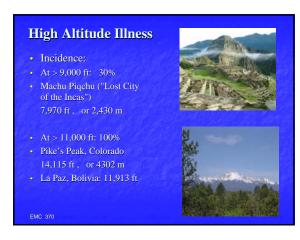
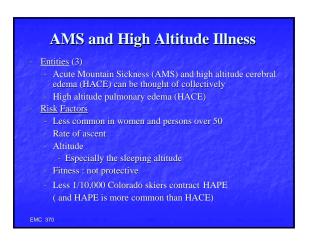
High Altitude Illness, AMS, and other Environmental Injuries Lecture 19 Unit 3

Outcomes At this lecture's completion, the learner will be able to: Integrate pathophysiological principles and the clinical presentations of environmental disorders of: Discuss the clinical picture and the implications for treatment of: Acute Mountain Sickness and the High Altitude Syndromes Lightning injuries Blast injuries





High Altitude Illness Pathophysiology Hypoxic ventilatory response (HVR) Hypoxia-induced over perfusion of the cerebral and pulmonary vascular beds with resulting: ↑ hydrostatic pressures ↑ interstial third-spacing and edema ↑ intracranial pressure → ↑ sympathetic reflex (vasopressin; renin,...) Systemic fluid retention → ↑ hydrostatic pressures in lung and brain

Acute Mountain Sickness An acute high altitude illness: Unacclimatized person Just traveled to altitude > 2500 m (8200 ft) HA and plus I or more of the following: GI (N,V, anorexia) Dizziness Lassitude; fatigue; insomnia Onset usually within 1 - 6 hr Signs: not helpful Cheyne-Stokes respirations (at > 9,000 ft 100% of persons) Significance of early sx. + signs; Harbinger of more serious high-altitude illnesses; HACE, HAPE

Prevention of AMS

- · Acclimation : Slow, staged, 3+ days ascent
- Acetazolamide (carbonic anhydrase inhibitor; sulfa...)
 (Diamox) 250mg BID-TID; (3) days: day before / day of arrival / 1day after ascent
 - . Renal bicarb excretion
 - · Metabolic acidosis
 - . H+ + HCO3- <--> H2O+CO
 - . Increased respiratory drive
 - . Decreased sleep-hypoventilation
 - . Decreased hypoxic ventilatory response
- Decr cerebral vasodilitation--> capillary leakage
- Other: Dexamethasone ; Ginkgo biloba
- Worse with hypoventilation (sedatives, hypnotics)

EMO 07

AMS Treatment

· O2

Avoid further ascent until symptoms resolve
 Consider descending

· 1000 - 1500 ft : usually adequate)

ASA, ibuprofen

· Dexamethasone 4-8 mg po, IV, IM

Start Acetazolamide, if not already begun

EMC 070

HACE High Altitude Cerebral Edema

- Progression of AMS sx . + signs :
 - HA, N, V
- · Ataxia
 - · a hard, end organ finding
- Stupor
- · Progression to seizures, coma
- · Papilledema, retinal hemorrhages
- · CN palsies

EMC 370

HACE Treatment

· Immediate *descent* (definitive Tx)

0

· Dexamethasone 8-12 mg IV, IM

· Consider

- Gamow bag (port. hyperbaric chamber)
- · Lasix 80 mg IV
- Mannitol 0.25 1.0 gram/kg IV
- · Intubation and hyperventilation

HACE Dx unlikely if:

- Onset > 3 days after ascent
- No HA

Failure to respond to O2, steroids, and descent

EMC 3

HAPE High Altitude Pulmonary Edema

Leading cause of high altitude-related deathsRisk factors

- Heavy exertion
- Rapid ascent
- · Cold exposure
- · Excessive salt intake
- · Use of sleeping meds, sedative-hypnotics (alcohol)

· Previous Hx of HAPE / HACE

- believed to be due to (1) a reduced ventilatory response to hypoxia; and (2) an exaggerated pulmonary vasoconstrictive response to both hypoxia and to exercise

EMC 3

Clinical Presentation of HAPE

Hackett, PH N Engl J Med 345 (2): 107, July 12, 2001 does not use a severity classification [Hultgren HN] exactly You may find it helpful to grade the severity: 1/2 or 2/2

Severity: mild to moderate

- · Dyspnea on exertion (DOE)
- Dry cough
- · Fatigue
- · Dusky nail beds

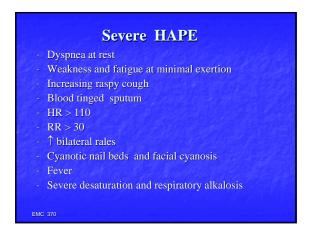
· Localized rales (typically beginning in right lung)

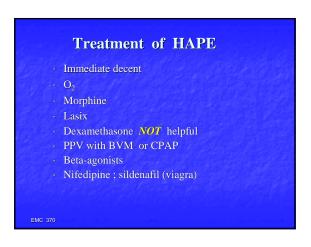
50% (but only 50%) have had prior AMS sx
Occurs on the second night ["2 lungs on night 2"]

