PREPARTICIPATION PHYSICAL EXAM

1. Objectives of the preparticipation physical exam (PPE):
   a. Primary objectives
      i. Screen for conditions that may be life-threatening or disabling.
      ii. Screen for conditions that may predispose to injury or illness during training or competition.
   b. Secondary objectives
      i. Determine general health.
      ii. Serve as an entry point to the health care system for adolescents.
      iii. Provide an opportunity to initiate discussion on health-related topics.

2. Timing, setting and structure
   a. The PPE should be performed at least 6 weeks prior to the start of preseason practice to allow time to further evaluate, treat, or rehabilitate any problem identified. Problems discovered during the PPE that are beyond the expertise of the responsible physician should be directed to an appropriate specialist for referral or consultation.
   b. No standard frequency has been established for PPE among state governing bodies and schools. At the secondary school level, state mandates will dictate PPE frequency. The most recent expert consensus recommends a comprehensive PPE should be performed every 2 years in younger student athletes and every 2-3 years in older athletes. Annual updates should include a comprehensive history questionnaire and a problem-focused examination of any concerns detected in the history.
   c. The PPE can be performed in an individual or group setting. The individual setting in the primary care office with a personal physician familiar with the athlete is preferred. A group setting requires oversight by a team or coordinating physician to ensure that all mandatory elements are performed. Group setting PPEs are not equivalent to the recommended periodic preventive health screening visits for children and adolescents.

3. Standardized form/History & Physical
   a. The medical history is the most important element of the PPE. Medical history alone leads to the diagnosis of 88% of medical conditions and 67% of musculoskeletal conditions detected during the PPE. Any positive responses on the history form should prompt further questions by the examining physician to clarify the issue of concern.
   b. Cardiac
      i. Cardiovascular disorders are the leading cause of sudden death in young athletes. Sudden cardiac death (SCD) is caused by a heterogeneous group of structural and electrical cardiac etiologies.
      ii. The goal of preparticipation cardiovascular screening is to identify or raise suspicion of abnormalities that could provoke disease progression or SCD.
1. In the United States, hypertrophic cardiomyopathy (HCM) and congenital coronary artery anomalies are the most common cause of SCD.

iii. A detailed patient and family history can identify athletes at risk for SCD.

1. Symptoms requiring cardiac workup before participation include exertional chest pain, exertional syncope or near-syncope, unexplained seizures, fatigue disproportionate to the level of exercise, and palpitations or irregular heartbeats.
2. A family history of sudden unexpected death in youth, death before age 50 due to cardiac reasons, sudden infant death, unexplained drowning, or unexplained seizures may indicate the presence of a genetic cardiovascular disorder.
3. Physical exam should focus on detecting the heart murmur of left ventricular outflow tract obstruction, palpation of femoral pulses to exclude aortic coarctation, and identification of physical characteristics suggestive of Marfan syndrome.
   a. Auscultation should be performed in both supine and standing positions (or with Valsalva maneuver). Standing is preferred to sitting because the murmur of HCM becomes louder when the patient is standing due to decreased venous return.

iv. Noninvasive cardiovascular screening as part of the PPE, including ECG and echocardiogram, is highly debated and no current consensus exists regarding universal use.

v. Hypertension

1. Elevated BP is one of the most common abnormal findings during the PPE. BP should be measured on a bare upper arm supported at heart level in the sitting position.
2. Athletes with persistently elevated BP should be questioned about family history and the use of stimulants and steroids.
3. All young athletes with persistent hypertension require evaluation for secondary causes and target organ disease, including serum chemistries, lipid profile, thyroid function tests, hematocrit, urinalysis, and ECG. Renal ultrasound is also recommended for athletes with stage 1 or stage 2 hypertension. Athletes with stage 2 hypertension or evidence of end-organ damage should be held from competitive sports until BP is further evaluated, treated, and under control.

c. Concussion

i. Concussions are common in young athletes, often unrecognized and underreported, and generally do not involve loss of consciousness.
ii. Concussion history is vital when determining participation, as athletes are more susceptible to subsequent concussions, may take longer to recover or occur with less trauma.
iii. Athletes should never return to sports after sustaining a concussion
until without concussion related symptoms and complete a graded return to play rehabilitation program. All return to play decisions are individual.

iv. If improperly treated, concussion sequela may include second impact syndrome, postconcussion syndrome, or persistent neurologic deficits such as decreased cognitive function.

d. Obesity

i. Although obese athletes may have associated conditions (i.e. hypertension, exercise-induced asthma, susceptibility to heat illness, diabetes, slipped capital femoral epiphysis), there is no reason to exclude them from sports participation because of their weight alone. Regular activity and sports participation should be encouraged.

e. Medications and Supplements (i.e. stimulants/PEDs)

i. The PPE is a great opportunity to discuss and counsel athletes and parents about the risks of medications, supplements, or ergogenic aids including steroids.

ii. Supplement use must be screened for both issues of safety and eligibility.

f. Anaphylaxis/allergies

i. Personal history anaphylaxis/allergic reactions and allergic triggers should be noted.

ii. Outdoor environments potentially expose the athlete to stinging insects and allergens that can induce anaphylaxis.

iii. Athletes with a history of true anaphylaxis should have an EpiPen or epinephrine device on-site for immediate use.

g. Heat Illness

i. Exertional heat stroke (EHS) can be fatal and is most often seen in preseason football. Major EHS risk factors include hot, humid conditions; poor aerobic fitness; football equipment; and inadequate heat acclimatization.

ii. The use of diuretics, caffeine, antihistamines, or stimulants increase the risk of heat illness.

h. Musculoskeletal

i. A general screening exam is reasonable for asymptomatic athletes with no previous injury. It can be used to quickly assess joint range of motion, gross muscle strength, and muscle symmetry.

ii. Stress fractures and recurrent soft tissue injuries can be associated with nutritional deficiencies or overuse.

i. Considerations for Female Athletes

i. Disordered eating is relatively common in female athletes. Eating disorders and energy imbalance may be associated with amenorrhea, persistent injury, recurrent injury, or stress fractures.

ii. Anemia is associated with heavy or frequent menstrual cycles and nutritional energy deficit.

iii. Vitamin D deficiency is increasingly common.

4. Clearance
a. Determining clearance is an important and sometimes difficult decision. Studies show that 3.1% to 13.9% of athletes require further evaluation before a final clearance status can be determined.

b. The initial clearance status for an athlete can be divided into 4 categories:
   i. Cleared for all activities without restriction
   ii. Cleared with recommendations for further evaluation or treatment
   iii. Not cleared - clearance status to be reconsidered after completion of further evaluation, treatment, or rehabilitation.
   iv. Not cleared for certain types of sports or for any sports

5. Medical conditions and sports participation
   a. Atlantoaxial instability
      i. Qualified Yes - needs evaluation to assess risk of spinal cord injury during sports participation, especially when using a trampoline.
   b. Cardiovascular disease
      i. Carditis
         1. No - may result in sudden death with exertion
      ii. Hypertension
         1. Qualified Yes - athletes with severe hypertension (>99th percentile for age plus 5 mmHg) should avoid heavy weight and powerlifting, bodybuilding, and high-static component sports. Those with sustained hypertension (>95th percentile for age) need evaluation.
      iii. Dysrhythmia.
         1. Qualified Yes - those with symptoms (chest pain, syncope, dizziness, shortness of breath) need evaluation with a cardiologist prior to sport participation.
   c. Structural/acquired heart disease
      i. All athletes with heart disease should be followed by cardiology, and decisions regarding athletic participation should be made in conjunction with a cardiologist.
         1. Hypertrophic cardiomyopathy
            a. Qualified No
         2. Coronary artery anomalies
            a. Qualified No
         3. Ehlers-Danlos, vascular involvement
            a. Qualified No
         4. Marfan syndrome
            a. Qualified Yes - athletes with Marfan syndrome are at increased risk for aortic aneurism, which can cause sudden death during intense exercise.
         5. Mitral valve prolapse
            a. Qualified Yes
         6. Anthracycline use
            a. Qualified Yes - athletes who have ever received chemotherapy with anthracyclines are at increased risk of cardiac problems due to the cardiotoxic effects of the
medication. Resistance training should be approached with caution. Cardiology evaluated is warranted.

d. Diabetes Mellitus
   i. Yes - all sports can be played, with particular attention to diet, blood glucose concentration, hydration, and insulin therapy.

e. Fever
   i. No - fever can increase cardiopulmonary effort, reduce maximum exercise capacity, make heat illness more likely, and increase orthostatic hypertension during exercise.

f. Heat illness, history of
   i. Qualified Yes - due to concern for recurrence, the athlete needs to be assessed for the presence of predisposing conditions, and develop a prevention strategy, including sufficient acclimatization, conditioning, hydration, and salt intake.

g. HIV
   i. Yes - however, if a viral load is detectable, these athletes should avoid high-contact sports such as wrestling or boxing which may create a situation that may favor viral transmission due to bleeding. Skin lesions should be properly covered and athletic personnel should use universal precautions.

h. Neurologic disorders
   i. Concussion, history of
      1. Qualified Yes - athletes need individual assessment prior to participation. Athletes should not participate while symptomatic following concussion, and complete graded return to play rehabilitation.
   ii. Recurrent plexopathy (burner or stinger)
      1. Qualified Yes - athlete needs individual assessment for collision, contact, or limited-contact sports. Regaining normal strength is an important benchmark for return to play.
   iii. Seizure disorder
      1. Qualified Yes - if seizures are poorly controlled, the athlete should avoid archery, riflery, swimming, weight or powerlifting, or sports involving heights.

i. Obesity
   i. Yes - because of increased risk of heat illness, the obese athlete particularly needs careful acclimatization, sufficient hydration, and potential activity and recovery modifications during competition and training.

j. Respiratory conditions
   i. Asthma
      1. Yes - with proper medication and education, only athletes with particularly severe asthma will need to modify their participation. Athletes with persistent asthma or exercise-induced asthma should have rescue inhaler available during exercise. Athletes with asthma may encounter risks when
scuba diving.

k. Sickle Cell Disease
   i. Qualified Yes - sports that place the athlete at risk for overexertion, overheating, dehydration, and chilling should be avoided. Participation at high altitude, especially when not acclimatized, also poses risk of sickle cell crisis.

l. Skin infections
   i. Qualified Yes - during the contagious period, all contact and limited-contact sports, and sports involving mats, such as gymnastics, should be avoided.
      1. These include, but are not limited to, molluscum contagiosum, staphylococcal and streptococcal infections (cellulitis, abscess, boils, impetigo) herpes, scabies, and tinea.

m. Spleen, enlarged
   i. Qualified Yes - if the spleen is acutely enlarged, participation should be avoided because of risk of rupture, i.e. athletes with infectious mononucleosis should be held out of competition for at least 1 month following onset to reduce risk of splenic rupture. If the spleen is chronically enlarged, individual assessment is needed before contact sports are played.
HEAT ILLNESS

Overview of Heat Illness
1. Physiology
   a. Body produces heat through exercise
   b. Body unable to dissipate heat into hot environment
   c. Eventual death if no cooling occurs
2. Types of heat illness - May occur simultaneously
   a. Sunburn
   b. Heat rash
   c. Dehydration
   d. Heat cramps
   e. Hyponatremia
   f. Heat exhaustion
   g. Heat stroke
      i. Exertional
      ii. Non-exertional
   h. Exercise-associated collapse
3. Management may include:
   a. First aid
   b. Stop activity / exercise
   c. Immediate cooling
   d. Transport to hospital

Mechanism
1. Heat generation
   a. At rest
      i. Blood transfers heat from internal organs to skin
   b. During exercise
      i. Increased heat production
      ii. Increased blood flow
         1. Muscle (deliver nutrients)
         2. Skin (cooling)
2. Cooling Mechanisms
   a. Vasodilation
      i. Air temperature
   b. Sweating
      i. Humidity
      ii. Rate
         1. Dependent on age
         2. Acclimatization to heat
3. Thermoregulatory failure
   a. Mechanism
      i. Increase blood flow to skin and muscle during exercise
      ii. Decreased central blood volume
      iii. Baroreceptors sense hypotension
iv. Vasoconstriction of peripheral vessels
v. Decreased heat transfer across skin
vi. Heat production exceeds dissipation
   1. Body temperature rises
   2. Proteins denature
   3. Cell apoptosis / necrosis
   4. Endotoxin release
   5. Generation of toxic free radicals
   6. Multi-organ failure
   7. Death

b. Risk Factors
i. High wet bulb globe temperature (WBGT)
   1. WBGT considerations
      a. Air temperature
      b. Humidity
      c. Radiant heat
   2. ACSM recommendations for exercise based on risk to athletes
      a. Green - lowest risk
      b. Yellow
      c. Red
      d. Black - highest risk, cancel activities
   3. Exposure to high WBGT the previous day

ii. Dehydration
    1. Lower plasma volume available for cooling
    2. How to monitor
       a. Pre and post-exercise weight
          i. No exercise if over 1-2% change from previous day
       b. Urine color / spec gravity

iii. Lack of acclimatization to weather conditions
    1. ACSM guidelines
    2. Takes longer in children than adults

iv. Pre-pubertal
   1. Greater surface area to body mass ratio
   2. Greater heat gain from environment
   3. Produce more heat per kg during exercise
   4. Decreased sweat rate
   5. Do not recognize dehydration
   6. Peer pressure to continue participation

v. Co-morbid conditions
   1. Sleep deprivation
   2. Recent febrile illness
   3. Recent GI illness
   4. Salt deficiency
   5. Malnutrition
   6. Skin condition
   7. Obesity
8. History of previous heat illness
   vi. Impermeable garments
   vii. Personality type that is reluctant to report problems
   viii. Medications / supplements
       1. Antihistamines
       2. Stimulants
       3. Diuretics
   ix. Improper training
       1. Low physical fitness
       2. Prolonged exercise
       3. Effort unmatched to fitness
       4. Improper work / rest cycles
       5. Training during hottest hours
       6. Disregard training regulations regarding WBGT
       7. Limited access to fluids
   x. Improper medical response increases risk of death due to heat stroke
       1. Absence of appropriate medical triage
       2. Improper diagnoses
       3. Improper treatment
       4. Lack of education
       5. No emergency plan

4. Prevention
   a. Hydration
      i. Easy access to fluids
      ii. Frequent water breaks
      iii. Water if exercising less than 1 hour
      iv. Sports drinks if exercising over 1 hour
         1. Replace electrolytes
         2. Taste encourages children to drink
   b. Proper acclimatization
      i. Body adapts cooling mechanism
         1. More efficient heat dissipation
            a. Expanded plasma volume
            b. Increased sweat rate
               i. Requires increased fluid and electrolyte intake to maintain
            c. Decreased core temperature at rest
            d. Decreased HR during exercise
            e. Sweating and vasodilation occur sooner during exercise
            f. More dilute sweat
      ii. Pre-pubertal children take longer to adapt
   c. Timing
      i. Exercise during cooler times of day
   d. Clothing
      i. Light-colored
      ii. Lightweight
iii. Replace if sweat-saturated
e. Minimize modifiable risk factors

Types of Heat Illness

1. Heat Exhaustion
   a. Epidemiology
      i. Common
      ii. Often unreported
      iii. No accurate data
   b. History
      i. Nausea / vomiting / diarrhea
      ii. Headache
      iii. Dizziness
      iv. Weakness
      v. Decreased coordination
      vi. Abdominal cramps
      vii. Dehydration
      viii. Fatigue
      ix. Syncope
   c. Physical
      i. Obvious difficulty continuing intense exercise
      ii. Tachypnea
      iii. Tachycardia
      iv. Hypotension
      v. Pale wet skin
      vi. Moderate hyperthermia
         1. Rectal temp ranging 37.7 - 39.4°C (100-103°F)
      vii. No severe CNS dysfunction
      viii. Symptoms quickly reverse with cessation of activity
   d. Treatment
      i. Stop exercise
      ii. Move to cool area
      iii. Remove extra clothing / equipment
      iv. Trendelenburg position
      v. Cool to rectal temp of 38.3°C (101°F)
      vi. Rehydrate
         1. PO
            a. Normal CNS
            b. No nausea / vomiting
            c. Sports drinks or water
         2. IV
            a. Unable to tolerate PO
            b. Normal saline or lactated ringers
   vii. Monitor
      1. Temperature
      2. CNS status
viii. Transfer - treat as heat stroke if growing worse or no rapid improvement

e. Complications
   i. Heat stroke

d. Outcomes / future risk
   i. No long term effects
   ii. Increased risk of future events

g. Return to Sports
   i. Symptom-free
   ii. Fully hydrated
   iii. Cleared by physician
   iv. Manage predisposing conditions
   v. Avoid intense exercise in the heat for at least 24 hours

2. Heat Stroke

   a. Epidemiology
      i. 3rd leading cause of death in adolescent athletes
      ii. 100% cure rate if managed properly

   b. Types
      i. Exertional
         1. Body generates extra heat due to exercise
         2. Occurs in many environmental conditions
      ii. More likely in hot / humid conditions
      iii. Non-exertional
         1. Body generates normal metabolic heat
         2. Unable to cool due to environmental conditions

   c. History
      i. Similar symptoms as heat exhaustion

   d. Physical
      i. Worsening heat exhaustion findings
      ii. Skin
         1. Exertional heat stroke
            a. Cool, clammy, pale skin
            b. Continues to sweat
         2. Non-exertional heat stroke
            a. Hot, red skin
            b. Does not sweat
      iii. Mild cognitive dysfunction
         1. Combative
         2. Confused
         3. Strong desire to continue exercise
         4. Refuse medical care
         5. Grows worse if untreated
            a. Loss of consciousness
      iv. Rectal temperature 40C (104F) or higher
      v. Does not improve upon cessation of exercise

   e. Treatment
i. Cool first
   1. Ice water submersion
   2. 100% survival if done within 10 minutes of onset
   3. Slow the cooling process when rectal temperature reaches 38.3C (101F)

ii. Monitor
   1. ABCs
   2. Core temperature
   3. CNS function

iii. IV fluids
   1. Rehydrates
   2. Cools

iv. Consider differential diagnosis for altered CNS in athletes
   1. Hyponatremia
   2. Concussion

v. Transport
   1. Evaluate for end-organ damage
      a. metabolic acidosis
      b. cardiac ischemia
      c. cerebral ischemia
      d. pre-renal azotemia
      e. anemia
      f. thrombocytopenia
      g. DIC
      h. GI mucosal swelling
      i. GI petechiae
      j. GI hemorrhage
      k. cholestasis
      l. liver necrosis
      m. elevated liver enzymes
      n. coagulopathy

f. Complications
   i. Multi-organ failure
   ii. Death

g. Outcomes / future risk
   i. Permanent organ damage possible
   ii. Permanent decreased heat tolerance possible

h. Return to Sports
   i. No universal guidelines
   ii. May have permanent compromised heat tolerance

iii. Recommendations
   1. Establish normal organ function, asymptomatic
   2. Rest 1 week
   3. Physician clearance and close supervision
   4. Light exercise in cool environment
   5. Intense exercise in cool environment
6. Exercise heat tolerance test
7. Light exercise in the heat
8. Light exercise in the heat, with equipment
9. Intense exercise in the heat, with equipment
10. Normal practice / games

3. Exercise-Associated Collapse
   a. Also called heat syncope
   b. Findings
      i. Exercise
      1. Peripheral vasodilation
      ii. Abrupt cessation of exercise
         1. Orthostatic hypotension
         2. Syncope or near-syncope
      iii. Corrected once athlete is supine
      iv. No further intervention needed

4. Heat Cramps
   a. Findings
      i. Sweat
         1. Fluid and electrolyte loss
         2. Muscle deficiency of fluids and electrolytes
         3. Painful spasms in isolated muscle groups
   b. Treatment
      i. Replenish fluids and electrolytes
         1. PO - sports drinks
         2. IV - normal saline or lactated ringers

5. Hyponatremia
   a. Presentation
      i. Fluid intake greater than loss
      ii. Inexperienced athletes
      iii. Endurance events
   b. Physical findings
      i. CNS changes
      ii. Weight gain
      iii. Extremity edema
      iv. Normal or slightly elevated rectal temperature
   c. Diagnosis
      i. Serum sodium
      ii. Begin cooling while waiting for results
Concussion

1. Definition: Concussion, as defined by the Consensus Statement on Concussion in Sport:
   - A complex pathophysiological process affecting the brain, induced by traumatic biomechanical forces
   - This definition includes several concepts that make it easier to understand:
     a. Force is either from a direct blow to the head or from transmission from the body to the head (e.g., whiplash injury). A blow to the head is not necessary to have a concussion.
     b. The brain injury induces functional, not structural, problems. This concept is important for understanding symptoms and treatment. Additionally, this limits the need for neuroimaging.
     c. Concussion symptoms are, by definition, temporary. They should resolve over a fairly predictable time period with the appropriate treatment. However, some patients go on to develop longer term symptoms (post-concussion syndrome).
   - The pathophysiology of concussion is that of a cascade of electrolyte and biochemical abnormalities that persist for days or weeks. A significant amount of energy is needed for neuron healing, while there is less energy available for healing (less blood flow to brain, resulting in decreased nutrient availability).
     a. An ‘energy crisis’ is created in the brain. Any mental or physical exertion during the recovery period may worsen the energy crisis, resulting in worsened symptoms and prolonged recovery.

