AUGUST 21-23, 2015
Loews Chicago O’Hare Hotel
Rosemont, IL

Improve Your Score - Sports Medicine Update
Brian Sokalsky, DO

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Name of CME Activity: AGOFP Intensive Update and Board Review in Osteopathic Family Medicine
Dates and Location of CME Activity: August 20-23, 2015, Loews Chicago O'Hare Hotel, Rosemont, IL

Topic(s):
Examination Techniques for Office Orthopedics—Primary Orthopedics: What You Need to Know

Friday, 8/21/15 7:30-9:30pm

Improve Your Score - Sports Medicine Update
Sunday, 8/23/15
9:45-10:15am

Name of Faculty/Moderator: Brian E. Sokalsky, DO

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Please indicate the name(s) of the organization(s) with which you have a financial relationship or interest, and the specific clinical area(s) that correspond to the relationship(s). If more than four relationships, please list on separate piece of paper.

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<th>Organization With Which Relationship Exists</th>
<th>Clinical Area Involved</th>
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Signature: ___________________________ Date: 8/25/15

Brian E. Sokalsky, DO

Please fax this form to AGOFP at 866-328-1835 or email to joank@acofp.org as soon as possible

Deadline: July 10, 2015
Sports Medicine Review

Brian Sokalsky, DO,
Primary Care Sports Medicine
Jersey Shore Sports Medicine
Team Physician, Jersey Shore Sharks
Rugby Club

Objectives

• Discuss common medical conditions seen in athletes
• Review diagnostic testing for these conditions
• Review treatment and return to play guidelines for these conditions

11 y/o c/o 3 wks of headaches

• HPI: Hit in forehead by opening door in school
  – ?LOC-sent to nurse’s office
    • Initial treatment unclear
  – Lethargic and crying upon return home on bus
  – Vomiting and increased sleep x3days
  – HA, photo-/phonophobia, decreased appetite and energy since
  – Not acting himself
  – CT Head normal
Concussions

New Definition

• 1st International Symposium on Concussion in Sport
  – A complex pathophysiological process affecting the brain, induced by traumatic biomechanical forces. Several common features may be used in defining the nature of a concussive head injury
  • Direct blow to head or elsewhere on body with impulsive force transmitted to head
  • Short lived impairment of neuro function that spontaneously resolves
  • Neuropathological changes with functional rather than structural disturbances.
  • Graded set of clinical symptoms that resolve sequentially
    – May include LOC
  • Typically associated with normal neuroimaging

Pathophysiology (cont.)

• Cellular level- metabolic dysfunction
  – Excitatory amino acid shifts → inc. glycolysis
  – Simultaneous dec cerebral blood flow
• Physiologic changes
  – Increased HR at rest and exertion
  – Increased Sympathetic NS stimulation
  – Altered cerebral autoregulation
  – Altered cytochrome P450 function
  – Changes in circadian rhythm and sleep
  – Increased pro-inflammatory cytokines
Concussion

Signs
- “Dinged”, “Bell rung”, “Don’t feel myself”
- Appears stunned or dazed
- Forgets plays
- Unsure of game, score, or opponent
- Moves clumsily
- Answers questions slowly
- Loses consciousness
- Behavior or personality change
- Forgets events prior to play (retrograde)
- Forgets events after hit (posttraumatic)

Symptoms
- Headache - most common
- Nausea
- Dizziness/Balance problems
- Blurry/double vision
- Photosensitivity
- Feeling sluggish or slowed down
- Feeling foggy
- Concentration problems
- Memory problems
- Fatigue
- Sleep problems

Concussion Evaluation

- ABC’s onfield
  - C-Spine Assessment
- SCAT3-onfield assessment tool
- Physical Exam
  - Cranial Nerves
  - Motor/Sensory exams
  - Cerebellar/Cerebral exam
  - Vestibular exam
- Grading
  - Old vs New

Zurich Conference Return to Play Protocol

- Removal from contest following any signs/symptoms of concussion
- No return to the same game
- Medical evaluation following injury
  - Rule out more severe intracranial injury
  - Neuropsychologic testing
- Stepwise return to play
  - Complete rest until asymptomatic
    - Devoid of clinical symptoms
    - Return to baseline function on neuropsych testing
25 y/o F c/o suprapubic pain x3wks

