Sudden Cardiac Death in the Athlete

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“Hearts too Young to Die”
Causes of SCD in athletes

• Coronary vessels
  – CAD
    • Arterial dissection
    • Arterial vasospasm
  – Congenital coronary artery malformations
    • LM from right coronary sinus
    • RCA from left coronary sinus
    • Intramural coronary artery (LAD)
• Endocardium
  – Valvular disease
    • AS
    • MVP
• Myocardium
  – Cardiomyopathies
    • Hypertrophic CM
    • Dilated CM
    • Arrhythmogenic right ventricular CM
  – Myocarditis
• Conduction system abnormalities
  – WPW
  – Long QT syndrome
• Aortic rupture (Marfan’s syndrome)
• Extrinsic
  – Medications
  – PES’s
  – Commotio cordis
Epidemiology of SCD

• Overall rate
  – Industrialized countries
    • 50-75:100,000
  – US
    • 166,000-300,000 / yr

• Incidence in age < 35
  – Einarsson, etal.
    • Iceland – 1.38:100,000
  – Wisten, etal.
    • Sweden – 0.93:100,000
  – Corrado, etal.
    • Italy – 0.9:100,000

SCD in athletes

- Incidence in young athletes
  - Corrado, et al.
    - 2.3:100,000
  - Van Camp, et al.
    - US high school males – 0.66:100,000
    - US college males – 1.45:100,000
  - Moran, et al.
    - Minnesota high school – 0.46:100,000

Causes of SCD in Athletes

< 35 y/o

Adapted from Maron, B; etal. Circulation. 2007 Mar 27. 115(12): 1643-1655.
Regional variation in cause of SCD

<table>
<thead>
<tr>
<th>Condition</th>
<th>US (n=134)</th>
<th>Italy (n=55)</th>
</tr>
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<tbody>
<tr>
<td>CAD</td>
<td>2%</td>
<td>18%</td>
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<tr>
<td>Anomalous coronary artery</td>
<td>23</td>
<td>16</td>
</tr>
<tr>
<td>MVP</td>
<td>2</td>
<td>11</td>
</tr>
<tr>
<td>AS</td>
<td>4</td>
<td>--</td>
</tr>
<tr>
<td>HCM</td>
<td>46</td>
<td>2</td>
</tr>
<tr>
<td>ARVC</td>
<td>3</td>
<td>22</td>
</tr>
<tr>
<td>Dilated CM</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Myocarditis</td>
<td>6.5</td>
<td>13</td>
</tr>
<tr>
<td>Conduction abnormality</td>
<td>0.5</td>
<td>9</td>
</tr>
<tr>
<td>Aortic rupture</td>
<td>5</td>
<td>2</td>
</tr>
<tr>
<td>Other</td>
<td>5</td>
<td>7</td>
</tr>
</tbody>
</table>


SCD in older athletes

• Maron, etal.
  – 80% of deaths due to CAD
    • Diagnosed via thrombus or plaque disruption
  – Of these, ½ had prodromal symptoms or previous knowledge of CAD

• Zipes, etal.
  – Incidence during exercise stress testing
    • 6.7-8.3:100,000


Causes of SCD in Athletes
> 35 y/o

- CAD: 80%
- HCM: 5%
- Valvular Disease: 10%
- Unknown: 5%

Adapted from Maron, B; etal. J Am Coll Cardio. 1986 Jan. 7(1): 204-214
“Athlete’s Heart” v. pathology

• Athlete’s Heart
  – Morphologic changes of heart due to athletic training
  – Inc. LV cavity dimension
  – Inc. LV wall thickness
  – Inc. cardiac mass
• Inc. CO / VO$_2$ with activity
  – No change at rest

• Pathologic heart
  – Asymmetric LVH
  – Compromised LV systolic or diastolic function
• Dec. CO / EF
Types of athletic activity

• Dynamic / isotonic
  – Change fiber length without changing tension
  – Ex: distance running

• Static / isometric
  – Change tension without changing fiber length
  – Ex: weightlifting
Physiologic changes with exercise

• **Dynamic exercise**
  – Inc. CO and inc. venous return
  – Due to peripheral muscle contraction and inc. cardiac activity
  – INCREASED PRELOAD

• **Static exercise**
  – Inc. SVR
  – INCREASED AFTERLOAD
    • SBP > 300 mmHg

Hypertrophy due to exercise

• Dynamic
  – Fibers added in series
  – Allows accommodation of increased volume

