Sports Medicine
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Mederic M. Hall

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Preface

When we conceptualized the first edition of *Sports Medicine: Study Guide and Review for Boards* there was a definite need for a comprehensive yet concise and easy-to-read reference and review book. Although there are now a number of excellent sports medicine books on the market, our niche is still necessary. We have received such flattering comments about the first edition, it was clear we needed to keep the format and hold our place in the sports medicine review category of thorough, yet succinct, texts.

For this second edition, we have again kept true to the sports medicine board exam content outline in order to cover all topics testable on the exam. Even the length of each chapter is designed according to how much that topic is weighted on the exam. We’ve also kept the easy-to-read outline format as well as the reference lists at the end of each chapter, which can be used for more in-depth study of the topics.

The general format of the original edition has also been retained, dividing the book into three primary sections: (I) General Topics; (II) Health Promotion and Injury Prevention; and (III) Diagnosis and Treatment of Sports Injuries and Conditions. The third section is divided into the following three subsections: (1) Musculoskeletal Injuries and Conditions; (2) Medical, Neurological, and Psychological Conditions; and (3) Special Populations.

Besides updating every chapter, we have added a couple of new elements to the second edition. The first is an additional (fourth) subsection entitled “Hot Topics in Sports Medicine.” The material in this subsection (chapters on “Sports Ultrasound” and “Regenerative Medicine”) is not testable information on the board exam yet; however, it is certainly information pertinent to every learner and practitioner of sports medicine.

The second new element is a practice test encompassing over 250 questions covering the breadth of sports medicine with an answer key including references to the appropriate chapters. This is a great addition for the reader who is studying for the board exam as well as anyone wanting to test their knowledge of sports medicine.

We are very proud of the final product and believe it provides the reader with an exceptional resource covering the entire breadth of sports medicine. The book is meant to be used as a study guide for primary care sports medicine physicians (family medicine, emergency medicine, internal medicine, pediatrics, and physical medicine and rehabilitation) and orthopedic sports medicine physicians preparing to take the sports medicine board examination for initial certification or recertification. It also can serve as a sports medicine reference for other medical professionals such as athletic trainers, physical therapists, physicians in training (ie, interns, residents, and fellows), and other physicians interested in sports medicine.

We, once again, would like to thank all the authors who contributed their expertise and time. Without their commitment to excellence, our hopes of enhancing this second edition and creating the best sports medicine review book on the market would have gone unrealized. We also thank our publisher, Beth Barry at Demos Medical Publishing, for her belief in our vision and continued support throughout the process. And, finally, we thank the readers of the first edition, including our own residents and fellows, whose comments and critiques have helped guide us to this second edition.

Mark A. Harrast, MD

Jonathan T. Finnoff, DO, FACSM
Cervical Spine Injuries and Conditions

Abby Cheng and Monica E. Rho

I. Structure and Function

A. Anatomy

1. Vertebral bodies (VBs)
   a. Seven cervical vertebrae, lordotic curve
   b. C1—Atlas
   c. C2—Axis—Transverse process palpable posterior to mandible angle
      i. Dens/odontoid process articulates with anterior arch of atlas
   d. C3—Posterior to hyoid bone
   e. C4, C5—Posterior to thyroid cartilage
   f. C6—Posterior to first cricoid ring
   g. C7—Most prominent cervical spinous process on palpation

2. Spinal canal (SC)
   a. Individual variance in size
   b. Normal lower anterior-posterior (AP) cervical canal diameter: 14 to 22 mm on lateral radiograph
   c. Narrowest during neck extension

3. Spinal nerve roots (Figure 18.1)
   a. Eight pairs of exiting spinal nerves

4. Intervertebral disks
   a. Annulus fibrosus—Outer fibrous ring
   b. Nucleus pulposus—Inner gelatinous material encased by the annulus fibrosus

5. Joints
   a. Zygapophyseal joints, also called Z-joints or facet joints
      i. Synovial joints
      ii. Oriented 45° to the coronal plane (in the cervical spine [C-spine])
      iii. Restrain forward translation
   b. Uncovertebral joints (of Luschka)
      i. Not true synovial joints
      ii. Arise from the posterolateral margins of the VBs
      iii. Present on C3–C7 only

6. Ligaments (Figure 18.2)
   a. Alar—Paired ligaments, limit head rotation
      i. From foramen magnum of skull to dens of C2
   b. Anterior atlanto-axial—Limits neck extension
      i. From C1 to C2 only
Figure 18.1  Anatomy of exiting spinal nerve roots of the cervical spine.

Figure 18.2  Sagittal view of the cervical spine and its ligaments, demonstrating the three-column concept of spine stability.
III: DIAGNOSIS AND TREATMENT OF SPORTS INJURIES AND CONDITIONS

7. Muscles

a. Splenius capitis, splenius cervicis, spinalis cervicis
   i. Head/neck extension
   ii. Lateral flexion and rotation to the same side of unilateral contraction

b. Semispinalis capitis, semispinalis cervicis
   i. Head/neck extension
   ii. Lateral flexion and rotation to the opposite side of unilateral contraction

c. Trapezius
   i. Scapular adduction and rotation
   ii. Head/neck extension
   iii. Head/neck lateral flexion to the same side of unilateral contraction
   iv. Head/neck rotation to the opposite side of unilateral contraction

d. Sternocleidomastoid
   i. Head/neck rotation to the opposite side of unilateral contraction
   ii. Head/neck flexion

e. Scalenes
   i. Anterior and middle scalenes: First rib elevation
   ii. Posterior scalene: Second rib elevation
   iii. Neck lateral flexion to the same side of unilateral contraction

B. Kinesiology

1. Greatest proportion of spine motion occurs in the C-spine

2. Flexion/extension
   a. 80° to 90° flexion, 70° to 85° extension from neutral
   b. Motion initiated in C6-C7
   c. Motion greatest at C5-C6
      i. 25% of all cervical flexion/extension
   d. Primary motion of atlanto-occipital joint (15°–20°)

3. Rotation
   a. 90° to each side
b. Greatest at C1-C2
   i. Primary motion of atlanto-axial joint
   ii. 45° to each side
c. Amount of rotation at each joint level decreases from superior to inferior

4. Lateral flexion (side bending)
   a. 40° to each side
   b. Greatest at C3-C4 and C4-C5

II. Evaluation of Neck Pain

A. History
   1. Mechanism of injury
   2. Pain intensity, quality, location, and duration
      a. Weakness, numbness, or tingling?
      b. Presence of radicular symptoms?
      c. How many limbs involved?
   3. Prior injury to the neck
   4. Known cervical structural abnormality
   5. Bowel and bladder abnormalities

B. Physical exam
   1. Inspection
      a. Posture
         i. Loss of normal cervical lordosis
         ii. Exaggerated thoracic kyphosis
         iii. Scoliosis
         iv. Rounded shoulders
         v. Depressed shoulder girdle (usually on dominant side)
      b. Asymmetry of head position
   2. Palpation
      a. Tenderness over bony landmarks (ie, occiput, spinous/transverse processes) and paraspinal muscles
      b. Identify muscle spasms and trigger points
   3. Active range of motion (AROM)
      a. Avoid passive ROM (PROM) if concern for unstable spine from ligament injury or fracture
   4. Manual muscle testing, sensation, and reflexes
      a. See Tables 18.1 and 18.2

Table 18.1 Manual Muscle Testing of the Cervical Spine

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<tr>
<td>Extensor digitorum communis</td>
<td>C7, C8</td>
<td>Finger extension</td>
</tr>
<tr>
<td>Flexor carpi ulnaris</td>
<td>C7, C8, T1</td>
<td>Wrist flexion, ulnar deviation</td>
</tr>
<tr>
<td>Flexor digitorum profundus</td>
<td>C8, T1</td>
<td>Distal interphalangeal finger flexion</td>
</tr>
</tbody>
</table>

Note: The bolded spinal nerve is the dominant contributor to the muscle contraction.
Table 18.2 Sensory Innervation and Reflexes Organized by Cervical Nerve Root

<table>
<thead>
<tr>
<th>Root Level</th>
<th>Sensory Innervation</th>
<th>Reflexes</th>
</tr>
</thead>
<tbody>
<tr>
<td>C5</td>
<td>Lateral arm</td>
<td>Biceps, brachioradialis</td>
</tr>
<tr>
<td>C6</td>
<td>Lateral forearm, thumb, index finger</td>
<td>Biceps, brachioradialis</td>
</tr>
<tr>
<td>C7</td>
<td>Dorsal forearm, dorsal and volar index/middle/ring fingers</td>
<td>Triceps</td>
</tr>
<tr>
<td>C8</td>
<td>Ring/little finger, medial forearm</td>
<td>–</td>
</tr>
<tr>
<td>T1</td>
<td>Medial arm</td>
<td>–</td>
</tr>
</tbody>
</table>

5. Special tests
   a. Suggest nerve root pathology
      i. Spurling’s neck compression test—Reproduces radicular symptoms
      ii. Neck distraction test—Relieves radicular symptoms (do not perform if suspicious of cervical instability)
      iii. Neurodynamic (dural tension) tests—Reproduce radicular symptoms
           (A) Median, ulnar, and radial nerve biases
   b. Suggests spinal cord pathology
      i. Lhermitte’s sign—Electrical “shock” down the spine during neck flexion
   c. Consider performing special tests to evaluate for thoracic outlet syndrome

C. Diagnostics
   1. Plain radiographs (Figure 18.3)
      a. Evaluate for fracture, dislocation, instability, or foraminal stenosis
      b. Views: AP, lateral, oblique, odontoid (open mouth), flexion/extension
         i. Flexion/extension view used to assess for ligamentous injury/instability
         ii. Oblique views assess for foraminal stenosis

Figure 18.3 Anterior-posterior (AP)/lateral cervical spine radiographs. The lateral cervical radiograph (B) demonstrates loss of the normal cervical lordosis.
2. CT
   a. Evaluates bony structures in more detail (may be necessary to assess injuries to C1 and C2)
3. MRI
   a. Evaluates soft tissues: intervertebral disks, nerves, muscles, ligaments, and spinal cord
   b. Evaluates spaces: foramina, central canal, and functional reserve volume (cerebrospinal fluid [CSF] around spinal cord)
4. Electodiagnostic testing (nerve conduction studies and electromyography [EMG])
   a. Assesses peripheral nerve conduction as well as nerve and muscle function
   b. Localizes injury to the nerve root, peripheral nerve, neuromuscular junction, or muscle level
   c. Often will not be abnormal until 3 to 4 weeks after initial injury; therefore, not to be used for acute assessment of injury

