DISASTER MANAGEMENT, EARTHQUAKES AND THE CRUSH SYNDROME

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I. **Course Description** - This course will:

A. Discuss the modes of injuries in natural disasters
B. Provide a description of the events surrounding two 20th Century Earthquakes and one 19th Century Earthquake
C. Discuss in detail the pathophysiology of the Crush Syndrome
D. Reflect on the current status of Disaster Preparedness in the U.S.
E. Suggest research endeavors to improve the medical response to natural disasters

II. **Natural Disasters**[1]

A. In order for natural events to be considered disasters, they must impact human populations[2]
B. Epidemiology
   1. Health decisions have been ineffective in the past.
      a. Often based on insufficient or false data
   2. Timing of the relief effort should parallel health requirements.
   3. Can vary by disaster and particularly by the day that the help arrives.
C. Floods
   1. the most common natural disaster
   2. 0.2-2% incidence of injuries requiring medical attention
D. Earthquakes
   1. high incidence of injuries and mortality
   2. Prediction
   3. Initial effects
      a. Property Damage with Entrapped population
      b. Injury data dependent on composition of collapsed structures
   4. Delayed effects
      a. lack of food, shelter, communication, transportation, sanitation
      b. disruption of medical care

5. Epidemiology [3]

Morbidity/Mortality after Earthquakes

Earthquake	Morbidity	Mortality	M/M Ratio
Peru 1970	143,331	66,974	2.2
Nicaragua '72	20,000	6,000	3.3
Pakistan '74	15,000	4,700	3.2
Guatemala '76	76,504	22,778	3.4
Tangshan '76	525,000	250,000	2.1

III. Armenia

A. Earthquake [4]

1. Occurred 11:35 AM, Dec 7, 1988
2. 12,000 hospitalized, 25,000 bodies recovered, 750,000 homeless, 11% housing destroyed
3. Yerevan - capital city, no damage
4. Leninakan - 290,000 with three hospitals destroyed and possibly 20,000 dead
5. Spitak - village of 25,000 with 8-10,000 dead

B. Soviet Treatment of Crush Injuries and Crush Syndrome

1. antibiotics, anticoagulants, IV fluids
2. non-use of mannitol
3. Pentoxifylline [5]
4. Surgical Management, Dialysis, CT

C. Injury Data

1. Three Villages Receiving No Foreign Aid[6]
   a. 5000 patients admitted
      (1) 20% head injury
          40% extremity injury with 440 amputations
          25% superficial injury
          11% crush injury, 50% developed renal failure
   b. 95% victims bystander rescue with no on-site medical care.
   c. Transportation to hospitals by private vehicles
   d. 3 dead to 1 injured
   e. Primary cause of death due to total collapse of buildings not adequately designed for earthquakes
   f. Entrapment: 80% trapped died, 50% in 2-6 hours
2. Total Relief Effort Including Foreign Aid[7]
   a. A large number died slowly due to shock, exsanguination, or head injury.
   b. early search and rescue primarily by uninjured co-victims

c. evacuations were rapid
d. hospital intensive care well organized

e. international aid had little impact due to late arrival

IV. Earthquakes in the U.S. - Building damage and mortality expected due to lack of seismic building codes and absence of earthquake preparedness.

A. Loma Prieta Earthquake 1989 - 43 killed
1. Santa Cruz (epicenter), San Francisco (highway)

B. New Madrid Earthquake 1811-12 - Missouri, largest in North America.[8] [9]
1. Three separate quakes greater than Richter 8
2. tremors lasted almost two years, thousands of individual quakes

V. Tangshan China 1976[10] [11] [12] [13]

A. Geography
1. Tangshan, 200 miles from Beijing, 1 million population

B. Earthquake Data
1. 7.8 Richter July 28, 1976 at 3:42 AM with 7.1 Richter aftershock
2. Epicenter directly under the city of Tangshan
3. 242,419 killed (1 death / 2 wounded)
a. 7218 households in which all members of the family died
b. many died from lack of medical care after rescue
4. 86% of population crushed under fallen debris
a. Entrapment - 20% died, 80% were rescued
(1) 16% died crushed by falling structures
(2) 35% died by suffocation (dust)
(3) 25% died slowly from entrapment and exposure
b. 525,000 wounded, 165,000 severely, 2200 paraplegics
(1) 50% had fractures
(2) 30% contusions
(3) 20% crush syndrome

