COMPREHENSIVE REVIEW

Prehospital Management of Earthquake Casualties Buried Under Rubble

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Abbreviations:
ATLS = advanced trauma life support
ECG = electrocardiograph
IV = intravenous

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Abstract

Earthquakes continue to exact a heavy toll on life, injury, and loss of property. Survival of casualties extricated from under the rubble depends upon early medical interventions by emergency teams on site. The objective of this paper is to review the pertinent literature and to analyze the information as a practical guideline for the medical management of casualties accidentally buried alive.

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Introduction

Earthquakes are among the most dangerous and destructive types of natural events. Earthquakes strike suddenly and without warning. Ninety percent of casualties result directly from the collapse of buildings. Secondary events, such as landslides, floods, fires, and tsunamis, account for the remainder (10%) of the casualties.

The need for specialization when confronting these calamities has led to the formation of various search and rescue teams worldwide. During the last two decades, there has been an increasing number of publications relating to the experiences accumulated by these teams. The objective of this paper is to summarize the medical experiences of these teams and to offer a practical protocol for the treatment of casualties that have been buried alive for both trained paramedics and other medical personnel responding to similar calamities.

Medical personnel working in search and rescue teams usually specialize in areas other than disaster medicine, such as emergency medicine, anesthesia, intensive care, surgery, and trauma medicine. Their professional life only is devoted episodically to disaster medicine. A protocol of treatment should accommodate itself as much as possible to treatment protocols proven in previous trauma scenarios. The Advanced Trauma Life Support (ATLS) protocol was selected as the basis of treatment because it is the standard of trauma management that is taught in many countries.[1] Whenever possible, a primary survey and resuscitation should be carried out. Nevertheless, medical personnel should be aware of the unique problems that earthquake casualties present. Most of the seriously wounded casualties are trapped under rubble. Both assessment and treatment are severely constrained by the confined surroundings in which the casualties are found. In all those extricated from under the rubble, crush injury should be suspected until proven otherwise. "Scoop and run" and the "golden hour" are terms that are not applicable in this setting. Nearby hospitals may have been devastated by the event or may be inaccessible. These and other

Primary Survey	Under Rubble	Just Extricated
Airway	Assume airway may be compromised	Assess
Breathing	Assume ventilation impaired secondary to dust and/or noxious gases inhalation and direct trauma	Assess
Circulation	Assume hypovolemia, crush injury	Assess
Disability	Assume neurologic examination incomplete	Assess
Exposure	Assume hypothermia, expose body parts only if deemed absolutely necessary for saving life	Expose and cover

Table 1—Assumptions made during primary survey

unique problems in the medical treatment that commonly arise during the extrication of earthquake casualties will be discussed.

Epidemiology

More than one million earthquakes occur each year, but major earthquakes, which cause massive devastation of life and property, have occurred on average once every three years worldwide.[2] Earthquakes exert their destructive effects during a period lasting 30 to 60 seconds. The first phase of community response is characterized by spontaneous rescue attempts by local survivors armed with simple tools, such as shovels, axes, and bare hands, who successfully extricate >90% of the trapped victims.[3-4] Death and injury rates are considerably higher among trapped as compared to non-trapped casualties.[5] Mortality increases with age, disability, and the degree of destruction of the structure in which the casualty was trapped.[6] Mortality also is related to the duration of entrapment.[3] Follow-up surveys of trapped victims rescued following the 1976 earthquake in Tangshan indicated that while survival was ≥80% for those extricated during the first three days, survival rates dropped to 19% and 7.4% of those extricated on the fourth and fifth day, respectively. The ratio between the incidences of death to injury varies between the different earthquakes, but averages one death to every three persons injured.[7] Overall survival among 1,892 victims extricated after the 1995 earthquake in Kobe, Japan was only 40%.[8]

The leading causes of injury are impact and crush by moving debris.[9-10] The most common injuries are fractures, contusions, abrasions, and lacerations. These account for >75% of the total number of injuries encountered by healthcare professionals. Less than 10% require major surgery.[9] In survivors, trauma to the extremities is the most common injury, especially the lower extremities.