2. Epidemiology:
   - The CDC estimates 1.6-3.2 million concussions occur in sport or recreation each year (adults and children).
   - The underreporting rate for concussion is thought to be at least 50%.
     a. Factors for underreporting include lack of athlete education/knowledge of concussion and fear of having to miss sports for recovery.
   - Concussions occur more often in organized sports than in non-organized leisure physical activities.
   - Female athletes have been shown to sustain more concussions in games than male athletes. High risk sports for concussion include football, soccer (men and women), lacrosse (men and women), and women’s basketball.

3. Pediatric and adolescent concussions
   - This curriculum focuses on the young patient with concussion. Adult concussions are discussed elsewhere. Differences between adult and pediatric concussions (in general) include:
     a. Recovery time is, in general, longer than for adult patients. There is good evidence showing average recovery (for uncomplicated patients) of 7-10 days for college age patients and 2 weeks for high school age patients.
There is little consistent data on length of recovery for patients prior to high school age; however, 3-4 week recovery periods are common.

b. Two to three times greater force to the brain is needed to produce clinical symptoms in children. When a child experiences concussion symptoms, it is likely that a large force has been sustained.

c. Cerebral swelling may occur more often in children than in adults. Second impact syndrome (discussed below) has been reported only in children and adolescents.

- The pediatric concussion patient has unique challenges in treatment.
  a. Academic considerations (missing school, delaying assignments) are a major concern.
  b. Comorbidities such as ADHD, depression, and learning disabilities may complicate recovery, especially academically.

4. History

- Concussion signs/symptoms:
  • Impaired consciousness (loss of consciousness is NOT necessary for concussion diagnosis)
  • Amnesia, pre- and/or post-traumatic (again, NOT necessary for diagnosis)
  • Easy distractibility/poor concentration
  • Short-term memory difficulties
  • Feeling ‘dazed’, ‘foggy’, ‘stunned’
  • Fatigue or feeling of slowness
  • Poor coordination/balance
  • Inappropriate emotions, irritability, or personality changes
  • Headache/pressure feeling
  • Dizziness
  • Nausea and vomiting

- Important History Items (have been shown to influence recovery time)
  • Personal history of concussion (number and length of recovery time for each)
  • Personal history of mood disorders, ADHD, learning problems, headaches
  • Family history of headache, particularly migraine
  • Academic history

- If head injury is suspected, always ask about neck injury.

5. Physical exam

- Exam is usually normal except for mental status. A full neurological exam should be done.
  • If neurological exam is abnormal (except for balance), consider neuroimaging to evaluate for intracranial bleeding.
  • Assess orientation, memory (immediate and delayed), and concentration.
• Balance has significant impairment after concussion and should be adequately assessed. The Balance Error Scoring System (BESS) is a validated tool used to assess deficiencies and improvement through recovery.
• For sideline/immediate evaluation, brief tools are available online.
  a. Standard Assessment of Concussion (SAC) - copyrighted
  b. Sport Concussion Assessment Tool 3 (SCAT3) – not copyrighted
• Scalp, facial, and neck exams are necessary with any head injury.

6. Differential diagnosis
• Concussion
• Headache/migraine
• Epidural hematoma
• Subdural hematoma
• Intracerebral hemorrhage
• Skull fracture
• Seizure

7. Need for urgent evaluation and neuroimaging
• ANY prolonged loss of consciousness (there is controversy over what constitutes ‘prolonged’, but anything over a few minutes is generally assumed), neurological abnormality on exam, or worsening state of consciousness requires urgent evaluation and likely neuroimaging.
  o If a sideline evaluation is being done, urgent transport to an emergency facility is warranted.
  o Need high degree of suspicion for intracranial hemorrhage with any head injury.
• Neuroimaging is being done less frequently in general, as better correlation of CT and cancer has been made. In addition, MRI is not usually available in the emergency environment.
  o Several criteria for neuroimaging are available but should be interpreted cautiously, as most include headache as criteria for imaging.
• Athletes with stable consciousness and ability to be reliably monitored can likely avoid neuroimaging.
• As above, carefully assess for neck injury in the head injured patient and evaluate with radiographs/CT if warranted.

8. Complications
• There are two main complications of concussion:
  a. Post concussion syndrome (PCS): When concussion symptoms go on longer than expected, post concussion syndrome should be diagnosed.
    i. Usual diagnosis made 3 months after concussion; however, some clinicians make diagnosis as early as 1 month after injury.
    ii. Involves significant life disruption, which may include cognitive (academic difficulties), physical (unable to exercise or play sports), somatic (chronic headache, sleep abnormalities), and psychosocial
(depression, worsened ADHD, difficulty participating in social events) abnormalities.
iii. Diagnosis is made clinically and should include formal neuropsychological consultation.

b. Second Impact Syndrome (SIS): A very rare but usually fatal condition occurring when a second brain injury is sustained prior to resolution of an initial head injury (within hours to days).
   i. Thought to occur from diffuse cerebral swelling.
   ii. Several cases are reported each year, and all have been in children or adolescents. No adult cases have been reported.
   iii. Can be totally prevented with recognition of concussion and appropriate restriction from further injury.

- Any neuropsychological condition complicates recovery and should be considered when counseling the patient/family.
- Concussion risk is thought to increase with each successive concussion;

9. Treatment
- Limiting physical and cognitive activity to symptom free or near symptom free likely accelerates, healing; however, there is no definitive research yet supporting this.
  a. Anything that worsens the ‘energy crisis’ may make recovery more difficult.
- Physical rest: No sports activity. No running, weight training, or drills. Activity restrictions may change based on recovery.
- Cognitive rest: Both academic and in activities of daily living
  a. Must be individualized and based on symptoms. Not every student needs significant reductions in cognitive activity and as symptoms worsen or improve, recommendations may change
  b. Brief time away from school or shortened school day may be necessary. Prolonged time away from school is generally unnecessary and may provoke depressive symptoms in children having been cut off from normal social situations. Also may increase stress related to getting caught up with work missed.
  c. Modifying reading, math, language skills, etc.
  d. Reduced duration homework, projects, and testing including breaks as needed.
  e. Reduce texting, computer work, video games, and television
  f. Modify or consider brief avoidance of socially stimulating environments, such as games, dances, church, etc.
  g. For first few days, extra sleep is acceptable. Encourage patients to reestablish normal sleep schedule as soon as possible. Significantly altered sleep schedules may worsen certain symptoms.
- Medications:
  a. Headache: Acutely, acetaminophen may be used if headache is severe; however, rest should be attempted first to control symptoms. Although
there is no concrete evidence that non-steroidal anti-inflammatory drugs
cause intracranial bleeding, there is some laboratory evidence of such,
and they should be generally limited in their use.
b. Sleep: Melatonin may be used for sleep initiation, if needed.

- PCS Treatment
  a. Medication is often needed to control headaches and to treat
     complications of PCS (e.g., depression, ADHD, etc.).
  b. Counseling (individual and family) is useful to help the patient deal with
     the diagnosis.
  c. Speech pathology, pediatric pain management, and occupational/physical
     therapy consultation may be helpful.

10. Use of neurocognitive and neuropsychological testing
    a. Neurocognitive testing: Brief (20-30 minute) computerized screening tools
       used to objectively measure brain processing speed and reaction time
       i. Recommended as a tool to assist with concussion management
       ii. Most commonly used: ImPACT, CogState, Concussion Vital Signs,
           ANAM
       iii. With athletes, post-concussion testing is often compared to a
            ‘baseline’ test obtained prior to the season in order to assess whether
            the athlete has recovered normal brain function.
       iv. Usually done with acute concussions to help assess resolution, as
           some children have shown abnormal cognitive function even after
           symptoms have resolved.
       v. Available through some primary care sports medicine physicians and
           at some high schools.
    b. Neuropsychological testing: Extended (several hours) testing done by a
       neuropsychologist to determine underlying cognitive or behavioral problems
       interfering with concussion recovery.
       i. Helps guide academic accommodations.
       ii. Usually not done until diagnosis of PCS is made.

11. Return to Play recommendations
    - An athlete should NEVER return to play while still symptomatic
      a. Increased risk for SIS and PCS
      b. The brain is likely more sensitized to injury during this time, and a lesser
         force will likely produce more injury.
    - Return to play should only be considered when three criteria are met:
      a. No concussive symptoms at rest
      b. No concussive symptoms with exertion (cognitive and physical)
      c. Normal brain function (normal neurocognitive testing)
    - Suggested protocol, with each step separated by ~24 hours (to watch for
      symptoms occurring after activity). Advance through the protocol only occurs if
      the patient remains symptom free. If symptoms recur, the patient should go
      back to the last asymptomatic step.
a. Light aerobic exercise
b. No resistance training
c. Sport-specific exercise – start resistance training
d. Non-contact training drills
e. Full-contact training
f. Game play

- Although specifics of the return to play protocol may vary slightly, it will take any concussed athlete multiple days to fully return to sport.

12. Strategies to decrease risk of concussion

- Very little evidence in this area
- Protective equipment
  a. Although helmets reduce the probability of a catastrophic head injury, no helmet definitively reduces concussions. No helmet can prevent all concussions.
- Rule enforcement in sport
- Strong skills and high fitness level can allow the athlete to avoid all types of injury, including head injury (avoiding the wrong place, wrong time, etc.)
- Poor neck strength in women, as compared to men, has been proposed as a reason for increased concussion incidence.
- Mouth guards have not been shown yet to definitively decrease concussion incidence but do decrease dental injuries.
APOPHYSEAL INJURIES – Acute and Overuse

1. Definitions
   a. Physis – The Growth Plate. Where new bone is formed for longitudinal growth
   b. Epiphysis – Area of bone between the physis and the joint
   c. Metaphysis – Area of bone between the physis and the shaft (diaphysis) of the bone
   d. Diaphysis – the shaft of the bone
   e. Apophysis – Area of growth that does not provide for longitudinal growth but is an attachment site for a tendon.

2. Apophyseal avulsion fracture vs Apophysitis
   a. Avulsion fracture
      i. An acute injury that results in a significant distraction/separation of the apophysis from its normal attachment site.
      ii. This injury is equivalent to a tendon rupture in an adult.
iii. Often can result in an acute significant loss of function of the tendon that attaches to where the injury has occurred.

b. Apophysitis
   i. Can occur with or without overuse but often is an overuse injury
   ii. A result of repetitive traction of the tendon on the apophysis
   iii. NOT a typical “itis,” as very little inflammation, if any, occurs

3. Osgood-Schlatter Apophysitis (Tibial Tuberosity Apophysitis)
   a. Anatomy
      i. Tibial tuberosity located on anterior portion of proximal tibia
      ii. Site of attachment for patellar tendon
   b. Epidemiology
      i. Most common source of anterior knee pain in preadolescent athlete
      ii. Males slightly more common than females
      iii. Bilateral in 20-30% of cases
      iv. Commonly occurs age 8-13 in females, age 10-15 in males
         1. Varies depending on individual’s skeletal maturation
      v. May have a genetic predisposition in families
   c. History
      i. Gradual onset of pain; usually no acute injury
      ii. Typically worst with running and jumping activities; Also can be aggravated by kneeling or applying direct pressure to the proximal tibia
      iii. May start limping
   d. Physical Examination
      i. May have prominence or swelling of tibial tuberosity
      ii. May be limping
      iii. Tender to palpation over tibial tuberosity of affected leg(s)
      iv. Often with pain to resisted leg extension
      v. May have poor flexibility of quadriceps and hamstrings
      vi. Be sure to examine hips as hip pathology may present as knee pain
   e. Differential diagnosis
      i. Patellofemoral pain syndrome
      ii. Sinding-Larsen-Johansson apophysitis
      iii. Proximal tibial stress fracture
      iv. Less common – bone or soft tissue tumors, leukemia, osteoid osteoma
   f. Imaging Studies
      i. X-rays – Osgood-Schlatter is a clinical, NOT x-ray diagnosis
         1. If x-rays obtained, may be normal or show prominence of tibial tuberosity. May also see fragmentation as a result of chronic stress.
      ii. CT or MRI – Generally not indicated for the diagnosis and management of Osgood-Schlatter
   g. Treatment
      i. Relative rest – decreasing the aggravating activity often can reduce or resolve pain. Complete rest may be needed if severe pain or causing athlete to limp with activity
ii. Stretching – Focused on quadriceps and hamstrings
iii. Icing – After activity; Can be done with ice bag (or bag of frozen peas or corn) for 15 minutes directly over tibial tuberosity; also can use ice massage with focal icing for 5 minutes with Dixie Cup filled ¾ full with water and frozen. Paper is peeled back on the cup to expose the ice to allow for ice massaging.
iv. Patellar tendon straps – Can be used with activity. Place between inferior pole of patella and tibial tuberosity – directly over patellar tendon.
v. NSAIDs/Acetaminophen – May be used for pain relief after activity

h. Return to Play Recommendations
i. Athletes may play with Osgood-Schlatter but should curtail activity if pain is affecting performance, reducing the “fun” of playing their sport, or causing them to limp while active.

i. Complications
   i. Avulsion of tibial tubercle
      1. Acute injury that produces avulsion of tibial tubercle.
      2. Athlete will be unable to bear weight or extend knee against resistance, equivalent to a patellar tendon rupture in an adult.
      3. Rare complication.
      4. Requires surgical management
   ii. Persistent pain after apophysis fuses
      1. Also not common but if does occur in some refractory cases may require surgical management

j. Outcomes/future concerns
   i. Athletes typically do well with proper treatment and maintenance of the condition
   ii. See complications for persistent pain

k. Prevention
   i. No specific way to prevent completely.
   ii. Stretching while going through growth spurts is helpful
   iii. Addressing pain early in course rather than reacting after more painful can help shorten duration of symptoms

4. Sinding-Larsen-Johansson (SLJ) Apophysitis
   a. Identical to Osgood-Schlatter but SLJ affects an apophysis localized to the inferior pole of the patella.
   b. Patients present identical to Osgood-Schlatter with tenderness to palpation over the inferior pole of patella
   c. May have complication of a patellar sleeve avulsion fracture. Many can be treated non-operatively but may need surgical management.
   d. Treatment of SLJ similar to Osgood.

5. Sever’s Apophysitis (Calcaneal Apophysitis)
   a. Anatomy review
      i. The calcaneal apophysis is at the most posterior aspect of the calcaneus
      ii. Site of attachment for the Achilles tendon
b. Epidemiology
   i. Most common source of heel pain in the pre-adolescent athlete
   ii. Frequently occurs in sports requiring the use of cleats (soccer, football, baseball, lacrosse, field hockey)

c. History
   i. Pain localized to heel
   ii. Frequently increases with running
   iii. May start walking on forefoot/toes to avoid putting pressure on the heel
   iv. May cause a limp
   v. Typically no history of acute injury

d. Physical exam
   i. Tender directly over calcaneal apophysis
   ii. Usually no soft tissue swelling or bruising
   iii. Calcaneal squeeze test elicits pain
   iv. Often have poor flexibility of calf muscles/Achilles tendon

e. Differential diagnosis
   i. Calcaneal stress fracture
   ii. Heel contusion/stone bruise
   iii. Posterior tibialis or peroneal tendonitis
   iv. Plantar fasciitis (not common in pre-adolescent age group)

f. Use of imaging studies
   i. X-rays – not necessary if classic history and exam. If obtained, may see appearance of increased density of the bone in the apophysis. Usually does not fragment or avulse.

g. Treatment
   i. Relative rest from offending activities
      1. May require time off if limping or in continuous pain
   ii. Ice after activity
   iii. Achilles (gastrocnemius and soleus) stretching
   iv. Viscoelastic gel cushions for shoes
   v. NSAIDs or acetaminophen after activity for pain, if needed
   vi. Consider using turf shoes instead of cleated shoes for sports

h. Return to play recommendations
   i. If no limp or not having severe or continuous pain, may consider return to activity

i. Complications
   i. Typically not common
   ii. May develop calcaneal stress fracture

j. Outcomes and future concerns
   i. Usually no long term issues

k. Prevention
   i. Reduction in time spent in running in sports
   ii. Adequate and appropriate footwear for sports
   iii. Stretching of calf

6. Little League Elbow (Medial Epicondylar Apophysitis)
a. Anatomy review
   i. The medial epicondyle is an apophysis that ossifies around age 5 and fuses to the distal humeral condyle typically by age 16.
   ii. Attachment site of the common flexor tendon
b. Epidemiology
   i. Most commonly seen in males since few females participate in baseball
   ii. Most common between age 9 and 13
   iii. Affects the throwing arm
c. History
   i. Pain along medial side of elbow
   ii. Typically hurts only with throwing; usually batting and fielding are pain free
   iii. Rarely hurts with daily activity
   iv. May report swelling
   v. May have decreased control or velocity on throws
   vi. Most often hurts at the late cocking and early acceleration phase of throwing
   vii. Most frequent in pitchers and catchers but any position player can develop this condition
   viii. Occasionally may have an acute injury; typically hear a pop, significant pain and unable to continue to throw
d. Physical exam
   i. Tender over the medial epicondyle to palpation
   ii. May have soft tissue swelling
   iii. Pain with resisted wrist flexion
   iv. Pain to valgus stress
   v. May have flexion contracture of the elbow
e. Differential diagnosis
   i. Osteochondritis dissecans of capitellum
   ii. Panner's disease
   iii. Olecranon apophysitis
   iv. Flexor-pronator tendinopathy
   v. Valgus extension overload
   vi. Avulsion fracture of medial epicondyle
   vii. Ulnar collateral ligament tear/sprain
   viii. Ulnar neuritis/ulnar nerve subluxation
f. Use of imaging studies
   i. X-rays – typically ordered for this condition. AP and lateral of elbow is obtained; May consider AP of unaffected elbow to document the widening of the apophysis that occurs on the affected side. May see fragmentation of the apophysis that may be a normal variant. In acute injury, may see significant displacement of the apophysis from its normal position.
   ii. CT and MRI – not routinely needed for this condition
g. Treatment
i. Rest from throwing. May allow a player to bat or field provided any throwing is underhand. Neither the batting or fielding should cause pain to allow the athlete to continue.

ii. Icing may reduce pain and swelling.

iii. Gradual resumption of throwing, usually after 4-8 weeks of rest from overhand throwing.

iv. Consider physical therapy to focus on forearm and rotator cuff strengthening. May benefit from throwing analysis.

v. Consider a return to throwers program when cleared to resume throwing.

h. Return to play recommendations
   i. When pain free and after the 4-8 week period of rest, gradual resumption of throwing may begin.
   ii. Consider return to throwing programs to allow for gradual controlled resumption of throwing.

i. Complications
   i. Avulsion fracture of medial epicondyle – typically an acute injury, may require surgical management if fragment is displaced significantly. Consultation with orthopedic surgeon or pediatric sports medicine specialist recommended.

j. Outcomes and future concerns
   i. Most cases do very well, but recurrence is common if the athlete does not follow appropriate throwing standards.
   ii. Future concerns consist of continued stress on elbow, likely related to poor mechanics, that may produce an injury to the ulnar collateral ligament over time.

k. Prevention
   i. Reduce throwing; Following guidelines provided by Little League Baseball with recommended number of pitches and days of rest needed between throwing outings.
   ii. Offseason strengthening programs.
   iii. Avoidance of the throwing of breaking pitches (curveball and sliders) until nearing skeletal maturity.

