Cervical Spine Injury Review and Case Presentation

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Emergency Medicine Resident

Objectives

• Appropriately assess for cervical spine injury
• Determine need to use spine board
• Illustrate proper spine boarding techniques
• Properly use cervical spine clearance rules
• Understand the importance of practicing proper technique during participation in athletic events

HPI

• 11 yo male participating in his 1st year of youth football. During the 2nd week of practice he sustains a hit to the facemask causing his head to snap back.
HPI continued

- He described an immediate “pinching” sensation with severe pain in the back of his neck. The injured player also described an electrical, “shock-like” sensation throughout his body that occurred on impact. He was physically unable to move or feel all 4 extremities for 10 seconds. He was then able to move his arms and legs but reported severe pain in the posterior portion of his neck.

WHAT DO YOU DO NOW?

Spine Boarding
• Long backboards are commonly used to attempt to provide rigid spinal immobilization among emergency medical services (EMS) trauma patients. However, the benefit of long backboards is largely unproven.
• The long backboard can induce pain, patient agitation, and respiratory compromise. Further, the backboard can decrease tissue perfusion at pressure points, leading to the development of pressure ulcers.
• Utilization of backboards for spinal immobilization during transport should be judicious, so that the potential benefits outweigh the risks.

Spinal Immobilization

**To Board**
- Blunt trauma and altered level of consciousness
- Spinal pain or tenderness
- Neurologic complaint (e.g., numbness or motor weakness)
- Anatomic deformity of the spine
- High-energy mechanism of injury and any of the following:
  - Drug or alcohol intoxication
  - Inability to communicate
  - Distracting injury

**Not to Board**
- Normal level of consciousness (GCS 15)
- No spine tenderness or anatomic abnormality
- No neurologic findings or complaints
- No distracting injury
- No intoxication

ED arrival

- Meds: none
- Allergies: none
- PMHx: normal development milestones
- Surgical Hx: none
- Social Hx: negative
- Fnhx: none
Physical Exam

**Vitals**
- HR: 110
- RR: 18
- BP: 102/60
- Temp: 99.5
- Pulse O2: 100

- A - patent
- B – present bilaterally
- C – tachycardic, distal pulses intact
- D – no obvious deformity, decreased sensation BUEs to light touch, normal rectal tone
- E – no step offs/non tender I/L spine

Clearing the C-spine

**NEXUS**
- 1. Midline cervical tenderness
- 2. Focal neurologic deficits
- 3. Altered level of consciousness
- 4. Evidence of intoxication
- 5. Painful distracting injury

**Canadian C Spine Rule**

1. Any high risk patient that induces you to order a C-spine xray
2. Key History Factor that would indicate need for a C-spine xray:       
   - High risk patient
   - Intoxication
   - Paralysis
   - Neurologic deficit
   - Evidence of intoxication
3. If you have any of the above, order a C-spine xray

Xray

![Xray image of cervical spine]
Patient Progress

• He was fitted Miami J collar for 10 weeks
• 10 week follow up he had slight decrease ROM after the collar was removed, motor and sensation was intact
• 12 week follow up FROM, normal neuro exam
• 9-month follow up x-rays showed complete healing of C7 fracture
• 1 year follow remained asymptomatic
• Parents discouraged future contact sports

Learning Points

• 60-80% spinal injuries in children are c-spine injuries — 50% fractures — SCIWORA
• Among high school and college football players about 6 players/year become quadriplegics
• Be sure athletes are appropriately taught and execute fundamentals in order to insure safety of all
• Appropriately assess for c-spine injury
• Maintain c-spine precautions
• Know when to use spine board
• Transport to appropriate trauma facility
• C-spine can only be cleared without radiographic studies with an awake, oriented, cooperative child

References

“A six hour hike…”
Sung to the tune of Gilligan’s Island theme song

Dad and Son

Exertional Heat-Related Injuries Treated in Emergency Departments in the U.S., 1997–2006
Nicolaas G. Nelson, MPH; Chirley L. Collins, MA; R. Dawn Comstock, PhD,