It appears that a significant proportion of mortality and morbidity could be prevented by implementation of early and appropriate medical response at the disaster site. Studies of the major earthquakes in Southern Italy (1980), Armenia (1988), Costa Rica (1991), Turkey (1992), and Japan (1995) indicate that up to 20% of the deaths occur after extrication.[11-15] Of these, 13-40% possibly could have been prevented by early implementation of medical and/or surgical interventions such as proper airway control, limitation of blood loss, treatment of crush injury, and prevention of hypothermia. Common causes of preventable deaths found in these studies include crush injury, hemopneumothorax, and slow exsanguination.

Management

Primary evaluation of casualties under rubble

Medical evaluation of a trapped victim starts whenever contact with the victim is established.[16] Communication usually is only verbal and auditory at first, and later becomes visual. Physical contact allowing a medic/paramedic to proceed with a formal primary survey comes last, minutes to hours after the initial contact. Even when physical contact is established with the victim, a full primary evaluation may not be possible because of the structural environment in which the casualty has been trapped.

As in the ATLS approach, verbal assessment should concentrate on assessing the level of consciousness and events related to the injury. It must be realized that even if the casualty is fully conscious and cooperative, symptoms may mislead the medical team.[16] Prolonged crushing of a limb may lead to sensory loss while the victim complains of pain in other body regions that have sustained minor trauma. Crush injury should be suspected until proven otherwise. The injury-producing mechanism may shed light on the victim's actual physiologic state just as in any other trauma scenario. The assumptions that should be taken into consideration in the primary survey both before and after extrication of the trapped earthquake victim are summarized in Table 1. Differentiation between those casualties still trapped and those who have just been released stems from the restrictions imposed upon both primary survey and resuscitation whenever the casualty remains trapped.

Airway control and ventilatory management

Both airway obstruction and impaired ventilation lead to life-threatening hypoxemia, hypercarbia, and acidosis. As long as the casualty is trapped, there is danger of further airway compromise by dust inhalation (possibly related to the extrication procedures) and impairment of pulmonary gas exchange by lack of ambient oxygen, exposure to noxious gases, and restriction of chest wall movement.[16,17] Combinations of the above are common. The assumption that a victim who can talk is well-oxygenated, has a secure airway and an intact ventilatory drive is not valid as long as the victim remains trapped.

Chest trauma occurs often in earthquake victims. Of 487 patients referred to Kobe University Hospital after the Great Hanshin-Awaji Earthquake (1995), 12.9% suffered from chest injuries.[18] Most of these patients sustained mild chest injuries such as broken ribs and clavicles, superficial lacerations, and contusions. Only 25% of these victims required hospitalization. In the two earthquakes that

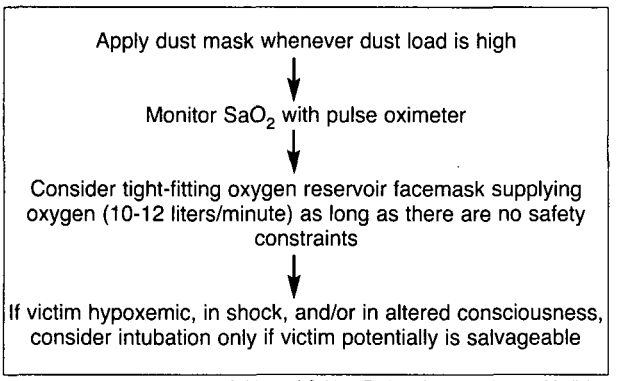

Figure 1—Airway and breathing—Primary resuscitation (SaO_2 = oxygen saturation)

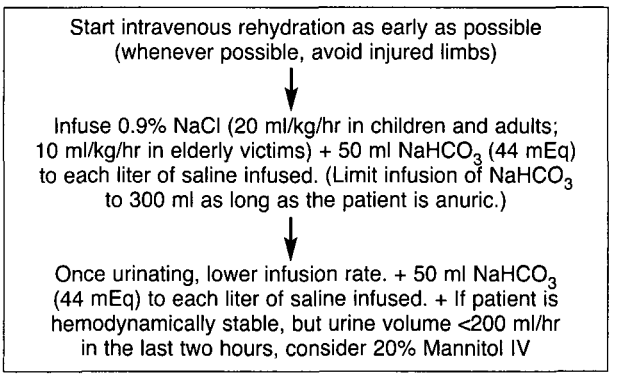

Figure 2—Rehydation protocol for victims entrapped under rubble

occurred in Turkey in 1999, thoracic injury was reported in 10% of those victims who were hospitalized.[19] Among these victims, pneumothorax and rib fractures were the two most frequent pathologies encountered.

