Rheumatoid Arthritis: Early Diagnosis, Early Treatment

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Disclosures

- None

At the end of this talk, you should be able to:

- Recognize the clinical features and differential diagnosis of early rheumatoid arthritis (RA)
- List some key factors in the pathogenesis of RA
- Describe the laboratory evaluation helpful in early diagnosis
- Have an understanding of treatment strategies in RA
- Know what you can do to help the rheumatologist in the co-management of RA

Case

- 37 year-old woman
- 7-week history of progressive polyarthralgias
- Pain in wrists, hands and feet
- Swelling in some joints, decreased hand function
- Morning stiffness for 3 hours
- Excessive fatigue
- ROS otherwise non-contributory
- Some relief from ibuprofen

Case

- Exam with swelling and tenderness at the bilateral wrists, 2nd MCPs, and 2nd PIPs, and bilateral MTP joints; no nodules
- Rest of exam including skin is normal
- Labs with mild normocytic anemia, mildly elevated ESR 28, normal renal and liver function tests
- Radiographs of hands with some soft tissue swelling around the wrists and PIPs, otherwise normal

Case

- Does she have inflammatory arthritis?
  - Yes
  - History of joint swelling, early morning stiffness lasting ≥30 minutes, systemic symptoms such as fatigue, improvement of symptoms with anti-inflammatory medication
  - Objective evidence of joint swelling and tenderness on examination
  - Raised ESR or CRP, normocytic normochromic anemia; could also have thrombocytosis, low albumin, raised alkaline phosphatase
- Could she have rheumatoid arthritis?
  - Yes

Rheumatoid Arthritis- Definition

- Chronic (>6 weeks), systemic, inflammatory arthritis
- Typically symmetrical joint involvement with polyarticular pattern
- Small joints of the hands and feet (wrists, MCPs, PIPs, MTPs)
- RF or CCP antibody may be positive (70-80%)
- Associated with erosive joint disease and extra-articular features

Reality can be more complex!

- Objective signs may be lacking or have been suppressed by anti-inflammatory medication
- Joint swelling can be difficult to identify in obese patients
- The sensation that joints are swollen may be reported even by some patients with fibromyalgia
- Osteoarthritis as well as RA can cause morning stiffness, though in osteoarthritis it usually lasts less than 30 minutes
- Inflammatory markers such as the ESR or C-reactive protein (CRP) are normal in about 60% of patients with early RA
- In a patient with preceding osteoarthritis, radiographic changes can be misleading, especially if those suggestive of inflammatory arthritis have not yet developed.

Furthermore… Variable RA presentations

- Polymyalgic onset- elderly, presents acutely with stiffness predominantly in the shoulders and pelvic girdle. ESR high usually. Good response to prednisone 15-20 mg/d. Later peripheral joint inflammation appears.
- Palindromic onset- recurrent episodes of pain, swelling, redness affecting any one joint or several joints at a time, lasting 24-48 hours, mimicking gout or pseudogout
- Systemic onset- non focal weight loss, fatigue, depression, fever, extra-articular features such as serositis, articular manifestations absent or subtle.
- Persistent monoarthritis- one single large joint, such as knee, shoulder, ankle or wrist. May mimic chronic infection.

Other conditions may look like RA

- Postviral arthritis—e.g. parvovirus, mumps, rubella, hepatitis B and C
- Seronegative spondyloarthritis—e.g. psoriatic arthritis, inflammatory bowel disease, reactive arthritis
- Connective tissue diseases—e.g. systemic lupus erythematosus, scleroderma, MCTD, vasculitis
- Inflammatory Osteoarthritis
- Crystal arthritis—e.g. polyarticular gout, pseudogout
- Miscellaneous—e.g. sarcoidosis, thyroid disease, infective endocarditis, paraneoplastic syndromes

Additional Investigations

- Review of Systems, social and exposure history
- Laboratory tests

Case

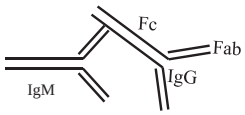
- Additional tests:
  - RF
  - CCP antibody
  - ANA
  - Urinalysis
  - Viral panel
  - Xrays

Additional Investigations

- Rheumatoid factor

Serologic Testing in RA: Rheumatoid Factor

- Rheumatoid Factor



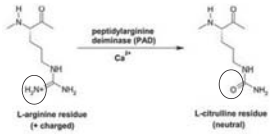
- Sensitivity 60-80%
- Specificity <70%
- Other causes of +RF
  - Infections
  - Malignancy
  - Other rheumatic diseases
  - Health

Additional Investigations

- Rheumatoid factor
- CCP antibody

Serologic Testing in RA: CCP Antibody

Anti-cyclic citrullinated peptide antibody



Citrullination:
- Post translational deamination of arginine to citrulline
-Occurs during cell-death and tissue inflammation
-Important consequences for the structure and function of proteins
-New epitopes, immunogenic

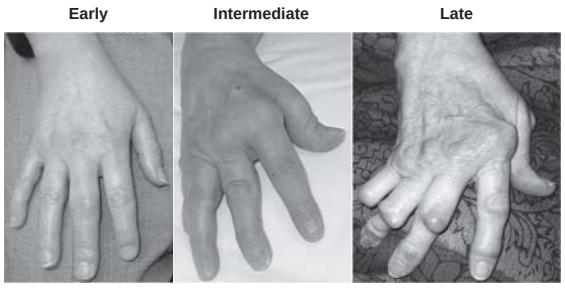
hopkinsarthritis.org

- Implicated in the pathogenesis of RA
- Sens 70% Spec 95%
- 40% of RF negative patients are ACPA+
- Detected in preclinical state
- Predicts more severe course and erosive disease

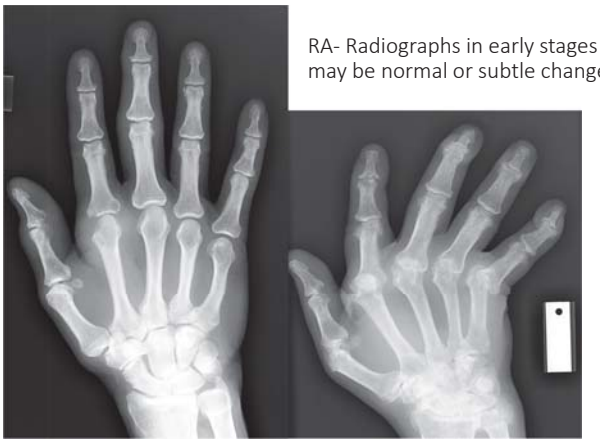
Additional Investigations

- Rheumatoid factor
- CCP antibody
- Antinuclear antibody—good screening test for SLE but sometimes positive in conditions including RA (30%) and in health
- Urinalysis—microscopic hematuria/proteinuria can indicate connective tissue disease or vasculitis
- Viral antibody titers—parvovirus IgM, hepatitis B/C, HIV
- Serum urate/synovial fluid analysis—to assess probability of gout/pseudogout
- Plain radiographs of hands and feet—can be normal in early RA or show periarticular soft tissue swelling/osteopenia/marginal erosions; erosions occur earlier in feet, so the feet should be X-rayed even in patients without foot symptoms

Stages of RA

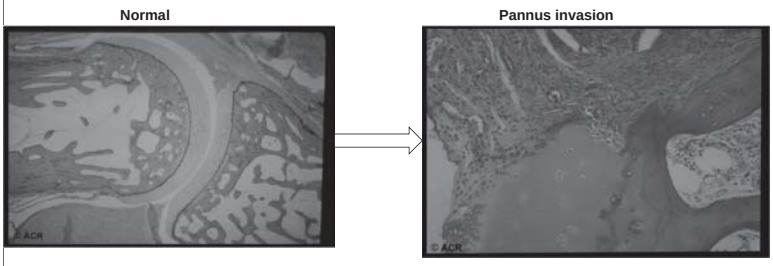


Courtesy of J. Cush, 2002.

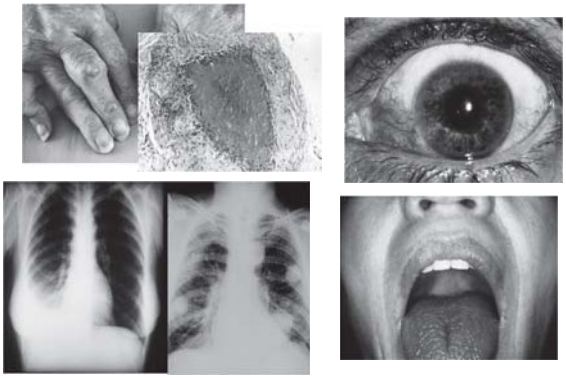


RA- Radiographs in early stages may be normal or subtle changes

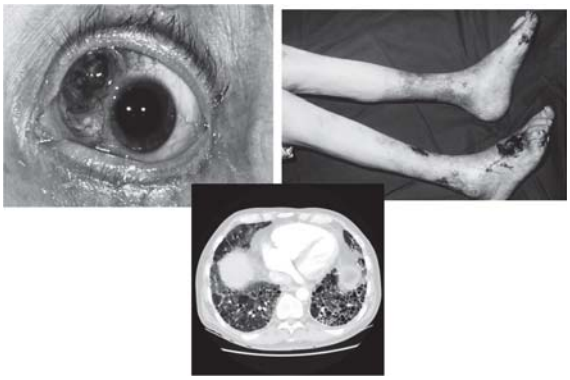
Cartilage and Subchondral Bone Invasion



RA- Extra-articular Manifestations



RA- Organ Threatening



ACR RA Classification- 1987

- Morning stiffness in and around the joints lasting greater than one hour
- Joint swelling of 3 or more joints
- Joint swelling of hand joints
  - wrist, MCP, PIP
- Symmetrical joint involvement
- Rheumatoid nodule
- Positive RA factor
- X-ray findings showing bony erosions

Symptoms must be ongoing for 6 weeks or longer; Need to have 4 out of 7 criteria for definitive diagnosis

ACR Classification- 2010

Table 3. The 2010 American College of Rheumatology/European League Against Rheumatism classification criteria for rheumatoid arthritis

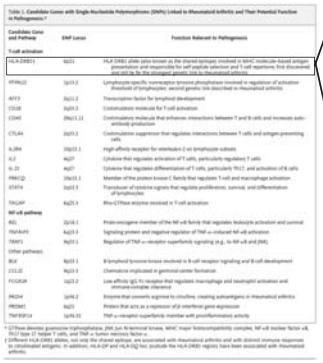
	Score
Target population (Who should be tested?): Patients who	
1) have at least 1 joint with definite clinical synovitis (swelling)*	
2) with the synovitis not better explained by another disease†	
Classification criteria for RA (score-based algorithm: add score of categories A–D; a score of ≥6/10 is needed for classification of a patient as having definite RA)‡	
A. Joint involvement§	
1 large joint¶	0
2–10 large joints	1
1–3 small joints (with or without involvement of large joints)#	2
4–10 small joints (with or without involvement of large joints)	3
>10 joints (at least 1 small joint)**	5
B. Serology (at least 1 test result is needed for classification)††	
Negative RF *and* negative ACPA	0
Low-positive RF *or* low-positive ACPA	2
High-positive RF *or* high-positive ACPA	3
C. Acute-phase reactants (at least 1 test result is needed for classification)‡‡	
Normal CRP *and* normal ESR	0
Abnormal CRP *or* abnormal ESR	1
D. Duration of symptoms§§	
<6 weeks	0
≥6 weeks	1

Aletaha et al. A&R 2010

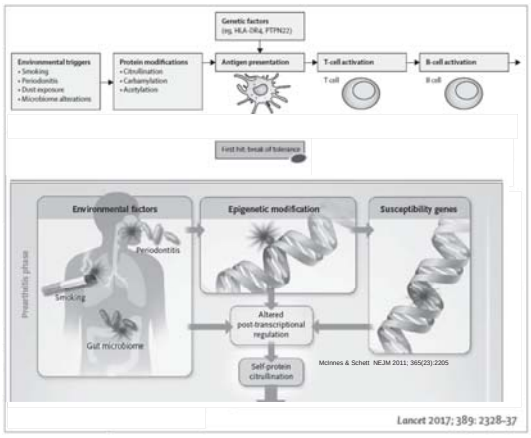
Epidemiology

- Worldwide, all ethnic groups
- Can occur at any age, but peaks between the 4th and 6th decades
- Prevalence in North America is 0.3-1.5%
- Prevalence is 2.5 times greater in women than in men
- Concordance rates among monozygotic twins 15-30%

Genes implicated in RA



- HLADRB1 (HLADR4)
  - Alleles that contain a common amino acid motif (QKRAA or shared epitope) in the antigen binding groove of the MHC molecule confer susceptibility to RA



Smoking, shared epitope, and risk of RA

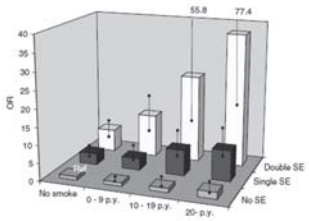
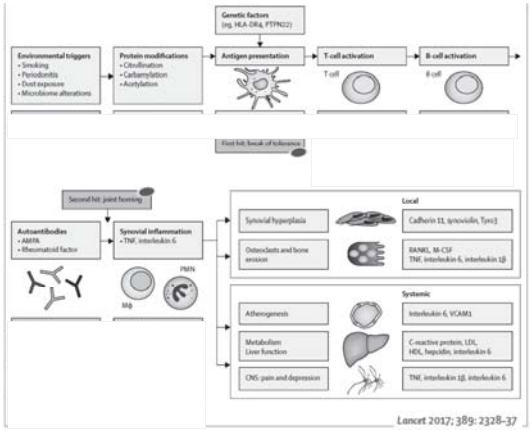


Figure 1 OR for different amounts of smoking (pack years (p.y.)) in combination with none (no shared epitope (SE)), one (single SE) or two (double SE) copies of SE alleles. The reference group was non-smokers without SE alleles.

Kallberg et al. Ann Rheum Dis 2010



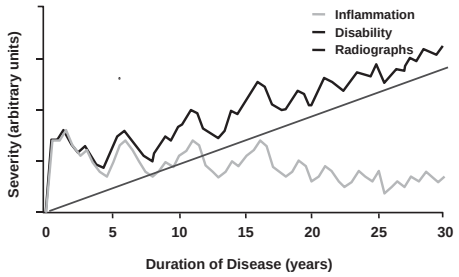
Goals in Treating RA

- Treat symptoms
  - Pain, stiffness, swelling, function
- Prevent long-term disability
  - Structural damage
- Prevent/Manage co-morbidities
  - Infections, cardiovascular risk, osteoporosis

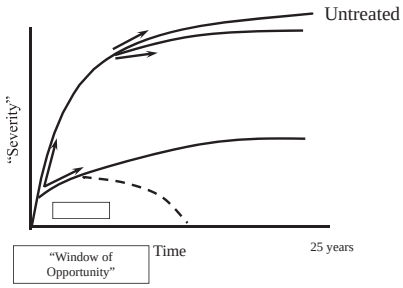
Principles to treatment of RA

- Diagnose early, refer early
- Initiate disease modifying anti-rheumatic drugs (DMARD) early
- Use combination therapy
- Treat-to-target (T2T)
- Aim for remission, or at least for low disease activity

Course of RA Progression

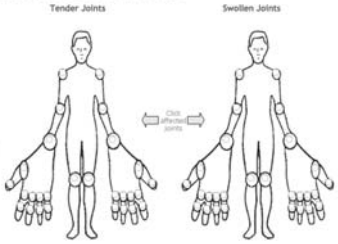


Preventing irreversible joint damage in RA: Window of Opportunity



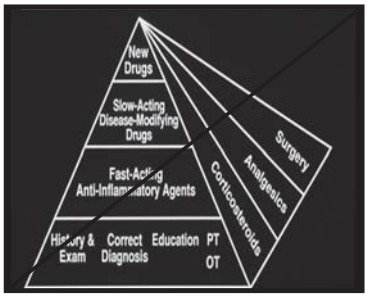
Evaluation of Response to Treatment in RA

- DAS 28
  - 28 joint count
    - # Swollen joints
    - # Tender joints
    - Global patient assessment (VAS 0-100 mm)
    - ESR



Remission	Low	Moderate	High
0	2.4	3.2	5.2

Turn the Old Treatment Pyramid (1960-90s) upside down



- Start with aspirin or NSAID
- Add a disease modifying anti-rheumatic drug (DMARD) only after damage is detected by X-rays
- Gradually escalate therapy

2012 RA Treatment Algorithm

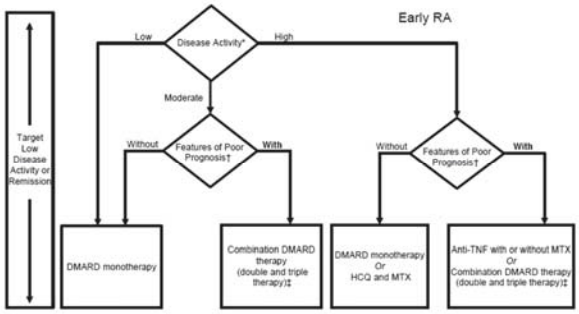


Figure 1. 2012 American College of Rheumatology recommendations update for the treatment of early rheumatoid arthritis (RA).

Singh et al. Arthritis Care & Res 2012; 64(5):625

RA Therapy: Traditional DMARDs

- Methotrexate (most commonly used, core RA drug)
- Hydroxychloroquine
- Sulfasalazine
- Triple therapy (MTX, HCQ, SZA combined)
- Leflunomide

RA Therapy- Biologic DMARDs

- Targeted inhibition of cytokines
  - TNF-alpha: Infliximab, etanercept, adalimumab, golimumab, certolizumab
  - IL-6: Tocilizumab, sarilumab
- Interfering with cytokine activation
  - JAK Kinase: Tofacitinib, Baricitinib
- Block T cell activation
  - CTLA-4Ig: Abatacept
- Deplete B cells
  - Rituximab

What to monitor?

- Lab monitoring
  - Cell counts, liver function, creatinine every 8-12 weeks
- Vigilant for infections
  - Common: Bronchitis, pneumonia, cellulitis
  - Viral: Zoster, influenza, hepatitis B reactivation
  - Atypical: Fungal, mycobacterial (esp. if travel)
- Vigilant for malignancy (skin)
- Vigilant for pulmonary toxicity (any), heart failure (anti-TNF), demyelination (anti-TNF), lupus like reaction (anti-TNF)
- Alcohol excess (methotrexate, leflunomide)
- Eye Exam (Hydroxychloroquine)

Vaccinations

- No live vaccines for patients on immunosuppressive treatment
- Influenza shot yearly
- Pneumonia vaccine (PCV13, PPV23)- best before starting methotrexate or rituximab
- Zoster vaccine- especially for patients on tofacitinib
  - Zostavax is a live vaccine, need to give before starting biologic drug, and wait 3-4 weeks after given to start biologic drug- ok to give while on methotrexate or low dose prednisone (refer to CDC page)
  - Shingrix is not a live vaccine and could be given while receiving biologic therapy in theory though not studied in those patients

Bone Health

- RA and prednisone use are additional risk factor in calculating FRAX score
- Advise on adequate calcium and vitamin D
- Advise on weight bearing or resistive exercise, and fall prevention
- Survey bone density regularly

Perioperative management

- Cervical spine
- Stop NSAIDs
- If on chronic prednisone, consider stress dose hydrocortisone
- Hold biologic medication, or time surgery for the end of the treatment interval; resume 2 weeks post op if no infections or wound complications
- Ok to continue oral DMARD for orthopedic surgeries

What about mortality in RA?

Table 3. Hazard ratios for total mortality for women with RA by serologic phenotype compared to women without RA in the Nurses' Health Study (1976–2012)*

	Deaths	Person-years	Mortality rate†	Age-adjusted HR (95% CI)	Multivariable HR (95% CI)‡
All RA					
No RA	28,501	3,678,801	775	1.00 (reference)	1.00 (reference)
RA	307	17,983	1,707	1.45 (1.30–1.63)	1.40 (1.25–1.57)
Seropositive RA					
No RA	28,492	3,425,924	832	1.00 (reference)	1.00 (reference)
RA	202	10,869	1,858	1.59 (1.38–1.82)	1.51 (1.31–1.74)
Seronegative RA					
No RA	28,501	3,666,676	777	1.00 (reference)	1.00 (reference)
RA	105	7,113	1,476	1.18 (0.97–1.43)	1.15 (0.95–1.39)

* RA = rheumatoid arthritis; HR = hazard ratio; 95% CI = 95% confidence interval.
† Per 100,000 person-years.
‡ Model adjusted for age, questionnaire cycle, census-tract family income (<$40,000 or ≥$40,000 per year), body mass index (<18.5, 18.5–24.9, 25–29.9, or ≥30 kg/m^2), cigarette smoking (never–10, 10.1–20, or >20 pack-years), postmenopausal hormone use (premenopausal, postmenopausal: never postmenopausal hormone use, postmenopausal: past postmenopausal hormone use, or postmenopausal: current postmenopausal hormone use), moderate to vigorous physical activity (0, 0.01–0.99, 1.00–3.49, 3.50–5.99, or ≥6 hours per week), cumulative average of Alternate Healthy Eating Index excluding alcohol component (quintiles), alcohol consumption (0, 0.1–4.9, 5.0–14.9, or ≥15.0 gm/day), cardiovascular disease (yes/no), and aspirin use (yes/no). Covariates were updated up to death, end of study period, censor, or loss to follow-up, whichever came first.

Sparks et al AC&R 2015

- Increased mortality associated with duration of rheumatoid arthritis
- Serologic status with greater risk of mortality in seropositive patients

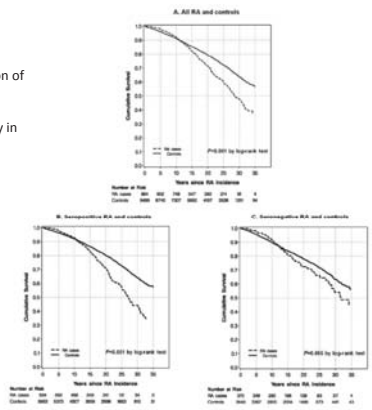


Figure 2. Kaplan-Meier curves for survival after incident rheumatoid arthritis (RA) diagnosis and age- and period-matched controls at index date of RA diagnosis for women in the Nurses' Health

Sparks et al AC&R 2015

Table 4. Hazard ratios for cause-specific mortality for women with incident RA by serologic phenotype compared to women without RA in the Nurses' Health Study (1976–2012)*

	Cancer mortality		Cardiovascular mortality		Respiratory mortality	
	Age-adjusted HR (95% CI)	Multivariable HR (95% CI)†	Age-adjusted HR (95% CI)	Multivariable HR (95% CI)†	Age-adjusted HR (95% CI)	Multivariable HR (95% CI)†
All RA						
No RA	1.00 (reference)	1.00 (reference)	1.00 (reference)	1.00 (reference)	1.00 (reference)	1.00 (reference)
RA	1.00 (0.80–1.25)	0.93 (0.74–1.15)	1.49 (1.18–1.89)	1.45 (1.14–1.83)	2.57 (1.91–3.48)	2.06 (1.51–2.80)
Seropositive RA						
No RA	1.00 (reference)	1.00 (reference)	1.00 (reference)	1.00 (reference)	1.00 (reference)	1.00 (reference)
RA	0.95 (0.71–1.26)	0.86 (0.65–1.15)	1.39 (1.02–1.90)	1.41 (1.03–1.93)	3.55 (2.55–4.95)	2.67 (1.89–3.77)
Seronegative RA						
No RA	1.00 (reference)	1.00 (reference)	1.00 (reference)	1.00 (reference)	1.00 (reference)	1.00 (reference)
RA	1.01 (0.72–1.42)	0.96 (0.68–1.35)	1.55 (1.08–2.21)	1.41 (0.98–2.02)	1.09 (0.54–2.19)	0.98 (0.49–1.99)

* RA = rheumatoid arthritis; HR = hazard ratio; 95% CI = 95% confidence interval.
† Model adjusted for age, questionnaire cycle, census-tract family income (<$40,000 or ≥$40,000 per year), body mass index (<18.5, 18.5–24.9, 25–29.9, or ≥30 kg/m^2), cigarette smoking (never–10, 10.1–20, or >20 pack-years), postmenopausal hormone use (premenopausal, postmenopausal: never postmenopausal hormone use, postmenopausal: past postmenopausal hormone use, or postmenopausal: current postmenopausal hormone use), moderate to vigorous physical activity (0, 0.01–0.99, 1.00–3.49, 3.50–5.99, or ≥6 hours per week), cumulative average of Alternate Healthy Eating Index excluding alcohol component (quintiles), alcohol consumption (0, 0.1–4.9, 5.0–14.9, or ≥15.0 gm/day), cardiovascular disease (yes/no), and aspirin use (yes/no). Covariates were updated up to death, end of study period, censor, or loss to follow-up, whichever came first.

Sparks et al AC&R 2015

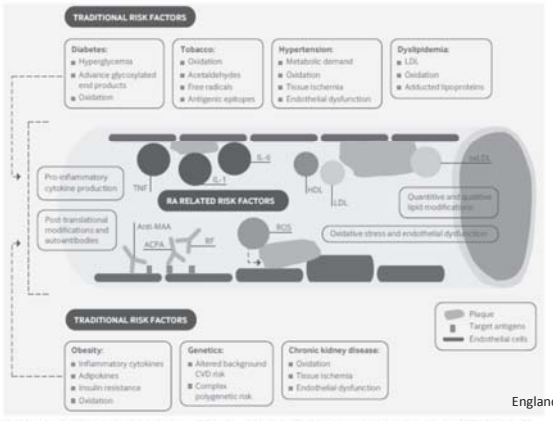


Fig 1 | Overview of mechanisms of cardiovascular disease (CVD) in rheumatoid arthritis (RA). Several mechanisms interact to amplify risk of CVD in RA. Higher RA

England et al BMJ 2018

Doing better…

Data from Olmsted County, MN

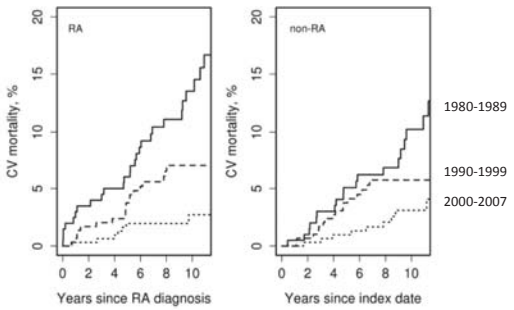
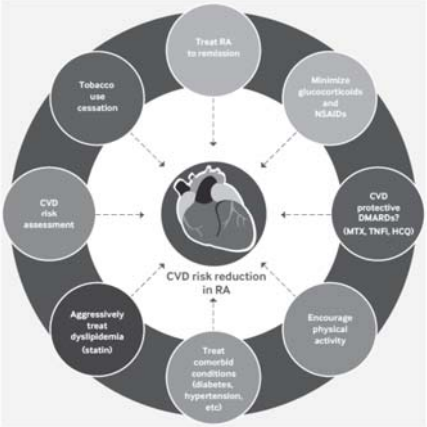


Figure 2. CV mortality in subjects with and without RA by decade of RA incidence/index: 1980–89 (solid line), 1990–99 (dashed line), and 2000–07 (dotted line). CV: cardiovascular; RA: rheumatoid arthritis.