7. Iselin's Apophysitis (Fifth Metatarsal Apophysitis)
a. Anatomy review
   i. Fifth metatarsal apophysis is located at the base of the fifth metatarsal (proximal aspect of the bone).
   ii. Site of attachment for the peroneus brevis tendon.
   iii. Oriented parallel to the fifth metatarsal.

b. Epidemiology
   i. Fifth metatarsal apophysis appears at around age 9 in females and 12 in males.
   ii. Typically will fuse to the shaft of the bone by age 12 in females and age 15 in males.

c. History
i. Pain with running, jumping, dancing activities at base of fifth metatarsal.

ii. Not typically an acute injury

iii. May become painful from an inversion ankle injury

iv. Also may hurt with cutting maneuvers in sports

v. May become painful enough to cause a limp

d. Physical exam

i. Tender to palpation at base of fifth metatarsal

ii. May have soft tissue swelling at the base

iii. Bruising not typical; If present, examiner should be more suspicious of an actual fracture

iv. May have pain to resisted eversion of the ankle

e. Differential diagnosis

i. Avulsion fracture base of fifth metatarsal

ii. Jones fracture

iii. Stress fracture of fifth metatarsal

iv. Peroneus brevis tendinitis

v. Ankle sprain

f. Use of imaging studies

i. X-ray – one of the more frequently misdiagnosed conditions of the foot. Frequently confused for an avulsion fracture. The apophysis runs parallel to the bone, most fractures at the base of the fifth metatarsal occur perpendicular to the bone. With no acute injury history, the examiner needs to reconsider the diagnosis of an avulsion fracture.

ii. CT, MRI and bone scan – not typically needed for this condition

g. Treatment

i. Relative rest

ii. Icing

iii. May consider orthotics, a post op shoe or walking boot if symptoms severe.

iv. Physical therapy may be of benefit

v. NSAIDs/acetaminophen for pain

h. Return to play recommendations

i. When able to walk and run without a limp, return to play may occur

i. Complications

i. None typically for this condition

j. Outcomes and future concerns

i. Typically this condition does quite well with no common long term problems

k. Prevention

i. Avoiding overuse

ii. Proper shoe wear
Shoulder Injuries

Shoulder Anatomy/Motions

1. Motions
   a. Ball-and-socket joint
      i. Abduction- 0 to 160-180 degrees
      ii. Forward Flexion- 0 to 160-180 degrees
      iii. Extension- 0 to 40-60
      iv. Internal Rotation- 0-90
      v. External Rotation- 0-90

2. Shoulder Anatomy-
   a. Bones
      i. Bones-
         1. Humerus
         2. Scapula
         3. Clavicle
         4. Thoracic Rib Cage
   ii. Joints-
      1. Sternoclavicular (SC)
      2. Acromioclavicular (AC)
      3. Glenohumeral (GH)
      4. Scapulothoracic (ST) pseudoarticulation
         a. Functional Anatomy- Golf ball (humeral head) on a golf tee (glenoid fossa)—
            i. Glenohumeral joint is considered a ‘ball-in-socket’ joint although the glenoid fossa is flat.
            This relationship allows for a large range of motion, but at the expense of stability.

b. Dynamic Stabilizers- Muscles
   i. Big Movers-
      1. Goal- Majority of the work and strength of shoulder movement
         a. Pectoralis Major
         b. Deltoid
         c. Biceps Brachii
         d. Latissimus Dorsi
         e. Triceps Brachii
         f. Trapezius
   ii. Small Movers- Rotator Cuffs-
      1. Goal: Compress the humeral head on the glenoid fossa (i.e. keep the golf ball on the tee) and internal/external rotation
         a. Supraspinatus
         b. Infraspinatus
         c. Teres Minor
         d. Subscapularis
iii. Other Scapular Stabilizers-
   1. Goal: Allow the scapula to provide a stable platform for the GH joint
      a. Serratus Anterior
      b. Levator Scapula
      c. Rhomboids
      d. Teres Major
   c. Static Stabilizers- Ligaments, Cartilage
      i. Glenohumeral Labrum- Fibrocartilaginous rim around the margin of the bony glenoid fossa.
         1. Increases the surface area of the GH joint (i.e. makes the tee larger) to create a deeper ‘socket’
      ii. Glenohumeral ligaments- Superior, Middle, Inferior
         1. Inferior GH Ligament is the most important and has three components- Anterior, inferior, anterior, posterior

**Physical Exam** -
1. Examine the cervical spine first
   a. Remember to examine the cervical neck first to make sure there is no neck pathology before the shoulder exam
2. Inspection of the Shoulder- Evaluate for atrophy, symmetry, discoloration, erythema, and ecchymosis
   a. Anterior- AC Joint symmetry, Clavicle
   b. Posterior- Resting Position of Scapula
      i. Dynamic- Evaluate for Scapular Dyskinesis with repeated abduction and/or forward flexion
   c. Lateral- Posture
3. Palpation- Landmarks-
   a. SC Joint
   b. Clavicle
   c. AC Joint
   d. Biceps Tendon
   e. Anterior Glenohumeral Joint Line
   f. Posterior Glenohumeral Joint Line
   g. Scapula
   h. Cervical Spine
4. ROM
   a. Abduction- 0 to 160-180 degrees
   b. Forward Flexion- 0 to 160-180 degrees
   c. Extension- 0 to 40-60
   d. Internal Rotation- 0-90
      i. Apley’s Scratch Test- (Tests Adduction and Internal Rotation)- Patient reaches behind the back to touch the inferior aspect of the opposite scapula
   e. External Rotation- 0-90
i. Superior Apley’s Scratch Test (Tests Abduction and External Rotation) - Patient reaches behind the head and touches the superior aspect of the opposite scapula

5. Provocative Tests - An attempt to isolate specific pathology
   a. There are various different ways of performing each of these provocative tests. This outline attempts to cover some of the more common maneuvers:
      i. Biceps Tendonitis
         1. Speed’s Test - Patient has arms 70-90 degrees of forward flexion with forearm supinated. A positive test is if there is pain or weakness with a resisted downward force from the examiner.
         2. Yergason’s Test - Patient’s elbow if flexed to 90 degrees with thumb up. Examiner grasps the wrist, and the patient attempts to actively supinate the arm and flex the elbow. A positive test causes pain.
      ii. Rotator Cuff Testing
         1. Supraspinatus test (AKA empty can test) - Patient forward flexes to 90 degrees and then moves about 30 degrees of abduction. Points thumb down toward the ground (full internal rotation). A positive test is if the application of downward force against resistance causes pain or weakness.
         2. Pain or weakness with resisted External Rotation at 0 degrees - Infraspinatus
         3. Pain or weakness with resisted External Rotation at 90 degrees abduction - Teres Minor (AKA Hornblower’s Test)
         4. Pain or weakness with resisted Internal Rotation at 0 degrees abduction - Subscapularis
      iii. Impingement
         1. Neer’s Impingement Test - Positive if passive forward flexion over 90 degrees causes pain
         2. Hawkins Impingement Test - Pain with passive internal rotation with the shoulder at 90 Degrees forward flexion
      iv. AC Joint
         1. Cross-Arm Test - Patient raises the arm to 90 degrees. Pain with active or passive adduction (forcing the acromion into the distal end of the clavicle) would be a positive test for AC pathology
      v. Glenohumeral Instability
         1. Sulcus Sign (inferior instability) - With the patient in neutral position, the examiner pulls downward on the elbow or wrist. A positive test occurs if there is a sulcus or depression lateral or inferior to the acromion
         2. Load and Shift Test - Similar to the Drawer test, except that the humeral head is gently pushed into the glenoid fossa (‘loaded’) before the test
3. **Apprehension Test (Anterior stability)**- With the patient supine or seated and the shoulder at 90 degrees of abduction. Examiner applies slight anterior pressure on the humerus and externally rotates the arm. Pain or apprehension about the feeling of impending subluxation indicates a positive test for anterior instability

vi. **Labral Pathology**
1. **O'Brien's Test**- With the patient at 90 degrees forward flexion, slight horizontal adduction, and thumb down, a positive test has pain or clicking with resisted downward force from the examiner. Repeat the same position except with palm up. The patient should have less pain with resisted downward force from the examiner if there is labral pathology.
2. **Clunk Test**- With the patient supine, stabilize the scapula and have the elbow at 90 degrees of flexion. The examiner brings the arm into full overhead abduction while rotating the humerus through varying degrees of internal and external rotation. A positive test is a clunk, clink, pop, or grind that may indicate a labral tear.

**Acute Injuries of the shoulder**

1. **Glenohumeral Instability (Dislocation and Subluxation)**
   a. **Anatomy**
   i. Glenohumeral joint- Head of the humerus interacting with the glenoid of the scapula like a golf ball on a golf tee. Labrum creates a ring around the glenoid to increase the surface area of this interaction
   ii. Muscles- Rotator Cuff muscles hold the head of the humerus on the glenoid. Glenohumeral ligaments add to stability.
   iii. Vast majority of acute dislocations involve anterior displacement of humeral head with respect to the glenoid
   b. **Definitions**-
   i. Dislocation- Complete separation of articular surfaces
   ii. Subluxation- abnormal translation of the humeral head without complete separation of articular surfaces. This will usually have spontaneous relocation.
   iii. Often referred to as TUBS
      1. T- Traumatic
      2. U- Unilateral dislocation
      3. B- Bankart lesion
      4. S- Surgery sometimes required
   c. **Epidemiology**
      i. High incidence of recurrence in adolescents
   d. **History**
i. Mechanism of injury
   1. Anterior force when shoulder is abucted and externally rotated (i.e. arm tackling in football) - Most common
   2. Fall on outstretched arm
   3. Blow to posterior shoulder

e. Physical Exam
   i. Intense pain
   ii. Anteriorly-inferiorly displaced head of the humerus usually looks like a gross deformity of the shoulder
   iii. Neurologic exam - often there is decreased sensation in the deltoid region (Shoulder patch)

f. Differential Diagnosis
   i. Fracture - Humerus or Clavicle
   ii. AC Separation
   iii. Rotator Cuff injury
   iv. Multidirectional Instability (Often associated with acute shoulder instability)
   v. Labral Injury (sometimes associated with Shoulder dislocation)

g. Imaging Studies
   i. Radiographs- AP and Axillary view
      1. Scapular Y is sometimes helpful
   ii. MRI- most helpful with intra-articular contrast
      1. Able to see secondary problems from the dislocation like Bankart lesion, Hills-Sachs defect, or Humeral avulsion of the Glenohumeral ligament (HAGL),

h. Treatment for dislocation
   i. Reduction - Reduce the dislocation as soon as possible. Muscles start to spasm as time passes so reduction becomes more difficult.
      1. Many different techniques for reduction
      2. Traction with counter traction is a common technique
      3. Sedation, muscle relaxant, or intra-articular lidocaine sometimes necessary if muscle spasms
      4. Repeat Neurologic exam after reduction
   ii. Immobilization/Rest Phase
      1. Sling and Swath immobilization (Possibly with slight abduction and external rotation) for several weeks
   iii. Recovery Phase- Physical Therapy
      1. Scapular stabilizers strengthening, Rotator Cuff strengthening
      2. Active/Passive ROM
      3. Sport specific activities
   iv. Surgical Interventions
      1. Recurrent dislocations
      2. Severe secondary defect/injury - Bankart lesion, Hills-Sachs Lesion, or HAGL (see below)

i. Return to Play Recommendations
i. Minimal requirements- Full ROM, Full strength, No pain with ADL’s
ii. Progressive return to activities is recommended
iii. Overhead activities and contact/collision participation are the final steps in a progressive return

j. Complications
i. Bony injury/Deformity-
   1. Bankart lesion- avulsion of the labrum (sometimes involves bone of the glenoid that is called a ‘Bony Bankart’) 
   2. Hill-Sachs Lesion- Chondral impact lesion on the humeral head
   3. HAGL- Humeral Avulsion of the Glenohumeral Ligament

ii. Fracture of the humeral head or proximal humerus
iii. Recurrence
   1. Recurrence more common with Multidirectional Instability, increased number of dislocations/subluxations, and younger age at first dislocation

iv. Axillary nerve injury

k. Outcomes/Future Concerns
i. Recurrence
ii. Maintenance physical therapy to keep shoulder strong

l. Prevention
i. Strengthening the rotator cuff muscles and scapular stabilizers
ii. Proper technique in sports- i.e. avoid arm-tackling
iii. Decreased participation in sports with high dislocation rates
iv. Some braces are available that decrease ROM, but have not been shown to completely prevent traumatic dislocation

2. AC Injury (AC Joint Sprain, AC Joint Separation = Shoulder Separation)
   a. Anatomy
   i. Joint between the clavicle and the acromion of the scapula
   ii. Ligaments
      1. Acromioclavicular (AC) ligament- Horizontal stability
      2. Coracoclavicular (CC) ligament- Vertical Stability
   iii. Muscles- Deltoid and trapezius

b. Definitions
   i. AC Joint Sprain- Stretching of the AC joint ligament
   ii. AC Joint Separation (AKA Shoulder Separation)- Partially or full tearing of the AC or CC ligaments
   iii. Grading System
      1. Grade 1- Simple Sprain of the AC joint with both ligaments still intact (Most common)
      2. Grade 2- Rupture of the AC ligament and sprain of the CC ligament
      3. Grade 3- Both AC and CC ligaments are disrupted. Distal clavicle is displaced above the acromion and shoulder
complex is displaced inferiorly. CC interspace is 25-100% greater than opposite shoulder

4. Grade 4- Complete AC dislocation and the clavicle is anatomically displaced posteriorly into the trapezius

5. Grade 5- Exaggerated Grade 3 with gross disparity between the clavicle and the acromion of 100%-300% displacement

6. Grade 6- Clavicle is displaced inferiorly to the acromion or coracoid process (extremely rare)

c. Epidemiology
   i. More males than females
   ii. Rare before 13 years of age
   iii. Most common in contact/collision sports (hockey or football)

d. History
   i. Mechanism- Direct blow to the AC joint on the shoulder
      1. Often while falling onto the shoulder directly (top or the side)
   ii. Pain or swelling over the AC Joint

e. Physical Exam
   i. Inspection- Abnormal contour of the shoulder compared to the contralateral side
   ii. Palpation- Pain over the AC joint or lateral clavicle
   iii. Provocative tests for stability
      1. Cross Arm Test (See Physical Exam Section above)
      2. AP stability- test the AC joint
      3. Vertical stability tests the CC joint

f. Differential Diagnosis
   i. Clavicle fracture
   ii. Dislocation/subluxation
   iii. SC joint injury
   iv. Rotator cuff injury
   v. Impingement

g. Imaging Studies
   i. Radiographs-
      1. AP- Comparing displacement of the acromion and the clavicle
      2. Zanca view- 10 degree cephalad view

h. Treatment
   i. Non-operative- For types I, II
      1. Rest, Ice, Sling immobilization for comfort
      2. Physical therapy- ROM, Strength
   ii. Grade III- Controversial treatment- surgical fixation vs non-operative care
   iii. Operative- Types IV, V, VI- ORIF or ligament reconstruction

i. Return to Play Recommendations
   i. Normal Range of motion, Normal strength, no pain with ADL’s
   ii. Progressive return to activities with a rough guideline of:
1. Grade 1 - 1-2 weeks
2. Grade 2 - 2-4 weeks
3. Grade 3 - 6-12 weeks

j. Complications
   i. AC joint arthritis
   ii. Chronic Subluxations/instability
   iii. Cosmetic- A visible bump is often seen after healing

k. Outcomes/Future Concerns
   i. Refer to orthopedic surgeon if significant displacement, continued recurrence, or not improving with conservative treatments

l. Prevention
   i. Appropriate sports equipment (i.e. shoulder pads in football/hockey/lacrosse)
   ii. Some bracing and taping techniques have been proposed but lack evidence

3. Clavicle Fracture-
   a. Anatomy
      i. Bone- Clavicle is S-shaped
         1. Group 1- Middle Shaft Fracture (Most common)
         2. Group 2- Lateral (lateral to the coracoclavicular ligament)
         3. Group 3- Medial (Medial to the sternocleidomastoid muscle)
            a. Medial epiphysis ossifies later than the rest of the clavicle and may not be completely fused until 22-25 years of age
      ii. Pathoanatomy-
         1. Often the SCM muscle pulls the medial fragment posterosuperiorly and the pectoralis pulls the lateral fragment inferomedially
         2. Clavicle fractures heal well due to excellent periosteal regenerative potential in pediatric patients
   b. Epidemiology
      i. One of the most frequently fractured bones in young, active patients
   c. History
      i. Mechanism-
         1. Direct blow to lateral aspect of the shoulder
         2. Fall on an outstretched arm
         3. Direct Trauma to the clavicle
      ii. Symptoms
         1. Shoulder pain after trauma
         2. Pain over the clavicle
   d. Physical Exam
      i. Inspection- Physical deformity of the clavicle
         1. Swelling or ecchymosis over the clavicle
2. Tenting of the skin above the clavicle
   ii. Palpation - Pain over the clavicle
      1. Crepitus
   iii. Neurovascular exam of the affected upper extremity (especially ulnar nerve)
   iv. Lung exam if having difficulty breathing

e. Differential Diagnosis
   i. AC or SC joint injury
   ii. Contusion
   iii. Brachial Plexus injury
   iv. Rib Fracture
   v. Rotator cuff injury
   vi. Glenohumeral Instability (Dislocation/Subluxation)
   vii. Physeal injury of the medial clavicular physis

f. Imaging Studies
   i. Radiographs - AP and serendipity view (40 Cephalic tilt)
   ii. CT scan - Evaluates displacement, shortening, comminution, articular extension, physeal involvement, and nonunion

g. Treatment
   i. Sling immobilization or Figure-of-eight brace
      1. Advantages and disadvantages of each of these devices
   ii. Progressive physical therapy starting with gentle ROM and then progressing to strength/functional activities
   iii. Surgery
      1. Surgical absolute indications: Open fracture or neurovascular/mediastinal structures at risk
      2. Surgical relative indications: displaced fractures with tenting, severely displaced fractures that are irreducible, 2 cm of shortening

h. Return to Play Recommendations
   i. Full ROM, normal shoulder strength, no tenderness to palpation, and evidence of bony healing (Clinical/Radiographic)
   ii. Usually about 6 weeks for ADLs and noncontact sports
   iii. Several months for contact/collision sports and you need to have evidence of bony healing

i. Complications
   i. Non-union (1-5%) - Higher risk if comminuted, displaced, extreme shortening

j. Prevention
   i. Re-fracture rate is high so be careful with return to contact/collision sports
   ii. Sometimes a donut pad or fiberglass shoulder shell may be used for extra protection

4. Proximal Humerus Fracture
   a. Anatomy
i. Proximal humeral physis- responsible for most of the growth of the humerus
   1. Closes at 14-18 years of age
ii. Salter Harris I and II are common (SH I younger age, SH II more common in teens) acute fractures
iii. Metaphyseal fracture common in grade school age
iv. Overuse Salter Harris I = Little Leaguer’s shoulder discussed in another section
v. Proximal humerus has excellent remodeling potential

b. Epidemiology
   i. Most common age is adolescents due to sport participation

c. History
   i. Mechanism- Direct trauma
   ii. Pain and weakness of the shoulder

d. Physical Exam
   i. Inspection- Ecchymosis of the proximal upper arm
   ii. Palpation- Pain in the proximal upper arm
   iii. Neurologic exam including axillary nerve distribution (Patch region of the lateral shoulder)

e. Differential Diagnosis
   i. Rotator cuff injury
   ii. Glenohumeral instability (Dislocation/Subluxation)

f. Imaging Studies
   i. Radiograph- Diagnostic for this injury

g. Treatment
   i. Rest, Ice
   ii. Immobilization with sling, sling and swathe, or shoulder immobilizer
   iii. ROM exercises in a couple weeks after pain is improved
   iv. Surgical intervention- Severely displaced, Open fractures, Vascular injury

h. Return to Play Recommendations
   i. Progressive return to play
   ii. Normal strength and ROM
   iii. Usually 6-12 weeks depending on the extent of the fracture and the bony healing

i. Complications
   i. Rarely with growth arrest
   ii. Nerve injury much less common in pediatric population

j. Outcomes/Future Concerns
   i. Malunion

k. Prevention
   i. Activity modification

Chronic/Overuse Injuries

1. Rotator Cuff Injury
a. Anatomy
   i. Muscles
      1. Supraspinatus- Initiation of abduction and humeral head stability during various movements
      2. Infraspinatus- External rotation
      3. Teres Minor- External rotation
      4. Subscapularis- Internal Rotation
      5. Rotator cuff muscles provide dynamic stabilization of the humeral head in the glenoid fossa.
   ii. Apophyseal insertions (location of possible apophyseal avulsion fractures)
      1. Supraspinatus, Infraspinatus, and teres minor insert on the greater tuberosity of the humerus
      2. Subscapularis inserts in the lesser tuberosity of the humerus

b. Definitions
   i. Tendinopathy- A broad term that describes any disease or dysfunction of a tendon
   ii. Tendonitis- Tendon injuries that involve larger-scale acute injuries often accompanied by inflammation
   iii. Tendonosis- Chronic overuse tendon injuries with damage to the tendon at a cellular level (Degeneration of the tendon’s collagen)
   iv. Rotator Cuff tear- Full, partial, or fraying of the rotator cuff’s musculotendinous unit
   v. Subacromial impingement- Rotator cuff muscles (Supraspinatus primarily) become irritated and inflamed due to friction against the undersurface of the acromion during overhead movements
      1. There are various other forms of impingement of the shoulder that are not as common in pediatrics

c. Epidemiology
   i. Fewer tears in younger patients (tears are rare in children)
   ii. Overuse as underlying cause in most cases
   iii. More common in overhead athletes

d. History
   i. Insidious onset of pain or weakness (especially with overhead activities)
   ii. Pain often over the anterolateral part of the shoulder
   iii. Repetitive overhead activities (Throwers, tennis/racket sports, water polo, swimmers...)