- 54,983 treated from 1997-2006
- Annual cases increased from 3,192 in 1997, to 7,452 in 2007
- Rate doubled from 1.2 to 2.5/100,000
Sudden Deaths in Young Competitive Athletes
Barry J. Maron, MD; Joseph J. Doerer, BS; Tammy S. Haas, RN

- A total of 1866 athletes aged 19 +/- 6 years died suddenly
- Sudden deaths were predominantly due to cardiovascular disease (1049 [56%]), but causes also included blunt trauma that caused structural damage (416 [22%]), commotio cordis (65 [3%]), and heat stroke (46 [2%]).

Circulation, 2009

Barry P. Boden, Ilan Breit, Jason A. Beachler, Aaron Williams and Frederick O. Mueller

Fatalities in High School and College Football Players

The most common causes of fatalities were:
Cardiac failure (n = 100, 41.2%)
Brain injury (n = 62, 25.5%)
Heat illness (n = 38, 15.6%)
SCT (n = 11, 4.5%)
Asthma and commotio cordis (n = 7 each, 2.9% each)
Embolism/blood clot (n = 5, 2.1%)
Cervical fracture (n = 4, 1.7%)
Intra-abdominal injury, infection, and lightning (n = 3, 1.2% each).

The Football quadruple whammy

- Deconditioned/Lack of acclimitization
- High heat and humidity
- High BMI
- Equipment
Affected patient populations

- Athletes-high school, college, professional, endurance
- Armed Services
- Firefighters
- Construction workers

epidemiology

- 19 or younger-47%
- Male-72%
- Sports and exercise-75%
- Yard work-11%
- Home maintenance-5%
- Other recreation-4%
- Miscellaneous-5%

Health care providers-Patient journey

- ATHLETIC TRAINERS
- PARAMEDICS
- ED RNs and PHYSICIANS
- INTENSIVISTS

spectrum

Heat Stroke
Heat Injury
Heat Exhaustion
Heat Syncope
Heat Associated Collapse
Heat Cramps

Relative incidence

- Heat exhaustion - 73%
- Dehydration - 19%
- Heat syncope - 10%
- Heat cramps - 5%
- Heat stroke - 2%

Heat cramps

- Intense muscle pain and spasm not due to injury, with prolonged contractions
- "Exercise induced muscle contractions"
- Multifactorial: dehydration, heavy sweating, Na+ and K+ depletion, extreme or de novo exertion, environment, fatigue
Exercise associated collapse (heat syncope)

- Athlete unable to stand or walk as a result of lightheadedness, syncope, or generalized weakness
- Immediately or soon after completing event
- Decreased venous return (dehydration, vasodilation, sudden decrease skeletal muscle tone)
- Temperature normal or near normal

Heat exhaustion

- Heat exhaustion (HE) is defined as a syndrome of hyperthermia (core temperature at time of event usually <40°C or 104°F) with physical collapse or debilitation occurring during or immediately following exertion in the heat,
- No more than minor central nervous system (CNS) dysfunction (e.g., headache, dizziness).
- Resolves rapidly with minimal cooling intervention.

Army medical department definition

Signs and symptoms of heat exhaustion

<table>
<thead>
<tr>
<th>Core Temperature</th>
<th>Symptoms</th>
<th>Signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>37°C to 40°C</td>
<td>Anorexia</td>
<td>Anxiety</td>
</tr>
<tr>
<td></td>
<td>Dizziness</td>
<td>Confusion</td>
</tr>
<tr>
<td></td>
<td>Fatigue and malaise</td>
<td>Hypotension</td>
</tr>
<tr>
<td></td>
<td>Headache</td>
<td>Oliguria</td>
</tr>
<tr>
<td></td>
<td>Nausea</td>
<td>Pyrexia</td>
</tr>
<tr>
<td></td>
<td>Visual disturbances</td>
<td>Vomiting</td>
</tr>
<tr>
<td></td>
<td>Weakness</td>
<td>Tachycardia</td>
</tr>
</tbody>
</table>
Heat INJURY

Heat injury (HI) is defined as HE with clinical evidence of organ (e.g., liver, renal, stomach) and/or muscle (e.g., rhabdomyolysis) damage without sufficient neurological symptoms to be diagnosed as heat stroke.