III. C-Spine Disorders
   A. Stingers and burners
      1. Definition
         a. Transient neurological event lasting seconds to minutes
         b. Characterized by pain, burning, and/or paresthesias in a single upper limb, sometimes accompanied by weakness
         c. Upper trunk of brachial plexus (C5, C6) most commonly involved
      2. Epidemiology
         a. Most common in contact sports that involve tackling; most common C-spine injury in football (most common in linemen and defensive ends)
         b. Estimated that >50% of collegiate football players sustain a stinger each year, and 87% have recurrence
      3. Mechanisms of injury
         a. Traction injury to the brachial plexus or nerve roots during forcible neck lateral flexion as the contralateral shoulder is depressed
            i. Potentially more common in young athletes due to less developed neck musculature
         b. Compression to nerve roots in their foramina during combined neck extension and lateral neck flexion
            i. Potentially more common in more experienced athletes with higher likelihood of preexisting degenerative C-spine changes
            ii. Neck extension narrows the cervical neural foramina, and neck rotation can further narrow this space
         c. Direct trauma to the brachial plexus in the supraclavicular fossa at Erb’s point
      4. Diagnosis
         a. Exam
            i. Potential weakness in the deltoid, biceps, and infraspinatus muscles if upper trunk of the brachial plexus is affected
            ii. Spurling’s test or Tinel’s test at Erb’s point may reproduce radicular symptoms
            iii. Sensation and reflexes in upper extremities can often be normal
            iv. C-spine ROM will help determine return to play (RTP)
         b. Imaging (rarely needed)
            i. Pursue if persistent signs/symptoms (>24–36 hours) or recurrent stingers
            ii. Radiographs
               (A) To rule out bony injury if presenting with severe neck pain with focal tenderness and limited C-spine ROM
               (B) Limited diagnostic value for stingers, but may reveal structural abnormalities that predisposed to the injury
iii. MRI
(A) C-spine—Evaluate for contributing foraminal stenosis or disk herniation that predisposes someone for a stinger
(B) Brachial plexus—Rarely needed but can evaluate for a lesion compressing the plexus

iv. Electrodiagnostic studies
(A) Consider only in those with persistent neurologic symptoms
(B) Highest diagnostic yield ≥3 weeks after injury
(C) Can help localize the lesion to nerve root versus brachial plexus
(D) Poor prognostic findings
   (1) Positive sharp waves and fibrillations—Indicate axonal injury, will delay RTP
   (E) Good prognostic findings
      (1) Absent positive sharp waves and fibrillation potentials
      (2) Sensory nerve action potential preservation, especially in the upper trunk, including lateral antebrachial cutaneous, median sensory to index finger, and superficial radial sensory nerves

5. Treatment
   a. Remove from sport, supportive therapy with nonsteroidal anti-inflammatory drugs (NSAIDs) and analgesics, consider physical therapy for athletes with persistent symptoms
   b. Symptom duration is often brief and self-limited; however frequent re-examination is recommended
   c. Ensure proper tackling technique
   d. Consider neck-strengthening program in youth athletes

6. RTP guidelines
   a. RTP—Only with full cervical ROM, full upper extremity strength, and no neurological symptoms
      i. Can RTP in same game if symptoms resolve within 15 minutes and no previous stingers that season
   b. Relative contraindication (CI)—Symptoms >24 hours, or ≥3 previous stingers/burners
      i. If second stinger in a season, do not RTP in the same game
      ii. If third stinger in a season, should have imaging evaluation before RTP and consider sitting out the rest of the season
   c. Absolute CI—Continued neck pain with decreased ROM, prolonged neurological deficit (even if eventually resolves), or evidence of instability

B. Cervical cord neurapraxia (CCN) and transient quadriparesis (TQ)
1. Definition
   a. Transient loss of motor function and/or dysesthesias in more than one limb
   b. Self-limited—Symptoms usually last <15 minutes, but have been reported lasting up to 2 days in adults and 5 days in children
   c. “Burning hands syndrome”—Typical initial presentation of CCN, consistent with central cord syndrome
2. Epidemiology
   a. Rare, but most common in high-velocity sports like football, rugby, and hockey
   b. Incidence in National Collegiate Athletic Association (NCAA) football: 7.3 per 10,000
   c. Recurrence rate after RTP in adults: 56%
      i. No recurrence in children
   d. Associated with cervical spinal stenosis in adults but not in children
3. Mechanisms of injury
   a. Cervical hyperextension
18. CERVICAL SPINE INJURIES AND CONDITIONS

i. Creates functional cervical spinal stenosis

ii. Preexisting spinal stenosis is a significant risk factor for permanent spinal cord injury

b. Axial loading

i. Occurs when the C-spine is slightly flexed or has loss of the normal cervical lordosis (see Figure 18.3)

ii. Fracture-dislocation can occur

iii. In the setting of a central disk herniation, there is risk for transient compression of the anterior cord and anterior spinal artery

4. Diagnosis

a. Exam

i. Weakness in a myotomal distribution

ii. Sensory loss in a dermatomal distribution

iii. Positive Lhermitte’s sign

b. Radiographs

i. Evaluate for acute changes (fractures, dislocations, instability)

ii. Torg ratio (see Cervical Stenosis section in the following text)

c. MRI (Figure 18.4)

i. Mandatory to evaluate for cervical stenosis and/or spinal cord injury/sequelae

ii. Used to assess the cervical SC “functional reserve” (ie, the presence of CSF around the spinal cord)

5. Treatment

a. Removal from sport, symptoms are often self-limited

b. Initially treat with full C-spine precautions until a more serious spinal cord injury is ruled out (see Chapter 19)

6. RTP guidelines—Athlete should never RTP on the same day

a. RTP—Cleared if first episode, full cervical ROM, full upper extremity strength, no residual neurological symptoms, stable C-spine, and no cervical stenosis on MRI

b. Relative CI—One prior episode, mild to moderate cervical stenosis

c. Absolute CI—More than one prior episode, persistent neurological deficit, or MRI evidence of cord edema, ligamentous instability, and/or severe cervical stenosis

d. Controversy regarding RTP in the setting of cervical stenosis

i. Before RTP, evaluate and correct the athlete’s sport-specific technique to minimize the risk of recurrent injury

ii. Can counsel the athlete and parents to consider transitioning to a noncollision, low-risk sport

Figure 18.4 Axial MRI images of cervical spine. (A) MRI T2-weighted axial image of C3-C4 decreased functional reserve or space available for cord. (B) MRI T2-weighted axial image of C3-C4 normal functional reserve or space available around the cord.
C. Cervical stenosis

1. Definition
   a. Narrowing of the central SC
   b. May be congenital or caused by degenerative changes such as osteophytes, disk herniation, and/or ligamentum flavum hypertrophy
   c. “Functional cervical stenosis”—canal narrowing resulting in the loss of CSF cushion around the spinal cord (ie, loss of “functional reserve”)
      i. Functional stenosis is the most important clinical indicator of increased risk due to stenosis

2. Diagnosis
   a. Presentation
      i. Can be asymptomatic
      ii. Symptomatic athletes will have neck pain and potentially radicular symptoms in one or both arms
   b. Lateral radiograph of C-spine
      i. Sagittal canal diameter
         (A) Diameter <14 mm
         (B) Measurement can be affected by positioning and target distance during imaging
      ii. Torg ratio—Ratio of SC diameter to VB diameter (Figure 18.5)
         (A) Ratio <0.8 consistent with stenosis
         (B) Very poor positive predictive value in football players because they often have large VBs
         (C) Historical tool; replaced by MRI

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**Figure 18.5** Lateral radiograph of the cervical spine demonstrating the Torg ratio. The Torg ratio is calculated by measuring the spinal canal (SC) and the vertebral body (VB) on a lateral radiograph of the cervical spine. SC is measured from the midpoint of the posterior aspect of the VB to the closest point of the corresponding spinolaminar line. VB is the anteroposterior width of the VB at its superoinferior midpoint. A Torg ratio <0.8 is considered to be consistent with cervical spinal stenosis.
c. MRI of C-spine (see Figure 18.4)
   i. Absence of “functional reserve”—lack of CSF signal surrounding the spinal cord—
   diagnostic for functional stenosis

3. Treatment
   a. Physical therapy to work on C-spine ROM and strengthening
   b. Stenosis in the setting of persistent neurologic symptoms or spinal cord edema may
   require surgical intervention

4. RTP guidelines
   a. Controversial
   b. Presence of asymptomatic functional cervical stenosis increases the risk of permanent
   neurological damage after C-spine trauma
      i. Often, previously asymptomatic stenosis is diagnosed retrospectively after the ath-
      lete sustains a traumatic C-spine injury (eg, CCN or spinal cord injury)
   c. If incidental finding and no history of neck injury
      i. May RTP, or consider having a discussion with the athlete and/or parents about
      increased risk of serious C-spine injury with continued involvement in collision/  
      contact sports. Consider a change in sport.
   d. If symptomatic and/or the athlete sustained a traumatic C-spine injury
      i. Generally considered a CI to RTP

D. Disk pathology
1. Definition
   a. Disk degeneration—Dessication, fibrosis, narrowing of the disk space, or diffuse
   bulging of the annulus
   b. Annular fissure—Separation of annual fibers
   c. Herniation—Displacement of disk material beyond the intervertebral disk space
      i. Protrusion
      ii. Extrusion
      iii. Sequestration
   d. Does not necessarily cause pain or other symptoms
   e. Discogenic pain—Pain directly due to disk injury (not due to secondary cervical
   nerve root irritation)

2. Epidemiology
   a. Up to 30% to 40% of asymptomatic adults have MRI evidence of disk degeneration
   b. Most common levels: C5-C6 and C6-C7

3. Mechanisms of injury
   a. Disk degeneration is a normal part of aging but can be accelerated by trauma

4. Diagnosis of discogenic pain
   a. History
      i. Axial neck pain, often worse with prolonged, fixed positions
      ii. Can be associated with muscle spasms/tightness
      iii. Pain can refer to the arms if there is associated radiculitis/radiculopathy
   b. Exam
      i. No neurological deficits if nerve roots are not compressed
      ii. Pain with cervical spinal ROM
   c. Imaging—Can be helpful, but does not identify a pain generator since many asympto-
   matic people have disk pathology
      i. Radiographs can demonstrate loss of disk height that is associated with
      degeneration
      ii. MRI can identify disk pathology

5. Treatment
   a. Only requires treatment if symptomatic
   b. Options include NSAIDs, analgesics, and physical therapy
6. RTP guidelines
   a. RTP—Full, painless cervical ROM
   b. Absolute CI—Herniation causing radicular symptoms, neurologic deficits, and/or functional cervical stenosis

E. Radiculitis/radiculopathy
1. Definition
   a. Pain and paresthesias typically starting at the neck and radiating down one arm due to irritation of the spinal nerve root (can also radiate to shoulder/scapula)
      i. Radiculitis—No associated strength or reflex deficits
      ii. Radiculopathy—Associated strength, reflex, and/or sensory deficits
   b. Most commonly due to disk herniation or foraminal stenosis
2. Epidemiology
   a. Most common levels: C6 and C7
   b. Most commonly caused by spondylosis (70%) or herniated nucleus pulposus (20%)
3. Diagnosis
   a. Exam
      i. Limited C-spine ROM
      ii. Positive Spurling’s maneuver and/or neural tension signs
      iii. Relief when symptomatic arm placed over head (Bakody’s sign)
   b. Imaging
      i. Radiographs
         (A) Evaluate for contributing disk space narrowing and/or vertebral endplate osteophytes
      ii. MRI
         (A) Evaluate for contributing disk bulge/herniation, foraminal stenosis, and/or central stenosis
   c. Electrodagnostic studies
      i. Can help distinguish between radiculopathy (abnormal findings) and radiculitis (normal findings)
      ii. Can localize specific nerve root affected
      iii. Can rule out other etiologies of symptoms (eg, plexopathy, peripheral nerve entrapment, etc.)
4. Treatment
   a. Remove from sport if there is persistent neurologic deficit
   b. NSAIDs and neuropathic pain medications
   c. Consider oral corticosteroids and/or short course of opioids (for severe pain)
   d. Physical therapy—Postural correction, cervical traction, and/or McKenzie method of Mechanical Diagnosis and Therapy
   e. Fluoroscopically guided cervical epidural steroid injection—In cases of severe pain that limits rehabilitation progress
   f. Surgery—If progressive neurologic deficit or signs of myelopathy (eg, weakness, poor coordination, gait impairment, bowel/bladder changes, or upper motor neuron [UMN] reflex changes), or if the athlete has persistent disability due to pain
5. RTP guidelines
   a. RTP—Pain-free full cervical ROM, full upper extremity strength, no neurological deficits
   b. Relative CI for contact sports
      i. Single-level or two-level cervical fusion
      ii. In professional athletes, mild residual pain/numbness/weakness may persist despite proper management. Okay to RTP if stable
   c. Absolute CI for contact sports
i. Multilevel fusion, cervical laminectomy, or C1-C2 fusion
ii. Cervical myelopathy
iii. Significant residual pain or weakness