Suffocation from earth used for walls or roofing material is a common cause of death in earthquake casualties.[3] Casualties buried inside structures built of reinforced concrete also are vulnerable to suffocation. Collapse of concrete, bricks, and adobe structures may raise a significant amount of dust.[16] Following building collapse, the dust load in the air within confined spaces may be overwhelming. Moreover, the activity of the search and rescue team may lead to resuspension of the dust in the air. Death due entirely to dust asphyxiation was reported in victims of the Great Tangshan Earthquake in 1976.[16]

Casualties trapped within confined surroundings may be exposed to air with a diminishing oxygen content.[16,17] Inhaled carbon monoxide, produced by fuel-powered rescue equipment being employed at the site, may aggravate already decreased tissue oxygenation. Exposure to other noxious gases should be assumed in sites that are not exclusively residential, such as factories, warehouses, laboratories, sewers, and commercial centers. These include a diverse group of compounds and waste products synthesized by the chemical industry. Some of these chemicals are characterized by high biological reactivity, and exposure may lead to acute respiratory distress.[20] Isocyanates, for example, are used in the plastics and pesticide industry. Bleaching solutions contain chlorine. Accidental inhalation of fumes produced by these compounds will cause severe respiratory irritation, which may culminate in pulmonary failure.[21–23]

The most basic procedure in protecting the airway of a casualty is to apply a simple dust mask (Figure 1). Casualties may resist placement of this mask due to a subjective feeling of suffocation when the mask is placed. An alternative strategy would be to place the mask only when the dust load around the victim is high.

The use of oxygen may be limited in the vicinity of rescue efforts since the oxygen source may explode if exposed to oil or discharges from the different machinery that is being used to free the casualty from entrapment. Still, whenever possible, a tight-fitting oxygen reservoir facemask should be applied that supplies nearly 100% oxygen at a flow rate of 10-12 liters/minute.

A pulse oximeter is best for monitoring the ventilation of the casualty since changes in oxygenation can fluctuate without clear-cut detectable clinical signs. Pulse oximetry is not always reliable. Profound anemia, hypothermia, and peripheral vasoconstriction impede accurate monitoring of oxygen saturation. Furthermore, pulse oximetry does not distinguish carboxyhemoglobin from oxyhemoglobin, which limits its use in carbon monoxide poisoning. Nevertheless, the pulse oximeter is an efficient piece of medical technology that should be taken to the scene of the damage. In most instances, the pulse oximeter allows continuous monitoring of the victim's most important physiological data: pulse and hemoglobin oxygen saturation.

According to the ATLS protocol, casualties who are hypoxemic, in shock, or have altered levels of consciousness must be intubated. Although hypoxemia may be ameliorated by the administration of oxygen, intubation, and ventilatory support may be required to treat hypercarbia and respiratory acidosis. Casualties suffering from massive dust inhalation will need positive pressure ventilation.[16] Of all the different methods available for securing the airway, orotracheal intubation is the most commonly used technique. Alternative methods include the insertion of an esophagotracheal Combitube® and the use of a laryngeal-mask airway. Use of any of these techniques requires prior experience.

A word of caution is necessary before proceeding with endotracheal intubation. Intubation is a difficult feat in confined surroundings and should not be used as liberally as in casualties who already have been extricated. Under the rubble, it is a dangerous procedure. The chances that an intubation attempt will fail are high. Moreover, failure to recognize that intubation has not succeeded also is substantial. This is due to difficult access and the hostile environment. Classically, intubation in trauma is a 3-person procedure during which one immobilizes the head and spine, another performs the Sellick maneuver, and the third performs the actual intubation. In restricted surroundings, access will determine how many rescuers actually can get

Under Rubble	Assume Hypovolemic
Oxygenation	
Induction of anaesthesia	Oxygenate only or Ketamine IV
Maintenance	Narcotics or/and Benzodiazepines IV

Table 2—Induction of anaesthesia in victims under rubble (IV = intravenous)

close enough to the casualty to perform the intubation. The trapped casualty in need of ventilatory support almost never is positioned in the neutral, supine position, which is best for intubation. Thus, before proceeding with intubation, alternative solutions should be considered. In the casualty who is apneic, oral intubation is the preferred mode of ventilatory support, since positive pressure ventilation is required for these patients. Whether an apneic casualty should undergo any kind of resuscitation under rubble depends on the circumstances. If the casualty only is partly comatose, with an intact gag reflex, sedation or anaesthesia is a pre-requisite for intubation. In this patient group, temporary maintenance of ventilation with a bag-valve-mask or other similar products is an alternative. Though these methods of ventilation are not as effective in reducing CO_2 accumulation as is positive pressure ventilation, they do allow temporary oxygenation without the risks associated with failed intubation.

