INJURY IS AN UNFORTUNATE, BUT INEVITABLE, REALITY IN THE SPORT OF AMERICAN FOOTBALL. INJURY RATES IN FOOTBALL ARE AMONG THE HIGHEST ACROSS ALL SPORTS. THE PURPOSE OF THIS REVIEW IS TO DISCUSS BIO-MECHANICAL PRINCIPLES, WITH PARTICULAR EMPHASIS ON FORCE AND MECHANICAL ENERGY, AS THEY APPLY TO MUSCULOSKELETAL INJURIES SUSTAINED BY PARTICIPANTS IN AMERICAN FOOTBALL AND TO PRESENT THE MECHANISMS OF INJURY FOR SOME COMMON FOOTBALL-RELATED INJURIES, INCLUDING INJURIES OF THE ANKLE, KNEE, HIP, SHOULDER, AND CERVICAL SPINE.

INTRODUCTION
American football arguably is the most popular sport in the United States. With more than a million participants at many levels, from youth football to high school, college, and professional play in the National Football League (NFL), and many millions more who support their favorite teams with great fervor, American football is a popular participant activity. Football has grown in recent decades beyond just a sport and into a phenomenon of financial and cultural significance.

Injury is an unfortunate, but inevitable, fact of everyday life, and especially so in the sport of American football. Football games and championships often are determined, at least in part, by injuries that either diminish players’ performance or preclude their participation. Studies have clearly shown that injury rates in American football are among the highest across all sports (8,11).

For the purposes of this review, musculoskeletal injury is defined as the damage sustained by tissues of the body in response to physical trauma. Given the nature of American football as a game involving frequent and violent impacts and collisions, injury is common. Studies consistently have shown that compared with other sports, American football has the highest injury rates at both the high school (20) and collegiate (26) levels. The type and extent of injury depends on myriad factors, including age, level of physical conditioning, position (e.g., quarterback, linebacker, lineman), playing surface type and condition, level of musculoskeletal maturity, and equipment.

Efforts have been made, and are ongoing, to reduce injury risk. In the late 1930s, for example, helmet use was mandated. Since then, required equipment (e.g., mouthpieces), published guidelines related to specific conditions such as heat illness and concussions, rule changes to prohibit deliberate use of the helmet to punish an opposing player (i.e., spearing), chop blocks, and horse-collar tackles, and conditioning guidelines (e.g., unrestricted player access to water), have all been developed to lessen the chance of injury. Despite these efforts, injury remains a fact of American football life.

The purpose of this review is to discuss biomechanical principles as they apply to musculoskeletal injuries sustained by participants in American football and present the mechanisms of injury for some common football-related injuries. This review focuses on musculoskeletal injuries affecting joints and body tissues such as bone, tendon, ligament, and cartilage. Other football-related conditions such as concussion and heat-related injuries, while critically important, are beyond the scope of this review.

BIOMECHANICAL CONCEPTS AND TERMINOLOGY
Biomechanics, broadly defined as the application of mechanical principles to problems of biological organisms...
and systems, has direct relevance to musculoskeletal injury. A full review of the many biomechanical factors involved in musculoskeletal injury is beyond the scope of this article. For a more detailed discussion, interested readers are directed to other sources (e.g., 25).

Force, the mechanical action or effect applied to a body that tends to produce acceleration, is a fundamental mechanical element involved in musculoskeletal injury. According to Newton’s Second Law of Motion, force = (mass) × (acceleration), the greater the mass of the players, and the faster they move, the greater the forces involved. In American football, forces abound in player-to-player and player-to-ground collisions. In injury-causing situations, 7 force-related factors combine to determine the nature and extent of an injury: (a) magnitude (how much force is applied?), (b) location (where on the body or structure is the force applied?), (c) direction (where is the force directed?), (d) duration (over what time period is the force applied?), (e) frequency (how often is the force applied?), (f) variability (is the force magnitude constant or variable over the application period?), and (g) rate (how quickly is the force applied?) (25).

