

---

# Prevention and Management of Common Musculoskeletal Injuries in Preadolescent and Adolescent Female Athletes

# 15

Mimi Zumwalt and Brittany Dowling

---

## Abstract

Females transitioning from childhood to adolescence undergo a dramatic change in their body. In fact, this transitional period is where males and females start to diverge in terms of body composition, muscular strength, and bone mass. With the start of menses, female hormones begin to take an even more significant role on the body by greatly affecting the development/function of skeletal, muscular, and nervous systems. This rapid physiologic change during menarche exposes the female athlete's body to musculoskeletal injury, i.e., tendons, ligaments, muscles, and bones are all at risk. The skeleton in particular, is at greater risk due to the presence of open physes or "growth plates" at the ends of growing long bones. The young female athlete is therefore more prone to the multitude of sports-related injuries, and in fact at higher risk for certain types of trauma than their adult counterparts. Heightened awareness and a certain level of precaution need to be taken to help prevent potential injury. However, if trauma does occur then appropriate steps need to be taken to treat as well as protect the injured site for optimal healing and recovery.

---

## Keywords

Adolescence • Menses • Menarche • Physes or "growth plate"

---

M. Zumwalt, M.D. (✉)  
Department of Orthopaedic Surgery and  
Rehabilitation, Texas Tech University Health  
Sciences Center, Lubbock, TX, USA  
e-mail: mimi.zumwalt@ttuhsc.edu

B. Dowling, M.Sc.  
Department of Health, Exercise, and Sports Sciences,  
Texas Tech University, Colorado Springs, CO, USA

---

## 15.1 Learning Objectives

After completion of this chapter, you should have an understanding of:

- The important changes from prepubescence to postpubescence in females as far as anatomy and physiology of the musculoskeletal system are concerned

- The pertinent differences AND similarities between young females and males in terms of anatomy, physiology, and biomechanics of the musculoskeletal system
- The occurrence of various common musculoskeletal injuries more unique to young female athletes, especially during the adolescent growth spurt
- Several measures for prevention of athletic injuries from occurring in preadolescent and adolescent females
- Different methods of orthopaedic treatment for musculoskeletal injuries incurred by young female athletes, including specific recommendations for a regular conditioning program once healing is complete in order to enhance their physical fitness profile

---

## 15.2 Introduction

The pubescent growth spurt, especially the period transitioning from late childhood to adolescence, represents a time of tremendous transformation. Generally speaking, growth, development, and maturation play such an important role during these ever so important teenage years. Specifically, growth refers to a size increase of the body or part(s) thereof, while development is the functional alterations occurring along with anatomical/structural growth. Maturation is reached when the body has achieved its full potential in terms of growth and development, i.e., attainment of the adult form. Specific bodily units involved in this early stage of life alteration include the reproductive/sexual, physiological, and musculoskeletal systems. Unique musculoskeletal conditions can occur while the body is growing, and they need to be recognized and addressed appropriately. In addition, with the increased emphasis in youth sports and female involvement with athletic activities, this comes with a higher risk/frequency of orthopaedic injuries [1]. This chapter focuses on several of the more common musculoskeletal concerns of the young female athlete, plus prevention and treatment for these orthopaedic issues.

---

## 15.3 Research Findings and Contemporary Understanding of the Issues

### 15.3.1 The Important Changes from Prepubescence to Postpubescence in Females as Far as Anatomy and Physiology of the Musculoskeletal System Are Concerned

After the initial rapid increase in height during the first 2 years of life (when 50 % of adult height is attained) and up until the onset of puberty, a female's body grows steadily in terms of height and weight. Once puberty starts, growth velocity rapidly rises causing a dramatic increase in height and weight, with peaking around the age of 12. In girls, final adult stature is reached between the ages of 16 and 17. It is an established fact that regular exercise, along with an appropriate diet, is essential for proper bone growth in terms of width, density, and strength by mineral (calcium among others) deposition into the skeletal matrix [1–3]. Alongside the bony framework providing structural support and protection for the body, musculotendinous and ligamentous attachment to the skeleton help synergistically by providing dynamic and static restraints while allowing motion/movement of the head, trunk, and limbs, respectively. Muscle mass also increases steadily (from hypertrophy or enlarging fiber size) in response to hormonal influence, reaching its peak between the ages of 16 and 20 years in females. As a corollary to the increase in muscle mass, an accompanied gradual rise in muscle strength occurs in conjunction as well, and reaches a maximum by 20 years of age in young female adults [1]. Prior to puberty, girls and boys are comparable in terms of muscular strength. Then around the age of 15 or 16, adolescent females are only about 75 % as strong as pubescent males. This strength difference is more marked in the upper versus the lower extremities [4].

In addition to longitudinal growth and gaining in body size/weight, morphologically the

young female body shape is further transformed during the adolescent growth spurt, partly in preparation for later childbearing [1, 4]. Under the influence of estrogen, fat deposition increases which more than doubles the percentage of total body weight present at birth (25 % rather than 10 to 12 %). Both sex specific (surrounding breasts and hips) and subcutaneous tissue fat accumulate throughout the body of a young woman. The mechanism of adipose tissue storage, unlike that of muscle, stems from both hypertrophy (increase in size of fat cells) and hyperplasia (increase in number of fat cells). The latter process can continue throughout one's life span, dependent on diet and activity which is among other factors. With continued enhancement from the developing neuromuscular and endocrine systems, motor control continues to develop; however, this process starts to plateau at the onset of pubescence in young females. Girls tend to slow down physically as a whole during adolescence, partly due to greater fat deposition [1].