Once the decision has been made to intubate, all of the equipment should be ready before the procedure is attempted. This includes: (1) a bag-valve device; (2) an appropriately sized mask; (3) an endotracheal tube with cuffs tested; (4) stylet; (5) laryngoscope; (6) oropharyngeal airway; (7) suction device; (8) high flow oxygen source; (9) 10 ml syringe; (10) electrocardiograph (ECG); (11) pulse oximeter; (12) intravenous access; (13) drugs; and (14) cricothyrotomy equipment. No other medical and rescue procedures should occur while intubation is attempted.

The most frequently used technique of intubation in trauma victims is rapid sequence induction (Tables 2 and 3). Whenever one attempts to intubate the casualty, one must differentiate between those who remain trapped and those who have just been extricated. Trapped casualties in need of intubation should be assumed to be hypovolemic. Whether the presence of head trauma should be assumed depends upon the mechanism of injury and the clinical signs.

For those victims who are not unconscious or who have an active gag reflex, sedation or anaesthesia is necessary to induce unconsciousness that enables intubation. The appropriate agent for induction depends upon two variables: (1) hemodynamic stability; and (2) suspected closed head injury (Table 3). If the victim is in shock, oxygenation prior to intubation may be all that is necessary. If pharmacological induction is considered, the three most commonly used drugs are: (1) thiopental; (2) etomidate; and (3) ketamine.[24] Thiopental reduces cerebral oxygen consumption, and it is commonly indicated in victims with a suspected

Just Extricated	Hypotensive		Normotensive	
	no head trauma	with head trauma	no head trauma	with head trauma
Oxygenation				
Induction of anaesthesia	Midazolam IV followed by Ketamine IV	Oxygenate only or Fentanyl IV	Midazolam IV followed by Ketamine IV or Fentanyl IV	Thiopental or Fentanyl IV
Maintenance	Narcotics or/and Benzodiazepines IV			

Table 3—Induction of anaesthesia in victims just extricated (IV = intravenous)

closed head injury. Its use is limited to normotensive victims, since it is a cardiac depressant and induces vasodilatation. Therefore, its use in patients under rubble should be questioned. Etomidate reduces cerebral oxygen consumption, as does thiopental. Unlike thiopental, it does not depress the myocardium and does not lower blood pressure. Etomidate is short acting. Apart from it being very expensive, a major disadvantage of the use of etomidate is that it may induce myoclonus.

Ketamine is a short-acting anaesthetic drug and has been suggested as the preferred drug for induction of anesthesia in primitive field conditions.[25] It does not depress the respiratory drive or reflexes. It increases the heart rate and blood pressure, a useful effect in hypovolemic patients. Ketamine's main disadvantages are that it increases tracheobronchial secretions, it increases both intracranial pressure and intraocular pressure, and causes hallucinations. The increase in intracranial pressure caused by Ketamine is of short duration. Furthermore, in resuscitating a hypovolemic victim with a possible head injury, increase in blood pressure actually may benefit the patient by increasing cerebral perfusion pressures. The psychomimetic effects of ketamine can be avoided by using this drug in conjunction with a benzodiazepine. Administration of midazolam followed two minutes later by ketamine has been suggested.[2]

If intubation is needed for a casualty who is hypotensive and is suspected to have sustained a closed head injury, fentanyl may be administered as an alternative since it provides good analgesia with minimal risk of hypotension in low doses. It is characterized by a rapid onset of sedation and analgesia.[27] Fentanyl also may attenuate the hemodynamic responses to laryngoscopy. Lidocaine is an alternative, which, when administered to hypotensive patients, ameliorates increases in intracranial pressure during intubation.[24,28] Its use also prevents bronchoconstriction.

