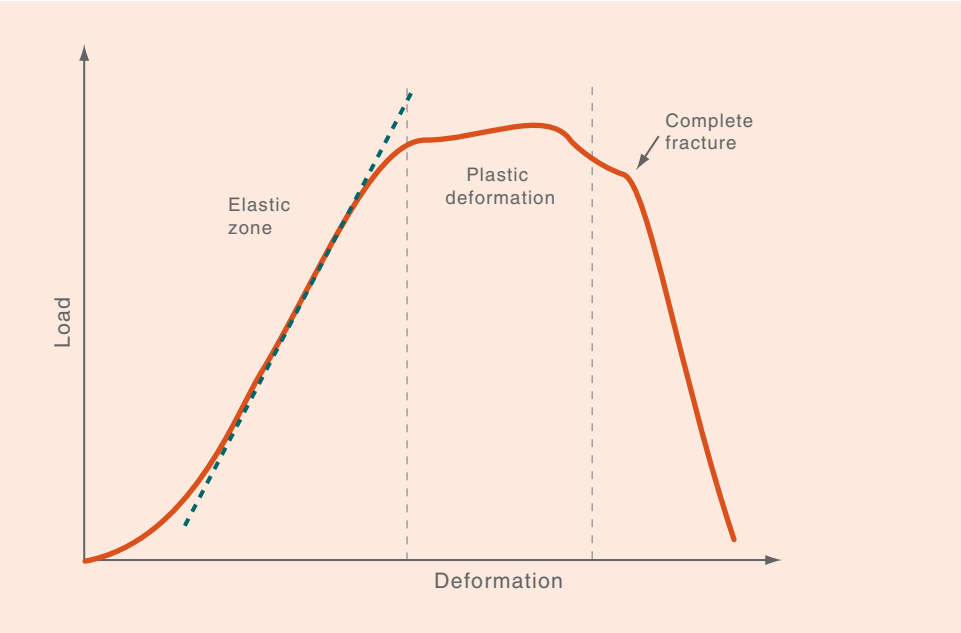

Figure 1.10 Acute stress–deformation curve for bone. (Reproduced with permission from the Norwegian Sports Medicine Association.)

Physical training increases bone mass (which can be measured as bone mineral density using a DXA scanner) and bone geometry (measured using a peripheral CT scanner). Training-related increases in bone strength are site specific to loaded bone. Jumping will not improve upper limb bone strength, tennis increases strength of the dominant arm only. Importantly, not all types of activity increase bone mass. Bone responds to fast signals—rapid deformation—not to slow, gentle loads.

Athletes in power and jumping sports, such as weight lifters, gymnasts, volleyball players, and squash players, have greater bone strength, all other things being equal (e.g., size and sex), than other athletes. Normal weight runners are in the midrange of athletes for bone strength; cyclists and swimmers have no higher bone mineral density than control groups. This pattern emphasizes the need for impact loading to promote bone strength.

With respect to the trajectory of change in bone strength over time, bone responds maximally to physical activity during the growing years. In just two peripubertal years (age 10–12 approximately in girls and 11–13 in boys), the individual can accumulate 25% of adult bone mass.

During the adult years (20s to 40s) intense training leads to preservation of bone strength—bone mass is retained and structural (shape) changes occur to maintain bone strength. In the postmenopausal years, strength training can largely prevent the natural decline in bone strength that occurs in nonexercising women. Thus, compared with women who do not exercise, older exercising women have a *relative* net benefit in bone strength because they avoid the loss that is "physiological" in their nonexercising counterparts.

Fractures

Fractures can be classified in various ways, but the most important difference is between acute fractures and stress fractures. Acute fractures are caused by trauma

that exceeds the tissues' ability for tolerance, direct trauma (e.g., a kick to the leg), or indirect trauma (e.g., twisting of the lower leg) (Figure 1.11).

Acute fractures can be broadly classified as transverse fractures, crushing fractures, oblique fractures, and compression fractures, depending on the type of force that caused the fracture, which usually contributes to giving them their characteristic appearance. Transverse fractures are generally caused by direct trauma to a small area, commuted fractures are caused by greater direct trauma to a larger area, oblique or spiral fractures are caused by indirect trauma with twisting (rotational, torsional) of the bone, and compression fractures are caused by vertical compression of the bone (e.g., by the femoral condyle being pressed down into the tibial plateau). Tearing of the tendon or ligament insertion causes avulsion fractures. In addition, two special types of fractures occur in children: (1) "greenstick fractures" (in which the bone is "bent" like a soft twig) and (2) epiphyseal plate fractures (loosening of and possibly a fracture through the growth zone).

Diagnostic signs of fractures are malalignment, abnormal movement, or shortening of an extremity. Pain, swelling, and reduced range of motion (ROM) are usually also present, but are less specific signs.

Figure 1.11 Torsional trauma like this can cause a fracture. (© Oslo Sports Trauma Research Center.)