### **15.3.2 The Pertinent Differences and Similarities Between Young Females and Males in Terms of Anatomy, Physiology, and Biomechanics of the Musculoskeletal System**

Before puberty, both growth and development in females and males parallel each other. During this childhood stage, prepubescent females and males are similar in stature and have the same relative muscle mass and strength. However, at the onset of puberty, due to the differing levels of secreted sex hormones, namely, the ratio of estrogen to testosterone, body composition changes start to separate young girls and boys in terms of maturation of the musculoskeletal system. Females have earlier onset of puberty than males; however, males undergo puberty for a longer time period, specifically 4 years as compared to 3 years [5]. Puberty for females starts around age 10–13 and ends at 15–16, whereas for males puberty begins at age 12–15 and ends at 17–18 years [1, 5, 6]. Because males grow for a longer

period of time than females, they are on average 10 % taller and 17 % heavier than females [6].

Likewise, under the influence of a sudden ten times increase in testosterone production during puberty, boys markedly gain muscle mass at an accelerated rate, resulting in 40 % of total body weight as compared to the 25 % present at birth. However, muscle mass does not reach its peak quantity until 18–25 years of age. Females do not undergo a significant change in muscle mass with puberty, resulting in only minor changes of muscular strength. Around age 12 or so, muscular strength in boys, and to lesser extent, in girls, starts to improve along with an increase in muscle mass. However, the rate of growth is compounded in males at a faster rate, stronger proportion, and longer duration, peaking between the ages of 20 and 30 [1, 7, 8]. Before puberty, males and females have similar muscular strength. However, at ages 11–12 females are 90 % as strong as males, then at age 13–14 females are 85 % as strong as males, and by age 15–16, females are only 75 % as strong as males [9]. This muscular strength difference between the sexes can be accounted for by differences in body composition. Adult males have muscle mass comprising 40 % of body weight, whereas females have only 23 % of total muscle mass [9]. This difference is due to estrogen, which increases adipose tissue and has been shown to have a slight reducing affect on lean tissue [6]. In contrast, due to lower estrogen levels as well as greater levels of androgens, males have an increase in lean tissue and do not tend to accumulate a large quantity of fat. Body fat in physically unconditioned males ultimately reaches 15 % of total body weight, whereas unconditioned females average about 25 % total body fat. For a conditioned male athlete, the percentage is lower at 7 % and females around 10–15 % [6, 9].

The difference in muscle mass and adipose tissue between females and males also accounts for difference in ability, coordination, and learning of motor skills starting between the ages of 9 and 12 for both sexes. After age 12, the physical performance in males continues to accelerate and far exceeds that of adolescent females during puberty due to more muscle mass/strength and

less fat accumulation [1, 7]. Along the same lines, development of speed also favors adolescent males, which ultimately results in higher fitness levels as compared to girls. In fact, the sprint velocity increases yearly starting at 5 years old in both sexes, but maxes out in females between the ages of 13 and 15, yet does not peak in boys until the age of 16. Two phases encompass the phenomenon of speed development; the first begins at about age eight in both girls and boys, most likely attributed to improved coordination aided by the maturing nervous system. The second phase occurs about age 12 in females, and anywhere from age 12 to 15 in males as a natural progression from the larger body size, muscle mass and, along with these changes, speed, strength, power, and endurance [7]. Morphologically, pubescent boys maintain their body structure similar to prepubescent girls, again affected by the ratio of testosterone to estrogen. Interestingly, recent studies have demonstrated that neuromuscular control and preference, especially in the lower limbs of adolescent females during certain sporting activities, differs from that of male athletes, potentially putting these young girls much more at risk for knee injuries [10–13].

### **15.3.3 The Occurrence of Various Common Musculoskeletal Injuries More Unique to Young Female Athletes Especially During the Adolescent Growth Spurt**

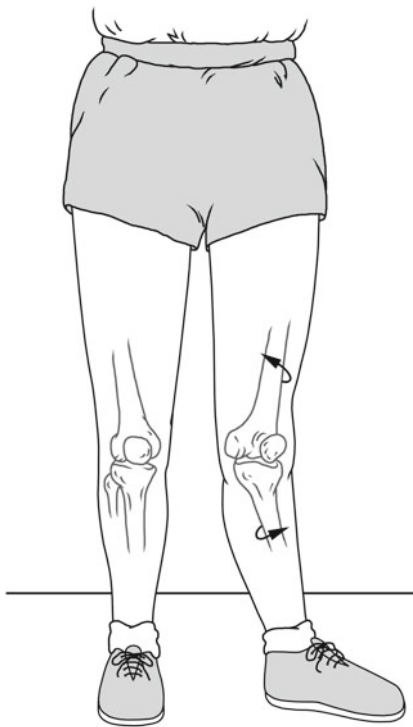
As previously outlined, the transition from prepubescence to postpubescence in females brings on a multitude of bodily changes, the majority of which involves the musculoskeletal system. While the skeleton is growing rapidly, muscles, tendons, ligaments, and other connective tissues must also grow to accommodate this accelerated bony growth. As a matter of fact, under endocrine and neural influence, locomotion and movement must be coordinated and adapted to the transforming skeletal framework in order to carry out life tasks and other physical endeavors, such as

recreational athletics or competitive sports. Consequently, teenage female athletes involved in certain sporting activities are exposed to a higher risk of orthopaedic injuries. This is partly due to a marked rise in the number of school-aged females playing sports, from one female participating in competition compared to 27 male athletes in 1972, to the ratio of one female athlete competing to three males 30 years later. Half of all children aged 5–18 years in the USA are thought to participate in organized sports; this is equivalent to a total of 30 million children [5].

Susceptibility to injury results from both intrinsic and extrinsic factors. Intrinsic factors include age, gender, strength, flexibility, and previous injury. Extrinsic factors include biomechanics of movement skills, equipment, environment, training schedule, and intensity of activity. Both intrinsic and extrinsic factors can work together or against each other to provide a mechanism of trauma or prevention of injury. For example, the repetitive nature of throwing and the acquired high velocity are coupled to cause chronic injury over time. Trauma can result from direct impact (such as a collision with another player) or an indirect impact (such as a force transmitted through an extremity causing injury at a different site) (Fig. 15.1).

