

# Heat Illness Fluids & Electrolytes in Exercising Individuals

The Sports Medicine Core Curriculum Lecture Series

Sponsored by an ACEP Section Grant

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# Objectives

Differentiate types of exertional heat illness and appropriate treatment.

Determine which types of exertional heat illness need to be transported to the hospital.

# PATIENT #1

18 yo FB player collapses during a pre-season non-contact practice after doing gassers.



# Patient #2

21 yo biker collapses after a work-out with the strength and conditioning coach.



# Patient #3

40 yo female runner crosses the marathon finish line in 3:59:30 and begins to get muscle cramping and feel a little light-headed and nauseous.



# Heat Stroke

Common and can be deadly

8 HS and college deaths from heat illness in 2008

31+ deaths from heatstroke since 1995

Highest incidence in late summer

i.e. football, soccer, track and field

\*National Center for Catastrophic Sport Injury Research



# Heat Production- Endogenous

Muscles can generate 20+ times as much energy at work than at rest;

Only 25% efficient- rest goes to heat release

Core temp +1`C per 5 min; 30 min -> 105`F

Basal Metabolic Rate: 100kcal/hr-> +2`F/hr

Intense exercise: 1000 kcal/hr, 90% by muscle

Dehydration- for each 1L deficit, temp +0.3`C

Heat dissipation impaired by clothing, ointments, etc.

# Heat Gain- Exogenous

## Environment- hot

- radiation (sunlight) +150kcal/hr
- conduction (temperature)
- convection (lack of wind)
- lack of evaporation (humidity)

Clothing- dark colors absorb heat/light rays



# Heat transfer

## Evaporation

Sweat vaporization, predominant at temps  $> 68$ ; 0.6 kcal/mL

## Radiation

Energy transferred via electromagnetic radiation from high to lower energy surfaces; may cause heat GAIN at temperatures  $> 90$  F;

65% heat loss in cool environment

## Convection

Heat transferred via circulating fluid/air, i.e. wind/fan

## Conduction

Heat transfers from warmer to cooler objects by direct contact, i.e. ice packs (water:air = 32:1)

# Heat transfer and dissipation

Body is like a thermostat

Rise in core temp

→thermodetectors in hypothalamus, spinal cord and limb muscles

→ sweating, vasodilation of skin vessels, increased HR and RR

# Cardiac output and plasma volume

Approx 15% of intravascular volume is shunted to working muscles

Additional 15-25% cardiac output shunted to skin

Sweating can produce losses of 1-2 L/hr

# Dehydration

Sweat is hypotonic; even less so in well-conditioned and heat-acclimatized athletes

May not be thirsty until lose 2% of body weight

Dehydration + exercise (heat stress) = decreased stroke volume

# Cumulative dehydration

Insidious onset

Most athletes replace only approximately  
50% volume lost during exercise

Can check urine dipstick to assess

Weigh in before and after workout to  
determine amount of fluid loss

# Hubbard's "energy depletion model"

Elevated skeletal muscle temperature increases metabolic rate

Elevated temperature causes increased cell membrane permeability and leakage of sodium, potassium ions → compensatory cell membrane ion pumping and energy drain

Build-up of lactic acid, fatigue and cell swelling

Muscles fail to relax and get weakness/tightness

# Acclimatization

Physiologic changes- response to heat exposure

Earlier vasodilation and sweating

Increased blood plasma volume

Increased sweat rate and decreased sodium losses

- Unacclimated 1.5L/h, Na<sup>+</sup> 100mEq/L
- Acclimated 2.5 L/h, Na<sup>+</sup> 75mEq/L

Core temperature lower at given work load/heat stress

After a week of daily exercise in the heat,

basal body temperature -0.9 °F (0.5 °C) (Buono et al 1998)



# Acclimatization

7-10 d heat exposure

75% effect first 4 days

100 minutes daily (Lind & Bass, 1963)

Effect persists 1-4 weeks (Pichan et al., 1985)

Every third day for 30 days same as exercising every day for 10 days  
(Fein et al 1975)

30 min per day at 75% VO<sub>2</sub>max as effective as 60 min at 50% (Houmard et al., 1990)

# Acclimatization- NCAA

## Division I: Football Training Camp Rules

first five days one practice/day

one practice/day during school + 1 week prior to game

practices limited to 3 hours

contact:

