**VOLUME 1 Chapter 34
Crush injury**

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**Definition and concepts**

*Crush injury* is the anatomical injury associated with direct trauma due to a compressive force. Extended entrapment with compression may cause crush syndrome, traumatic rhabdomyolysis, or compartment syndrome. *Crush syndrome* is the systemic manifestation of skeletal muscle injury from extended compression. Crush injury-induced traumatic rhabdomyolysis is one form of rhabdomyolysis; however, prolonged immobilization of an individual against a surface (e.g. due to altered level of consciousness) and agitated delirium can cause rhabdomyolysis without external trauma. *Compartment syndrome* is the increase in pressure within a fascial compartment eventually compromising venous outflow then arteriolar inflow with progressive capillary leak and edema leading to skeletal muscle injury that can progress to rhabdomyolysis. All three of these clinical entities may be encountered by EMS physicians, potentially in the same patient.

**Pathophysiology of crush injuries**

The systemic manifestations of crush syndrome are due to ischemia/reperfusion injury of skeletal muscle and the intense local and systemic inflammatory response due to the physiological, biochemical, and immunological changes that accompany the ischemic and reperfusion periods [1]. Ischemia/reperfusion injury is encountered in EMS practice in acute stroke, head injury, myocardial infarction, and crush injury with crush syndrome. Reactive oxygen species and activated neutrophils are the main contributors to local and systemic effects of ischemia reperfusion. Oxygen is the substrate initiating this response and is provided upon reperfusion. All tissue is sensitive to ischemia reperfusion but skeletal muscle injury can cause major systemic complications.

From the EMS perspective, the critical factors placing a patient at risk of crush syndrome are mass of muscle injured and ischemia time. The critical muscle mass necessary to put an entrapped patient at risk for crush syndrome is poorly defined, but qualitatively requires more than that of a hand or foot. Critical ischemia time is better defined but variable. At body temperature, critical ischemia time (the maximum time a tissue can tolerate ischemia and remain viable) of skeletal muscle is 4 hours [2]. However, critical ischemia time can be shorter when direct trauma is the cause of ischemia rather than just vascular occlusion.

The compression-stretch myopathy and ischemia of crush injury with muscle compression result in sarcolemma membrane leak and the release of myoglobin, urate, potassium, and phosphate out of muscle cells. Water, calcium, and sodium leak into muscle cells. Fluid and electrolyte shifts, myoglobinuria, and hyperkalemia then contribute to the systemic manifestations of crush syndrome after muscle compression is relieved and perfusion is reestablished. Large volumes of intravascular fluid shift into the injured muscle, leading to hypovolemia and hypotension. Hyperkalemia and metabolic acids cause bradydysrhythmias and reduced cardiac output while uric acid from muscle purines and myoglobin cause acute renal injury. From a clinical management perspective, the early consequences of crush syndrome are hypovolemia, hyperkalemia, and metabolic acidosis and the late consequence is acute renal failure due to myoglobinia and uricosuria.

**Clinical setting**

Because the compression-stretch myopathy and reperfusion injury of crush injury that goes on to become crush syndrome requires muscle compression of the order of hours rather than minutes, the clinical setting of EMS patients in which crush syndrome is to be suspected will be somewhat unique. Mostly these patients will be entrapped in some way, requiring disentanglement and extrication involving technical rescue, or may require a search operation before extrication is possible. In single-patient or small multicasualty incidents this may involve traffic collisions, industrial, construction or machinery incidents, explosions, structural collapse, debris flows, and below-grade or confined space entrapments.

Although the critical mass of skeletal muscle necessary to cause the systemic effects of crush syndrome is uncertain, the clinical setting would likely involve entrapment of an extremity, possibly as far as the shoulder, hip, or gluteus. Torso compression associated with traumatic asphyxia would be rapidly fatal.

In the large multicasualty or mass casualty/disaster setting, crush syndrome is associated with structural collapse due to earthquakes, floods, tornadoes, hurricane/tropical cyclones, or events of war. Because substantial time might pass before survivors are found in search operations following a disaster, crush syndrome can be a major contributor to delayed morbidity and mortality. Crush syndrome that leads to acute renal failure has been reported to be the second most frequent cause of mortality following disasters, after direct trauma [3–5].