Aside from acute orthopaedic trauma occurring by sudden explosive episodes, more gradual, chronic bouts of repeated force over and over can also cause damage to the musculoskeletal system, resulting in overuse injuries and can eventually progress to stress fractures [13, 14].

Inflammation is the body's natural response to tissue trauma, which can develop in response to an acute injury or from repeated, chronic irritation. The primary functions of this physiologic reaction are to protect the body from harmful released histamines, dispose of dead/dying tissue, and promote the regeneration of new tissue. The first step of inflammation is an increase in blood flow and capillary permeability at the site of injury due to release of chemicals by injured tissue cells. This causes swelling or edema with a subsequent rise in osmotic pressure, in turn causing an increase in movement of proteins, white blood cells, and fluid to the site of injury.

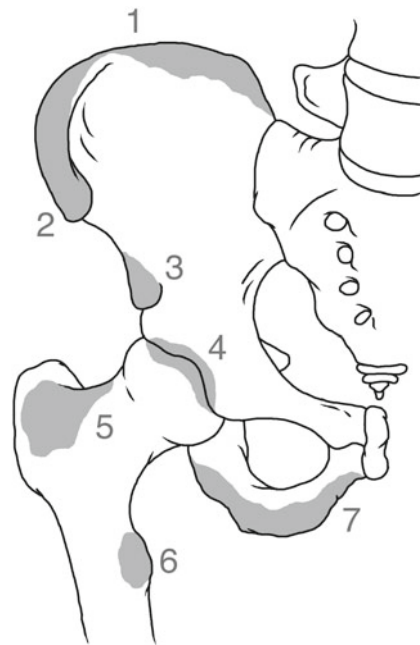


**Fig. 15.1** Risky limb landing attitude

Edema and associated chemicals heighten the sensitivity of pain receptors, causing an increase in pain of the involved area. The next step is coagulation, causing fluid to be trapped at the injury site. This captured fluid, also known as exudate, dilutes and inactivates toxins, provides nutrients for cells, and contains antibodies. The final phase of inflammation is the breakdown of coagulation with a decrease in osmotic pressure causing chemicals, toxins, and dead cells to leave the injured site [13].

The greatest difference between the immature skeleton and the adult skeleton is the presence of physes, or open growth plates. The physis is comprised of cartilage cells that proliferate to create longitudinal growth. Apophysis is a similar structure but differs in that it is in close proximity to the tendinous attachment on bone (Fig. 15.2).

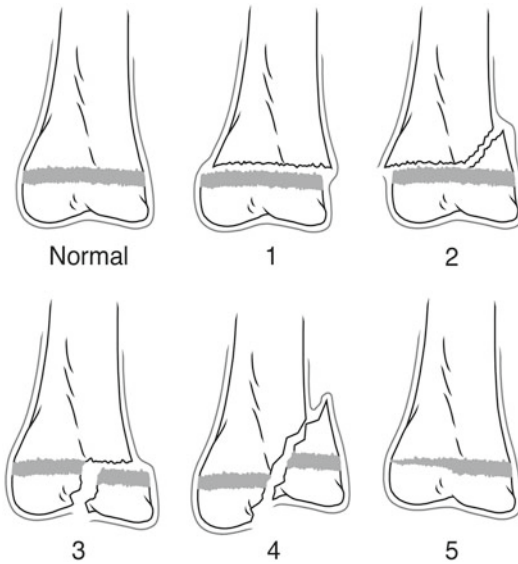
As discussed in Chap. 7, there is an inherent weakness of the physis during the adolescent growth spurt because the peak velocity height is obtained prior to peak bone mass. These growing



**Fig. 15.2** Pelvic apophyses

anatomical structures are also at more risk of injury when exposed to excessive force because they are inherently weaker than the surrounding ligaments and tendons [9]. The enveloping tissue, including capsules (connective tissue around joints), has sufficient strength and therefore can resist an application of force better than the weaker physal plate plus adjacent cartilage and bone. Consequently, any stress of sufficient magnitude to tear ligaments in adults tends to disrupt the physal cartilage in bone of younger children, resulting in growth plate injuries or avulsion fractures (Fig. 15.3).

The latter results from sudden violent muscular contractions transmitted through various tendons inserting onto bone. Physal injuries account for about 15 % of all fractures in children; with girls more prone to injury from ages 9 to 12 years and boys from ages 12 to 15 years [15]. Physiologic fusion of growth plates begins during the preteen years and is completed by the early twenties, occurring several years earlier in girls than boys. Prior to completion of the fusing process, any physal damage incurred while the body is changing may lead to temporary or even permanent growth disturbance, which could

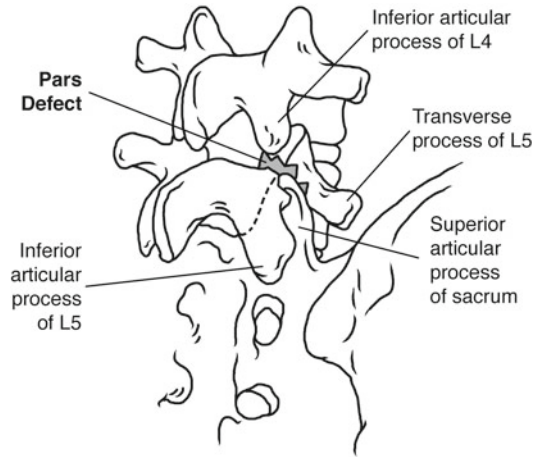


**Fig. 15.3** Growth plate injuries

result in leg length discrepancy or angular deformity of the affected limb [9, 15].

Bone of the immature skeleton is more porous and not as dense as adult bone due to the fact it is more vascular with less mineral content, which translates to being more flexible but structurally weaker. The offset of this characteristic is that the periosteum or envelope surrounding growing bones is thicker, stronger, and biologically more active; thus, greatly promotes the process of healing at a much faster rate [9, 15].