- helmets only day 1,2
- helmets + shoulder pads day 3,4

no back to back two-a-day practices

3 hours recovery between practices

# Heat shock proteins

Respond to stress and synthesize proteins to protect against normally lethal conditions

Refold denatured proteins into their normal configuration and stabilize them

Threshold temperature for HSP induction not constant

Type of cell, duration, rate of rise, normal environment

Different proteins for short vs long-term acclimation

# Risk factors for Heat Stroke

Hot, humid environments  
(previous day is best predictor)

Dehydration

Barriers to heat  
evaporation-equipment

Illness

History of heat illness

Increased BMI

Poor physical condition

Lack of acclimation

Dark clothing

Over zealous

Medications/drugs

Caffeine, theophylline

Electrolyte imbalance

Predisposing Illnesses

Cystic fibrosis

Sickle cell trait

# What is the spectrum of heat illness?

Heat edema

Heat rash

Heat cramps

Heat syncope

Heat exhaustion

Heat stroke

Hyponatremia

# Heat Edema

Heat acclimation ->

Peripheral vasodilation ->

Increased aldosterone ->

Sodium and water retention

Dependent edema

Rx: supportive care

*No diuretics*

# Heat Rash=Prickly Heat=Miliaria Rubra

Erythematous papules

Clothing covered areas

Keratin plugs

Itching/burning

\*Impairs heat dissipation

Rx: cleanse, cool, dry skin

Sweat gland recovery 7-10 d



# Heat cramps

Acute, painful muscular tightening and spasming

Lower limbs most commonly affected

Dehydration, increased lactic acid, electrolyte depletion, neuromuscular fatigue, and failure of body's thermoregulatory mechanism

# Heat cramps- Treatment

Rest

Massage/stretch/Ice

Replete electrolytes: sodium, potassium, magnesium, and calcium

Salt solution- 1 tsp table salt/1 qt water

Rehydration- po versus IV fluids

\*If prolonged cramping consider sickle trait or rhabdomyolysis

# Heat syncope

Transient hypovolemic syncopal episode due to decreased blood volume returning to heart

\* worse with unacclimated, CV disease, diuretics

At END of race/competition

Maximal vasodilation leads to pooling of blood in lower limbs when exercise stops

Rx-Lie down, elevate legs, rest in cool place, drink cold fluids

# Heat Exhaustion

## Sodium depletion

Rehydration with water

## *Hyponatremia*

Weakness/Fatigue

Headache

Nausea/Vomiting

Muscle cramps

## Water depletion

Low fluid intake

Large sweat loss

## *Hypernatremia*

Weakness/Fatigue

Mild Confusion

Thirst

# Heat Exhaustion

## Mild

profuse sweating  
headache  
dizziness  
nausea/vomiting  
weakness/fatigue  
visual changes  
cutaneous flushing

## Treatment

remove clothing  
cool down  
oral rehydration  
rectal temperature

# Heat Exhaustion

## Severe

temperature <104`F (40.0`C)

tachycardia

hyperventilation

hypotension

decreased urine output

decreased sweating

## Treatment

remove clothing

cool down

oral rehydration

iv rehydration

rectal temperature

+/- transport to ER

# Heat Stroke

## Classic

exogenous heat  
impaired dissipation  
dry skin  
peripheral vasoconstriction

## Exertional

endogenous heat  
sweating  
peripheral vasodilation



# Heat Stroke

## Criteria

Temp >104°F, rectal

CNS changes- confusion,  
behavioral changes,  
irritability, apathy,  
irrational, ataxia,  
delirium,  
seizure (25-50%),  
LOC, coma

## Treatment

remove clothing

*rapid cool down:* fanning,  
ice packs, immersion

avoid shivering

iv rehydration

rectal temperature

transport to ER

seizure/muscle cramping control

-benzodiazepines

core cooling

# Heat Stroke

Above 42`C:

Uncoupled oxidative phosphorylation

Proteins denature- enzymes impaired

Lipid liquefaction- cell membrane integrity

Organ Failure

# Heat Stroke-Complications

heart failure, arrhythmias

rhabdomyolysis, renal failure

hepatic failure

brain

pulmonary-ARDS

hematologic-DIC

electrolytes- hyperkalemia (2<sup>o</sup> to cell death)

lactic acidosis

# Heat Stroke

## Treatment Caveats

Avoid excessive hydration which can cause pulmonary and cerebral edema

Alkalinize IVF when treating rhabdomyolysis

Administer benzodiazepines to stop shivering

Monitor hydration

- Swan Ganz/Foley-urine output

Do not give Ringer's lactate (due to K<sup>+</sup>):