A muscle bruise (contusion) results from direct impact and results in intramuscular hemorrhage. In American football, a common example is when a player’s knee, helmet, or shoulder pads violently impact the anterior or lateral aspect of another player’s thigh. This results in a quadriceps contusion. Repeated impacts before healing may worsen the injury and lead to a serious secondary condition such as myositis ossificans, wherein an ossified mass develops within or at the edges of the impacted muscle.

As noted above, we define injury as damage to body tissues caused by physical trauma. Musculoskeletal injury typically happens as a result of some event or mechanism. In general, a mechanism is defined as the fundamental physical process responsible for a given action, reaction, or result. More specifically, injury mechanisms establish the cause-and-effect relationship between an action (e.g., direct impact) and the effect (e.g., bone fracture). A comprehensive list of potential injury mechanisms would be extensive and include mechanisms such as crushing deformation, bending, twisting (or torsion), pulling or stretching, rapid acceleration or deceleration, and energy absorption. In the following sections, we explore injuries and associated mechanisms common to American football.

EXEMPLAR INJURIES AND MECHANISMS

CONTUSION

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The term bruise (or contusion) also is used to describe bone lesions that typically result from direct impact, most often at the knee and ankle and less often from shear forces resulting from twisting and turning movements. Bone bruises are classified based on the area of injury and include bleeding between the periosteum and bone (subperiosteal hematoma), bleeding and microfracture of trabecular bone (interosseous bruising), and bleeding between the articular cartilage and underlying bone (subchondral fracture).

STRAIN

A strain injury refers to damage to a musculotendinous unit (or muscle-tendon complex). Acute muscular strain can result from overstressing an inactive muscle or dynamically overloaded an active muscle either in concentric (active shortening) or eccentric (active lengthening) action.
The severity of musculotendinous strain depends on the level of structural disruption, pain, and functional loss. Mild strains are characterized by minimal structural damage and pain and rapid return to play. Moderate strains exhibit partial tearing of the muscle tissue, often at or near the musculotendinous junction. There is some functional loss and delay in return to normal function. In a severe strain, muscle tissue is completely, or near-completely, torn and accompanied by considerable hemorrhage and swelling and a long period of repair and rehabilitation (25).

Certain muscles are more susceptible to strain injury than others. The hamstring muscle group (semimembranosus, semitendinosus, biceps femoris) is at particular risk (17). Football players who sprint at full speed (e.g., running backs, wide receivers) are prone to hamstring strains. All of the muscles in the hamstring group, with the exception of the short head of the biceps femoris, are biarticular (i.e., have action at 2 joints, the hip and knee). Simultaneous hip flexion and knee extension (as seen in the late swing phase when the foot is off the ground and early in the stance phase right after the ground contact) conjointly lengthen these hamstring muscles. During this time, the hamstrings act eccentrically to decelerate both the thigh and lower leg and prepare for the ground contact. An additional contributing factor to the hamstrings’ susceptibility to strain injury is their relatively high proportion of fast-twitch muscle fibers, which allow for higher levels of intrinsic force. These factors combine to help explain the frequency of hamstring strain in football players whose positions demand frequent sprinting (17,25).

SPRAINS

Injury to a ligament is termed a ligamentous sprain. Ligaments are collagenous tissue structures that span between bones to help stabilize and maintain the structural integrity of joints. Ligamentous sprains, especially when moderate and severe, compromise joint function. Sprains most commonly occur at the ankle joint complex and involve both the ankle and subtalar joints. The ankle joint’s relative instability, especially when the foot is in a planter-flexed position, contributes to its susceptibility to injury.