**Management of crush injury**

The field management of crush injury will depend upon the immediacy of disentanglement and extrication, extent of the anatomical injury, access to and availability of definitive health care infrastructure, transport time, number of casualties, scope of practice of field EMS providers, and availability of advanced field EMS response resources.

Fundamentally, treatment begins with control of external hemorrhage and stabilization of orthopedic and soft tissue injuries following stabilization of the airway, assisted ventilation, and decompression of a tension pneumothorax as needed. Early and effective hemorrhage control can be life-saving.

Following external hemorrhage control, stabilization of the soft tissue and orthopedic injuries through dressings and splinting will contribute to reducing any further injury during patient movement and to pain control. These interventions should not, however, independently delay transport to definitive trauma care, especially when the crush injury is part of other multisystem trauma.

Pain management is both therapeutic and humanitarian during EMS operations. The operational use of analgesics in EMS is defined by two principal considerations: the analgesics available within the scope of practice of the EMS provider, and the spectrum of adverse effects as they relate to the patient and to the rescue environment. Opiates are a common and effective analgesic widely available within EMS practice. In the rescue environment one important consideration is respiratory depression. Access to and management of the airway may be limited during disentanglement and extrication of an entrapped patient, so escalating doses of opiates and their associated effect of respiratory depression must be considered. When scope of practice or availability of advanced EMS responders allows, adjunctive ketamine can be useful during EMS rescue operations. Ketamine in subanesthetic doses (0.1–0.2 mg/kg intravenous or intraosseous) as an adjunct to opiates can reduce the dose of opiates needed to achieve the degree of analgesia required to improve patient comfort and tolerance of movement necessary during the rescue evolution.

During mass casualty/disaster operations, patient evacuation to definitive care can be delayed by hours or days. If there are open soft tissue injuries or open fractures, empirical antimicrobial therapy can be given. Literature on infection prevention, morbidity, and mortality following crush injury in disasters is lacking but pending clinical evidence, it is reasonable to administer intravenous or intramuscular antistaphylococcal antibiotics, and tetanus toxoid or tetanus immune globulin if those resources are available. Of note, soft tissue injuries with heavy soil contamination (e.g. injuries from tornado debris) have resulted in fungal infections and soft tissue injuries following improvised explosive devices with cross-contamination from bone and tissue of other persons will require HIV, HBV, and HCV prophylaxis and postinjury surveillance.

During the field care of a rescued entrapped survivor, an extremity crush injury may begin to show signs of compartment syndrome. Compartment pressure measurement may not be practical, and the classic signs of pulselessness, pallor, paresthesia, paralysis, and pain out of proportion to injury are all late signs. If compartment syndrome is clinically suspected in association with crush injury in the mass casualty/disaster setting based upon mechanism of injury and pain on passive movement of a distal digit, the therapeutic decision is between adequate and monitored fluid resuscitation and field fasciotomy. Given the absence of outcome data on field fasciotomy and the risk of infection, nerve and other iatrogenic injury, it is likely better to address fluid resuscitation requirements and keep the affected extremity at heart level than to perform a field fasciotomy.

## Management of crush injury with suspected crush syndrome

When extremity entrapment has been of the order of hours or longer, treatment extends to the prevention of morbidity and mortality from two additional mechanisms: sudden hypotension and cardiovascular collapse upon extrication, and late renal failure. In the individual patient or multicasualty setting, the duration of entrapment that puts the patient at risk for sudden hypotension and cardiopulmonary arrest has not been well established but traumatic rhabdomyolysis has been reported to occur in less than 1 hour [6]. Myoglobinuric acute renal failure following rhabdomyolysis has been reported to occur in up to 33% of cases and to account for up to 50% of fatalities [7], and was prevalent following the earthquakes in Armenia, China, and Turkey [8]. Morbidity and mortality from immediate postextrication hypovolemia and hyperkalemia as well as late myoglobinuric acute renal failure can be reduced through field interventions.