Myasoedova et al. J Rheum 2017



England et al BMJ 2018

Take Home Messages

- Diagnose and refer RA early
- Think of atypical presentations
- Labs and x-rays not always supportive, so rely on history and exam
- Key to treating RA is early initiation of disease modifying drugs
- Treating with prednisone and NSAIDs only is not acceptable
- DMARDs prevent joint damage, extra-articular complications, and cardiovascular disease and likely improve mortality
- Have a low threshold to evaluate for infection
- Assess safety labs, bone health, vaccinations

Questions?

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Gout Management: Treat to Target!

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09/05/2019

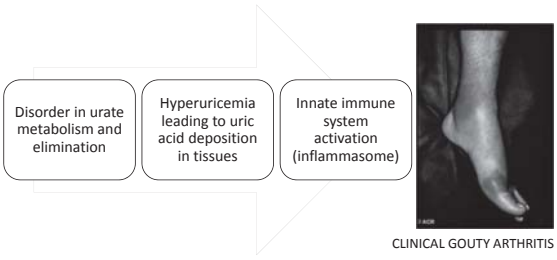
Disclosures

- None

By the end of this talk, you should be able to:

- Describe mechanisms of hyperuricemia and gouty arthritis
- Recognize the risk factors and epidemiology of gout
- Compare and contrast clinical features of gout with other arthropathies
- Master treatment strategies for acute and chronic gout management

Gout is the end-result of hyperuricemia.



CLINICAL GOUTY ARTHRITIS

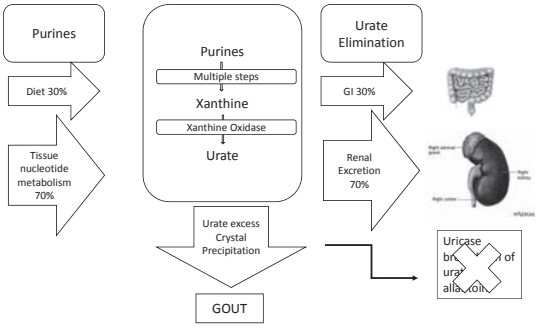
Hyperuricemia ≠ Gout

Prolonged hyperuricemia predisposes to gout

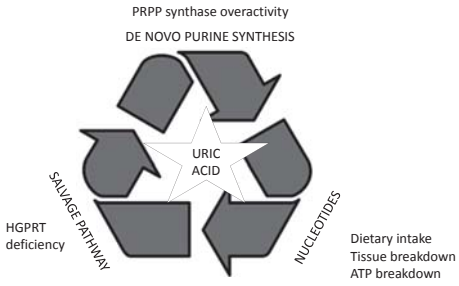
Uric acid level	Cumulative Incidence at 5 yrs
<6	0.5%
6-6.9	0.6%
7-7.9	2%
8-8.9	4.1%
9-9.9	19.8%
>10	30.5%

Campion et al, Normative Aging Study, *Am J Med*, 1987

Urate level depends on purine intake, metabolism, and elimination.



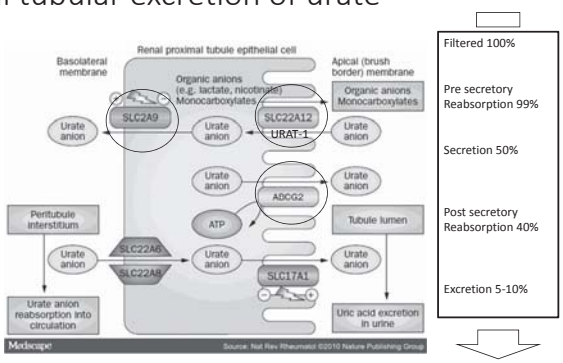
Purine source



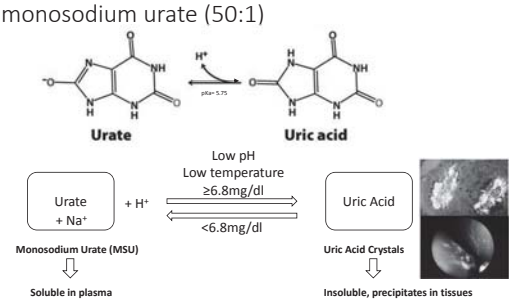
Why do some people have hyperuricemia?

- The minority (10%) are overproducers (inherited disorders of purine metabolism or high cell turnover state)
- The vast majority (90%) of patients with hyperuricemia are underexcretors

Renal tubular excretion of urate



Uric acid is a weak organic acid which in body fluid exists mostly as monosodium urate (50:1)

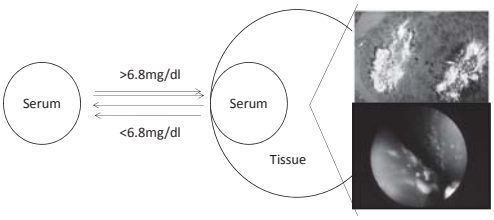


At the pH 7.4 of most body fluids, urate ion levels exceed those of un-ionized uric acid in a ratio of approximately 50 : 1. Because of the high concentration of sodium in extracellular fluid, urate functions as monosodium urate (MSU). Urate sodium solubility is quite low (~6.8 mg/dL or 400 µM). At a concentration above 6.8mg/dl, monosodium urate becomes uric acid which is insoluble and precipitates in various tissues especially joints and periarticular tissues.

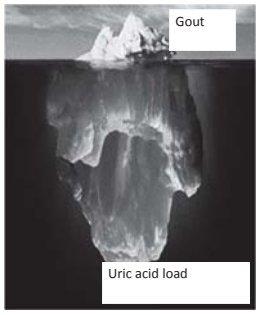
Pearl

- Uric acid tends to precipitate in the lower extremity joints, possibly because of repeated stress to the great toe/foot, reduced pH and cooler temperatures peripherally.

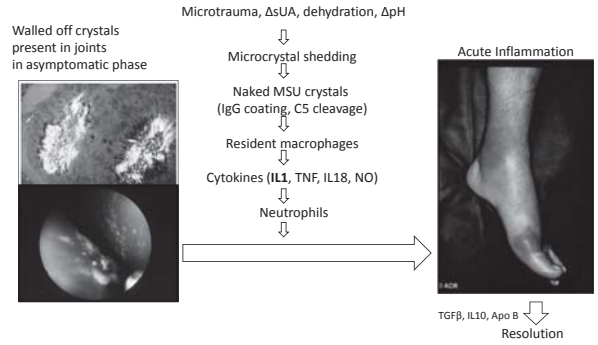
Gout is not a serum urate level disease but a urate deposition disease.



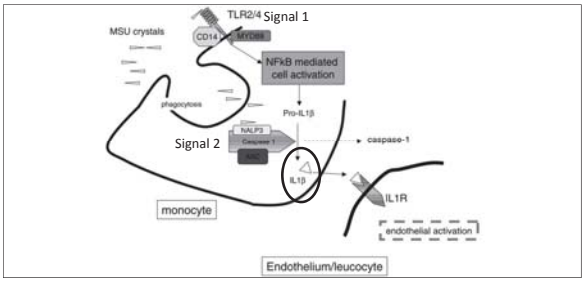
Gout is the tip of the hyperuricemia iceberg



Acute Gout and the Inflammasome



Uric acid crystals are sensed as danger signals by the innate immune system, resulting in IL-1β release.

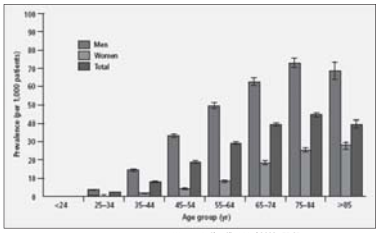


Pearl

- Interleukin-1 is a key cytokine behind crystal induced inflammation

Gout prevalence is rising.

- Gout affects 4% of the US population (8.3 million Americans).
- Most common cause of inflammatory arthritis in men over age 40
- Male gender and age are greatest risk factors.



Weaver A. Clev Clin J Med 2008 ; 75:S5

Pearl

- Premenopausal women should not get gout

Risk factors for hyperuricemia and gout

Non-modifiable
- Male gender
- Age
- Heredity (urate tubular transport)
- High blood pressure
- Chronic kidney disease

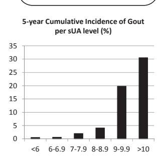
Risk factors for hyperuricemia and gout

Non-modifiable
- Male gender
- Age
- Heredity (urate tubular transporters)
- High blood pressure
- Chronic kidney disease

Modifiable
- Obesity, insulin resistance
- Medications (low dose aspirin, diuretics, cyclosporine)
- Alcohol Consumption (beer > liquor > wine)
- Diet high in purines (red meat, shellfish)

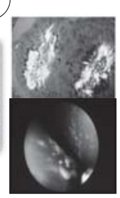
Gout: Clinical Stages

Asymptomatic hyperuricemia: Not all patients with hyperuricemia get gout



Acute gout:
Age at onset 30-40
Acute Monoarthritis
Peaks at 4-12 hours
Self-limited or easily treated
1MTP, foot, ankle, knee

Intercritical: Asymptomatic

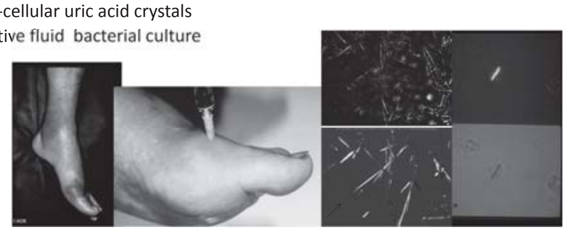


Advanced gout:
Multiple joints
Wrist, elbows, fingers
Prolonged episodes
Harder to treat
Tophi
Erosions

Gout Features	ACUTE	INTERCITICAL PHASE
History	Acute onset (<24 hours to peak) No trauma or minor Lower extremity Risk factors for gout	Episodic acute arthritis, resolves within 14 days, asymptomatic between episodes 90% of the time, MTP affected at some point along the way
Exam	Red, exquisitely tender, swollen joint	Normal exam Possible tophi or joint deformities if long standing, chronic, untreated
Labs	Uric acid may be normal in 30% in the acute phase Synovial fluid aspiration	Uric acid is > 6.8 mg/dl
X-rays	Soft tissue swelling	Normal, or abarticular erosions with overhanging edge

Gout Diagnosis: Joint aspiration is the gold standard for a definitive diagnosis of gout.

- Inflammatory fluid
  - Cell count: WBC >2000, neutrophil predominance
  - Polarized microscopy: Needle shaped, negatively birefringent, intra- and extra-cellular uric acid crystals
  - Negative fluid bacterial culture



Pearl

- One cannot differentiate gout from septic arthritis
- Aspiration is recommended to establish a diagnosis, especially if first presentation, or as guided by risk factors
- Gout and septic arthritis can co-exist

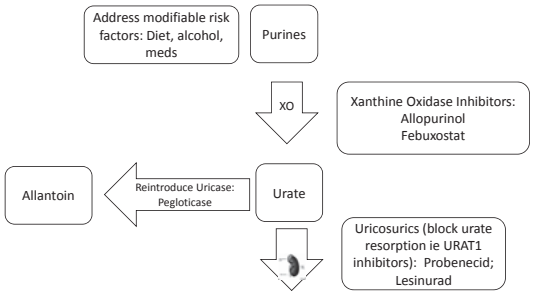
Goals of Gout Treatment

- Treat acute arthritis to relieve pain and restore function
- Prevent future attacks
- Prevent development of chronic advanced gout which can lead to permanent joint damage

Acute Gout Treatment: Know the co-morbidities!

- NSAIDs (full dose, continue for a few days after resolution of flare usually about 5-7 days)
- Corticosteroids (systemic or intra-articular)
- Colchicine (to be given within 12 hours of onset of the flare to be effective; inhibits microtubules and the inflammasome)
- IL1 blocking drug (anakinra, non-FDA approved)
- ACTH (rarely used, expensive; increases endogenous steroid production, direct anti-inflammatory effects)

The key to preventing future gout flares is to treat the hyperuricemia!



Diet and gout

- Reduce red meat (including organ meat)
- Reduce shellfish
- Reduce alcohol
- Reduce high fructose drinks, and fruit juices
- Increase low fat milk and vegerables

Choi et al. Health Professionals Follow-Up Study *NEJM* 2004; 350:1093

When to initiate Urate Lowering Therapy?

- Patient with established diagnosis of gout AND
  - Tophus
  - CKD stage 2 or greater
  - Nephrolithiasis
  - 2 or more attacks per year

Khanna et al. ACR Gout Guidelines 2012 AC&R

Arthritis Care & Research
Vol. 64, No. 10, October 2012, pp 1431–1446
DOI 10.1002/acr.21772

SPECIAL ARTICLE

2012 American College of Rheumatology Guidelines for Management of Gout. Part 1: Systematic Nonpharmacologic and Pharmacologic Therapeutic Approaches to Hyperuricemia

DINESH KHANNA,[1] JOHN D. FITZGERALD,[2] PUJA P. KHANNA,[1] SANGMEE BAE,[2] MANJIT K. SINGH,[3] TUHINA NEOGI,[4] MICHAEL H. PILLINGER,[5] JOAN MERILL,[6] SUSAN LEE,[7] SHRADDHA PRAKASH,[2] MARIAN KALDAS,[2] MANEESH GOGIA,[2] FERNANDO PEREZ-RUIZ,[8] WILL TAYLOR,[9] FRÉDÉRIC LIOTÉ,[10] HYON CHOI,[4] JASVINDER A. SINGH,[11] NICOLA DALBETH,[12] SANFORD KAPLAN,[13] VANDANA NIYYAR,[14] DANIELLE JONES,[14] STEVEN A. YAROWS,[15] BLAKE ROESSLER,[1] GAIL KERR,[16] CHARLES KING,[17] GERALD LEVY,[18] DANIEL E. FURST,[2] N. LAWRENCE EDWARDS,[19] BRIAN MANDELL,[20] H. RALPH SCHUMACHER,[21] MARK ROBBINS,[22] NEIL WENGER,[2] AND ROBERT TERKELTAUB[7]

Pearls in initiating urate lowering therapy

- Pick a urate lowering drug: start low, go slow
- Do not forget to concomitantly start an anti-inflammatory drug ("prophylactic therapy") to prevent gout flares as uric acid levels begin to drop: colchicine 0.6mg daily or qod; or low dose NDAID eg naproxen 500 mg daily; or low dose prednisone 5 mg/day.

TABLE 3
Some important drug interactions with colchicine*

Drug	Likely interaction mechanism†	Comments‡
Cyclosporine	P-glycoprotein	Shown in PK studies; clinically important interactions reported[18]
Clarithromycin	CYP 3A4	Shown in PK studies; fatal interactions reported[19,20]
Azithromycin	P-glycoprotein	Shown in PK studies
HMG-CoA reductase inhibitors (statins)	Unknown	Myotoxicity/ rhabdomyolysis[21]
Fibric acid derivatives	Unknown	Myotoxicity/ rhabdomyolysis[22]
Verapamil	CYP 3A4, P-glycoprotein	Shown in PK studies; clinically important interactions reported[23]
Diltiazem	CYP 3A4, P-glycoprotein	Shown in PK studies; clinically important interactions reported[17]
Ketoconazole	CYP 3A4, P-glycoprotein	Shown in PK studies
Carvedilol	CYP 3A4	

* In addition to the medications listed here, other drugs are anticipated to interact with colchicine, in the absence of pharmacokinetic studies or clinical reports, based on strong CYP 3A4 or P-glycoprotein inhibition; these drugs include itraconazole, nefazodone, telithromycin, ranolazine, and several antiretroviral drugs used to treat HIV.

Mandell et al. CCJM 2010

Pearls in initiating urate lowering therapy

- Pick a urate lowering drug: start low, go slow
- Do not forget to concomitantly start an anti-inflammatory drug ("prophylactic therapy") to prevent gout flares as uric acid levels begin to drop: colchicine 0.6mg daily or qod; or low dose NDAID eg naproxen 500 mg daily; or low dose prednisone 5 mg/day.
- Continue prophylactic therapy for 6 months after initiating urate lowering therapy, and for at least 12 weeks post achieving uric acid goal
- Check uric acid level 2-4 weeks after each dose adjustment
- Target a uric acid level of < 6 mg/dl (if tophi, < 5mg/dl)

Pearl

- The most common mistake in gout treatment is the failure to start prophylactic anti-inflammatory treatment together with ULT

Pearl

- The second common mistake in gout treatment is the failure to start treat to target and aim to reduce sUA < 6 mg/dl

Allopurinol: First line Urate lowering Therapy

- Xanthine Oxidase Inhibitor
- Cheap
- Once a day
- Contraindicated with azathioprine and 6-MP (relative)
- Start with 100 mg daily (50 mg daily if GFR <30)
  - Increase by 100 mg (50 mg if GFR <30) every 4 weeks to reach target sUA
  - Monitor cbc with diff, creatinine, LFTs
  - Warn about hypersensitivity reaction (rash, fever, abnormal labs, first 6 mos)
    - At risk: HLAB5801 carriers (test if Chinese, Thai, South Korean), CKD, high starting dose
  - FDA max dose 800 mg/day

Febuxostat- Alternative ULT

- Xanthine Oxidase Inhibitor
- Once a day
- Expensive
- Contraindicated with azathioprine and 6-MP (absolute)
- Start 40 mg/day, increase to 80 mg/day if sUA > 6 mg/dl after 2 weeks
- May cause GI side-effects
- Monitor LFTs
- Black box warning re: increased risk for CV mortality compared with allopurinol

Probenecid- Second line ULT

- URAT-1 inhibitor, so increases urinary excretion of uric acid
- Seems like we should use it in everyone then
- But… ineffective GFR <60 and…
- BID dosing
- Drug-drug interactions
- Risk of nephrolithiasis (need to check baseline 24 hour urine uric acid excretion; not to be used if > 800 mg per 24 hours)
- Expensive
- Can be combined with allopurinol or febuxostat

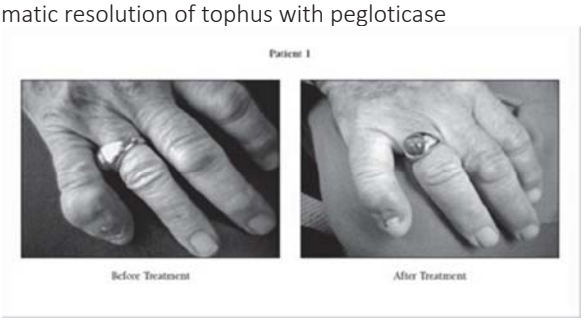
Lesinurad (FDA approved alternate uricosuric)

- URAT-1 inhibitor, so increases urinary excretion of uric acid
- Expensive
- Has to be used WITH a xanthine oxidase inhibitor!
- Risk of AKI

Pegloticase

- Recombinant uricase
- Infusion, 8 mg every 2 weeks
- High risk of allergic and anaphylactic reaction
- Should not be used to XOI or uricosuric
- Uric acid drop dramatically to 0.5-1mg/dl and tophi will shrink
- May be used for 6-12 months then transition back to oral agent
- Reserved for severe refractory tophaceous gout
- Expensive

Dramatic resolution of tophus with pegloticase



70 yo man with 25 yr h/o gout and nephrolithiasis, allergic to allopurinol received 8mg IV q 2 weeks for 12 wks. Uric acid level fell from 9.3 to <0.1 and remain that low even 2 weeks post last infusion. Baraf et al *Arth Rheum* 2008; 11:3632.

When to refer?

- Unclear etiology of hyperuricemia
- Refractory gout
- Inability to reach target sUA despite titration of ULT
- CKD
- Adverse effects of treatments

Take Home

- Treat the acute flare first
- When indicated, start urate lowering therapy: start low, titrate slow
- Treat to target sUA < 6mg/dl
- Do not forget prophylactic therapy for 6 months after ULT started
- Remember to think of comorbidities and drug-drug interactions

Questions?

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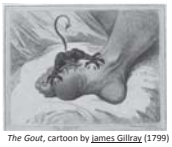
Origin of the gout, cartoon by Henry Bunbury (1786)



The Gout, cartoon by James Gillray (1799)

Making Sense of Hypermobility Syndromes:

MARCIA FRIEDMAN, MD

ANNUAL MSK UPDATE FOR PRIMARY CARE

SEPTEMBER 5, 2019

Disclosures

I have no relevant disclosures

Objectives

1. Diagnosing hypermobility: the Beighton score
2. Joint hypermobility vs joint hypermobility *syndrome* (JHS)
3. JHS vs Ehlers Danlos-hypermobility type (EDS-HT)
4. DDx of hypermobility
5. Management: MSK and associated features
6. When and where to refer?

Case:

- 25 year old man with years of generalized pain.
- Reports dislocating his shoulder twice, has never been the same.
- Feet, knees, back hurt, can't do a lot of exercise without being in pain.
- ROS: often feels lightheaded, constipation alternating with diarrhea, generally fatigued

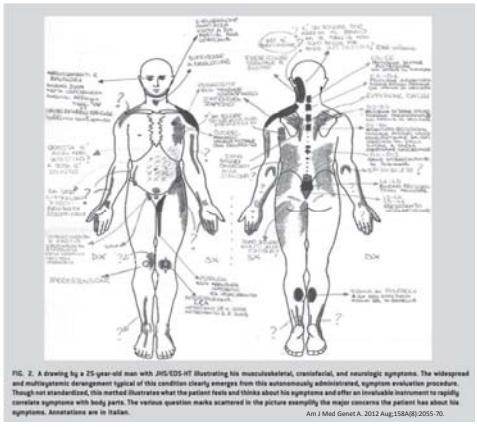


FIG. 2. A drawing by a 25-year-old man with JHS/EDS-HT illustrating his musculoskeletal, craniofacial, and neurologic symptoms. The widespread and multisystemic derangement typical of this condition clearly emerges from this autonomously administrated, symptom evaluation procedure. Though not standardized, this method illustrates what the patient feels and thinks about his symptoms and offer an invaluable instrument to rapidly correlate symptoms with body parts. The various question marks scattered in the picture exemplify the major concerns the patient has about his symptoms. Annotations are in Italian.

Am J Med Genet A. 2012 Aug;158A(8):2055-70.

Case con't

- On exam:
  - Thin, tired, but otherwise healthy-appearing young man
  - Tender over the left knee, left shoulder, and low spine/paraspinal muscles.
  - No joint effusions, no synovitis, has flexible joints.
  - Few bruises, otherwise normal appearing skin
  - Remainder of his exam is normal
- Is he hypermobile? Can we be any more specific?
- Does he have an underlying heritable connective tissue disorder?
- Are his complaints connected? How can we help him?

Joint hypermobility = Beighton score ≥ 4/9

https://www.ehlers-danlos.org/about-eds/getting-a-diagnosis/beighton-score/

Hypermobility vs. Joint Hypermobility *Syndrome* (JHS)

- Hypermobility is relatively common, especially in young people, women, and Asian/African ancestry.
- Most hypermobile people are asymptomatic and do not have an underlying connective tissue disorder.
- Joint hypermobility syndrome: heritable connective tissue disorder with pain, dislocations, bursitis, tendonitis, fatigue, dysautonomia, and multi-system involvement.

Am J Med Genet C Semin Med Genet. 2015 Mar;169C(1):6-22.
Am J Med. 2017 Jun;130(6):640-647.

Joint hypermobility syndrome (JHS): Brighton criteria

(...not to be confused with Beighton score)

Brighton Criteria

- **Major Criteria**
  - Beighton score of >4 *Figure 4*)
  - Arthralgia for longer than 3 months in 4 or more joints
- **Minor Criteria**
  - Beighton score of 1, 2, or 3 (*Figure*\t>4)
  - Arthralgia (>3-month duration) in one to three joints or back pain (>3-month duration)or spondylosis, spondylolysis/spondylolisthesis
  - Dislocation or subluxation in more than one joint, or in one joint on more than one occasion
  - Three or more soft tissue lesions (eg, epicondylitis, tenosynovitis, bursitis)
  - Marfanoid habitus (tall, slim, span greater than height (>1.03 ratio), upper segment less than lower segment (<0.89 ratio), arachnodactyly)
  - Skin striae, hyperextensibility, thin skin, or abnormal scarring
  - Ocular signs: drooping eyelids, myopia, antimongoloid slant
  - Varicose veins, hernia, or uterine or rectal prolapse
  - Mitral valve prolapse
- **Requirement for Diagnosis**
  - Any one of the following:
    - two major criteria
    - one major plus two minor criteria
    - four minor criteria
    - two minor criteria and unequivocally affected first-degree relative in family history

J Am Osteopath Assoc. 2006 Sep;106(9):531-6.
J Rheumatol. 2000 Jul;27(7):1777-9.

Now that you've diagnosed JHS...

"I looked this up, and I'm pretty sure I have EDS!"

Does this patient have EDS-hypermobility type (EDS-HT)..?