Succinylcholine is the agent of choice for induced paralysis in most trauma scenarios in hospitals and its administration has been reported to be safe in prehospital care.[29,30] One should be cautious when choosing succinylcholine as the agent of choice in paralyzing victims trapped in collapsed buildings. Cell depolarization in normal patients may increase plasma concentrations of potassium by 0.5

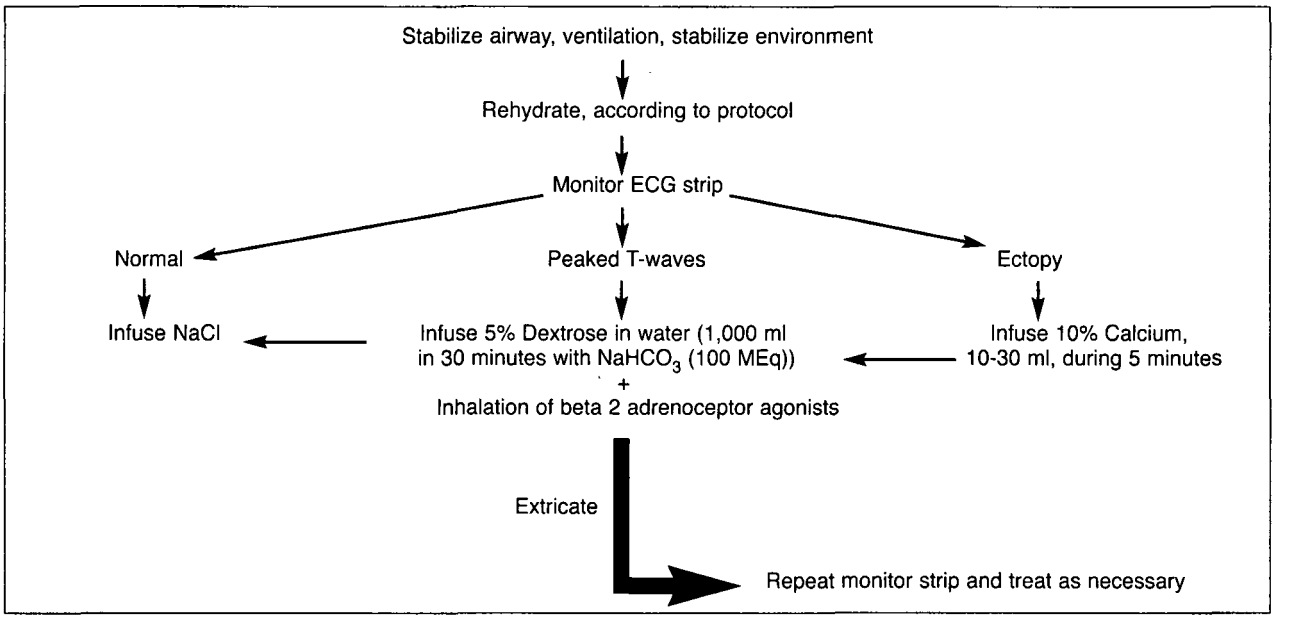

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Figure 3—Extrication protocol

mEq/dl.[24] Those with prolonged muscle denervations and burns may increase potassium levels, but these effects usually are not seen during the first 24 hours of injury. Still, the danger of hyperkalemia developing in extricated casualties suffering from crush injury makes succinylcholine a bad choice. Other problems with administration of this drug are fasciculations that may increase intraocular and intragastric pressures and may displace long-bone fractures. Concern over rises in intracranial pressure secondary to the use of succinylcholine has not been substantiated. In-hospital, if succinylcholine is believed to be relatively contraindicated, vecuronium may be used. It is longer acting, and it does not induce cardiovascular instability. It induces paralysis within 1–3 minutes that may last more than 30 minutes. Vecuronium has been used in mass-casualty situations.[26] Vecuronium has the added benefit of having a long shelf-life within a wide range of temperatures, and it may be reconstituted from powder when required. The main disadvantage with the use of vecuronium is that prolonged paralysis in patients under rubble is very dangerous. Accidental dislodgement of the endotracheal tube during efforts to extricate the victim is possible. Therefore, the administration of vecuronium should be avoided in the prehospital setting for either facilitation of intubation or for maintenance of ventilation. If maintenance of ventilation is desired, narcotics and benzodiazepines are the agents of choice.

Restriction of space may make traditional laryngoscopic intubation impractical.[16,17] Oral intubation alternatively may be achieved using a lighted stylet or tactile intubation. Other alternatives to oral intubation are nasal intubation, cricothyrotomy, and percutaneus, translaryngeal jet insufflation.[26]

Circulation and Crush Injury

After securing the airway, the primary survey should target possible sources of external hemorrhage that require prompt control. If possible, at least two peripheral intravenous lines should be inserted. Insertion of intravenous lines in traumatized extremities should be avoided.