When crush syndrome is clinically suspected during the management of crush injury, based upon time of entrapment and mass of skeletal muscle compressed, the principal intervention is intravascular fluid resuscitation [7]. Substantial fluid shifts from the intravascular compartment to the interstitial and intracellular compartments during entrapment can result in a precipitous drop in blood pressure following disentanglement and release of the compressing force. A reduction in cardiac output can result from hypovolemia as well as dysrhythmias from hyperkalemia and metabolic acidosis when the injured extremity is reperfused. In single-patient or multicasualty incidents involving crush injury, access to the vascular space will be intravenous or intraosseous. Intraosseous vascular access will have to be in an ipsilateral or contralateral largely uninjured extremity. Proximal and distal long bone as well as manubrium access sites may be limited by local scopes of practice. If advanced practice EMS providers or EMS physicians are available, as well as during mass casualty/disaster events, then central venous access as well as hypodermoclysis (with or without hyaluronidase) may become options for intravascular volume resuscitation.

From a medical logistics perspective, intravenous and intraosseous fluid resuscitation in the rescue environment is probably best done using intermittent bolus infusions. Depending upon the patient’s physical position and confinement during entrapment, it may not be practical to hang intravenous solutions to be delivered by gravity, and intravenous infusion pumps may not be available. Pressure infusion bags are a consideration but are difficult to control. Additionally, there may be technical rescue operations going on in close proximity to the patient and intravenous lines, monitoring cables, and oxygen tubing are prone to cutting and dislodgment. Medical and rescue personnel will need to coordinate operations to keep the rescue effort moving. Well-coordinated drug and intravenous fluid bolus therapy along with interval patient assessment will help to optimize patient care as well as rescue operations.

When intravenous or intraosseous vascular access is impossible and central venous access and hypodermoclysis are unavailable, then applying an arterial tourniquet close to the time of disentanglement and release of the compressing force may prevent sudden fluid and electrolyte shifts. Literature on the safety and efficacy of arterial tourniquets for this purpose is lacking, but they should be applied at any point when hemorrhage uncontrolled by other means is part of the problem.

The endpoint of intravascular resuscitation is more difficult to define. Conceptually the goal is to maintain cerebral, coronary, and renal blood flow during the compartmental fluid shifts and/or hemorrhage associated with crush injury, crush syndrome, and extremity reperfusion. The difficulty is defining field-expedient measurable endpoints that reflect those goals. Currently heart rate and heart rate trends, target systolic blood pressure, and presence of pulse oximetry waveforms and measurements of waveform quality from digital capillary beds are practical measureable endpoints in EMS practice. Patient access may be limited if entrapment includes confined space operations. It is also conceivable to monitor urine output, or at least obtain a colorimetric measurement of urine specific gravity if that equipment is available.

Further confounding decision making regarding intravenous fluid resuscitation of a crush injury patient is the concomitant occurrence of head injury, blunt and/or penetrating multisystem trauma, or underlying illness such as chronic heart failure or renal disease. The goal of maintaining end-organ perfusion is favorable when head injury and crush syndrome occur together. However, the strategy of permissive hypotension in penetrating trauma, and perhaps in blunt trauma as well, is in direct conflict with the strategy of intravascular fluid resuscitation for crush syndrome. Evidence-based composite endpoints for systolic blood pressure or other vital signs and fluid resuscitation in the comorbidities of head injury with penetrating or blunt trauma associated with crush syndrome have not been established.

The other intra- and postrescue short-term risk in crush syndrome is hyperkalemia. Whether hyperkalemia evolves during the extrication or is a consequence of limb reperfusion following disentanglement and removal of the compressing force, it can result in bradydysrhythmias and, along with postextrication hypovolemia, contribute to sudden death. Therapeutic options for hyperkalemia will depend upon scopes of practice and availability of medical resources during the rescue.