The most common musculoskeletal injury found in young athletes involves chronic, repetitive, submaximal mechanical load applied to the same area, causing microtrauma. Continued microtrauma culminates in bursitis of soft tissues, strains of tendons, sprains of ligaments, and stress reactions or even frank fractures of bone. This type of orthopaedic injury tends to occur more often in the lower rather than upper extremities and results primarily from overtraining. The anatomical sites that are more prone to fatigue fractures depend on the types of movement performed in specific athletic activities. For example, in the lower extremity, volleyball and basketball players are at increased risk of stress injury to the tibia due to repeated jumping/landing. Long distance runners, especially those athletes engaged in training over 20 miles weekly,



**Fig. 15.4** Spondylolysis

can suffer stress fractures to their femoral neck or fibula from excessive impact from running. In a similar fashion, because ballerinas have to be “en pointe” while dancing on the tips of their toes, they tend to sustain stress fractures of the second metatarsal more readily [14].

This does not mean that the upper extremities are exempt from microtrauma due to overuse. Stress fractures are commonly seen in the ulna from participation in tennis and fast-pitch softball players due to the nature of their racquet striking the ball or mechanism of pitching with their forearms, respectively. Along the same lines, the risk of radial fatigue fractures is higher in gymnasts as a result of repetitive load-bearing maneuvers on their wrists. Although quite rare, swimmers can injure their humeri from repeated overhead striking the water [14]. Furthermore, other athletic activities involving repetitive back extension maneuvers, i.e., dance, skating, and gymnastics can potentially place excessive load on the lumbar spine, resulting in a stress fracture of the pars interarticularis (spondylolysis) [4] (Fig. 15.4).

Additional variables contributing to other overuse type of injuries include environmental factors such as inadequate playing equipment, difficult/uneven surface terrain, faulty footwear, and inappropriate technique. The bodily internal environment also contributes to overuse injuries such as genetics, anatomic structure, and prior injury. Females in particular have altered internal

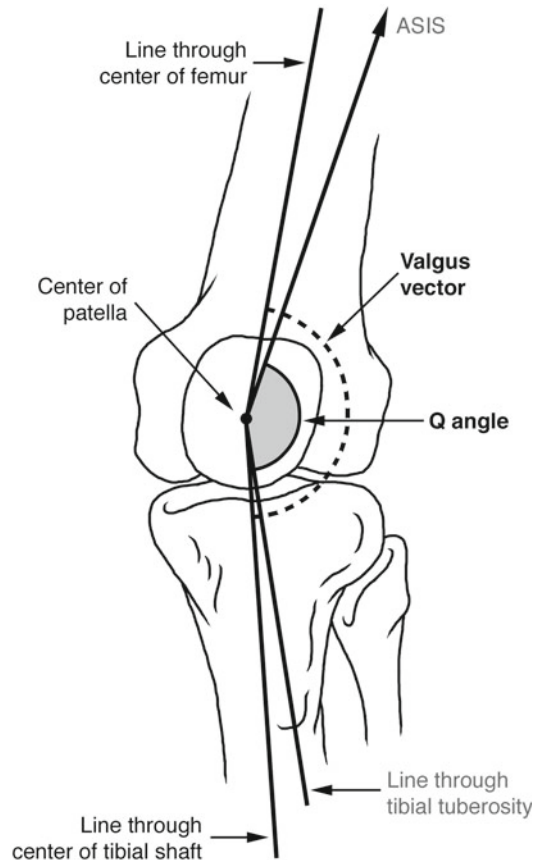


milieu due to monthly hormonal changes (as discussed in Chap. 7). Varying hormones in the circulation have different effects on soft tissue, timing of muscular contraction, and stability of joints. For instance, with the upper extremity, structural differences in young female athletes place them more at a mechanical disadvantage and, therefore, at an increased risk for certain types of overuse injuries. This stems from a shorter humerus compared to total arm length, less muscular strength, and more joint laxity. Consequently, sports involving excessive overhead activity such as throwing and swimming can contribute to rotator cuff tendinosis and shoulder subluxation [4].

The “miserable malalignment syndrome” is quite unique to young female athletes and is a constellation of lower extremity anatomical structures being misaligned. This syndrome is associated with primary quadriceps neuromuscular dominance, placing an increased risk for the knee, especially anterior cruciate ligament (ACL) injuries during various sporting activities [7, 11, 13]. The structural malalignment consists of a widened pelvis, genu valgum, increased internal tibial torsion, and pes planus. The Q-angle is accentuated, contributing to patellar maltracking which can cause anterior knee pain as well [4] (Fig. 15.5).

Additionally, adolescent female athletes tend to recruit their quadriceps prior to their hamstrings (the reverse occurs in males) when they land from a jump, along with a decrease in hip and knee flexion and an elevated valgus knee moment. The resulting misalignment of the lower body places a higher anterior shear force on the tibia which puts more strain on the knee, leading to one of the theories behind a several fold increased incidence of ACL tears in females involved in certain sports, i.e., basketball, soccer, handball, and softball [16, 17] (Fig. 15.6).

Osgood–Schlatter disease is commonly seen in young athletes aged 10–15 years. The patella tendon transfers high amounts of force generated by quadriceps contraction to the tibial tuberosity. Muscular contractions from sprinting and jumping in particular create high forces irritating the tibial tubercle, causing it to become tender and painful with subsequent quadriceps contraction. Increased force can also stimulate bone growth,



**Fig. 15.5** Alignment/forces across the knee joint

increasing the size of the tuberosity. Prominence of the tibial tubercle is seen around the adolescent growth spurt. Treatment for Osgood–Schlatter disease is conservative with stretching, ice, anti-inflammatory drugs, and rest. Knee symptoms begin to abate, then sequester after the adolescent growth spurt, and the disease disappears with the closure of the physis [5, 18].