- HPI: No history of injury
  - Intermittent sharp pain
  - Increased pain with running
    - Runs 5-6x/wk (approx 25-30mi/wk)
  - No groin/rad pain, no numb/ting
  - h/o 4 stress fractures over last 3 years
- PE: TTP over pubic symphysis
  - 5'6" 110 lbs → BMI 17.8
- Bone scan demonstrates stress fx
- DEXA scan: T-score -1.5

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**Female Athlete Triad**

**Disordered Eating**

- Intentional deficit of energy intake compared to energy expenditure
  - With or without eating disorder
- Minimum energy availability requirement for an athlete is 30 kcal/kg of LBM/day
  - Energy availability = Dietary Energy Intake – Exercise Energy Expenditure
  - Can be caused by decreased intake or increased expenditure
- Start of the cascade of deleterious effects of the female athlete triad
Menstrual Irregularities

- Low energy availability
  - Louks, et al: reproductive disturbances with energy deficits due to either increased exercise or dietary restriction, but no disturbances with increased energy along with dietary supplementation
- Decreased energy → decreased GnRH pulsatility
  - LH pulsatility affected with diets below 30 kcal/kg-LBM/day
  - LH more affected than FSH
- Amenorrhea
  - Primary-no menstrual cycles by age 16 with other normal pubertal changes
  - Secondary-persistent absence of menstrual cycles
    - Often defined as 3 months
- Oligomenorrhea-cycle>35 days
- Luteal Suppression and Anovulation-asymptomatic

Altered Bone Mineral Density

- Can be caused by accelerated bone loss as an adult, or insufficient BMD accumulation as a child
- Pathophysiology
  - Negative correlation between number of missed cycles and bone mineral density
  - Estrogen prevents bone resorption
    - Changes not fully reversed with estrogen replacement
  - Other hormones affected by negative energy balance
    - Osteocalcin
    - Carboxyterminal propeptide of type I collagen
    - Insulin, T3, IGF-I
    - Estradiol
  - Decreased Ca and Vitamin D intake
- In premenopausal females and children-use Z-score instead of T-score: < -2.0 considered low bone density below the expected range for age
- Bone density loss may not be fully reversible

Female Athlete Triad

- Screening
  - Annual exam
  - Preparticipation Exam
  - Problem visit with one component of Triad
- History
  - Musculoskeletal
  - Menstrual
  - Psychosocial
  - Nutritional
  - Endocrine
  - Performance
  - Medications
Female Athlete Triad

- Labs
  - B-HCG, CMP, CBC, Thyroid panel, FSH/LH, Prolactin, Testosterone
  - UA
  - Euel and monitoring
  - DEXA
    - Dysmenorrhea-6 months
    - disordered eating-6 months
    - Stress fracture or fracture with minimal trauma
- Physical
  - BMI and percent body fat
  - Eyes and visual fields
  - Parotids
  - Thyroid
  - Heart
  - Skin
    - Russell's sign
    - Scarred knuckles
    - Lanugo
    - Hirsutism
    - Bruising/stra
  - Tanner staging
  - Pelvic exam

Treatment

- Nutritional counseling
  - Increase energy availability to as high as 45cal/kgFFM
- Eating disorder-mental health practitioner
- MVI
- Calcium + Vitamin D
- Monitor urine for ketones
- Continue exercising if no fracture
- ?OCP's?
  - Improved hormone balance and ?BMD
  - ? Increase body fat and decrease performance
- Bisphosphonates-questionable use secondary to long half-life and potential teratogenicity

21 y/o c/o tight chest/SOB x15min

- HPI: Halftime of rugby game
  - No SIGNIFICANT chest trauma
  - Cold, rainy weather
  - Has had similar episodes in cold weather before
  - No previous evaluations
- PE
  - Mild distress
  - +wheezes B/L
Exercise-Induced Bronchospasm

- **Asthma**: chronic disease characterized by 3 features
  1. airway obstruction (may or may not be reversible)
  2. hyperresponsiveness
  3. airway inflammation

- **Exercise Induced Bronchoconstriction**: transient increase in airway responsiveness following 5-8 minutes of strenuous exercise; EIA—such a response in individuals w/ known asthma