e. Physical Exam
   i. Inspection- Evaluate posture (forward positioned neck/head, anteriorly rotated shoulders) and protracted scapulae
      1. This might predispose to impingement
   ii. Rotator cuff strength testing
   iii. Impingement
      1. Neer Impingement sign- Positive if passive forward flexion over 90 degrees causes pain
2. Hawkins impingement test- Pain with passive internal rotation with the shoulder at 90 Degrees forward flexion

f. Differential Diagnosis
   i. Biceps tendonitis
   ii. Neck injury
   iii. Little League shoulder
   iv. Apophyseal avulsion fracture

g. Imaging Studies
   i. XR- Usually normal, but you can identify the anatomy of the acromion to assess for impingement risk
   ii. MRI- Diagnostic study for rotator cuff inflammation or tear
   iii. Ultrasound- dynamic testing available
      1. Similar diagnostic capabilities as MRI, but operator dependent

h. Treatment
   i. Rest/Activity modification/Ice
   ii. Physical therapy with rotator cuff strengthening and strengthening of the scapular stabilizers
   iii. Surgical options only for severe cases or cases that don’t improve with conservative treatments

i. Return to Play Recommendations
   i. Progressive return to activities with the overhead activities last

j. Complications
   i. Recurrence
   ii. Worsening injury/tears

k. Prevention
   i. Proper posture
   ii. Stretching and warming up properly
   iii. Continued scapular strengthening to prevent scapular dyskinesis
   iv. Pitch limits for pitchers
   v. Appropriate sport-specific techniques to utilize core muscles

2. Labral Injury- SLAP Tears- Superior Labrum from Anterior to Posterior tears
   a. Anatomy
      i. Glenoid labrum- fibrocartilagenous tissue that creates a ring around the bony glenoid
      ii. Biceps tendon anchors into the superior labrum (usually at 12 o’clock)
   b. Epidemiology
      i. Most commonly in throwers or other overhead athletes
   c. History
      i. Symptoms could start acutely or chronically
      ii. Deep shoulder pain especially with overhead activities or cross body activities
      iii. Mechanical symptoms (popping, clicking, catching)
      iv. Decreased athletics performance
   d. Physical Exam- Several provocative maneuvers are described in the literature
      i. O’Brien’s test- See above Section on Physical Exam
ii. Clunk test- See above Section on Physical Exam
iii. Speed’s test for biceps tendonitis is often positive
iv. Apprehension test positive as a sign for glenohumeral instability
v. Underlying biomechanics- Glenohumeral Internal Rotation Deficit
   1. Weak scapular stabilizers or scapular dyskinesis
e. Differential Diagnosis
   i. Rotator cuff tendonitis
   ii. Impingement
   iii. Subluxation/dislocation
f. Imaging Studies
   i. Radiographs- Usually normal
   ii. MRI with intra-articular contrast is good way to evaluate for a SLAP tear
g. Treatment
   i. Physical therapy
      1. Underlying Biomechanics- GIRD, scapular stabilizer strengthening, rotator cuff strengthening
   ii. Activity modification- Stop the offending action (throwing)
   iii. Surgical repair- Arthroscopic debridement and stabilization of the labrum and biceps tendon
h. Return to Play Recommendations
   i. If conservative treatment is effective a progressive return to play and a progressive throwing program
      1. Progressive throwing program- Start throwing a short distance and low velocity with a progressive increase in velocity and distance.
   ii. If surgical treatment needed
      1. Immobilization for several weeks
      2. Physical therapy for several months
      3. Progressive return to activities and a progressive throwing program
         a. Total time post-surgery is usually 4-6 months depending on sport and severity
i. Complications
   i. Treatment failure and re-injury
j. Prevention
   i. Stretching exercises to improve the posterior capsular tightness (GIRD)
   ii. Improving strength of scapular stabilizers
   iii. Adhering to pitch counts
   iv. Proper sport-specific mechanics

3. Little Leaguer’s Shoulder
   a. Anatomy
      i. Proximal humeral physis- responsible for most of the growth of the humerus
         1. Closes at 14-18 years of age
   b. Definition
Little Leaguer's Shoulder - Stress injury of the growth plate of the proximal humerus

1. Usually described as a Salter-Harris I fracture

c. Epidemiology
   i. Most common with adolescent throwers- Baseball (pitchers/catchers)
   ii. Less commonly- Athletes with overhead activities- racquet sports, volleyball, swimming, gymnastics
   iii. Age 9-16

d. History
   i. History of pitching/throwing or other repetitive overhead activity
   ii. Usually a gradual pain onset
   iii. Pain over the lateral shoulder, especially with overhead activities
   iv. Pain improves with rest

e. Physical Exam
   i. Point tenderness over the lateral proximal humerus
   ii. Pain with shoulder rotation

f. Differential Diagnosis
   i. Rotator Cuff injury
   ii. Multi-directional Instability
   iii. Subluxation/Dislocation
   iv. Proximal Humerus Fracture
   v. Labral injury

g. Imaging Studies
   i. AP X-ray with widening of the proximal humeral physis
      1. Sometimes bilateral view is helpful
   ii. MRI- Usually not necessary, but more helpful to rule out other pathology (labral injury)

h. Treatment
   i. Relative Rest usually for minimum of 2-3 months
   ii. Physical Therapy- Strengthening rotator cuff muscles, strengthening of scapular stabilizers, improving Glenohumeral internal rotation deficit if possible
      1. Core strengthening and stretching
   iii. Progressive throwing program- Start throwing a short distance and low velocity with a progressive increase in velocity and distance.

i. Return to Play Recommendations
   i. Progressive throwing program after rest and physical therapy
   ii. Start in the field before pitching
   iii. Re-introduction of pitching is the final step

j. Complications
   i. Premature growth arrest- Rare

k. Prevention
   i. Strengthening rotator cuff muscles, good core strength, posterior capsule stretches
   ii. Correct throwing mechanics
   iii. Pitch count enforcement
iv. Adequate rest between pitching/throwing outing

4. Multidirectional Instability
   a. General- Multidirectional Instability- generalized laxity (anterior, posterior, inferior) of the glenohumeral joint that is due to increased mobility and joint weakness
      i. Often caused by generalized ligamentous laxity (genetic), overuse that causes pain (especially in overhead athletes), and shoulder muscle weakness
      ii. Sometimes referred to as AMBRII
          1. A- Atraumatic
          2. M- Multidirectional
          3. B- Bilateral
          4. R- Rehabilitative initial management
          5. I- Inferior capsular shift as a surgical repair
   b. Anatomy of the glenohumeral joint
      i. Glenohumeral joint- Head of the humerus interacting with the glenoid of the scapula like a golf ball on a golf tee. Labrum creates a ring around the glenoid to increase the surface area of this interaction
      ii. Muscles- Rotator Cuff muscles hold the head of the humerus on the glenoid. Glenohumeral ligaments add to stability.
   c. History
      i. Generalized pain
      ii. Instability
   d. Physical Exam
      i. Sulcus Sign- See the Physical Exam Section above
      ii. Apprehension Test- See the Physical Exam Section above
      iii. Drawer test or Load And Shift test- See the Physical Exam Section above
      iv. Beighton Score- To evaluate for generalized signs of hypermobility
         1. Palms on the ground on forward bend (1 point)
         2. Hyperextension of the elbows past 10 degrees (1 point each for each elbow)
         3. Genu Valgum (1 point each)
         4. Ability to passively touch the thumb to the flexor aspect of the forearm with the wrist flexed (1 point each)
         5. Passive Extension of the fifth finger past 90 degrees (1 point each)
   e. Differential Diagnosis
      i. Neck injury
      ii. Rotator cuff tendonitis
      iii. Unidirectional instability
      iv. Acute Dislocation/subluxation
   f. Imaging Studies
      i. X-rays- Often are normal
      ii. MRI- Often are normal, but can rule out other pathologies (Rotator cuff injury or SLAP tears)
   g. Treatment
i. Rest, Ice, Activity modifications

ii. Physical therapy
   1. Strengthening of the dynamic stabilizers
   2. Closed kinetic chain exercises
   3. 3-6 months minimum

iii. Operative stabilization procedures
   1. Only if failure of extensive non-operative management

h. Return to Play Recommendations
   i. Progressive return to activities as tolerated by pain

i. Complications
   i. Dislocation or subluxation is often associated with MDI
   ii. Axillary nerve injury
   iii. Bankart/Hills-Sachs/HAGL if dislocation (see Glenohumeral Instability Section)

j. Outcomes/Future Concerns
   i. Maintenance PT is often needed and surgical interventions are limited

k. Prevention
   i. Strengthening of the dynamic stabilizers
   ii. Activity modifications (especially decreasing overhead activities)

5. Distal Clavicle Osteolysis (Weight Lifter Shoulder)
   a. Definition
      i. Distal Clavicle Osteolysis- osteolysis of the distal clavicle due to repetitive microtrauma
   b. Epidemiology
      i. Common in weight lifters
   c. History
      i. Pain in the distal clavicle or around the AC joint
   d. Physical Exam
      i. Tenderness to palpation of the distal clavicle
      ii. Cross chest activities cause pain
   e. Differential Diagnosis
      i. AC joint injury
      ii. Clavicle fracture
   f. Imaging Studies
      i. Radiograph- Osteolysis of the distal clavicle
   g. Treatment
      i. Rest, Ice
      ii. Activity modification- Stop weight lifting or modify technique
   h. Return to Play Recommendations
      i. Progressive return to activities
   i. Complications
      i. Cyst formation of the distal clavicle
   j. Prevention
      i. Modify weight lifting technique or avoiding lifts that cause pain
BACK PAIN

1) Overview of the pediatric spine, highlighting development and the differences between the adult and pediatric spine with review of anatomy. The primary focus is the lumbar spine.

2) Anatomy
   a) The spine is a mechanical structure composed of bones, joints, ligaments and muscles surrounding and distributing neural elements.
   b) The spinal column is composed of 33 vertebrae divided into 5 sections: cervical (7), thoracic (12), lumbar (5), sacral (5) and coccygeal (4). [Netter Plates 142-147]
   c) Bones and Bony Prominences
      i) Vertebral body: Large, strong, anterior weight bearing structure
      ii) Posterior vertebral arch: Semi-circular-shaped structure surrounding the central canal
      iii) Vertebral foramen: Area through which the roots pass
      iv) Pedicles: Project dorsally off the vertebral body on each side
      v) Lamina: Bony structure that connect the pedicles
      vi) Transverse process: Bony projections on either side of the pedicles/lamina
      vii) Spinous process: Bony projection from the lamina
      viii) Facet joints: Articulation of the superior and inferior lamina
      ix) Pars interarticularis: Part of vertebra located between the inferior and superior articular processes of the facet joint; susceptible to injury and stress fracture (spondylolysis/spondylolysthesis)
      x) Sacrum: Coalesced inferior spinal segments that articulate with the pelvis
   d) Ligaments
      i) Disc: Major ligament connecting each vertebral body. Comprised of two types of tissue:
         (1) Nucleus Pulposus: Gelatinous structures located in the annulus
         (2) Annulus Fibrosis: Outer layer of fibers that functions to hold the nucleus pulposus and restrains the vertebral bodies
      ii) Anterior longitudinal ligament: Fibrous tissue that runs the length of the spine along the vertebral bodies
      iii) Posterior longitudinal ligament: Runs along the posterior surface of the vertebral bodies; weaker than the anterior longitudinal ligament
      iv) Ligamentous flavum: Very strong ligament attaching the lamina above to the lamina below
      v) Facet capsule: Connects each superior articulating process with its corresponding inferior articulating process
      vi) Interspinous ligament: Connects each spinous process to the one below
e) Muscles and Fascia
   i) Thoracolumbar fascia: Tissue that separates the muscle compartments and fuses the aponeuroses of several muscles
   ii) Anterior Muscle Group: Psoas, intertransversalis, quadratus and levator costae
   iii) Posterior Muscle Group: Erector spinae (semispinalis, longissimus, iliocostalis), multifidus, rotaires, interspinalis
   iv) Accessory Muscle Group: Abdominal muscles, latissimus dorsi, rhomboids, gluteus maximus

f) Neural Elements
   i) Spinal cord
   ii) Conus medullaris
   iii) Cauda equine
   iv) Nerve roots: Exit the spine at each level on both sides of the canal via the vertebral foramina

3) Biomechanics
   a) Functions of the spine:
      i) Support head, abdominal contents and pelvic girdle
      ii) Point of attachment for muscles and ribs
      iii) Protection of the spinal cord and neural elements within while allowing movement
      iv) During periods of growth, young athletes are prone to loss of flexibility and muscle imbalances.
   b) How movement affects the structures of the spine:
      i) Flexion
         (1) Increases the size of the intervertebral canal and foramina
         (2) Increases dural sac and nerve root tension
         (3) Causes annulus to bulge anteriorly
      ii) Extension
         (1) Decreases the size of the intervertebral canal and foramina
         (2) Decreases dural sac and nerve root tension
         (3) Causes annulus to bulge posteriorly
         (4) Applies more pressure on the pars interarticularis
      iii) Forward flexion, axial loading and upright posture increase intradiscal pressure
           → Pressure is greatest in sitting, less in standing, least in lying supine
      iv) Rotation and torsion: Produce annular tears and disc herniations

4) Patterns of back pain in young athletes are significantly different than in adults.
   a) The rate of mechanical low back pain in children approaches the rate in adults however the etiologies are quite different.
   b) Pars interarticularis injuries are more common in young athletes (Responsible for back pain in up to 47% of young athletes)
c) Disc-related problems are uncommon in young athletes (Disc-related problems are the cause of back pain in 11% of children versus 48% of adults).

d) The pediatric spine is more flexible than the adult spine and this accounts for the greater susceptibility to traction injuries than the adult spine. Examples of this flexibility include:
   i) The facet joints in a child’s spine are more horizontal.
   ii) The soft tissues, particularly those in the neck, are more elastic than those in adults.

5) History
   a) What is the exact location of the pain?
      i) If pain is reported as radiating to the legs, neurological involvement is more likely.
   b) When does it hurt?
      i) Pain that occurs with motion or activity suggests overuse and/or a mechanical cause.
      ii) Night pain or pain at rest signals more ominous etiologies of pain.
   c) What makes the pain worse?
      i) Pain with extension suggests spondylolysis/spondylolisthesis.
   d) What makes the pain better?
   e) When and how did your symptoms begin?
      i) Gradual onset is associated with low-grade stresses that occur after long periods of training suggest a stress reaction.
      ii) Sudden-onset during anaerobic training or lifting single weights may indicate a more acute injury.
   f) How do you train for your sport?
      i) What activities are you doing to train?
      ii) Are there any new activities you have incorporated into your regimen?

6) Physical exam
   a) Inspection
      i) Posture and Stance:
         (1) Leaning back or tripod position when seated help to unload the spine
         (2) Listing to one side while standing suggests nerve root compression
         (3) Appearance of a flat back with vertical sacrum may be seen with spondylolisthesis
         (4) Uneven shoulder height, rib hump on forward bend and asymmetric flank creases may indicate scoliosis
      ii) Gait:
         (1) A forward flexed posture can be seen with stenosis
         (2) Antalgic, slow gait occurs with severe back pain
   b) Palpation
c) Range of Motion
   i) Flexion: 40-60°
   ii) Extension: 20-35°
   iii) Lateral flexion: 20° to each side
   iv) Rotation: 90° to each side

d) Strength Testing

e) Neurological Function
   i) Upper motor neuron (spinal cord pathology) findings: spasticity, weakness, numbness, hyper-reflexia, clonus
   ii) Lower motor neuron (cauda equine or nerve root injury) findings: flaccid muscles, weakness, numbness, hypo-reflexia

f) Provocative Maneuvers
   i) Straight leg raise (SLR): Tension test with hip flexion of the extended leg that indicates sciatic nerve irritation when radicular pain occurs at <60-70 degrees of hip flexion/leg elevation. May be performed with patient seated or supine.
   ii) Lasegue’s Test (SLR with foot dorsiflexion): Tension test with hip flexion of the extended leg that indicates sciatic nerve irritation when radicular pain occurs with foot dorsiflexion.
   iii) Crossed Straight Leg Raise: Performed by lifting the unaffected leg while patient is in a seated position; production of pain on the affected leg may be pathognomonic for a herniated disc.
   iv) Single Leg Hyperextension Test: Localized pain to the lumbar spine, suggests possible pars interarticularis stress fracture (spondylolysis).
   v) FABER (Flexion, ABduction, External Rotation): Suggests hip joint or sacroiliac dysfunction.

7) Waddell’s signs
   a) Waddell, et al. (1980) described five categories of signs:
      i) Superficial non-anatomic tenderness
      ii) Overreaction
      iii) Pain on simulated maneuvers
         (1) Pain on axial loading of skull
         (2) Pain on passive rotation of shoulders and pelvis
      iv) Straight Leg Raise testing discrepancy
         (1) Sitting exam and supine exam are not consistent
         (2) Sitting test performed while distracting patient
      v) Non-physiologic examination
         (1) Non-dermatomal sensory loss
         (2) Cogwheel or give-way weakness
b) Any individual sign marks its category as positive. When three or more categories were positive, the finding was considered clinically significant. Positive test implies psychogenic back pain.

8) “Red Flags” of Pediatric Back Pain

a) Night pain
b) Pain that interferes with activity, especially in young children
c) Frequent pain at rest (However pain with prolonged sitting is extremely common in teens with mechanical back pain)
d) Back pain associated with an abnormal gait, limp, and unsteadiness.
e) Any associated constitutional symptoms: Fever, lethargy, weight loss, loss of appetite
f) Important associated cutaneous findings: High and deep dimple, hairy patch, café-au-lait spots suggestive of neurofibromatosis
g) Positive “finger test.” If a child localizes the pain to one spot it is more worrisome than if the pain is diffuse.