F. Spear tackler’s spine
1. Definition (must meet all four criteria)
   a. Cervical canal stenosis
   b. Persistent straightening or reversal of the normal cervical lordotic curve on an erect lateral radiograph in neutral position
   c. Evidence of minor posttraumatic abnormalities on radiographs of the C-spine
   d. Use of the “spear tackling” technique—Tackling or blocking in a head-down, neckflexed position (see Section IV. Safety Considerations)
2. Mechanism of injury
   a. Head-down “spearing” position results in slight neck flexion and loss of the normal C-spine lordotic curve. Loss of the lordotic curve decreases the spine’s shock-absorbing capability
   b. Injury occurs during axial load to the C-spine during intentional or unintentional head-down contact
   c. Greatly increases an athlete’s risk of CCN, TQ, and serious irreversible spinal cord injury when the C-spine is exposed to axial load
3. Treatment
   a. Can attempt restoration of the normal cervical lordosis with therapy and/or manipulation
   b. Can counsel the athlete on proper tackling technique
4. RTP guidelines for contact sports
   a. Controversial
   b. Usually considered an absolute CI, even if an incidental finding
   c. Some allow RTP if
      i. Normal cervical lordosis is restored
      ii. Athlete refrains from spearing technique

G. Fractures
1. Epidemiology
   a. In sports, most commonly in lower vertebrae (C4–C7)
   b. In the National Football League (NFL), fractures most commonly occur during blocking and tackling
   c. High-risk positions for fractures, from greatest to least: Offensive linemen, defensive backs, defensive linemen, linebackers
2. Diagnosis
   a. History
      i. Elicit mechanism of injury (eg, flexion, flexion-rotation, extension, compression) to narrow the differential of possible fractures
   b. Imaging
      i. Radiographs
         (A) National Emergency X-Radiography Utilization Study (NEXUS) low-risk criteria—Radiographs not necessary if all the following criteria are met (negative predictive value 99.9%)
            i. No posterior midline cervical tenderness
            ii. No neurologic deficits
            iii. Normal alertness
            iv. Not intoxicated
            v. No painful distracting injuries
      ii. CT/MRI
3. Treatment
   a. Depends on stability of the spine
   b. Rule out concomitant ligamentous injury that could contribute to instability
   c. Three-column concept of spine stability: Spine is unstable if at least two columns disrupted (see Figure 18.2)
      i. Anterior column
         (A) ALL, anterior two-thirds of VBs
      ii. Middle column
         (A) Posterior one-third of VBs, PLL
      iii. Posterior column
         (A) Pedicles, transverse processes, facet joints, laminae, spinous processes
   d. Treatment for stable fractures
      i. C-collar for immobilization until pain-free
      ii. Once pain-free, repeat flexion/extension lateral radiographs to confirm spinal stability
   e. Treatment for unstable fractures
      i. Reduce quickly, stabilize, surgical referral
4. Fracture types
   a. Stable fractures
      i. Spinous process fracture
         (A) May occur after strong contraction of the trapezius or rhomboid muscles or after hyperflexion leading to avulsion
         (B) C7 spinous process fracture—Clay-shoveler’s fracture
      ii. C1 posterior arch fracture—Due to hyperextension
      iii. Chip or minor wedge/compression fracture—Due to hyperflexion
      iv. Unilateral laminar fracture—Due to flexion, distraction, and rotation
         (A) Isolated facet dislocation may occur, as well
   b. Less stable fractures
      i. Bilateral C2 pars fracture (Hangman’s)—Due to hyperextension
         (A) Most common C-spine fracture
         (B) If not significantly displaced, requires C-collar only
         (C) Unstable but rarely associated with spinal cord injury because AP diameter of SC is large at this level, and fractured pedicles allow decompression
         (D) Unstable when associated with C2 facet dislocation
      ii. Burst fracture—Due to compression
         (A) Anterior and middle columns disrupted. Variable degree of posterior column disruption
         (B) Usually stable, but posteriorly displaced fracture fragments may impinge on the spinal cord and cause anterior cord syndrome
         (C) Unstable if
            i. >50% loss of vertebral height
            ii. Multiple adjacent compression fractures
      iii. C1 burst fracture (Jefferson)
         (A) Stable if displacement of lateral masses <6.9 mm (transverse ligament still intact)
            i. Treat with rigid brace
         (B) Unstable if displacement of lateral masses >6.9 mm (indicating ligament disruption)
            i. Requires reduction and halo
      iv. Odontoid fracture—Due to forceful flexion or extension; evaluate with open mouth view on radiographs (see Figure 18.6)
18. CERVICAL SPINE INJURIES AND CONDITIONS

(A) Type I—Tip avulsion above transverse ligament
   i. Rare, stable

(B) Type II—Fracture through base of the dens
   i. Most common, unstable

(C) Type III—Fracture through axis body
   i. Can be unstable, but heals better than type II

C. Unstable fractures (and dislocations)
   i. Flexion teardrop
      (A) Severe flexion and compression causes one VB to collide with the VB below,
         leading to anterior displacement of a wedge-shaped “teardrop” fragment of
         the anteroinferior portion of the superior vertebra
   ii. Extension teardrop
       (A) Hyperextension mechanism, causing displaced avulsion of anteroinferior
           bony fragment
       (B) Common after diving, tends to occur at lower cervical levels
       (C) May be associated with central cord syndrome due to ligamentum flavum
           buckling
       (D) Stable in flexion but highly unstable in extension
   iii. Atlanto-occipital dislocation—Due to flexion or flexion-rotation
   iv. Rotatory atlanto-axial dislocation—Due to hyperflexion
   v. Bilateral facet dislocation—Extreme form of subluxation resulting in ligamentous
      disruption

5. RTP guidelines
   a. RTP—Wait 8 to 10 weeks for bone healing. Full ROM, no tenderness, no neurologic
      deficits
   b. Relative CI—With a relative CI, need radiographic evidence of healing/fusion before
      RTP
      i. Healed posterior element fracture (excluding spinous process fracture, which is
         always okay for RTP)
      ii. Healed, minimally displaced compression fracture
      iii. Healed upper cervical fracture (nondisplaced Jefferson, type I or II odontoid, or
           C2 lateral mass fracture)
III: DIAGNOSIS AND TREATMENT OF SPORTS INJURIES AND CONDITIONS

C. Absolute CI
   i. Healed fracture with permanent cervical kyphosis, coronal plane abnormality, or cord encroachment
   ii. Atlanto-occipital fusion or atlanto-axial rotatory fixation
   iii. Persistent instability or neurologic deficit

D. After unstable fractures, caution against RTP, even if healed after surgical or halo stabilization
   i. Persistent altered surrounding biomechanics and loss of normal ROM increases athlete’s risk of future injury

H. Sprains and strains
1. Definition
   a. Sprain
      i. Ligamentous stretch injury affecting spinal ligaments or capsular structures of facet joints
   b. Strain
      i. Muscular stretch injury, usually occurring at the musculotendinous junction
      ii. Commonly affects the trapezius, rhomboids, levator scapulae, scalenes, erector spinae, and sternocleidomastoid muscles

2. Diagnosis
   a. History
      i. Often presents with localized neck/upper back pain, tenderness, weakness, and/or limited ROM without neurologic deficits
      ii. Suboptimal posture and cumulative microtrauma due to muscle imbalance can predispose one to this injury
   b. Exam
      i. Do not test PROM due to the risk for instability from ligamentous laxity
      ii. Test active ROM or gentle isometric contraction to assess for laxity
   c. Imaging
      i. Radiographs
         (A) Indicated if a history of trauma, point tenderness over a bony prominence, limited ROM, or prior cervical surgery
         (B) Rule out coexisting fracture, subluxation, or ligamentous instability
      ii. Radiographic evidence of ligamentous laxity
         (A) Criteria for diagnosis
            i. AP displacement >3.5mm or 20%, or >11° rotation
            ii. Sagittal plane rotation >20°
         (B) Limitations in diagnosis by radiograph
            i. CI to PROM for adequate flexion/extension views
            ii. Active ROM may be limited by pain and spasm
               1. 30% to 70% of films show inadequate gross motion to determine instability
            iii. May require MRI/CT confirmation
            iv. Youth often have physiologic hypermobility of C2–C4 (pseudo-subluxation)

3. Treatment
   a. Remove from sport if the athlete has restricted ROM, immobilize C-spine if concern for ligamentous injury and/or instability
   b. Consider NSAIDs and modalities
   c. If no concomitant fracture/dislocation/subluxation
      i. Consider C-collar until acute symptoms subside (7–10 days), then proceed to ROM and isometric strengthening exercises
   d. If concomitant subluxation
1. Wear hard C-collar for 2 to 4 weeks
   ii. If no evidence of progression on repeat flexion/extension radiographs and if symptoms have resolved, then unlikely a significant injury occurred and RTP is acceptable

4. RTP guidelines
   a. RTP—Pain-free full cervical ROM and upper extremity strength
   b. Absolute CI—Cervical instability such as asymptomatic ligamentous laxity

I. Whiplash
   1. Definition
      a. Whiplash—Acceleration–deceleration mechanism of energy transfer to the neck
      b. Whiplash-associated disorders (WADs)—Clinical manifestations of bony or soft tissue injuries sustained due to whiplash
         i. Can include the development of alterations in motor function, chronic widespread sensory hypersensitivity, posttraumatic stress reactions, and biopsychosocial dysfunction
   2. Epidemiology
      a. In sports, most frequently documented in indoor soccer, basketball, wrestling, and diving
   3. Mechanism of injury
      a. Commonly due to sudden contact from behind, resulting in cervical hyperextension, or flexion/extension
      b. Injury thought due to abnormal facet posterior translation, rather than physiologic sliding
   4. Diagnosis
      a. History
         i. Neck pain/stiffness, possibly delayed 24 to 48 hours after injury
         ii. Can be associated with decreased ROM, headaches, dizziness, and/or blurred vision lasting <24 hours
         iii. Pain/stiffness may last a few days/weeks
      b. Exam
         i. Usually no neurological deficits, but can present with transient vision changes, Horner’s syndrome, or cranial nerve palsies
   5. Treatment
      a. Rest, ice, compression, elevation (RICE) therapy, consider NSAIDs
      b. Early, gentle, pain-free ROM promotes faster recovery than immobilization in soft C-collar
      c. Can consider deep tissue massage or manipulation, if no concern for fracture, dislocation, or instability
      d. Lower risk of developing WADs if: <35 years old, low symptom severity immediately after injury, no baseline headache/neck pain
   6. RTP guidelines
      a. RTP—Full pain-free ROM and strength

J. Congenital anomalies
   1. Atlanto-axial instability from Down syndrome
      a. Definition
         i. Increased atlanto-axial laxity due to abnormalities of the transverse ligament of the atlas, vertebral shapes, or both
      b. Epidemiology
         i. Affects 10%–20% of people with Down syndrome
         ii. Symptomatic in up to 1%–2% of people with Down syndrome
      c. Diagnosis
         i. History/exam
(A) Neck pain, decreased ROM, torticollis, UMN signs

ii. Imaging
(A) Screening radiographs (flexion/extension)—Increased atlanto-odontoid distance (upper limit of normal is 3–4 mm)
(B) MRI if symptomatic
(C) With time, radiographic abnormalities can develop or resolve

d. Treatment
i. If symptomatic, recommend surgical stabilization
e. RTP guidelines
i. Preparticipation screening is mandatory for Special Olympics and recommended before beginning sports in any context
(A) C-spine radiographs—If atlanto-odontoid distance >3.5 mm in adults or 4 to 5 mm in children
   i. Restricted from contact events (eg, floor hockey, soccer, high jump, alpine skiing, squat lifting, soccer, gymnastics, diving, swimming using flip turns/diving starts/butterfly stroke)
   ii. Can still participate in noncontact sports such as cross-country skiing, distance running, bowling, and so on
(B) Controversial because of unclear benefit of screening in preventing spinal cord injuries
   ii. CI—Symptomatic instability