• Static
  – Fibers added in parallel
  – Provides added strength to overcome SVR
Cardiac morphologic changes

- **Dynamic**
  - Inc. LV dimension
    - Inc. dimension correlates with inc. VO2 max
  - Inc. septal and posterior wall thickness
    - Maintains LaPlace’s Law

- **Static**
  - Minimal change in LV dimension
  - Markedly inc. septal and posterior wall thickness

Physiologic v. pathologic hypertrophy

- Vinereanu, et al.
  - HCM v. systemic HTN v. athletes v. controls
  - Evaluated morphology with echocardiogram
  - Pathologic hypertrophy
    - Subendocardial fibrosis
    - Diastolic dysfunction
      - Decreased mitral annular velocity
  - Physiologic hypertrophy
    - No change in contractility
    - Normal diastolic function

Evaluation Methods

• Comparison between 2D echo, 3D echo and MRI to evaluate LV dimensions
• LV mass, LV volume (end systolic and end diastolic), EF
• 18 elite Italian male athletes and 12 sedentary age matched controls
• 3D echo and MRI results correlate closely

Evaluation Methods

- LV volume (end systolic and end diastolic), EF, LV mass index, stroke volume index

- 120 total subjects
  - 18 healthy volunteers
  - 25 competitive athletes
  - 35 patients with HCM
  - 24 patients with aortic stenosis
  - 18 patients with systemic hypertension

Evaluation Methods

• ROC diastolic wall to volume ration of < 0.15 mm x m² x mL⁻¹ differentiated athlete’s heart from other forms of hypertrophy
• 99% specificity

Evaluation Methods

- 60 total subjects
  - Hypertensive patients with LVH
  - Semi professional triathletes with LVH
  - Professional soccer players
  - Sedentary controls without CAD

- Myocardial contrast echo at rest and after adenosine

- Relative blood volume (rBV) and exchange frequency measured
  - rBV corresponds to the intravascular volume
  - Exchange frequency corresponds to resistance to blood flow

Evaluation Methods

• Hypertensive patients had lower rBV and higher exchange frequency than other groups

• rBV < 0.114 ml mL⁻¹ 93% sensitive and 100% specific for differentiating hypertensive patients from athletes

The Literature

• Pelliccia et al
  – 1309 elite Italian athletes
  – 55% with normal LV end diastolic cavity dimension (<54 mm)
  – 14% with LV dimension > 60 mm
  – Systolic function normal; no wall motion abnormalities noted
  – No cardiac symptoms over follow up period (mean 4.7 years)

Sport Specific Differences

- Spirito et al

- Rowers, swimmers, and distance cyclists have greatest increase in LV cavity dimension and wall thickness
- Wrestlers and weightlifters had highest wall thickness
- Females had smaller increases in both measures than males in the same sport

More Literature

- Iglesias-Cubero et al
  - Cyclists, soccer players, canoeists
  - H & P, EKG, echocardiogram, exercise testing
  - Cyclists had significantly higher LV diastolic dimensions and wall thickness
  - Significant correlation between morphologic changes and VO$_2$ max

More Literature

• Douglas et al
  – 235 Ironman triathletes
  – Testing within one week of competition
  – 61 showed increased ventricular dimensions
    • 22 % of all men
    • 43 % of all women
  – Significant correlation between VO₂ max and cavity dilation
  – No change in cardiac function

More Literature

- Macchi et al
  - National team members vs. matched controls
  - LV cavity diameter, septum, posterior wall and LA cavity diameter all significantly higher in athletes
  - Female athletes had significantly higher LA cavity dimensions vs. controls than did males
  - Significantly more valvular regurgitation in athletes than controls

Effect on Presentation
Another EKG
Associated EKG Changes

• 91 % of all athletes have sinus bradycardia; sport dependent
• 10-33 % of athletes have some conduction delay; first degree AV block most common
  – 40 % of athletes with first degree block actually have Mobitz 1 rhythm; abnormality disappears with exercise and deconditioning. Benign condition
  – Mobitz 2 is not associated with athletic hypertrophy and should be evaluated as a pathologic condition
• Incomplete RBBB associated with athletic hypertrophy; complete RBBB is not
More on EKG Changes