Rarely occurs after four nights

EMC 370

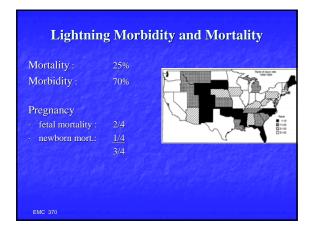
Environmental Emergencies

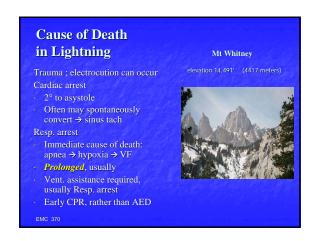


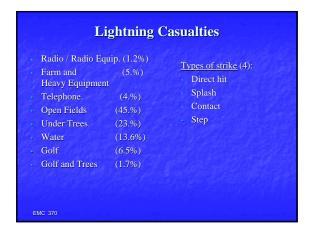


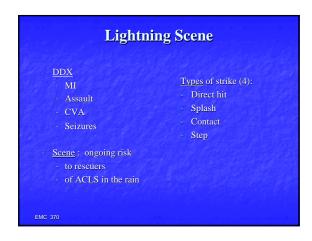


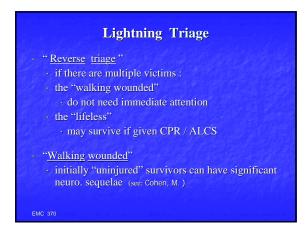


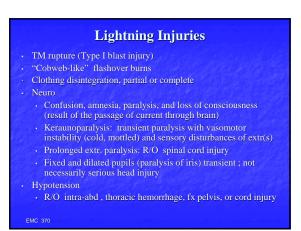


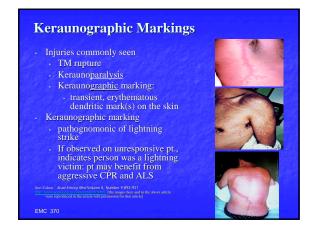




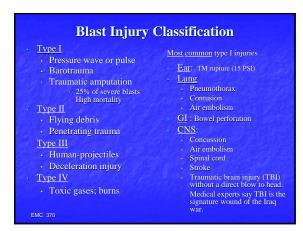












Traumatic Brain Injury (TBI) "A blast creates a sudden increase in air pressure by heating and accelerating air molecules and, immediately thereafter, a sudden decrease in pressure that produces intense wind. These rapid pressure shifts can injure the brain directly, producing concussion or contusion. Air emboli can also form in blood vessels and travel to the brain, causing cerebral infarcts..." (Mayorga MA: The pathology of primary blast overpressure injury, Totic 1997;121:17:29) "... 22,600 U.S. soldiers wounded in the conflicts in Iraq, Afghanistan, and other locations as of Nov. 4, 2006, blasts have been by far the most common cause of injury, and 59% of blast-exposed patients at Walter Reed have been found to have a TBI...". Traumatic Brain injury in the War Zone Okas, S. N. Engl. J. Med. 2005; 352: 2043-2057 "... Kevlar body armor and helmets are one reason for the high proportion of TBIs among soldiers wounded...." usua "... because mortality from brain injuries among U.S. combatants in Vietnam was 75 % or greater, soldiers with brain injuries made up only a small fraction of the casualties treated in hospitals ..." usua

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Patient and Event History in Blast

History:
- nature of explosive
- closed / open
- distance

Symptoms:
- hearing↓
- dyspnea
- chest pain
- hemoptysis
- LOC
- stroke sx

History:
Conduction medium (water)
- Hollow organ (ear, lung, Gl, CNS) injuries will occur:
- with > severity in water and
- at > distances in water (than in air)
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Blast Injury Treatment

• A / C

• Facial fractures / FB

• B

• O2 100%

• R/O pnthx. / tension pnthx.

• Cautious ventilation (Risk of air embolism)

• C: IV

• Support BP

• Monitor 4

• D:

• Disability (cortex / brain stem / cord)

• Drums
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Blast Injuries

D:

Assess the D rums (as an indicator of psi.)

If TMs are intact:

pulmonary injury very unlikely

TM rupture:

if P > 15psi.: 50 % will have TM rupture

Lung injury:

if << 30 psi:

unlikely

if P > 75 psi.:

very likely

75% of victims will have lung injury
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Notes: Blast and TBI

Okie S. Traumatic brain injury in the war zone. N Engl J Med 2005;352:2043-2047.

Mayorga MA. The pathology of primary blast overpressure injury. Toxicology 1997;121:17-28.

CDC web pages:

Blast Lung Injury: What Clinicians Need to Know. http://www.bt.cdc.gov/masscasualties/blastlunginjury.asp

See also;
Explosions and Blast Injuries: A Primer for Clinicians http://www.bt.edc.gov/masscasualties/exotesions.asp
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Environmental Emergencies

Summary

Acute Mountain Sickness: a "headache plus" that can develop into severe HACE.

HAPE, which may or may not be preceded by AMS, results from the HVR (hypoxic ventilatory response

· HAPE has the highest lethality

HAPE is a non-cardiogenic pulmonary edema which may respond more to O2, CPAP, and beta agents than to a standard NTG-lasix approach-but NOT to steroids

vent. assistance. Reverse triage. And tell-tale signs of injury. Blast injuries of hollow organ (ear / lung / GI / CNS); and what to use as indicator of psi. and of possible lung burst