9) Muscular low back pain

a) Overview – Strain of the muscle or tendon unit
b) Epidemiology
c) Muscle tendon strains account for about 6% of adolescent back pain, compared to 27% of adult back pain.
d) History
   i) Acute-onset pain after bending, rotation and/or improper lifting
   ii) Poorly localized diffuse pain
   iii) No neurologic symptoms
e) “Red Flags” that indicate more significant pathology:
   i) Pain that awakens patient from sleep
   ii) Pain radiating to the buttock or legs
   iii) Bowel/bladder incontinence
   iv) Distal tingling or numbness
   v) Increased pain with extension
f) Common findings on physical exam
   i) Localized tenderness to palpation with or without palpable muscle spasm
   ii) Decreased range of motion
   iii) Normal neurological evaluation
g) “Red Flags” that indicate more significant pathology:
   i) Decreased sensation
   ii) Decreased reflexes
   iii) Weakness
   iv) Positive straight leg raise
   v) Positive single-leg hyperextension
h) Differential diagnosis
   i) Spondylolysis/spondylolysthesis
   ii) Muscle strain
   iii) Disc herniation
   iv) Infection (discitis or osteomyelitis)
   v) Inflammation (seronegative spondyloarthropathies)
   vi) Tumors (osteoid osteoma, osteoblastoma, bone cysts, Ewing sarcoma, osteogenic sarcoma)
   vii) Sacroiliitis
i) Use of imaging studies
   i) Xrays are not required by history or physical exam if no “red flags” are present.
      If performed, xrays may show decreased normal lordosis due to muscle spasm or may appear completely normal.

j) Treatment: Conservative care
   i) Analgesia
      (1) Tylenol or NSAIDs
      (2) Short-term muscle relaxants for older adolescents
   ii) Relative rest
      (1) Pain as the limiting factor for activity
   iii) Massage
      (1) May improve pain levels to enable participation in exercise therapy programs
   iv) Physical therapy
      (1) Exercise therapy programs to incorporate stretching and strengthening
      (2) Return to play
         (a) Cleared to gradually return to activity when pain-free with full range of motion and full strength and no neurological symptoms.

k) Complications
   i) If back pain is ignored for a prolonged period, additional injuries often occur as athletes attempt to compensate for the initial injury

j) Outcomes and future risks
   a. Natural history of mechanical back pain, regardless of treatment
      i. 33% resolves within 1 week
      ii. 70% resolves by 3 weeks
      iii. 90-95% resolves in 3 months

k) Prevention
   i. Good back hygiene with proper sports and lifting technique
   ii. Maintaining a back and core stability training program

10) Spondylolysis and Spondylololithesis
   a) Overview
b. Injury to the posterior elements of the spine (pars interarticularis).
c. Increased risk: Repetitive extension and rotation of the lumbar spine, such as dance, figure skating, and gymnastics.
d. Spondylolysis:
   i. Defect in the pars interarticularis, a stress fracture caused by repetitive extension and torsion of the spine
e. Spondylolisthesis:
   i. Forward translation of 1 vertebra on the next caudal segment results when bilateral spondylolysis occurs at the same vertebral level
   ii. Graded according to the percentage of slip:
      1. Grade I: 0-25% slip
      2. Grade II: 25-50% slip
      3. Grade III: 50-75% slip
      4. Grade IV: >75% slip

b) Epidemiology
   i) A large percentage (47% has been reported) of young athletes with back pain will have spondylolysis
   ii) L5: Most common level of spondylolysis
   iii) Occurs most often on the left.
c) History
   i) Insidious onset of extension-related low back pain
   ii) Increased pain with the landing component of running and jumping
   iii) Occasionally, radiating pain, numbness or weakness may be present, however these symptoms may also represent disc herniation
   iv) Associated with hamstring tightness
d) Common findings on physical exam
   i) Hyperlordosis
   ii) Ipsilateral muscle spasm
   iii) Hamstring tightness
   iv) Pain elicited with hyperextension
   v) Single-legged hyperextension testing localizes the pain to the spine when standing on the ipsilateral leg
   vi) Focal tenderness to palpation
e) Differential diagnosis
   i) Spondylolysis/spondylolisthesis
   ii) Muscle strain
   iii) Disc herniation
   iv) Infection (discitis or osteomyelitis)
   v) Inflammation (seronegative spondyloarthropathies)
vi) Tumors (osteoid osteoma, osteoblastoma, bone cysts, Ewing sarcoma, osteogenic sarcoma)

vii) Sacroiliitis

f) Use of imaging studies

i) Plain radiographs
   (1) AP: Identify anatomic variants or developmental defects such as transitional vertebrae or spina bifida occulta.
   (2) Lateral: May demonstrate spondylolisthesis or a lytic lesion
   (3) Oblique views: May demonstrate a stress fracture, the pathognomonic “neck of the Scotty dog” lesion. However, because only 1/3 of stress fractures can be seen on plain radiographs, oblique views are often not worth the extra radiation exposure.

ii) SPECT (Single-photon emission computed tomography)
   (1) Increased uptake on bone scan indicates active bony turnover that occurs with stress reaction/fracture.

iii) CT
   (1) Used to confirm the presence of a pars interarticularis fracture and to monitor the healing process.
   (2) Many physicians reserve CT for patients not responding to treatment because of the additional radiation exposure of CT.

iv) MRI
   (1) Historically, MRI was not as sensitive as SPECT bone scan. However, when using STIR sequence and thin cuts through the lower lumbar spine, MRI is now approaching sensitivity of SPECT/CT without the ionizing radiation exposure.

g) Treatment

i) Activity modification:
   (1) Avoid all high impact activity until pain resolved.
   (2) Avoid all aggravating activities.
   (3) Avoid extension of the spine.

ii) Home and formal physical therapy program to improve abdominal muscle and core strength – including back extensors, hip flexor and hamstring stretches, and anti-lordotic exercises for the lumbar spine.

iii) Anti-lordotic Bracing: Controversial—Medical literature is mixed regarding the effect of bracing on resolution of symptoms and fracture healing.

iv) Surgical stabilization is indicated if spondylolisthesis progresses beyond 50%, or if neurological symptoms or persistent pain.

h) Return to play

i) Once the athlete is pain-free at rest, gradual return to activity can begin.
ii) If bracing is used, bracing continues during activities, until the athlete has resumed full activity without pain; then the brace is gradually weaned until pain-free without bracing and participating in full activities.

i) Complications
   i) Untreated unilateral spondylolysis can progress to bilateral spondylolysis and ultimately spondylolisthesis.
      (a) j. Outcomes and future risks
      ii) Athletes are at low risk for worsening of spondylolisthesis.
      iii) Spondylolisthesis should be followed every 4 to 6 months with standing lateral films until skeletal maturity to assess for the progression of slip.

j) Prevention
   i) Avoid overuse.
   ii) Do not push through the pain; pain is the limiting factor for all activity.
   iii) During periods of growth, address young athletes’ loss of flexibility and muscle imbalances.
   iv) Observe proper technique

11) 5. Disc Pathology

a) Overview-- There are 2 discogenic syndromes that can occur in the young athlete:
   i) Annular tear
      (1) Tear of the annulus fibrosis
      (2) Nucleus material may or may not be extruded or displaced
      (3) Because tear occurs on periphery, it is more likely to be associated with radicular pain or chemical neuritis
   ii) Herniated nucleus pulposis
      (1) The nucleus pulposis extrudes partially or completely through the annulus fibrosus, protruding into the neural elements or extruding into the canal
      (2) Herniation usually occurs to one side or the other of the posterior longitudinal ligament
      (3) Most common in the 3rd and 4th decades

b) Epidemiology
   i) Acute herniation of the nucleus pulposis is uncommon in young athletes.

c) History
   i) Flexion-related back pain
   ii) Radicular symptoms may occur, but often are not present in the young athlete
   iii) Associated with back muscle spasm, hamstring tightness and buttock pain
   iv) Increased pain with prolonged flexion, sitting or Valsalva maneuvers

d) Common findings on physical exam
   i) Decreased lumbar motion
   ii) Positive straight leg raise
   iii) Decreased reflexes and strength on the affected side
iv) Presentation can be variable and may include only one or two positive findings.

e) Differential diagnosis
   i) Spondylolysis/spondylolisthesis
   ii) Muscle strain
   iii) Disc herniation
   iv) Infection (discitis or osteomyelitis)
   v) Inflammation (seronegative spondyloarthropathies)
   vi) Tumors (osteoid osteoma, osteoblastoma, bone cysts, Ewing sarcoma, osteogenic sarcoma)
   vii) Sacroiliitis

f) Use of imaging studies
   i) Lumbar radiographs: Rule out osseous injury
   ii) MRI:
      (1) Indicate the extent of the herniation and show nerve root impingement
      (2) Obtain if refractory or progressive symptoms

g) Treatment
   i) Conservative management
      (1) Relative rest with pain as the limiting factor for participation
      (2) Home and/or Formal Physical therapy program to improve abdominal and core muscle strength
      (3) NSAIDs as needed for pain
      (4) Consider oral or epidural steroids for refractory cases
   ii) Surgical intervention indicated if:
      (1) Cauda equina syndrome
      (2) Progressive neurological deficit
      (3) Pain refractory to conservative management

h) Return to play
   i) Athletes with disc herniation may gradually return to full activity when they have achieved pain-free full range of motion, full strength and have progressed through sport-specific activities in a controlled setting.

i) Complications
   i) Cauda equina syndrome:
      (1) Caused by compression of nerves in the lower portion of the spinal canal
      (2) Can result in loss of bowel/bladder function and paralysis of the legs
      (3) Considered a surgical emergency because deficits may be permanent

j) Outcomes and future risks
   i) Degenerative disk changes are two-times more commonly seen in MRI studies done on gymnasts as compared with non-athletes.
ii) The exact relationship between pain and degeneration is not known, however lifetime incidence and prevalence of low back pain is higher in retired wrestlers than in the general population.

I) Prevention
   a. Fully rehabilitate previous injuries, muscle weakness and poor flexibility to avoid future injury.
Hip Injuries

Overview of the hip and anatomy

1. Motions
   a. Ball-and-socket joint
      i. Abduction (45-50 degrees)
         1. Gluteus medius
         2. Gluteus minimus
      ii. Adduction (20-30 degrees)
         1. Adductor longus
         2. Adductor brevis
         3. Adductor magnus
         4. Pectineus
         5. Gracilis
      iii. Flexion (120 degrees)
         1. Iliopsoas
         2. Rectus femoris
         3. Sartorius
      iv. Extension (30 degrees)
         1. Gluteus maximus
         2. Hamstrings
      v. Internal Rotation (35 degrees)
         1. Adductor longus
         2. Adductor brevis
         3. Adductor magnus
      vi. External Rotation (45 degrees)
         1. Gluteus maximus
         2. Piriformis
         3. Obturator externus
         4. Obturator internus
         5. Superior gemellus
         6. Inferior gemellus

2. Hip and Pelvis Anatomy
   a. Bones and Bony Prominences
      i. Femur
      ii. Pelvic acetabulum
      iii. Greater trochanter
      iv. Lesser trochanter
      v. Ischium
      vi. Ilium
      vii. Pubis
   b. Muscles
      i. Superficial
         1. Tensor fasciae latae
         2. Sartorius
3. Gluteus maximus
   ii. Deep
      1. Anterior
         a. Rectus femoris
         b. Iliopsoas
      2. Medial
         a. Adductor longus
         b. Adductor magnus
         c. Adductor brevis
         d. Pectineus
         e. Gracilis
      3. Lateral
         a. Gluteus medius
         b. Gluteus minimus
      4. Posterior
         a. Piriformis
         b. Obturator internus
         c. Superior gemellus
         d. Inferior gemellus
         e. Obturator externus
         f. Quadratus femoris
         g. Semimembranosus
         h. Semitendinosus
         i. Biceps femoris
   c. Ligaments (bone to bone attachment)
      i. Iliofemoral ligament (anterior)
      ii. Ischiofemoral ligament (posterior)
      iii. Pubofemoral ligament (inferior)
      iv. Inguinal ligament
   d. Other Structures
      i. Iliotibial band
      ii. Greater trochanter bursa
      iii. Gluteus Medius Bursa
      iv. Acetabular labrum
3. Important to assess both the knee and hip in children and adolescents presenting with knee pain as hip pain can be referred to the knee.
   a. Hip injuries that may present as knee pain
      i. Slipped Capital Femoral Epiphysis (SCFE)
      ii. Legg Calve Perthes
      iii. Stress Fracture
      iv. Septic Hip
      v. Synovitis

**Physical Exam**
1. Evaluate both lower extremities, using non-injured side as a comparison
   a. Visualize joint below during exam
b. Loose fitting shorts  
c. Socks and shoes off  

2. Gait Evaluation  
   a. Evaluate weight-bearing status, limp, range of motion  
      i. Inability to bear weight is red flag  
      ii. Inspection  
   b. Evaluate foot type and motion  
      i. Pes planus / pes cavus  
      ii. Hyperpronation  
      iii. Hypersupination  

3. Inspection  
   a. Evaluate for pelvic asymmetry, bruising, redness, swelling, muscle atrophy  

4. Hip range of motion  
   a. If a joint lacks full active range of motion, the examiner should passively move the joint to see if full motion can be obtained.  
   b. 0 to 120 degrees of flexion  
   c. 0 to 45 degrees of internal and external rotation  
      i. Important to evaluate in skeletally immature patients  
      ii. Limited motion, especially reduced and/or painful internal rotation may indicate intra-articular pathology  

5. Palpation  
   a. Apophyses  
      i. Iliac crest, anterior superior iliac spine, anterior inferior iliac spine, ischial tuberosity, pubic symphysis, greater trochanter, lesser trochanter  
         1. Acute onset pain, tenderness, may indicate avulsion  
         2. Gradual onset pain, tenderness, may indicate apophysitis  
      ii. Anterior hip joint, gluteal muscles, lower back  

6. Special tests  
   a. Single leg hop  
      i. Pain on single leg hop concerning for stress fracture  
   b. Trendelenburg test  
      i. Athlete stands on one leg. If the pelvis of the non-weight bearing leg sags, indicates weakness of gluteus medius on weight-bearing leg  
   c. Ober’s test  
      i. Patient lies on side with affected hip up. The leg is grasped, knee flexed and hip extended and abducted. When released, the knee should fall back to neutral. Failure to return to neutral indicates tight IT band.  
   d. Modified Thomas test  
      i. With patient supine and buttocks on table with legs hanging off, one knee is pulled to the chest. The contralateral knee and hip should remain extended; if the knee and hip flex, this indicates tight hip flexors  
   e. Impingement test  
      i. Hip flexion to 90 degrees. Pain with adduction and internal rotation may indicate femoral acetabular impingement
**Hip Injuries**

1. Hip Pointer
   a. Overview
      i. Contusion over iliac crest or greater trochanter
   b. Epidemiology
      i. Common in adolescent athletes involved in contact/collision sports
   c. History
      i. Direct blow to lateral hip, i.e. tackle
   d. Physical Exam
      i. Hematoma or ecchymosis may be present over iliac crest or lateral hip
      ii. Tender to palpation
      iii. Pain with bending or rotation of the trunk
   e. Differential Diagnosis
      i. Fracture if significant trauma and/or inability to weight bear
   f. Imaging
      i. Not usually indicated
   g. Treatment and Return to Play
      i. RICE
      ii. Crutches for support
      iii. Physical therapy active ROM, strengthening
      iv. RTP 2-8 weeks
   h. Complications
      i. Myositis ossificans
      ii. Lateral femoral cutaneous nerve palsy
      iii. Bursitis
      iv. Compartment Syndrome rare
   i. Prevention
      i. Adequate padding

2. Slipped Capital Femoral Epiphysis (SCFE)
   a. Overview
      i. Displacement of upper femoral epiphysis on the metaphysis
      ii. Caused by increased stress across weakened physis
      iii. May present as knee pain - always examine hip in pediatric or adolescent with knee pain
   b. Epidemiology
      i. Overall incidence - 11:100,000 children
      ii. Males more common than females
      iii. Peak age corresponds to period of maximal skeletal growth
      iv. Peak age males age 13
      v. Peak age females 11
   vi. Risk factors
      1. Obese or excessively tall and thin
      2. Endocrine disorders
      3. History of radiation therapy
      4. Renal failure
   c. History
i. Knee, thigh, or groin pain with weight bearing
ii. Insidious or acute onset
d. Physical exam
   i. +/- ability to weight bear
   ii. Inability to weight bear in acute slip
   iii. May walk with leg externally rotated
   iv. Hip falls into external rotation on passive hip flexion
   v. Passive range of motion decreased internal rotation
e. Differential diagnosis
   i. Knee injury
   ii. Femoral neck fracture
   iii. Septic arthritis
   iv. Osteomyelitis
   v. Legg-Calve-Perthes
   vi. Juvenile Rheumatoid Arthritis
   vii. Transient Synovitis
   viii. Tumor
f. Imaging
   i. Pelvic radiographs: AP pelvis and frog-leg lateral
      1. “ice cream slipping off of the cone”
      2. Klein's line on AP film-line drawn along superior border of femoral neck; normal line should transect proximal epiphysis
      3. Pre-slip may see widening and irregularity of physis
g. Treatment and RTP
   i. Immediate non-weight bearing
   ii. Immediate referral to Pediatric Orthopedics
   iii. Emergent operative stabilization
h. Complications
   i. Avascular necrosis (AVN)
i. Outcomes and future risks
   i. Good outcome if SCFE is mild or moderate and treated early
   ii. If AVN develops, risk or significant early degenerative change
j. Prevention
   i. Early detection/high index of suspicion and immediate referral can prevent devastating consequences if left untreated
k. Prophylactic pinning of contralateral hip being considered more commonly in United States

3. Legg-Calve-Perthes Disease
   a. Overview
      i. Idiopathic avascular necrosis of femoral head in the skeletally immature, due to decreased blood supply
      ii. Epidemiology
         1. Incidence approximately 1:25,000 children
         2. Males 4 x more common than females
            a. Typical age between 4-10
            b. Can occur bilaterally
c. Worse prognosis in females
d. More common in Asians, Eskimos, and Caucasians

b. History
   i. Classic initial symptom is a painless limp
   ii. Mild hip, groin, or knee pain
   iii. Stiffness
   iv. Better with rest, worse with activity

c. Physical exam
   i. Limp
   ii. Stiffness with passive range of motion
   iii. Loss of internal rotation initially
   iv. Later loss of hip abduction and hip flexion
   v. Weakness on resisted motion

d. Differential diagnosis
   i. Septic arthritis
   ii. Osteomyelitis
   iii. Sickle cell disease
   iv. Spondyloepiphyseal dysplasia
   v. Gaucher's disease
   vi. Eosinophilic dysplasia
   vii. Transient synovitis
   viii. Hypothyroidism
   ix. Multiple epiphyseal dysplasia

e. Imaging
   i. Hip/pelvis radiographs
      1. Flattening of femoral head
      2. Sclerosis
      3. Loss of smooth contour of femoral head

f. Treatment and RTP
   i. Crutches, limited weight-bearing
   ii. Referral to Pediatric Orthopedics
   iii. Physical therapy, NSAIDS, occasionally casting
   iv. Occasionally surgery may be required for advanced stages, older age
   v. Most children can return to all activities in 18 months-2 years

g. Complications
   i. Osteoarthritis if residual deformity of femoral head
   ii. Better outcome if in the very young

4. Femoral Neck Stress Fracture
   a. Overview
      i. High risk stress fracture due to repetitive impact activities
   b. Epidemiology
      i. Frequent in long distance runners, dancers, military recruits
      ii. Often seen with the “female athlete triad”
   c. History
      i. Insidious onset of hip or groin pain related to impact activities
      ii. Often related to increase in training
iii. Better with rest
iv. Pain worsens with further training
v. Females may have history of amenorrhea
d. Physical Exam
i. Gait may be antalgic; if displaced, inability to bear weight
ii. Pain with passive range of motion, especially internal rotation
iii. Inability to perform a single leg hop without pain
iv. Bony tenderness of anterior hip often absent
e. Differential Diagnosis
i. Osteitis pubis
ii. SCFE (in early adolescents)
iii. Snapping Hip Syndrome
iv. Femoral Acetabular Impingement
v. Labral tear
f. Imaging
i. Plain films with poor sensitivity, but first step
ii. Bone scan or MRI if x-rays negative and high suspicion
iii. MRI most sensitive and specific
g. Treatment and RTP
i. Orthopedic consultation
ii. Non-weight bearing
iii. Tension sided (superolateral) requires immediate fixation
iv. Compression sided (inferomedial) may be treated with NWB 6-12 weeks
v. RTP based on clinical healing if nonsurgical, typically many months before impact
h. Complications
i. If undetected, can go on to displacement
ii. Non-union, re-fracture, osteonecrosis, AVN
iii. Maintain high index of suspicion for femoral neck stress fracture
5. Apophyseal Avulsion and Traction Injuries
a. Overview
i. Pelvic apophyses may not close until early 20’s
ii. Apophysis subject to acute avulsion or chronic traction
iii. Cartilaginous area weaker than tendinous attachment
iv. Whereas adult would experience muscle strain, apophysis is the weak link
b. Apophyses and attachments
i. Iliac crest - abdominal musculature
ii. Anterior superior iliac spine (ASIS) - sartorius
iii. Anterior inferior iliac spine (AIIS) - rectus femoris
iv. Pubic symphysis - adductors
v. Ischial tuberosity - hamstrings
vi. Greater trochanter – gluteal muscles
vii. Lesser trochanter- iliopsoas
c. Epidemiology
i. Adolescents and young adults, skeletally immature
ii. Soccer, football, track; sports with sprinting, jumping, kicking

d. History
i. Apophyseal traction injuries (apophysitis) presents with gradual onset pain without clear mechanism of injury in discrete area of apophysis
ii. Avulsion injuries present with sudden pain following forceful muscle contraction
   1. Doing the splits, kicking a ball, jumping over a hurdle
iii. May have heard or felt a “pop”
iv. Avulsion injuries may have difficulty weight-bearing

e. Physical Exam
i. Avulsion injuries may have antalgic gait
ii. Swelling may be visible
iii. Both avulsion and apophysitis with discrete tenderness at apophysis
iv. Both with pain +/- weakness with resisted motion

f. Differential Diagnosis
i. For ischial tuberosity avulsion, proximal hamstring injury
ii. AIIS and lesser trochanter apophysitis difficult to differentiate from other hip etiologies, i.e. femoral neck stress fracture, SCFE, FAI

g. Imaging
i. Hip/Pelvic radiographs: AP pelvis and AP frog-leg
   1. Evaluate bilaterally with same film
   2. May reveal avulsion
ii. May or may not see widening of apophysitis
iii. Additional imaging usually not required
iv. Frog leg lateral if SCFE in differential (anterior hip/groin pain in adolescent)

h. Treatment and RTP
i. Majority treated conservatively
ii. Protected weight bearing until pain resolves, may be few weeks on crutches in avulsion
iii. If greater than 2 cm displacement of avulsion, consider surgical consult
iv. Once clinically healed, initiate physical therapy
v. RTP may be few weeks for apophysitis
vi. RTP may be 6 weeks-6 months for avulsion injuries