• J point elevation
  – Common in anterior and inferior leads as opposed to global elevation in pericarditis
• ST segment elevation common, ST segment depression rare and warrants further evaluation
• LVH
• Increased P wave amplitude and notched P waves
• PACs and PVCs common, particularly in distance runners
QT Changes

• Spataro et al
  – Italian rowers
  – All with evidence increased LV diameter on echocardiogram
  – Increased T wave voltage during low intensity training
  – Transient flattening of T waves during high intensity training
  – No change in QT, QTc interval or QT dispersion
  – No change in cardiac function
More on QT Changes

- Mayet et al
  - Endurance athletes vs. soccer players vs. controls
  - Echocardiogram, EKG
  - Athletes with increased LV diameter
  - No change in QT interval, QTc or QT dispersion between groups
  - Important because QT dispersion is a potent arrhythmic stimulus

Congenital Long QT Syndrome

- 150 known mutations to cardiac ion channel mutations
- Autosomal dominant inheritance most common
- Palpitations, presyncope/syncope, seizures and cardiac arrest most common manifestations
  - Different manifestations with different mutation patterns
- EKG: QTC >440 msec in males and >460 msec in women is diagnostic
- Beta blocker is treatment of choice; ICD placement common
- Risk stratify based on QTC and genotype
  - Most sports participation prohibited

Importance to General Internists

- New role of general internists in sports medicine
  - Role of the crowd
  - Role as team physician
- 3 million high school and college athletes in 10 year period
  - 160 non-traumatic deaths; cause of death known in 136
  - 100 cardiac related, 51 from hypertrophic cardiomyopathy (HCM); 5 others likely due to HCM

Commotio Cordis

- Normal heart subjected to blunt chest trauma directly over heart (R on T phenomenon)
- Ventricular fibrillation most common rhythm
- Greater chest wall compliance places athlete at higher risk (greater displacement of forces directly onto myocardium)
- Younger athletes at significantly higher risk
Commotio Cordis

- Link et al
- Swine study
- 30 mph, timed with cardiac cycle
- 6 with multiple impacts, 12 with single impact
- 9/10 impacts 30-15 msec before T wave peak --> v tach
- No impact at any other time generated v tach

Coronary Artery Anomalies

- Anomalous origin of left coronary artery
- Slit present to supply anterior heart
- Unable to maintain adequate cardiac output during exercise
Origin LM from Right Coronary Sinus

Aorta

RC

Pulmonary Trunk

LC

LAD
Aorta

RC

Pulmonary Trunk

LC

LAD

Origin RC from Left Coronary Sinus
Arrhythmogenic RV

- Increased fatty infiltration of right ventricle
  - Fibrofatty infiltrate possible
- Trigger unknown
- Likely autosomal dominant
- Linked to ryanodyne receptor
- EKG normal
- First presentation usually cardiac death
SCD, AEDs and the NCAA

- 91% NCAA Division 1 schools with AEDs
- Average of 4 AEDs per school (range 1-30)
- Training room most common location
- 35 reported SCD uses
  - 27/35 (77%) in older, non-athletes
- Immediate resuscitation in 19/35 (54%)
- AED delivered shock improved survival to 71%
- 5 athletes with SCD; none resuscitated

Endurance Events & the Heart

• Belonje et al
• 5 year review of 36 km beach cycle race and 21 km run (half marathon)
• 62,862 athletes
• 3 runners, 1 cyclist collapsed
• 2 MIs, 1 catecholamine induced collapse, 1 heat stroke without cardiac changes
• Prevalence = 0.006%

Marathons and the Heart

- 30 non-professional runners in the 2005 Boston Marathon
- Troponin-T, d-dimer, BNP, matrix metallproteinase-9 (MMP-9)
- Significant increases in MMP-9 and d-dimer
- 7 runners with troponin > 0.05 ng/dl
- Myocardial stunning
- Systemic inflammatory response +/- exertional rhabdomyolysis

Marathons and the Heart

- 27 marathoners
- BNP, cortisol, troponin-T
- Blood drawn before, immediately and 24 hours after the race
- All levels significantly increased immediately after the run
- All levels returned to normal 24 hours after the run

Marathons and the Heart

- 60 nonelite runners in the 2004 and 2005 Boston Marathons
- Echocardiography, troponin-T, NT-BNP Echo: altered diastolic filling, increased pulmonary pressures, increased right ventricular dimensions, decreased right ventricular systolic function
- 60% with post-race troponin-T elevations, 40% to level of ischemia
- NT-BNP increases significant
- Training >45 miles/week had fewer increases than those running <35 miles/week

The Role of Stimulants

- Caffeine
- Pseudoephedrine
- Ephedra

- Removed from the banned substance list by the NCAA, US ADA, WADA

- Heat injury risk from tachycardia/ hypertension
Caffeine: Is It Safe?