2. Klippel-Feil syndrome
a. Definition
i. Congenital fusion of adjacent cervical vertebrae at one or more levels, resulting in increased risk of spinal cord injury from minor trauma (similar to effects of surgical cervical fusion)

   ii. Commonly associated with a low hairline

   iii. Associated with increased risk for cardiac, pulmonary, urologic, or other malformations

b. Epidemiology
   i. Incidence—1:40,000 with a female predominance
c. Diagnosis
   i. Radiographs, including flexion/extension views
d. Treatment
   i. Activity modification and/or bracing, if symptomatic
   ii. Surgery—If progressive instability or neurologic compromise
e. RTP guidelines
   i. Okay to play if single-level fusion not involving C0–C1 articulation
      (A) Monitor for adjacent-level instability with periodic radiographs
   ii. Absolute CI
      (A) Symptomatic
      (B) Multilevel Klippel-Feil anomaly
      (C) Atlanto-occipital fusion
      (D) Brain stem signs, for example, Arnold-Chiari malformation or basilar invagination

K. Vascular injuries
1. Definition
   a. Blunt cerebrovascular injuries (BCVIs) usually involve carotid or vertebral artery occlusions or dissections

2. Epidemiology
   a. Incidence of traumatic BCVIs is unclear because unilateral injuries can be asymptomatic and underdiagnosed if there is adequate contralateral blood supply
b. More common in athletes who develop posttraumatic neurological deficits
c. Incidence of vertebral artery thrombosis with concomitant cervical fracture: 13% to 24%
d. Rate of ischemic stroke in untreated patients: as high as 14% to 54%
e. Stenotic lesions eventually resolve in >90% of cases, and 67% of occluded vessels recanalize

3. Mechanism of injury
a. Usually occurs due to collisions with other players or the environment, but cases have been reported after strenuous weight lifting and repeated Valsalva maneuvers
b. Can cause brain ischemia or stroke due to vessel occlusion or thrombus embolism

4. Diagnosis
a. History
i. Cervical injuries associated with higher incidence of BCVIs—Facet dislocation, fractures through the transverse foramen, C1–C3 fractures, and craniocervical distraction
ii. Suspect a BCVI if an athlete presents with delayed-onset, acute, focal neurologic deficits (especially involving the head and cranial nerves) even after relatively minor neck trauma
b. Imaging
i. CT or MR angiogram of the neck
   (A) Consider as part of the initial imaging protocol after polytrauma
   (B) Strongly consider if the athlete has a facet or transverse foramen dislocation
ii. Denver radiological grading scale of BCVIs
   (A) Grade I—Irregularity of vessel wall or dissection/intramural hematoma with <25% stenosis
   (B) Grade II—Intramural thrombus or raised intimal flap or dissection/intramural hematoma with >25% stenosis
   (C) Grade III—Pseudoaneurysm
   (D) Grade IV—Vessel occlusion
   (E) Grade V—Vessel transection

5. Treatment
a. Controversial
i. Efficacy of heparin, aspirin, and clopidogrel in preventing thromboembolic events in BCVIs has not been studied sufficiently. The effectiveness of endovascular stenting/coiling versus medicines alone has also not been studied sufficiently
ii. One study showed no difference in clinical outcomes between aspirin and anticoagulation
b. Early treatment improves long-term outcomes
c. Consider anticoagulation for any BCVI unless CIs (eg, due to coexisting injuries)
d. Proposed treatment algorithm
   i. Grade I—No treatment, or anticoagulation and conversion to antiplatelet therapy (regardless whether symptomatic)
   ii. Grades II–IV—Anticoagulation and/or endovascular surgery (stenting or coiling)
   iii. Grade V—Anticoagulation and endovascular or open surgery
e. Fracture reduction may reduce turbulent blood flow and also minimize risk of thrombus formation

6. RTP guidelines
a. No published guidelines
b. Consider RTP if
   i. Resolution of neurologic deficits
   ii. Imaging-proven resolution of vascular injury
c. Relative CI—Ongoing anticoagulation or antiplatelet therapy
IV. Safety Considerations

A. Equipment—Cowboy/Bullock/Kerr collars and neck rolls
1. Worn by football players to limit neck ROM in an attempt to prevent stingers/burners
2. Use is controversial
3. No good evidence they are effective
4. Limiting neck extension may result in undesired neck flexion that can potentially increase athletes’ risk of severe spinal cord injury

B. Technique—Spearing
1. Spearing is a tackling and blocking technique in football characterized by making first contact with the opponent using the top of the helmet
2. High-impact contact while in spearing position is the primary mechanism for catastrophic C-spine injuries in sports
3. Spear tackling was prohibited by the NCAA and high-school regulatory organizations in 1976, which resulted in a significant decrease in the incidence of catastrophic C-spine injuries
4. Proper tackling technique—Head up, trunk flexed 45°, first contact using shoulder

C. Classification of C-spine risk by sport
1. High-risk contact/collision sports
   a. Football, rugby, hockey, lacrosse, skiing, snowboarding, pole vaulting, gymnastics, cheerleading, wrestling
   b. May be a direct relationship between long-term participation in contact sports and the development of premature (radiographic) osteoarthritis
2. Medium-risk contact sports
   a. Soccer, basketball, baseball, equestrian
3. Low-risk noncontact sports
   a. Running, swimming, biking

D. RTP principles
1. Guidelines for many C-spine injuries remain controversial due to the relatively low frequency of injuries and resulting lack of randomized trials
2. Most RTP guidelines predominantly apply to high-risk sports. Noncontact sports usually do not increase the risk of C-spine injury
3. In general, may RTP when the athlete has pain-free full ROM and normal or near-normal strength

Recommended Reading


Knee Injuries and Conditions

Ashwin N. Babu and Cheri A. Blauwet

I. Clinical Anatomy

A. Joints

1. Tibiofemoral joint: Modified hinge joint
   a. Medial and lateral tibiofemoral compartments
   b. Normal range of motion (ROM): 135° of flexion, 5° to 10° of hyperextension, 10° of internal/external rotation
   c. Screw home mechanism: Locks the knee during terminal 20° of knee extension
      i. Kinetics vary, depending on open versus closed chain
         (A) Open chain: External rotation and anterior glide of the tibia
         (B) Closed chain: Internal rotation and anterior glide of the tibia
      ii. Unlocking of the knee is via popliteus muscle activation

2. Patellofemoral joint: Modified plane joint
   a. Medial stabilizers
      i. Dynamic—Vastus medialis obliquus (VMO)
      ii. Static—Medial femoral condyle, medial retinaculum, especially medial patellofemoral ligament (MPFL)
   b. Lateral stabilizers
      i. Dynamic: Vastus lateralis, iliotibial band (ITB)
      ii. Static: Lateral femoral condyle, lateral retinaculum
   c. Q angle: Formed by lines that connect the anterior superior iliac spine (ASIS) to the midpatella and the midpatella to the tibial tubercle
   d. Patellar loading varies and thus symptoms depend on activity
      i. Walking: 0.3 to 0.5 × body weight
      ii. Ascending stairs: 2 to 4 × body weight
      iii. Descending stairs: 3.5 × body weight
      iv. Squatting: 7 to 8 × body weight

3. Proximal tibiofibular joint: Synovial joint
   a. Stabilized by anterior and posterior ligaments of the fibular head

B. Ligaments

1. Anterior cruciate ligament (ACL)
   a. Originates at the posteromedial aspect of the lateral femoral condyle and courses anteromedially to insert anterior to the intercondylar eminence
   b. Two bundles
      i. Anteromedial: Tight in flexion
      ii. Posterolateral: Tight in extension
   c. Function
III: DIAGNOSIS AND TREATMENT OF SPORTS INJURIES AND CONDITIONS

1. Primary: Prevents anterior displacement of the tibia on the femur
   ii. Secondary: Limits varus/valgus stress and rotation, prevents hyperextension, assists in screw home mechanism and proprioception

2. Posterior cruciate ligament (PCL)
   a. Originates at the anterolateral aspect of the medial femoral condyle, courses posterolateral to insert on the posterior surface of the tibial eminence
   b. Two bundles
      i. Anterolateral: ~65% of substance of PCL, tight in flexion
      ii. Posteromedial: ~35% of substance of PCL, tight in extension
   c. Function
      i. Primary: Prevents posterior displacement of tibia on femur
      ii. Secondary: Limits varus/valgus stress, assists in proprioception and screw home mechanism

3. Medial collateral ligament (MCL)
   a. Two components
      i. Superficial: Originates at medial epicondyle, inserts at the proximal tibia, attaches broadly 5 to 7 cm below joint line
      ii. Deep: Contiguous with medial meniscus, consists of tibiomeniscal and meniscofemoral ligaments
   b. Primary restraint to valgus stress

4. Lateral collateral ligament (LCL)
   a. Originates at lateral femoral condyle and inserts on fibular head
   b. Primary restraint to varus stress

5. Ligaments of the posterolateral corner (PLC)
   a. Anterolateral ligament (ALL)
      i. Originates at the lateral femoral condyle, courses anteroinferior, inserts midway between the fibula and Gerdy’s tubercle
   b. Popliteofibular ligament
   c. Fabellofibular ligament

6. Meniscofemoral ligament
   a. Courses from posterior horn of lateral meniscus to medial femoral condyle
   b. Anatomic variants lie either anterior (ligament of Humphrey) or posterior (ligament of Wrisberg) to PCL

C. Menisci
   1. Medial meniscus
      a. More “C” shaped, covers 50% to 60% of medial tibial plateau
      b. Less excursion than lateral meniscus due to attachment of the deep component of the MCL

2. Lateral meniscus
   a. More “O” shaped, covers 70% to 80% of lateral tibial plateau
   b. More excursion than medial meniscus
   c. Discoid meniscus variant: Thickened, ovoid variant of the lateral meniscus where the attachment of the posterior horn is absent, predisposing to increased motion and injury

3. Vascular supply
   a. Outer one-third has good blood supply (red zone) and potential to heal
   b. Inner two-thirds have poor supply (white zone) and limited healing potential

4. Function: Load transmission, knee stability, proprioception

D. Capsule
   1. Composed of ligaments and tendons surrounding the knee joint
   2. Lined with synovium that secretes synovial fluid
3. Synovial plicae: Redundant folds of synovium; during fetal development, plicae are membranes that separate the knee into compartments, which typically diminish in size during the second trimester, but may resorb incompletely
   a. Locations: Mediopatellar (most common), suprapatellar, lateral, infrapatellar

II. Knee Injuries and Conditions

A. Fractures

1. Patellar fracture
   a. Types
      i. Direct trauma (e.g., fall onto patella): Often causes comminution, but typically not displaced; articular cartilage often damaged
      ii. Indirect trauma (e.g., forceful quadriceps activation): Less likely to be comminuted but more likely displaced fragments; less damage to articular cartilage
   b. Imaging: Plain radiographs
      i. Anterior–posterior (AP) view: Difficult to visualize patella and peripheral fractures may be mistaken for bipartite patella (check bilateral films)
      ii. Lateral view: Often reveals comminution or displacement
      iii. Sunrise view: Reveals vertical fractures
   c. Treatment
      i. Nonoperative: Minimal or nondisplaced fractures with minimal disruption of articular surface, preserved extensor mechanism
         (A) Strict splinting in extension
         (B) Gradually increase flexion range as healing progresses
      ii. Operative: Avulsion of extensor mechanism, displaced transverse fracture
   d. Complications: Infection, avascular necrosis, posttraumatic arthritis of patellofemoral compartment, quadriceps weakness