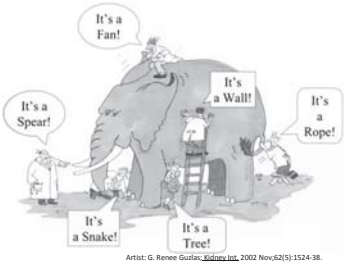
Must meet 1, 2, and 3:

1. ☆ **Beighton score ≥ 4/9**
2. **Two or more of the following:**
   - A. ? **Systemic manifestation of a connective tissue disorder** with ≥ 5 of the following: unusually soft/velvety skin, mild skin hyper-extensibility, unexplained striae, bilateral piezogenic papules of the heel, atrophic scars, recurrent or multiple abdominal hernias, pelvic floor/rectal/uterine prolapse without clear cause, dental crowding or high/narrow palate, arachnodactyly, arm span to height ≥ 1.5, MVP, or aortic root dilatation
   - B. ? **Positive family history** with 1 or more 1º relatives affected
   - C. ☆ **MSK complications** ≥1 of the following: MSK pain in two or more limbs for at least 3 months, chronic widespread pain for ≥ 3 months, recurrent joint dislocations in the absence of trauma
3. **None of the following :**
   - A. ☆ **Unusual skin fragility** (consider other types of EDS)
   - B. ? **Other heritable or acquired connective tissue disorder** (if you do, must have both 2A and 2B)
   - C. ? **Alternative diagnosis associated with hypermobility**

Am J Med Genet C Semin Med Genet. 2017 Mar;175(1):8-26.

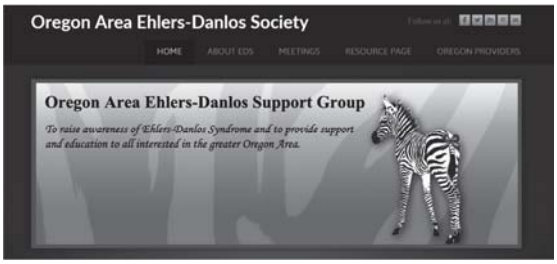
Are JHS and EDS-HT the same condition?

- Clinically indistinguishable
- Neither has an objective diagnostic test, molecular cause, or known gene mutation
- JHS: Brighton criteria (from rheum literature)
- EDS-HT: Villefranche criteria (from peds/genetics literature)
- Is JHS a mild variant of EDS-HT? Are they the same condition?
- **Increasingly these are thought of as interchangeable or at least part of the same spectrum.**



Artist: G. Renee Guzlas; Kidney Int. 2002 Nov;62(5):1524-38.

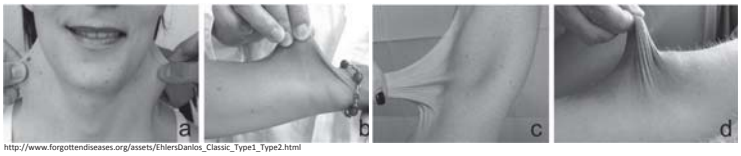
EDS Support Systems



What else could this be? Ddx..

1. Other forms of EDS:
   - Many other forms, but most are extremely rare and have severe phenotypes from a young age.
   - Classic EDS: **skin** is a significant finding
   - Vascular EDS: **arterial ruptures/dissections**
2. Marfan Syndrome:
   - **Tall stature**, family history of Marfan or **aortic dissection**, pectus deformities, arachnodactyly, myopia, **lens dislocations**
3. Loey's Dietz:
   - Widely spaced eyes, cleft palate/bifid uvula, **severe arterial tortuosity**. Arterial aneurysms/dissections
4. Osteogenesis Imperfect:
   - **Fragile bones, frequent fractures**, blue sclera

Skin Hyper-extensibility: Classic EDS



http://www.forgottendiseases.org/assets/EhlersDanlos_Classic_Type1_Type2.html

Test at a neutral site, such as volar surface of the forearm or neck.

Pull skin up until resistance is felt.

Ability to stretch the skin 4cm or more

Difficult in children due to subcutaneous fat

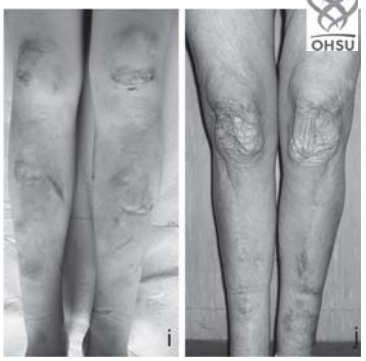
Am J Med Genet. 1998 Apr 28;77(1):31-7.

Tissue Fragility: Classic EDS

Presence of easy bruising and dystrophic scars

Scars are mostly on pressure points: knee, elbow, forehead, chin and have a thin papyraceous appearance.

Scars are frequently wide and discolored, wound healing is impaired

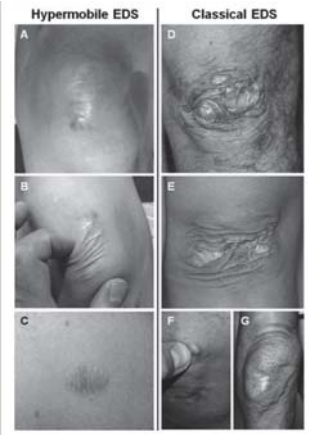


http://www.forgottendiseases.org/assets/EhlersDanlos_Classic_Type1_Type2.html

A. Post-traumatic, atrophic and widened scar

B. Skin stretching between examiner's fingers shows mild atrophy of the underlying dermis

C. Atrophic and widened scar due to wound healing delay after nevus excision



D. Papyraceous and hemosideric scar after repetitive wound opening with molluscoid psuedotumor

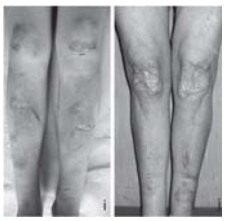
E. Papyraceous scar and cutis laxa

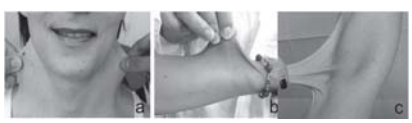
F. Subcutaneous spheroid

G. Huge molluscoid psuedotumor

Am J Med Genet C Semin Med Genet. 2017 Mar;175(1):8-26.

Classic EDS





Genetics

COL5A1/COL5A2
1/20,000
Autosomal dominant

Classification criteria

Major:
1. **Skin hyper-extensibility and wide atrophic scars**
2. Joint hypermobility

Minor:
1. Easy bruising
2. Soft, doughy skin
3. Skin fragility (traumatic tearing)
4. Molluscoid psuedotumors
5. Subcutaneous spheroids
6. Hernias
7. Epicanthal folds
8. Complications of joint hypermobility

Positive family hx

Criteria to prompt confirmatory testing:
- Both major criteria OR
- Skin hyper-extensibility and atrophic scars + ≥ 3 minor

Genetic testing: COL5A1/5A2

Am J Med Genet C Semin Med Genet. 2017 Mar;175(1):8-26.

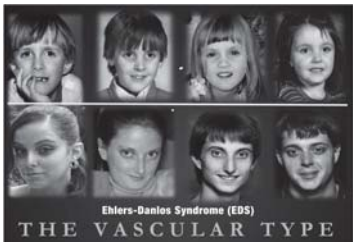
http://www.forgottendiseases.org/assets/EhlersDanlos_Classic_Type1_Type2.html

Vascular EDS


Acrogeria

Genetics

COL3A1
1/100,000
Autosomal dominant



Classification criteria

Major criteria:
1. Family history of vascular EDS
2. **Arterial rupture** at a young age
3. Spontaneous **sigmoid colon perforation**
4. **Uterine rupture** during 3rd trimester
5. **Carotid-cavernous sinus fistula**

Minor criteria:
1. Excessive bruising without trauma
2. **Thin, translucent skin with venous visibility**
3. Characteristic facial features
4. Spontaneous pneumothorax
5. **Acrogeria**
6. Hypermobility of small joints
7. Club foot
8. Congenital hip dislocation
9. Small joint hypermobility
10. Tendon/muscle rupture
11. Keratoconus
12. Gingival recession
13. Early onset varicose veins (<age 30)

Diagnostic testing: COL3A1 gene

http://www.forgottendiseases.org/assets/EhlersDanlos_Vascular_Type4.html

Am J Med Genet C Semin Med Genet. 2017 Mar;175(1):8-26.

DDx of hypermobility

1. **Other forms of EDS:**
   - Classic EDS: skin is a significant finding
   - Vascular EDS: arterial ruptures/dissections
   - Many other forms, but most are extremely rare and have severe phenotypes from a young age.
2. Marfan Syndrome:
   - **Tall stature**, family history of Marfan or **aortic dissection**, pectus deformities, arachnodactyly, myopia, **lens dislocations**
3. Loey's Dietz:
   - Widely spaced eyes, cleft palate/bifid uvula, **severe arterial tortuosity**. Arterial aneurysms/dissections
4. Osteogenesis Imperfecta:
   - **Fragile bones, frequent fractures**, blue sclera

Back to EDS-HT / JHS:

- Prevalence:
  - EDS literature estimates 1:5,000
  - JHS estimates up to 3% of the population (self reported questionnaire)
- Patients with self-reported hypermobility are 40% more likely to have chronic widespread pain.
- Multi-system involvement

Arthritis Care Res (Hoboken). 2013 Aug;65(8):1325-33.

Systemic manifestations of EDS-HT/JHS

- Fatigue: >80%
- Dysautonomia: 75%
  - Orthostatic intolerance, PoTS, pre-syncope/syncope, dizziness, palpitations
- Headaches: 75% of female patients suffer from migraines
- Abdominal pain: >80%
  - Partially related to dysautonomia
  - Constipation, diarrhea, hernias, pelvic prolapse, nausea/vomiting, bloating
- Structural cardiac disease: Mitral valve prolapse in 6%
- Psychiatric: increased anxiety, depression, kinesiophobia

Rheum Dis Clin North Am. 2013 May;39(2):419-30.
Clin Rheumatol. 2014 Jul;33(7):981-7.
Arthritis Care Res (Hoboken). 2013 Aug;65(8):1325-33.

Management: general principles

- MSK symptoms: search for underlying cause
- Musculoskeletal pain management centers on physical therapy and lifestyle modification: strengthening, proprioception, limited stretching
- Recognize signs of autonomic dysfunction (PoTS) and functional GI disorders
- Cognitive behavior therapy to help learn to cope and live with chronic condition

MSK complications

- MSK symptoms—search for an underlying cause:
  - Recurrent and chronic joint injuries, dislocations, soft tissue injuries
  - Neuropathic pain: compressive neuropathies, axonal neuropathies (spine hypermobility, disk herniation)
- Headaches: c-spine hypermobility, migraine headaches, occipito-atlantoaxial instability, Chiari 1 malformations
- Avoid opiate pain medications—often ineffective and may worsen GI complications
- Chiropractic manipulation: general avoided due to intrinsic connective tissue laxity

https://balanceexerciseskomehitsu.blogspot.com/2017/09/balance-exercises-using-wobble-board.html

Management: MSK

- **Physical Therapy**:
  - Exercise: specific, isolated, low-level, stabilization training.
  - Building deconditioned muscles, focus on **postural muscles**
  - **Proprioception exercises** improve joint stability—weight bearing, closed kinetic chain exercises:
    - Standing on one leg, mini squats, single knee bend, heel walking, walking backward, walking with eyes closed, slowly sitting/standing
    - Rocker-bottom wood, board-balance wood, foam rollers, etc
- Joint protection/avoiding injury:
  - Avoid excessive splinting/bracing– prefer muscle strengthening, however splints/braces can be used for stabilization or acute injury
- Education: avoid harm, don't overstretch

Curr Opin Rheumatol. 2011 Mar;23(2):131-6.
Rheumatol Int. 2008 Aug;28(10):995-1000.

Taping: can help with pain, mechanical stability, joint laxity, and proprioception

Ther Adv Musculoskelet Dis. 2015 Feb; 7(1): 3–10.

Sports: Aquatic exercises, T'ai Chi, Pilates/yoga/dance (but avoid excessive stretching).

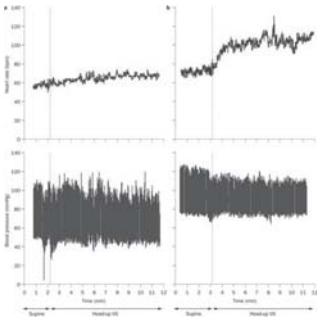
GI/GU complications

- Anatomic complications:
  - Higher rates of abdominal hernias
  - Rectal and uterine prolapse/pelvic floor dysfunction are more common
- Functional complications:
  - Pathogenesis is poorly understood
  - Nausea, vomiting, bloating, constipation, IBS-symptoms and diarrhea are common
- Treat functional complications symptomatically
- Dysautonomia may contribute to GI complications?

Neurogastroenterol Motil. 2015 Nov;27(11):1657-66

PoTS: symptoms and diagnosis

- Symptoms: palpitations, dizziness, pre-syncope/syncope, fatigue, headaches
- Worsened by: heat, eating, exertion
- Diagnosis:
  - Head-up tilt-table test:
  - Tilt to 60^0 for 10 minutes → sustained 30bpm rise in BP, or max HR of ≥120bpm during the first ten minutes.
  - There is usually NO orthostatic hypotension



Nat Rev Neurol. 2011 Dec 6;8(1):22-34.

PoTS: non-pharmacologic management

Avoid	Encourage
Sudden head-up postural changes	Head-up tilt at night
Excessive exertion	Regular gradual exercise (swimming)
Large meals (especially simple carbs)	Small, infrequent meals
EtOH	High salt intake (if no HTN)
Hot temperatures	Water repletion
Vasodilator or vasodepressors (nitrates, Ca channel blockers, diuretics)	Elastic stockings/compression

Nat Rev Neurol. 2011 Dec 6;8(1):22-34.

PoTS: Pharmacological Management		
First line:	**Fludricortisone:** 0.05-0.2mg/day	Avoid in HTN, may cause fluid retention
Second line:	**Midodrine:** vasoconstriction, prevents pooling, raises BP 2.5-10mg 3x/day	Avoid in HTN May cause urinary retention in older men
Third line: Low BP	**Cardioselective ß1-blockers**: atenolol, metoprolol Start low, gradually increase	Avoid in asthma
Third line: High/ normal BP:	**Non-selective ß-blockers**: propranolol	Avoid in asthma
	Clonidine: central sympatholytic	
	Ivabradine: selective sinus node inhibitor, for substantial tachycardia	May cause symptomatic bradycardia
Post-prandial tachycardia	Octreotide: somatostatin analog	
Other	Pyridostigmine: 30mg/day	Common GI side effects

Nat Rev Neurol. 2011 Dec 6;8(1):22-34.

Management: structural heart disease

Mitral valve prolapse: 6.4% of EDS-HT; 4.7% of JHS
- Generally mild

Aortic root dilatation: 1.6% of EDS-HT/JHS patients
- Rarely requires surgical repair.

Routine screening echo?
- Used to be routinely recommended but increasingly echo is only advised if symptoms warrant.

Am J Med Genet A. 2018 Jul 31. doi: 10.1002/ajmg.a.40364.

When/where to refer:

- Medical genetics:
  - There is no genetic testing for hypermobility EDS—these patients do not need to be referred to medical genetics
  - If you suspect other forms of EDS, Marfan syndrome, etc. → refer to medical genetics for detailed assessment, genetic testing, & family planning advice
- Cardiology:
  - Marfan's syndrome, vascular EDS, Loey's Dietz
- Rheumatology:
  - If an inflammatory connective tissue disease is suspected → refer to rheumatology to evaluate.

Take home points

- Beighton score to dx hypermobility: 60 second exam!
- JHS and EDS-HT are probably interchangeable.
- DDx: examine skin, stature, ask about vascular disease & family history
  - Other types of EDS, Marfan, Loey's Dietz
- Management—recognizing that this is a multi-organ system disease:
  - Consider dysautonomia & PoTS—can make a big difference in symptoms
  - MSK: Think about the cause: entrapment neuropathies, spinal disease, dislocations/enthesitis/bursitis/tendonitis
  - **Physical therapy & lifestyle modifications are the mainstay of treatment**
- Referral to genetics is not necessary for EDS-HT, but can be useful for other forms of EDS, or other heritable connective tissue disorders.

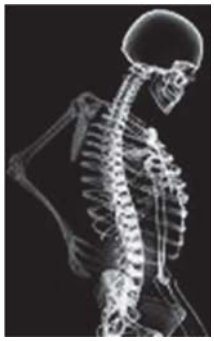
MUSCULOSKELETAL UPDATE FOR PRIMARY CARE

Hans Carlson, MD
Associate Professor
Physical Medicine & Rehabilitation
OHSU

INTERESTING CASES / PANEL

Making the diagnosis with musculoskeletal pain

AN OVERVIEW OF THE MANAGEMENT OF PERSISTENT MUSCULOSKELETAL PAIN



Objectives

- Understand the importance of differentiating accurate vs. specific diagnoses of musculoskeletal symptoms.
- Discuss the progression of diagnostic tools available when presented with musculoskeletal pain.
- Describe the general treatment options for musculoskeletal pain and the role of the patient and clinician in pursuing treatment.
- Effectively educate the patient regarding their diagnosis, imaging and management.

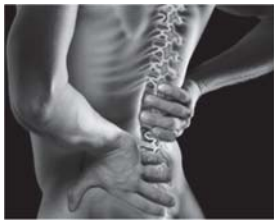
Musculoskeletal Complaints

One of the most common reasons for patients to see health care providers.

Larsson *BMC Musculoskeletal Disorders* 2009
Walsh *Arch PM&R* 2008

Musculoskeletal Complaints

One of the most common reasons for patients to see health care providers.



Larsson *BMC Musculoskeletal Disorders* 2009
Walsh *Arch PM&R* 2008

Musculoskeletal/Spine Pain: Goals

- Patient Goals:

  **What's the diagnosis?*

  **What are the treatment options?*

- Communication regarding goals is key to successful outcomes.
  - Walsh *Arch PM&R* 2008

Etiology of Musculoskeletal / Spine Pain

Often <u>unclear</u> in the absence of fracture, tumor, infection, significant arthritis

- Predominantly non-traumatic.
- Broad differential diagnosis despite careful history and exam.
  - Gaeta *Am J Roent* 2008
  - Schoffl *J Sport Med Fit* 2007
  - O'Connor *Phys Sports Med*1997

Etiology of Musculoskeletal / Spine Pain

Often <u>unclear</u> in the absence of fracture, tumor, infection, significant arthritis

- Discouraging for patient's with:
  - Chronic symptoms
  - Uncertain etiology
  - Numerous diagnostic studies
  - Extensive treatments
    - Lillrank *Soc Sci Med* 2003

Etiology of Musculoskeletal / Spine Pain

Often <u>unclear</u> in the absence of fracture, tumor, infection, significant arthritis

- Results in decreased function and further impairment secondary to poor mental and emotional wellbeing.
  - Foster *Pain* 2010

PERSISTENT MUSCULOSKELETAL / SPINE PAIN

<u>Significant utilization of healthcare resources often without meaningful improvements!</u>

Von Korff *Pain*1988

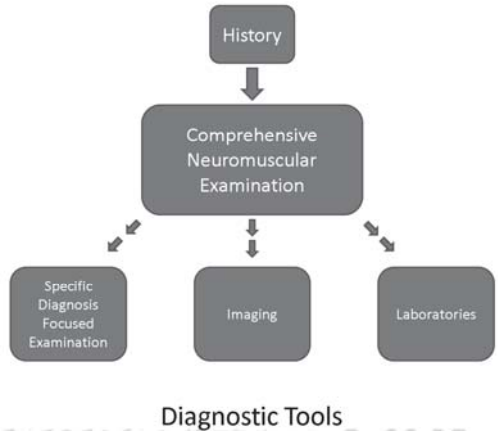
It is difficult to find a black cat in a dark room…

It is difficult to find a black cat in a dark room… especially if there is no cat. *Confucius*

Louis Pasteur

- *Chance favors the prepared mind*

What are our Diagnostic Tools?

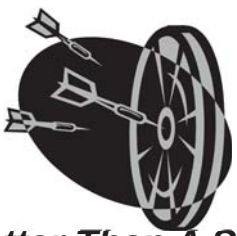


Establish Diagnosis

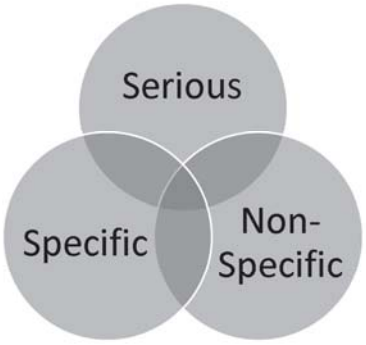
- Activity/event related to onset
- Symptoms: intermittent vs. constant
- Extremity & kinetic chain evaluation
- Evaluate during activity, fatigue
- Ancillary studies

Avoid over-diagnosing

An <u>Accurate</u> Diagnosis



Is Better Than A <u>Specific</u> Diagnosis



Accurate Musculoskeletal Diagnosis

The majority of conditions that lead to persistent musculoskeletal / spine pain ….

…are not confirmed by imaging studies

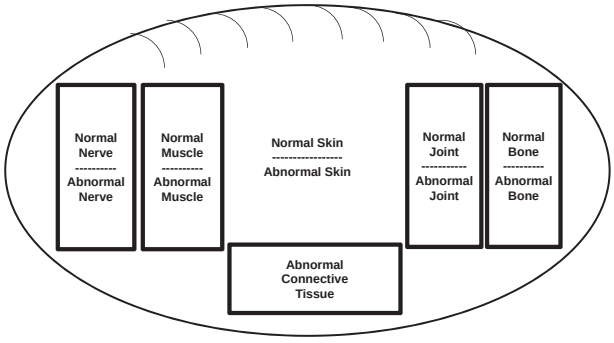
The majority of conditions that lead to persistent musculoskeletal / spine pain ….

… may be further complicated by psychological factors

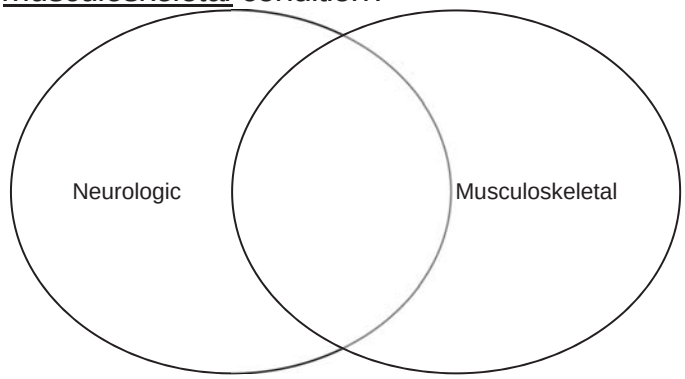
Don't *memorize* the phonebook…

…*learn* the alphabet!

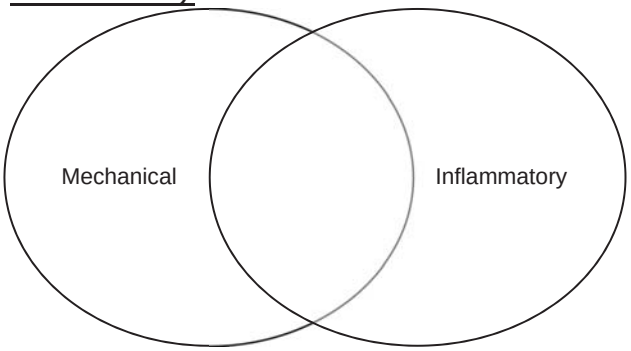
Skin, Bone and Musculature Roadmap



Roadmap: Is this a <u>*neurologic*</u> or <u>*musculoskeletal*</u> condition?



Musculoskeletal conditions: Is this ***mechanical*** or ***inflammatory***?



The prepared mind…

Where does it hurt?

What's there that can hurt?

Musculoskeletal / Spine Injuries

Tendons
Muscles
Ligaments
Joints
Bursa
Nerves
Referred Pain

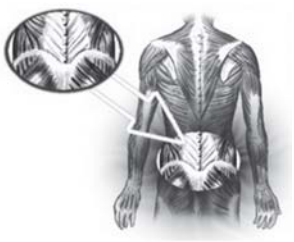
Musculoskeletal / Spine Injuries

Tendons
Muscles
Ligaments
Joints
Bursa
Nerves
Referred Pain

Know the patterns!

Musculoskeletal / Spine Injuries

- Tendons
  - Pain with palpation
  - Pain with passive stretch
  - Pain with contraction against resistance

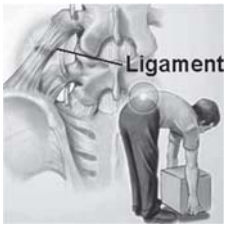


Musculoskeletal / Spine Injuries

- Muscles
  - Pain with palpation
  - Pain with passive stretch
  - Pain with contraction against resistance

Musculoskeletal / Spine Injuries

- ◆Ligaments
  - ◆Pain with palpation
  - ◆Pain with passive stretch
  - ◆Joint instability



Musculoskeletal / Spine Injuries

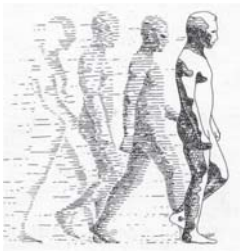
- ◆Joints
  - ◆Pain with motion
  - ◆Pain with loading

Musculoskeletal / Spine Injuries

- ◆Nerves
  - ◆Numbness
  - ◆Weakness
  - ◆Radiating pain

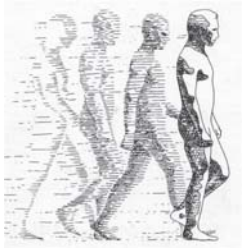
Chronic orthopedic conditions

- Physical Examination
  - Rule out referred pain



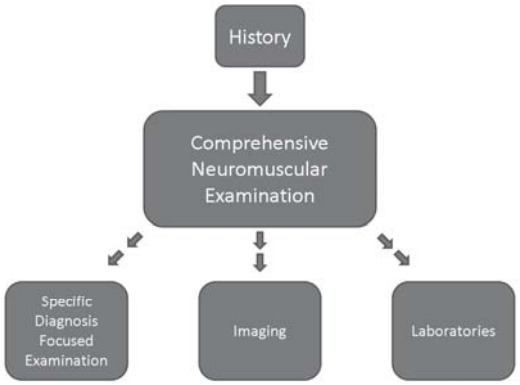
Chronic orthopedic conditions

- Physical Examination
  - *Recreate the symptoms… and know what you did!*

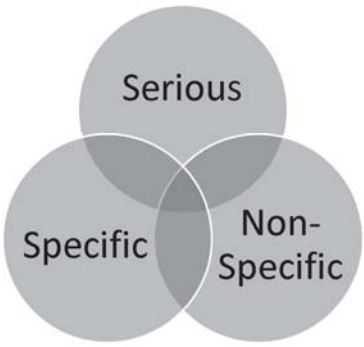


"Red Flags"

- Trauma
- Age > 50 yo
- Fever
- Weight Loss
- Night/Rest Pain
- Hx of Cancer
- Recurrent Pain or Failure to Improve
- Bowel or Bladder Dysfunction
- Sensory or Motor Deficits
- Leg > Back Pain



Diagnostic Tools



Accurate Musculoskeletal Diagnosis

The prepared mind…

Where does it hurt?