Unlike acute fractures, stress fractures do not necessarily have any specific triggering trauma. In addition, there is a continuum of clinical reactions to loading. As mentioned in the preceding text, bone remodels continuously throughout life. Increased loading results in microinjuries, circulatory injuries, and accelerated remodeling, with increased osteoclast and osteoblast activity. At first, symptoms are absent despite accelerated remodeling. Routine X-rays do not demonstrate any changes, although magnetic resonance imaging (MRI) will demonstrate bone marrow edema, and scintigraphy will demonstrate increased uptake of technetium. If excessive loading continues, mild pain will set in a while after the training session begins, and eventually earlier and earlier into the training session. This is different from pain from soft tissues (such as the tendons), which typically occurs at the beginning of training and usually decreases after warm-up. Continued training will increase the intensity of pain, so that the pain will also be present after training and during other activities such as regular walking. In these cases, both MRI and scintigraphy will usually be positive, whereas plain X-rays often do not show any changes except a subtle periosteal reaction. Positive X-rays will, of course, be seen if there is a complete fracture. The development of stress fractures represents a physiological and clinical continuum from normal remodeling via accelerated remodeling, stress reaction, and stress fractures to complete fractures. Early diagnosis reduces treatment time.

As with other loading injuries, a combination of factors contributes to stress. Key among these are training errors ("too much, too often, and too quickly, and with too little rest"), muscle fatigue (which presumably affects the shock-absorbing ability of the foot when running), and malalignment in the lower extremities, surface, and equipment (particularly footwear). If training is accurately documented, it will usually be seen that the athlete has made significant changes in training during recent weeks. Menstrual and eating disorders can cause reductions in bone mineral density and increase the risk of stress fractures.

Cartilage

Structure and Function

Cartilage consists of the basic elements in connective tissue, cells, and extracellular matrix. There are three types of cartilage—elastic, hyaline, and fibrocartilage—of which hyaline is the most important. Hyaline cartilage consists of several layers characterized by a horizontal organization of cells in the extracellular matrix in the surface layer and a vertical organization in the deeper layers (Figure 1.12).

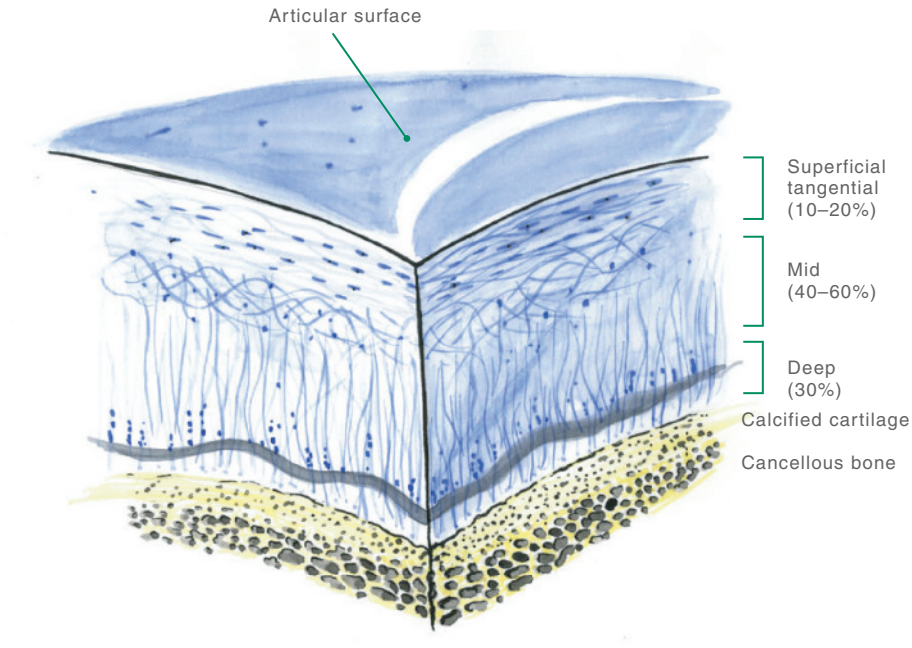

Figure 1.12 Structure of cartilage. (© Medical Illustrator Tommy Bolic, Sweden.)

The articular surface of most joints is covered by hyaline cartilage that is 1–5 mm thick. Cells constitute less than 10% of the volume of the hyaline cartilage, the remainder consisting of macromolecules

(20%) and water (70%). The macromolecules are primarily collagen fibers and proteoglycan. Cartilage strength is mainly due to collagen–primarily type II, which is organized like a network of long fibrils. The proteoglycans are woven into this network and have two important properties: (1) they bind water and (2) they are negatively charged, so that they repel each other. This causes the cartilage to naturally absorb water and swell up. The amount of proteoglycan and water is greater in younger than in older athletes.

Hyaline cartilage does not have a nerve supply, blood supply, or lymph drainage. The cartilage cells obtain oxygen and nutrients from the surrounding tissue and articular fluid and dispose of waste matter through diffusion. When a joint is loaded so that the cartilage surfaces are pressed against each other, the fluid is pumped out. The cartilage receives its nutrient supply through this process of cyclic loading and unloading. Another key element of joint function is that the filmy synovial fluid between the two hyaline cartilaginous surfaces makes friction very low, as low as wet ice on glass.

To understand the relationship between loading and deformation of hyaline cartilage, it is important to remember that the collagen fibers are organized as a network–horizontally on the surface, more multidirectional in the middle section, and more vertical in the deep layer. When loading begins, the fibers will be organized in a wavy pattern (Figure 1.13). Eventually, the fibers will straighten out, and deformation increases linearly with the increase in load until tearing occurs–initially among individual fibers and later among larger groups of fibers.