C. Medical Relief Effort
1. Three phases
a. Initial Response (days 1-5)
(1) Bystander rescue
(2) surviving medical workers, police, military
b. Second Phase (days 3-6)
(1) 280 medical teams from surrounding area arrived
(2) 30,000 cases treated with bandaging and dressing, antiseptics and suturing, splinting, CPR, tetanus

c. (1) Third Phase (Week two to week eight)
(2) 1.6 billion cases treated, 156,000 serious
(3) 21,000 patients transported to outside hospitals
(4) Disease control (enteritis and dysentery)
(5) Body disposal - no disease or contamination
(6) Sprayed area to kill flies and mosquitoes

D. Military Relief Effort - Rescue teams were mainly organized by the army.[14]
1. 16,500 local soldiers, 100,000 total
2. Provided assistance in search and rescue, body disposal
3. caused considerable problems
a. did not bring food or water
b. caused serious traffic jams

VI. Crush Syndrome: Historical Perspective
A. Initially described by Von Colmers after the 1909 Messina, Italy earthquake
B. After WWI, the Germans[15] and the British[16] described the features of the Crush Syndrome
C. Renal failure following crush injuries was first described in 1941 by after the German bombing of London.[17] Bywaters[18] predicted a 5% incidence of the crush syndrome after civilian bombing in this conflict.

VII. Crush Syndrome: Definition[19] - Prolonged continuous pressure on the extremities results in rhabdomyolysis (death of skeletal muscle) with the release of muscle cell content into plasma, myoglobinuria, and eventually acute renal failure. Consequences of prolonged crush injuries:

A. Local Injury - Crushed muscle tissue with increased pressures cause death of that muscle. Skin is remarkably resilient to crush.
B. Systemic Injury -
1. Early - Crush injuries that are prolonged can result in rhabdomyolysis with associated release of myoglobin, potassium, phosphorus, and creatinine into the circulation. Calcium enters the muscle cell. This can result in hyperkalemia, hypocalcemia, hyperphosphatemia, and metabolic acidosis. Skeletal muscle becomes extremely edematous and contributes to hypovolemia. Cardiac arrhythmias or hypovolemic shock can cause **sudden death**.
2. Late - This volume contracted state in an acidic environment with myoglobinemia can lead to oliguria, deposition of myoglobin in the renal tubules, and eventually **renal failure**.

VIII. Crush Syndrome: Causes

A. Natural - Earthquakes and tornadoes can result in the collapse of steel-reinforced masonry structures.

Earthquakes

1990	Iran	40,000 dead
1988	Soviet Armenia	25,000 bodies recovered
1980	Compania, Italy	256 dead
1976	Tangshan, China	250,000 dead
1989	California La Prieta	43 dead
1906	San Francisco	700 dead
1896	Charleston, S.C.	60 dead
1811	New Madrid	8 dead (deaths on the River not accounted for)

2-5% of injured in an earthquake can be expected to develop the Crush syndrome.

B. Motor vehicle accidents
C. Military -
D. MAST trousers - contraindicated in the Crush Syndrome.[20]

IX. CRUSH SYNDROME: PATHOPHYSIOLOGY[21] [22]

A. Rhabdomyolysis -
   1. influx from ECF to muscle cells
      a. Na+ and Ca++ - hypovolemia, prerenal azotemia and oliguria, hypocalcemia
   2. efflux to ECF from Damaged muscle cells
      a. Potassium - hyperkalemia
      b. Purines - hyperuricemia
      c. Lactic Acid and Phosphate - metabolic acidosis
      d. Myoglobin - nephrotoxicity
      e. Thromboplastin - DIC
      f. Creatinine - increased serum creatinine

B. Effects of Reperfusion - Skeletal muscle damage greatest after reperfusion.
   1. The Oxygen Paradox: Oxygen Free Radicals -
      a. Xanthine Dehydrogenase is converted to Xanthine Oxidase in the presence of Calcium proteases and ischemia.
      b. Xanthine oxidase plus Oxygen (after reperfusion) results in superoxide radicals.
      c. Lipid peroxidation - Oxygen free radicals attack the unsaturated bonds of free fatty acids.
         (1) damage to cell membrane causing cell swelling, interstitial edema, cell death and necrosis.