What's there that can hurt?

Roadmap

A. **Neurologic**
   CNS/PNS
B. **Musculoskeletal**
   Mechanical
   Intra-articular
C. **Musculoskeletal**
   Mechanical
   Extra-articular
D. **Musculoskeletal**
   Inflammatory
   Intra-articular
E. **Musculoskeletal**
   Inflammatory
   Extra-articular

- 32 y.o. woman
- Pain and stiffness in hands for 8 weeks
- Gradual increase in symptoms over time, some relief with ibuprofen
- Difficulty holding heavy pots while cooking
- Stiffness in morning lasting 3-4 hours
- Significant fatigue over past few months

Roadmap

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C. **Musculoskeletal**
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D. **Musculoskeletal**
   Inflammatory
   Intra-articular
E. **Musculoskeletal**
   Inflammatory
   Extra-articular

- Social history
  - married, 2 children (3 m.o., 3 y.o.)
  - no alcohol, no tobacco
- Medications
  - occasional ibuprofen
- Family history
  - mother with autoimmune thyroid disease

Roadmap

A. **Neurologic**
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C. **Musculoskeletal**
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   Extra-articular
D. **Musculoskeletal**
   Inflammatory
   Intra-articular
E. **Musculoskeletal**
   Inflammatory
   Extra-articular

- Physical exam:
  - Mild tenderness of wrists, fingers
  - Tenderness bilaterally at MTPs, warmth of bilateral knees without effusions
  - Mild bilateral synovitis of wrists, 2nd and 3rd MCPs bilaterally and majority of PIPs

Roadmap

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E. **Musculoskeletal**
   Inflammatory
   Extra-articular

- Xrays
  - Bilateral hands - normal
- Labs
  - HCT 33% (mild anemia)
  - Normal wbc & platelets
  - Creatinine 1.0 (0.8-1.3 mg/dL)
  - Urinalysis negative
  - ANA 1:40 (nl <1:160)
  - RF 120 (nl <40)
  - Anti-CCP antibody 99 (>60 strong positive)

Roadmap

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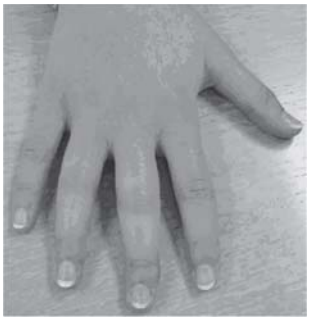
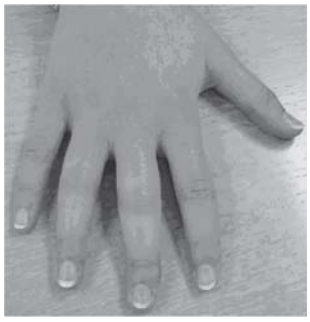


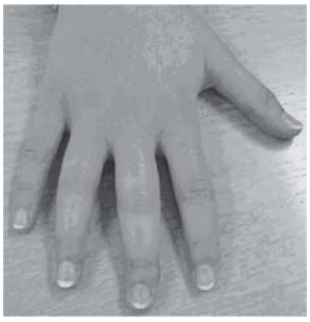
Diagnosis?

A. **Osteoarthritis**
B. **Aseptic toxic synovitis**
C. **Rheumatoid arthritis**
D. **Stenosing tenosynovitis**
E. **Cellulitis**



Rheumatoid Arthritis

Musculoskeletal
Inflammatory
Intra-articular



Rheumatoid Arthritis

Roadmap

A. **Neurologic**
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B. **Musculoskeletal**
   Mechanical
   Intra-articular
C. **Musculoskeletal**
   Mechanical
   Extra-articular
D. **Musculoskeletal**
   Inflammatory
   Intra-articular
E. **Musculoskeletal**
   Inflammatory
   Extra-articular

- 35 year old female
- 1 month history of right greater than left shoulder pain
  - Started after increasing her swimming
- Pain at the posterior lateral shoulder
- Increased with overhead activity

Roadmap

A. **Neurologic**
   CNS/PNS
B. **Musculoskeletal**
   Mechanical
   Intra-articular
C. **Musculoskeletal**
   Mechanical
   Extra-articular
D. **Musculoskeletal**
   Inflammatory
   Intra-articular
E. **Musculoskeletal**
   Inflammatory
   Extra-articular

- Significantly increased right shoulder pain 2 weeks ago
- Started with sudden onset of pain and crepitus while lifting toddler
- Associated with 4 days of diffuse RUE paresthesias
- Mild right sided neck pain with radiation to the clavicle

Roadmap

A. **Neurologic**
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   Intra-articular
C. **Musculoskeletal**
   Mechanical
   Extra-articular
D. **Musculoskeletal**
   Inflammatory
   Intra-articular
E. **Musculoskeletal**
   Inflammatory
   Extra-articular

- Medical History
  - History of remote left shoulder dislocation while competitive gymnast
  - Chronic intermittent low back pain relieved with rest
  - History of cervical disc herniation and radiculitis – resolved with conservative treatment
  - Left foot stress fracture – in boot
  - Allergic rhinitis – on medication

Roadmap

A. **Neurologic**
   CNS/PNS
B. **Musculoskeletal**
   Mechanical
   Intra-articular
C. **Musculoskeletal**
   Mechanical
   Extra-articular
D. **Musculoskeletal**
   Inflammatory
   Intra-articular
E. **Musculoskeletal**
   Inflammatory
   Extra-articular

- Physical Exam
  - Normal BUE motor
  - Mildly positive left and right impingement maneuvers with decreased AROM, PROM
  - Decreased cervical ROM without pain
  - Negative Spurling's maneuver
  - Normal sensation and reflexes of the BUE
  - Negative Speed's, positive O'Brien's
  - Shoulder X-ray unremarkable

Roadmap

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C. **Musculoskeletal**
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D. **Musculoskeletal**
   Inflammatory
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   Inflammatory
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- Imaging?

Roadmap

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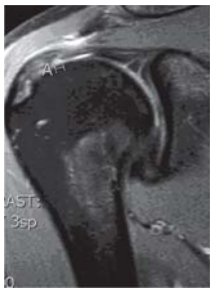
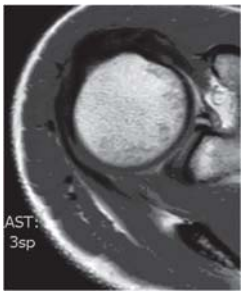
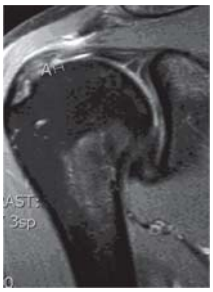
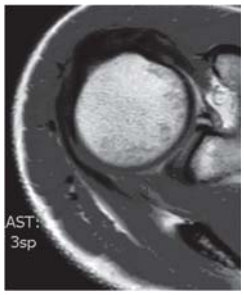




Diagnosis?

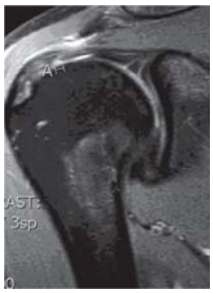
A. **Supraspinatus tendinitis**
B. **Glenohumeral osteoarthritis**
C. **Superior labral tear**
D. **Avascular necrosis**
E. **Complex regional pain syndrome**

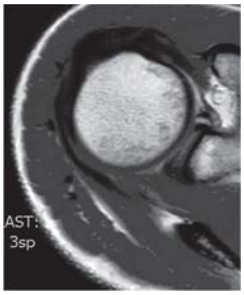




Supraspinatus tendinopathy
Superior labral tear

Musculoskeletal
Mechanical
Intra-articular
Musculoskeletal
Mechanical
Extra-articular





Supraspinatus tendinopathy
Superior labral tear

Musculoskeletal
Mechanical
Intra-articular
Musculoskeletal
Mechanical
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Roadmap

A. **Neurologic**
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B. **Musculoskeletal**
Mechanical
Intra-articular
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Extra-articular
D. **Musculoskeletal**
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E. **Musculoskeletal**
Inflammatory
Extra-articular

- 48 y.o. male
- 4 month history of
  - Neck pain
  - Left shoulder pain
- No acute event or injury
- Pain at night & neck/shoulder ROM
- Taking ibuprofen
  - With minimal help
- PMHx unremarkable
  - No prior hospitalizations or surgeries
  - No prescription medications

Roadmap

A. **Neurologic**
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Mechanical
Intra-articular
C. **Musculoskeletal**
Mechanical
Extra-articular
D. **Musculoskeletal**
Inflammatory
Intra-articular
E. **Musculoskeletal**
Inflammatory
Extra-articular

- FHx
  - History of “arthritis”, diabetes
- SHx
  - Computer programmer
  - Sedentary work
  - Increased symptoms with computer use
- 2 month history of
  - Paresthesias left thumb, index finger
  - Left arm weakness

Roadmap

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Mechanical
Intra-articular
C. **Musculoskeletal**
Mechanical
Extra-articular
D. **Musculoskeletal**
Inflammatory
Intra-articular
E. **Musculoskeletal**
Inflammatory
Extra-articular

- Physical Exam
  - Normal vitals, gait, transferring
  - Decreased neck ROM due to pain
  - Normal shoulder ROM
    - Negative Spurling’s Maneuver
  - Symmetric 1+ reflexes biceps, triceps, brachioradialis
  - Decreased sensation left thumb and index finger
  - 4/5 left wrist extensors

Roadmap

A. **Neurologic**
CNS/PNS
B. **Musculoskeletal**
Mechanical
Intra-articular
C. **Musculoskeletal**
Mechanical
Extra-articular
D. **Musculoskeletal**
Inflammatory
Intra-articular
E. **Musculoskeletal**
Inflammatory
Extra-articular

- Imaging

• MRI – cervical spine

Roadmap

A. **Neurologic**
   CNS/PNS
B. **Musculoskeletal**
   Mechanical
   Intra-articular
C. **Musculoskeletal**
   Mechanical
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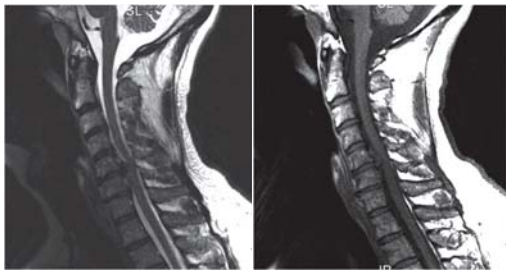

• MRI – cervical spine

Roadmap

A. **Neurologic**
   CNS/PNS
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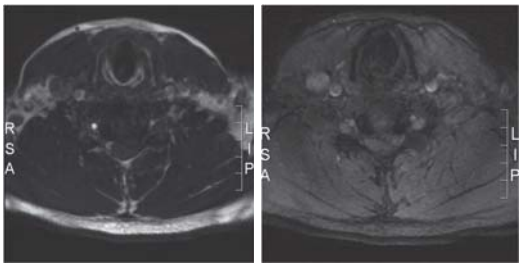
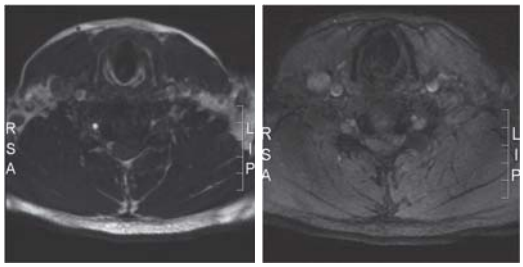


• MRI – cervical spine

Diagnosis?

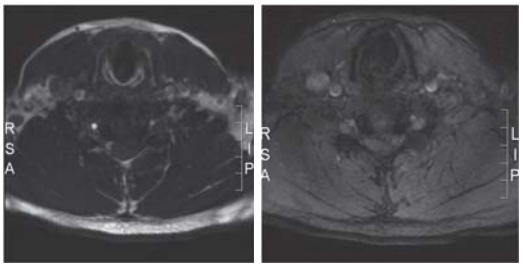
A. **Brachial neuritis**
B. **Left C6 radiculopathy**
C. **Proximal median neuropathy**
D. **Left C8 radiculopathy**
E. **Cervical myelopathy**



• MRI – cervical spine

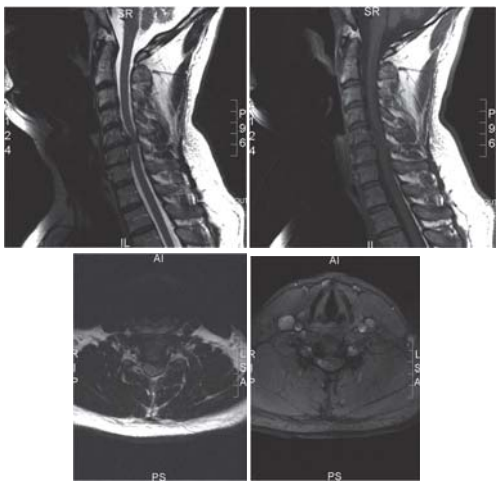
Left C6 Cervical Radiculopathy

Neurologic
CNS/PNS



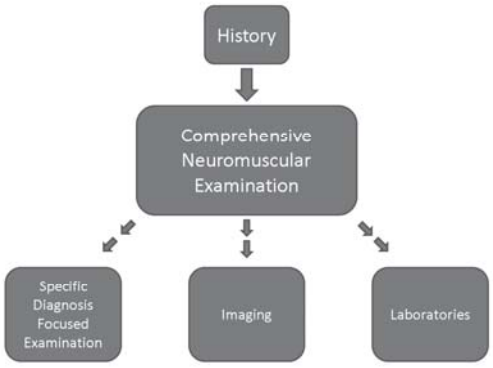
Left C6 Cervical Radiculopathy

Neurosurgery Consult
Surgery recommended
Patient declined
5 months later
No arm/neck pain
4/5 left wrist extensor weakness
9 months later
No pain or numbness
5/5 Strength
Full pain free cervical spine ROM

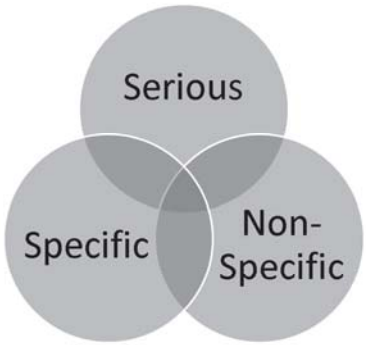


Left C6 Cervical Radiculopathy

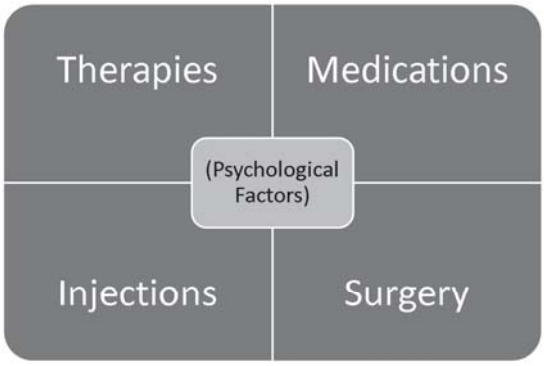
Neurologic
CNS/PNS



Diagnostic Tools



Accurate Musculoskeletal Diagnosis



Treatment Options

Overuse Injuries
and Strategies for Treating Them

Ryan Norton, DO, CAQSM
Assistant Professor
Family and Sports Medicine
Oregon Health and Sciences University

OHSU

No financial disclosures

- Medical Director, OHSU Human Performance Lab
- Co-Medical Director, Portland Fire and Rescue
- Team Physician, Mountainside High School
- Endurance Athlete

Objectives

- Define overuse injury
- Most common
- Risk factors
- Diagnosis, treatment, and management
- When to get imaging?
- When to refer?
- Treatment pearls
- Stay awake

What is an overuse injury?

DEMAND >> recovery

Microtrauma to soft tissue and/or bone

Occurs over a period of time (non-acute)

Repetitive movement

Most Common Overuse Injuries

- Achilles
- IT band
- Plantar fasciitis
- Tennis elbow
- Rotator cuff

- ✓ Shin splints
- ✓ Stress fracture
- ✓ Patellofemoral syndrome

- Osgood-Schlatter
- Sever
- Sinding-Larsen-Johansson
- Little league elbow
- Little league shoulder

How commonly do they occur?

- TRAILS study followed 300 runners for 2 years. 66% sustained an overuse injury during the study period
- 45-54% of all sports injuries are due to overuse
- Survey of musicians found up to 68% report MSK complaint

Can they be prevented?

Intrinsic Risk Factors
- Growth plate
- Age (puberty and elderly)
- Level of conditioning
- Anatomical factors
- Psychological factors

Extrinsic Risk Factors
- Training workload
- Competition schedule
- Sport specialization
- Equipment
- Footwear
- Environment / playing surface
- Psychological factors

The run down

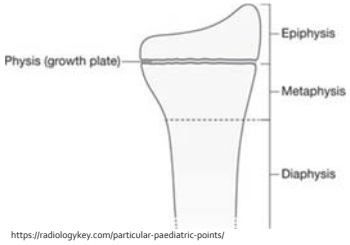
- Pediatric overuse
- Achilles
- Plantar fascia
- Lateral epicondylitis (Tennis elbow)
- Stress fracture
- Patellofemoral syndrome

Pediatric Overuse Injuries

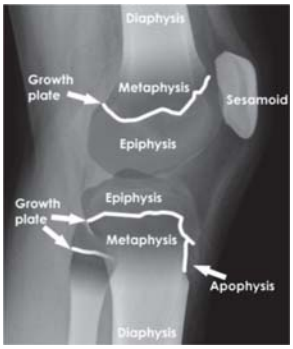
Osgood-Schlatter disease
Sever disease
Sinding-Larsen-Johansson
Little League Elbow and Shoulder

Common theme

- Physis, or growth plate, is the weakest part of the bone
- Traction at the physis leads to separation
- Often site of major tendon insertion, where tension is high
- Osseous growth outpaces muscle / tendon lengthening and muscle hypertrophy
- Highest risk during periods of high growth velocity
- Repetitive running, jumping, throwing
- Coaches and parents play a major role
- If sudden onset or pop, rule out apophyseal avulsion fracture
- Bilateral imaging



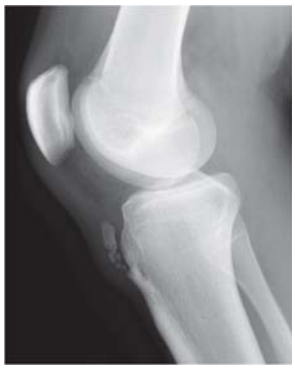
https://radiologykey.com/particular-paediatric-points/



https://d1yboe67510e2cu.cloudfront.net/i/100b1498e27630356c42f584f8c62c3efb34322c

Osgood-Schlatter

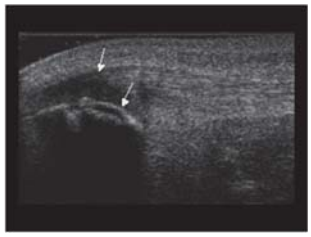
- Traction apophysitis at the patellar tendon insertion on tibial tuberosity
- Fragment and irregularity at ossification center
- Girls 8-13 and Boys 10-15
- Activity modification, ice, NSAIDs, padding
- Clinical diagnosis; delay imaging if typical presentation
- Resolves weeks to months, or when physis closes
- May result in permanent prominence



https://radiopaedia.org/articles/osgood-schlatter-disease

Sinding-Larsen-Johansson

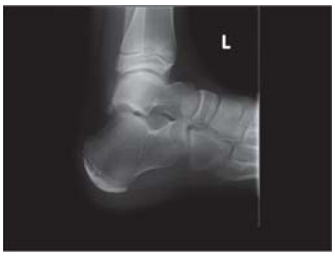
- Inferior pole of patella
- Similar age range
- Usually a clinical diagnosis
- If acute onset and associated with hyperextension, rule out patellar sleeve fx (check extensor mechanism)
- Clinical diagnosis; delay imaging if typical presentation
- Activity modification, ice, NSAIDs, padding
- Weeks to months
- Completely resolves when physis closes

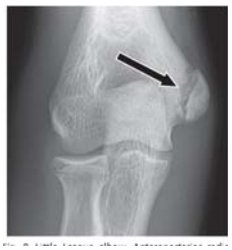


http://www.ultrasoundcases.info/Slide-View.aspx?cat=59&case=3303

Sever Disease

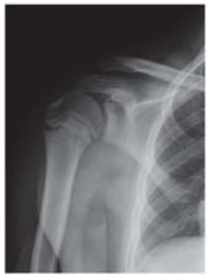
- Traction apophysitis at the Achilles tendon insertion on the calcaneus
- Predominantly Boys, 8-12
- Clinical diagnosis; delay imaging if typical presentation
- Activity modification, ice, NSAIDs, padding, heel lift
- Resolves when physis closes





Little League Elbow

- Repetitive traction on medial epicondylar apophysis



Little League Shoulder

- Tension across proximal humeral epiphysis

https://www.semanticscholar.org/paper/Imaging-pediatric-sports-injuries%3A-upper-extremity.-Davis/67984a0145d99b8c644d7d3511a7a16c5ef0a93d3/figure/7

- Cumulative stress
- Progressive arm pain with throwing
- Bilateral plain films
- Early detection is key
- Treatment is prolonged rest (up to 3-5 months)
- ROM > Strength > Return to throwing
- Preventable!
- Know the risk factors

https://abc7news.com/sports/hawaii-shuts-out-georgia-3-0-to-move-to-llws-championship/4052527/

Pitch Smart Risk Factors

1) Pitching while fatigued
2) Too many innings per year
3) Not enough time off
4) Too many pitches, not enough rest
5) Pitching on consecutive days
6) Excessive throwing when not pitching
7) Multiple teams
8) Injuries to other body regions
9) Poor upper extremity strength and conditioning
10) Showcases
11) Breaking pitches at young age
12) Radar gun use

"…age, height, playing for multiple teams, pitch velocity, and arm fatigue were found to be independent risk factors for throwing arm injuries…"

Clinical Sports Medicine Update

Risk Factors for Elbow and Shoulder Injuries in Adolescent Baseball Players

A Systematic Review

Ryan Norton,*† DO, Christopher Honstad,† MD, Rajat Joshi,† BS, Matthew Silvis,† MD, Vernon Chinchilli,‡ PhD, and Aman Dhawan,† MD
Investigation performed at Penn State Health Milton S. Hershey Medical Center, Hershey, Pennsylvania, USA

"One additional consideration is the biological development of an immature athlete, who during the critical high school years undergoes a growth spurt, allowing for a pitch to be thrown with suddenly increased velocity before the growth plates have closed or the supportive musculature has adapted to the increased stress placed on the elbow"

"This scenario, in which a young athlete has the ability to perform at a level before his body has fully developed to withstand the resulting stress, is one that likely occurs during a finite period of time during development, is difficult to predict, and is even harder to prevent"

Conclusions

- Playing for multiple teams, pitch velocity, arm fatigue, and pitches per game (elbow) appear to be modifiable extrinsic risk factors
- Great utility in Pitch Smart guidelines, but caution against too much focus on pitch counts alone
- Find the sweet spot!
- 15-19 yo age group

Achilles Tendinopathy

But first….

Tendonitis vs Tendinosis vs Tendinopathy?