[5.96g NaCl, 3.1g sodium lactate (NaC<sub>3</sub>H<sub>5</sub>O<sub>3</sub>), 0.3g KCl and 0.2g CaCl<sub>2</sub>]

# Heat Stroke- Cooling

Cool first, transport second

Avoid overcooling- stop active cooling at 102`F (38.9`C)

Avoid shivering

# Cooling

## Non-Invasive

Cool environment

Misting + Fans:  $-0.2^{\circ}\text{C}/\text{min}$

Ice packs over major vessels

Cool p.o. fluids

Immersion: up to  $-2.5^{\circ}\text{C}/\text{min}$



# Cooling

## Invasive

Foley irrigation

Gastric lavage:  $-0.15^{\circ}\text{C}/\text{min}$

Rectal lavage

DPL

Dialysis



# Heat Stroke- Cooling

Ice-water immersion cools 2X evaporation

Cold water cools as fast as ice water

Falmouth Road Race (Roberts, 1998)

10-15 runners/yr

rectal temperatures 106-110 °F

observed 20-60 minutes after cooling

walked away, no hospitalizations

# Heat Stroke- Prognosis

Every minute counts

Morbidity correlates with duration and magnitude of hyperthermia  
(area under the curve)

With fast cooling, survival rate approaches 100%

(Kark et al., 1996)

Febrile @2 hours exposure- 70% mortality

Poor outcome if:

$T > 42.2^{\circ}\text{C}$ , SGOT  $> 1000$ , coma  $> 4$  h, DIC

Overall mortality 15%

# Medications & Heat Illness

Uncouple oxidative phosphorylation

salicylates

Blunted cardiac response

calcium channel/beta blockers

Increase heat production/muscle activity

stimulants, thyroid hormone, neuroleptics,

drugs of abuse- cocaine, PCP, LSD, ephedra

# Medications & Heat Illness

Decrease heat loss

inhibited sweat response

- anticholinergics/ antihistamines/tricyclic antidepressants

vasoconstrictors

- cocaine, (pseudo)ephedrine

hypovolemia

- diuretics/caffeine/alcohol

# Prevention of Exertional Heat Illness

Progressive intensity and duration of training over 10-14 days prior to event under same heat conditions

Important to drink and cool body down between practices on same day/successive days due to cumulative losses

Match fluid losses with sodium-containing fluids

Urine should be light yellow

< 2% body weight change- no practice unless within 2% weekly baseline body weight

# Prevention

Sleep 6-8 hrs/night in cool environment

Well-balanced diet

NATA statement, “If the WBGT is greater than 28 C (82F, or “very high”) an athletic event should be delayed, rescheduled, or moved into an air-conditioned space, if possible.”

# Wet Bulb Globe Temperature Index

$$WBGT = 0.1DBT + 0.7WBT + 0.2BGT$$

Dry Bulb Temperature- mercury thermometer

Wet Bulb Temperature (humidity)

Black Globe Temperature (solar radiant heat)

American College of Sports Medicine Risk Category	Wet Bulb Globe Temp	National Weather Service Heat Index
Dangerous Zone	>90°F	>115°F
Very High Risk Zone	82 - 90°F	98 - 115°F
High Risk Zone	73 - 82°F	80 - 98°F
Moderate Risk Zone	65 - 73°F	65 - 80°F
Low Risk Zone	< 65°F	< 65°F



# *Exertional Heat Stroke during a Cool Weather Marathon*

*Med Sci Sports Exerc* 38(7):1197–1203, 2006

3X yo M well trained, marathon time 3:15

Collapsed near finish line, agitated, diaphoretic

Start time: Temp 43`F, humidity 99%

End time: Temp 49`F, humidity 62%

27 min later rectal temp in ER= 105.3`F

Intubated, echo showed stunned myocardium

# Fluid Requirements

Sedentary adult, temperate climate: 2 L/day

Sedentary adult, hot climate: 6 L/day

Athlete, hot climate: 10-18 L/day

Sweat losses during exercise: 2-3 L/hr

one pound=450ml=15 oz

# Voluntary Intake Not Enough

Thirst response @ plasma osmolality +2%

Runners drink < 500 mL/h (Noakes et al 1991/1993)