2. The **Calcium Paradox:** Increased intracellular Calcium -
   a. Damaged Na-K-ATP Pump - due to decreased Ph and ischemia.
   b. Sodium-Calcium Exchange Pump - can survive ischemia. Increased intracellular sodium is exchanged for Ca++.
   c. Increased pressure on muscle causes Stretch Myopathy - increases the leakiness of the membrane to calcium.
   d. Effects of intracellular Hypercalcemia -
      (1) decreases ATP production
      (2) increased phospholipase A2 promoting cellular injury
      (3) calcium-activated proteases stimulate production of free radicals which increases cellular injury after reperfusion
3. Activated Leukocytes - in response to Interleukin I and TNF
   a. produce oxygen free radicals
   b. increase permeability of the endothelium

C. Hypocalcemia - cardiac arrhythmia, especially with hyperkalemia
D. Hyperkalemia - cardiac arrhythmia
E. Hyperphosphatemia - interferes with renal function, metastatic calcification
F. Nephrotoxicity -
   1. Deposition of myoglobin casts at ph < 6.5 with hypovolemia.
   2. Prerenal - hypovolemia if untreated can cause secretion of constrictor hormones (vasopressin, thromboxane)
   3. Oxygen Paradox - oxygen free radicals from the muscles may directly harm the kidney

*** Systemic effects of the Crush Syndrome do not occur **until the extremity is perfused after the crushing object is removed.**

X. Crush Syndrome - Clinical Presentation[23]

A. The Crushing injury
   1. Primary - The crushing object creates a sandwich of dead muscle tissue between the object and bone.
   2. Secondary - a transitional zone of dead or dying tissue surrounding the crushed area (stretch myopathy).
   3. Ischemia - total ischemia causes muscle death after six hours.
   4. Reperfusion - Increased microvascular permeability leads to interstitial edema
   5. compression necrosis, nerve constriction, rhabdomyolysis

6. decompressive fasciotomy, excision of necrotic tissue, amputation

B. Appearance
   1. Limbs - may be grossly edematous with tense, shiny skin. Hypesthesia and decreased range of motion are common.
   2. Overlying Skin - thin and shiny to abraded to deeply contused or obviously necrotic. Any laceration greatly increases morbidity.

C. General Presentation
   1. Alert, intoxicated, hypothermic, hypotensive, comatose, confused, combative. Language may be a problem. History of allergies, present medications, and immunization status.

XI. CRUSH SYNDROME: MANAGEMENT [24] - Relief from the crushing object releases numerous cardiotoxic substances into the circulation. (hyperkalemia, hyperphosphatemia, and metabolic acidosis can lead to lethal cardiac dysrhythmias)

A. Hypovolemia
   1. Sudden reperfusion of the crushed extremity results in an acute hypovolemia. Movement of fluid from the ECF to the edematous muscle cell increases fluid shift.
      a. Large volumes of Lactated Ringer's solution even before patient freed. 1.5 liter bolus initially, then 300-550 cc/hr, with CVP at least 5 mm Hg.
      b. 12 liters (equivalent to entire extracellular volume) may be required in 24 hours.
      c. Decision Tree for initial fluid resuscitation[25] is dependent upon monitoring and lab capabilities

B. Treatment of Acidosis
   1. Urine Alkalinization Ph > 6.5 with IV sodium bicarbonate can prevent myoglobin deposition in the renal tubules.
   2. The brisk diuresis will correct most metabolic aberrations. Acetazolamide administered in boluses of 250 mg IV can prevent systemic alkalosis if serum Ph > 7.45.