2. Tibial plateau fracture
   a. Types
      i. Sports related: Often accompanied by ligamentous injury
      ii. Low energy/osteoporotic
         (A) Typically a depressed fracture
      iii. High energy
         (A) Often due to motor vehicle accidents, pattern is typically a bicondylar fracture
   b. Treatment
      i. Nonoperative if minimal displacement or depressed fracture
         (A) Non–weight-bearing with brace × 6 weeks
      ii. Operative management
         (A) Absolute indications: Open plateau fractures, associated compartment syndrome, associated vascular injury
         (B) Relative indications: Displaced bicondylar fractures, displaced medial condylar fractures, lateral fractures with joint instability

3. Segond fracture
   a. Features
      i. Small capsular avulsion fracture of the lateral tibial plateau
      ii. Results from abnormal varus stress to the knee combined with internal rotation of the tibia
      iii. Likely to be associated with ACL tear (75%–100%) and/or meniscal injury (66%–75%)
      iv. Can also be associated with avulsion at the fibular head or Gerdy’s tubercle
   b. Treatment: Typically requires surgical intervention, given concomitant injuries
4. Stress fractures
   a. Types
      i. Patellar: High-risk stress fracture due to risk of nonunion
      ii. Medial tibial plateau: Commonly misdiagnosed as pes anserine bursitis
   b. Diagnosis should prompt evaluation for low energy availability

B. Dislocation
   1. Patellar dislocation: Patella displaced laterally out of the trochlear groove
      a. Types
         i. Traumatic: Often occurs with twisting or jumping, presents with hemarthrosis
         ii. Atraumatic: Occurs mainly in young females with generalized ligamentous laxity
      b. Features
         i. Associated with injury to medial retinaculum, particularly MPFL, which inserts onto the adductor tubercle
         ii. Osteochondral fracture occurs in one-third of cases
         iii. Usually reduced spontaneously with knee extension
         iv. Risk factors include shallow trochlear groove, generalized ligamentous laxity, patella alta, femoral anteversion, loose medial or tight lateral retinaculum
      c. Physical examination pearl
         i. Lateral apprehension test: Sense of apprehension with passive lateral displacement of patella in knee in extension
      d. Imaging
         i. X-rays should be obtained after reduction to evaluate for osteochondral fracture or avulsion (AP, lateral, and sunrise view)
         ii. Obtain MRI if concern for osteochondral defect on x-ray, persistence of symptoms, or for evaluation of medial retinaculum/ MPFL in recurrent dislocations (Figure 25.1)

Figure 25.1  T2-weighted MRI image of a knee in the axial plane in a patient with a patellar dislocation. Note the increased signal intensity over the lateral aspect of the trochlea and the medial aspect of the patella, representing a characteristic bone contusion pattern (arrows) seen in lateral patellar dislocations.
e. Treatment
   i. Nonoperative: Consider knee brace/immobilizer to maintain extension in acute phase, then progress to rehabilitation program
   ii. Operative: For recurrent patellar dislocations or presence of an osteochondral defect

2. Knee dislocation
   a. Types: Described by position of the tibia relative to femur
      i. Anterior: Occurs in hyperextension injuries
      ii. Posterior: Dashboard-type injuries
      iii. Medial, lateral, and rotatory dislocations may also occur
   b. Features
      i. High-force injury often associated with severe multiligamentous injury, nerve injury (fibular), vascular injury (popliteal artery), fracture
      ii. Consider immediate arteriography to evaluate popliteal artery (medical emergency)
   c. Treatment
      i. Emergent surgical intervention: Open dislocation, popliteal artery injury, irreducible dislocations, compartment syndrome
      ii. Delayed intervention: Ligamentous reconstruction

C. Muscle/tendon injuries
1. ITB friction syndrome
   a. Features
      i. Typically seen in athletes requiring repetitive knee motion such as running, cycling, rowing
      ii. Patients present with pain over lateral knee, usually absent at rest
      iii. Traditionally thought to be caused by friction-induced irritation of a bursa deep to the ITB as it courses over the lateral femoral epicondyle; current evidence conflicting
   b. Physical exam pearls
      i. Tenderness over ITB, commonly at insertion and 3 cm proximal to the lateral joint line
      ii. Ober’s test: Demonstrating ITB tightness
      iii. Noble’s test: Passively extend knee from 90° of flexion while palpating over the lateral femoral epicondyle; lateral knee pain reproduced at 30° of flexion
   c. Treatment
      i. Mainstay is conservative management including biomechanics evaluation, activity modification, equipment evaluation, and local anti-inflammatory modalities
      ii. Surgical intervention: Recalcitrant cases only, several surgical options exist

2. Patellar tendinopathy (jumper’s knee)
   a. Features
      i. Usually insidious onset in younger athletes
      ii. Repetitive knee flexion/extension, such as with jumping
      iii. Most commonly occurs at patellar insertion
   b. Treatment
      i. Nonoperative
         (A) Relative tendon unloading
         (B) Eccentric strengthening and motor control exercises
         (C) Correction of biomechanical factors
         (D) Injection therapy use debated in literature; if utilized is typically a regenerative injection
      ii. Operative: Often debridement of abnormal tissue, only for recalcitrant cases
3. Popliteus tendinopathy
   a. Features
      i. Rare cause of posterolateral knee pain
      ii. Typically a chronic/overuse injury, exacerbated with excessive downhill walking/running
   b. Physical examination pearl
      i. Garrick’s test: Place leg in “figure four” position (crossed position) and palpate just posterior to the LCL while resisting tibial internal rotation
   c. Treatment: Relative rest, nonsteroidal anti-inflammatory drugs (NSAIDs), functional rehabilitation

4. Extensor mechanism injury
   a. Types
      i. Quadriceps tendon rupture
         (A) More common in older males (average age 60)
      ii. Patellar fracture
         (A) More common in women with osteoporosis
      iii. Patellar tendon rupture
         (A) More common in younger males (average age 40)
   b. Features
      i. In older individuals, often associated with medical comorbidities such as diabetes mellitus and atherosclerosis
      ii. Quadriceps and patellar tendon ruptures are often the result of a forcible contraction of the quadriceps across a flexed knee
   c. Treatment
      i. Often requires early operative repair
      ii. Post-op: Start early weight bearing in extension with limited ROM

D. Instability/ligament injuries
   1. ACL injuries
      a. Features
         i. Most common knee ligament injured in sports
         ii. Females two to eight times more likely than males to experience ACL injury
         iii. Often a noncontact injury after cutting, landing, or rapid deceleration
         iv. Contact injuries often result from an external force causing knee valgus
         v. Patients may report hearing a “pop” followed by a feeling of instability
         vi. Hemarthrosis/swelling occurs within hours
         vii. >50% of ACL injuries occur with a meniscal tear
         viii. “Terrible triad” is a buzzword for concomitant tears of ACL, MCL, and medial meniscus
         ix. ACL-injured knees are at higher risk for the development of premature osteoarthritis
      b. Diagnosis
         i. Physical examination pearls
            (A) May be difficult to perform complete examination in the acute phase due to pain and swelling
            (B) Presence of effusion (hemarthrosis)
            (C) Positive Lachman test (sensitivity 95%–99%)
               (1) Grade 1+: 0 to 5 mm displacement
               (2) Grade 2+: 6 to 10 mm
               (3) Grade 3+: >10 mm
            (D) Positive anterior drawer (sensitivity 22%–95%, specificity 53%–95%)
            (E) Positive pivot shift is pathognomonic, particularly when performed under anesthesia (sensitivity 35%–98%, specificity 98%–100%)
ii. Imaging
   (A) Plain radiographs: Evaluation for bony injuries such as tibial spine avulsion, tibial plateau, or Segond fractures
   (B) MRI
      (1) Often demonstrates classic bone contusion pattern (anterior lateral femoral condyle and posterior lateral tibial plateau) (Figure 25.2)
      (2) Also evaluates for concomitant injuries to the meniscus, PLC, and other ligaments

iii. Arthroscopic evaluation is the gold standard

c. Treatment
i. Acute/initial
   (A) Rest, ice, compression, elevation (RICE)
   (B) Knee immobilizer or hinged knee brace and crutches if painful or subjectively unstable
   (C) Early gentle knee ROM exercises
   (D) Exercises to prevent quadriceps inhibition/atrophy

ii. Subacute/long term
   (A) Operative: ACL reconstruction
      (1) Considered in athletes playing cutting sports, with multiligamentous injury, having a repairable meniscus injury, or having functional knee instability
      (2) Prehabilitation is important to regain ROM and decrease swelling prior to surgery
      (3) Delayed surgery has decreased risk of arthrofibrosis

Figure 25.2  T2-weighted MRI image of a knee in the sagittal plane in a patient with an ACL tear. Note the increased signal intensity over the anterolateral femoral condyle and the posterolateral tibia, known as a “kissing contusion” (arrows).

ACL, anterior cruciate ligament.
(4) Surgery does not appear to prevent the development of osteoarthritis
(5) Postoperative rehabilitation program lasts 6 to 9 months prior to full return to play

(B) Nonoperative: Aggressive rehabilitation program
(1) Can be considered in sedentary patients, athletes participating in noncutting sports (eg, cycling), those without significant instability
(2) Hamstring strengthening to aid ACL in its role in preventing anterior tibial translation
(3) Patient should be followed regularly to assess for knee instability or symptomatic meniscus injury
(4) Bracing for ACL-deficient athletes, though commonly used, is controversial

2. PCL injuries
   a. Features
      i. Less common than ACL injuries
      ii. Majority associated with injury to other structures, need to evaluate PLC when PCL injury is suspected
      iii. Typically from an impact to anterior tibia with knee flexion (eg, dashboard injury), less commonly from knee hyperextension
      iv. Often presents with minimal swelling and poorly defined posterior knee pain
   b. Physical examination pearls
      i. Posterior drawer test
         (A) Grade 1+: 0 to 5 mm displacement
         (B) Grade 2+: 6 to 10 mm
         (C) Grade 3+: >10 mm
      ii. Positive quadriceps activation test (sensitivity 54%–98%, specificity 97%–100%):  
         (A) Patient is supine with knee flexed to 90°, foot stabilized by examiner, then asked to slide foot gently down the table
         (B) In a PCL-deficient knee, quadriceps contraction results in anterior shift of tibia ≥2 mm
      iii. Reverse Lachman’s test
      iv. Posterior sag sign (quadriceps spasm may cause false-negative)
   c. Imaging
      i. Plain radiographs
         (A) Usually negative in isolated PCL injuries
         (B) Can evaluate for concomitant avulsion injury of the tibial insertion
      ii. MRI
         (A) Useful to diagnose PCL injury and evaluate for associated ligamentous injuries
      iii. Arthroscopic evaluation is gold standard
   d. Treatment
      i. Acute/initial
         (A) RICE
         (B) Early ROM exercises
         (C) Consider knee brace (in full extension)/crutches × 1 to 2 weeks if significant functional limitation
      ii. Subacute/long term
         (A) Functional rehabilitation, especially quadriceps strengthening to help support PCL’s role in preventing posterior tibial translation
         (B) Surgical reconstruction for PCL rarely required, but useful if there is persistent instability or other surgically repairable injuries
3. MCL injuries
   a. Features
      i. Caused by impact to lateral knee causing valgus stress, especially with foot planted in knee flexion
      ii. Concomitant ACL injuries can be seen with cutting, rotational movements
   b. Diagnosis
      i. Physical examination pearls
         (A) Tenderness along course of MCL
         (B) Valgus stress test: Performed with knee flexed to 30°
            (1) Grade 1: Pain but no laxity
            (2) Grade 2: Some laxity but clear end point
            (3) Grade 3: Complete disruption of ligament with gaping of medial joint line without end point
         (C) Valgus stress test should also be performed with knee in full extension; if laxity or gapping, a concomitant ACL, PCL, or PLC injury may be present
         (D) Minimal effusion in isolated injury given MCL is extraarticular
      ii. Imaging
         (A) X-ray usually normal: AP and lateral views can rule out associated bony injury (epiphyseal fracture)
         (I) Pellegrini-Stieda sign (Figure 25.3): Typically asymptomatic, posttraumatic ossification of the MCL near its origin at the medial femoral condyle can be seen in those with a history of MCL injury
         (B) MRI: Usually obtained if there are other suspected injuries
   c. Treatment
      i. RICE in acute phase
      ii. Consider hinged knee brace × 4 to 6 weeks
      iii. Early gentle knee ROM exercises (within 1–2 weeks)
      iv. Gradual advance to higher-level activities as tolerated (next 1–4 weeks)
      v. Isolated MCL injuries rarely require surgery unless tibial-sided avulsion is present, for which acute surgical repair is indicated