Once external bleeding is controlled, the main treatment issues concerning the circulatory management of the casualty under rubble are internal bleeding and the sequelae of crush syndrome. The current trend in volume resuscitation of trauma victims in other scenarios has been to avoid aggressive volume resuscitation. Vigorous volume resuscitation has been condemned as a factor aggravating blood loss and increasing mortality. This has been confirmed in laboratory animals and in a prospective study of penetrating trauma in humans in urban scenarios.[32–34] The problem with the recommendation for withholding fluid resuscitation is that it takes into consideration that the trauma casualty will be transported promptly to a nearby hospital where definitive surgical treatment can be undertaken. In the case of the casualty buried under rubble, this is not a real possibility. Unlike trauma victims from other scenarios, trapped casualties may experience a lengthy delay from the onset of injuries until resuscitation efforts begin, making it almost impossible for anyone suffering from serious internal bleeding to survive.[17] Thus, once the trapped casualty has been reached, it may be assumed that aggressive volume resuscitation, which is indicated to ameliorate the systemic sequellae of crush injury, probably will not aggravate clinically significant internal bleeding.

The history of crush syndrome recently has been reviewed.[35] Crush injury was first described by Larrey in 1812, the lead military physician in Napoleon's army.[36] Larrey described the appearance of gangrene in the skin and the muscles in dependent areas in soldiers who were comatose due to carbon monoxide poisoning. It is fascinating that in the first scientific description of crush injury, the etiology of "crushing" of tissues was not caused by actual crushing of the soft tissue by external objects, but by gravitational forces acting on the immobilized body. Bywaters *et al* provided the first modern descriptions of crush syndrome in their classic studies of the London Blitz in the autumn of 1940.[37–40] They showed that all of those sur-

vivors extricated from the rubble who suffered extensive muscle crush injury ultimately developed renal failure and died. Autopsies showed widespread muscle necrosis and acute myoglobinuric renal failure. In an experiment considered to be a landmark in the elucidation of the pathophysiology of crush injury, Bywaters *et al* infused myoglobin in rabbits.[41] Rabbits having acidic urine developed renal failure, while rabbits with alkaline urine did not develop nephropathy. The results of this experiment serve as the basis for the current treatment plan for suspected crush injury that recommends the addition of alkaline fluid to volume resuscitation with the aim of alkalinizing the urine and preventing acute myoglobinuric renal failure.

Previous reports have suggested ischemia as the primary insult in the crushed muscle, but crush injury and its systemic sequelae have been shown to occur in extremities with a normal blood supply. It currently is believed that crush injury results from increased sarcrolemmal stretch rather than from ischemia.[42] It is believed that increased stretch in the muscle interferes with the selective impermeability of the sarcolemma. This causes influx of sodium, water, and extracellular calcium down their electro-chemical gradients from the extracellular fluid into the sarcoplasm. The cationic extrusion pumps, operating under maximal capacity, cannot cope with this influx and rapidly deplete the intracellular energy stores. The end result is cellular swelling and an increase in intracellular calcium that disrupts cellular function and mitochondrial respiration, leading rapidly to myocytic death. Muscle swelling within non-compliant fascia may lead to a devastating compartment syndrome that may develop hours and sometimes days following extrication. This may not become obvious until well after the victim has been extricated.[43]

The systemic squellae of crush injury result from the death of muscle cells and leak of intracellular metabolites into the systemic circulation.[44] Common metabolic derangements include hyperkalemia, hypocalcemia, hyperphosphatemia, metabolic acidosis, myoglobinemia, and myoglobinuria. Hyperkalemia, aggravated by hypovolemia and hypocalcemia, may cause cardiac arrhythmias that potentially are lethal.[40] Lethal hyperkalemia, an early sequel of crush injury, may occur before acute renal failure and without obvious clinical signs of compartment syndrome. The approach to hyperkalemia is described below. Crush injury has been reported to appear in up to 40% of survivors extricated from under collapsed buildings.[45] As many as half of these survivors will develop renal failure and at least 50% of the latter will require dialysis.