Fibrocartilage is strong and flexible; and it is located near joints, tendons, ligaments, and in the intervertebral disks, where it forms a protective surface between the tendons, ligaments, and bone. Therefore, fibrocartilage is primarily found in larger joints, such as the hip, shoulder (glenoid lip), knee (menisci), and wrist (triangular fibrocartilage complex). In the knee, fibrocartilage contributes to improving the articular congruence between the hyaline cartilaginous surfaces and to absorbing shock, whereas in the hip, shoulder, and wrist it contributes to expanding the articular surface, as well, thereby increasing stability. Unlike hyaline cartilage, fibrocartilage can have a blood and a nerve supply. For example, the nucleus fibrosis has a

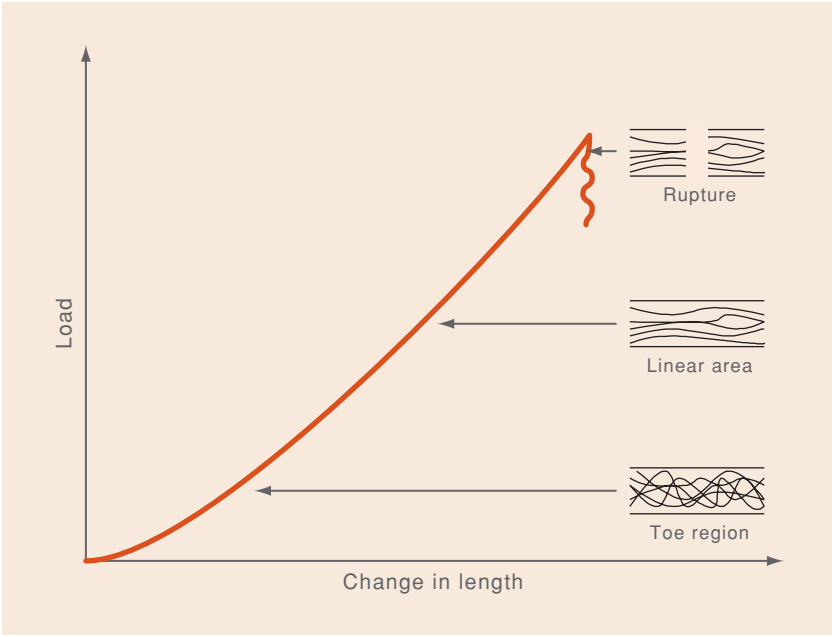

Figure 1.13 The stress–deformation curve for hyaline cartilage shows the relationship between loading and changes in length. (Reproduced with permission from the Norwegian Sports Medicine Association.)

nerve supply in the outer superficial portion, whereas the menisci in the knees have a blood supply in the inner capsular portion.

Adaptation to Training

Active loading of the articular cartilage causes the nutrients to diffuse in and outside of the cartilage. Consequently, regular loading is necessary for normal cartilage function. Cartilage adapts to activity (Figure 1.14). Immobilization, such as in a cast, reduces function. It is also assumed that too much loading reduces biological properties.

Cartilage Injury

In acute injuries, hyaline cartilage can be destroyed through contusion, which causes cracks, or when shearing forces in the joint cause vertical or horizontal rifts. Cartilage injuries occur often in connection with acute joint injuries. Patients with acutely sprained ankles that result in lateral ligament injuries often have macroscopic cartilage injuries. Of patients who have an arthroscopic examination after having sustained an acute knee ligament injury, full-thickness cartilage injuries are common. Some patients have an isolated cartilage injury; others have osteochondral injuries in which the underlying bone is also injured.

Articular cartilage injuries are classified on the basis of the size and depth of the lesion and the cause and accompanying pathology of the injury. The most important is to distinguish between degenerative cartilaginous injuries (osteoarthrosis), where changes are found at several places in the joint, and focal articular cartilage injuries, where localized changes are found in one or two places in the joint. If the patient has osteoarthrosis, hyaline cartilage degeneration, sclerosis of the underlying bone, and the development of ossified cartilage in the outer edges of the joints (osteophytes) occur. Large acute ligament injuries, such as anterior cruciate ligament injuries, increase the risk of secondary osteoarthrosis later on. However, it is not known whether this occurs because the acute injury starts the degenerative processes in

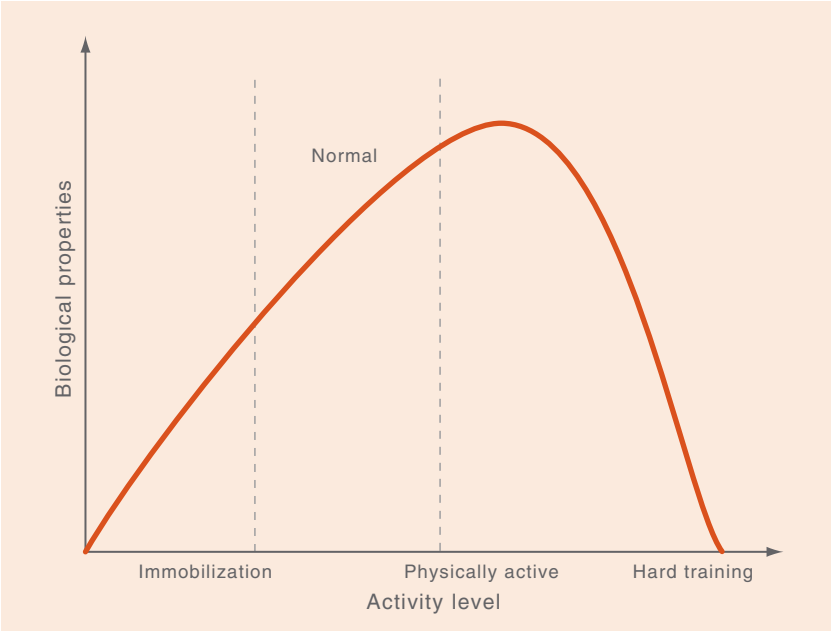

Figure 1.14 Hypothetical relationship between physical loading and the development of biological properties in hyaline cartilage. (Reproduced with permission from the Norwegian Sports Medicine Association.)

the knee joint or because the loading pattern in the knee is changed as a result of increased laxity. The cause of primary osteoarthrosis is still unknown, yet the process may be due to increased loading of a normal joint or to cartilage failure despite normal loading. Even without a recognized injury, it appears that the occurrence of osteoarthrosis is more prevalent in former athletes than in the general public.