- Army Medical Department Definition

Heat stroke

- Heat stroke (HS) is defined as a syndrome of hyperthermia (core temperature at time of event usually >40°C or 104°F), physical collapse or debilitation, and encephalopathy as evidenced by delirium, stupor, or coma.
- Occurring during or immediately following exertion or significant heat exposure.
- Can be complicated by organ and/or tissue damage, systemic inflammatory activation, and disseminated intravascular coagulation.

- Army Medical Department Definition

Heat Stroke

<table>
<thead>
<tr>
<th>Core Temperature</th>
<th>Symptoms</th>
<th>Signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt;40°C (104°F)</td>
<td>(same)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Anhydrosis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cardiac arrhythmias</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Disseminated intravascular coagulation</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hepatic failure</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hyperpyrexia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hyperventilation</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Mental Status Changes</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ataxia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Coma</td>
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<tr>
<td></td>
<td></td>
<td>Confusion</td>
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<tr>
<td></td>
<td></td>
<td>Irritability</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Seizures</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Pulmonary edema</td>
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<tr>
<td></td>
<td></td>
<td>Renal failure</td>
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<tr>
<td></td>
<td></td>
<td>Rhabdomyolysis</td>
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<tr>
<td></td>
<td></td>
<td>Shock</td>
</tr>
</tbody>
</table>
Physics/physiology

- Heat Exchange:
  - Conduction
  - Convection
  - Radiation
  - Evaporation

- Depend on gradients of heat and moisture
- Hypothalamic thermoregulation:
  - Peripheral vasodilation, sweating, cardioresp. changes (HR, CO, MV)

-Glazer. American Family Physician. 2005

Environment

Heat and Humidity \[\uparrow\] Heat Transfer

WBGT - wet bulb globe temperature

A composite temperature used to estimate the effect of temperature, humidity, wind speed (wind chill) and visible and infrared radiation (sunlight) on humans.

<table>
<thead>
<tr>
<th>Category</th>
<th>WBGT °F</th>
<th>WBGT °C</th>
<th>Flag color</th>
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<tbody>
<tr>
<td>1</td>
<td>79.9</td>
<td>26.6</td>
<td>White</td>
</tr>
<tr>
<td>2</td>
<td>80.84.9</td>
<td>26.7-29.3</td>
<td>Green</td>
</tr>
<tr>
<td>3</td>
<td>85-87.6</td>
<td>29.4-31.0</td>
<td>Yellow</td>
</tr>
<tr>
<td>4</td>
<td>88-88.6</td>
<td>31.1-32.1</td>
<td>Red</td>
</tr>
<tr>
<td>5</td>
<td>90+</td>
<td>32.2+</td>
<td>Black</td>
</tr>
</tbody>
</table>

Wikipedia (sorry!)
**pathophysiology**

- Heat illnesses occur when thermoregulatory processes are inadequate to preserve core temp homeostasis
- Thermal maximum refers to magnitude and duration of heat that cells can endure before damage occurs (42°C/107°F for 45 min-8 hours)
- Heat exposure causes cardiovascular challenge by diverting visceral blood flow to surface in attempt to lower core temp
- Visceral hypoperfusion can result in tissue ischemia; intestinal ischemia can result in release of gut endotoxins, with resultant pro-inflammatory cytokines leading to SIRS with liver kidney intestine spleen and brain involvement

**Differential Diagnosis**

- Infection (Meningitis, Encephalitis, other)
- Neuroleptic Malignant Syndrome
- Malignant Hyperthermia
- Thyroid storm
- CNS event
- Pheochromocytoma
- Anticholinergic poisoning
- Drug ingestion