Tendinopathy: generic term for disease or injury to the tendon

Tendonitis: inflammatory response to tendon injury

Tendinosis: chronic degeneration without inflammation

>>>Tendinosis<<<

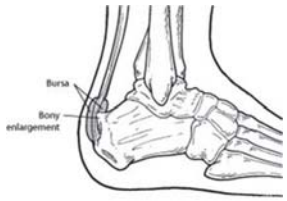
- Repetitive forces cause micro tears of the collagen matrix, disrupting cross links
- Bleeding may or may not occur; if significant bleeding occurs from larger partial tear or injury to sheath, the body may activate true inflammatory response
- Fibroblast cells within the tendon can be enough for self-healing; they differentiate into other cell types necessary for repairing tendon
- Tendinosis occurs when the self healing properties fail to create healthy collagen matrix and instead scaring occurs

Achilles Tendinopathy

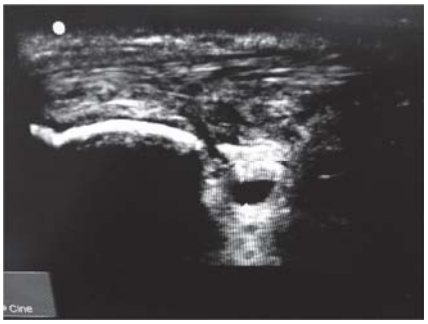
- **Anatomy and Physiology**
  - Muscle / tendon crosses two joints
  - involved in locomotion (push off phase of gait cycle)
  - watershed area just proximal to insertion
- **Epidemiology**
  - 50% of runners during their career, older athletes
- **Risk factors**
  - repetitive eccentric stress from uphill running or sprinting
  - Haglund deformity
  - Hyperpronation

- **Presentation**
  - pain at insertion or mid portion
  - +/- swelling
  - Calf raises or push off
- **Diagnosis**
  - clinical exam (palpation)
  - +/- imaging (US, Xray)
  - Bursitis? Haglund? Rupture?
- **Treatment**
  - Relative rest +/- NSAIDs
  - Flexibility and strength
  - Heal lift, footwear (correct overpronation)
  - Shock wave therapy
  - Regenerative injection
  - Surgery last resort

Achilles Tendinopathy



https://www.foothealthfacts.org/conditions/haglund's-deformity

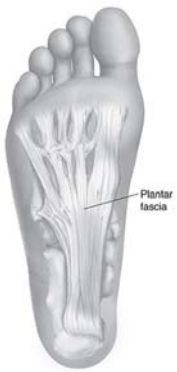


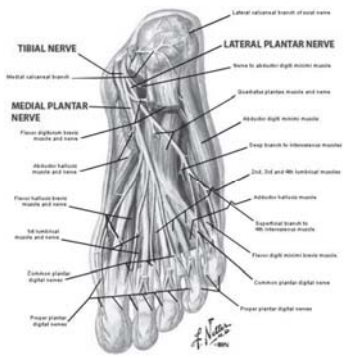
Plantar fasciopathy

- **A&P –**
  - Originates from calcaneus, extends to insertion of proximal phalanges
  - Largest band originates on medial calcaneus
  - Maintains rigid arch from heel lift to toe-off
- **Epidemiology –**
  - 7% of Hood to Coast runners
  - Most common cause of heel pain
- **Risk factors –**
  - Pes planus and pes cavus
  - Decreased ankle dorsiflexion
  - Decreased foot and ankle strength
  - Running, walking barefoot

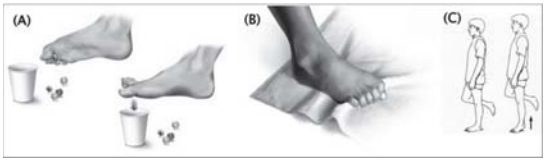
Plantar fasciopathy

- **Presentation**
  - Pain at origin on medial calcaneus
  - First steps in the morning
  - May improve with exercise
- **Diagnosis**
  - Clinical exam
  - +/- imaging (US, Xray, MRI)
  - Medial calcaneal and lateral plantar nerve
- **Treatment**
  - Patient education
  - Non surgical 90% of the time
  - Flexibility, strength, massage
  - Orthosis, night splints
  - ?injection ?surgery ?gait analysis

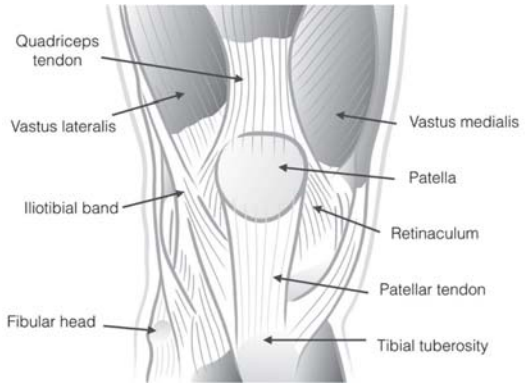




https://www.fairview.org/patient-education/83735



https://www.roguefitness.com/pro-tec-spiky-ball
http://runfonefoot.com/plantar-fascia-and-metatarsal-bones-strengthening-exercises-for-forefoot-running/
https://eliminateheelpain.com/massage-therapy-for-plantar-



https://humananatomylibrary.co/photos/7538/soft-tissue-knee-patient-information-gavin-mchugh-anterior-knee-anatomy-picture.asp

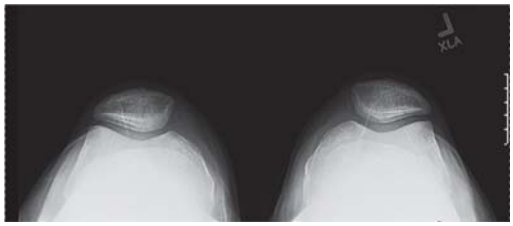
Patellofemoral Syndrome

- **A&P –**
  - Patella articulates with distal femur
  - Insertion of quadriceps tendon and origin of patellar tendon
- **Epidemiology –**
  - Most common overuse injury in recreational runners
  - Chondromalacia only 45%
- **Risk factors –**
  - Females
  - Patella alta
  - Hypermobility or malalignment
  - Overpronation
  - Femoral anteversion or tibial torsion
  - Quad or hip muscle weakness
  - IT band or quad tightness

Patellofemoral Syndrome

- **Presentation**
  - Diffuse anterior knee pain
  - Not acute, not swollen
- **Diagnosis**
  - Clinical exam
    - single leg squat, Trendelenburg
    - J sign, hypermobility
    - VMO atrophy
    - Ober
  - Imaging only if recalcitrant to rule out other causes
- **Treatment**
  - Patient education is key
  - Activity modification
  - Hip and quad strength / flexibility to correct imbalance
  - McConnell vs Kinesio taping
  - Gait analysis
  - Lateral retinacular release?

Merchant (Skyline) Views



https://openi.nlm.nih.gov/detailedresult.php?img=PMC3445133_10.1177_1941738110393875-fig8&req=4

Rehab

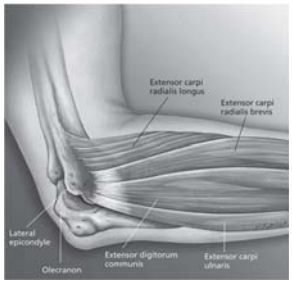
- Squat with hip adduction
- Wall sits
- Single leg squat / step down (not pistol squat)
- Single leg deadlift
- Supine leg lifts, leg externally rotated for VMO
- 7-way hip series

Lateral epicondylitis (misnomer)

- **A&P –**
  - Extensor tendons at lateral epicondyle
  - Extensor carpi radialis brevis degeneration
  - Crosses two joints
- **Epidemiology –**
  - Tennis only 5-10% of cases
- **Risk factors –**
  - Repetitive wrist extension
  - Racquet sports
  - Manual labor

Lateral epicondylitis (misnomer)

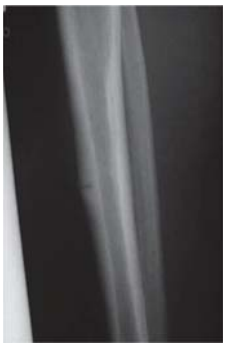
- **Presentation**
  - Lateral elbow pain
- **Diagnosis**
  - Clinical exam –
    - TTP lateral epicondyle
    - Extended elbow, middle finger and wrist extension
    - Rule out posterior interosseous nerve entrapment
  - Consider US to confirm diagnosis
- **Treatment**
  - Activity modification
  - Home therapy
  - Counterforce strap
  - Wrist brace
  - PRP vs AWB vs Prolotherapy



https://www.otsbraces.com/epifoam-tennis-elbow-support

Stress Fractures

- Definition
- Risk factors
- The spectrum
- Diagnosis
- Grading of fractures
- High risk sites
- Return to play



- The rate of breakdown exceeds the remodeling of bone
- Overuse / overload

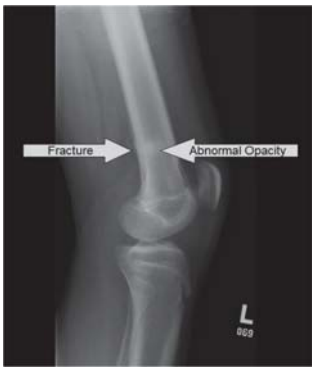
- Spectrum
  - Impact > microfracture > remodeling > overuse > imbalance > stress reaction > Fracture

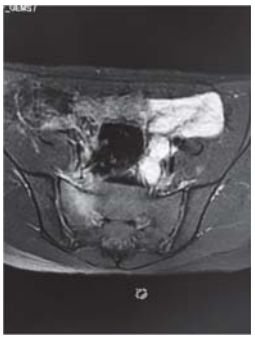
Risk Factors

Extrinsic	Intrinsic
• Training errors (total mileage vs rate of increase) • Recovery time • Nutrition • Shoes	• Muscle imbalance • Foot strike • Gait • Menstrual irregularities • Caucasian • Psychological

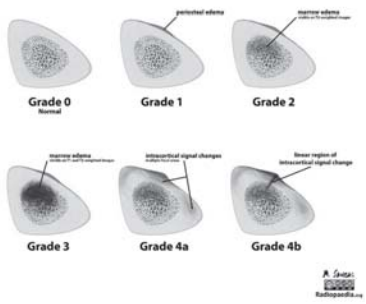
Diagnosis

- Pain that increases during workout, or does not resolve post work-out
- Cannot be explained by other cause
- Brief period of rest ineffective
- Tenderness to palpation of the bone
- Hop test
- Xray
- Presume and treat, repeat xray
- High risk area, or clinically necessary, obtain MRI
- Check labs and ?DEXA





Fredericson classification system
for medial tibial stress syndrome on MRI



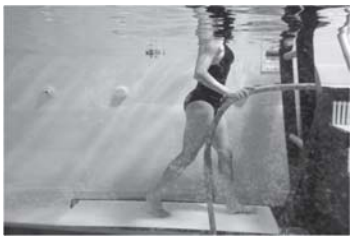
Location, location…

Low Risk
- Medial femoral neck
- Femoral shaft
- Posteromedial tibia (shin)
- Metatarsals 1-4 (feet)
- Calcaneus (heal)
- Fibula
- SI joint
- Pelvis

High Risk
- Lateral femoral neck (tension)
- Anterior tibia (shin)
- Navicular
- 5th metatarsal
- Medial malleolus

Treatment

- Low risk – at least 3-6 weeks
  - non weight bearing until able to walk pain free
  - Introduce non weight bearing / low impact activity to maintain fitness
  - 2 weeks before impact training
  - Gradual return to sport
- High risk - at least 10 -12 weeks
  - Surgical consultation
  - Non weight bearing at least 4-6 weeks
  - Partial weight bearing
  - Low impact activity



Patience is key

Education

Gradual return

Utilize cross training to build fitness

Take home point…

The body's ability to withstand physical stress, and thus the volume and intensity of exercise, should be individualized to the patient

References

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- Running Medicine. Wilder et al. 2017. Healthy Learning
- ACSM Sports Medicine: A comprehensive review. O'Conner et al. 2013. American College of Sports Medicine
- Netter's Sports Medicine. Madden et al. 2010. Saunders

Thank you!!

Ryan Norton DO
nortonry@ohsu.edu

Dazed and Confused - New Insights in Concussion Evaluation - Active Rehab

Jim Chesnutt, M.D.
OHSU Concussion Program
Orthopaedics & Rehabilitation
Neurology, Family Medicine
Rebound Sports Medicine

OR Governor's Task Force TBI
OCAMP: Co-Director
OHSU TBI Inititiave Co-Chair

Learning Objectives:

- Update on new state laws and policies
- Highlight recent OHSU research and collaborations
- Implement active strategies for concussion recovery
- Become familiar with Return to Learn strategies
- Learn about rehabilitation roles and protocols

OHSU Concussion Center

The NW's most comprehensive, multidisciplinary concussion care center

- Cutting edge research and clinical care
  - Concussion rehab and clinical outcomes
  - Sensory Integration: balance & auditory processing
  - Chronic traumatic encephalopathy(CTE)-tau protein
  - Informatics and clinical guidelines

Yearly TBI Scientific Symposium- research to rehab

Partnerships- academics, community and industry

Brain Injury Rehabilitation Center (BIRC)

OCAMP

Stanford

REED COLLEGE

cbirt

Univ Hawaii

ThinkFirst
National Injury Prevention Foundation

Brain Injury Alliance of Oregon

OHSU TBI- PTSD Research to Rehabilitation Scientific Symposium

Dec 13-14, 2019 Dr Steve Broglio, ATC PhD

Collaboration Advancement Award

School of Medicine

Research Roadmap

OHSU TBI Initiative

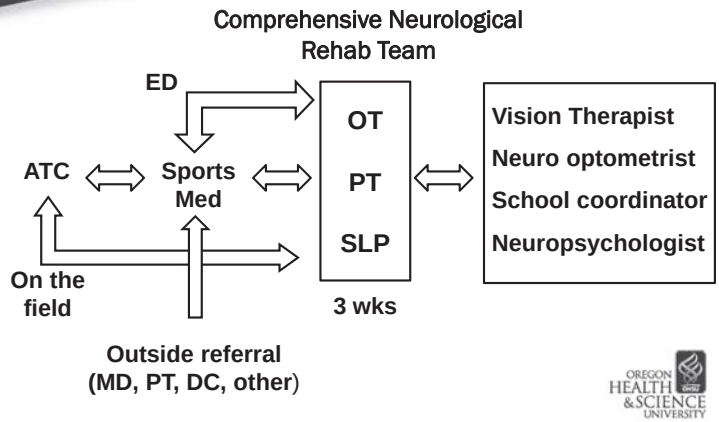
Dr. Jim Chesnutt, SM, Ortho Rehab, FM, Neuro
Dr. George Keepers, Chair of Psychiatry
Dr Nathan Selden, Vice chair Neurosurgery

- Over 150 clinicians and researchers
- Research on basic science pathophys & imaging
- Clinical research: trauma, balance, education
- Multidisciplinary teams, inpt, outpt, outreach
- VA Collaboration, auditory processing, neuro trauma, PTSD, research, rehab protocols
- Medical Informatics, EBM, policy

OHSU Concussion Management

- Pre-season Impact baseline testing
  - Can do whole team or individuals
- Athletic trainers on- field and in injury clinic
- Post –concussion evaluations
  - Physician and ATC evaluations & Impact testing
- Concussion Rehabilitation Team
  - PT, Vestibular/ENT, SLP/ cognitive,OT/vision
- Severe/Chronic: Neuropsych, Neuro, NSurg Sport Concussion Support Group (student/family)

Interdisciplinary approach: OHSU Model



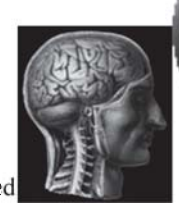
Concussions: The Problem

- We now realize concussions occur more often than previously thought
- Young athletes are at risk for serious short-term and long-term problems
- There is much variation in the knowledge of Health Care Providers managing concussed athletes
- New and emerging technologies will lead to a continuing evolution of care

Concussions: The Oregon Plan

State-wide concussion management program involving all high schools

- Establish state-wide physician network
- Uniform evaluation and management protocol
- Consultation service for coaches, athletes, parents, and physicians
- ImPACT baseline suggested for contact and collision sport athletes



Oregon Concussion Awareness and Management Program (OCAMP)

Multi-disciplinary group across the state:

Educators, Physicians, Neuropsychologists, Certified athletic trainers, Rehab Therapists

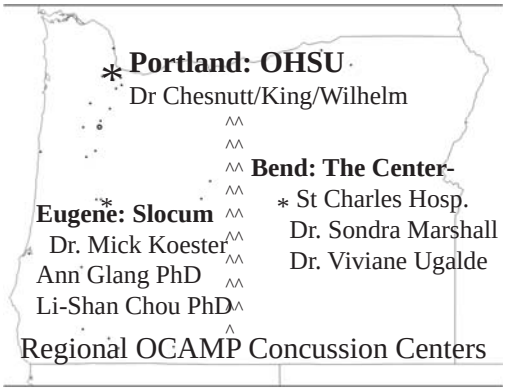
Brain Injury Association of Oregon

Athletic Directors(OADA),

Center for Brain Injury Research and Teaching

Representatives from OSAA , OR Dept of Ed.

Concussions: The Oregon Plan



Each helps "oversee" programs at the "satellite" sites and help local doctors/trainers care for their own athletes

Max's Law: Sports Concussion
(SB 348- April 2009, 1st one passed!)

Max Condradt is an OR brain- injured athlete hurt in football.

- No return-to- play the same day as concussion
- Medical release needed to return to play
- Yearly coach concussion education required
  - Free for coach : www.osaa.org/healthandsafety/concussion.asp
- Effective: July 2009

Left to right: David Kracke, Max Conradt, Governor Ted Kulongoski, Tootie Smith, Sherry Stock

Max's Law: The 4 R's

1. **RECOGNIZE:**
   - all coaches must receive annual training in recognizing the symptoms of concussion.
2. **REMOVE**:
   - no same day return to play
3. **REFER** :
   - must be evaluated by a properly trained medical professional.
4. **RETURN :**
   - all symptoms resolved, graded return to play over about one wk and a medical release has been obtained

Zachery Lystedt Law
Washington HB 1824 5-14-2009

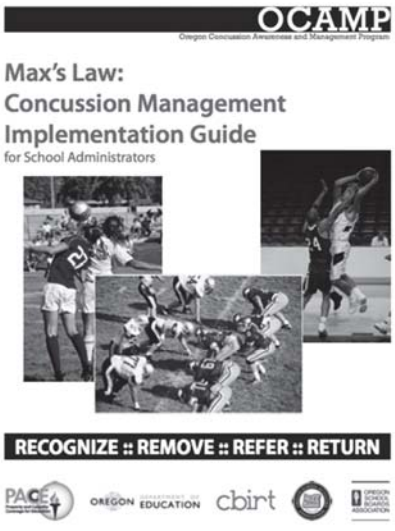
- Youth athletes who are suspected of sustaining a concussion or head injury be removed from play. "When in doubt, sit them out"
- School districts to work with the Washington Interscholastic Activities Association (WIAA) to develop information and policies on educating coaches, youth athletes and parents about the nature and risk of concussion, including the dangers of returning to practice or competition after a concussion or head injury.
- All student athletes and their parents/guardians sign an information sheet about concussion and head injury prior to the youth athlete's initiating practice at the start of each season.
- Youth athletes who have been removed from play receive written medical clearance prior to returning to play from a licensed health-care provider trained in the evaluation and management of concussion.

New 2013 Oregon Bill
Concussions in Club sports
Jenna Sneva, ski racer, >12 concussions
Jenna's Law

What's new in 2018:

SB 217- Add naturopath/ Chiro/ ~~AT~~/ PT to clear mTBI

OHSU to provide concussion education for state law



Concussion Management Team:
Healthcare professional, Physician, Neuropsychologist, Athletic Trainer, Nurse Practitioner, Physician Assistant, Coach, HS Counselor, Teachers and Parents

IMPLEMENT CONCUSSION MANAGEMENT PLAN
TO ADDRESS AND ASSESS PHYSICAL & COGNITIVE NEEDS OF ATHLETE
(Share plan with coach, school, athlete & parent)

Follow a Graduated Return to Exertion & Academics
Follow-up concussion management assessment

- Consider formalized support if symptoms last more than 2-3 months:
  - Contact OR TBI Team
  - 504 Plan or referral to SPED

When symptom free and released by Concussion Management Team proceed to full activity level

GOVERNOR'S TASK FORCE ON TRAUMATIC BRAIN INJURY

REPORT ON FINDINGS AND RECOMMENDATIONS

OUR VISION

The vision of the Governor's Task Force on TBI is that Oregon will establish and maintain a comprehensive, public-private system of coordinated care and supports for individuals with brain injury of all ages, severity levels, and backgrounds that facilitates maximum community engagement and quality of life.

GOVERNOR'S TASK FORCE ON TRAUMATIC BRAIN INJURY: EXECUTIVE ORDER NO. 13-02, 2013

REPORT ON FINDINGS AND RECOMMENDATIONS

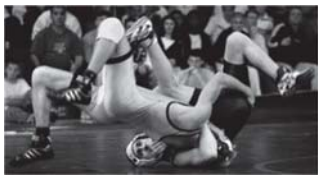
August 2016

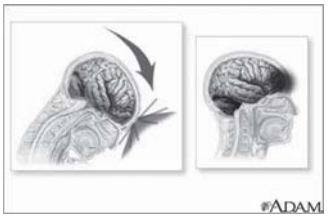
RECOMMENDATIONS AT A GLANCE

1. Increase educational outreach
2. Establish a TBI Clinical Registry
3. Establish a centralized "road map" of services and resources
4. Establish a statewide program of care coordinators
5. Develop an equitable system of care and services
6. Develop a communication system to improve coordination across agencies
7. Establish sustainable, equitable funding mechanisms
8. Establish the Governor's Traumatic Brain Injury Coordinator and Advocate in the Office of the Governor

What is a Concussion?

- A concussion is a **mild traumatic brain injury** that interferes with normal function of the brain
- Evolving knowledge- "dings" and "bell ringers" are brain injuries- no such thing as a *mild concussion*
- Loss of consciousness **is not common** in concussion(<90%)
- (GCS 13-15)

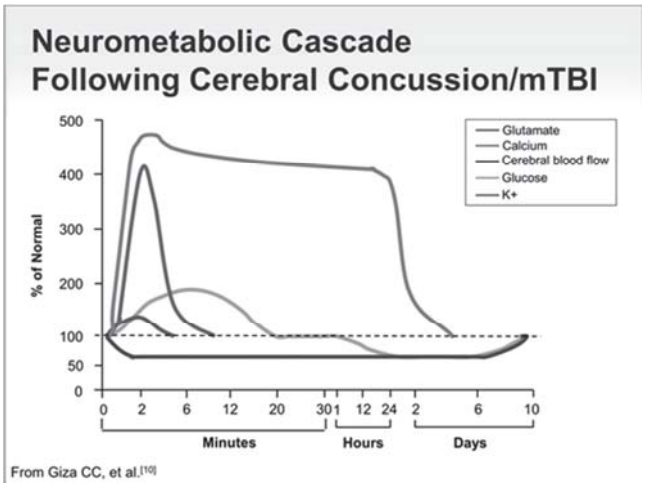




Concussion Mechanics/ Biology

- Aceleration/ Deceleration
- Linear/ rotational
- Neurometabolic energy crisis
- Decreased cerebral blood flow, glucose
- Abnormalities Glutamate, K, Na, Ca, etc
- Endocrine, neurochemical abnormalities
- Neuron injury and Axon shearing
- Prefrontal motor cortex, corpus callosum and central processing network

Metabolic Crisis in Brain post TBI-



Concussion: Helmet to helmet hit

Newer Data High School RIO 2015-6

Injury rate per 10,000 player exposures competition

- Boy's Football-- 10.5
- Girls' soccer 9.2
- Girls' lacrosse 8.6
- Boys' Ice Hockey – 7.6
- Boys' Wrestling – 5.5
- Girls' basketball 5.5
- Boys' Lacrosse – 5.0
- Boys' Soccer – 4.2
- Girls' field hockey 4.1
- Boys' basketball 3.9

Soccer-Football and Concussion

World Soccer and Science Conf -2016

- Estimate 270 million players world wide
- 27 mil in N America
- 40 % concussions for arm/ elbow to head
- 60% contact related to headers but not headers themselves
- Female more ground contact
- Heading *may* be related to brain injury
  - Usually at least 1000yr worse if over 1800?

Concussion Symptoms

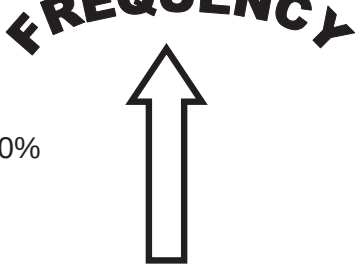
variable for each individual in terms of type, intensity and duration:

- **Symptoms:** (eg, headache, foggy or emotional) l
- **Physical signs** (eg, loss of consciousness, amnesia, neurological deficit)
- **Balance impairment** (eg, gait unsteadiness)
- **Behavioral changes** (eg, irritability)
- **Cognitive impairment (**eg, slowed reaction times)
- **Sleep/wake disturbance** (eg, somnolence,drowsiness)

Berlin 2016. McCrory P, *et al. Br J Sports Med* 2017.

Symptoms

- Headache 75%
- Blurred vision 75%
- Dizziness 60%
- Nausea 54%
- Memory/ confusion 40-60%
- Double vision 11%
- Noise sensitivity 4%
- Light sensitivity 4%
- Loss of consciousness 5-10%



Carney, Ghajar et al 2014

New Definition
evidence- based systematic review

1.) a change in brain function;

2.) following a force to the head(+/- hit)

-a potentially concussive event;

3). may (or may not) be accompanied by temporary LOC;

4.) identified in awake individuals; and

5.) includes measures of neurologic and cognitive dysfunction. (Carney, Ghajar et al., 2014.)

Concussion -consistent and prevalent diagnostic indicators

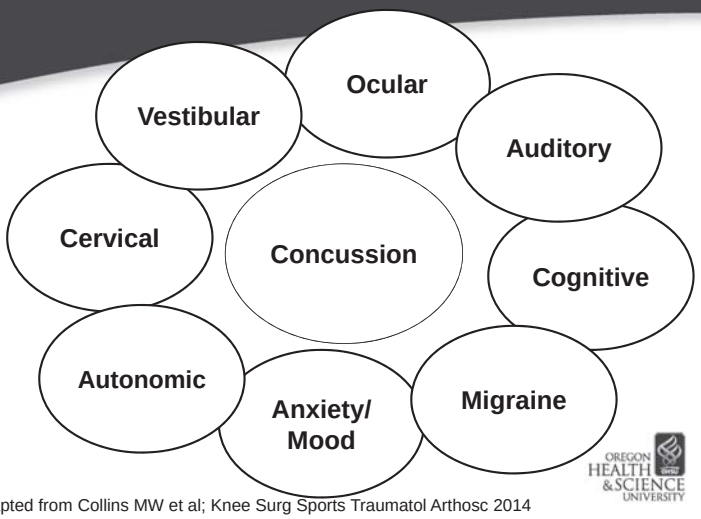
1.) observed and documented disorientation or confusion immediately after the event;

2.) impaired balance within 1 day after injury;

3.) slower reaction time within 2 days after injury; and /or

4.) impaired verbal learning and memory within two days after injury. (Carney,Gjahar, et al., 2014.)