The ability of hyaline cartilage to repair is limited after injuries. This is attributed to the lack of blood and nerve supply and the relative lack of cells in the cartilaginous tissue. The inability to regenerate increases the risk that osteoarthrosis will develop after a cartilage injury.

Fibrocartilage is also regularly injured in meniscal injuries and labrum injuries. In most cases, these injuries are acute, but degenerative changes also occur. The blood supply to fibrocartilage varies. In the meniscus of the knee, blood supply is good in the capsular portions ("red meniscus"), where the possibilities for repair are good. However, central portions ("white meniscus") have a less good blood supply and consequently poor potential to repair.

Muscle

Structure and Function

Muscles make up 40–45% of body mass. The structure of the musculature (Figure 1.15) reflects its central function–to generate power. The muscle fibers

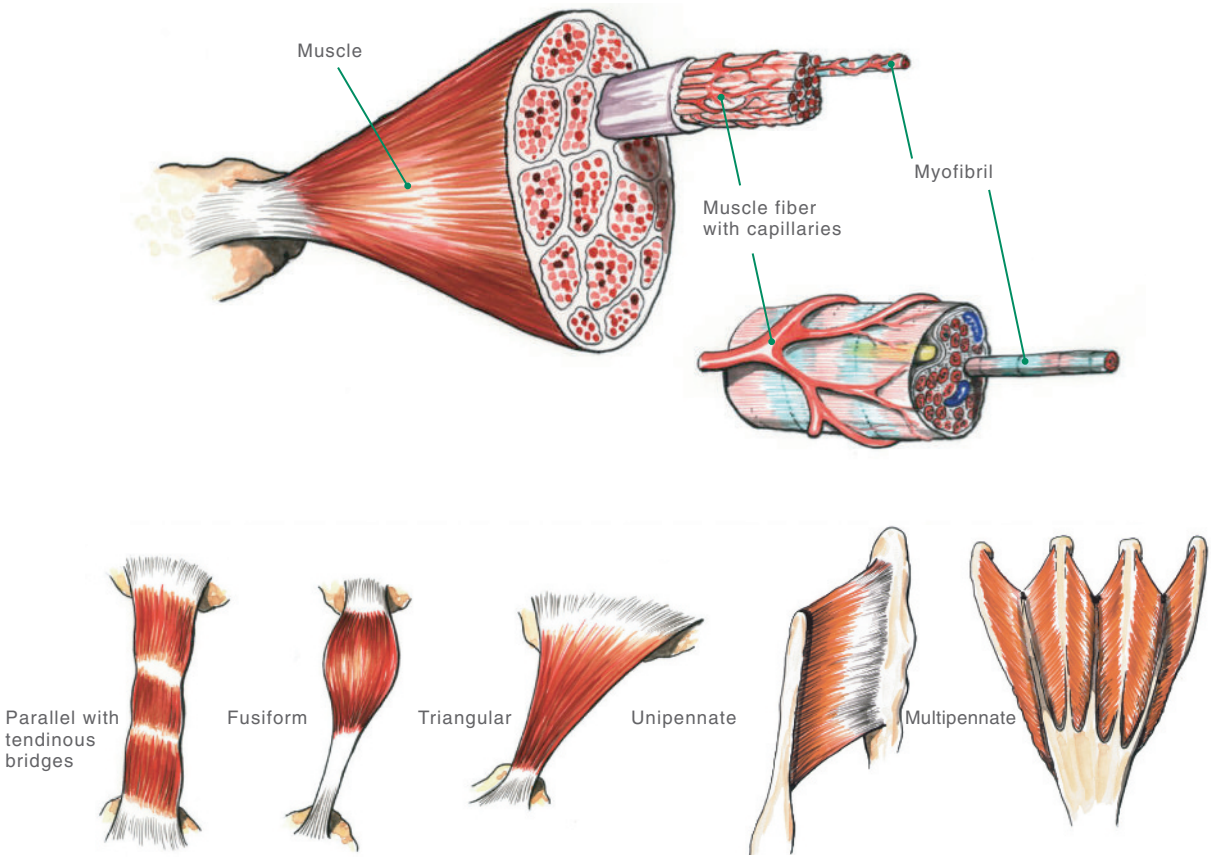

Figure 1.15 Schematic overview of the structure of musculature. (© Medical Illustrator Tommy Bolic, Sweden.)

(muscle cells) are the muscles' central unit, and these can be organized in several ways, such as unipennate, multipennate, or fusiform patterns. Pennate muscles are generally stronger than fusiform muscles, because several muscle fibers can work parallel to each other. However, because they contain shorter fibers, the maximum contraction speed is lower. The striated muscle cell is a fiber with a diameter of 10–100 µm and a length up to 20 cm. The primary elements in the muscle fibers are myofibrils, which are composed of protein filaments (mainly actin and myosin). Capillaries surround the muscle fibers, so that the ability to supply the fibers with oxygen and nutrients is very good.