- 15 healthy subjects
- Supine bicycle testing at sea level and altitude simulated conditions
- Testing with and without 200 mg caffeine (2 8 oz cups of coffee)
- Significant decreases in myocardial blood flow at sea level; greater difference at altitude

Screening Guidelines

• Several protocols but no consensus
  – AHA (1)
  – NCAA (2)
  – Bethesda Conference (3)
  – The British (4)
Georgia Tech/Atlanta Braves Approach

- Screening questionnaire
- Annual H & P with focus on cardiovascular exam
- Baseline EKG when entering college/joining the Braves
- Echo with any potential EKG abnormality
- Particular focus on African-American males

The Italian Screening Protocol

• 4450 national caliber athletes passed initial screening
  – History, physical examination and EKG

• Echocardiogram to evaluate for undetected HCM

• No HCM but 13 in the gray zone
  – Myocarditis (4)
  – MVP (3)
  – Marfan’s (2)
  – Aortic regurgitation with bicuspid valve (2)
  – Arrhytmogenic RV (1)

Cost Effective?

• General screening examination: 25 euros
• With EKG: 40 euros
• Athletes < 18 covered by the National Health System

Importance of EKGs

• 12,550 athlete database
  – 229 age and gender matched athletes with normal EKGs
  – 81 with ≥ 2 mm ST depressions in at least 3 leads
    • No known cardiac disease
    • Yearly exams (EKG and echo) for 9 ± 7 years

• 5 (study group) ultimately diagnosed with HCM
  – 1 sudden death at age 24
  – Symptoms in 4 after 12 ± 5 years

• No controls developed HCM
  • Pelliccia A et al. NEJM. 358(2);152-161.
Ryan Shay?
Injection and Splinting Basics

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Scott Vargo, MD
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Supplies

• Local anesthetic = lidocaine 1%
• Steroid = triamcinalone 40 mg/mL
• Needle guage and length listed
• Prep and drape area in normal sterile fashion
• Use sterile gloves for procedure
• Consider hurricane spray
## Injection Basics

<table>
<thead>
<tr>
<th>Location</th>
<th>Landmark</th>
<th>Local</th>
<th>Steroid</th>
<th>Needle</th>
</tr>
</thead>
<tbody>
<tr>
<td>AC joint</td>
<td>Superior aspect of AC joint; insert at 30° angle</td>
<td>1 mL</td>
<td>10 mg</td>
<td>25 g 0.5 inch</td>
</tr>
<tr>
<td>Shoulder</td>
<td>Posterior angle of acromion</td>
<td>4 mL</td>
<td>30 mg</td>
<td>21 g 2 inch</td>
</tr>
</tbody>
</table>
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<th>Local</th>
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<th>Needle</th>
</tr>
</thead>
<tbody>
<tr>
<td>Elbow</td>
<td>Space between radial head and olecranon</td>
<td>1.5 mL</td>
<td>20 mg</td>
<td>25 g 0.5 inch</td>
</tr>
<tr>
<td>Lateral epicondyle</td>
<td>Lateral epicondyle and extensor origin</td>
<td>1 mL</td>
<td>10 mg</td>
<td>25 g 0.5 inch</td>
</tr>
</tbody>
</table>
## Injection Basics

<table>
<thead>
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<th>Location</th>
<th>Landmark</th>
<th>Local</th>
<th>Steroid</th>
<th>Needle</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thumb CMC</td>
<td>Base of thumb; distract to open joint</td>
<td>1 mL</td>
<td>10 mg</td>
<td>25 g 0.5 inch</td>
</tr>
<tr>
<td>DeQuervain’s</td>
<td>Distal radius proximal to proximal carpal row</td>
<td>1 mL</td>
<td>10 mg</td>
<td>25 g 0.25 inch</td>
</tr>
</tbody>
</table>
## Injection Basics

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<thead>
<tr>
<th>Location</th>
<th>Landmark</th>
<th>Local</th>
<th>Steroid</th>
<th>Needle</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carpal tunnel</td>
<td>Radial aspect of palmaris longus</td>
<td>0.5 mL</td>
<td>20 mg</td>
<td>23 g 1.5 inch</td>
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</table>
# Injection Basics