Lateral ankle sprains typically happen when the foot and ankle complex is in a supinated position (Figure 1A), which combines subtalar joint inversion, ankle plantar flexion, and internal rotation of the foot. Placing the foot and ankle in this position, either when landing from a jump or stepping in a hole or on an opponent’s foot, places tensile forces on structures on the lateral side of the foot. Because of its orientation in this position and its relatively low tissue strength, the anterior talofibular ligament (Figure 1B) is predictably the first and most often injured lateral ligament. If the foot and ankle continue into further supination, the calcaneofibular ligament is next involved, followed, rarely, by injury to the posterior talofibular ligament. Given the close proximity of players to one another, the frequent

Figure 1. (A) Player’s foot forcibly put in a position conducive to a lateral ankle sprain. (B) Mechanism of supination involved in a lateral ankle sprain.
contact with other players and the ground, the players’ size and speed, and the violent nature of the game, it is not surprising that lateral ankle sprains are common in American football (18). Another sprain associated with the ankle is the so-called high (also syndesmotic) ankle sprain (4, 18). In a high ankle sprain, applied loads wedge the tibia and fibula apart just above the ankle joint (Figure 2). This can tear the interosseous membrane and tibiofibular ligaments. The most likely mechanisms of high ankle sprains are talar torsion (i.e., twisting of the talus between the distal end of the tibia and fibula when the ankle is in a dorsiflexed position) and forced ankle dorsiflexion. These mechanisms are present in football when linemen, for example, plant and cut while in a squat position with hips and knee flexed and ankles dorsiflexed.

**FRACTURES**

A bone fractures, or breaks, when an applied load exceeds the bone’s ability to resist the force. Fracture may happen as a result of a single high-magnitude load (as in a violent collision) in what is termed an acute fracture. Bone also may fracture in response to repeated low-magnitude loadings in what is called a chronic fracture. In American football, bone fractures most commonly are acute and result from direct impact forces or forceful bending.

**TENDON RUPTURE**

Tendons are responsible for force transmission from skeletal muscles to bones and thus play an essential role in movement of the musculoskeletal system. If the forces being transmitted through a tendon exceed that tendon’s strength, the tendon may tear. The calcaneal (Achilles) tendon is the largest and strongest tendon in the body. Its strength notwithstanding the calcaneal tendon can fail in response to excessive loads. The calcaneal tendon transmits forces from the triceps surae muscle group (gastrocnemius and soleus) to its attachment on the posterior aspect of the calcaneus. Numerous studies (3, 5, 9, 10, 19) have estimated the forces transmitted through the calcaneal tendon. Forces up to 10 times body weight have been noted.

Four mechanisms have been implicated in calcaneal tendon failure: (a) sudden dorsiflexion of a plantar-flexed foot, (b) pushing off the weight-bearing foot while extending the ipsilateral knee joint, (c) sudden excess tensile forces applied to an already taut tendon, and (d) a taut tendon struck by a blunt object (13).

The first mechanism is exemplified by a quarterback dropping back and planting his rear foot as he throws (Figure 3).

**KNEE INJURY**

Among the most recognized and researched of sports-related injuries are tears of the anterior cruciate ligament (ACL). The ACL is a complex knee ligament that connects the femur and tibia. Proximally, the ACL attaches to the medial surface of the lateral femoral condyle. The ACL attaches distally...
to the anterior surface of the midtibial plateau. The ACL functions as the primary restraint to anterior tibial translation (relative to the femur) or, conversely, posterior femoral translation (relative to the tibia). Secondarily, the ACL resists valgus-varus angulation of the knee and works in concert with the posterior cruciate ligament in limiting knee hyperextension and hyperflexion.

Most often, ACL injury happens in response to valgus (knock-knee) loading combined with external tibial rotation or to hyperextension with internal tibial rotation (Figure 4). The first mechanism (valgus rotation) typically occurs in what is termed a noncontact injury when the foot in planted on the ground with the tibia externally rotated, the knee near full extension, and the knee collapsing into valgus (Figure 5A). This mechanism is seen in plant-and-cut or change-of-direction tasks. In American football, the situation can be exacerbated in what is termed a contact injury (Figure 5B). With the foot planted on the ground, another player impacts the lateral aspect of the knee, thus accentuating the knee valgus and rotation and greatly increasing ACL injury risk. For more detail on the anatomy, physiology, and biomechanics of ACL injury, interested readers are referred to the study by Siegel et al. (24).