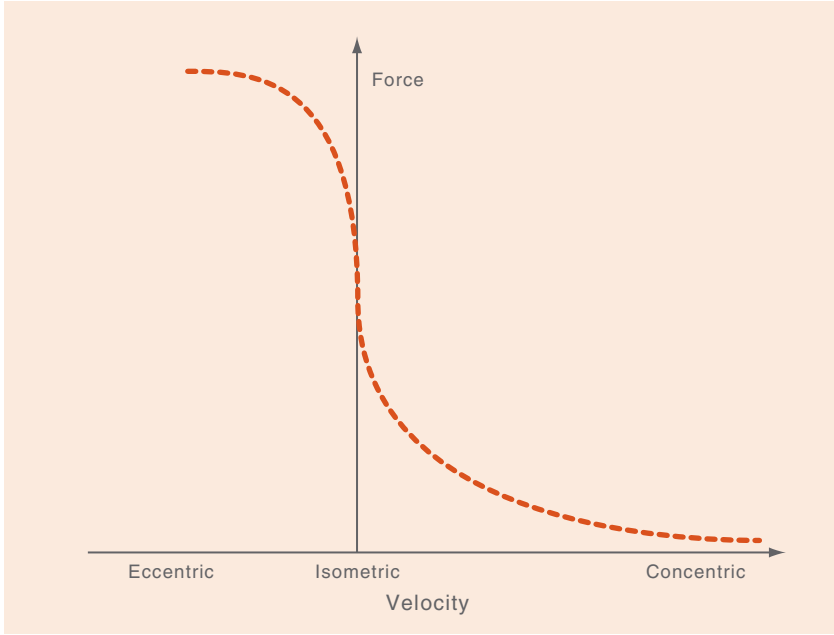

Figure 1.16 The relationship between force and speed in different types of muscular exertion. (Reproduced with permission from the Norwegian Sports Medicine Association.)

The ability to generate force depends on the working conditions, as shown in Figure 1.16. The generation of force without changes in the joint angle is called an "isometric" or "static" muscle action (the length of the muscle is constant, but the tension changes), whereas the muscle contraction where the length changes but tension remains constant is called "isotonic." The generation of power while the muscle is shortened is called "concentric" muscle contraction, whereas the term "eccentric" is used when the muscle is extended while it offers resistance. For concentric muscle action, maximal muscle force is reduced when the speed of contraction increases, whereas in eccentric muscle activity, muscle force increases with increasing speed. This means that the risk of muscle injuries is greater with eccentric than with concentric muscle action.

That working conditions play a decisive role in the generation of force can be illustrated by comparing various types of jumps. Figure 1.17 shows a notable difference between the generation of force against the surface from a squat jump (a strict concentric jump from a 90° knee bend), a countermovement jump (a continuous eccentric–concentric movement), and a drop jump (jumping after dropping down from a height). The greater force generated from a drop jump significantly increases the risk of acute strains, and the risk of overuse injuries is high in sports characterized by this type of muscle action. This is true not only of the muscles but also of other structures, such as tendons, cartilage, and bone.

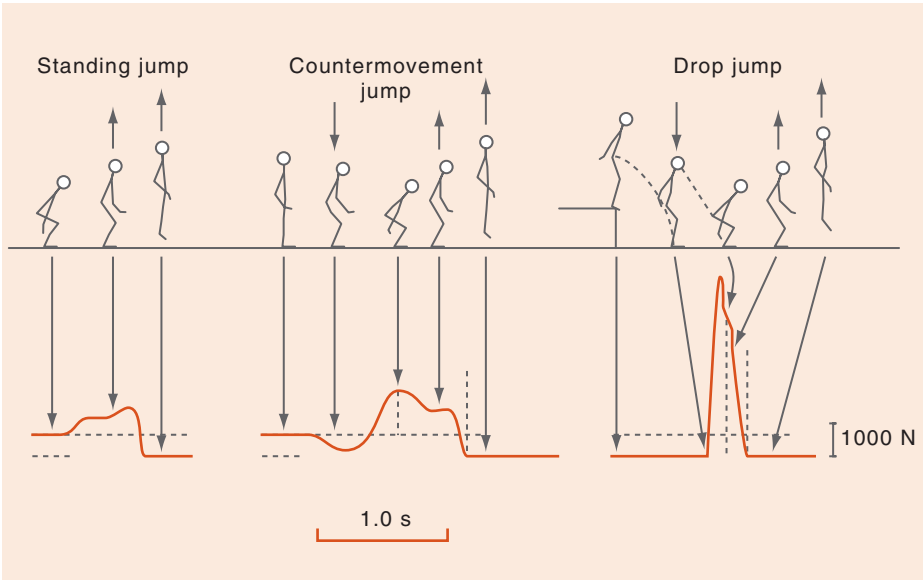

Figure 1.17 Force generation in various types of jumps. (Reproduced with permission from the Norwegian Sports Medicine Association.)

Adaptation to Training

Muscle is the tissue that shows the greatest and most rapid response to training. Muscle volume and strength increase significantly after a short period of specific strength training (Figure 1.18). Two factors contribute to increasing strength: (1) the ability to recruit several muscle fibers at the same time for the contraction (neural factors) and (2) muscle volume (muscular factors). Muscle volume primarily increases as a result of individual muscle fibers increasing their cross-sectional area (hypertrophy), and also by forming new muscle cells (hyperplasia) from stem cells (satellite cells) in the musculature. Neural factors contribute most to the initial strength increase, whereas hypertrophy is primarily responsible for the subsequent strength increase.