Collateral ligaments (medial and lateral) are at high risk of injury in adolescent athletes because they originate and insert into the inherently weak physis of the femur and tibia. The distal femoral physis is susceptible to any varus or valgus stress. The collateral ligaments are able to withstand higher amounts of tensile strength compared to the physis; therefore, the physis will fail first with sufficient force. When an injury has occurred to the collateral ligaments, evaluation of the physis needs to be done as well. With isolated collateral ligament injury, treatment can be initiated with



**Fig. 15.6** ACL (anterior cruciate ligament) tear

bracing, ice, rest, and, only in rare instances, surgical repair of high-grade tears [5, 18].

Anterior cruciate ligament (ACL) injuries are not seen in high numbers in adolescent athletes, occurrence of ACL ruptures is rare before age 11 but the incidence increases with age. Female athletes experience ACL rupture at least three to seven times more often than males, most likely due to sex-specific hormones, ligament strength and size, anatomical alignment, and landing differences (discussed further in Chap. 16). Injury to the ACL is seen in deceleration, change in direction (cutting), and in hyperextension during simple leg landing. Issues arise in terms of ligament surgery because most surgical techniques for reconstruction involve crossing the physis. Alterations of the physal plate create concern for premature closure of the physis. Therefore, delay in ligament reconstruction is common in adolescent athletes until the growth plates have closed. However, delaying surgical treatment can cause an increase in knee injuries to cartilage due to the subsequent decline of joint stability [9]. Nonsurgical intervention can include knee

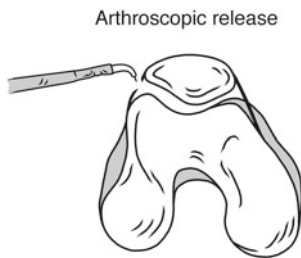
bracing and reduction of activity to ensure there is no more tissue damage (especially the meniscus). Studies have found that injured adolescents treated with knee bracing alone have higher rates of further meniscal damage and in the long term, earlier osteoarthritis [19]. In fact, young athletes treated 12 weeks after ACL injury are four times more likely to develop medial meniscal tears and 11 times more likely to have lateral compartment chondral injuries at the time of surgery [20].

Dislocation of the patella is most common in females aged 14–18 years, most often occurring laterally. The mechanism of injury is most often due to internal rotation of the femur with a fixed foot causing the quadriceps to pull the patella laterally. A less common mechanism is a medial blow to the knee causing lateral dislocation. The dislocated patella, in general, usually spontaneously reduces; however, if it does not reduce, slow extension of the knee with medial force on the lateral patella will cause reduction [9]. The occurrence of subsequent dislocations is between 15 % and 44 %, with higher rates being associated with younger age [21].

Patellofemoral pain syndrome (PFPS) is most common in adolescent female athletes characterized by anterior knee pain. Pain is caused by activities such as repetitive knee flexion, jumping, climbing stairs, and sitting for long periods of time [9]. Treatment for PFPS includes strengthening the quadriceps (especially the vastus medialis oblique), restraint bracing, and patellar taping. Bizzini et al. found that, for adolescent female athletes, the use of orthotics for excessive pronation can help to relieve knee pain [22]. In extreme cases, surgery might be needed to correct PFPS such as lateral release, proximal patellar realignment, and medial tibial tubercle transposition [21] (Fig. 15.7).

Lateral ankle sprains are among the most common adolescent sports-related injury. This injury usually results from inversion of a plantar-flexed foot injuring the anterior talofibular ligament (ATFL) and the calcaneofibular ligament (CFL). Symptoms of an ankle sprain include swelling, bruising, tenderness, and decreased range of motion. Studies have shown that bony tenderness in adolescent sprains is correlated





**Fig. 15.7** Surgical treatment for patellofemoral joint incongruity

with fracture; therefore, any ankle injury with extreme pain and difficult weight bearing should be radiographed to detect fractures. Treatment for ankle sprains consists of rest, ice, elevation, and compression [9].

The radius and the ulna comprise the bones of the forearm. The relative length difference between these two bones is known as ulnar variance (UV). When the ulna is longer than the radius, UV is positive; and, vice versa, when the radius is longer than the ulna, UV is negative. Variation in UV is determined by age, gender, ethnicity, and loading history. While the wrist is not designed to be a load bearing joint, in sports such as gymnastics, a great amount of force is applied to the wrist then transmitted to both the ulna and radius, with the radius receiving 80 % of the load. Particularly in those young athletes with immature bones, repetitive loading of the wrist can cause premature closing of the distal radial growth plate. As the athlete matures, the ulna continues to grow, thus causing a positive UV. Continual loading of the wrist causes other issues such as ulnar impaction syndrome, with progressive degeneration of the fibrocartilage and bones of the ulnar-sided carpus [9].

Injuries to the mid clavicle are one of the most common fractures seen in adolescent athletes. These fractures generally occur from a fall on the shoulder or from direct impact on the bone itself [18]. Surgical fixation of a clavicle fracture is not necessary for healing; therefore, treatment usually consists of bracing, ice, and pain medicine. Another common place for upper extremity fracture is the distal radius, account-

ing for one sixth of all fractures [9]. Symptoms of a fracture to the radius consist of tenderness, swelling, and inability to move the wrist [18]. The most common treatment is casting typically for 4–6 weeks, depending on the severity of the fracture and age of the patient [5].