**Medications/Substances**

- Alcohol
- Alpha Adrenergics
- Anticholinergics
- Amphetamines
- Beta Blockers
- Calcium Channel Blockers
- Cocaine
- Diuretics
- Neuroleptics
- Thyroid supplements
- Tricyclic antidepressants
Risk factors

- Strenuous exercise in high temperature and humidity
- Lack of acclimatization
- Poor physical fitness
- Obesity
- Dehydration
- Comorbid conditions
- Medications

TREATMENTS of EXERTIONAL ILLNESSES

Treatment - Heat cramps

- Hydrate-oral as good as IV
- Sodium repletion-sports drink or other source
- Relax, stretch, massage
Treatment - exercise associated collapse

- Shade/cool environment
- Supine
- Elevate legs
- Hydrate
- Optional: Cheeseburger, Fries, Milkshake (chocolate preferably)
- Failure to recover fully in 20 minutes or high risk: treat for Heat Exhaustion

Treatment - heat exhaustion

- Shade/cool environment
- Supine
- Raise legs
- Remove excess clothing
- Rehydrate: oral if tolerated, IV if not
- Active recooling: mode not as important as with heat stroke (comfort vs. life saving)
- Monitor vitals
- Transport to ER if improvement not rapid or high risk

Treatment - heat stroke

- Guiding Principles:
  1) Severity may not be initially apparent
  2) Morbidity and Mortality directly related to duration of elevated core temp
- “Heat Attack” and “The Golden Hour”

-Bouchama NEJM 2002
-Heled Military Medicine 2004
Treatment-heat stroke-prehospital

- DRABC
- Rectal temp!
- Blood glucose, sodium if available
- Rapid cooling!

IS ORAL TEMPERATURE AN ACCURATE MEASUREMENT OF DEEP BODY TEMPERATURE? A SYSTEMATIC REVIEW

- Context: “Oral temperature might not be a valid measure to assess core temperature. However, many clinicians, including athletic trainers, use it rather than criterion standard methods, such as rectal thermometry.”

- Conclusion: “Evidence suggests that... oral temperature is an unsuitable diagnostic tool for determining body temperature...”

Mazarolle J Athl Training 2011

Temperature

- Rectal temp only!
- If not available: cool until patient shivers
- Approximately 20 minutes
- Rate 1°C over 5 minutes, total 3° to 4°C
Cold water immersion: the gold standard for exertional heatstroke treatment


Treatment - heat stroke:
Rapid cooling

- Heat attack! Golden Hour!
- Activate EMS, but: cool first, transport second
- Remove clothing (achieve nakedness)
- Ice water immersion preferred! Stir vigorously
- Alternate cooling methods: cool bath, cold shower, hose, ice packs
- Maintain patient safety
- Target temp 38 to 39°C (101 to 102°F)

-Smith Br J Sports Med 2005
-McDermott J Athl Trainers 2009
-Newport Emerg Med J 2009

Treatment - hospital

- ABC’s
- Continue ice water immersion in cooling tub if available
- Adjunctive cool IV fluids
- Cardiac and vital sign monitoring
- Foley to monitor urine output
- Central line to monitor CVP for volume status
- Core temp monitoring
Treatment – supportive

- Hyperthermia
- Volume status
- Electrolyte abnormalities
- End organ complications (CNS, kidney, liver, DIC)

Labs

- CBC
- Electrolytes
- Renal Function
- LFTs
- UA
- Total CK
- Coags
- Urine myoglobin
- EKG
- Lactate

Pharmacological Interventions

- No role
- Antipyretics contraindicated—pyrexia not hypothalamus driven
- NSAIDS—Renal
- Tylenol—Liver
- Immune modulators?
Sickle trait

- SSCT considered benign
- Army recruits with SCT 28 fold higher risk of exertional death
- NCAA Division I players with SCT 22 x higher risk
- Army Way vs NCAA Way: universal vs targeted precautions