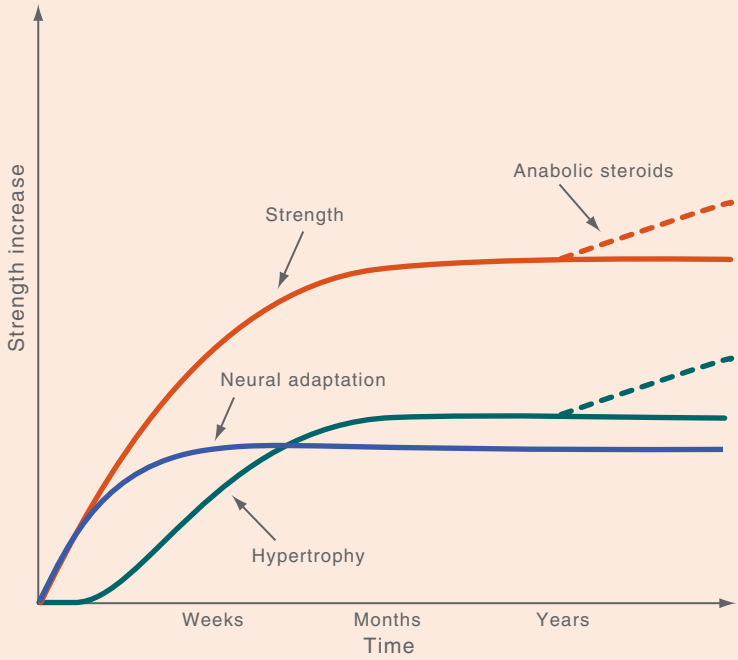

Figure 1.18 Increase in strength as a result of systematic strength training. (Reproduced with permission from the Norwegian Sports Medicine Association.)

The enhancement of endurance capacity of muscles, in turn, involves training-induced increases in the oxidative capacity of the muscles (e.g., increased capillary density and number of mitochondria). Both main types of training, endurance (low-intensity, high volume) and strength (high-intensity, short duration) training, are known to improve the energy status of working muscle, subsequently resulting in the ability to maintain higher muscle force output for longer periods of time. Recent experimental data demonstrate that strength training can also lead to enhanced long-term (>30 min) and short-term (<15 min) endurance capacity in well-trained individuals and elite endurance athletes when high-volume, heavy-resistance strength training protocols are applied. The enhancement in long-term endurance capacity appears to involve training-induced increases in the proportion of type IIA muscle fibers, as well as gains in maximal muscle strength and rapid force characteristics, while also likely involving enhanced neuromuscular function.

Because strength increases after a few weeks but tendons, cartilage, and bone require months to adjust, there is a danger that overuse injuries will occur in these structures in connection with the beginning of systematic strength and jump training. The patellar tendons and the Achilles tendons are examples of structures that are especially vulnerable in adult athletes. This is particularly true when the patient uses anabolic steroids, where there seems to be an increased risk for a total rupture of muscle or tendons (e.g., of the quadriceps or the pectoralis major) is present. In children and adolescents (e.g., Osgood–Schlatter disease and Sever disease), these types of overuse problems usually affect apophyseal disks.

Muscle Injuries

Muscle injuries generally occur in two ways: (1) distension ruptures (pulled muscles, i.e., strains) and (2) by direct trauma that results in contusion ruptures. Muscular

lacerations also occur, although they are rare in sport. In addition, the musculature is sometimes injured as a result of unusual and hard training, especially eccentric training. This may cause muscular soreness called delayed onset muscle soreness (DOMS).

Distension ruptures (strains) usually occur close to the myotendinous junction in connection with maximum eccentric muscle action, such as in sprinters. The usual locations are the hamstrings, adductor, and gastrocnemius muscles, but ruptures may affect a large number of muscle groups. The athlete experiences immediate pain from the muscle at the moment of impact, followed by tenderness and reduced contraction strength. The athlete can sometimes feel a bump in the muscle right away. Eventually, this is replaced by swelling due to bleeding.

Contusion ruptures primarily occur in the quadriceps muscles, which are exposed frontally and laterally on the thigh and, therefore, can easily be hit, for example, by an opponent's kneecap. The severity of contusion injuries varies from very mild strain injury like DOMS to "real" strains, shearing type of muscle injuries, in which myofibers and the associated connective tissue structures including blood vessels are ruptured. Muscle injuries involving rupture of blood vessels cause internal bleeding in the musculature. This is because the musculature is well vascularized and the blood flow is usually high when the injury occurs. Therefore, a hematoma will occur almost instantly with this type of injury. Bleeding may be either intramuscular, if there is no injury to the muscle fascia, or intermuscular, if the blood can escape from the muscle compartments through an injured fascia (Figures 11.1 and 11.4). In general, healing time is significantly longer with intramuscular bleeding than it is for intermuscular bleeding.

What distinguishes the healing of injured skeletal muscle as well as the other soft tissues from that of fractured bone is that the skeletal muscle heals by a repair process, whereas the bone heals by a regenerative process. When most of the musculoskeletal tissues are being repaired, they will heal with a scar, which replaces the original tissue, whereas when a bone regenerates, the healing tissue is identical to the tissue that existed there before.

