CRUSH SYNDROME

PRESENTED BY DEREK HICKS

HISTORY

Crush syndrome (C.S.) is described as a life-threatening condition caused by extensive crushing trauma, characterized by destruction of muscle and bone tissue, bleeding, and fluid loss.

The first documented cases were found trapped in collapsed structures following a 1909 earthquake in Messina, Sicily. In 1941, Bywaters and Beall published a comprehensive description of this syndrome following observations during the Second World War. Crush Syndrome is also called Bywaters Syndrome.

PATHOPHYSIOLOGY

Crush syndrome occurs slowly and after only prolonged, continuous compression of a muscle mass in one or more extremities. C.S. does not occur in the torso, head, or neck injuries. In order for C.S. to occur, the extremity is usually compressed for a minimum of four (4) hours.

The compression of an extremity causes obstruction of venous return (blood returning back to the heart), while arterial supply supply continues. The result of poor venous return and continued arterial pumping is increased internal pressure in the extremity. It appears that full arterial obstruction does not cause C.S.

Due to the extremity compression and decreased venous return, the supply of oxygen-rich blood is diminished and carbonic acid begins to rise. Muscle-cell metabolism changes from aerobic to anaerobic metabolism. This causes metabolic acidosis. During this event, the metabolic work in the extremity continues. The muscle cells continue to consume energy and produce metabolic waste, which only adds to the increasing acidosis.

Pressure within the muscle compartment begins to increase, causing the muscle cells to stretch, which in turn causes the cell membranes to leak myoglobin into the circulation. Within the compressed tissue, there are a number of minerals, enzymes, lactic acids, and myoglobin. Also contained are oxygen-free radicals, which are destructive, toxic substances produced by incomplete metabolism. It is the combination of these substances the cause significant cellular damage of the injured extremity after the compression is released.

It is following the pressure release, fresh oxygen-rich blood rapidly reperfuses the extremity. As the new pH-balanced blood fills the injured extremity, the highly toxic, hyperkalemic (abnormally elevated levels of potassium), and acidotic blood is flushed out of the crushed extremity and into the central circulation. A common result is sudden ventricular fibrillation (cardiac arrest) due to the blood's acidity and high levels of potassium.

If the patient survives the reperfusion of the extremity, hypovolemia is a common complication as blood pools in the injured muscle mass. Pulmonary embolism and adult respiratory distress syndrome (ARDS) are possible late complications. If the patient survives the initial injury, the possibility of death due to renal failure and associated infections secondary to immune system compromise is greater than 40% in patients that are misdiagnosed and under-treated following the injury.

Myoglobin, which is normally soluble in normal-pH blood, becomes a solid in acidic blood, and in turn clogs the kidneys. This will cause life-threatening kidney failure.

PROGNOSIS

The expected outcome for C.S. victims following proper pre-hospital and hospital care is excellent. This of course is dependent on the length of compression, and the amount of muscle mass involved. Without rapid and proper assessment and care, almost 80% of patients with prolonged compression injuries will develop severe complications. Of these, more than 40% will die. It is obvious that with simple and effective recognition and treatment of potential crushing injuries, C.S. can be prevented or greatly reduced.

PATIENT ASSESSMENT

Early recognition and proper pre-hospital treatment remains key in preventing or reducing C.S. Rescuers must maintain a high index of suspicion of C.S. when there is a muscle mass compression injury of an extremity. Crush syndrome can occur when circulation is restricted for more then four (4) hours. It can also occur in any condition that causes an altered level of consciousness that makes the patient lay in a supine (flat) position for more than four hours. Some of these conditions include strokes, alcoholism, hypothermia, trauma, and muscular overexertion. Other rare occurrences

include long distance flights (i.e. 18 to 24 hour flights), and casts on fractured extremities.