Prevention

- Providers: available, familiar, authorized
- Pre-Participation screening: risk factors, previous history
- Acclimitization: 10-14 days, gradual increase in intensity and duration
- Trainers and coaches: educated in recognition and treatment
- Athletes: educated in fluid intake, urine output, weight, sleep
- Environmental: heat and humidity limits
- Clothing: “light and loose,” weight, color, new fabrics
- Rest breaks and rehydration
- Public Awareness Campaigns

Return to duty/play

- Refrain from exercise for at least 7 days following release from medical care
- Follow up one week post release for physical exam and lab testing targeted to specific clinical course
- When cleared to return, begin in cool environment, gradually increase duration, intensity, heat exposure over 2 weeks
- If return to vigorous activity not tolerated by 4 weeks, consider laboratory exercise heat tolerance test
- Clear for full competition if heat tolerant at 2-4 weeks

American College of Sports Medicine Conf Proceedings 2010
Sickle cell Trait associated Death

Michele Kirk, MD
JPS Sports Medicine Fellowship Director
TCU Team Physician
**HISTORY**

- 14 y/o Female
- African American
- Girls basketball player
- Normal PPE
- History of Migraines
  - Topamax

**HISTORY**

- Pre-Season Conditioning
- Timed 2-mile run
- Ambient Temp 86 F
- Humidity 65 %
- Heat Index = 93 F

**HISTORY**

- Collapsed after run
- Assisted to water station by teammates
- Escorted to Athletic Training facility by coach/teammates
DIFFERENTIAL DIAGNOSIS

- AT considerations
  - Exertional Heat Illness
  - Electrolyte deficiency
  - Hypoglycemia
  - Dehydration
  - Cardiac Anomaly

MANAGEMENT

- Oxygen placement
- Blood Glucose = 78 mg/dL
- AED Placement
  - PEA (pulseless electrical activity)
  - EMD (electromechanical dissociation)
- Immediate Resuscitation and ACLS
- Transportation to local hospital

FOLLOW UP

- Southwestern Institute of Forensic Sciences
  - Widespread intravascular sickled thrombi
  - Hemoglobin solubility and hemoglobin electrophoresis both consistent with sickle cell trait (heterozygous) – Hb AS
    - Hemoglobin A – 55%
    - Hemoglobin S – 39%
OUTCOME

• Conclusion – Medical examiner

  – . . . "a 14-year-old black female, died of a sudden cardiac death due to widespread sickled thrombi associated with physical exertion, due to sickle cell trait (heterozygous)."

History

• First death associated with SCT in 1974
• Polie Poitier, Colorado
  – Defensive back
  – Previous collapse on opening day of practice in 1973
  – Second collapse/death in August 1974 doing conditioning drills
  – NCAA guidelines recommended African-American be offered testing for awhile

Jospin Milandu, NC A&T
History

• Most recent collegiate athlete to die from SCT complications
• Track tryouts
• None of the 29 athletes had physicals on file with the school (NCAA mandates sickle testing)
• Athlete’s family claimed no knowledge of SCT status
• NC (the state) has tested for sickle cell (trait & dz) since 1994

History

• NCAA football deaths since 2000
  – 0 from games
  – 0 from practice
  – 16 from conditioning: 15 high-speed drills and 1 weight-lifting
    • 4 cardiac, 1 asthma, 1 exertional heat stroke (EHS)
    • 10/16 deaths were from exertional sickling
    • 3-4% of NCAA football players have SCT, and SCT accounts for 63% of the deaths (16-21 fold increase

History

• Athletes as young as 12 have died from SCT exertional sickling
• 14 yo girl from DFW area, 2002
• 15 yo male football player, Florida, 9/2010
• For every fatal SCT collapse, 3-5 nonfatal collapses
  – Lumbar paraspinal myonecrosis, splenic infarction, leg compartment syndrome
How does exertional sickling happen?