Epidemiology

- Prevalence: Over 22 million people in the US (7% of pop) Dx w/ asthma and 90% have EIA if provoked
- 40% of individuals w/ allergic rhinitis have EIB
- Increasing prevalence in athletes reaching over 20% in elite Olympic athletes w/ EIB
  - As high as 50% in winter sports
Clinical Presentation

- Symptoms of bronchoconstriction occur as soon as 3 min after exercise peaking @ 10-15 minutes and resolves spontaneously over 30-90 minutes after completion
- High intensity of exercise (max HR >85%) needed to produce EIB
- Most common symptoms are cough and wheezing
  - Dyspnea, congestion, chest tightness
  - Feeling out of shape, inconsistent performances

Hx and Physical

- Detailed history—include prior attacks or events, fam hx, meds, other medical hx (AR, eczema, etc.)
- Suspicions by trainers, family, coaches
- Screening questionnaires-PPE
- PE typically normal
  - ck for AR signs, complete resp tract (upper and lower) including nasopharynx, sinuses

Diagnosis

- Dx confirmed by demonstration of reduction in PFT’s of 15% in comparing baseline readings w/ post exercise readings
  - Exercise Challenge Test-most common
  - Methacholine Challenge Test
  - Eucapnic Voluntary Hyperpnea Challenge Test
    - Used by IOC to confirm need for bronchodilator
Treatment

• Nonpharmacologic
  - Counseling athletes re: appropriate sport
  - Improve/maintain aerobic conditioning- reducing stimuli for EIB
  - Breathe through scarf or mask in cold/dry air to help warm and moisten air
  - Nasal breathing
  - Avoid pollutants if possible
  - Control assoc. problems (i.e AR, sinusitis, URIs)
  - Avoid smoking

Treatment

• Pharmacologic
  - Short-acting β₂-agonist is treatment of choice
    • Complete prevention of EIB in 80-90% patients
  - AntiLeukotrienes - montelukast/zafirlukast
    • Well tolerated, safe with kids
  - Cromolyn Sodium - mast cell stabilizer
  - Inhaled corticosteroids - stabilize underlying asthma
    • No immediate effect

17 y/o M wants to return to football

• HPI: Recovered from mono
  - Diagnosed 2 weeks ago
    • Symptoms x4wks
  - Feels great, no current symptoms
    • Returned to school 2 days ago
    • No athletic activity for 3 weeks
• PE: normal
Infections

- Mono
  - Viral infection caused by Epstein-Barr Virus
  - Transmitted by oral secretions—“the kissing disease”
  - Classic triad of symptoms—fever, pharyngitis and lymphadenopathy
  - Diagnosed clinically and confirmed with blood test
  - RTP
    - Biggest concern is risk of splenic rupture
    - Greatest risk is 1st 3 weeks of illness, but most athletes still too weak to compete
      - Average symptom resolution is 4-8 weeks
    - Return to light activities after 3 weeks
    - Return to contact less clear, but athlete must at least be asymptomatic

Infections

- URTI’s/Fevers
  - Above the Neck Rule
    - Symptoms above the neck
      - Train at 50% normal intensity for 15 minutes
      - If symptoms improve—increase intensity as tolerated
        - If not (or worsen)—rest and try again when symptoms improve
    - Symptoms below the neck
      - Rest until symptoms resolve
  - If fever—rest
    - Some viral infections can cause myocarditis
Cardiology

Sudden Cardiac Death

• #1 cause of death in young athletes
• 2.3-4.4/100,000 per year
• Strong male predominance
• Football and basketball most common sports
• Majority of athletes are asymptomatic prior to the cardiac event
• Warning signs include exertional chest pain, exertional syncope/presyncope, SOB, fatigue and palpitations

Hypertrophic Cardiomyopathy

• #1 cause of SCD in young athletes in the US
• Pathology
  – Asymmetrical LV hypertrophy-usually involving the septum
  – Disorganized cellular architecture
• Most athletes asymptomatic
• Characteristic exam finding is harsh systolic murmur worsening with decreased venous return (Valsalva/squat→stand)
Hypertrophic Cardiomyopathy

- **EKG**
  - Abnormal up to 95%
  - Prominent Q-waves, deep neg T-waves, high voltage QRS voltage
- **Echo-gold standard**
- **RTP**
  - Low dynamic, low static sports only (maybe)
  - Bowling, golf