Presentation Of Compressed Extremity In Crush Syndrome

- Cold
- Hard
- Insensible (no sensation in the extremity)
- Diaphoretic
- Painless; and/or
- Distal pulses present

Deceptive Symptoms

- Patients appear quite well when first discovered and during the extrication phase
- Frequently alert and oriented, and sometimes even in good spirits
- typically free of pain
- Distal pulses are frequently present in the compressed extremity beyond the crushing object
- Vital signs are typically normal, unless there are other traumatic injuries, or other complications
- If the extremity remains pulseless after compression is released, suspect other injuries to the extremity

Further Assessment and Treatment

- Do not rule out C.S. if there is not a detectable pulse in the affected extremity. The pulses may be to weak to feel, or have become absent do to increasing pressure within the extremity.
- Always perform a complete head-to-toe exam both before and after the release of the pressure. This includes the ABC's and the search for any other injuries. Do not be fooled by the victim's lack of pain.
- Inspect the crushed extremity for open wounds, fractures, pulses, and sensation beyond the injury site.
- After the compression is released, constantly monitor the patient's ABC's, vital signs, and level of consciousness. Cardiac arrest (V.-Fib) is a great risk due to the high levels of potassium (hyperkalemia), and the acidity of the blood released from the injury site. Always have a defibrillator at hand during compression release and transportation to the hospital.
- If the patient losses consciousness, or begins to have difficulty breathing after the compression release, immediately give high flow oxygen, assist ventilations when required, place the patient in shock position (legs elevated 45%), and run IV fluids wide open.
- Always obtain a thorough past medical history, medications, and medical allergies prior to releasing compression in the case the patient losses consciousness.
- In most cases, edema (swelling) is not event during compression, however, following the release, edema may occur rapidly.
- Do not give anything by mouth during the extrication (including water). Always anticipate that the patient will require surgical intervention. Also, remember to ask the patient when they last ate or had anything to drink.

Additional Advanced Life Support Intervention

- Oxygen be non-rebreather mask (treatment in all trauma)
- I.V. fluids (500 ml/hr)
- Sodium Bicarbonate, 44 meq added to every second 1000 ml bag of saline or R/L. This is to maintain urine pH greater than 6.5.
- Monitor urine output (you may be there a long time). Dark urine is an indication of myoglobinuria.

SUMMARY

Early suspicion, assessment, and intervention will prevent the onset of crush syndrome. If left untreated, the risk of death due to sudden cardiac arrest, hypovolemia, acidosis, pulmonary embolus, renal failure, and infection, is very high. The mortality rate exceeds 40% if this condition is not detected or not treated. On scene recognition and early intervention results in an excellent prognosis. The best way to achieve this outcome is the teamwork involving both firefighters and EMS paramedics, and hospital personnel.

GLOSSARY

Aerobic metabolism: Chemical process at the cellular level involving the consumption of oxygen to promote growth, generate energy, and eliminate waste.
Anaerobic metabolism: Chemical process at the cellular level that takes place in the

absence of oxygen.
Carbonic acid: Water and carbon dioxide compound; normal byproduct of cellular metabolism.
Hypovolemia: The lack of circulating blood volume most commonly caused by hemorrhage. The common cause of shock is hypovolemic shock.
Metabolic Acidosis: Acidosis resulting from excess acid due to abnormal metabolism.
Myoglobin: Oxygen-binding protein in muscle cells; similar to hemoglobin in red blood cells.
Oxygen-free radicals: Partially reduced oxygen reduced species that are extremely toxic. These are produced due to incomplete metabolism as the result of partial burning.
Pulmonary Embolism: Blockage of the pulmonary artery by foreign matter such as fat, air, tumor tissue, or a thrombus that arises from a peripheral vein.

Derek Hicks is a Firefighter with the City of Niagara Falls, Ontario. He has brought of wealth of knowledge through his years as an Advanced Care Paramedic with the Niagara Regional EMS system. Presently, Derek is an active member with the Niagara Falls Fire Dept. Extrication Team, in the Medic position.

Note about the research:

The majority of the information contained in this presentation was derived from the research of Mr. William Raynovich, senior program manager of the University of New Mexico's EMS Academy.