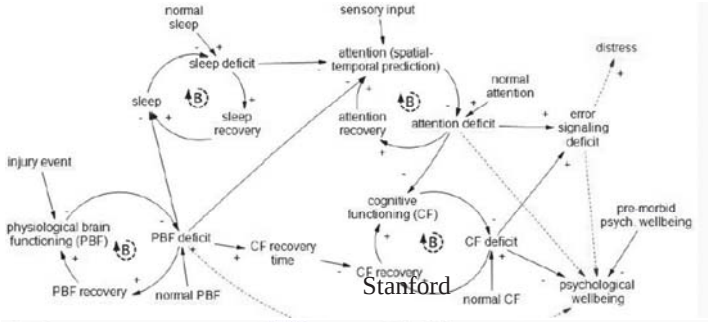
Interdisciplinary Approach: Concussion Symptoms



Adapted from Collins MW et al; Knee Surg Sports Traumatol Arthosc 2014

Dynamic Model of Concussion

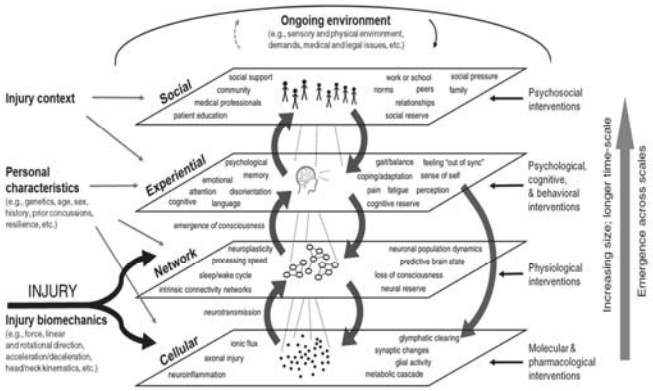
work started in 2013 on scale model and causal loop diagram model and finally papers published 2017 and 2018



Concussion As a Multi-Scale Complex System: An interdisciplinary Synthesis of Current Knowledge

Erin S. Kenzie, Elle L. Parks, Erin D. Bigler, Miranda M. Lim, James C. Chesnutt, and Wayne Wakeland

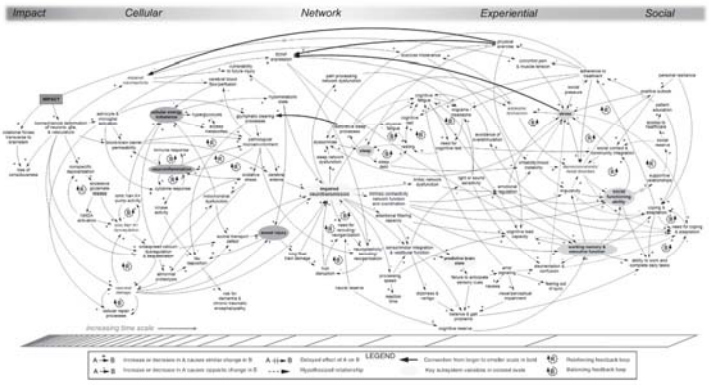
Frontiers in Neurology September 2017 | Volume 8 | Article 513



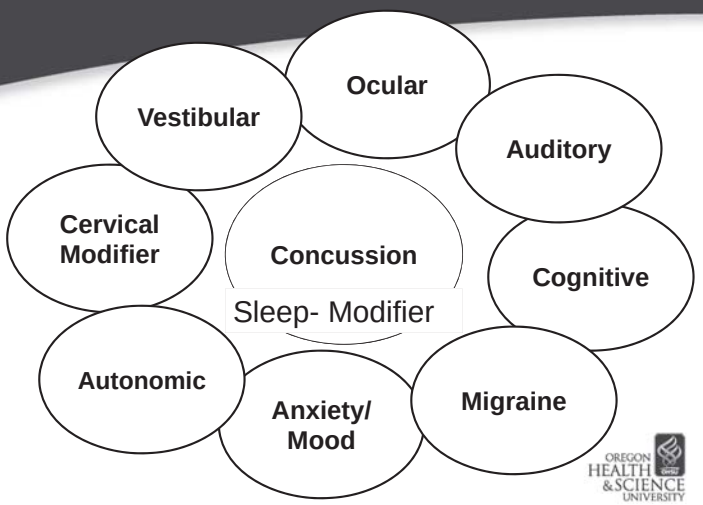
The Dynamics of Concussion: Mapping Pathophysiology, Persistence, and Recovery With Causal-Loop Diagramming

Benzie ES, Parks EL, Bigler ED, Wright DW, Lim MM, Chesnutt JC, Hawryluk GWJ, Gordon W and Wakeland W (2018). Front. Neurol. 9:203.

www.Dynamicsofconcussion.com



Concussion Subtype Development



OREGON HEALTH & SCIENCE UNIVERSITY

GUIDELINES

Concussion Guidelines Step 2: Evidence for Subtype Classification

Angela Lumba-Brown, MD
Masaru Teramoto, PhD, MPH, PStat
G. Josh Bloom, MD, MPH
David Brody, MD, PhD
James Chesnutt, MD
James R. Clugston, MD, MS
Michael Collins, PhD
Gerard Gioia, PhD
Anthony Kontos, PhD
Avtar Lal, PhD
Allen Sills, MD
Jamshid Ghajar, MD, PhD

Department of Emergency Medicine, Brain Performance Center, Stanford University, Stanford, California; Division of Physical Medicine & Rehabilitation, University of Utah, Salt Lake City, Utah;
(Continued on next page)

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Received, February 14, 2019.
Accepted, June 23, 2019.

BACKGROUND: Concussion is a heterogeneous mild traumatic brain injury (mTBI) characterized by a variety of symptoms, clinical presentations, and recovery trajectories. By thematically classifying the most common concussive clinical presentations into concussion subtypes (cognitive, ocular-motor, headache/migraine, vestibular, and anxiety/mood) and associated conditions (cervical strain and sleep disturbance), we derive useful definitions amenable to future targeted treatments.

OBJECTIVE: To use evidence-based methodology to characterize the 5 concussion subtypes and 2 associated conditions and report their prevalence in acute concussion patients as compared to baseline or controls within 3 d of injury.

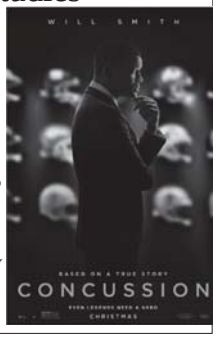
METHODS: A multidisciplinary expert workgroup was established to define the most common concussion subtypes and their associated conditions and select clinical questions related to prevalence and recovery. A literature search was conducted from January 1, 1990 to November 1, 2017. Two experts abstracted study characteristics and results independently for each article selected for inclusion. A third expert adjudicated disagreements. Separate meta-analyses were conducted to do the following: 1) examine the prevalence of each subtype/associated condition in concussion patients using a proportion, 2) assess subtype/associated conditions in concussion compared to baseline/uninjured controls using a prevalence ratio, and 3) compare the differences in symptom scores between concussion subtypes and uninjured/baseline controls using a standardized mean difference (SMD).

RESULTS: The most prevalent concussion subtypes for pediatric and adult populations were headache/migraine (0.52; 95% CI = 0.37, 0.67) and cognitive (0.40; 95% CI = 0.25, 0.55), respectively. In pediatric patients, the prevalence of the vestibular subtype was also high (0.50; 95% CI = 0.40, 0.60). Adult patients were 4.4, 2.9, and 1.7 times more likely to demonstrate cognitive, vestibular, and anxiety/mood subtypes, respectively, as compared with their controls ($P < .05$). Children and adults with concussion showed significantly more cognitive symptoms than their respective controls (SMD = 0.66 and 0.24; $P < .001$). Furthermore, ocular-motor in adult patients (SMD = 0.72; $P < .001$) and vestibular

Neurosurgery 0:1–12, 2019

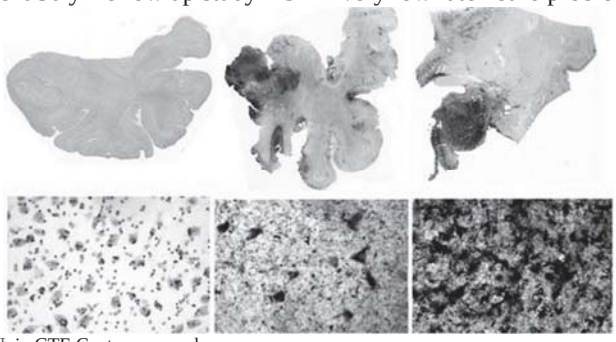
Concussion in the Media

- Baseline neuropsych testing of Pro Athletes in NFL, NHL, Baseball and other sports
- Higher incidence (3X) of depression if >3 concussions in Pro athletes in some studies
- Risk of premature dementia
- Possible brain damage
  - Brain lesion : *tau protein* deposition
  - Similarities to Alzheimer's/Parkinson's
  - Higher risk if certain genes (APO E -4)
  - CTE chronic traumatic encephalopathy



CTE- Tau deposition

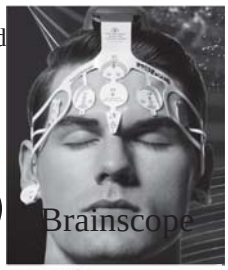
perivascular and frontal/temporal sulci
assoc with repetitive TBI but no direct cause
Recent article suggests possible very high rate in FB??
JAMA. 2017;318:360–370.
Recent 50 yr follow up study HS FB very low rate neuro problems



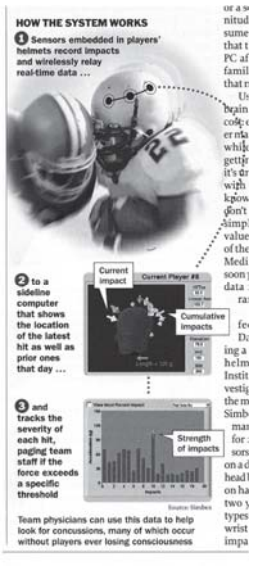
Boston Univ CTE Center normal | Tau tangles | Tau plaques

NY Time, LA Times, etc, 2/18
FDA approves first blood test that can help diagnose a concussion , what????It actually does not diagnose concussion at all.. Only brain bleeds and structural brain injury

?


Brainscope

e to learn more about the impact indic



AAN 2013 Concussion Guideline

http://www.neurology.org/content/80/24/2250.full.pdf+html

SPECIAL ARTICLE

Summary of evidence-based guideline update: Evaluation and management of concussion in sports

Report of the Guideline Development Subcommittee of the American Academy of Neurology

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ABSTRACT

Objective: To update the 1997 American Academy of Neurology (AAN) practice parameter regarding sports concussion, focusing on 4 questions: 1) What factors increase/decrease concussion risk? 2) What diagnostic tools identify those with concussion and those at increased risk for severe/prolonged early impairments, neurologic catastrophe, or chronic neurobehavioral impairment? 3) What clinical factors identify those at increased risk for severe/prolonged early postconcussion impairments, neurologic catastrophe, recurrent concussions, or chronic neurobehavioral impairment? 4) What interventions enhance recovery, reduce recurrent concussion risk, or diminish long-term sequelae? The complete guideline on which this summary is based is available as an online data supplement to this article.

Methods: We systematically reviewed the literature from 1955 to June 2012 for pertinent evidence. We assessed evidence for quality and synthesized into conclusions using a modified Grading of Recommendations Assessment, Development and Evaluation process. We used a modified Delphi process to develop recommendations.

Results: Specific risk factors can increase or decrease concussion risk. Diagnostic tools to help identify individuals with concussion include graded symptom checklists, the Standardized Assessment of Concussion, neuropsychological assessments, and the Balance Error Scoring System. Ongoing clinical symptoms, concussion history, and younger age identify those at risk for postconcussion impairments. Risk factors for recurrent concussion include history of multiple concussions, particularly within 10 days after initial concussion. Risk factors for chronic neurobehavioral impairment include concussion exposure and APOE ε4 genotype. Data are insufficient to show that any intervention enhances recovery or diminishes long-term sequelae postconcussion. Practice recommendations are presented for preparticipation counseling, management of suspected concussion, and management of diagnosed concussion. *Neurology*® 2013;80:2250–2257

Published 2107 - newest guidelines!!

Consensus statement on concussion in sport—the 5th international conference on concussion in sport held in Berlin, October 2016

Paul McCrory,[1] Willem Meeuwisse,[2] Jiří Dvorak,[3,4] Mark Aubry,[5] Julian Bailes,[6] Steven Broglio,[7] Robert C Cantu,[8] David Cassidy,[9] Ruben J Echemendia,[10,11] Rudy J Castellani,[12] Gavin A Davis,[13,14] Richard Ellenbogen,[15] Carolyn Emery,[16] Lars Engebretsen,[17] Nina Feddermann-Demont,[18,19] Christopher C Giza,[20,21] Kevin M Guskiewicz,[22] Stanley Herring,[23] Grant L Iverson,[24] Karen M Johnston,[25] James Kissick,[26] Jeffrey Kutcher,[27] John J Leddy,[28] David Maddocks,[29] Michael Makdissi,[30,31] Geoff Manley,[32] Michael McCrea,[33] William P Meehan,[34,35] Sinji Nagahiro,[36] Jon Patricios,[37,38] Margot Putukian,[39] Kathryn J Schneider,[40] Allen Sills,[41,42] Charles H Tator,[43,44] Michael Turner,[45] Pieter E Vos[46]

McCrory P, et al. Br J Sports Med 2017

PREAMBLE

The 2017 Concussion in Sport Group (CISG) consensus statement is designed to build on the principles outlined in the previous statements[1–4] and to develop further conceptual understanding of sport-related concussion (SRC) using an expert consensus-based approach. This document is developed articles were screened by the expert panels for the Berlin meeting. The details of the search strategies and findings are included in each of the systematic reviews.

The details of the conference organisation, methodology of the consensus process, question development and selection on expert panellists and

Sport Concussion Assessment Tool (SCAT5)

SCAT5© SPORT CONCUSSION ASSESSMENT TOOL – 5TH EDITION

DEVELOPED BY THE CONCUSSION IN SPORT GROUP

FOR USE BY MEDICAL PROFESSIONALS ONLY

supported by

FIFA

STEP 1: RED FLAGS

RED FLAGS:

- Neck pain or tenderness
- Double vision
- Weakness or tingling/ burning in arms or legs
- Severe or increasing headache
- Seizure or convulsion
- Loss of consciousness
- Deteriorating conscious state
- Vomiting
- Increasingly restless, agitated or combative

Sideline Evaluation

STEP 2: OBSERVABLE SIGNS

Witnessed ☐ Observed on Video ☐

Lying motionless on the playing surface	Y	N
Balance / gait difficulties / motor incoordination: stumbling, slow / laboured movements	Y	N
Disorientation or confusion, or an inability to respond appropriately to questions	Y	N
Blank or vacant look	Y	N
Facial injury after head trauma	Y	N

STEP 3: MEMORY ASSESSMENT MADDOCKS QUESTIONS[2]

"I am going to ask you a few questions, please listen carefully and give your best effort. First, tell me what happened?"

Mark Y for correct answer / N for incorrect

What venue are we at today?	Y	N
Which half is it now?	Y	N
Who scored last in this match?	Y	N
What team did you play last week / game?	Y	N
Did your team win the last game?	Y	N

Note: Appropriate sport-specific questions may be substituted.

Also GCS, 13-15, neck exam

2

STEP 2: SYMPTOM EVALUATION

Please Check: ☐ Baseline ☐ Post-Injury

Please hand the form to the athlete

	none	mild	moderate	severe
Headache	0	1	2	3	4	5	6
"Pressure in head"	0	1	2	3	4	5	6
Neck Pain	0	1	2	3	4	5	6
Nausea or vomiting	0	1	2	3	4	5	6
Dizziness	0	1	2	3	4	5	6
Blurred vision	0	1	2	3	4	5	6
Balance problems	0	1	2	3	4	5	6
Sensitivity to light	0	1	2	3	4	5	6
Sensitivity to noise	0	1	2	3	4	5	6
Feeling slowed down	0	1	2	3	4	5	6
Feeling like "in a fog"	0	1	2	3	4	5	6
"Don't feel right"	0	1	2	3	4	5	6
Difficulty concentrating	0	1	2	3	4	5	6
Difficulty remembering	0	1	2	3	4	5	6
Fatigue or low energy	0	1	2	3	4	5	6
Confusion	0	1	2	3	4	5	6
Drowsiness	0	1	2	3	4	5	6
More emotional	0	1	2	3	4	5	6
Irritability	0	1	2	3	4	5	6
Sadness	0	1	2	3	4	5	6
Nervous or Anxious	0	1	2	3	4	5	6
Trouble falling asleep (if applicable)	0	1	2	3	4	5	6
Total number of symptoms:				of 22
Symptom severity score:				of 132
Do your symptoms get worse with physical activity?				Y N
Do your symptoms get worse with mental activity?				Y N

If 100% is feeling perfectly normal, what percent of normal do you feel?

If not 100%, why?

COGNITIVE & PHYSICAL EVALUATION

Cognitive assessment

Standardized Assessment of Concussion (SAC)*

Orientation (1 point for each correct answer)

What month is it?	0	1
What is the date today?	0	1
What is the day of the week?	0	1
What year is it?	0	1
What time is it right now? (within 1 hour)	0	1
Orientation score		of 5

Immediate memory

List	Trial 1	Trial 2	Trial 3	Alternative word list		
elbow	0 1	0 1	0 1	candle	baby	finger
apple	0 1	0 1	0 1	paper	monkey	penny
carpet	0 1	0 1	0 1	sugar	perfume	blanket
saddle	0 1	0 1	0 1	sandwich	sunset	lemon
bubble	0 1	0 1	0 1	wagon	iron	insect
Total						

Immediate memory score total of 15

Concentration: **Digits Backward**

List	Trial 1	Alternative digit list		
4-9-3	0 1	6-2-9	5-2-6	4-1-5
3-8-1-4	0 1	3-2-7-9	1-7-9-5	4-9-6-8
6-2-9-7-1	0 1	1-5-2-8-6	3-8-5-2-7	6-1-8-4-3
7-1-8-4-6-2	0 1	5-3-9-1-4-8	8-3-1-9-6-4	7-2-4-8-5-6
Total of 4				

Concentration: **Month in Reverse Order** (1 pt. for entire sequence correct)

Dec-Nov-Oct-Sept-Aug-Jul-Jun-May-Apr-Mar-Feb-Jan	0	1
Concentration score		of 5

ImPACT© Clinical Report

Mark

Exam Type	Baseline	Post-concussion	Post-concussion	Post-concussion	Post-concussion	Post-concussion
Date Tested	09/21/2004	10/08/2004	10/12/2004	10/15/2004	10/19/2004	10/27/2004
Last Concussion		10/07/2004	10/07/2004	10/07/2004	10/07/2004	10/07/2004
Exam Language	English	English	English	English	English	English
Test Version	2.2.729	2.2.729	2.2.729	2.2.729	2.2.729	2.2.729
Composite Scores *						
Memory composite (verbal)	93 75%	**66** 1%	**57** <1%	**63** <1%	87 55%	88 55%
Memory composite (visual)†	70 23%	**41** <1%	**49** 1%	**47** <1%	**55** 3%	66 12%
Visual motor speed composite	45.88 85%	46.38 86%	**40.13** 65%	**38.93** 57%	45.85 85%	41.90 72%
Reaction time composite	0.54 46%	0.60 22%	**0.66** 6%	0.54 46%	**0.62** 15%	0.54 46%
Impulse control composite	8	14	10	16	10	11
Total Symptom Score	0	**14**	3	1	0	0

* Scores in **bold** type indicate scores that exceed the Reliable Change Index score (RCI) when compared to the baseline score. However, scores that do not exceed the RCI index may still be clinically significant. Percentile scores, if available, are listed in small type. Please consult your ImPACT User Manual for more details.

The Canadian Head CT rule

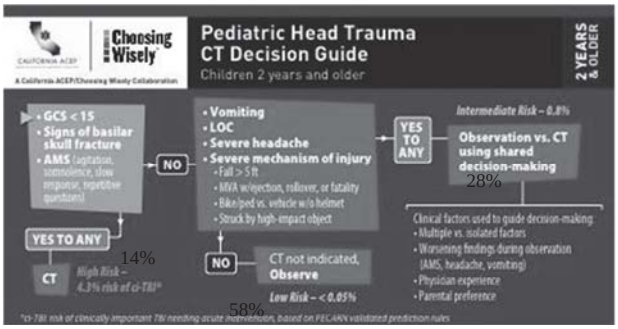
100% sensitive abnormalities that need neurosurgery.

Major Criteria:

- 1.)GCS < 15 at 2 hours post-injury; 2.)Suspected open or depressed skull fracture; 3.) Any sign of basilar skull fracture; 4.) Blood in the middle ear(hemotympanum), or around eyes (raccoon eyes) or back of head(Battle's sign) or cerebral spinal fluid drainage from the nose/ ears); 5.)vomiting (≥ 2 episodes); 6.) Age ≥ 65.

Minor Criteria:

- 1.)Retrograde Amnesia to the Event ≥ 30 minutes; 2.)"Dangerous" Mechanism; 3.)Pedestrian struck by motor vehicle; 4.)Occupant ejected from motor vehicle; 4.)Fall from > 3 feet or > 5 stairs. (Stiell et al. 2001).



PEDIATRIC EMERGENCY CARE APPLIED RESEARCH NETWORK

PECARN CT rule

PECARN CT Rule

Pediatric Emergency Care Applied Research Network

- GCS <15
- Signs of basilar skull fx
- AMS-agitation, somnolence, slow response,repetive questions

YES- 14% positive

Needs CT scan

High risk 4.3% of clnically signif TBI needing acute intervention

NO->

- Vomiting
- LOC
- Severe Headache
- Severe mechanism of injury- fall> 5 ft, MVA ejection, rollover, fatality, high speed, bike crash no helmet

NO- 58%

CT not Needed

Observe

Low risk <0.05%

YES to any-> 28%

Observe vs CT

Intermediate risk 0.8%

Depends on worsening, doctor experience, family concern

Brain Imaging in Acute TBI

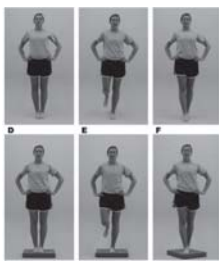
- The decision rule-predicting positive CT
  - 100% sensitive (picks up all problems)
  - 46% specific (picks up more unrelated problems)
  - 13.8 % positive if meet criteria
  - All negative if no criteria

Balance screening after concussion

- Balance assessment recommendations in 2009
- Now required for NCAA sports

OREGON HEALTH & SCIENCE UNIVERSITY

- BESS (modified) –balance error scoring system
- count errors - can be variable
- Sensitivity 34%–64% to detect mTBI
- Balance reportedly resolves after 3-5 days
  But may be due to insensitive testing



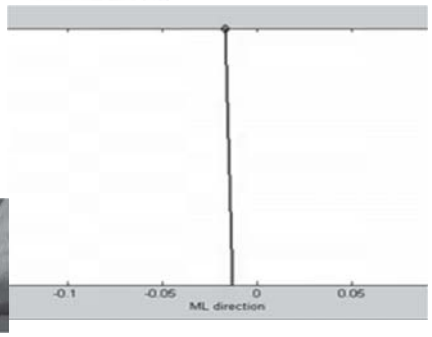
**Need better balance tests- goal to find more sensitive test that can be easily done

Many investigators trying objectify balance better.

(Zurich 2008; McCrory 2009, Guskiewicz 2001, Finnoff 2009, King 2013, Giza 2013)

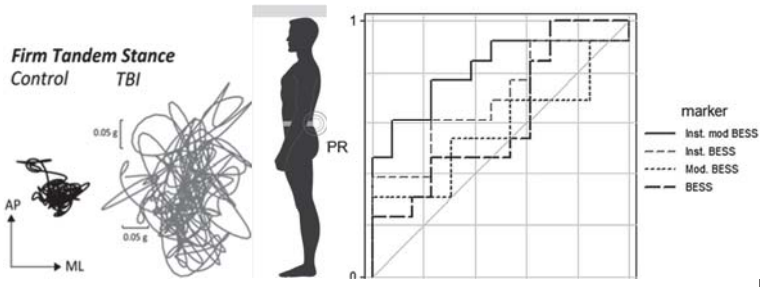
Instrument the BESS using an inertial sensor

New portable, wearable and wireless technology

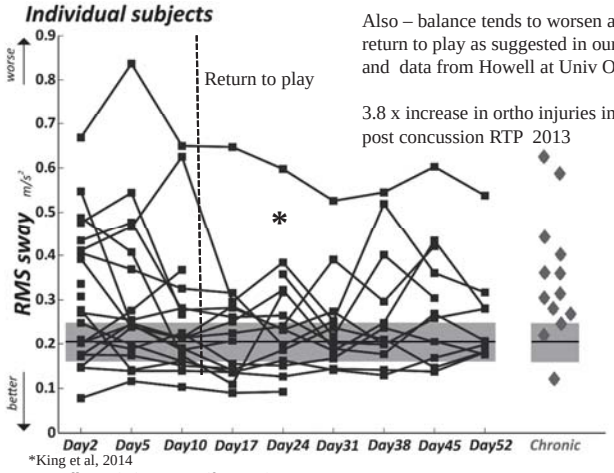


Inertial sensors:
similar to force plate
-Portable
-Automatic analysis

Instrumenting the BESS could best classify chronic concussion vs healthy



King LA, Horak FB, Mancini, Pierce D, Priest KC, Chesnutt JC, Sullivan P, Chapman JC. Instrumenting the Balance Error Scoring System for use with patients reporting persistent balance problems after mild traumatic brain injury. Arch Phys Med Rehabil. 2013 Nov 4. (Mancini et al., 2012, King et al 2013)



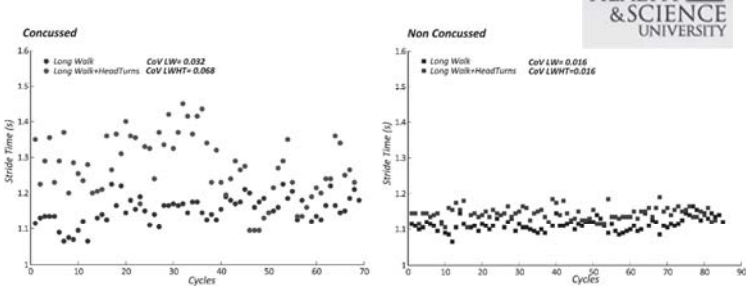
Also – balance tends to worsen after return to play as suggested in our data and data from Howell at Univ Oregon*

3.8 x increase in ortho injuries in 90 days post concussion RTP 2013

*King et al, 2014
*Howell D. MS, Osternig L. Chou L. 2015 .