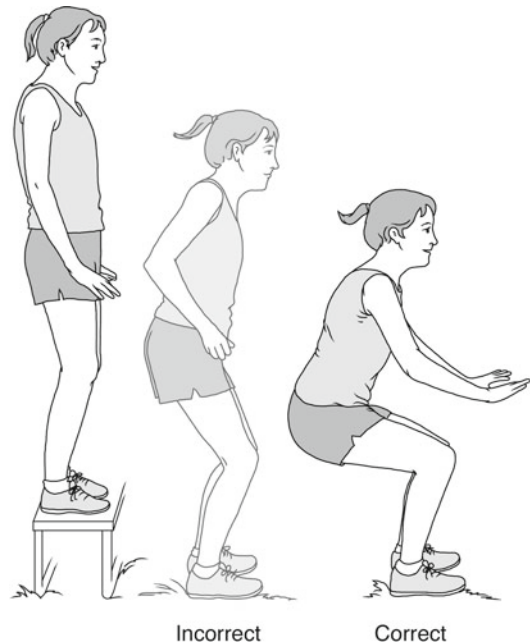
Rotator cuff injuries are uncommon in adolescent athletes. However, injury to the shoulder is common in overhead sports. For example, little league's shoulder causes pain in the proximal humerus due to repetitive throwing. This is thought to be an overuse injury involving the physis and is not limited to baseball players but is also seen in swimming, gymnastics, volleyball, and tennis [5]. Treatment consists of rest, ice, and anti-inflammatory medications. Even though traumatic shoulder dislocations are rare in adolescents, it is the most common dislocated large joint involving athletes in general [9]. Dislocation usually results from an indirect force caused by landing on an abducted externally rotated arm [9]. After a shoulder dislocation, the issue of recurrence arises especially in young athletes. One study found recurrence rates of at least 75 % in athletes younger than 20 years of age [23].

Common back injuries that affect a large portion of adolescents and athletes are spondylolysis and spondylolisthesis involving the lumbar vertebrae, particularly at L4-L5 and L5-S1 levels. Spondylolysis is characterized by a defect in the lamina between the superior and inferior articular facets; whereas spondylolisthesis is the translational motion between adjacent vertebral bodies. There are different classifications of injury for these two mechanisms; however, for the young athlete, repetitive axial loading or hyperextension force causes microfractures and eventually progresses to complete bone failure [9, 24]. Treatment for both of these lumbar spine injuries consists of back bracing and hamstring stretching. Acute trauma to the back and neck (cervical spine) can result from high impact collisions and acrobatic sports. While it is uncommon to see fractures of the thoracic spine (since it is protected by the ribcage), when this traumatic injury does occur, it can result in devastating consequences [24].

### 15.3.4 Several Measures for Prevention of Athletic Injuries from Occurring in Preadolescent and Adolescent Females

The first step in musculoskeletal injuries for adolescent female athletes is prevention. As discussed above, there are different types of orthopaedic trauma incurred by these females during various sporting activities. Acute injuries while playing team sports are difficult to control since other players are involved. However, preventive measures should include employing appropriate safety equipment and avoiding extreme surrounding environmental conditions during actual competitions. In terms of the more chronic type of overuse injury, consider instituting and/or modifying the six S's: altered Structure or anatomic malalignment; Shoe wear pattern or status; Surface type or irregular topography; Stretching for flexibility; Strengthening of imbalanced muscle groups; and avoiding too much Speed too soon. As an example, for those participating in track and field events, correctly fitted footwear, along with correct running stride mechanics is extremely important in the prevention of uneven force distribution and excessive stress transmission through the feet with regard to shoe–ground interaction [25]. Beyond these external variables, the main factors which can be modified through training are: correct technique/appropriate skill level, altered parameter of play, and physical condition of the athlete [10, 26, 27]. For example, at the beginning of the regular sports season, athletes should not increase the volume of their workouts more than 10 % weekly to avoid overtraining [13].

The other very important variable to consider as far as prevention of musculoskeletal trauma in adolescent female athletes is their individual fitness profile. Studies have shown that preseason (several weeks) workouts, i.e., strength, endurance, conditioning, and plyometrics programs can increase physical fitness and help in decreasing the risk of knee/ACL injuries [27–29]. A previous study has shown that even only 8 weeks of training in preadolescents can result in increased muscle strength of ~75 % [8]. Since the period of training is relatively short, these regimens represent the ini-



**Fig. 15.8** Risky versus safe landing positions

tial physiologic adaptations involving the neuromuscular system, incorporating resistance/flexibility exercises, jumping/landing drills, and speed/agility maneuvers; all of these measures contribute toward improving muscular strength/endurance, anaerobic power, and sprint velocity [1, 30]. One of the most crucial elements involved with typical conditioning programs is teaching an athlete how to land with the lower limbs in a “safer” position to help protect against potentially devastating ACL injuries [11, 29] (Fig. 15.8).

### 15.3.5 Different Methods of Orthopaedic Treatment for Musculoskeletal Injuries Incurred by Young Female Athletes, Including Specific Recommendations for a Regular Conditioning Program Once Healing Is Complete in Order to Enhance Their Physical Fitness Profile

Once a female athlete has sustained an acute or overuse type of injury, proper treatment is imperative for a full recovery. Conservative steps can

be implemented if the injury is acute. First, strive to follow PRICE: pain control and Protect the injured part from further harm; relative Rest for the limb (not absolute immobility since the surrounding joints will become stiff) while maintaining gentle ROM (range of motion); apply intermittent Ice for inflammation (20–30 min every 4–6 h); use Compression wraps for swelling; and Elevate the limb above heart level to limit edema. This is basic first-aid type treatment for any kind of soft tissue trauma. If a fracture occurs, whether due to a sudden force or from gradual, repeated stress application, then add the following precaution: no weight bearing on the injured extremity and, if deemed severe enough, strict immobilization with external splinting/bracing or casting. For more specialized management, proceed toward orthopaedic consultation. If the fracture displacement is of sufficient magnitude, it could ultimately require surgical internal fixation [13].