C. Treatment of Hyperkalemia
   1. High volume crystalloid therapy in addition to urine alkalinization may be adequate.
   2. 5cc calcium chloride IV may be necessary for severe Hyperkalemia with EKG changes. This can be measured (level greater than 7.5 with EKG changes) or estimated (ventricular ectopy with high peaked T waves resembling ventricular tachycardia).
      a. Calcium should be used to treat hyperkalemia only. The hypocalcemia should not require therapy. Ectopic calcification in the kidney and muscle tissue may further complicate clinical picture.

3. 25cc D50W IV followed by 10 units regular insulin IV is helpful for severe hyperkalemia.

D. Treatment of Oliguria
   1. Maintaining a diuresis of 300cc/hour with IV fluids and mannitol in an average dose of 160 grams/day.

E. Hyperbaric Oxygen[26]
   1. preserves adenosine triphosphate (ATP) function in the cell membrane
   2. maintains tissue oxygenation despite hypoperfusion
   3. decreased tissue necrosis in dog leg crush
   4. increased superoxide dismutase to inhibit lipid peroxidation from O2 free radicals.
   5. arrests progression of muscle edema and can obviate the need for surgical decompression of compartment syndrome.

F. Treatment of Calcium Paradox
   1. Amiloride - potassium-sparing diuretic drug
      a. inhibits sodium-calcium exchange
      b. net reduction in intracellular calcium loading
      c. volume loading is essential
      d. EXPERIMENTAL - may elevate K+, treat at scene?????

G. Treatment of Oxygen Paradox
   1. Free-radical scavengers (superoxide dismutase, catalase, allopurinol, Mannitol)
      a. may attenuate or prevent reperfusion-induced injury to ischemic skeletal muscles and the kidneys
      b. given before crush is relieved or as early as possible

H. Renal Failure
   1. Oliguria, myoglobinuria, and acidosis with myoglobin in the renal tubules leads to acute renal failure. 6 hour delay of fluid therapy, 50% develop renal failure. 12 hours delay, 100% develop renal failure. Renal failure with rhabdomyolysis has a mortality of 20-40%. **Prevention is the key!**
   2. Hemodialysis[27]
      a. No permanent renal damage if sepsis is avoided.
      b. Graded Response to need is essential for efficient utilization of resources.
      c. 150 patients dialyzed after the Armenian Earthquake. 300-500 may have suffered from acute renal failure.

I. Local Injury: Management of the **Compartment Syndrome**
   1. Compartment Syndrome: Definition
      a. An anatomical Compartment consists of soft tissue contained in unyielding envelope of fascia, bone and skin.

b. Pressures greater than 30 mm Hg for six hours or longer[28] prevent venous drainage and result in muscle necrosis (Compartment Syndrome) and require fasciotomy.
   (1) Pressures above 55 m Hg are associated with loss of neuromuscular function without surgical decompression.[29]
   (2) Fasciotomy has been recommended for pressures
      (a) greater than 45 mm Hg (Mubarak[30])
      (b) within 20 mm Hg of diastolic (Whitesides)
   (3) Whitesides[31] [32] maintains that pressures that would require fasciotomy are actually within 20 mm Hg of diastolic pressure. In addition, he notes that significant crush injuries that are longer than 8 hours old would be dangerous to have fasciotomy due to:
      (a) increased tissue necrosis requiring disfiguring debridement
      (b) increased risk of sepsis
   (4) Mubarak[33] and Schwartz[34] recommends early fasciotomy a) to prevent Volkmann's Contracture and
      (a) to reduce the systemic effects of the crush syndrome.

c. pain, paresthesia, pain with passive stretch, increased intracompartmental pressure, paralysis, progression of symptoms.

d. Pressures can best be measured in the field by the Whitesides technique.[35]

e. If a fasciotomy is performed, all fascial compartments must be opened as well as the overlying skin[36]

2. <u>Closed injuries</u>
   a. <u>Fasciotomy</u> necessary with elevated intracompartmental pressure, obliterates the blood supply
   b. <u>Two Controversial Views</u>
      (1) <u>Currently Accepted</u> - Early fasciotomy preserves the limb, avoids Volkmann's contracture, and maintains cutaneous sensation.
      (2) <u>Alternate view</u> - If distal pulses are present, then fasciotomy in a closed wound can be avoided. Fasciotomy converts closed wound to open wound - sepsis.