Sweat rates average 1,500+ mL/h

Marathoners dehydrated @ >1,000 mL/h



# Dehydration & Exercise Capacity

2.5% water loss induced prior to exercise:

30% reduction in power output (Nielsen et al 1982)

2% body weight loss dehydration prior to 1500 meter race:

3.7% slower = 6 seconds at world-class pace

(Armstrong et al 1985)

# Complete v Partial Volume Replacement

50 min exercise at 80%  $\text{VO}_2\text{max}$  followed by brief, high power cycling test

1300ml v 200ml replacement during first 35 min

heart rate lower 4bpm

core temperature  $-0.33^\circ\text{C}$

6% faster during last leg

70 g of carbohydrate

6% improved performance

# Salt Supplementation

Average American diet ~10-20g

Sodium loss with exercise ~10L=29g

0.1% oral salt solution:

2 ten grain tablets or 1/4 t in one quart

Salt tablets

Generally not advised

Can be useful in hot, humid climates

Need free water access!

# Water Absorption Rates

Volume: ~20ml/min; 1200 ml/hr

Carbohydrate:

>8% slows absorption

7% solution absorbed 30% faster than water

# Fluid replacement

Modify drinking behavior by:

Education

Availability

Palatability

Consume 500-600 mL fluid 2-3 hours prior to activity,  
then 200-300 mL 10-20 min prior to activity

200-300 mL every 10-20 min during exercise





# Fluid replacement

Non-competitive athletes: replace at  $\sim 500$  mL/hour

Cold water and well-balanced diet

Intense exercise for  $> 1$  hour:

Ingest beverages containing 4-8% carbohydrates  
(14g/8 oz serving) and 0.5-0.7g/L  $\text{Na}^+$

Stimulate thirst centers and supplement  $\text{Na}^+$  losses

Consume  $\sim 20$  oz fluid for every pound lost

Before, during and after meal

# Locker Room Guidelines

Body weight must be within 4% baseline  
*prior to leaving area*

Body weight must be within 1-2% baseline  
*prior to next practice*



# Fluid Comparisons

Solution	Sodium mEq/L	Potassium mEq/L	Glucose Gm/dL	mOsm/L
WHO	90	20	2.0	310
Gatorade	20	3	2.1	
Gatorade + Gatorlytes	80		2.1	
Normal saline	154	0	0	300
Table salt 1 tsp	125	0	0	

# Hyponatremia

AKA: “Water intoxication”

Ultra-endurance, marathon runners

Incidence up to 30% (most clinically insignificant between 125-135)

Etiology not entirely clear

- Excessive hypotonic fluid consumption

- Excessive sweat loss

- Normal levels of vasopressin and aldosterone

Slower runners and females at greater risk

# Hyponatremia

Prolonged exercise prevents complete suppression of ADH

Even small amounts of ADH reduce renal excretion of water

Athlete retains ingested fluids

Na<sup>+</sup> loss in sweat is a minimal factor in reducing serum sodium concentration

# Signs and symptoms of (EAH)

Many similar to dehydration/heat illness

Early signs and symptoms:

Bloating

“puffiness”

Nausea/vomiting

Headache

More serious signs and symptoms:

Altered mental status due to brain swelling

- Confusion/disorientation
- Agitation

Seizures

Respiratory distress (pulmonary edema)

Obtundation



# Hyponatremia

2002 Boston Marathon study (Almond, N Engl J Med 2005)

Prospective study 488 runners

13% with sodium levels  $< 135\text{meq/L}$

3 runners with sodium levels  $< 120\text{meq/L}$

Associated with weight gain during race, race time, consumption of  $> 3\text{L}$  of fluid, or consumption of fluids every mile

Not associated with type of fluid consumed

# Hyponatremia

## 2002 Boston Marathon study (Almond, N Engl J Med 2005)

Weight gain 2-2.9kg , 10% had Na < 130meq/L

Weight gain >3kg, 30% had Na < 130meq/L

Race time < 3:30, 8/195 finishers were  
hyponatremic (4%)

Race time > 4:00, 32/140 finishers were  
hyponatremic (22%)

Longer time to consume more fluids?



# Hyponatremia

## 2002 Christchurch Marathon study

(Reid, et al. Clin J Sport Med 2004).