Administration of medications may help with pain and speed up recovery of the injured body part. Over-the-counter nonsteroidal anti-inflammatory (NSAID) drugs can be used for swelling and pain; however, caution needs to be taken as not to remove the body's natural protective mechanism (masking effect). Another consideration is the medication's potential toward a delayed healing response due to blunting of the body's natural process of inflammation. Administration of vitamin C has been shown as a strong anti-inflammatory supplement plus aids in the process of bone building and scar tissue formation. Vitamin C is also an anti-oxidant, which helps in the repair of tissues. However, results are contradictory as far as the role of vitamin C in exercise recovery. In a similar fashion, recent studies have shown both anti-inflammatory as well as anabolic affects of omega-3s, seen particularly in fish oil. Like NSAIDs, caution needs to be taken when consuming these minerals as not to remove the body's response to inflammation and subsequent healing [13].

After an injury has occurred, modification of the training regimen must be modified as to not add further insult to the already injured extremity. The first step is reduction in the levels of

training, i.e., magnitude, intensity, duration, and/or frequency to allow for full recovery after tissue healing. Cross training with a low or no impact type of activity (such as swimming or stationary bicycling) can be beneficial to keep from losing cardiorespiratory fitness. Administration of a formal physical therapy rehabilitation program to regain joint range of motion plus muscular strength/endurance is begun and aimed to progress towards sports-specific exercises after the initial period of recovery. Once functional testing of the affected extremity documents the restoration of objective measures of strength, girth, flexibility, endurance, among other parameters, demonstrating that an athlete is indeed physically recovered back to pre-injury status and again ready to participate in sports, clearance can be granted for gradual return to play [13].

Of utmost importance is the prevention of another injury to the same site; therefore, a conditioning program should be instituted and continued throughout the sports season, as well as extended through the off-season to help maximize performance and minimize injury [12, 13]. Warming up before any exercise by activating muscles around the involved joints appears to afford some protection from risk of injury due to improved proprioception of surrounding soft tissue restraints. However, there is debate on what type of warm-up is most beneficial in terms of maximizing performance and injury prevention. Recent movements in the athletic world have steered away from any type of passive, static stretching. In fact, studies have found that static passive stretching can inhibit neural muscular feedback and decrease peak muscular force up to 120 min after the stretch session [31]. Another study looked at the differences between static and dynamic stretching in 200-m race times; they found that runners who performed dynamic stretches before the race ran faster than those who stretched passively [32]. Contradiction exists in research regarding stretching as injury prevention; some studies have found stretching has a minimal prophylactic protective effect [33, 34], while others have shown better protective results [35]. Several studies have demonstrated the beneficial effects of strength training in children and adolescents in terms of enhancing athletic performance;

with minimal risk of trauma or growth disturbance as long as these programs are well supervised and submaximal exercises are being performed [5, 8, 12, 36, 37]. Specific guidelines regarding a resistance program for building muscular strength include the following: begin at age 7 or 8; add warm-up and cool down periods prior to and after exercise; use own body weight, light medicine balls or dumbbells; perform workouts that last between 20 and 30 min no more than 2–4 days/week (with rest days in between); initially, start with 1 set of 10–15 repetitions 2 times a week; progress to 1–3 sets of 6–15 repetitions concentrating on 6–8 different exercises, focusing on major muscle groups with mild tension/resistance while learning the correct technique of lifting; finally, sequentially accelerate training by small increments of weight (2–5 pounds), or 5–10 % increase in the exercise load weekly once the amount lifted is no longer challenging/fatiguing. For those young athletes who desire to maximize performance in various competitions, they should also practice sports-specific moves/skills as well. Additionally, engaging in any type of aerobic or other endurance type training is also well advised to enhance overall cardiac health in the long term [5, 8, 26, 27, 36]. The only caveat to this principle is that once training stops, regression or detraining occurs after a few to several weeks of inactivity, so the athlete must continue with the conditioning program to retain the highest level of fitness benefits. For all adolescent athletes, power-lifting type maneuvers/drills (i.e., single maximal lifts) should be avoided in order to avoid the potential of growth plate injury to their limbs [5, 26, 27].

## 15.4 Future Directions and Concluding Remarks

In summary, young females, during the time span from childhood to adolescence is accompanied by a myriad of bodily alterations, some of which resemble the changes experienced by males, while others are totally different. The main components involved with this life transition period of puberty include reproductive, hormonal, nervous, and musculoskeletal systems.

The latter undergoes an extreme makeover to involve morphological and neuromuscular transitions. Growth, development, and then eventually maturation into the adult form is faced with an inherent risk, mainly orthopaedic trauma from soft tissues (ligament and musculotendinous structures) lagging behind the fast growing skeletal system (bones). Acute and chronic injuries may occur in preadolescent and adolescent female athletes participating in certain sporting activities. It is important to recognize these unique patterns of musculoskeletal trauma in order to protect the athlete and prevent injury, plus treat the offending problem if need be. When in doubt or if the injury is severe, seek appropriate orthopaedic consultation. The bottom line lies in helping to maximize gains and minimize risks by involving these young females in a well-supervised maintenance conditioning program for the musculoskeletal system. As such, they can compete and perform their best, while adapting to their ever-changing body during their teenage years and even further beyond that into adulthood.

## References

1. Wilmore J, Costill D. Growth development and the young athlete: chapter 17. Special populations in sport and exercise. *Physiology of sport and exercise: human kinetics*. Champaign, IL: Human Kinetics; 1994. p. 401–21.
2. Rogol A, Clark P, Roemmich J. Growth and pubertal development in children and adolescents: effects of diet and physical activity. *Am J Clin Nutr*. 2000; 72:521S–8.
3. Schoenau E, Frost HM. The “muscle-bone unit” in children and adolescents. *Calcif Tissue Int*. 2002 ;70: 405–7.
4. Timmerman M. Medical problems of adolescent female athletes. *Wis Med J*. 1996;95(6):351–4.
5. Cassas KJ, Cassettari-Wayhs A. Childhood and adolescent sports-related overuse injuries. *Am Fam Physician*. 2006;73(6):1014–22.
6. Brooks GA, Fahey TD, Baldwin KM. *Exercise physiology: human bioenergetics and its applications*. 4th ed. New York, NY: McGraw-Hill; 2005.
7. Borms J. The child and exercise: an overview. *J Sports Sci*. 1986;4:3–20.
8. Bencke J, Damsgaard R, Saekmose A, Jorgensen P, Klausen K. Anaerobic power and muscle strength characteristics of 11 years old elite and non-elite