Maintaining perfusion with intravenous volume resuscitation is the essential therapy. A second generally available strategy is empirical treatment of metabolic acidosis or blood alkalinization using intravenous or intraosseous sodium bicarbonate (e.g. 1 mEq/kg intermittent bolus therapy every few hours). Although field-expedient point-of-care blood analyzers are available, measurement of acid-base parameters is usually limited to specialized medical resources that may not be part of the initial response. Another strategy would be to add 50–100 mEq of sodium bicarbonate to 1 liter of 0.45% sodium chloride, making it approximately isotonic, and use that solution for intravenous fluid replacement. Better goal-directed therapy would be possible if a point-of-care blood analyzer were available, or with continuous or intermittent ECG precordial lead V1 and V2 monitoring for T-wave shape and amplitude; however ECG monitoring for hyperkalemia lacks sensitivity and specificity [9]. Hypotension in spite of intravenous volume resuscitation accompanied by atrioventricular block, tachycardia or bradycardia, or wide QRS complex (‘sine wave’ ECG appearance) would usually require intravenous or intraosseous administration of calcium chloride or calcium gluconate; however, in the setting of skeletal muscle crush syndrome, calcium is taken up by the injured muscle, making it difficult to raise blood calcium levels, and can aggravate the calcium-dependent apoptosis of myocytes [7].

Another generally available treatment for hyperkalemia is inhaled beta2-agonists like albuterol (e.g. 5 mg every few hours). Atmospheric monitoring and ventilation are important considerations if the rescue involves confined space operations and oxygen is used to drive a nebulizer. Compressed air (breathing air quality) is an alternative to oxygen if hypoxia by pulse oximetry is not present, and is more likely to be a renewable resource in the mass casualty/disaster setting. When available, a very effective treatment of hyperkalemia is intravenous or intraosseous glucose and regular insulin. This therapy requires frequent monitoring of blood glucose.

Late morbidity and mortality from crush injury and crush syndrome can have many causes including sepsis, brain injury, and organ system failure from multisystem trauma. However, myoglobinuric acute renal failure can be prevented or can be reversed by renal replacement therapy (peritoneal dialysis, hemofiltration, or hemodialysis) when fluid resuscitation is begun prior to disentanglement and extrication [7]. Early intravenous or intraosseous fluid resuscitation is the mainstay of treatment, along with empirical sodium bicarbonate to alkalinize the urine to protect the kidneys from the nephrotoxic effects of myoglobin and uric acid. When used early, mannitol, as both an osmotic agent and a polyalcohol free radical scavenger, can be nephroprotective by reducing interstitial fluid volume and muscle compartment pressure, thereby reducing release of myoglobin and purines, and by maintaining renal perfusion [10]. Mannitol can be given alone (e.g. 0.5 g/kg of a 20% solution) or 20–30 g of mannitol and 50–100 mEq of sodium bicarbonate added to 1 L of 0.45% sodium chloride to be given as an infusion or intermittent boluses.

## Transport destination considerations

Even in the absence of multisystem trauma, the potential for complex soft tissue, orthopedic, nerve, and vascular injuries resulting from crush injuries makes a designated trauma center the preferred acute care destination. Designated trauma centers will have access to the specialties of plastic and reconstructive surgery, microvascular surgery, and orthopedic surgery as well as critical care, nephrology, and infectious disease consultants. Depending on local standards of care, isolated extremity injury involving crush injury, compartment syndrome, or crush syndrome could be definitively managed at non-trauma-designated hospitals with access to specialty surgical and medical services, or could be stabilized and transferred to a referral hospital. Other than multisystem trauma and head injury, time-sensitive injuries associated with crush injury include uncontrollable external hemorrhage, vascular disruption or occlusion requiring surgical or interventional radiology vascular repair, and compartment syndrome requiring fasciotomy. Hyperbaric oxygen therapy has been used as part of the management of microvascular surgical repair of muscle flaps, partial traumatic amputations, vascular and ischemic muscle injury but its efficacy has only been demonstrated in skin grafts and flaps and chronic ischemic ulcers in patients with diabetes [11]. Hospital destination determination should be based on the availability of surgical and critical care resources rather than the availability of hyperbaric oxygen therapy.

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