The healing of an injured skeletal muscle follows a fairly constant pattern irrespective of the underlying cause (contusion, strain, or laceration). As described in the preceding text for the soft-tissue injuries in general, three phases have been identified in this process. These are (1) inflammatory (destruction), (2) proliferative (repair), and (3) maturation (remodeling) phases.

In short, the natural course of muscle injury healing takes place as follows. After the initial trauma, the ruptured myofibers contract and a hematoma fills the gap between the myofiber stumps. The injured ends of the myofibers undergo only local necrosis, because the torn sarcolemma is rapidly resealed allowing the rest of the ruptured myofibers to survive. Activated platelets secrete growth factors that function as chemoattractants for the inflammatory cells. Macrophages, having first invaded the injury site from the torn blood vessels, remove the cell debris and secrete growth factors that initiate angiogenesis, that is, blood supply to the injured area and also activate the satellite cells, that is, the regenerative (reserve) stem cells of the muscle tissue. The satellite cells reside between the sarcolemma and the basal lamina of the myofibers and can survive even though the surrounding tissue undergoes necrosis. There are two different populations of satellite cells, *committed* and *stem* satellite cells, with very defined functions: *committed* satellite cells begin to differentiate into myoblasts immediately after injury, whereas *stem* satellite cells begin to proliferate first. After the round of proliferation, *stem* satellite cells contribute one daughter cell

to the formation of regenerating myoblasts, at the same time providing new satellite cells by asymmetric cell division for future needs of regeneration. Thus, the regenerating myoblasts arise from both the *committed* and *stem* satellite cells then fuse to form myotubes within a couple of days. The regenerating young myotubes grow in length and size and, finally, mature into myofibers.

Simultaneously with the muscle fiber regeneration, the concomitant production of a connective tissue scar by fibroblasts takes place between the regenerating muscle fibers that try to pierce into connective tissue. Thus, the ends of the regenerating myofibers do not usually reunite, but instead their ends attach to the extracellular matrix of the interposed scar via adhesion molecules at the newly formed myotendinous junctions. Thus, each ruptured myofiber remains divided into two independent fibers bound together by the interposed (small) scar. Finally, the maturation of the regenerated myofibers, retraction and reorganization of the scar tissue, and recovery of the functional capacity of the muscle occur over time during the maturation phase (Figure 1.19).

Tissue injury and bleeding result in an inflammatory reaction with the formation of scar tissue. After this type of injury, there is little muscle tissue regeneration, so that the injured muscle tissue is replaced by fibrous scar tissue without contractile properties. This contributes to the highly increased risk of recurrent injuries, for example, hamstrings strains.

Occasionally, muscle hematomas lead to an unfortunate complication: myositis ossificans (calcification or ossification of the injured tissue). The most common

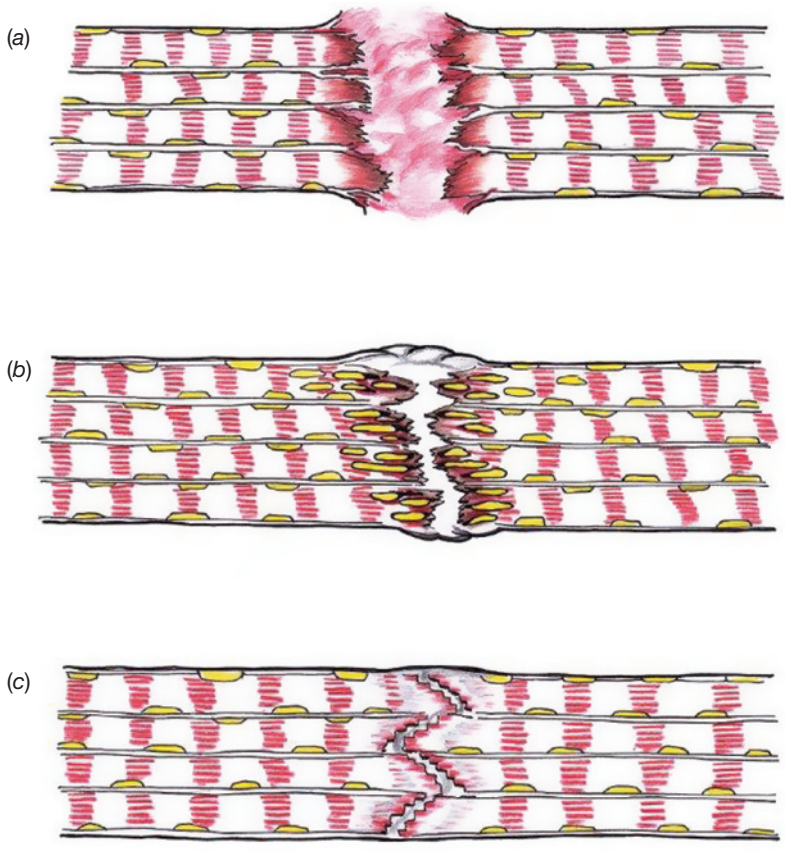

Figure 1.19 In strains not only the myofibers rupture but also their basal lamina as well as mysial sheaths and blood vessels running in the endo- or perimysium are torn (*a*). The ruptured myofibers become necrotized only over a short distance. The injured part of the ruptured myofiber inside the remaining old basal lamina is replaced by the regenerating myofiber, which then begins to penetrate into the connective tissue scar between the stumps of the ruptured myofibers (*b*). The maturation of the regenerating myofibers includes formation of a mature contractile apparatus and attachment of the ends of the regenerated myofibers to the intervening scar by newly formed myotendinous junctions (*c*). The retraction of the scar pulls the ends closer to each other, but they appear to stay separated by a thin layer of connective tissue to which the ends remain attached by newly formed myotendinous junctions. (© Medical Illustrator Tommy Bolic, Sweden.)