4. LCL injuries
   a. Features
      i. Typically the result of an acute varus stress
      ii. Rarely injured in isolation
   b. Physical examination pearls
      i. Positive varus stress test, performed at 30° of knee flexion (grading system is the same as for MCL, noted in the preceding text)
      ii. Varus stress test should also be performed with knee in full extension; if laxity or gapping, a concomitant ACL, PCL, or PLC injury may be present
      ii. Evaluate for associated fibular nerve injury

Figure 25.3 Tunnel radiograph of the knee with findings of a Pellegrini-Stieda sign, indicating a history of MCL injury. MCL, medial collateral ligament.
c. Treatment
   i. RICE
   ii. Early knee ROM exercises
   iii. Functional rehabilitation
   iv. Surgical intervention typically depends upon concomitant injuries

5. PLC injuries
   a. Features
      i. Injury to the PLC may be associated with severe injury to other structures (especially PCL or ACL)
      ii. Untreated PLC injuries are a major source of failure for ACL reconstructions
   b. Physical examination pearls
      i. Dial test (Figure 25.4): Assess external rotation (ER) of the tibia compared to the contralateral side at 30° and 90° of knee flexion
         (A) Increased external rotation at 30°, but not 90°, indicates isolated PLC injury
         (B) Increased external rotation at both angles suggests injury to both the PLC structures and the PCL
   c. Imaging
      i. MRI is needed to determine the specific structures involved
   d. Treatment: If injury is associated with cruciate ligament tear, surgical intervention is typically warranted

E. Anterior knee pain
1. Plica syndrome
   a. Features
      i. Most synovial plicae are asymptomatic; however, may progress to be painful, inflamed, and eventually hypertrophic
      ii. Mediotibial plica is most commonly involved
   b. Physical examination pearl: Taut band of tissue that reproduces concordant pain with palpation
   c. Imaging: Plain radiographs or MRI to rule out more common sources of anterior pain
   d. Treatment
      i. RICE, NSAIDs; consider steroid injection
      ii. Functional rehabilitation

Figure 25.4 The dial test. In the left panel, the examiner passively externally rotates the tibia on the femur at 30° of knee flexion. In the right panel, the examiner repeats the maneuver at 90° of knee flexion. Note the increased external rotation of the right knee with 30° of knee flexion but not at 90° of knee flexion, suggesting an isolated posterolateral corner injury.
25. KNEE INJURIES AND CONDITIONS

iii. Arthroscopic removal is reserved for recalcitrant cases and may have a high rate of failure

2. Fat pad impingement (Hoffa’s syndrome)
   a. Features
      i. Hoffa’s fat pad, located deep to the patellar tendon, can become inflamed/swollen due to direct trauma, hyperextension, or chronic irritation
      ii. When inflamed, it may impinge between the inferior pole of the patella and femoral condyle
      iii. Causes anterior/inferior knee pain worsened with knee extension
      iv. Commonly irritated by transitioning from sit to stand
         (A) Contrast with patellofemoral pain, which is more symptomatic with prolonged sitting in knee flexion
   b. Physical examination pearls
      i. May have genu recurvatum
      ii. Positive Hoffa’s test
         (A) Patient lies supine with knee flexed, and examiner presses both thumbs along the sides of the patellar tendon, just distal to the patella
         (B) Pain/apprehension with active knee extension is considered positive
   c. Treatment
      i. RICE
      ii. Patellar taping
      iii. Surgical removal of fat pad in recalcitrant cases

3. Patellofemoral syndrome
   a. Features
      i. Anterior knee pain in region of patella
      ii. Typically insidious onset, often bilateral, worse with prolonged sitting (positive “theater sign”) or activities that load the patellofemoral joint (see Section I.A.2, patellofemoral joint: modified plane joint)
      iii. Likely combination of poor patellar tracking/malalignment within femoral groove as well as peripatellar synovial irritation
      iv. May be associated with patellar hypermobility/subluxation
      v. Chondromalacia patellae: Degeneration of the patellar cartilage, often associated with patellofemoral syndrome, particularly in aging athletes
   b. Risk factors: Consider the entire kinetic chain
      i. Proximal factors: Femoral anteversion, weak hip abductors and external rotators, tight hip flexors, tight ITB
      ii. Local factors: Patella alta/baja, increased Q angle, genu valgum, weak quadriceps, hypermobile patella, tight hamstrings, tight quadriceps
      iii. Distal factors: Increased subtalar pronation, tibial external rotation, weak foot intrinsics, tight gastrocnemius/soleus
   c. Physical examination pearls
      i. Important to evaluate kinetic chain assessing for proximal hip strength, femoral rotational control, dynamic knee stability, ankle and foot mechanics (Figure 25.5)
      ii. Evaluate patellar position both statically and dynamically
         (A) J sign: Lateral translation of the patella at terminal extension, with medial motion during early flexion
      iii. Positive patellofemoral grind/compression test
   d. Imaging: Can be used to assess for intra-articular pathology, bipartite patella, patellar malalignment
   e. Treatment
      i. RICE, activity modification
ii. Rehabilitation program, focusing on risk factors listed in Subsection b in the preceding text

iii. Taping: Based upon position of the patella in relation to the femur
   (A) Need to evaluate tilt, glide, and rotation

iv. Bracing: Patellar tracking brace provides firm lateral support to facilitate medial glide of the patella in flexion; should be used only after athlete’s own intrinsic strength is optimized

v. Orthotics: Consider correction of overpronation

vi. Surgical interventions such as lateral release or tibial osteotomy rarely indicated

F. Meniscal pathology
1. Medial and lateral meniscus tears
   a. Features of medial meniscus injury
      i. More common than lateral meniscus injury
      ii. Often a history of cutting activity, tibial rotation with knee flexion during weight bearing (eg, soccer, football)
      iii. Commonly associated with ACL injury
   b. Features of lateral meniscus injury
      i. Injury after squatting, full flexion with rotation (eg, wrestling)
      ii. Discoid meniscus variation can be cause of lateral knee pain in a young athlete
   c. Features common to medial and lateral meniscus injuries
      i. Present with mechanical symptoms: Painful clicking, popping, catching
      ii. Degenerative tears typically occur at >40 years old, minimal trauma
   d. Physical examination pearls
      i. Effusion ± restricted ROM
      ii. Pain with squatting
      iii. Joint line tenderness
      iv. Provocative maneuvers
         (A) McMurray test: Audible and palpable click is considered a positive test, although more often it produces pain only
         (B) Apley grind: Tenderness with flexion, rotation, and axial compression
         (C) Bounce home test: Pain with terminal extension
         (D) Thessaly test: Pain with rotation on a partially flexed knee
   e. Imaging
      i. Plain radiographs evaluate for degenerative changes, loose bodies
      ii. MRI: Grading system used for meniscal signal intensity
         (A) Grade 1: Small focal area of hyperintensity, no extension to the articular surface

Figure 25.5 A patient with hip abductor and external rotator weakness (ie, poor proximal control) performing a single-leg squat. Note the contralateral pelvic tilt as well as femoral adduction/internal rotation with resultant valgus moment at the knee, highlighted by the lines.
25. KNEE INJURIES AND CONDITIONS

(B) Grade 2: Linear area of hyperintensity, no extension to articular surface
(C) Grade 3: Hyperintensity extends to at least one articular surface, is referred to as definite meniscal tear

f. Treatment
i. Nonoperative for those with minor tears and without significant functional disability
ii. Surgical intervention: For those with functional limitation not responding to conservative management or with large tears causing mechanical symptoms
(A) Attempt to maximize preservation of meniscus
(B) Inner two-thirds poorly vascularized, may require removal of damaged tissue (ie, partial meniscectomy)
(C) Outer one-third can often be repaired

G. Degenerative joint disease

1. Osteoarthritis
a. Features
i. Medial compartment most commonly affected
ii. Involvement of lateral, patellofemoral, or a combination of all compartments possible
iii. Osteoarthritis present in a relatively young patient is often due to history of joint trauma/acute ligamentous injury

b. Imaging
i. Plain radiographs: Weight bearing films, sunrise view to evaluate patellofemoral compartment
(A) Hallmarks: Joint space narrowing, subchondral cysts, sclerosis, marginal osteophytes or spoking of the tibial spines

c. Treatment
i. Conservative management
(A) Activity modification
(B) Weight loss
(C) Rehabilitation (quadriceps and gluteal strengthening)
(D) Unloader bracing (limited evidence)
(E) Assistive devices (cane in contralateral hand)
(F) Heel wedge (lateral wedge to offload medial compartment)
(G) Oral medications (acetaminophen/NSAIDs)

ii. Intra-articular injections
(A) Steroid injection commonly used
(B) Other options include viscosupplementation and regenerative injections, although evidence remains limited

iii. Operative management: Knee arthroplasty
(A) Indications: Failure of nonsurgical management, significant functional limitations, notable radiographic degenerative changes

H. Bursitis

1. General principles
a. Bursae typically provide an interface between bony surfaces and ligaments or tendons in areas of friction
b. Acute traumatic injury, infection or systemic disease, abnormal biomechanics, or chronic overuse can cause bursitis/irritation of bursa
c. Examination typically demonstrates focal tenderness and swelling
i. Warmth or erythema may indicate infection
d. Treatment typically consists of RICE, NSAIDs, and occasionally aspiration with corticosteroid injection if noninfectious

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2. Prepatellar bursitis (housemaid’s knee)
   a. Due to trauma or chronic irritation from extensive kneeling
   b. May have urate crystals due to gout
3. Pes anserine bursitis
   a. Difficult to differentiate between bursal pathology and tendon insertional pathology of the sartorius, gracilis, or semitendinosus
4. MCL bursitis (Voshell’s bursitis)
   a. Located between superficial and deep portions of MCL

Recommended Reading

Chest Trauma: Thoracic Injuries and Conditions

Kristi Colbenson

ANATOMY

I. General Considerations

A. Chest wall—musculoskeletal structures
   1. Twelve ribs—first 7 (true) ribs articulate anteriorly with sternum via the costochondral cartilage; ribs 8 to 10 (false) articulate anteriorly to the costal cartilage; ribs 11 to 12 (floating) have no anterior articulation
   2. Posteriorly—ribs 2 to 10 articulate with two adjacent vertebral bodies and transverse processes via costovertebral and costotransverse joints; remaining ribs articulate with only one vertebra
   3. Anteriorly—sternum, manubrium, and xiphisternum—attached to body of sternum by fibrocartilage; sternum articulates proximally with clavicles via synovial joints (sternoclavicular joints)
   4. Thoracic musculature—internal and external intercostal muscles, overlying serratus anterior, latissimus dorsi, trapezius muscles, pectoralis major and minor
   5. Neurovascular—intercostal neurovascular bundle lies inferior to each rib; intercostal nerves supply thoracic wall; cervical nerves 3, 4, 5 innervate the diaphragm.