6. Snapping Hip Syndrome
a. Overview
i. External: snapping of either iliotibial band or gluteus maximus over the greater trochanter
ii. Internal: iliopsoas tendon over iliopectineal eminence

b. Epidemiology
i. Common in female ballet dancers, most often ages 15-40

c. History
i. Describe painless or painful snapping of hip (audible)
ii. Typically long history of symptoms rather than acute onset
iii. Certain dance maneuvers will predictably reproduce symptoms
iv. Better with rest

d. Physical exam
   i. Gait is typically non-antalgic
   ii. Patient may be able to reproduce snapping
   iii. May be able to palpate snap
   iv. May have tenderness in area of snapping

e. Differential diagnosis
   i. Iliotibial band syndrome
   ii. Iliopsoas tendinitis
   iii. Intra-articular loose body
   iv. Acetabular labral tear
   v. Femoracetabular impingement

f. Imaging
   i. Radiographs of hip if diagnosis unclear
   ii. MSK ultrasound may help identify area of snapping
   iii. MRI with arthrogram may be indicated if source of snapping not clear;
      i.e. concern for acetabular labral tear

g. Treatment and RTP
   i. Physical therapy mainstay of treatment
   ii. May continue to participate as tolerated, avoid provocative maneuvers

h. Complications
   i. Chronic snapping can lead to bursitis and more limiting pain

7. Acetabular labral tear
   a. Overview
      i. Hip socket lined by acetabular cartilage subject to tearing with turning/twisting sports
      ii. May be associated with abnormally shaped femoral neck or acetabulum (femoracetabular impingement, FAI)
      iii. History of developmental hip dysplasia, Legg-Calve-Perthes disease, SCFE, or other risk factors
   b. Epidemiology
      i. Unknown incidence, but not infrequent
      ii. Labral tears found on 55% of arthroscopies for intractable hip pain
   c. History
      i. May have history of acute trauma involving twisting or planting
      ii. May be more insidious onset of pain
      iii. Vague hip or groin strain
   d. Physical Exam
      i. Physical exam findings may be few
      ii. Internal and external rotation may be painful
      iii. Impingement sign: Hip flexion to 90. Pain with adduction and internal rotation (may be seen with associated FAI)
   e. Differential Diagnosis
i. Internal Snapping Hip
ii. Iliopsoas tendinitis
iii. Femoral neck stress fracture
iv. Hip OA in adults

f. Imaging
   i. Hip/pelvis radiographs may show evidence of FAI
   ii. MRI arthrogram for labral tear

 g. Treatment and RTP
   i. If suspect labral tear in young athlete, consider PT
   ii. If definitive labral tear and fail conservative treatment, refer for hip arthroscopy

h. Complications
   i. Acetabular labral tear and/or FAI may lead to early hip osteoarthritis
Knee Injuries

Overview of the knee and anatomy
1. Motions
   a. Hinge joint
      i. Flexion
      ii. Extension
2. Most commonly injured joint
3. Knee Anatomy
   a. Bones and Bony Prominences
      i. Femur
      ii. Tibia
      iii. Fibula
      iv. Patella
      v. Tibial tuberosity
      vi. Gerdy's tubercle
   b. Muscles
      i. Quadriceps
         1. Rectus femoris
         2. Vastus medialis
         3. Vastus lateralis
         4. Vastus intermedius
      ii. Hamstrings
         1. Semitendinosus
         2. Semimembranosus
         3. Biceps femoris
      iii. Calf
         1. Gastrocnemius
         2. Soleus
   c. Ligaments (bone to bone attachment)
      i. ACL-anterior cruciate ligament
         1. Keeps tibia from translating anteriorly off the femur
      ii. PCL- posterior cruciate ligament
         1. Keeps tibia from translating posteriorly off the femur
      iii. MCL- medial collateral ligament
      iv. LCL- lateral collateral ligament
   d. Tendons (muscle/tendon to bone attachment)
      i. Quadriceps tendon
      ii. Patellar tendon (actually a ligament but called a tendon)
   e. Other structures
      i. Medial meniscus
      ii. Lateral meniscus
      iii. Prepatellar bursa
      iv. Patellar retinaculum
      v. Pes anserine bursa
vi. Iliotibial (IT) band  

vii. Infrapatellar fat pads  

4. Important to assess both the knee and hips in children and adolescents presenting with knee pain as hip pain can be referred to the knee  
a. Hip injuries that may present as knee pain (see hip section)  
   i. Slipped capital femoral epiphysis (SCFE)  
   ii. Legg-Calve-Perthes disease  
   iii. Stress Fracture  
   iv. Septic hip  
   v. Synovitis  

**Physical Exam**  
1. Evaluate both lower extremities, using non-injured side as a comparison  
a. Visualize joint above during exam  
b. Loose fitting shorts  
c. Socks and shoes off  
2. Gait evaluation  
a. Evaluate foot type and motion  
   i. Pes planus / pes cavus  
   ii. Hyperpronation  
   iii. Hypersupination  
b. Weight-bearing status, limp, range of motion  
3. Hip internal and external rotation  
a. Important in skeletally immature patients  
b. Limited motion, especially reduced and/or painful internal rotation may indicate hip pathology  
4. Inspection  
a. Evaluate for physical deformity, bruising, redness, swelling, muscle atrophy  
5. Knee range of motion  
a. 0 to 130 degrees of flexion  
b. If a joint lacks full motion actively, the examiner should passively move the joint to see if full motion can be obtained.  
6. Strength  
a. Quadriceps  
b. Hamstring  
7. Effusion  
a. Milking of the knee joint to look for intra-articular effusion  
b. Differentiate between intra-articular effusion and localized extra-articular swelling (ex. fat pad)  
8. Palpation  
a. Tibial tuberosity  
   i. Acute injury/pain may indicate fracture  
   ii. Chronic injury/pain may indicate Osgood-Schlatter disease in skeletally immature  
b. Patellar tendon  
   i. Pain can indicate patellar tendonitis or tendonosis
c. Medial joint line
   i. Medial collateral ligament (MCL) injury
   ii. Medial meniscus injury, especially if posteromedial pain (very non-specific)
   iii. Hamstrings cross mid-medial joint line
d. Lateral joint line
   i. Lateral collateral ligament injury
   ii. Lateral meniscus injury, especially if posterolateral pain (very non-specific)
   iii. IT band crosses lateral joint line
e. Distal femoral and proximal tibial physes near MCL and LCL attachments
   i. Physeal pain may indicate fracture
f. Patella
   i. Step-off signifying fracture
   ii. Pain at inferior pole of patella
      1. Acute pain and swelling may indicate sleeve fracture or avulsion fracture
      2. Chronic pain may indicate Sinding Larsen Johansson Syndrome
   iii. Medial and lateral facet pain - patellofemoral stress syndrome

9. Flexibility
   a. Quadriceps
   b. Hamstring
      i. Popliteal angle of less than 160 degrees indicates hamstring tightness
c. Hip flexors (Thomas test)
d. IT band (Ober’s test)

10. Special tests
    a. Patella
       i. Medial and lateral patellar glides (patellar mobility)
          1. Hypermobility - more than ½ the diameter of patella movement in either direction; may be at risk for subluxation/dislocation
       ii. Compression test - pain suggests patellofemoral stress syndrome
       iii. Apprehension test - pain/apprehension or patellar subluxation/dislocation indicates patellar instability/maltracking
   b. Ligaments
      i. Lachman’s test - increased laxity/no endpoint indicates injury to ACL
         1. More specific and sensitive than anterior drawer
      ii. Anterior drawer test - increased laxity may indicate ACL damage
         1. At 90 degrees (angle in which to do anterior drawer test) most people's hamstrings are tight (the hamstrings also prevent the tibia from moving forward from the femur) which can make results inconclusive
      iii. Posterior drawer test - increased laxity indicates PCL damage
         1. Starting point – tibia should sit slightly forward on femur
         2. If starting point reveals tibia flush or behind femur, then the examiner may be misled into feeling a positive anterior drawer test, when the injury is to the PCL
      iv. Valgus stress test at 0 and 30 degrees – to assess MCL
         1. Increased pain and laxity at 30 degrees signifies injury to MCL
2. Increased pain and laxity at 0 degrees signifies injury to MCL and other structures (ACL, capsule) that stabilize knee with valgus stress applied

v. Varus stress test at 0 and 30 degrees – to assess LCL
   1. Increased pain and laxity at 30 degrees signifies injury to LCL
      a. Rare to damage LCL in isolation
   2. Increased pain and laxity at 0 degrees signifies injury to LCL and other structures (posterolateral corner) that stabilize the knee with varus stress applied

c. Meniscus
   i. McMurray’s test - pain or click/clunk during internal and external rotation may indicate meniscal pathology
      1. Many with patellofemoral pain or Osgood Schlatter disease may also experience pain with full flexion of the knee
   ii. Apley’s compression test – pain may indicate meniscus pathology
      1. Since done with patient prone and knee flexed to 90 degrees, this test is more helpful in patients that have lost knee ROM

d. Single leg squat
   i. Assess for general core body strength
      1. Knee that moves into valgus (increased Q angle) and does not maintain a straight line with the hip and ankle indicates pelvic/core body weakness
      2. Truncal instability (needed to lean or bend at the waist) indicates insufficient core strength

**Traumatic injuries**

1. Anterior cruciate ligament injuries
   a. Overview
      i. ACL maintains proper relationship between tibia and femur
      ii. Prevents tibia from moving anteriorly off the femur
      iii. Adolescent females are 2-8x more likely to injury the ACL versus their male counterparts
      iv. 1/3000 people tear their ACL
   b. History
      i. Noncontact injury- foot planted, athlete twists, turns or pivots and hears or feel a "pop"
      ii. Contact Injury
      iii. Difficulty walking
         1. Feeling of instability, shifting or giving way with walking, running, pivoting
      iv. Knee swelling within 24 hours of injury
   c. Physical exam
      i. +/- Inability to bear weight, limp
      ii. Limited ROM secondary to pain and swelling
      iii. Large effusion within 24 hours of injury
      iv. Positive Lachman’s test (compare to contralateral knee)
      v. Consider the “unhappy triad” of concomitant MCL and meniscal injury
d. Differential diagnosis
   i. Patellar dislocation
   ii. Fracture
   iii. MCL sprain
   iv. Meniscal injury

e. Imaging
   i. Knee radiographs (AP, lateral, tunnel/notch and sunrise)
      1. Joint effusion
      2. Tibial eminence fracture – more common in skeletally immature
      3. Segond fracture on AP view
         a. Lateral capsule/tibial fracture which is pathognomonic for ACL injury
   ii. MRI

f. Treatment
   i. Immediate
      1. PRICES
      2. May need crutches for assisted weight bearing
      3. May begin knee ROM immediately
      4. May not return to cutting and pivoting sports unless surgery
         a. Risk for further damage to meniscus and increased risk for arthritis
   ii. Surgical treatment
      1. Autograft (own tissue) or allograft reconstruction using patellar tendon, hamstrings or quadriceps tendon
      2. Other appropriate surgical intervention as warranted (meniscal etc.)
      3. Controversy regarding surgical management of skeletally immature secondary to risk of physeal damage
         a. Physeal sparing procedures
         b. Waiting until skeletal maturity before surgical involvement
         c. Rehabilitation difficulties in young patients
   iii. Return to play
      1. May return once fully functional (6-9 months post surgery)
         a. Core strengthening and functional rehabilitation (running, cutting, pivoting, jumping)
      2. Some use an ACL stabilizing brace for support/proprioceptive effect
         a. No studies to support idea that bracing in and of itself prevents further injury or damage or can replace actual surgical repair

g. Complications
   i. Damage to other ligaments/soft tissues
   ii. Increased risk for future arthritis
iii. Increased risk for patellar tendon pain (use of autograft patellar tendon for repair)

iv. Inability to return to previous level of activity secondary to pain, stiffness, or instability

h. Outcomes and future risks
   i. 80-90% success rate with reconstructive surgery
   ii. Up to 8% may re-tear graft
   iii. Increased risk for arthritis

i. Prevention
   i. ACL risk reduction programs
      1. Focus on flexibility, core strengthening, agility and proprioception to prevent injury
      2. Some studies show effectiveness at injury reduction, but further research needed
   ii. ACL bracing
      1. No studies to support idea that bracing prevents further injury or damage or can replace actual surgical repair
         a. May provide proprioceptive feedback

2. Medial and lateral collateral ligament sprains
   a. Overview
      i. Skeletally immature may be at risk for physeal fracture (distal femur or proximal tibia) as opposed to MCL/LCL ligament sprain
      ii. MCL commonly injured ligament in isolation secondary to valgus stress (cutting injury)
         1. Primary restraint to valgus stress; secondary stabilizer to anterior translation; resists external rotation of tibia
      iii. LCL uncommonly injured ligament in isolation secondary to varus stress
         1. Primary restraint to varus stress; resists external rotation of tibia
   b. History
      i. Pain/feeling of instability with walking, running, cutting
      ii. May complain of swelling
      iii. Often have altered weight bearing
      iv. MCL
         1. Valgus stress to knee
         2. Most common in football, basketball, soccer
      v. LCL
         1. Varus stress combined with other forces
         2. Rarely injured in isolation
            a. Concern for posterolateral corner injury, which necessitates an urgent orthopedic referral
   c. Physical exam
i. MCL
   1. +/- limping/altered weight bearing
   2. Often decreased knee range of motion
   3. May have an effusion or swelling
   4. Pain with palpation of MCL or insertion/origin sites
   5. Positive valgus stress test at 30 degrees: pain and increased laxity (compare to contralateral knee)
   6. Consider further injury (ACL, anterior capsule) if positive valgus stress test at 0 degrees: pain and increased laxity (compare to contralateral knee)

ii. LCL
   1. +/- limping/altered weight bearing
   2. Often decreased knee range of motion
   3. May have an effusion or swelling
   4. Pain with palpation of LCL or insertion/origin sites
   5. Positive varus stress test at 30 degrees: pain and increased laxity (compare to contralateral knee)
   6. Consider further injury (posterolateral corner, popliteus) if positive varus stress test at 0 degrees: pain and increased laxity (compare to contralateral knee)

D. Differential diagnosis
   i. Meniscal injury
   ii. ACL injury
   iii. Salter Harris type fracture of distal femur
   iv. Tibial plateau fracture
   v. Knee dislocation
   vi. Popliteus avulsion

E. Imaging
   i. Knee radiographs (AP, lateral, tunnel/notch and sunrise) – consider oblique views to further assess for fracture; stress views not indicated in pediatric population
      1. Radiographs typically normal except for soft tissue swelling
   ii. MRI
      1. As needed, used to assess soft tissue damage and further injuries

F. Treatment and return to play
   i. PRICES
   ii. Rehabilitation for ROM, flexibility, strengthening, core strengthening and functional return to activity (ideally full, pain free range of motion and strength)
   iii. Often takes 4-8 weeks
      1. Consider bracing in a hinged knee brace for 3-4 weeks after recovery
      2. Typically MCL heals on own without need for surgery
      3. Higher grade injuries take longer to recover
         a. Grade 3 injuries may require surgery
4. LCL sprain is more likely to require surgical intervention due to concomitant injuries
g. Complications
   1. Incomplete rehabilitation may lead to further injuries
   2. Missed physeal fractures may cause growth arrest
h. Prevention
   1. Some studies suggest bracing may decrease risk of MCL sprain
      a. Commonly used in collegiate football lineman
   2. No known effect of bracing at decreasing LCL sprain rates
3. Patellar dislocation/subluxation
   a. Overview
      i. Dislocation/subluxation typically occurs when patella displaces laterally
         1. Dislocation occurs when patella moves completely out of place
         2. This may spontaneously reduce or need to be reduced by a medical professional
      ii. Subluxation occurs when patella moves partially out of place and usually pops back in on own (less severe than dislocation)
      iii. A history of dislocation/subluxation increases the risk of recurrence
   b. History
      i. Typically twist or turn causes athlete to hear or feel a “pop”
      ii. May appreciate patella being out of place or moving out of place
      iii. Immediate pain, swelling and difficult weight bearing
      iv. Pain/feeling of patellar instability with walking, running, cutting
c. Physical exam
   i. +/- Ability to bear weight, limping
   ii. Decreased ROM
   iii. Effusion/swelling within hours of injury
   iv. Pain around medial retinaculum
   v. Pain along medial and lateral facets
   vi. Positive apprehension test
   vii. Positive compression test
   viii. +/- VMO atrophy with inability to fire VMO
d. Differential diagnosis
   i. ACL tear
   ii. MCL sprain
   iii. Meniscal injury
   iv. Fracture
   v. Osteochondral defect
e. Imaging
   i. Knee radiographs (AP, lateral, tunnel/notch and sunrise)
      1. Radiographs may only show soft tissue swelling
      2. May see lateral tilt of patella or shallow femoral trochlea/groove on sunrise view
      3. May see fragments of patellar fracture
   ii. MRI
1. As needed, but obtain if concerned about osteochondral injury, fracture or concomitant injury

f. Treatment and return to play
   i. PRICES
   ii. Subluxation
      1. May begin ROM within 24-48 hours
      2. Rehabilitation for ROM, flexibility, strengthening, core strengthening and functional return to activity
      3. Typically 4-8 weeks
      4. Patellar stabilizing brace or McConnell taping may be useful to help keep patella properly placed

iii. Dislocation
   1. Many use knee immobilizer for 1-3 weeks
      a. Will prolong recovery as they shut down quadriceps and do not allow resorption of effusion secondary to limited motion
      b. However, may improve healing
   2. Rehabilitation for ROM, flexibility, strengthening, core strengthening and functional return to activity
   3. Typically 6-8+ weeks
   4. Patellar stabilizing brace or McConnell taping may be useful to help keep patella properly placed

iv. Return to play once fully functional
v. Surgical intervention
   1. Surgery considered if anatomic factors and failure of physical therapy (daily exercises for 6 months)
   2. Recurrent/repeat dislocations

g. Complications
   i. Osteochondral fractures behind patella
   ii. Recurrent subluxations/dislocations

h. Outcomes and future risks
   i. Incomplete rehabilitation increases risk for future subluxations/dislocations
      1. There is risk of recurrence even after full recovery
   ii. Recurrent subluxations/dislocations may lead to need for surgical intervention (lateral release, distal osteotomy)

i. Prevention
   i. Bracing may help prevent severe future dislocations in those with a history of dislocations
   ii. Inconclusive studies regarding injury prevention exercises