155 runners

No cases of hyponatremia

Water stations every 5km, compared with every 1.6km in Boston and most North American marathons

Use of NSAID in 24 hours prior to race associated with significantly elevated creatinine levels

# Sodium losses in sweat

Wide range of sodium concentration 20–110 mmol/L

Sodium losses not shown to be a primary cause of  
Exercise Associated Hyponatremia.

Armstrong, et al. Med Sci Sports Exerc. 1993;25;543-49.

No difference in sodium losses between athletes who  
develop EAH and those who don't.

Speedy, et al. Clin J Sports Med. 2000;10;272-278.

# Prevention of Hyponatremia

Do not drink  $> 500-1000\text{cc/hr}$

Increase distance between drinking stations

Hypotonic sodium/carbohydrate solutions instead of water may help

# Are NSAIDS a Risk Factor for Development of Hyponatremia?

# NSAIDs and Hyponatremia

Runners using any type of NSAID (including COX2) more likely to become hyponatremic after 60km mountain run.

Page, et al. Clin J Sports Med. 2007 Jan; 17(1): 43-8.

Athletes using NSAIDs had lower sodium and higher creatinine levels after New Zealand Ironman triathlon.

Wharam, et al. Med Sci Sports Exerc. 2006 Apr; 38(4):618-22.

330 participants

30% NSAID use

1.8% hyponatremic → Statistical significance

# NSAIDs as a risk factor?

Similar incidence in hyponatremia b/w

+NSAID and -NSAID use

Almond et al. Boston Marathon Study

NSAID use associated with elevated creatinine levels.

2002 Christchurch Marathon study (Reid, et al. Clin J Sport Med 2004).

155/296 participants

No incidence of hyponatremia

# Does Creatine Increase Risk of Heat Illness?

# Creatine

95% of body's creatine stored in muscle cells

Buffer

Brief, intense bouts of exercise rely heavily on this system

> 300 studies: “creatine loading”

Increase levels by 25%

Up to 5 days before a competition



# Does creatine increase risk of heat illness?

Theory: increases intracellular water

*Greenwood et al 2003*: NCAA DI athletes over 3 years. No significant difference b/w creatine and no creatine users on heat illness/muscle injuries

*Rawson and colleagues 2001*: Eccentric muscle contractions and measured levels of markers of muscle damage. Found no difference in creatine supplementation vs placebo

# Does creatine increase risk of heat illness?

*Volvek et al 2001*: 20 cyclists in hot, humid environment and cycled 30 min at 60-70% peak VO<sub>2</sub> max. Placebo vs creatine groups showed no incidence of heat illness

*Kreider and colleagues 2003*: 98 college FB players over 21 months and looked at urine and serum markers of muscle damage and found no difference on creatine vs placebo or length of creatine supplement use

If we suspect heat illness, do we really need to take a rectal temperature?

# YES!

- \*Important to measure CORE temperature
- \*Oral, axillary, tympanic are inaccurate in the exercising athlete

# IV versus Oral Rehydration

Conscious, cognizant athlete without nausea and vomiting can orally rehydrate

# IV vs Oral Rehydration?

Rate of Perceived Exertion:

CONTROL > ORAL & IV at 5 and 15 min

IV significantly higher than oral at 15 min

Thirst Ratings:

Lower ( $p < 0.05$ ) during ORAL than CONTROL and IV at min 0, 5, and 15.

Thermal sensation:

CONTROL > ORAL & IV at 5 and 15 min

IV > ORAL at 15 min

Herrera et al., "PERCEPTUAL RESPONSES TO EXERCISE IN THE HEAT FOLLOWING RAPID ORAL AND INTRAVENOUS REHYDRATION," *Med Sci Sports Exerc.* 1998;30(5s):6.

# IV vs Oral Rehydration?

Castellani JW, Maresh CM, Armstrong LE, et al. Intravenous versus oral rehydration: effects on subsequent exercise heat stress. *J Appl Physiol.* 1997;82:799–806.

Casa et al, “Intravenous versus oral rehydration during a brief period: responses to subsequent exercise in the heat,” *Med Sci Sports Exerc.* 2000;32:124–133.

# Take-home points

Exertional Heat Illness can be deadly

Keep athletes hydrated, but not overly hydrated

Any persistent or profound change in mental status =  
call 911

Education

Acclimation can improve adaptations to heat stress