Other Causes of SCD

- **Arrhythmogenic Right Ventricular Cardiomyopathy**
  - 4% in US but 22% in Veneto region of NE Italy
  - Prodromal symptoms often present
    - Syncope, chest pain, palpitations
  - EKG-right precordial t-wave inversions, epsilon wave, QRS prolongation
  - No competitive athletics
- **Aortic rupture**-due to aortic root dilation as part of Marfan Syndrome

Marfan Syndrome

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<thead>
<tr>
<th>Category</th>
<th>Major Criteria</th>
<th>Minor Criteria</th>
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<tr>
<td>Skeletal system</td>
<td>Pathologic ectasia (dilated aortic root, dilated aortic root segment)</td>
<td>Facial appearance, chest pain, palpitations, aortic root dilation</td>
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<tr>
<td>Heart</td>
<td>Cardiomegaly, arrhythmias, atrial fibrillation, atrial flutter, QRS prolongation</td>
<td>Soft tissue fibrosis, chest pain, palpitations, aortic root dilation</td>
</tr>
<tr>
<td>Other</td>
<td>Evidence of Marfan syndrome (family history)</td>
<td>Soft tissue fibrosis, chest pain, palpitations, aortic root dilation</td>
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<td>Marfan syndrome</td>
<td>Evidence of Marfan syndrome (family history)</td>
<td>Soft tissue fibrosis, chest pain, palpitations, aortic root dilation</td>
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- **Early onset**
  - Before age 6 years
  - Calf length/height ratio > 0.5
  - Calf length/height ratio > 0.5
  - Calf length/height ratio > 0.5
  - Calf length/height ratio > 0.5

- **Late onset**
  - After age 6 years
  - Calf length/height ratio > 0.5
  - Calf length/height ratio > 0.5
  - Calf length/height ratio > 0.5
  - Calf length/height ratio > 0.5
Athlete’s Heart

- Physiologic and morphologic changes in response to intense regular exercise
  - Increased vagal tone-lower resting HR
  - LV enlargement and increased wall thickness
    - Maintains normal LV filling
    - Larger end-diastolic cavity dimensions
    - Changes resolve with deconditioning over 3-6 months
  - EKG changes include sinus bradycardia, sinus arrhythmia, 1st degree AV block, criteria for LVH

Exertional Heat Illness

Heat Cramps

- Painful muscle spasms, most commonly in the calves, thighs and shoulders that occur several hours after vigorous exertion and begin during rest or showering.
  - Typically last only a few seconds but may last longer.
- Thought to be caused by electrolyte abnormality
- Treatment
  - Prevention
  - Passive stretch/massage
  - Rest
  - rehydration
Heat Syncope

• Results from volume depletion, peripheral vasodilatation which increases blood flow to the periphery of the body (pooling in the legs) decreasing central venous return all causing the athlete to fall.

• Treatment
  – ABC’s !!!
  – Move to cool place
  – Elevate legs
  – Rehydration

Heat Exhaustion

• Elevated temp <104 F with cramps, N/V, HA, malaise
  – Symptoms can be very non specific so a high index of suspicion is required.

• Athletes with heat exhaustion will usually have profuse sweating, dry mucous membranes, flushed skin and muscle tenderness.

• Treatment
  – Must move to cool location immediately
  – Cool body
    • Immersion vs Evaporative cooling
  – Rehydrate
  – If CNS symptoms-treat as heat stroke

Heat Stroke

• MEDICAL EMERGENCY!
• Temp>104 F + CNS dysfunction
  – Ataxia and confusion most common
  – Must r/o hyponatremia with sodium level

• Characteristically present with anhidrosis, tachycardia and hypotension.

• Risk for major organ damage
  – ARF
  – Rhabdomyolysis
  – DIC
Prevention

• How much fluid?
  – 15-20 oz, 2-3 hours before activity
  – 7-10 oz every 15-20 minutes during activity
  – Thirst is a poor indicator of acute hydration status
  – Urine should be pale yellow
• Weigh the athlete before and after the activity.
  – Afterwards they should drink enough fluid to replace the
    weight loss within two hours: 12-24 oz/lb lost.
• Monitor heat and humidity
  – Practice early morning or late afternoon
• Light clothing

References