The actual proportion of those suffering from crush injury compared to the rest of the wounded is unknown. The Mexico City earthquake of 1985 resulted in >3,000 deaths and >30,000 persons were injured. Acute renal failure, however, was not mentioned as a major clinical concern.[46] In Armenia, the 1988 earthquake caused 25,000 deaths and 100,000 persons were reported injured. At least 600 casualties were reported as suffering from acute renal failure requiring dialysis.[47] This amounts to <0.5% of the total number of casualties. Similarly, only one case of acute renal failure was identified due to the 1989 earthquake in San Francisco, and no case was reported from the 1990 earthquake in the Philippines in which 1,500 people died.[46] Acute renal failure was reported after the 1990 earthquake in Iran in which 13,888 persons died and 33,616 needed hospitalization, with 156 patients nationwide (0.46% of those hospitalized) requiring dialysis.[48] In the Great Hanshin-Awaji earthquake in 1995, 6,308 were killed and 35,000 persons were injured.[49] Of these, 6,107 were hospitalized. 372 (6.1%) hospitalized patients were identified as suffering from crush injury, of whom 202 (3.3%) suffered from renal failure, but only 123 (2.0%) needed hemodyalysis.[50] Fifty of these victims died (0.8% of hospitalized patients and 13.4% of those sustaining crush injury). In the 1999 earthquake in Marmara, Turkey, a survey conducted by the Turkish Society of Nephrology Task Force identified 639 victims with acute renal failure due to crush injury.[51] Of these, 477 required dialysis. From these numbers, one can extrapolate that the incidence of renal failure due to crush injury is approximately 1% of hospitalized patients of whom more than half will end up requiring dialysis.

Experience from Japan shows that major risk factors for renal failure are massive muscle damage (two or more extremities crushed), insufficient initial fluid resuscitation (<10,000 ml/2 days), and a long lag time between the onset of trauma and arrival at the hospital.[50,52]

As described, the Marmara earthquake produced a high incidence of renal problems and the need for dialysis support. Still, the incidence of renal failure in children younger than 10 years of age and in victims older than 60 years of age was significantly lower compared with the resident population of the affected area.[53] Of 639 patients who suffered from acute renal failure following the Marmara earthquake, 71% were young adults within the range of 16–45 years.[51] Infants seem to be relatively immune to the development of severe acute renal failure after suffering from crush injury. Of patients hospitalized due to musculoskeletal trauma up to 20% were under the age of 16 years.[54] Though most of these children sustained the crush syndrome, only 18.5% ended up needing hemodialysis. In comparison, up to 93% of adult victims with crush injury required hemodialysis. Thus, although the types of injuries seen in children were similar to those in adults, the consequences of these injuries were very different as far as crush injury leading to acute renal failure is concerned. The relative immunity of children and the elderly may be secondary to the fact that their muscle mass is smaller than is that of young adults.

Crush injury should be suspected in any victim who either has had trapped parts of the body compressed or has layed immobile against a hard surface for several hours.[55] The main indicator hinting at the possibility of crush injury is the mechanism of trauma. The major sites of crush injury reported are the lower extremities, followed far behind by the upper extremities and trunk.[50] Extremities suffering from crush injury may have loss of sensation and flaccid paralysis. Pelvic fractures, limb fractures and abdominal injuries frequently are associated. Crush injury and appropriate treatment should be considered in all survivors who were buried under rubble for >2 hours or in those buried for a shorter time but with evidence of exten-

sive damage to the limbs.

Adequate intravenous access should be secured as soon as the casualty is reached (Figure 2). Prompt deployment of intravenous access is especially important in the casualty who has been trapped for a long time and is suspected of suffering from crush injury. If possible, intravenous volume resuscitation should be started prior to extrication. Once the casualty is extricated, areas of the body previously entrapped will be reperfused, allowing solutes to be released from injured tissue, such as potassium and myoglobin, to leak into the systemic circulation and to exert their toxic effects on the heart and later on the kidneys.[45] Volume resuscitation is the mainstay of treatment for shock, hypovolemia, and the metabolic derangements commonly presents in casualties suffering from crush injury.[56] It must be stressed that a volume equivalent to the entire extracellular fluid volume may be sequestered into the crushed muscles within a single day. This severe intravascular hypovolemia may be aggravated by vasodilatation due to excessive production of nitric oxide.[57]

Once adequate intravenous access has been achieved, normal saline (0.9% NaCl) is infused to replenish intravascular volume. Lactated Ringer's solution contains potassium, which although in small concentration, its use may add unnecessarily to the existing potassium load. Up to 10 liters of intravenous crystalloid fluid may be needed to counteract the hemodynamic instability in some casualties after extrication. An initial infusion rate of 1 to 1.5 L of saline per hour (20 ml/kg/hr in adults and children, 10 ml/kg/hr in elderly victims) is recommended. The amount of fluid infused before and after extrication should be monitored so to avoid unnecessary volume overload, especially in older individuals. Urine volume can be monitored using an intravesical catheter. Alternately, simple verification of urine output may be an acceptable compromise in an environment where each invasive procedure may become complicated by infection.