<table>
<thead>
<tr>
<th>Location</th>
<th>Landmark</th>
<th>Local</th>
<th>Steroid</th>
<th>Needle</th>
</tr>
</thead>
<tbody>
<tr>
<td>Greater trochanter</td>
<td>Greater trochanter (point of maximum tenderness)</td>
<td>2 mL</td>
<td>20 mg</td>
<td>23 g 1.25 inch</td>
</tr>
<tr>
<td>Knee</td>
<td>Superiolateral aspect of patella</td>
<td>4 mL</td>
<td>40 mg</td>
<td>21 g 1.5-2 inch</td>
</tr>
</tbody>
</table>
# Injection Basics

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<thead>
<tr>
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<th>Local</th>
<th>Steroid</th>
<th>Needle</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ankle</td>
<td>Plantar flexed ankle; lateral to tibialis anterior tendon</td>
<td>1.5 mL</td>
<td>30 mg</td>
<td>23 g 1.25 inch</td>
</tr>
<tr>
<td>First metatarsal</td>
<td>MTP joint; distract to open joint</td>
<td>1 mL</td>
<td>20 mg</td>
<td>25 g 0.5 inch</td>
</tr>
</tbody>
</table>
Splinting
Splinting Basics

- Padding is critical, particularly over bony prominences
- Plaster with padding vs. precombined vs. pre-made splint
- 3 inch, 4 inch or 5 inch plaster/precombined materials available
- Consider patient habitus when choosing materials
Thumb Spica

- DIP to proximal 1/3 of forearm
- Wrist extended 15
- Thumb abducted
- May split material to wrap around thumb or use separate piece
- Indications: Suspected/confirmed scaphoid fracture, ulnar collateral ligament injury
Volar Splint

• Mid PIP-DIP to proximal 1/3 of forearm
• Wrist extended $15^0$
• Indications: Wrist trauma without fracture, 2nd or 3rd metacarpal fracture
Sugar Tong

- Volar splint extended around the elbow and the dorsal aspect of the forearm to the mid PIP-DIP
- Indications: Radial head fractures, elbow dislocations
Lower Leg Splint

• 2 components
  – Posterior component: metatarsals to mid gastrocnemius
    • Ankle and subtaus should be in neutral position
  – Sugar tong: medial-lateral u shaped piece, including sling under the arch

• Indications: ankle sprains, foot fractures
  – Can splint ankle/subtalus in plantar flexion for Achilles tear, popliteus rupture, gastrocnemius injury
Goals

• Review basic anatomy of the shoulder and the knee
• Discuss common musculoskeletal conditions
• Consider treatment options available
• Practice techniques
Shoulder Basics

• Roof: acromion and clavicle
• Socket: glenoid
  – Glenoid is very shallow
  – Labrum is cartilaginous lining; increases the depth of the capsule
• Ball: humeral head
Rotator Cuff

• Four components
  – Supraspinatus
  – Infraspinatus
  – Teres Minor
  – Subscapularis
Shoulder Basics: Range of Motion

• Abduction
  – 0-180\(^0\)
  – Deltoid abducts to 60\(^0\)
  – Supraspinatus completes the arc of motion

• Forward elevation
  – 0-180\(^0\)
  – Supraspinatus

• Internal rotation
  – To lumbar spine
  – Subscapularis

• External rotation
  – 0-45\(^0\)
  – Infraspinatus and teres minor
The Shoulder Exam

• Supraspinatus
  – Resistance with elbow extended and thumbs to the floor

• Infraspinatus/teres minor
  – Externally rotate against resistance

• Subscapularis
  – Push against resistance while in full internal rotation
  – Can be performed against abdomen if internal rotation limited
Rotator Cuff Tears

- **Traumatic**
  - Fall to shoulder
  - Forced abduction
    - Starting lawn mower

- **Chronic**
  - Overhead activities

- **Supraspinatus** most commonly torn
  - Limited abduction
  - Weakness with abduction

- **MRI**

- **Surgical repair usually needed**
Rotator Cuff Tendinopathy

• Supraspinatus most commonly affected
  – Chronic overhead activity
• Anterior shoulder pain
• Weakness but motion maintained
• Treatment
  – NSAIDs
  – Periscapular strengthening
  – Relative rest
To Inject or Not to Inject?