- boys and girls from gymnastics, team handball, tennis and swimming. *Scand J Med Sci Sports*. 2002; 12:171–8.
9. Whiting WC, Zernicke RF. Biomechanics of musculoskeletal injury. Champaign, IL: Human Kinetics; 1998.
  10. Ahmad C, Clark M, Heilmann N, Schoeb S, Gardner T, Levine W. Effect of gender and maturity on quadriceps-to-hamstring strength ratio and anterior cruciate ligament laxity. *Am J Sports Med*. 2006; 34(3):370–4.
  11. Barber-Westin S, Noyes F, Galloway M. Jump-land characteristics and muscle strength development in young athletes a gender comparison of 1140 athletes 9 to 17 years of age. *Am J Sports Med*. 2006;34(3): 375–84.
  12. Hewett T, Ford K, Myer G. Anterior cruciate ligament injuries in female athletes. Part 2, a meta-analysis of neuromuscular interventions aimed at injury prevention. *Am J Sports Med*. 2006;34(3):490–8.
  13. Withrow T, Huston L, Wojtys E, Ashton-Miller J. The relationship between quadriceps muscle force, knee flexion, and anterior cruciate ligament strain in an in vitro simulated jump landing. *Am J Sports Med*. 2006;34(2):269–74.
  14. Verma R, Sherman O. Athletic stress fractures: part II. The lower body part III. The upper body—with a section on the female athlete. *Am J Orthop*. 2001;30: 848–60.
  15. Ogden JA. Anatomy and physiology of skeletal development. In: Ogden JA, editor. *Skeletal injury in the child*. 2nd ed. Philadelphia: Saunders; 1990. p. 42.
  16. Hewett T, Zazulak B, Myer G, Ford K. A review of electromyographic activation levels, timing differences, and increased anterior cruciate ligament injury incidence in female athletes. *Br J Sports Med*. 2005;39:347–50.
  17. Powell J, Barber-Foss K. Sex-related injury patterns among selected high school sports. *Am J Sports Med*. 2000;28(3):385–91.
  18. Kaeding CC, Whitehead R. Musculoskeletal injuries in adolescents. *Adolesc Med*. 1998;25(1):211–23.
  19. Maffulli N, Del Buno A. Anterior cruciate ligament tears in children. *Surgeon*. 2012. doi:10.1016/j.surge.2012.02.003.
  20. Henry J, Chotel F, Chouteau J, Fessy MH, Berard J, Moyer B. Rupture of the anterior cruciate ligament in children: early reconstruction with open physes or delayed reconstruction to skeletal maturity? *Knee Surg Sports Traumatol Arthrosc*. 2009;17:748–55.
  21. Cofield RH, Bryan RS. Acute dislocation of the patella: results of conservative treatment. *J Trauma*. 1977;17:526–31.
  22. Bizzini M, Childs JD, Piva SR, Delitto A. Systematic review of the quality of randomized controlled trials for patellofemoral pain syndrome. *J Ortho Sports Phys Ther*. 2003;33:4–20.
  23. Deitch J, Mehlman CT, Foad SL. Traumatic shoulder dislocation in the adolescents. *Am J Sports Med*. 2003;31:758–63.
  24. Patritti B. Running shoe cushioning impacts foot-ground interface. *Biomechanics*. 2004; 57–67.
  25. Benjamin H, Glow K. Strength training for children: risks versus benefits. Illinois chapter AAP. Illinois pediatrician sports medicine articles. <http://www.illinoisAAP.org/sportsarticles.htm>.
  26. Faigenbaum A, Kang J. Youth strength training: facts, fallacies and program design considerations. *Am Coll Sports Med*. 2005;15(4):5–7.
  27. Faigenbaum A., Chu D. Plyometric training for children and adolescents. *Am Coll Sports Med*. 2001.
  28. Myer G, Ford K, McLean S, Hewett T. The effects of plyometric versus dynamic stabilization and balance training on lower extremity biomechanics. *Am J Sports Med*. 2006;34(3):445–55.
  29. Balabinis C, Psarakis C, Moukas M, Vassiliou M, Behrakis P. Early phase changes by concurrent endurance and strength training. *J Strength Cond Res*. 2003;17(2):393–401.
  30. Bernhardt D. Strength training by children and adolescents. *Am Acad Pediatr*. 2001;107(6):1470–2.
  31. Nelson AG, Driscoll NM, Landin DK, Young MA, Schexnayder IC. Acute effects of passive muscle stretching on sprint performance. *J Sports Sci*. 2005; 23(5):449–54.
  32. Bartlett MJ, Warren PJ. Effects of warming up on knee proprioception before sporting activity. *Am J Sports Med*. 2002;36(2):132–4.
  33. Herbert R, Gabriel M. Effects of stretching before and after exercising on muscle soreness and risk of injury: systematic review. *BMJ*. 2002;325:1–5.
  34. McHugh MP, Cosgave CH. To stretch or not to stretch: the role of stretching in injury prevention and performance. *Scand J Med Sci Sports*. 2010; 20:169–81.
  35. Izquierdo M, Hakkinen K, Gonzalez-Badillo J, Ibanez J, Gorostiaga E. Effects of long-term training specificity on maximal strength and power of the upper and lower extremities in athletes from different sports. *Eur J Appl Physiol*. 2002;87:264–71.
  36. Guy J, Micheli L. Strength training for children and adolescents. *Am Acad Orthop Surg*. 2001;9(1): 29–36.
  37. Power K, Behm MD, Cahill F, Carroll M, Young W. An acute bout of static stretching: effects on force and jumping performance. *Med Sci Sports Exerc*. 2004;36(8):1389–96.