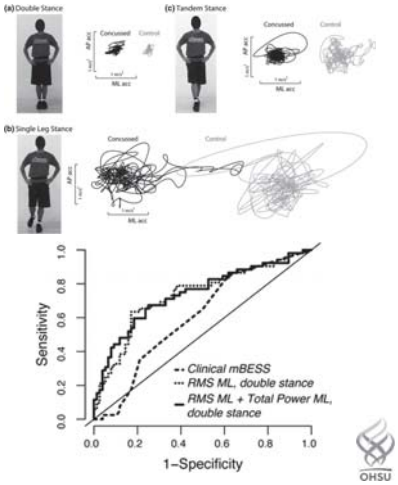
Dynamic balance during walking

Gait variability larger after concussion
worse with dual task



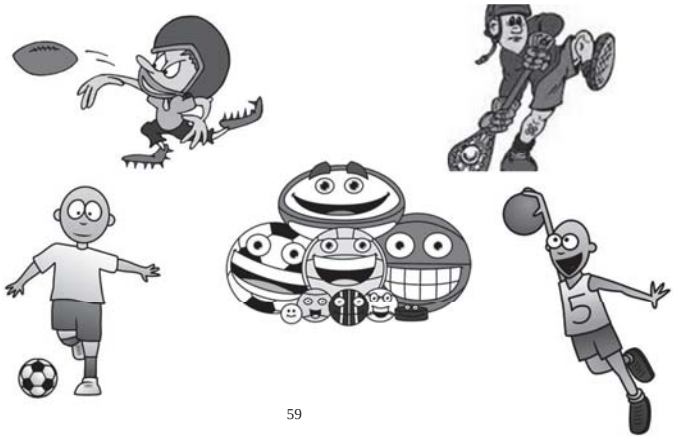
Static balance: take home

Sensor with double limb stance on firm: best at diagnosing concussion and tracking recovery



King LA et al; Ann Biomed Eng 2017

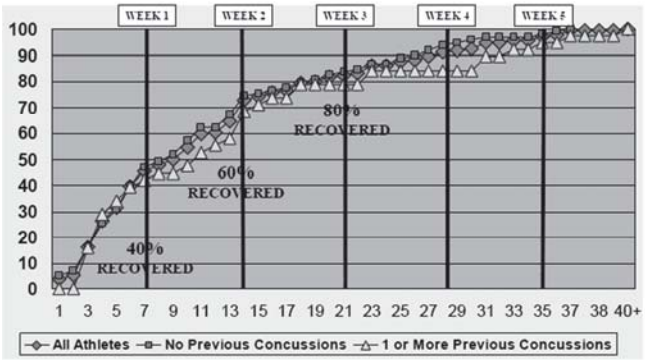
Active Management of Concussions: Return to Learn and Play



The Goal of Appropriate Treatment

- Minimize the duration of symptoms
- Return to play as soon as safely possible
- Avoid entirely the risk of second impact syndrome
- Minimize the rate of chronic post concussion syndrome

Concussion recovery: How long



n = 134 male football athletes

Collins et al; Neurosurgery 2006

Concussion prognostic Factors suggesting slower recovery

- Athlete pre-injury characteristics:
  - Previous concussions
  - Migraine (personal and family)
  - Vestibular or occular issues
  - ADD or learning issues
  - Genetics (apoE 4)
  - Age/ gender

- Am J Sports Med 399110:2311-2318

Concussion Prognostic Factors suggesting slower recovery

- Post concussion symptoms:
  - Early dizziness/ imbalance(7x risk >21 days)
  - Nausea and Vomiting
  - Diff concentrating and fogginess
  - Photo/ phonosensitivity

  - Early intervention seems to impact recovery

  Am J Sports Med 399;110:2311-2318

"Rest or not to rest?"

- Recognize role of relative rest: avoid "cocoon" or "black box" theories
- "best evidence suggests complete rest exceeding 3 days is probably not helpful"
  - Silverberg. J Head Trauma Rehab. 2013
- Encourage return to some activity
  - ↓ mood disorders/social isolation
- Avoiding contact sports
  - Decreased reaction speed
  - Brain more vulnerable to injury , 2nd impact

Exercise and Concussion recovery

- Post concussion treadmill tests in first week are safe per Dr Leddy study
- Exercise tolerance associated with successful RTP- Leddy
- Improved aerobic fitness associated with fewer headaches, esp migraine

Aerobic Exercise: Buffalo Concussion Treadmill Test (BCTT) (Modified Balke protocol)

Provocative exercise test: help to determine if ready for RTP

- Protocol: Measure BP, HR and RPE
  - Treadmill: 3.6 mph, 0.0% incline
  - Minute 2: 3.6 mph, 1.0% incline
  - Minute 3: 3.6 mph, 2.0% incline
  - Minute 4: 3.6 mph, 3.0% incline
  - Keep going: ↑ 1.0% incline every min until:
    - Symptomatic (≥ 3 points)
    - Exhaustion (Borg 19/20 or 85% of age-predicted max HR)
- High inter-rater reliability (95%) and sensitivity (99%)

Leddy JJ et al 2013
Baker JG et al 2012

Vestibular Rehabilitation with Concussion is effective

Recommended by 5th Consensus statement on concussion in sport

Vestibular therapy + cervical PT vs sham

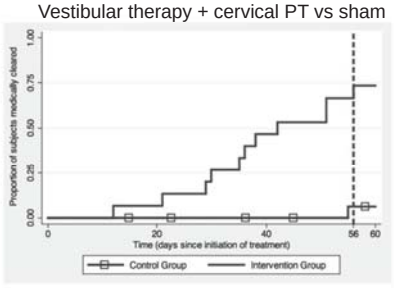
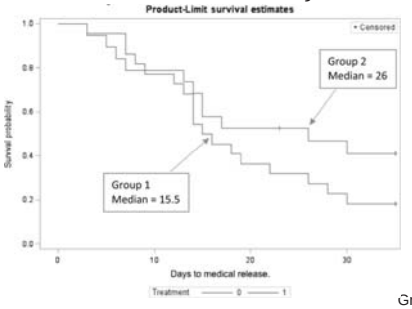


Figure 1 Proportion of patients medically cleared over time.

Schneider KJ et al; Br J Sports Med 2014
Alsalaheen BA et al; J Neurol Phys Ther. 2010
McCroy P, Meeuwisse W, Dvorak J et al; Br J Sports Med 2017

Early Physical Therapy in concussion is safe

- **PT:** 10 days post injury
  - Safe
  - Shortened recovery



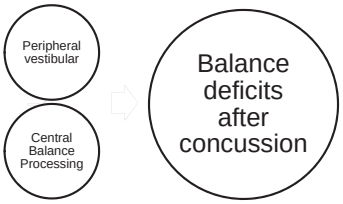
- **Return to activity in kids within** 7 **days:** ↓ symptoms vs no activity
- **Aerobic exercise**
  - Buffalo protocol (treadmill) 1-9 days post injury **did not harm** patients

Grool AM et al; JAMA 2016
Reneker JC et al; Scand J Med Sci Sports 2017
Leddy et al; Clin J Sports Med 2018

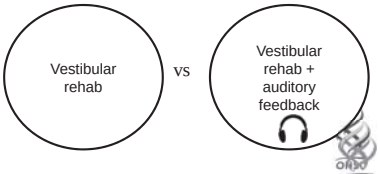
Assessment and Rehabilitation of Central Sensory Impairments for Balance in mTBI

PI: Laurie A King, PT, Ph.D. DOD Award: $2 million

Study Aim 1: To characterize the differences in sensory weighting between people with and without chronic mTBI



Study aim 2: To evaluate whether sensory weighting changes with rehabilitation



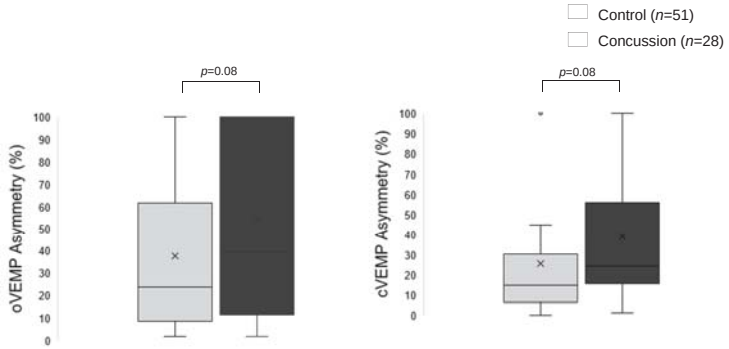
Fino PC et al; BMC Neurol 2017

Vestibular rehabilitation program – progressing in difficulty over 6 weeks

	Eyes Open				Eyes Closed			
	Feet Together (DS), Firm							
Static	Standing Still	Tossing Ball	Rotating Head (L/ R) Smooth Pursuit Gaze Stabilization Saccades	Bobbing Head (U/ D) Smooth Pursuit Gaze Stabilization Saccades	Standing Still	Rotating Head (L/ R)	Bobbing Head (U/ D)	
	Feet Together (DS), Foam							
	Standing Still	Tossing Ball	Rotating Head (L/ R)	Bobbing Head (U/D)	Standing Still	Rotating Head (L/ R)	Bobbing Head (U/D)	
Dynamic	**Tandem Gait, Firm**							
	Walking	Tossing Ball	Rotating Head (L/ R)	Bobbing Head (U/D)	Walking			
	Tandem Gait, Foam							
	Walking	Tossing Ball	Rotating Head (L/ R)	Bobbing Head (U/D)	Walking			
Bending	Chair	Side of Treadmill	Floor		Chair	Side of Treadmill	Floor	
Squatting	**Squat Firm**							
	Sit to stand (mini squat)	Lunge	Lunge onto unstable surface	Lunge + Twist	Sit to stand (mini squat)	Lunge	Lunge onto unstable surface	Lunge + Twist
	Squat Foam							
	Sit to stand (mini squat)	Lunge	Lunge onto unstable surface	Lunge + Twist	Sit to stand (mini squat)	Lunge	Lunge onto unstable surface	Lunge + Twist

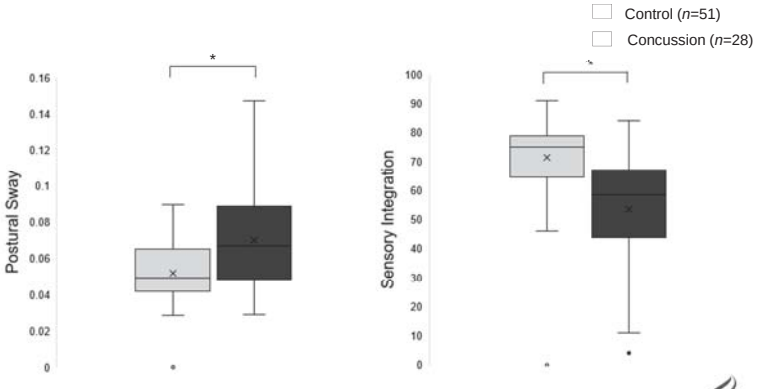
(Fino, et al., 2017)

Results: No abnormal otolith function



- No difference in the amplitude of cVEMP or oVEMP (p>0.05)

Results: Abnormal central control of balance post concussion

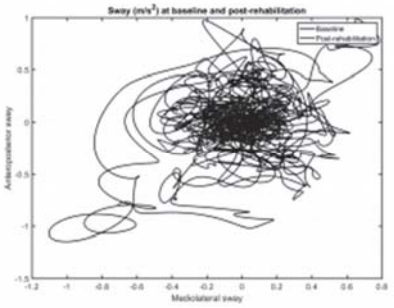


mBESS: Feet together, EC + Foam

Composite score of SOT

Preliminary results:

Auditory biofeedback may be useful to augment real-time sensory feedback for balance



Take homes

*Take home message

- Chronic mTBI:
  - Central dysfunction of vestibular system
  - No peripheral otolith dysfunction
- People with chronic mTBI improved!
  - Repetition/frequency higher in research protocol
- Outcome measures:
  - SCAT symptom checklist
  - SOT/CSMI

Rehabilitation of Complex TBI with Sensory Integration Balance Deficits: Can Early Initiation of Rehabilitation with Wearable Sensor Technology Improve Outcomes?

Log #PT160104
Award # W81XWH-17-1-0424

PI: Laurie A King, PT, Ph.D. **DOD Award:** $4,652,124

Why early rehab?

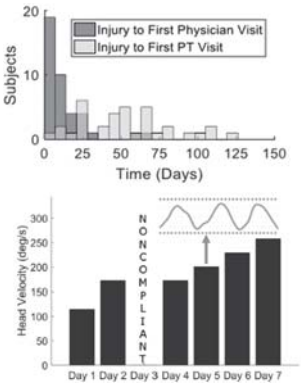
- Waiting can encourage maladaptive strategies
- Early promotes neuroplasticity
- After peripheral vestibular disorder: improved outcomes when treated early vs late
- Negative outcomes: hours post injury (not days) based on animal models

- **Early is the new Normal:**
  - **Early** mobilization in the ICU on mechanical ventilation improves LOS
  - **Early** (2 d) vs Delayed (9 d) rehab for muscle injury : faster RTP
  - Current model for neurological rehabilitation

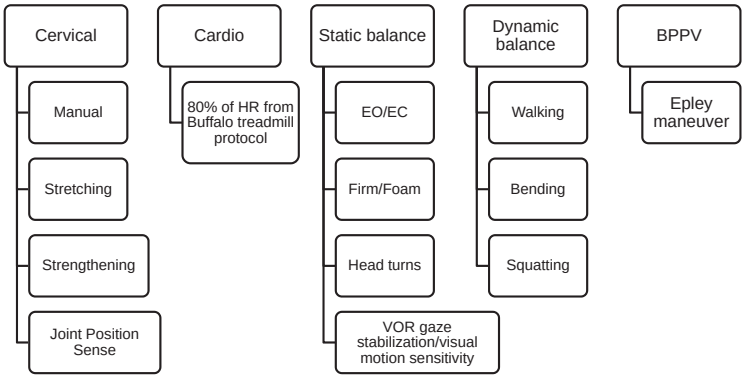
Bamiou DB et al Scand Audio 2000; Shen J et al; Brain Research 2016
Hashem MD et al; Respir Care 2016; Bayer et al; N Engl J Med 2017

Study Aims

1. Does early rehab improve outcomes vs standard care?
2. Are patients doing their home program and are they doing it correctly?
3. Develop technology for PTs to get real time feedback on head/trunk motion



Protocol



Early Concussion Intervention: Study Overview

Population	Intervention	Comparison	Outcome
• 18-60 yo • < 12 wks from concussion	• Vestibular x 30 min • Cervical x 15 min • Cardiovascular (sub threshold) x 15 min	• "Early" vs "Standard" care • With and without home monitoring using sensors	• Buffalo treadmill • SCAT2 • VOMS • Instrumented BESS and walking tests • CSMI

Contact Info

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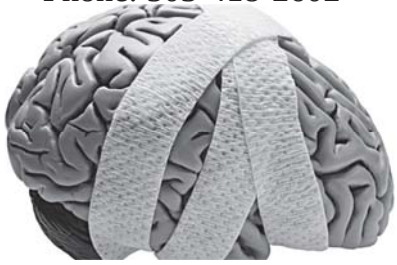
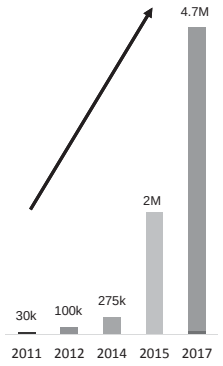
Phone: 503-418-2602



Grants awarded for mTBI and Balance (OHSU: Neurology King Lab)

Grants

- NIH: Center for Translation of Rehabilitation Engineering Advances and Technology (TREAT)
  Title: Wearable Sensor to Detect Postural Instability in People after Concussion
  PI: Laurie King PT, Ph.D
- OCTRI K22
  Title: Quantification of balance deficits after concussion; implications in return to play determination
  PI: Laurie King PT, Ph.D
- NIH R21
  Title: Wearable Sensor to Detect Postural Instability in People after mTBI
  PI: Laurie King PT, Ph.D
- Department of Defense
  Title: Assessment and Rehabilitation of Central Sensory Impairments for Balance in mTBI
  PI: Laurie King PT, Ph.D
- Medical Research Foundation
  Title: Perturbations and mTBI
  PI: Peter Fino PhD
- Department of Defense
  Title: Rehabilitation of Complex TBI with Sensory Integration Balance Deficits; Can Early Initiation of Rehabilitation with Wearable Sensor Technology Improve Outcomes?
  PI: Laurie King PT, Ph.D



Publications

•Fino P.C., Parrington L., Walls M., Sippel E., Hullar T.E., Chesnutt J.C., King L.A. Abnormal turning and its association with self-reported symptoms in chronic mTBI. Journal of Neurotrauma 2017

•Fino PC, Peterka RJ, Hullar TE, Murchison C, Horak FB, Chesnutt JC, King LA. Assessment and rehabilitation of central sensory impairments for balance in mTBI using auditory biofeedback: a randomized clinical trial. BMC Neurology 2017, 17(1):41.

•King, L. A., Mancini, M., Fino, P. C., Chesnutt, J., Swanson, C. W., Markwardt, S., & Chapman, J. C.. Sensor-based balance measures outperform modified balance error scoring system in identifying acute concussion. Annals of Biomedical Engineering 2017 45(9) 2135-2145.

•Haran, FJ, Slaboda, JC, King, LA, Wright, WG, Houlihan, D, & Norris, JN. Sensitivity of the Balance Error Scoring System and the Sensory Organization Test in the Combat Environment. Journal of Neurotrauma, 2015 33(7):705-11

•King LA, Horak FB, Mancini, Pierce D, Priest K, Chesnutt, Chapman J. Instrumenting the balance error scoring system for use with patients reporting persistent balance problems after mild traumatic brain injury. Archives of Physical Medicine and Rehabilitation 2014 95(2):353-9

•Fino P.C., Parrington L., Pitt W., Martini D.N., Chesnutt J., Chou L., King L.A. (Under Review). Detecting Gait Abnormalities After Concussion or Mild Traumatic Brain Injury: A Systematic Review of Single-Task, Dual-Task, and Complex Gait.

•Parrington L, Fino PC, Swanson C, Murchison CF, Chesnutt JC, King LA; Longitudinal assessment of balance and gait following concussion in college athletes; In preparation.

•Parrington L, Fino NF, Fino PC, Murchison CF, Chesnutt JC, King LA. Inflection points in longitudinal models: Tracking recovery and Return to Play following concussion; submitted and under review

•Fino PC, Wilhelm J, Parrington L, Stuart S, Chesnutt JC, King LA. Horizontal head turns after concussion: insight from inertial sensors. Archives of Physical Med and Rehabilitation; In preparation

Balance Disorders Laboratory

Funding: This work was supported by the Assistant Secretary of Defense for Health Affairs under Award No.W81XWH-15-1-0620. Opinions, interpretations, conclusions and recommendations are those of the author and are not necessarily endorsed by the Department of Defense.

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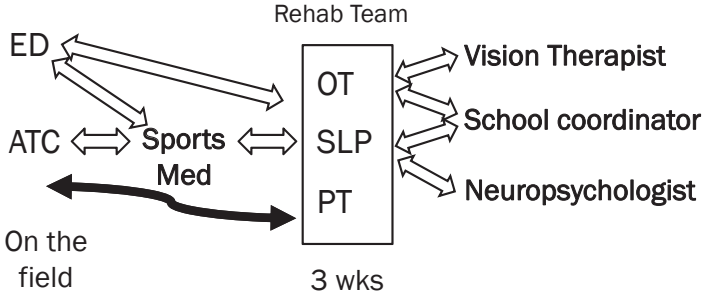
Interdisciplinary approach: OHSU Model

Comprehensive Neurological Rehab Team



OHSU Concussion Rehab Team

A. Speech -language pathologist: for evaluation and treatment to address cognitive and executive function and school and work issues.

B. Physical therapy: for vestibular therapy and neck and associated orthopaedic issues and exercise testing and prescription.

C. Occupational Therapy: for visual and functional therapy , s sensory integration/ overload and driving evaluations.

D. Concussion Coping Clinic and Support Group

This is on the 1st floor of OHSU Center for Health and Healing.
Please call 503-494-3151 to schedule an appointment but this will likely need to be approved by your insurance

Return to Play Protocol

Stage	Aim	Activity	Goal
1	Symptom limited activity	Daily activities that do not increase symptoms (after about 1-2 d rest)	Gradual re-introduction of work/school activities
2	Light aerobic exercise	Walking or stationary bike. No resistance training	Increase in HR <70% max Walk, jog, exercise bike
3	Sport-specific exercise	Running/skating drills; no head impact activities	Add movement
4	Non-contact training drills	Harder training drills (passing drills). Start progressive resistance training	⇧Exercise, coordination and cognitive load add wt lifting, passing, plays
5	Full contact practice	*Requires medical clearance; can participate in normal training activities	Restore confidence and asses functional skills by coaching staff/ ATC
6	Return to sport	Normal unrestricted game play	

McCrory P, et al; Br J Sports Med. 2017

*Start with 24-48 hrs of rest before initiating stage 1
*Minimum 24 hrs per stage

OSAA Concussion Return to Play Form

OSAA Concussion Return to Play Form

Oregon School Activities Association
25200 SW Parkway Avenue, Suite 1
Wilsonville, OR 97070
503.682.6722 FAX 503.682.0960 http://www.osaa.org

CONCUSSION – RETURN TO PARTICIPATION MEDICAL RELEASE

Student Name: _____ **Date of Birth:** ___/___/___ **School/Grade:** _____

Date of Injury: ___ / ___ / ___ **Sport/ Injury Details:** _____

At this time, the student is:
- ☐ symptom-free at rest
- ☐ symptom-free at exertion
- ☐ scoring within a normal range on ImPACT
- ☐ NOT symptom-free at rest
- ☐ NOT symptom-free at exertion
- ☐ NOT scoring within a normal range on ImPACT

When ImPACT is utilized, please either attach or allow access to baseline and post concussive scores with percentiles.

Comments: _____

Completed by (Printed name): _____ Signature: _____ Date: _____

☐ Registered Athletic Trainer ☐ Coach ☐ Athletic Director ☐ Other: _____

Graduated, Step-wise Return-to-Participation Progression

1. **No activity**: Complete rest, both physical and cognitive. This may include staying home from school or limiting school hours and/or homework as activities requiring concentration and attention may worsen symptoms and delay recovery.
2. **Light aerobic exercise**: Walking or stationary bike at low intensity; no weight lifting or resistance training.

Before progressing to the next stage the student must be healthy enough to return to school full time

3. **Sport-specific exercise**: Sprinting, dribbling basketball or soccer; no helmet or equipment, no head impact activities.
4. **Non-contact training**: More complex drills in full equipment. Weight training or resistance training may begin.
5. **Full contact practice**: Participate in normal training activities.
6. **Unrestricted Return-to-Participation/full competition. (Earliest Date of Return-to-Participation**: _____)

The student should spend a minimum of one day at each step. If symptoms re-occur, the student must stop the activity and contact their trainer or other health care professional. Depending upon the specific type and severity of the symptoms, the student may be told to rest for 24 hours and then resume activity one-step below where he or she was when the symptoms occurred. ***Graduated return applies to all activities including sports and PE classes.***

This section to be completed by Physician/Health Care Professional:

☐ Student **may NOT return** to any sport activity until medically cleared.

☐ Student should **remain home from school** to rest and recover with a projected return date _____

☐ Please **allow classroom accommodations,** such as extra time on tests, a quiet room to take tests, and a reduced workload when possible.

Additional Recommendations: _____

☐ Student **may begin graduated return at stage circled above.** If symptom free at rest and with graded exertion, can return to participation on date above.

☐ Student is now **cleared for full contact practice/participation**: symptom free at rest and exertion and has completed a graduated Return-to-Participation protocol.

Physician/Health Care Professional Signature: _____ **Date**:_____

Physician/Health Care Professional Name/Title: _____ **Phone**:_____

Per OAR 581-022-0421 "Health Care Professional" means a Physician (MD), Physician's Assistant (PA), Doctor of Osteopathic (DO) licensed by the Oregon State Board of Medicine, nurse practitioner licensed by the Oregon State Board of Nursing, or Psychologist licensed by the Oregon Board of Psychologist Examiners.

Return to Academic Plan

1. **RECOGNIZE:**
   Concussion management team identifies student's concussion and informs teachers

2. **REMOVE/REST**:
   Students remain home for 2 days or more with physical and cognitive rest

3. **REFER** :
   Students suspected of sustaining a concussion must be evaluated and cleared by a properly trained medical professional.

4. **RETURN :**
   Develop return to academic plan with educational accommodations with modified environment and work load. Consider freezing grades early and be flexible with transitions. **Back to school before athletics!**

cbirt

Return to Academics after Concussion

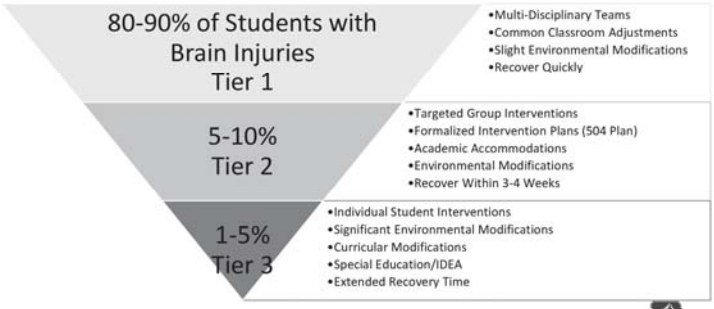
When students have symptoms after a concussion they may need a gradual return to their pre-injury academic load. This progression can speed recovery and support the student's return to a full academic load. Important things to remember:

- The stages are flexible based on the student's tolerance to school activities.
- Depending on symptoms, a student may start at any step and remain at each step as-long-as needed.
- If symptoms worsen, the student should return to the previous step.
- Daily check-ins with the student regarding how they are tolerating school is recommended.
- Depending on symptoms, some students can begin limited physical activity early after injury.