B. Thoracic spine:
   1. Twelve vertebrae—composed of spinous process, transverse process, vertebral arch, vertebral body, and articular process
   2. Articulations—zygapophyseal joint (facet); costovertebral junctions

C. Lung structures:
   1. Pleura—parietal (attached to chest wall, diaphragm, and mediastinum) and visceral (covering lungs), pleural cavity—potential space between the two pleura
   2. Trachea, bronchi, bronchioles, alveoli (location of gas exchange); bronchovascular system

D. Mediastinum:
   1. Bordered by sternum anteriorly, vertebral column posteriorly, thoracic inlet superiorly, and diaphragm inferiorly
   2. Anterior—thymus, lymphatics, internal mammary artery, intercostal neurovascular bundle
   3. Middle—pericardium, myocardium, ascending aorta, right brachiocephalic artery, left internal carotid, and subclavian artery (comes directly off the aorta), superior vena cava (SVC), subclavian vein, trachea
   4. Posterior—descending aorta, azygous vein, sympathetic chain, esophagus, gastroesophageal junction
EPIDEMIOLOGY, DIAGNOSIS, AND TREATMENT

II. General Considerations

A. Epidemiology of chest/thoracic pain

1. Can occur due to:
   a. Trauma (usually acute onset)
   b. Overuse (more chronic insidious onset)
   c. Referred visceral pathology

2. Chest pain can be due to:
   a. Musculoskeletal causes: bone/cartilage, joints, myofascial
   b. Gastrointestinal disorders: gastrointestinal esophageal reflux disease (GERD), esophagitis, Boerhaave’s syndrome (esophageal rupture), esophageal dysmotility disorders, esophageal spasm, hiatal hernia, peptic ulcer, biliary colic/cholecystitis, pancreatitis
   c. Respiratory conditions: lower respiratory tract infection, asthma, exercise-induced bronchospasm (EIB), spontaneous or traumatic pneumothorax, hemothorax, pneumomediastinum, subcutaneous emphysema
   d. Cardiac conditions: myocardial infarction, myocardial contusion, myocarditis, pericarditis, pericardial effusion, cardiac tamponade, pneumopericardium, aortic dissection, apical ballooning syndrome
   e. Miscellaneous: malignancy, herpes zoster, spontaneous pneumomediastinum, Mondor’s disease (idiopathic thrombosis of subcutaneous veins of anterior chest wall), SVC syndrome

B. Diagnosis

1. History
   a. Trauma: penetrating, blunt, barotrauma (ie, scuba diving), blast
   b. Insidious onset (overuse mechanism)
   c. Exertional
   d. Comorbidities/associated symptoms

2. Physical examination
   a. Observation—ecchymosis, “splinting,” paradoxical motion of “flail chest” segment, signs of respiratory distress (accessory muscle use and diaphragmatic breathing), cyanosis (lips and nail beds), bulging neck veins, plethoric face
   b. Palpation—areas of tenderness, rib “springing,” chest wall instability, carotid and peripheral pulses (rhythm, intensity, variation), tracheal deviation, subcutaneous emphysema
   c. Auscultation—quality of breath sounds, air movement, crackles, inspiratory wheeze, expiratory wheeze, inspiratory stridor, dysphonia, cardiac murmurs, and heart sounds

III. Fractures

A. Rib fractures

1. Basics
   a. Fractures occur at site of trauma or the posterolateral bend where the rib is the weakest
   b. First rib fractures—require considerable traumatic force—high risk for associated neurovascular injuries—subclavian artery/vein, aortic arch aneurysm, Horner’s syndrome, and brachial plexus
   c. Ribs 9 to 12 should raise suspicion for intra-abdominal injury
   d. “Flail” chest—two adjacent ribs broken in two sites—creates unstable segment that moves paradoxically in response to intrathoracic pressure
   e. Rib fractures can have many associated injuries—pulmonary contusion, pulmonary laceration, hemothorax, pneumothorax, hemothorax, pneumomediastinum, subcutaneous emphysema, liver laceration, splenic laceration
II: DIAGNOSIS AND TREATMENT OF SPORTS INJURIES AND CONDITIONS

2. Diagnosis
   a. Physical examination
      i. “Rib springing”—compression perpendicular to the site of concern recreates the pain
   b. Imaging
      i. Plain radiographs have low sensitivity
      ii. Ultrasound has high sensitivity and specificity for rib fractures
      iii. Dedicated rib series increase sensitivity by utilizing bone exposure—increased radiation exposure
      iv. Inspiration and expiration views can reveal underlying pneumothorax
      v. CT scan for suspected first rib fracture

3. Management
   a. Pain management (opioids, nonsteroidal anti-inflammatory drugs [NSAIDs]) to allow adequate inspiratory effort—prevents splinting resulting in atelectasis and subsequent pneumonia
   b. Elderly, multiple rib fractures (3+), or ventilation compromise—admission with consideration for intercostal nerve blocks
   c. Normally heal within 6 weeks, no indication for routine radiographs
   d. Return to activity as tolerated—consider flak jacket for protection

B. Sternal fracture
   1. Basics
      a. Mid-body followed by manubrium fractures is most common
      b. Blunt trauma, large amount of force needed
      c. High risk for associated injuries to underlying cardiac structures and lungs
      d. Stress fractures—reported in wrestlers and overhead athletes
   2. Diagnosis
      a. Standard chest x-ray (CXR) and dedicated lateral sternal views
   3. Cardiac contusion
      a. Displaced sternal fractures—myocardial trauma—cause dysrhythmia, conduction abnormalities, ST segment changes, and vital sign instability
      b. Diagnosis
         i. Initial ECG and 6-hour follow-up
         ii. Troponin 4 to 6 hours posttrauma
   4. Treatment
      a. Conservative with analgesics
      b. Return to play—per radiographic and clinical healing—usually 8 to 12 weeks

C. Thoracic vertebrae
   1. General consideration
      a. Denis’s three-column model
         i. Anterior—anterior longitudinal ligament and anterior half of vertebral body
         ii. Middle—posterior component of vertebral body to the pedicles
         iii. Posterior—bony vertebral arch and associated ligaments
      b. Two-column involvement is considered unstable
   2. Vertebral body fractures
      a. Burst-type fracture
         i. Stable (middle column intact)
         ii. Unstable (>2 columns)—with retropulsion of posterior fragments into spinal canal, damaging or compressing spinal cord—can cause neurological sequelae in up to 60% of patients
         iii. Difficult to diagnose and visualize on x-ray—two-column involvement—obtain CT scan to visualize posterior components
   3. Compression fracture—most common
32. CHEST TRAUMA: THORACIC INJURIES AND CONDITIONS

a. Basics
   i. Combined axial flexion and compression resulting in anterior wedge fracture of the vertebral body
   ii. Among the most common back injuries in snow sports
   iii. More common in athletes with long-term corticosteroid use, postmenopausal women, or athletes with osteopenia or osteoporosis (female athlete triad)
   iv. Unstable—greater than 50% loss of vertebral height, greater than 25 degrees of fracture kyphosis, or multiple acute compression fractures

b. Imaging—plain radiographs—consider CT scan if any instability criterion is met
   i. Fractures of T11 and T12 can easily be missed on x-ray, because of overlying shadow of the diaphragm

c. Treatment
   i. Stable—one column—rest, pain control, extension orthosis to limit flexion
   ii. Unstable—nonsurgical—thoracolumbosacral orthosis (TLSO) brace (T7 and distal injuries) or cervicothoracolumbosacral (CTLSO) brace (proximal to T7); surgical—warranted to restore structural integrity with possible decompression of foraminal or central canals

4. Spinous process fracture
   a. Direct trauma to posterior spine, forced flexion with rotation
   b. Stable fracture

IV. Stress Fractures

A. General considerations
   1. Overload mechanism—need to understand specific stresses of sport; usually occur with increases in training, change in equipment or technique

B. Sternum—uncommon—repetitive forward flexion of the thoracic spine combined with protraction of the scapula (wrestlers, gymnasts)

C. Ribs
   1. First rib
      a. Common in golfers and overhead athletes
      b. Most commonly found at subclavian sulcus—anatomically the thinnest portion, between insertions of scalenus anterior and medius and serratus anterior muscles—“fatigue failure”
      c. Diagnose with “trapezius squeeze” test—focal tenderness
      d. Can develop painful hypertrophic pseudarthrosis
   2. Other rib stress fractures
      a. Golfers (posterolateral ribs 4–6)
         i. Due to repeatedly striking ground (novice players)
      b. Most common in rowers and other paddlers (kayak, canoe, dragon boat, outrigger canoe):
         i. Fifth to ninth ribs posterolaterally—most common location; but also seen in anterior axillary line
         ii. Site of rib cage compression (bending) with isometric cocontraction of serratus anterior muscles and trapezius during the “drive” phase, and/or opposite pull of external abdominal oblique muscle—various theories
         iii. Risk factors—increased training regimen on ergometers, use of large blades, long (>10 K) slow (stroke rate 16–18/min) steady-state rows

3. Diagnosis
   a. High index of suspicion in appropriate sport setting
   b. Insidious onset, localized tenderness, pain with “rib springing,” deep inspiration, cough, even rolling over in bed
   c. X-rays negative early on
d. Technetium bone scan is diagnostic

4. Treatment
   a. Rest from aggravating activities—may take 4 to 6 weeks
   b. Pain management—analgesics, NSAIDs
   c. Physiotherapy—correction of underlying biomechanics

V. Dislocations/Subluxation/Inflammation

A. Sternoclavicular joint
   1. General considerations
      a. Synovial joint, epiphysis does not fuse until age 22 to 25
      b. Posterior dislocations are rare but are a medical emergency due to potential for injury to the great vessels and airway immediately posterior to the joint
      c. Anterior/superior dislocations can also occur and are treated conservatively
      d. Sternoclavicular subluxation common in overhead sports and swimming—may be unilateral or bilateral
      e. Traumatic—fall on outstretched arm or direct blow to the shoulder
   2. Diagnosis
      a. Physical examination—localized swelling/tenderness over sternoclavicular joint; deformity; arm paresthesias, venous congestion, or decreased pulses; respiratory distress, dysphonia; and dysphagia
      b. X-rays—“serendipity” view (40 degrees cephalic tilt), CXR to rule out associated injury
      c. Gold standard is spiral CT; angiography if suspect vascular injury
   3. Treatment
      a. Posterior dislocations—intraoperative reduction
         i. If neurovascular compromise and no option for emergent transfer, consider reduction—supine position, abduct and extend the arm, apply axial traction, and manipulate the medial clavicle with a towel clip
      b. Anterior/superior dislocations acute—reduction is less urgent
      c. Sternoclavicular subluxation—chronic—conservative treatment

B. Costovertebral subluxation—seen in rowers, swimmers
   1. Localized tenderness, pain increased with side flexion of thoracic spine, rotation away from or into painful side
   2. Treatment—manipulation and prevention by therapeutic exercises and ballistic rotational stretches away from painful side

C. “Slipping-rib” syndrome—abdominal and/or thoracic pain—due to hypermobility of anterior ends of false rib costal cartilages—leads to slipping of affected rib under superior adjacent rib (usually ribs 8–10)
   1. Diagnose with “hooking maneuver”—clinician slips fingers under lower costal margin and pulls anteriorly; pain and “click”
   2. Treatment primarily conservative—reassurance, avoidance of postures that cause symptoms; may require nerve block or surgical resection

D. Costochondritis
   1. Basics
      a. Inflammation of the costochondral or costosternal joints—2 to 5 most common
      b. Benign cause of chest pain
      c. Tietze’s syndrome—type of costochondritis—nonsuppurative swelling of the costochondral joints—usually self-limited
   2. Diagnosis
      a. Clinical diagnosis—reproducible chest wall pain
      b. X-ray not helpful
   3. Management
a. Rest from aggravating activities, anti-inflammatories, reassurance
b. Tietze’s syndrome—can consider corticosteroid injection