Another knee ligament commonly injured in football is the medial collateral ligament (MCL). The MCL provides primary restraint to valgus in response to an impact on the lateral aspect of the knee. A lateral impact and resulting knee valgus places tensile loads on the medial structures of the knee. Excessive tensile forces can tear the MCL.

Although injury to the ACL and MCL can happen in isolation, they sometimes are injured together, along with the medial meniscus, in what has been termed the “unhappy” triad (Figure 6). Damage to the medial meniscus usually occurs when the meniscus is subjected to a combination of flexion and rotation or tension and rotation during weight bearing, the former being consistent with knee valgus. Given the interconnectedness and similar functions of these 3 structures, the ACL, MCL, and medial meniscus often are injured simultaneously in valgus rotation situations commonly seen in football.

**HIP INJURY**

Given the hip joint’s relative stability, because of the relative good bony fit of the femoral head in the acetabulum of the pelvis and the substantial nature of the hip’s periarticular structures (e.g., large muscle mass), the hip is rarely dislocated. Most hip dislocations arise from high-energy impacts in automobile collisions. Although relatively rare, hip dislocations have been noted in football. One study (16) reported 8 subluxations (i.e., partial dislocations) in American football and identified the most common mechanism as falling and landing on a flexed and adducted hip.

**SHOULDER COMPLEX PATHOLOGIES**

The shoulder girdle contains 2 bones: the scapula and the clavicle. The clavicle attaches medially to the sternal manubrium and laterally to the acromion process of the scapula at the acromioclavicular (AC) joint. The humerus of the upper arm articulates at the glenohumeral (GH) joint where the humeral head fits loosely into the shallow glenoid fossa of the scapula. The GH joint is the most mobile, and least stable, joint in the body. The joint’s exceptional mobility and
limited stability have injury implications. Shoulder injuries common to American football include AC sprains, GH dislocations, and rotator cuff pathologies.

Sprain of the AC joint is referred to as a separated shoulder (or shoulder separation) and should not be confused with a GH dislocation. Acromioclavicular injury most commonly results from direct force applied to the point of the shoulder with the arm in an adducted position (6). The impact force drives the acromion inferior relative to the clavicle. Such forceful impacts in football can be seen in player-to-player collisions and by direct contact between a player’s shoulder and the ground. Less frequently, AC injury results indirectly from forces transmitted up to the shoulder by a player falling on an outstretched arm. In the NFL, the highest incidence of AC injury is found in quarterbacks, special team players, and wide receivers (12).

The ability of a joint to resist dislocation determines its inherent stability. The GH joint’s extensive mobility, because of poor bony fit between the humeral head and the shallow glenoid fossa, and lack of supporting structures contribute to the GH joint’s propensity for dislocation. Most GH dislocations involve anterior, or anteroinferior, displacement of the humeral head relative to the glenoid fossa. In football, the primary mechanisms for GH dislocation are anterior dislocation from direct force applied to the posterior aspect of the shoulder (e.g., a quarterback hit from behind by a rushing lineman or linebacker) or forced horizontal abduction with external rotation, as when a linebacker reaches out to tackle a running back. Much less frequently, posterior dislocation happens from indirect force applied through the arm in a flexed, adducted, and internally rotated position (e.g., a receiver diving for a pass and landing on a forward reaching arm) or posterior GH dislocation from direct force applied to the anterior aspect of the shoulder (e.g., a running back’s anterior shoulder hit head-on by a defensive lineman).