location is on the thigh. After quadriceps contusion, it can affect as many as one of five patients. Myositis ossificans is a nonneoplastic proliferation of bone and cartilage within the skeletal muscle at the site of a previous single major trauma or repeated injury or/and hematoma. Being a relatively rare complication of muscle injury, the scientifically valid evidence regarding either the pathogenesis or the most optimal treatment is virtually nonexisting. In sports, myositis ossificans is typically associated with prior sports-related muscle injury (i.e., re-injury to the same location), the incidence being the highest in the high-contact sports in which the use of protective devices is uncommon (e.g., rugby). The most common muscle involved is the quadriceps femoris, where even up to 20% of injuries could lead into myositis ossificans. Increased susceptibility to myositis ossificans has also been described in individuals with hemophilia or other bleeding disorder in conjunction with a soft-tissue injury.

Clinically, myositis ossificans should be suspected if pain and swelling are not clearly subsiding 10–14 days after an injury to a skeletal muscle or if the healing does not seem to progress normally despite the execution of a proper conservative treatment. One should be particularly alert if the symptoms intensify weeks (or months) after the trauma, especially if the site of injury becomes more indurated and the injured extremity displays reduced joint ROM. Although it is sometimes possible to detect the first signs of the ectopic bone in radiographs as early as 18–21 days after the injury, the formation of ectopic bone usually lags behind the symptoms by weeks, and thus, a definite radiographic diagnosis can only be made substantially later, even months after the actual injury. It is important to be aware that the radiographic appearance of the bone mass in the early stage can be confused with osteogenic sarcoma. The conditions can be difficult to distinguish on histology as well.

Due to its rarity, the treatment principles of myositis ossificans are based more on empirical experience than on clinical or experimental evidence than any other type of muscle complaint. The proper first aid of muscle trauma (the prevention of formation of a large hematoma) naturally creates the foundation for the treatment of this complication. However, if the myositis ossificans still occurs despite the best prevention efforts, there is little that can or should be done in the acute phase. Although indomethacin is quite commonly used in orthopedics in preventing heterotopic ossification, it has not been validated for the prevention and/or treatment of myositis ossificans. The surgical excision of the bone mass can be considered at later phases, if the symptoms do not reside despite 12 months of watchful waiting. However, surgery should not be performed until the ectopic bone has fully "matured," which is 12–24 months after the onset of the symptoms, as the excision of immature bone often results in recurrence. Overall, the myositis ossificans could be considered to underscore the importance of proper initial treatment of athletes with muscle injury. Despite the fact that a great majority of muscle injuries heal virtually irrespective of the primary treatment, compromised healing of muscle injury (myositis ossificans) results in a delay in return to sports that is highly comparable–and often even longer–than that associated with the failed treatment of other sports-related major injuries.

Another complication of intramuscular hematomas is compartment syndrome. Bleeding and intracellular and intercellular edema can cause such an increase in pressure that circulation in a muscle compartment is compromised. This affects the muscle primarily on the capillary level, rarely the large vessels. Thus, a good pulse distal to the hematoma does not necessarily exclude the possibility of compartment syndrome. The primary symptom is pain, eventually extreme pain, and the muscle compartment is hard on palpation. Nerve function may be affected so that the patient feels paresthesia distally. If it is untreated, compartment syndrome may result in necrosis of the muscles and major sequelae in the long term.

Muscle lacerations are rare in sport but may occur as a result of cuts from the edge of a skate or a downhill ski. Transverse lacerations cut across the muscle fibers. The wound muscle rupture is repaired as described in the preceding text for contusions and strain, that is, with a fibrous scar tissue without contractile properties. This may have consequences for muscle function.

Muscle stiffness (DOMS) is a troublesome but generally harmless symptom or the mildest type of muscle injury that all active sports people will have experienced at some point. DOMS is commonly a consequence of an overenthusiastic exercise of untrained muscle, which is tolerated while engaged in that activity, but followed by muscle soreness 1–3 days after the exercise. This phenomenon strikes especially if the exercise includes eccentric work, that is, lengthening of contracted muscles like in running downhill or squatting with weights. The symptoms of stiffness, soreness, and tenderness with palpation develop during the first 1–2 days with a peak on days 2 or 3, and they disappear usually with no treatment by days 5–7. The pain is aggravated by passive stretch of the sore muscle and the strength of the muscle is decreased. This is usually associated with a rise in serum creatine kinase (CK), which is usually modest but sometimes up to 20-fold. CK values peak around days 3–6 and usually return to normal during the first week after the eccentric exercise. Inflammatory reaction has been reported in both experimental animals and in humans. The pain in DOMS is mediated by nociceptors, which in DOMS are most likely stimulated by factors (such as bradykinin, prostaglandins, and serotonin) released from the inflammatory cells. Nonsteroidal anti-inflammatory drugs (NSAIDs) have been used to reduce the pain, but the relatively mild inflammation does not actually need any alleviation by treatment with NSAIDs. In humans, DOMS develops after eccentric work excessive for the fitness level of the muscle. Even though DOMS is associated with CK rise, which must indicate some degree of sarcolemmal damage inducing leak of sarcoplasmic proteins, it has been demonstrated that in DOMS usually no frank necrosis of myofibers ensues. The main structural finding has been focal loss of the myofibrillar (sarcomeric) structures.