3. <u>Open Crush Injuries</u>
   a. antibiotics, tetanus toxoid, and thorough cleansing are mandatory[37]. Debridement of nonviable tissue protects against sepsis.

b. Compartment Syndrome
(1) Fasciotomy necessitates radical excision of all dead muscle tissue to prevent sepsis. Split thickness skin excision may be utilized to determine viable tissue borders.[38]
c. Early amputation - severely injured limb limits sepsis.

J. Summary of Treatment of the Crush Syndrome
1. Volume - CVP > 5, 300 cc/hour urine output
2. Mannitol
a. diuretic action
b. decrease oxygen requirements of renal tubules
c. oxygen free radical scavenger to treat oxygen paradox
3. sodium bicarbonate - urine Ph > 6.5
4. consider fasciotomy or amputation

XII. Disaster Management in the U.S. [39] [40]

A. Disaster Planning
1. Cooperation of community and local, state, and federal governments
2. Identify available resources and special needs based on specific disaster models to predict injury patterns
3. Utilize disaster drills to evaluate local response variables
4. Establish priorities, triage procedures, and levels of response
a. Level I Disaster - Local EMS and Community
b. Level II Disaster - Regional response
c. Level III Disaster - Involves state and federal resources
5. Absolute Priorities
a. Assessment of existing resources
b. Pre-arranged mutual aid agreements
c. Identification of lead agencies and individual leaders

B. Incident Command System - a management system for crisis response
1. common organizational structure
2. standard language and communications
3. Unified command utilized "Management-by-objective-style"

C. NDMS - National Disaster Medical System [41] - A partnership of FEMA, DOD, VAH, HHS and private medical volunteers to supplement the local and state response to the medical needs of victims of large-scale natural disasters or to assist existing agencies with survivors of a conventional military conflict.

1. DMAT's - Disaster Medical Assistance Teams [42]
   a. Stabilize the victims
   b. Arrange transfer to prearranged hospitals for definitive care
   c. There are currently 58 teams either operational or forming. 8 to 15 are ready for immediate mobilization. NDMS is currently working to increase the total number of DMAT's and to redefine their composition and role.[43]
   d. Problems - transportation, funding, communication and leadership.

D. Special DMAT's
   1. FEMA announced in February 1991 through the work of Drs. Claude Cadoux and Joseph Barbera that 26 Search and Rescue teams will be formed and completely funded. There will be a medical component led by emergency physicians.
   2. Dr. Susan Briggs leads the Special DMAT subcommittee of the NDMS Steering Committee.[44] As a trauma surgeon, she will coordinate the activities of this special contingent to include trauma surgeons, burn surgeons, emergency physicians, anesthesiologists, critical care specialists, etc.

E. Future Considerations
   1. Institutional Sponsorship - University or Corporate
   2. Private Funding
   3. Involvement of the Military [45]
   4. Heavy equipment for search and rescue
   5. Medical Equipment
      a. Only 30% of the 5000 tons of medical equipment sent by the international community to Armenia was able to be utilized.[46]
      b. Antibiotic Selection[47]
      c. Mannitol, IV Fluids, Foley Catheters
      d. Nutritional Requirements - dictated by need and by individual evaluation.[48]
   6. Specialized Equipment
      a. Flexible fiberoptic scopes, bedside ultrasound[49] [50]
   7. Practice, Practice, Practice!!

XIII. Who to Contact (Information on Disaster Medicine and disaster teams)

A. National Disaster Medical System
   1. Federal, Health and Human Services
      a. Harold Rebuck or Thomas Reutershan (NDMS)
      b. 301-443-1167 (call for information)

B. American College of Emergency Physicians
   1. Section of Disaster Medicine
      a. 1-800-798-1822
      b. Chair - Paul Roth, MD, FACEP
      c. ViceChair - Henry J. Siegelson, MD, FACEP
      d. Secretary - Frederick Burkle, MD, FACEP

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