Stage	Suggested Accommodations	Criteria for Progression
Rest Limited mental activity	Limited mental exertion (computer, texting, video games, or homework), no driving.	30 minutes of mental exertion without symptom exacerbation
Part-time school with accommodations	Accommodations based on symptoms (e.g., shortened day/schedule, built-in breaks, no significant classroom or standardized testing)	Full day of school with accommodations
Full-time school with accommodations	Accommodations based on symptoms (e.g., shortened day/schedule, built-in breaks, no significant classroom or standardized testing)	Handles all class periods in succession without symptom increase.
Full pre-injury academic load	Complete return to pre-injury status	NA

CBIRT- www.osaa.org Return to Learn

A school-wide academic accommodation protocol for students with concussions or brain injuries can be effectively implemented in most schools using the following progression.



OREGON HEALTH & SCIENCE UNIVERSITY

Acute Concussion Evaluation (ACE) Care Plan

Gerard Gioia, PhD[1] & Micky Collins, PhD[2]
[1]Children's National Medical Center
[2]University of Pittsburgh Medical Center

Patient Name_____
DOB: _____ Age:_____
Date:_____ ID/MR#_____
Date of Injury:_____

You have been diagnosed with a concussion (also known as a mild traumatic brain injury). This personal plan is based on your symptoms and is designed to help speed your recovery. Your careful attention to it can also prevent further injury.

Rest is the key. You should not participate in any high risk activities (e.g., sports, physical education (PE), riding a bike, etc.) if you still have any of the symptoms below. It is important to limit activities that require a lot of thinking or concentration (homework, job-related activities), as this can also make your symptoms worse. If you no longer have any symptoms and believe that your concentration and thinking are back to normal, you can slowly and carefully return to your daily activities. Children and teenagers will need help from their parents, teachers, coaches, or athletic trainers to help monitor their recovery and return to activities.

Purpose of Care Plan: Guide recovery , Educate, Manage exertional and school activity

Educational resources: State TBI Teams

Develop concussion education programs and return to academic programs, and assist with 504 plans if needed.

Call : 541-346-0597 or Email: **www.ocamp.org**

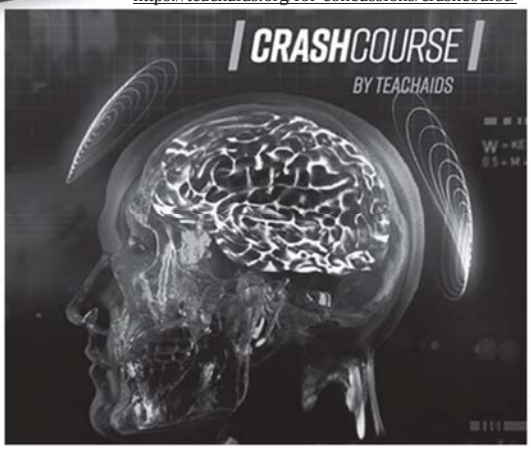
http://brain101.orcasinc.com

The Effectiveness of a Web-Based Resource in Improving Post-concussion Management in High Schools. Glang AE, Koester MC, Chesnutt JC, Gioia GA, McAvoy K, Marshall S, Gau JM, *Journal of Adolescent Health* , Volume 56, Issue 1 , 91 – 97, Jan 2015.

Crash Course- new for concussion education

What is CrashCourse?

https://teachaids.org/for-concussions/crashcourse/



OREGON HEALTH & SCIENCE UNIVERSITY

USA FOOTBALL

OREGON SCHOOL ACTIVITIES ASSOCIATION OSAA SINCE 1918

HEADS UP

USA FOOTBALL- Heads Up program

Video on OSAA website- www.osaa.org

- OSAA- Concussion guidelines and return to learn info from CBIRT/ OCAMP
- www.osaa.org/health-safety

CDC Concussion Tool Kit

- Videos and Educational/ media programs

www.cdc.gov/ncipc/tbi/Coaches_Tool_Kit.htm

Summary

- Must improve early identification & diagnosis
  - Coach, athlete, parent, Correction officer, medical education
- Careful individualized clinical assessment and tracking from time of injury
  - SCAT5
  - Neuropsychological Testing (Impact, Axon, or full)
- Interdisciplinary Team- OT, PT, SLP
- Implement active treatment in home & school
  school accommodations, 504 plan, OCAMP.org
  Max's Law Implementation Guide- download
- Free coaches education: You too can take this…
  www.osaa.org/healthandsafety/concussion.asp

OHSU Concussion Team

Sports Medicine Doctors

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Sean Robinson, M.D.
Carol Federiuk, M.D.
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Rachel Cohen, PT, DPT
Carly Lochala, PT, DPT
Kristin Moore, PT, DPT
Bill Rubine, MSPT
Jeff Schlimgen, MSPT, NCS
Marvin Smith, PT, DPT
.Kate Scanlan, PT, DPT, NCS
Amy Woods, PT, DPT
Margaret McReynolds, PT, M.S.ED

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Family Medicine
Neurology Peds/ Adult
Anesthesia/ Pain Center
Neurosurgery
Psychiatry
Trauma
ENT/ vestibular
Ophthalmology
Emergency Med
Internal Medicine
Pediatrics
Radiology

Neuro-psychology

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Muriel Lezak, PhD
Leeza Maron, PhD
Bonnie Nagel, PhD
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James Carson, PhD
Tyler Duffield, PhD

Occupatioanl / Visual Therapy

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Anne-Marie Banasky, OTR-L, CHT
Kelly Dunbar, OTR-L, CHT
Kyla Laraway, OTR-L, OTD

Speech/ Cognitive Therapy

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Kristin Knight, MS, CCC-SLP

Certified Athletic Trainers

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OHSU

Thank You

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2019 OHSU TBI Symposium: Dec 13-14

What Was That Pop? Acute Knee Injuries

Andrea Herzka, MD
September 6, 2018

Disclosure

- none

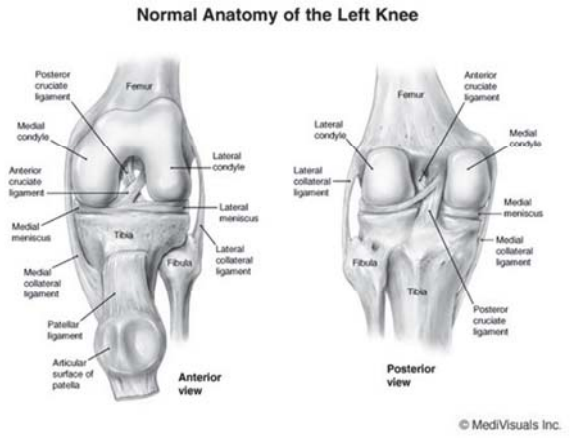
Objective: Build confidence in managing the patient with acute knee injury

- Integrate the history, physical exam and radiographs to sharpen a differential diagnosis
- Build confidence in the nuances of the physical exam: Recognize presentation

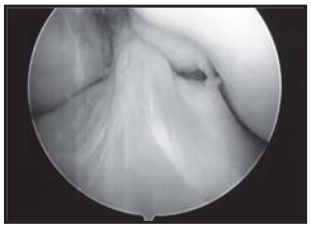
Agenda

- Review Knee Anatomy
- Creating a Differential
- Clues in the History
- Physical Exam Review
- MRI findings
- When to Refer to Orthopaedic Surgery

Knee Anatomy



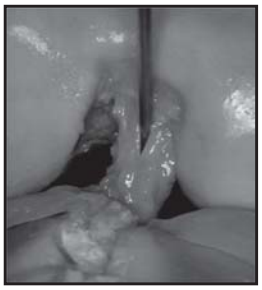
ACL



ACL

Prevents Anterior Translation of the Tibia

PCL



PCL
Prevents Posterior tibial translation

MCL and FCL

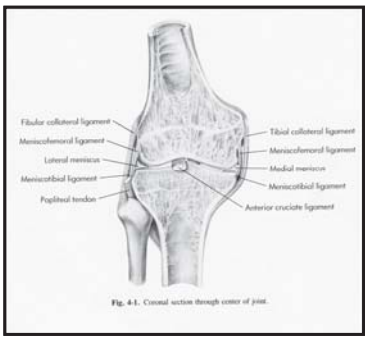
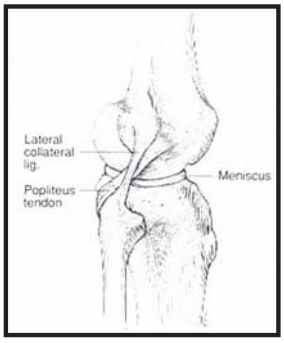


Fig. 4-1. Coronal section through center of joint.



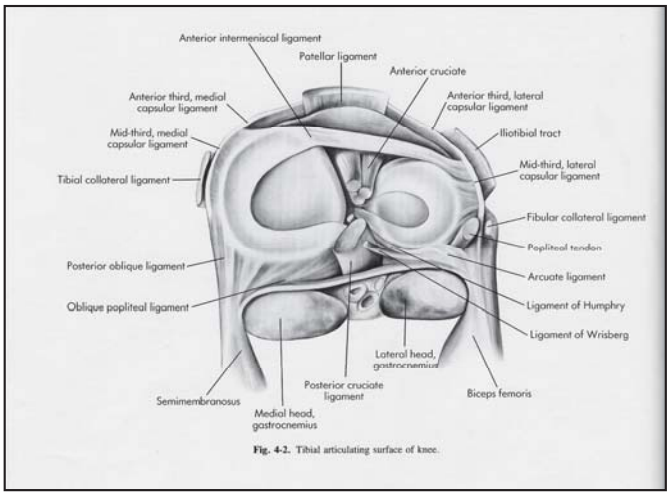
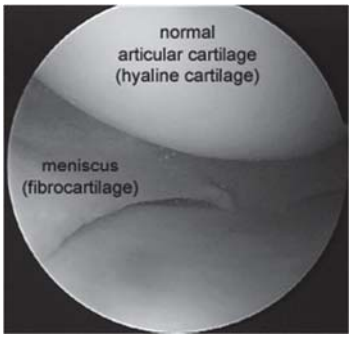
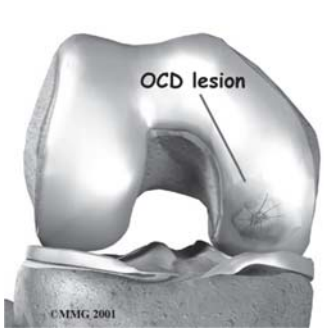


Fig. 4-2. Tibial articulating surface of knee.

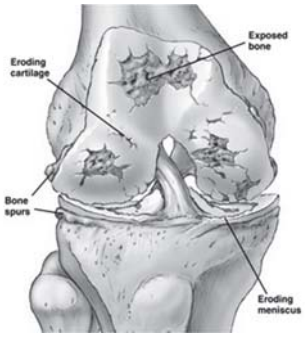
Types Of Cartilage



Articular Cartilage Problems

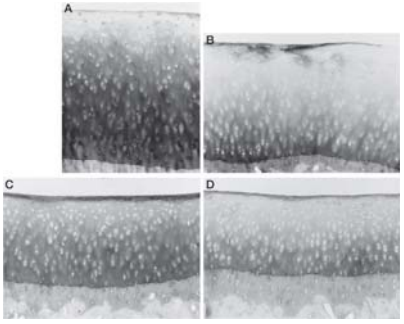


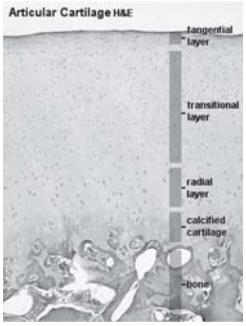
Focal Injury



Diffuse Injury

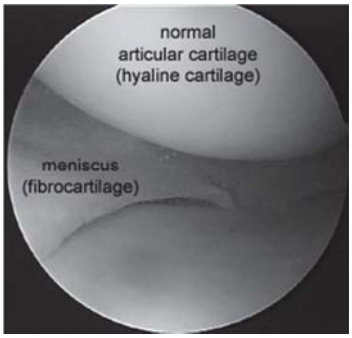
Articular Cartilage: Hyaline Cartilage / "Teflon Coat"



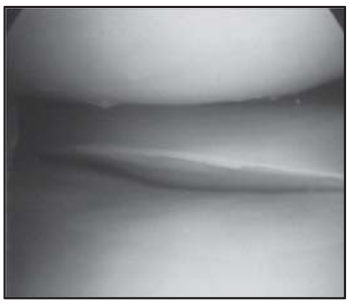


Fibrocartilage = Meniscus: "Shock Absorber"

Crescent shaped
Medial = C-shaped
Lateral = Semicircular

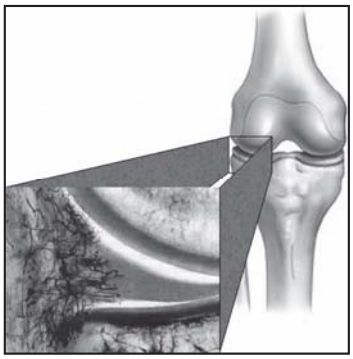


KNEE ANATOMY : Meniscus



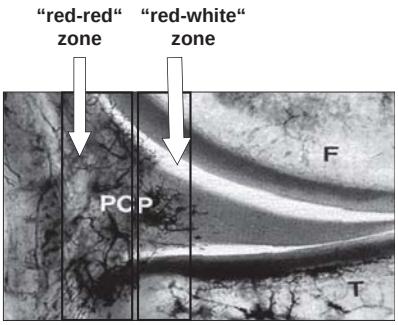
- Deepens tibial surface
- 2º stabilizer

Meniscus= Fibrocartilage: "Shock Absorber"



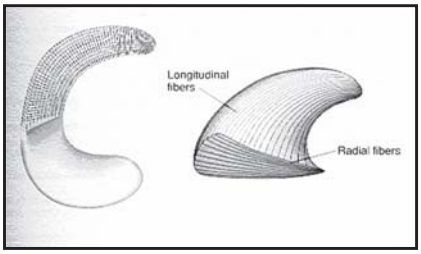
Vasculature

- Perimeniscal capillary plexus (med and lat genic) provides blood supply to the periph. 1/3 of the meniscus



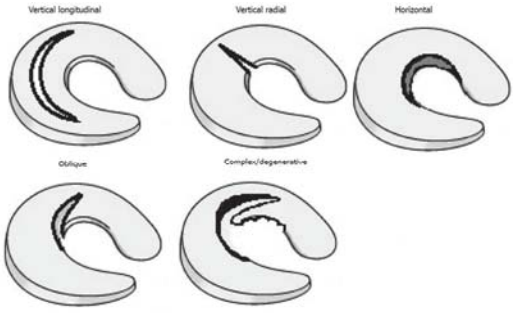
Photos by McGinty: Operative Arthroscopy (1991) (from Arnoczky and Warren)

Meniscus



Longitudinal & Radial fibers
Dissipate hoop stress
Expand under compressive force
Type I collagen

Radial Split Tear: Poor Prognosis
Root Avulsion: Difficult
Longitudinal Tear: ? Zone



MENISCUS: LOAD TRANSMISSION

- 50% joint load transmitted in full ext.
- 85% joint load transmitted in 90º flex.
- 15-34% (small) partial meniscectomy increases contact pressure to articular cartilage dramatically

"POP": Differential Diagnosis

- Ligament Tear
  - ACL
  - MCL
  - PCL
- Patellar Dislocation
- Meniscus Tear/ Avulsion
- Cartilage Flap Tear
- Patellar/ Quad Tendon Tear
- Ruptured Baker's Cyst
- Fracture

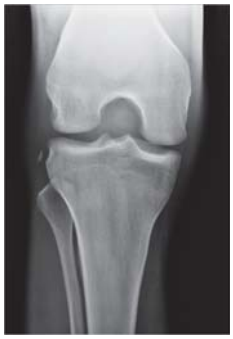
Acute Injury

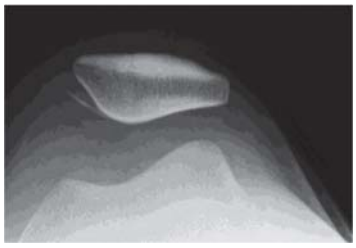
- Tried to cut or stop abruptly and felt a pop
  - ACL tear
  - Patellar Dislocation
  - Meniscus
  - Displaced Osteochondral Injury
- Landed on my Anterior Shin/ Knee felt a pop
  - PCL
  - Patella Fracture

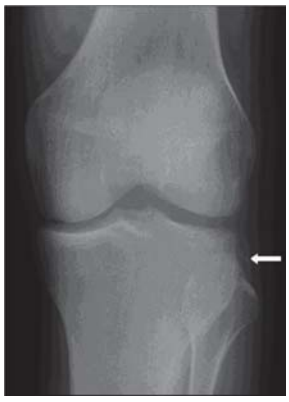
Acute Injury

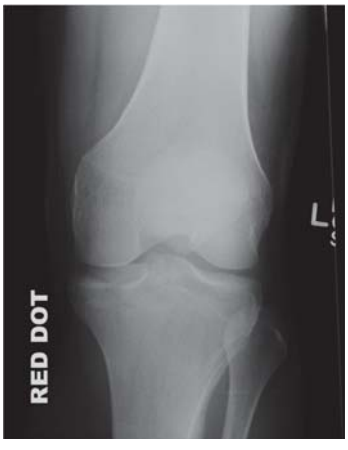
- My knee was bent while wrestling and I felt a pop
  - Meniscus tear
- I felt a sharp pain in the back of my knee and my calf
  - Ruptured Baker's Cyst
- Felt a pop and now even just the weight of a sheet at night on my inner knee causes severe pain
  - Inferiorly displaced medial meniscal fragment
- My kneecap popped out and I had to shove it back in

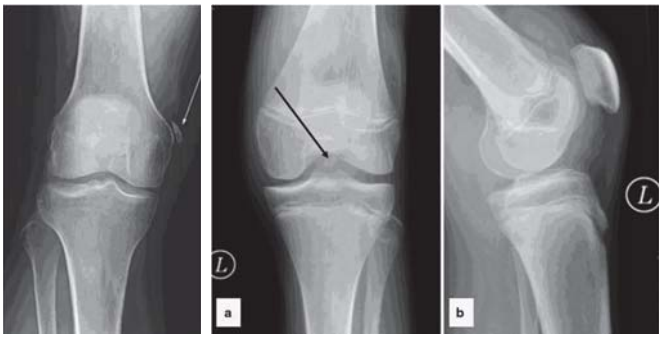
Acute Xrays











Physical Exam

EFFUSION: trace, small, moderate, large, tense
SKIN:

WEIGHTBEARING
RIGHT: Alignment
LEFT: Alignment
(varus= bowlegged; valgus= knock-kneed)

FEET: Arch height (pronation vs supination)
SQUAT: Pain generated? Where?
GAIT: Antalgic, flexed knee
ACTIVE ROM: Right: ***, Left: ***

PATELLA:		
	RIGHT	LEFT
NORMAL		
TILT	Neutral	Neutral
GLIDE	2 quadrants	2 quadrants
CREPITATION	None	None
J SIGN	Absent	Absent
TENDERNESS	None	None
APPREHENSION	Neg	Neg
LIGAMENTS:		
	RIGHT	LEFT
VALGUS AT 30˚	Neg	Neg
VARUS AT 30˚	Neg	Neg
LACHMAN	0	0
ANT. DRAWER	Firm endpoint, symmetric	Firm endpoint, symmetric
PIVOT SHIFT	Absent	Absent
POST. DRAWER	Negative	Negative
PRONE DIAL 30˚	Symmetric	Symmetric
PRONE DIAL 90˚	Symmetric	Symmetric

PERIARTICULAR TENDERNESS:

	RIGHT	LEFT
JLT MEDIAL	Neg	Neg
JLT LATERAL	Neg	Neg
MCMURRAY	Neg	Neg
WILSON	Neg	Neg
PAT TENDON	Neg	Neg

NVS: Intact

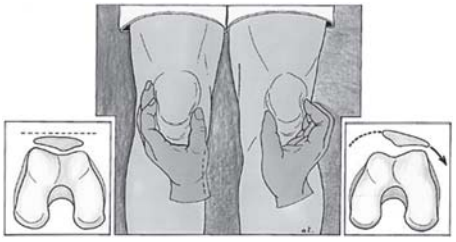
Ober Sign: ? Tight IT band

Inspection

- Effusion
- Alignment
- Gait (if able)
  - Antalgic
  - Flexed knee
    - ?DDX

Patella

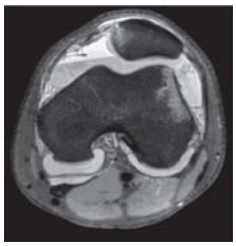
- **Apprehension**
- **Glide/ translation**
- **Tilt**

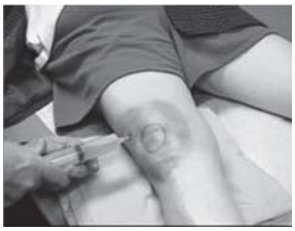


Patellar Translation/ Apprehension

Patellar Dislocation: Initial Treatment

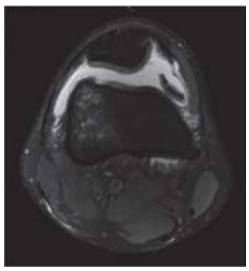
- Aspirate Hemarthrosis
  - (look for fat droplets)
- Use small rolled kerlix to make a lateral patellar bumper and ace wrap for

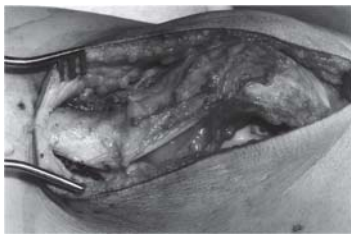


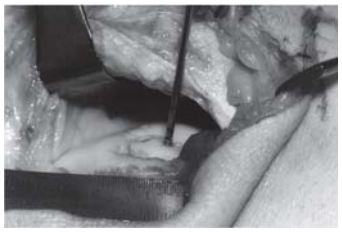


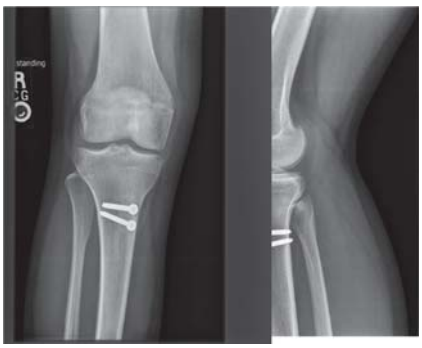
Patellar Dislocation: Treatment

- Conservative if traumatic etiology normal anatomy and no osteochondral injury
  - PT
  - Short term bracing
- Surgical options
  - Soft tissue procedure
    - MPFL
  - Bony procedure
    - Tibial Tuberosity Osteotomy



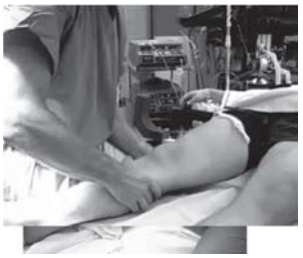


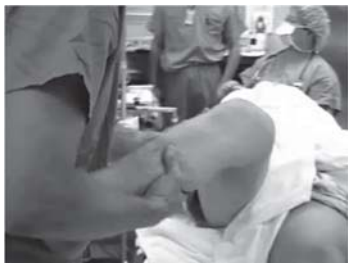




ACL tear

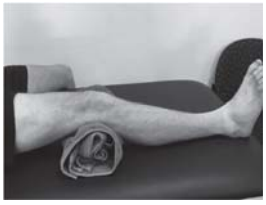
- **LACHMAN**
- **ANT. DRAWER**
- **PIVOT SHIFT**





ACL Presentation

- Pop- then knee felt wobbly
- Gradual swelling
- Flexed knee gait
- Full extension very uncomfortable- feels like deep pinch
- Guards against lachman



Management of Acute ACL Tear

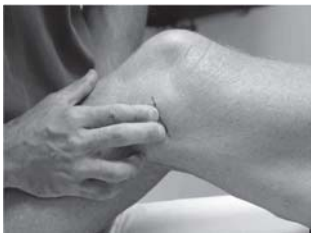
- Order MRI
- Referral to ortho
- Can aspirate knee if large hemarthrosis
- Knee immobilizer or hinged knee brace if poor quad function but encourage ROM
- If MRI shows no displaced meniscus tear OK to WBAT and start ACL “prehab” PT

Definitive Treatment of ACL Tear

- Surgery prevents recurrent giving way and meniscus tears, allows more rigorous sports and cutting
- Surgery does not prevent arthritis and does not change the timeline of post traumatic OA
- There is a small cohort of patients who can do well with conservative treatment

Meniscus pathology

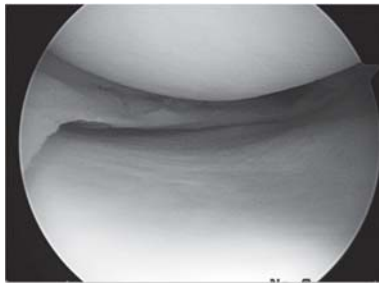
- Jointline tenderness
- McMurray
- Appley



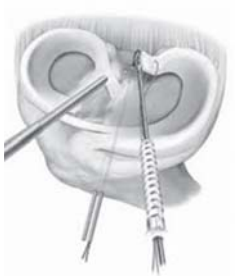
Meniscus Tears

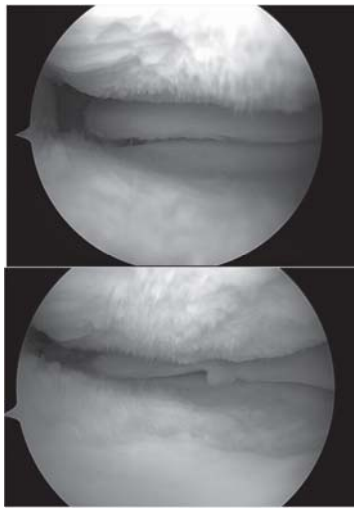
- Nondisplaced oblique or horizontal tears can usually be treated conservatively
- Displaced Bucket Handle requires urgent referral and usually surgical intervention
- Peripheral longitudinal- ? Repair
- Root Avulsion- ? Repair
  - Prognosis deeply affected (extrusion)

Peripheral Longitudinal Tear



Meniscal Root Injuries





Radial Split Tear: The Kiss of Death