VI. Pulmonary Conditions

A. Pneumothorax

1. General considerations
   a. Air or gas within the pleural space
   b. Nonspontaneous—traumatic—usually due to penetrating or direct blunt trauma
   c. Spontaneous
      i. Primary spontaneous (PSP)—rupture of blebs or bullae; risk factors include: tall, thin; Marfan’s syndrome; smoking
      ii. Secondary spontaneous (SSP)—secondary to underlying lung conditions; chronic obstructive pulmonary disease (COPD), cystic fibrosis
      iii. Occurs after episodes of increased intrathoracic pressure and shear force on the apex of the lung—coughing, straining, vomiting, Valsalva maneuver (weight lifting), breath holding
   d. Tension pneumothorax—air under positive pressure in the pleural space causing collapse of the lung parenchyma and displacement of the mediastinal structures

2. Diagnosis
   a. Clinical exam—acute chest pain, dyspnea, cough, hemoptysis, tachycardia
      i. Tension pneumothorax
         (A) Respiratory distress, tachypnea, unilateral decreased breath sounds, hyperresonance on percussion, hypotension, tachycardia, jugular venous distention, laryngeal shift
   b. Imaging
      i. X-ray—deep sulcus sign; visceral pleural edge, lack of lung markings—may need inspiration/expiration views for small pneumothorax to reveal hyperexpansion (Figure 32.1)
      ii. Ultrasonography—diagnostic aid—look for lack of lung sliding

3. Treatment
   a. Oxygen therapy—threefold increase in absorption rate
   b. Observation with expectant reabsorption—consider if PSP, stable vital signs, and small pneumothorax (<3 cm between lung and chest wall)
   c. Aspiration—large pneumothorax (>3 cm from lung to chest wall) or SSP; pigtail catheter or tube thoracostomy for decompression
   d. Tension pneumothorax—medical emergency
      i. Needle decompression—14-gauge, long, angiocatheter into the second intercostal space, mid-clavicular line
      ii. Tube thoracostomy
   e. Recurrent spontaneous pneumothorax—surgical intervention—video-assisted thorascoscopic surgery

4. Return to play—dependent on resolution of symptoms and x-ray findings, usually 4 weeks
   a. PSP—at increased risk for reoccurrence; history of multiple PSP, consider surgical correction

5. Air travel prohibited until x-ray resolution of the pneumothorax

B. Pneumomediastinum

1. Basics
   a. Dissection of free air into the mediastinal space and subcutaneous tissue
   b. Categorized as spontaneous or traumatic
   c. Risk factors: asthma, cystic fibrosis, recreational inhalational drug use (cocaine, methamphetamine, marijuana), intense physical activity, high-altitude or underwater activity
d. Associated injuries—pneumothorax, esophageal rupture, gastric perforation, foreign body aspiration

2. Diagnosis
   a. Clinical exam—chest pain; dyspnea; dysphagia; subcutaneous emphysema (crepitation on palpation of soft tissues over chest wall and in neck region); reduced or muffled heart sounds; and systolic crepitation (Hamman’s sign)
   b. Imaging
      i. CXR—radiolucent bubbles or streaks that outline the heart margins and great arteries (ring around the pulmonary artery)
      ii. Contrast esophagography to rule out esophageal rupture

3. Treatment
   a. Conservative treatment—analgesia, rest, avoidance of provocative maneuvers; usually resolves within 2 weeks.
   b. Consider and address possible underlying conditions

C. Hemothorax
   1. Basics
      a. Can occur with blunt or penetrating chest trauma—may be in association with pneumothorax or injuries to great vessels or cardiac structures
   2. Diagnosis and treatment
      a. Physical examination: dullness to percussion, diminished breath sounds at lung bases, respiratory distress, tachypnea
      b. Imaging—x-ray: upright and lateral decubitus, blunting of costophrenic angle
      c. Ultrasound can aid in diagnosis
   3. Management—tube thoracostomy: more than 1,500 mL of immediate drainage is an indication for surgical thoracotomy

D. Pulmonary contusion
   1. Basics
      a. Traumatic injury to the lung parenchyma leading to accumulation of blood or edema into the alveolar space; interferes with gas exchange

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Figure 32.1  Acute left-sided tension pneumothorax exhibiting hyperexpansion of the left lung, a visceral pleural edge, absence of lung markings lateral to the pneumothorax, and tracheal deviation.
b. CXR findings lag behind clinical picture—extent of injury not fully appreciated until 24 to 48 hours after injury

2. Diagnosis
   a. Physical exam—tachypnea, hypoxia, respiratory distress, hemoptysis
   b. Imaging—CXR: limited sensitivity acutely, patchy opacity
   c. CT—gold standard; nonsegmental, irregular opacities at lung periphery (Figure 32.2)

3. Management
   a. Supportive—pain control with analgesics, oxygen therapy, and hospital observation

4. Return to play
   a. Cleared to return 1 week after resolution of symptoms and CXR findings

E. Pulmonary embolism

1. Basics
   a. Fat embolus—from trauma/surgery to long bone elsewhere
   b. Most commonly from deep venous thrombosis (DVT) originating in the lower extremities
   c. Risk factors—genetic thrombophilia (factor V Leiden, deficiencies of protein C or S); oral contraceptive, hormone replacement, smoking, air travel greater than 4 hours, and pregnancy

2. Diagnosis
   a. High level of suspicion and predisposing clinical setting
   b. Physical exam—pleuritic chest pain, dyspnea, tachypnea, cough (may be nonproductive), hemoptysis, tachycardia, lower extremity swelling/pain
   c. CXR—low sensitivity may see Hampton’s hump (shallow wedge-shaped opacity in periphery of lung with base against pleural surface)
   d. ECG—sinus tachycardia, S1Q3T3, right axis deviation, T wave inversion in the anterior and inferior leads
   e. Ultrasound—right ventricle enlargement, plethoric inferior vena cava (IVC)
   f. Laboratory—D dimer: only beneficial in low-risk patients; high sensitivity but low specificity; negative predictive value nears 100%
      i. Risk stratification—Pulmonary Embolism Rule-out Criteria (PERC), Wells or Geneva rules
   g. Imaging—CT pulmonary angiography (gold standard) or ventilation—perfusion (V/Q) scan

![Figure 32.2](image)

Figure 32.2 A CT scan revealing diffuse, nonsegmental consolidation within the alveoli consistent with acute pulmonary contusion.
3. **Treatment**
   - a. Supportive—supplemental oxygen, analgesia
   - b. Anticoagulation
      - i. Immediate—subcutaneous low-molecular-weight heparin
      - ii. Long term (3–6 months) with warfarin
         - (A) Monitor with international normalized ratio (INR)
         - (B) Caution with contact sports—increased risk of bleeding from injuries while anticoagulated; wait 2 weeks after stopping
   - c. Discontinue oral contraceptives—especially preoperatively
   - d. Hypercoagulability evaluation includes homocysteine, antithrombin III, anticardiolipin antibody, prothrombin 20210A, factor V Leiden, and lupus anticoagulant levels (antithrombin mutations, prothrombin mutations)

**VII. Heart and Great Vessels**

A. **General considerations**
   1. Pericardium and pericardial space—pericarditis, pericardial effusion, cardiac tamponade
   2. Heart muscle—laceration, ventricular rupture, myocardial contusion
   3. Great vessels—ascending and descending aorta, vena cava, jugular, and subclavian veins

B. **Commotio cordis**
   1. **Basics**
      - a. Direct high-speed impact to the chest overlying the cardiac silhouette—greatest risk when object speeds reach 40 to 50 mph
      - b. Mechanism—increased left ventricular pressure 15 to 30 msec prior to the peak of the T wave induces inappropriate ventricular repolarization and ventricular fibrillation
   2. **Management**
      - a. Advanced Cardiovascular Life Support (ACLS) algorithm with emergent defibrillation
         - i. Time to automated external defibrillator (AED) is critical—if delayed more than 3 minutes less than 3% chance of survival
      - b. Precordial thump

C. **Pericarditis**
   1. **Basics**
      - a. Inflammation of the pericardial sac
      - b. Causes—infectious, autoimmune, neoplastic, myocarditis, or trauma
   2. **Diagnosis—clinical diagnosis**
      - a. Clinical exam—pleuritic chest pain, positional chest pain (worse when supine and improved with leaning forward), pericardial friction rub
      - b. EKG—diffuse ST elevation with PR depression (Figure 32.3)
      - c. Ultrasound—to rule out associated pericardial effusion
      - d. Laboratory—elevation in white blood cell count, inflammatory markers, and cardiac biomarkers
   3. **Treatment**
      - a. Symptomatic management—NSAIDs and colchicine
   4. **Return to play**
      - a. Current guidelines suggest hold from play for 3 months after normalization of biomarkers and resolution of symptoms

D. **Pericardial effusion and cardiac tamponade**
   1. **Basics**
      - a. Pericardial effusion—pathologic fluid within the pericardial sac; consider in athletes with trauma, autoimmune disease, hypothyroidism, or acute pericarditis
      - b. Pericardial tamponade—large pericardial effusion under pressure; constricts ventricle size, inhibits diastolic compliance

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2. Diagnosis
   a. Clinical exam—not sensitive with small pericardial effusion
      i. Cardiac tamponade—muffled heart sounds, elevated jugular venous pressure, arterial pulsus paradoxus (significant decrease in systolic blood pressure and pulse amplitude with inspiration), hypotension
   b. ECG—low voltage or electrical alternans
   c. CXR—enlarged cardiac silhouette
   d. Echocardiogram diagnostic—defines size of effusion and effect on ventricle compliance

3. Treatment
   a. Unstable—ultrasound guided percutaneous pericardiocentesis; decompress heart, increase stroke volume, filling pressure, and restore adequate circulation
   b. Surgical exploration to repair any laceration/rupture that resulted in bleeding and tamponade

E. Aortic dissection

1. Basics
   a. Intimal tear of the aorta creating a false lumen through the media
   b. Caused by acute traumatic deceleration, underlying connective tissue disease (Marfan's), valve abnormality (congenital bicuspid aortic valve)
   c. Stanford classification: Type A—ascending aorta (surgical intervention); Type B—beyond brachiocephalic (medical management)

2. Diagnosis
   a. Clinical—maximal onset, tearing chest pain with radiation to the back, chest pain with neurologic deficit, aortic regurgitation murmur
   b. Vital signs—20 mmHg blood pressure variation between the upper extremities; hypotension is late finding
   c. Imaging
      i. CXR—widening of mediastinum, abnormal aortic contour, pleural effusion, pleural apical cap
      ii. CT angiography—gold standard

3. Management
   a. Systolic blood pressure control—goal less than 110 mmHg

VIII. Penetrating Trauma

   A. Indoor sports—fencing or fixed stationary object

Figure 32.3  EKG showing diffuse ST elevation and PR depression suggestive of pericarditis.
III: DIAGNOSIS AND TREATMENT OF SPORTS INJURIES AND CONDITIONS

1. Outdoor—field events (javelin), archery, motor vehicle equipment
2. Associated injuries: laceration—arterial or venous bleeding; pulmonary—hemothorax, pneumothorax, pulmonary laceration, hemopneumothorax; tracheobronchial injuries; cardiac—ventricle rupture resulting in tamponade
   a. Open pneumothorax—communication between the environment and pleural space; creates a “sucking chest wound” where air is drawn into the pleural cavity

B. Management
1. Leave object in place until transfer to nearest emergency room (ER) for more definitive care
2. May require surgical exploration and treatment of associated injuries
3. Open pneumothorax—three-sided occlusive dressing; creates a flap valve, prohibiting air entrance, but allowing air to escape during expiration

IX. Injuries to the Breasts
A. Breast contusion—direct trauma; symptomatic management
B. Breast protection
1. For contact sports—chest protectors; may be modified for women
C. Lactating women
1. Moderate exercise—no effect on milk supply or milk composition
2. Maximum intensity exercise—increased lactate levels and decreased immunologic factors in breast milk
D. Breast cancer survivors
1. Tissue scarring “pulling” with activity—treat with physiotherapy
2. Lymphedema—resistance, aerobic, and flexibility exercise do not increase lymphedema; consider compression garments

Recommended Reading

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