4. Meniscus pathology
   a. Overview
      i. The medial and lateral meniscus are wedge shaped and C-shaped stabilizers and shock absorbers of the knee located on superior aspect of the tibia
      ii. They are usually injured with twisting and flexion maneuvers
iii. Meniscus tears may be associated with other injuries (ACL tear, MCL sprain)
iv. Meniscus lacks a strong blood supply which usually prevents non-surgical healing

b. Discoid meniscus
i. Affects 2% of population
ii. Thicker, disc-shaped lateral meniscus (developmental)
iii. Increased risk for damage with normal forces secondary to pressure on misshaped meniscus

c. History
i. Twisting, cutting, squatting mechanism causing pain and/or a “pop”
ii. Mild-moderate effusion usually occurs within several hours
iii. Loss of knee motion, most commonly flexion
iv. Difficulty with weight bearing
v. True catching or locking of knee (knee stuck in flexion for at least a few minutes (not usually instantaneous relief) with relief after wiggling it around to get it to unlock
   1. Suggestive of a bucket handle tear or flipped meniscus

d. Physical Exam
i. +/- Ability to bear weight, limping
ii. Decreased knee ROM
   1. Pain with flexion
   2. Inability to fully straighten (extension)
   3. “Surgical emergency” if can’t fully extend secondary to possibility to flipped meniscus
      a. Make non-weight bearing on crutches
      b. Can damage articular surface of knee if walking on a partially bent knee
iii. Mild-moderate effusion, especially when acute injury
iv. Posteromedial or posterolateral joint line tenderness
v. Positive McMurray’s test or Apley’s compression test

e. Differential Diagnosis
i. Loose Body
ii. Ligament injury
iii. Fracture
iv. Osteochondral Injury

f. Imaging
i. Knee radiographs (AP, lateral, tunnel/notch and sunrise)
   1. Radiographs typically normal except for soft tissue swelling
ii. MRI
   1. Needed to help evaluate injury

g. Treatment and return to play
i. Immediate treatment
   1. PRICES
   2. Weight bearing as tolerated on crutches
   3. Unforced ROM exercises
ii. Surgical treatment
   1. Repair vs resection (removal)
      a. Depends on location, type and extent of injury
      b. Repair is preferred when possible, as it may decrease risk of arthritis
         i. Repair has a longer recovery and return to play timeline compared to resection
      c. Both will need potential immobilization, crutch use and rehabilitation
      d. Return to play depends on surgical findings / procedure
         i. May return once fully functional

h. Complications
   i. Further damage to meniscus / knee without proper treatment
   ii. Increased post-operative risk for arthritis

i. Outcomes and future risks
   i. Incomplete rehabilitation can lead to increased risk for future injury

j. Prevention
   i. Inconclusive studies regarding injury prevention exercises

Overuse injuries and chronic pain
1. Osgood Schlatter disease (see apophyseal injury section)
2. Sinding Larsen Johansson syndrome (see apophyseal injury section)
3. Patellofemoral stress syndrome (PFSS)
   a. Overview
      i. Also known as patellofemoral pain syndrome (PFPS), patellofemoral syndrome (PFS)
      ii. Most common injury seen in general sports medicine clinics
      iii. Common in growing adolescents (female > male)
      iv. Usually begins after 8 years
   b. History
      i. Usually insidious onset, rarely traumatic
      ii. Anterior knee pain around and underneath patella with walking, running, jumping and weight bearing activity
      iii. Increased pain with walking stairs or hills
      iv. Pain with prolonged sitting (theater sign) and prolonged standing
      v. No night pain
      vi. No swelling, catching or locking
      vii. Commonly report “knee popping”, “knee giving way”
      viii. Activities may be limited secondary to pain
   c. Physical exam
      i. Usually normal weight bearing, but may cause limp
      ii. No joint effusion
      iii. Full knee ROM
      iv. +/- positive compression test
      v. May have patellar hypermobility
         1. May have patellar maltracking (j-tracking / lateral tracking)
2. +/- positive apprehension test (usually negative)
   vi. Pain on palpation of medial or lateral patellar facets
   vii. May have pain in full knee flexion, but negative McMurray’s test
   viii. Often have abnormal single leg squat testing
   ix. Typically have tight hamstrings and hip flexors
   x. May have genu recurvatum, increased femoral anteversion, pes planus, hyperpronation on gait evaluation

d. Differential diagnosis
   i. Osgood-Schlatter disease
   ii. Sinding Larsen Johansson syndrome
   iii. Patellar tendonopathy
   iv. IT band tendonopathy/friction syndrome
   v. Osteochondritis dissecans
   vi. Stress fracture
   vii. Underlying osteochondral injury

e. Imaging
   i. Knee radiographs (AP, lateral, tunnel/notch and sunrise)
      1. Radiographs typically normal
      2. May have lateral patellar tilt or shallow femoral groove/trochlea noted on sunrise view
   ii. MRI
      1. Usually not helpful

f. Treatment and return to play
   i. Relative rest may be needed – limit activities secondary to pain
      1. Cross training (swim, bike, elliptical) can be helpful for aerobic fitness
   ii. Pain control with ice and OTC analgesics
      1. Daily NSAID use indicates too much activity
   iii. Rehabilitation focusing on lower extremity flexibility (hamstrings, hip flexors and IT Band) and core/hip strengthening
      1. Need to do home exercise program (HEP) on daily basis
      2. Typically 6-8 weeks before seeing results
      3. Must be maintained for life of sport to decrease risk of recurrence
      4. Work to improve running, jumping and landing mechanics
   iv. OTC orthotics may be helpful if significant pes planus and hyperpronation on gait evaluation
   v. Patellar bracing or McConnell taping may be helpful if patellar hypermobility
   vi. Rarely requires surgical evaluation for intractable pain/dysfunction

g. Outcomes and Future Risks
   i. Recurrence or chronic pain into adulthood if non-compliant with rehabilitation and home exercise plan
   ii. Not necessarily “outgrown” after child and adolescent years

h. Prevention
i. More research is needed but flexibility and good core strength combined with proper running/jumping/landing mechanics may decrease risk for injury

4. Osteochondritis dissecans (OCD)
   a. Overview
      i. Lesion affecting the subchondral bone and articular cartilage
      ii. Etiology is unknown – hereditary, overuse, traumatic, vascular
      iii. Presents in a variable fashion, often mimicking PFSS
         1. Frequently a “missed” diagnosis
      iv. Most common in knee
         1. Posterolateral aspect of the medial femoral condyle
      v. Estimated to occur in 15-30/100,000 people
   b. Classification
      i. Juvenile (open physes)
         1. Usually 10 to 15 years of age
         2. More likely to heal without surgical intervention
         3. Open distal femoral physis is best predictor of successful non-operative management
      ii. Adult (skeletally mature)
         1. More likely to require surgery
   c. History
      i. Usually insidious onset of pain, usually activity related
      ii. Vague/poorly localized knee pain, but most commonly anterior or anteromedial pain
      iii. May report episodic catching, clicking or locking
         1. Mechanical symptoms may herald more advanced disease
      iv. May report intermittent effusions
         1. Significant swelling may herald more advanced disease
   d. Physical exam
      i. May have limp or altered weight bearing status
      ii. May have a loss of knee ROM
      iii. Often have joint effusion
      iv. May have tenderness localized to medial femoral condyle, medial patellar facet
      v. Wilson’s test (pain with internally rotating tibia during knee extension between 90 and 30 degrees that is relieved by external rotation of tibia) may be positive, but low sensitivity
   e. Imaging
      i. Knee radiographs (AP, lateral, tunnel/notch and sunrise)
         1. Posterior femoral condyle lesions best seen on tunnel/notch view
         2. Difficult to see on AP and lateral views
         3. Patellar OCD best seen on sunrise and lateral views
      ii. MRI
         1. Need to further evaluate the lesion
a. Determine if stable or unstable (loosening or loose bodies) – seen by signal intensity surrounding lesion
b. Further details on size and status of subchondral bone and articular cartilage
c. Evaluate for loose bodies

f. Treatment and return to play
   i. Immediately begin rest from activity (similar to non-operative treatment)
   ii. Consultation with specialist advised
      1. Treatment can be controversial
   iii. Non-operative
      1. Skeletally immature/juvenile with small, stable lesions
         a. Good blood flow to area near physis
      2. Asymptomatic lesions
      3. Limit activity (running, jumping, athletics, PE class)
         a. Typically 3-12 months
         b. Some recommend crutches and non-weight bearing
         c. Some recommend bracing with restricted motion
      4. Follow imaging to assess healing
      5. Once documented healing and asymptomatic, slowly begin to work on full functional recovery in rehabilitation
   iv. Operative
      1. Not responsive to non-operative management
      2. Large or unstable lesions in the juvenile patient
      3. Symptomatic lesions in skeletally mature patient
      4. Operative techniques and post-operative treatments vary
         a. Microfracture
         b. Internal fixation
         c. Osteochondral grafting
   v. Complications
      1. Damage to articular surface
         a. Increased risk of arthritis, especially when ignored or improperly treated
Ankle Injuries

Overview of the ankle and anatomy

1. Motion
   a. Hinge Joint
      i. Dorsiflexion
      ii. Plantarflexion
   b. Subtalar and Calcaneonavicular joints
      i. Inversion
      ii. Eversion

2. Ankle Anatomy
   a. Bones and Bony Prominences
      i. Tibia and Fibula
         1. Physis closes in girls between 12 and 14 years old, and in boys between 15 and 18.
      ii. Talus
      iii. Calcaneus
      iv. Navicular
      v. Base of 5th metatarsal
   b. Lower extremity compartments – muscles / nerves / artery
      i. Anterior compartment
         1. Extensor hallucis longus
         2. Extensor digitorum longus
         3. Peroneus tertius
         4. Anterior tibialis
         5. Deep peroneal nerve
         6. Anterior tibial artery
      ii. Lateral compartment
         1. Peroneus longus
         2. Peroneus brevis
         3. Superficial peroneal nerve
         4. Peroneal artery branches
      iii. Superficial posterior compartment
         1. Gastrocnemius
         2. Soleus
         3. Plantaris
         4. Sural nerve
      iv. Deep posterior compartment
         1. Posterior tibialis
         2. Flexor digitorum longus
         3. Flexor hallucis longus
         4. Posterior tibial nerve
         5. Posterior tibial artery
   c. Ligaments (bone to bone attachment)
      i. Lateral ligament complex
1. Anterior talofibular ligament (ATFL)
   a. Main stabilizer against inversion when foot is plantarflexed
2. Calcaneofibular ligament (CFL)
3. Posterior talofibular ligament (PTFL)
   a. Main stabilizer against inversion when foot is dorsiﬁxed

ii. Medial ligament
1. Deltoid ligament
   a. Prevents eversion

iii. Interosseous connection
1. Anterior inferior and posterior inferior tibiofibular ligaments
   a. Main stabilizers against external rotation when foot is dorsiﬁxed

**Physical Exam**

1. Key exam concepts
   a. Exposure of patient
      i. Socks and shoes off of both feet
      ii. Loose-ﬁtting shorts
   b. Always exam knee joint, especially the proximal ﬁbula, whenever ankle pain is present.
   c. Evaluate both ankles, using non-injured side as a comparison
2. Gait evaluation
   a. Evaluate foot type and motion
      i. Pes planus / pes cavus
      ii. Hyperpronation
      iii. Hypersupination
   b. Evaluate weight-bearing status, limp, range of motion
3. Inspection
   a. Evaluate for physical deformity, bruising, redness, swelling, warmth, muscle atrophy
4. Ankle range of motion
   a. Normal active range of motion of the ankle
      i. Dorsiﬁexion 20 degrees
      ii. Plantarflexion 50 degrees
      iii. Inversion 40 degrees
      iv. Eversion 20 degrees
   b. If a joint lacks full motion actively, the examiner should passively move the joint to see if full motion can be obtained.
5. Strength
   a. Dorsiﬁexion - tibialis anterior
   b. Plantarflexion – gastrocnemius/soleus complex
   c. Inversion - tibialis posterior
   d. Eversion - peroneal tendons
6. Palpation
a. Start proximally at fibular head, palpate entire fibula and tibia, including both lateral and medial malleolus. Pay attention to distal physes if open.
b. Syndesmosis including anterior tibiofibular ligament
c. Lateral ligament complex – anterior talofibular ligament, calcaneofibular ligament, posterior talofibular ligament
d. Deltoid ligament
e. Anterior joint line
f. Gastrocnemius / soleus muscles down to achilles tendon, achilles attachment on calcaneus and retrocalcaneal space
g. Peroneal tendon and base of 5th metatarsal
h. Anterior and posterior tibialis tendons and navicular

7. Flexibility
a. Gastroc/soleus flexibility should be tested with the knee in full extension and the ankle passively dorsiflexed. Measure the angle between the plantar surface of the foot and a perpendicular line drawn through the axis through the lower leg. Normal dorsiflexion is to 20 degrees on average.

8. Special Tests
a. Stability Tests
   i. Comparison to unaffected side as well as prior injury history are crucial for proper interpretation of stability tests
   ii. Anterior drawer test
      1. Assesses integrity of the anterior talofibular ligament
      2. A few millimeters of anterior translation of the talus with a solid endpoint is considered normal, however there is great variation between individuals.
   iii. Talar tilt test
      1. Assesses integrity of the calcaneofibular ligament
      2. Also known as the inversion stress test or varus stress test
      3. Should reveal minimal movement unless the CFL is torn
b. Syndesmosis
   i. Squeeze test
      1. Medial / lateral compression of the tibia and fibula is performed at the level of the mid-calf
      2. Reproduction of pain signifies a positive test
   ii. External rotation stress test
      1. Knee should be bent to 90 degrees
      2. Foot is dorsiflexed and externally rotated
      3. Reproduction of pain signifies a positive test
      4. However it often is positive in individuals with a fibular fracture
b. Peroneal tendon instability test
   1. Also known as a circumduction test
   2. Active circumduction of the foot in the clockwise and counterclockwise directions is performed. Palpation posterior to the lateral malleolus and assessment of patient's pain in helpful when interpreting the test.
3. Assesses for subluxation or dislocation of the peroneal tendon over the lateral malleolus
4. Indicative of a retinacular tear if true dislocation occurs

9. Functional assessment
   a. Walk on tip toes
   b. Hop on two feet
   c. Hop on a single foot
   d. Jog

10. Proprioceptive skills
    a. Single foot balance on firm and / or uneven surface
    b. Single foot toe-raises

11. Neurovascular
    a. Check capillary refill, posterior tibialis or dorsalis pedis pulse
    b. Assess achilles reflexes
    c. Check for sensory changes

12. Skin
    a. Assess for bruising

**Traumatic Injuries**

1. Ankle Sprains
   a. A sprain is a disruption of a ligament, which connects bone to bone.
   b. Three types of ankle sprains:
      i. Lateral (ATFL, CFL, and / or PTFL)
      ii. Medial (Deltoid ligament)
      iii. High ankle sprains (AKA syndesmotic injury)
   c. Epidemiology
      i. All types of ankle sprains make up 15-25% of all musculoskeletal injuries
   d. Lateral ankle sprains
      i. Overview
         1. Ankle is inverted on a plantarflexed foot causing the lateral ligament complex to have excessive forces on it, and potentially tear.
         2. Lateral ankle sprains make up 85% of all ankle sprains. They are most common in basketball, football, and volleyball, but they can affect athletes in virtually any sport.
         3. The lateral ligament complex is composed of the anterior talofibular ligament (ATFL), calcaneofibular ligament (CFL), and posterior talofibular ligament (PTFL). The ATFL is the weakest and most commonly torn.
         4. Grading
            a. Grade I - ligament stretching, mild to moderate swelling, mild loss of function / ROM, no instability, and minimal difficulty weight bearing.
b. Grade II - partial ligament tear. Moderate to severe swelling and bruising, moderate loss of function / ROM, moderate instability, difficulty weight bearing.

c. Grade III - complete ligament tears. They result in complete loss of function, severe limitations in ROM, severe instability, and inability to bear weight.

ii. History
1. Landing on an inverted and plantarflexed ankle is the typical mechanism. This typically occurs due to an awkward land or uneven terrain.
2. Patients often report “rolling” the ankle and sometimes feeling a “pop”
3. Swelling and bruising are common

iii. Physical Exam
1. Bruising and swelling occur if a ligament is partially or completely torn. Palpate for bony tenderness vs. ligament tenderness.
2. Range of motion is often limited
3. Strength testing may reveal no significant deficits for Grade I or II sprains
4. Anterior drawer test will reveal laxity if ATFL torn.
5. Talar tilt will reveal laxity if CFL also torn.
6. Gait and functional exam will be variable depending on severity and timing of injury

iv. Differential Diagnosis
1. Fibular shaft fracture
2. Salter Harris type 1 fibular physis fracture
3. Syndesmotic injury
4. Peroneal tendon strain / subluxation
5. Talar OCD
6. Base of 5th metatarsal fracture

v. Imaging
1. X-ray indications
   a. Suspicion of open distal tibial / fibular physes
   b. Extensive bruising, swelling, or any blistering
   c. Inability to bear weight
   d. Bony tenderness
   e. Persistent pain
2. Ottawa Ankle Rules have been applied to validated for midfoot and ankle fracture in pediatric patients
3. X-rays should include AP, lateral, and mortise view of the injured ankle. Include foot AP, lateral, and oblique if base of 5th metatarsal is tender or foot is tender or swollen.
4. Lateral malleolus swelling is common
5. Attention should be paid whether the lateral malleolus is open, as a Salter Harris type 1 fracture is a clinical diagnosis.

vi. Treatment
   1. PRICES
   2. Immobilization in a brace for compression (rigid or functional)
   3. Crutches as needed
   4. Early range of motion and strengthening will speed recovery and return to play for most mild and moderate sprains.
   5. Physical therapy can help minimize pain, restore function, and help prevent re-injury through proprioceptive training.

vii. Referral
   1. Patients with recurrent sprains, grade III sprains, or ongoing pain / dysfunction should be referred to a specialist.

viii. Return to Play
   1. Full motion, full strength, and a normal functional exam are required for return to sports.
   2. Recovery times are variable. Grade I injuries typically take up to 14 days for recovery. Grade II injuries take 2-4 weeks and Grade III injuries require more than 4 weeks for full rehabilitation.
   3. Bracing and / or taping are recommended when an individual is returning to sports.
      a. Semi-rigid braces, such as a lace up brace with lateral support or a hinged brace with rigid sides, are typically the best for wearing during sports.
      b. Taping can be beneficial also, but tape is proven to loosen within 20 minutes.

ix. Complications / Outcomes / Future Risk
   1. There is a high reoccurrence rate for lateral ankle sprains.
   2. Chronic complications may occur in up to 40% of ankle sprain athletes. This is usually due to lack of proper rehabilitation and / or a missed concomitant diagnosis.
   3. Individuals are at risk of: recurrent ankle sprains, chronic ankle instability, talar OCD.

x. Prevention
   1. Balance exercises have been proven to decrease reoccurrence of ankle sprains.
   2. Bracing for a minimum of 6 months post-injury may also be helpful.

   e. Salter Harris (SH) Type I Fracture – Distal Fibula
      i. Overview of SH Classification – description of physeal injuries
         1. Type I goes through the physis.
2. Type II goes through the physis and the metaphysis
3. Type III goes through the physis, the epiphysis, and joint space
4. Type IV goes through the metaphysis, physis, the epiphysis, and the joint space
5. Type V is a crush injury to the physis

ii. Overview of SH type I fracture
   1. Ankle inversion on a plantarflexed foot
   2. By definition, distal fibular physis must still be open, so any child who is skeletally immature at the ankle can develop this injury
   3. The physis is the weak link in growing individuals. Therefore a SH fracture is more likely than an ankle sprain in individuals with open physis.
   4. It is a clinical diagnosis based on consistent mechanism, open physis on x-ray, and tenderness of physis

iii. Epidemiology
   1. Salter-Harris Type I fractures are the most common ankle fracture in children.

iv. Patient History
   1. Inversion ankle injury due to awkward land or uneven terrain
   2. Patient may complain of swelling, pain over the distal fibula, and trouble bearing weight.

v. Physical Exam Findings
   1. + / - Inability to bear weight
   2. Swelling over lateral malleolus
   3. Bruising may be present
   4. Tender distal fibular physis

vi. Differential Diagnosis
   1. Lateral ankle sprain
   2. Syndesmotic sprain
   3. Peroneal tendon strain

vii. Imaging
   1. X-rays should include AP, lateral, and mortise view of the injured ankle
   2. By definition, distal fibular physis must be open.
   3. Initial x-rays are typically normal, although the physis may appear widened if comparison views of the uninjured ankle are obtained
   4. Repeat x-rays in 4 weeks may show new bone / callus formation over distal fibula but often remain normal

viii. Treatment
   1. Fracture boot or casting for 3-4 weeks
2. Physical therapy for range of motion, strengthening, and balance exercises should be encouraged after boot / cast removal

ix. Referral
   1. Specialty referral is recommended secondary to physeal involvement.

x. Return to Play
   1. After 4 weeks immobilization, re-examination should occur
   2. Full motion, full strength, and a normal functional exam must be present before they can safely return to sports
   3. Bracing and / or taping is recommended
      a. Semi-rigid braces, such as a lace up brace with lateral support or a hinged brace with rigid sides, are typically the best for wearing during sports.
      b. Taping can be beneficial also, but tape is proven to loosen within 20 minutes.

xi. Complications / Outcomes / Future Risk
   1. There is a high reoccurrence rate for inversion injuries
   2. SH I injuries have low risk of affecting physeal growth

xii. Prevention
   1. Balance exercises have been proven to decrease reoccurrence of ankle sprains.
   2. Bracing for a minimum of 6 months post-injury may also be helpful.

f. Peroneal Tendon Injury
   i. Overview
      1. The peroneal longus and brevis both run along the posterior border of the lateral malleolus. The brevis then inserts at the base of the 5th metatarsal and the longus inserts at the base of the first metatarsal.
      2. Common peroneal injuries
         a. Tendinosis
            i. An overuse injury. It commonly occurs in individuals with chronic ankle instability due to repetitive ankle sprains.
         b. Strains
            i. An acute strain may occur as a result of an inversion injury that places a stretch on the tendon group.
         c. Subluxation/Dislocation
            i. Subluxation/dislocation may occur with an inversion injury that can tear the peroneal retinactulum. A pronounced pop usually occurs.
   3. Patient History
a. Overuse tendinosis will cause insidious onset of pain over or just posterior to the lateral malleolus. The pain may extend up the lateral leg or down towards the 5th metatarsal. Swelling in the tendon sheath may be present.

b. Strain occurs after an acute inversion injury that results in lateral pain.

c. Subluxation/dislocation may result in a sense of instability. Patients may complain of recurrent popping or snapping of the tendon of the lateral malleolus. The injury frequently presents after it has become chronic in nature.

ii. Epidemiology

1. The peroneal tendon is infrequently injured compared to other tendons within the body.

2. However, recognition of an injury, particularly a retinaculum tear, is important.

iii. Physical Exam Findings

1. Tendinosis or strain may result in tender peroneal tendon, tendon sheath swelling, pain with resisted eversion, pain rising up on toes.