• Consider remaining evaluation
  – Injection may limit MRI interpretation; is the fluid an effusion or is it the injection?
Impingement

- Acromion morphology
  - Type 1: flat
  - Type 2: slightly curved
  - Type 3: large hook
- Clavicle rarely responsible
  - Osteolysis possible
- Pain with abduction and cross body adduction
Biceps Tendinopathy

- Anterior shoulder pain
- Chronic upper extremity activity
- Positive Speed’s test
  - Pain with forward elevation of supinated hand

Treatment
- Relative rest
- NSAIDs
- Therapy
  - Ultrasound
Adhesive Capsulitis

• Inability to range shoulder without any preceding trauma or overuse
  – Mechanism unknown

• Common in diabetics and those with autoimmune diseases
  – 2% incidence in general population, 11% in diabetics
Adhesive Capsulitis

• Three stages
  – Freezing: increasing pain and decreasing ROM
  – Frozen: greatest loss of motion
    • 3-4 months after onset of pain
  – Recovery: spontaneous return of ROM
    • Return to full ROM rare without other interventions
Adhesive Capsulitis

• Examination
  – No bony or soft tissue tenderness
  – Distal neurovascular exam intact

• Treatment
  – Physical therapy
    • Climbing the wall
    • Concentric circles
  – Examination under anesthesia
Patellofemoral Pain Syndrome

- The most common diagnosis for knee pain
- Anterior knee pain
- Pain after prolonged sitting
  - Theater sign
- Pain with stairs
  - Particularly when walking down stairs
- Women >> men
Why the Gender Difference?

• Quadriceps muscles are main controllers of patellar motion
  – Vastus medialis oblique
    • Key muscle
  – Vastus intermedius
  – Vastus lateralis
  – Rectus femoris
More on Gender Differences

• Q angle
  – Intersection of line drawn from the anterior superior iliac spine to the tibial tubercule and line down the center of the femur to the tibial tubercule

• Normals
  – Men: < 12°
  – Women: < 15°
  – Women’s quadriceps inherently weaker due to the greater force lever
Patellofemoral Pain

• Examination findings
  – Lack of defined VMO
  – Abnormal patellar motion (j tracking)
  – Positive single leg squat

• Treatment
  – Injections not helpful
  – Weight loss
  – STRENGTHENING
    • Wall slides
    • Leg extensions/curls
Osteoarthritis

- Loss of articular cartilage
- 3 compartments
  - Medial
    - Most commonly affected
  - Lateral
  - Patellofemoral
- Valgus deformity in extreme cases
- Increasing knee pain
  - Generalized pain
  - Decreased activity
X-ray Findings

• AP, lateral and sunrise views
  – WEIGHT BEARING AP VIEW CRITICAL
• Loss of joint space
  – Bone on bone in extreme cases
  – Valgus deformity
• Subchondral sclerosis
• Subchondral cysts
• Osteophyte formation
Treatment

• Physical therapy
• Pain control
• Unloading brace
• Injections
  – Steroids/local anesthetic
  – Hyaluronic acid
• Surgery
  – Arthroscopic debridement
  – Replacement
    • Unicompartment vs. total knee
Meniscal Tears

- C shaped cartilaginous shock absorbers between the tibia and femur
- Limited blood supply
- Pie crust analogy
- Tears common
  - Traumatic: axial load and twist
  - Degenerative tears
Meniscal Tears

• Chief concern
  – Joint line knee pain
  – Locking
  – Clicking

• Physical examination
  – Mild effusion
  – Joint line tenderness
  – McMurray’s test
    • Extend and rotate the flexed knee while holding the knee at both the medial and lateral joint lines
    • Positive = Appreciable clunk as meniscal fragment slides between the tibia and femur
      – May recreate pain
Meniscal Tears

• Imaging
  – MRI is not always a definitive study
  – Arthroscopy is the gold standard

• Treatment
  – Pain control
  – Physical therapy
  – Arthroscopy
    • Usually excision of fragment
    • Repair possible in white zone if diagnosed early
Summary

• Anterior shoulder pain is a common presenting concern
  – Rotator cuff pathology must be differentiated from biceps tendinopathy

• Patellofemoral pain is the most common diagnosis in those with knee pain
  – Pain with stairs and single leg squat most sensitive findings
Questions?