Athletes who perform repeated overhead motions, such as a quarterback throwing a football (Figure 7A), are at risk for GH impingement syndrome. An impingement syndrome, in general, occurs when increased pressure develops within a confined anatomical space and produces deleterious effect on the enclosed tissues (Figure 7B). There are 2 major GH impingement types: subacromial impingement and internal impingement. In subacromial impingement, repeated shoulder abduction
results in suprhumeral structures (primarily the distal supraspinatus tendon, subacromial bursae, and proximal tendon of the long head of the biceps brachii) being forcibly pressed against the coracoacromial arch (comprised the anterior surface of the acromion and the coracoacromial ligament). Another form of GH impingement, internal impingement, happens when the supraspinatus tendon contacts the posterior-superior rim of the glenoid fossa. This mechanism is commonly seen in throwing (e.g., quarterback passing), when the shoulder is abducted and externally rotated during the cocking phase of an overhead throw.

Glenohumeral impingement syndromes can play a role in a sequence of events that eventually lead to lesions in muscles of the rotator cuff (i.e., supraspinatus, subscapularis, infraspinatus, teres minor). Intrinsic and extrinsic factors are involved in the cascade of events that lead to eventual rotator cuff ruptures.

**CERVICAL SPINE TRAUMA**

Although extremely rare, injury to the cervical spine (neck) is one of the most potentially catastrophic conditions in football. Severe cervical spine injuries with concomitant spinal cord lesions can result in paralysis and even death.

The complex structure and intricate motion of the cervical spine presents special challenges in describing mechanisms of cervical injury. Classification of cervical spine injury mechanisms requires caution and care because (a) the overall motion of the head relative to the trunk may not be indicative of the local motion of structural elements within the cervical spine, (b) small deviations in the location and direction of forces applied to the spine may change the injury mechanism, for example, from flexion compression to extension compression, and (c) observed head motions may happen after the instant of injury and therefore not reflect the true mechanism of injury (15).

Cervical injuries in American football used to be ascribed to a hyperflexion mechanism or a hyperextension mechanism in which the posterior rim of the helmet acted as a pivot during cervical hyperextension and created what was termed a “guillotine” effect. These mechanisms have been discounted as a primary cause of cervical injury (7). Currently, a flexion compression (also compression flexion) mechanism is generally accepted as the most common mechanism of cervical spine injury. When the neck is slightly flexed, the normal cervical curvature (lordosis) disappears and the cervical vertebrae become aligned in a column-like arrangement (Figure 8). In
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this position, the cervical spine lacks the curvature required for energy-absorbing bending. If a compressive load is applied at the instant of cervical vertebral alignment, all of the energy must be accepted by the aligned vertebra. If the applied energy exceeds the capacity of the cervical structures, vertebral fracture and intervertebral disc failure can occur. These injuries carry the risk of causing impingement of the spinal cord and spinal nerves. The cervical alignment required for a flexion compression injury is present in football, for example, when a tackle lowers his head and helmet and impacts (“spears”) another player or the ground.

One final football-related injury of note is the so-called “stinger” or “burner.” Although technically a neural, rather than musculoskeletal injury, the frequency of stingers in American football supports brief mention of the injury here. Well more than 50% of football players have experienced this injury, primarily while tackling or blocking. The most common mechanism is when a direct blow on the top of the shoulder drives the shoulder inferiorly and simultaneously bends the head and neck to the opposite side and results in a compressive or stretching insult to the brachial plexus, a group of nerves that innervates muscles of the neck and shoulder, and manifests as a shooting or stinging pain traveling down the arm. Symptoms include pain, weakness, numbness, and tingling, which typically subside within minutes, hours, or a few days after onset.

SUMMARY
Although musculoskeletal injury has been, and always will be, a fact of everyday athletic life in general, and American football in particular, an understanding of functional anatomy, biomechanical principles, and mechanisms of musculoskeletal injury can help reduce the risk and incidence of injury and improve the effectiveness of treatment and rehabilitation protocols for injuries when they happen.

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