2. Subluxation/dislocation of the peroneal tendon over the fibula may be evident with a painful eversion and dorsiflexion of ankle or resisted circumduction of ankle.

iv. Differential Diagnosis

1. Fibula fracture

   a. Fibula bears 5% of body weight with ambulating.

   b. Inversion injury may produce acute fibula fracture.

   c. Chronic stress may produce stress fracture of fibula.

   d. Signs and symptoms mimic peroneal injury.

   e. X-ray is indicated to minimize chance of missing this injury. Stress fractures may not show up on x-ray until the healing process has begun. Within the first few weeks of pain – they will show up on MRI.

2. Dancer's fracture

   a. A fracture to the proximal 5th metatarsal at the base, through the proximal metaphysis.

   b. Due to an acute inversion injury.

   c. Crack or pop may be felt, but swelling and bruising may be minimal.

   d. Patient will be tender on base of the 5th metatarsal.

   e. Foot x-rays should include a weightbearing AP, lateral, and oblique views.

   f. Fracture boot can be used to immobilize patient for up to 6 weeks.
g. Conservative care is usually successful in achieving bony healing.

3. Jones fracture
   a. A fracture within 1.5cm of tuberosity at the metaphyseal / diaphyseal junction
   b. It is usually an acute injury. However, chronic stress injury to the bone may be an underlying problem.
   c. The mechanism is a lateral force to forefoot with the ankle plantarflexed
   d. The patient develops swelling, bruising, and tenderness over 5th metatarsal proximally.
   e. Foot x-rays should include a weightbearing AP, lateral, and oblique views.
   f. Conservative treatment in a non-weightbearing cast for 6 - 8 weeks is appropriate for an acute fracture
   g. However, non-union risk is high and surgical fixation is the treatment of choice if conservative treatment fails or the individual is a high-performance athlete
   h. Referral to specialist is required

v. Imaging
   1. Normal ankle x-rays are present in most cases of a peroneal injury, although a fibular avulsion fracture may be present.
   2. X-ray is recommended since a fibular fracture can mimic a peroneal injury. Foot AP, lateral, and oblique views may be necessary if 5th metatarsal is tender.
   3. MRI imaging of the peroneal retinaculum is recommended if subluxation / dislocation is present and conservative treatment fails.

vi. Treatment
   1. J-shape felt or foam pad may be used in conjunction with taping or bracing to control peroneal tendon
   2. Tendinitis / strains will improve with physical therapy.
   3. Subluxation may respond with use of a short leg cast for 4-6 weeks followed by physical therapy.
   4. Retinacular tears require surgical reconstruction of the retinaculum if conservative care fails.

vii. Referral
   1. Specialist referral recommended

viii. Return to Play
   1. Full motion, strength, and ability to perform functional activity is required for return.

ix. Complications / Outcomes / Future Risk
   1. Overuse strains may recur if proper physical therapy is not done. Encourage proprioceptive skills, assess need for custom orthotics.
2. Recurrent subluxation of the peroneal tendon may result in ongoing pain and a sensation of instability.

x. Prevention
   1. Ankle strengthening and balance skills may help individuals who have developed overuse tendinitis or strained the peroneal tendon.

g. Syndesmotic Ankle Sprain (AKA High Ankle Sprain)
   i. Overview
      1. Tear of the anterior and/or posterior inferior tibiofibular ligaments that connect the tibia and fibula
      2. Occurs when the individual is weightbearing on an ankle that is dorsiflexed and subsequently externally rotated
      3. Because there is an anterior and a posterior component to the ligament, a partial tear can occur.

   ii. Epidemiology
      1. Account for up to 10% of all ankle sprain
      2. There is a higher incidence in contact sports (being tackled is a common mechanism)

   iii. Patient History
      1. The patient may report a mechanism consistent with dorsiflexion and external rotation, such as being tackled with their foot planted
      2. They feel immediate pain and frequently can not bear weight

   iv. Physical Exam Findings
      1. This type of ankle sprain may or may not have swelling and bruising associated with it, therefore pain is often out of proportion to the appearance of the injured leg
      2. Patients are tender along anterior inferior tibiofibular ligament and along the interosseous space between the tibia and fibula
      3. Most athletes cannot walk on the injured limb
      4. Positive external rotation stress test and tibia – fibula squeeze test are typically positive

   v. Differential Diagnosis
      1. Fibula fracture
         a. See above section on differential diagnosis of peroneal injuries

   vi. Imaging
      1. Ankle series with AP, lateral, and mortise view is imperative.
         a. Tibiofibular overlap should be > 1mm when measured 1cm proximal to the plafond on mortise view, and >6mm on the AP view
b. Medial clear space (between medial malleolus and medial talus) should be between 2-4mm. If there is greater than 2mm difference compared to unaffected side, that is also considered positive.

c. The tibiotalar clear space (between tibia and fibula 1 cm proximal to the top of the talus) should be ≤ 6mm. Anything greater is concerning for a syndesmotic injury.

2. Comparison of unaffected leg with weightbearing or stress views are helpful to determine if syndesmotic widening is present.

3. If a syndesmotic injury is suspected clinically and on x-ray, the proximal fibula should be palpated – if tenderness is present, x-ray is required to rule out a fracture of the proximal fibula, called a Maisonneuve fracture. This requires surgical intervention.

vii. Treatment

1. A complete tear requires surgery. Partial tear requires immobilization in a walking boot with crutches for 4-6 weeks.

2. General principles of protect, rest, ice, compress, and elevate should be followed.

3. Physical Therapy should be started as soon as pain allows.

viii. Referral

1. Any individual suspected of having a syndesmotic injury should be referred to a specialist.

ix. Return to Play

1. Conservative treatment requires about 6 weeks before resolution of symptoms.

2. When athlete has full motion, full strength, and a normal functional exam, they can safely return to sports.

3. Bracing and / or taping is recommended. Semi-rigid braces, such as a lace up brace with lateral support or a hinged brace with rigid sides, are typically the best for wearing during sports.

4. Taping can be beneficial also, but tape is proven to loosen with 20 minutes.

x. Complications / Outcomes / Future Risk

1. Low reoccurrence risk.

xi. Prevention

1. No known prevention exists.
FEMALE ATHLETE TRIAD

As defined in the 1997 American College of Sports Medicine (ACSM) Position Stand, the Female Athlete Triad consists of disordered eating, amenorrhea, and osteoporosis. Since that time there has been a plethora of research in this area. Therefore the ACSM redefined the Female Athlete Triad in 2007 as a spectrum of low energy availability (with or without eating disorders), amenorrhea, and osteoporosis. Each clinical condition is now understood to comprise the pathological end of a spectrum of interrelated subclinical conditions between health and disease. These components each pose significant health risks to female athletes and need to be addressed to maximize prevention, early diagnosis, and treatment. Following this review of the Female Athlete Triad will be an in depth discussion on stress fractures.

1. Disordered Eating

When the ACSM redefined the Female Athlete Triad in 2007, the “disordered eating” category changed to become a "spectrum" ranging from optimal energy availability to low energy availability with or without an eating disorder.

a. As there is a spectrum of energy availability, there is also a spectrum of disordered eating which may range inadvertent calorie restriction to anorexia nervosa or bulimia nervosa.

b. "Energy availability" is the amount of energy remaining for all other bodily functions such as cellular maintenance, thermoregulation, growth, and reproduction

i. Energy availability (kcal/kg/lean body mass) = Dietary energy intake – Exercise energy expenditure

c. Energy deficit may be secondary to:

i. Increased total output.
   1. Increased exercise (kcal).

ii. Reduced total energy intake.
   1. Calorie restriction.
2. Fasting.
4. Use of diet pills and laxatives.
   iii. Combination of the above.

d. An athlete can still be at a stable body weight while energy availability is low by compensatory mechanisms to restore energy balance:
   i. Reducing metabolism.
   ii. Decreasing energy available for reproduction.
   iii. Decreasing energy available for cellular maintenance.

2. Menstrual Dysfunction

Functional hypothalamic amenorrhea is the second component of the Female Athlete Triad and is a common occurrence among female athletes. In 2007, the ACSM Position Stand broadened the previous term amenorrhea to include a spectrum of menstrual irregularities ranging from anovulatory eumenorrhea to amenorrhea

a. There are two types of amenorrhea:
   i. Primary amenorrhea: menstruation cycles not starting by age 15 years.
   ii. Secondary amenorrhea: absence of ≥ 3 consecutive menstrual periods after menarche

b. Oligomenorrhea: menstrual cycles with > 35 day intervals.

c. Athletic associated amenorrhea is a complex multi-factorial condition with several possible causes:
   i. Low energy availability—this is the most likely predisposing factor.
      1. Alterations in resting energy expenditure and metabolic hormones (energy conservation) are increasingly evident across the spectrum of menstrual irregularities including luteal-phase defects, anovulation, and amenorrhea in exercising women.
   ii. Extreme weight loss.
   iii. Excessive exercise.
   iv. Physical and emotional stress.
   v. Genetics.

d. Low energy availability results in endocrine and metabolic changes often leading to hypothalamic induced athletic amenorrhea often referred to as functional hypothalamic amenorrhea (FHA) which is the most prevalent cause of amenorrhea in the adolescent age group.
   i. Proposed mechanism:
      1. Low energy availability leads to →
      2. Inhibition of hypothalamic Gonadotropin Releasing Hormone (GnRH) →
      3. Decreased Luteinizing Hormone (LH) secretion from pituitary →
4. Ovarian suppression → decreased estrogen.

3. Low Bone Mineral Density (BMD)
   Disordered eating and menstrual irregularities with estrogen deficiency predispose women to the third component of the female athlete triad, osteoporosis. In 2007, the ACSM Position Stand expanded the female athlete triad definition to include a spectrum ranging from optimal bone health to osteoporosis. Additionally in 2007, the ACSM defined the term "low BMD" as a Z-score between -1.0 and -1.9 and "osteoporosis" as Z-scores < -2.0 with secondary risk factors such as chronic malnutrition, eating disorders, hypogonadism, amenorrhea, glucocorticoid exposure, and previous fracture

   a. Osteoporosis does not occur instantaneously with the onset of amenorrhea; however, there is a direct correlation between length of athletic amenorrhea and reduction in BMD.
   b. Athletes have higher BMD than sedentary premenopausal women
   c. The largest increases in BMD in female athletes occur when impact sports are started 5 years before menarche
   i. Sports with a high amount of "irregular weight bearing" have been shown to increase bone mineral density to a greater degree in both male and females
   d. A significant increase in BMD occurs with pubertal growth. At age 12, young girls have 83% of their total body BMD. Two years after menarche, 95% of their BMD has formed. Bone growth in females ceases approximately at age 20 years.
   e. Although genetics determine 60-80% of peak bone mass, lifestyle choices including diet (energy availability, calcium and vitamin D) and physical activity (athletes have a 5-15% higher BMD than non-athletes) are also predictors of bone accrual during growth.
   f. A study by Ihle and colleagues found that the rate of bone resorption increased and the rate of bone formation declined within 5 days after energy availability was reduced below 30 kcal·kg⁻¹ FFM·d⁻¹ in exercising women.
   g. Estrogen is important in regulation of BMD since it limits bone resorption, stimulates calcitonin, and promotes renal retention of calcium. When energy availability is low enough to suppress estrogen, resorption increases and bone formation is suppressed in dose-response relationships similar to those of insulin, T₃ and IGF-1.

4. Prevalence
   a. A significant number of high school athletes (78%) have one or more components of the female athlete triad.
   b. Disordered Eating:
   i. A study looking at the prevalence of female athlete triad characteristics in a club triathlon team found that 60% of the triathletes were in a calorie deficit.
ii. Another study of 300 female cross-country runners found 19.4% of the runners had previous or current eating disorders.

iii. Yet, another study of more than 300 females between ages 13-39 years found that more elite athletes in leaness sports (46.7%) had clinical eating disorders than athletes in non-leaness sports (19.8%) and controls (21.4%) (P<0.001).

c. Menstrual Dysfunction:
   i. The prevalence of athletic associated amenorrhea has been reported as high as 60-66%, with runners having the highest prevalence.
      1. Amenorrhea is of great concern for runners as it puts them at increased risk for reduced BMD and stress fracture.
      2. Studies have found that 20% of casual runners and 50% of elite runners report irregular menses.
   ii. Nichols et al found that 23.5% of high school athletes had evidence of amenorrhea or oligomenorrhea.
   iii. Hoch et al found a much higher prevalence of 53% in high school athletes, but also found that 21% sedentary high school students had evidence of menstrual dysfunction.

d. Low Bone Mineral Density:
   i. Wiksten-Almstromer and colleagues evaluated the long-term effects on BMD in females diagnosed with menstrual disorders in their adolescence and demonstrated a high frequency of osteopenia/osteoporosis (52%) in adulthood with the strongest predictor of low BMD being a restrictive eating pattern in adolescence.
   ii. In 2006, Beals et al [28] reported 10% of US college athletes from a variety of sports had low BMD defined as Z score = (-1.0 and -1.9).
   iii. In high school athletes Hoch and Nichols [13,26] found similar degrees of low bone mineral density Z score = (-1.0 and -1.9) reporting 16% and 21.8% respectively.

e. Triad*:
   i. Studies of high school, college and elite European athletes have found the prevalence of simultaneously having all three components of the triad to be low.
   ii. Hoch and Nichols had similar findings in high school athletes of 1% and 1.2% respectively.
   iii. Beals et al found 2.6% of college athletes she studied met the criteria for all three components.
   iv. Torstvei found 4.3% of elite Europeans had evidence of all three components.

*Unfortunately, each study used different criteria for each component making comparisons between studies difficult.

5. Risk factors for disordered eating:
a. Body image dissatisfaction.
b. Drive for thinness.
c. Preoccupation with weight.
d. Participation in sports with an aesthetic component.
e. Pressure from parents, coaches, judges, and peers.
f. Low self-esteem.
g. Depression.

a. History
i. Menstrual history.
   1. Age of menarche.
   2. Date of last menstrual period.
   3. Number of periods in the last year.
   4. Number of days between periods.
   5. Symptoms associated with periods.
   6. Any missed periods.
   7. Previous or current hormonal therapy.
ii. Exercise history.
   1. Current/previous sports.
   2. Time spent exercising (hours/week).
   3. Exercise intensity.
   4. Goals for upcoming events.
iii. Nutritional history.
   1. Weight changes, including athlete’s highest and lowest weight.
   2. Ideal weight.
   3. Calcium requirements, actual consumption.
   4. Bingeing or purging behaviors.
   5. Use of diet pills, laxatives, or diuretics.
iv. Injury profile.
   1. Stress fractures, low impact fractures.
   2. Other overuse injuries.
v. Physical and emotional stressors.
vi. Medications and supplements.
vii. Family history.
viii. Symptoms of estrogen deficiency.
   1. Hot flashes, vaginal dryness, dyspareunia.
ix. Symptoms of androgen excess
   1. Acne, excessive hair, striae.
x. Symptoms of pituitary tumor
   1. Galactorrhea, headaches, loss of sense of smell, visual disturbances.
xi. Symptoms of cardiac involvement
   1. Shortness of breath, chest pain, palpitations, dizziness.
b. Physical exam
   i. Height, Weight, Vital Signs, Abnormal habitus
ii. Tanner stage
iii. Dental caries, parotid gland enlargement
iv. Sclera injection
v. Thyroid
vi. Finger callous
vii. Acne, hirsutism
viii. Pelvic exam
c. Differential diagnosis
   i. Polycystic ovary syndrome*
   ii. Hypothalamic amenorrhea*
      1. Stress
      2. Exercise
      3. Nutrition-related
   iii. Hyperprolactinemia*
   iv. Ovarian failure*
   v. Pregnancy/lactation
   vi. Thyroid disease
   vii. Pituitary tumor
   viii. Congenital adrenal hyperplasia
   ix. Genitourinary/anatomic abnormalities
      1. Mullerian agenesis
      2. Complete androgen resistance
   x. Turner’s syndrome

*Top 4 causes of amenorrhea with the exception of pregnancy.
d. Utilization of further testing
   i. Laboratory studies:
      1. Pregnancy test
      2. CBC
      3. CMP
      4. Prolactin level
         a. If prolactin level is > 50 ng/mL rule out microadenoma with MRI.
      5. Follicular stimulating hormone (FSH)
         a. Low or normal FSH levels are seen with hypothalamic amenorrhea and polycystic ovary syndrome.
         b. High FSH levels = lack of gonadal function/ovarian failure
      6. Luteinizing hormone (LH)
         a. Decreases with low energy availability.
         b. High levels are seen with polycystic ovary syndrome.
      7. Free testosterone and Dihydroepiandosterone (DHEA)
         a. Testosterone > DHEA = polycystic ovary syndrome
         b. Testosterone < DHEA = adrenal source
      8. LDL/HDL
   ii. Additional studies:
1. **DEXA scan**
   a. Assessment of bone density is recommended for athletes with a 6-12 month history of disordered eating, amenorrhea or oligomenorrhea.

2. **Pelvic ultrasound**
   a. Evaluate for polycystic ovarian syndrome.

3. **EKG**
   a. Prolong QT interval can be seen with severe eating disorders (i.e. anorexia nervosa).

4. **CT of the adrenal glands**
   a. Evaluate adrenal source.

**e. Treatment**

i. Multidisciplinary approach including the athlete, physician, dietician, athletic trainer/physical therapist, psychologist, psychiatrist, parents and coaches.

ii. **Disordered eating**
   1. Dietician consultation.
      a. Assess energy balance.
      b. Recommendations on achieving a positive balance.
         i. Pre/Post-exercise meal planning.
      c. Education of athlete and parents on energy needs.
   2. Involvement of psychologist and/or psychiatrist for moderate to severe eating disorders.

iii. **Amenorrhea**
   1. Rule out medical cause of amenorrhea.
   2. Correct energy deficits/nutritional issues.
      a. Reduction of training (1 day/week)
      b. Addition of a sports drink (360 kcal/day)
      c. Dietician consultation
         i. Assess negative/positive energy balance
   3. Increase weight and body fat
   4. Involvement of psychologist and/or psychiatrist for stress reduction techniques.

iv. **Osteoporosis**:
   1. Treat the underlying problem.
   2. Optimize nutritional status.
      a. Focus on weight gain/obtaining a positive energy balance.
      b. Calcium requirements: 1500 mg/day
      c. Vitamin D requirements: 2000 IU/day
   3. Establish normal menses.
   4. Avoid excessive exercise.
   5. Add a weight-bearing and resistive exercise program.
   6. May need serial bone densities taken >1.5 years apart to monitor treatment.
7. Return to play
   a. Athletes with the Female Athlete Triad may continue to participate in their sports as long as they have no cardiovascular symptoms.
      i. Chest pain, shortness of breath, syncope/pre-syncope, dizziness, etc.
      1. If these symptoms are present, athlete should be held from play and a cardiology consultation should be placed.

8. Outcomes and future risk
   a. Low bone mineral density may set up the athlete for sub-optimal bone mineral density leading to early onset osteoporosis.
   b. Low bone mineral density may set up the athlete for stress fractures.
   c. Infertility—reversible if exercise related.
   d. Athletic amenorrhea has been associated with reduced endothelium-dependent dilation of the brachial artery.
      i. Endothelial dysfunction is a precursor of early cardiovascular disease.

9. Prevention
   a. Education of physicians, parents, coaches, athletic trainers, and athletes on the 3 components of the triad.
   b. Screen for the female athlete triad.
      i. Preparticipation physical examinations.
      ii. Annual physical examination with primary care physician.
      iii. When athlete is evaluated for other sports related injuries.

10. Promote healthy eating and exercise habits.