- As little as 2-3 min of maximal, sustained effort
- Severe Hypoxemia
- RBC dehydration
- Hyperthermia of muscles
- Metabolic acidosis
- Risk increased by: heat, dehydration, altitude, asthma

Athlete Presentation

- No trauma, otherwise healthy
- Muscle weakness, tightness, and/or pain
- Doesn’t feel right
- Anxious
- Rapid breathing
- Most often confused with EHS, cardiac cause, asthma
Telltale features among common nontrauma causes of on-field collapse.

<table>
<thead>
<tr>
<th>Sickling</th>
<th>Cardiac</th>
<th>Heat Stroke</th>
<th>Asthma</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weakness/pain</td>
<td>No cramping</td>
<td>Fuzzy thinking</td>
<td>Usually known asthma</td>
</tr>
<tr>
<td>Stumps to ground</td>
<td>Falls suddenly</td>
<td>Unconscious</td>
<td>Breathless, may/may not wheeze</td>
</tr>
<tr>
<td>Can talk at first</td>
<td>Uncoordinated</td>
<td>Incoherent</td>
<td>Gasping, panicky, on hands/knees</td>
</tr>
<tr>
<td>Temp&lt;103F</td>
<td>Temp irrelevant</td>
<td>Temp often&gt;106F</td>
<td>Auscultate: moving little air</td>
</tr>
<tr>
<td>Can occur early</td>
<td>No warning</td>
<td>Usually occurs late</td>
<td>Usually occurs after sprinting</td>
</tr>
</tbody>
</table>

Settings and heart weights in NCAA Division I football sickling collapses.

<table>
<thead>
<tr>
<th>Year</th>
<th>State</th>
<th>Setting</th>
<th>Age</th>
<th>Body Weight (lb)</th>
<th>Heart Weight (gm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2000</td>
<td>Tennessee</td>
<td>Ran 800 m</td>
<td>18</td>
<td>190</td>
<td>----</td>
</tr>
<tr>
<td>2001</td>
<td>Florida</td>
<td>1-h mat drill</td>
<td>18</td>
<td>220</td>
<td>400</td>
</tr>
<tr>
<td>2004</td>
<td>Ohio</td>
<td>Ran 10 min</td>
<td>18</td>
<td>190</td>
<td>----</td>
</tr>
<tr>
<td>2004</td>
<td>Texas</td>
<td>Gassers 30 min</td>
<td>20</td>
<td>300</td>
<td>----</td>
</tr>
<tr>
<td>2005</td>
<td>Missouri</td>
<td>1-h station drill</td>
<td>19</td>
<td>220</td>
<td>500</td>
</tr>
<tr>
<td>2006</td>
<td>Texas</td>
<td>Ran 16 sprints</td>
<td>19</td>
<td>190</td>
<td>425</td>
</tr>
<tr>
<td>2008</td>
<td>Florida</td>
<td>Drills, running</td>
<td>19</td>
<td>185</td>
<td>375</td>
</tr>
<tr>
<td>2008</td>
<td>North Carolina</td>
<td>Ran hill 15 times</td>
<td>22</td>
<td>280-290</td>
<td>550</td>
</tr>
<tr>
<td>2009</td>
<td>North Carolina</td>
<td>Ran 700 yd</td>
<td>20</td>
<td>185</td>
<td>460</td>
</tr>
<tr>
<td>2010</td>
<td>Mississippi</td>
<td>Station drills</td>
<td>20</td>
<td>190</td>
<td>480</td>
</tr>
</tbody>
</table>

Controversy

- Not everyone feels exertional sickling from SCT is the cause of death in athletes/recruits (military, physicians)
- Concern for discrimination of persons with SCT if mandatory testing
- Argument that we don’t mandatory test for other diseases that kill athletes-heart disease
- Argument that precautions are needed for all
Supporting Evidence

What Can We Do?

- Education!-testing and risks
  - Physicians
  - Parents
  - Athletes
- NATA precautions
  - www.nata.org/sites/default/files/sicklecelltraitandtheathlete.pdf

PHYSICAL EXAM

- Evaluation by ATC
  - Dizziness
  - Profuse sweating
  - Shortness of breath
  - Weakness (ambulatory)
  - Lethargy
  - Elevated HR