This recommendation for fluid resuscitation has been criticized as being excessive.[58] Yet, not only are these victims dehydrated, tremendous amounts of fluids will be sequestered into injured muscles once they are freed from the pressure imposed upon them. Early rehydration is the best method for avoiding acute renal failure and the need for dialysis. The aim, therefore, is not only to achieve the desired level of hydration, but also to reach the rate of diuresis necessary to eliminate the toxic metabolites of myoglobin. Still, the aggressiveness of fluid rehydration should be dependent on the caregiver's ability to monitor the victim. Full rehydration should be provided only in those victims in whom pulse, blood pressure, urinary output, oxygen saturation, ventilatory rate, and chest auscultation can be continuously assessed.

The addition of bicarbonate to the intravenous solution will avoid precipitation of toxic myoglobin metabolites in nephrons.[38,59] The administration of bicarbonate also will ameliorate acidosis and hyperkalemia. Potassium levels will decline within one hour of administration.[60] Although great caution should be exercised in the administration of bicarbonate to avoid both iatrogenic metabolic alkalosis and sodium overload, addition of up to 300 mEq of bicarbonate per day potentially is more beneficial than dangerous, even in the anuric patient. Bicarbonate solutions include isotonic concentrations of sodium and water. Sodium and water retention with secondary edema may occur during therapy whenever saline and bicarbonate are administered in large doses. Casualties with pre-existing renal insufficiency or congestive heart failure are predisposed to develop pulmonary edema. In addition, the need to store bicarbonate solutions at 15–30°C should be taken into account.

Mannitol is an osmotic diuretic that, when used judiciously, may have an advantageous impact on the treatment of casualties suspected of suffering from crush injury. It is an important adjunct to the administration of crystalloids and bicarbonate in the treatment of crush injury. Mannitol may help prevent acute renal failure secondary to myoglobinuria by flushing of myoglobin casts from the tubules and increasing the elimination of myoglobin by the kidney.[61] If rehydration using crystalloid solutions is unsuccessful in achieving adequate diuresis within four hours (urinary output remains <2 ml/kg/hour), mannitol 20% solution 0.25 g/kg, is administered intravenously over 10–30 minutes.[62] Onset of diuresis usually will occur within 15–30 minutes. If urine volume diminishes again, the patient should be assumed to be dehydrated. Only after aggressive rehydration should a second, similar dose of mannitol be administered. The maximal dose recommended per day is 2 g/kg/day (no more than 200 g per day). Mannitol is contraindicated in casualties with established anuric renal failure and is relatively contraindicated in victims with heart failure. In patients with heart failure, a sudden increase in effective blood volume induced by the hyperosmolar effect of mannitol may lead to pulmonary edema. If the casualty has received plenty of fluids but has not started to pass urine, a small single test dose of 0.25 g/kg of mannitol may be given with the hope of initiating diuresis.[62] Because of the possible dangers associated in the injudicious administration of mannitol, only personnel experienced with the use of this hypertonic solution should be allowed to administer this solution in the prehospital setting.

Mannitol has extra beneficial renal effects as well. Mannitol has been shown to facilitate decompression of experimental compartment syndrome in a dog model and a few case-reports suggest that it may be beneficial in humans as well.[63,64] This may be due to its hyperosmotic properties, which allow fluid to be mobilized from edematous tissues. Mannitol may delay or even preempt fasciotomy. Whether this is applicable to patients extricated under rubble is unknown. Mannitol also may act as a scavenger of free radicals produced in the muscle by reperfusion, thus limiting ongoing muscle necrosis.[65] Another beneficial effect of mannitol is its positive inotropic effect on the heart.[66]

Disability

Many entrapped victims will suffer from traumatic injuries caused by the impact of falling objects. Head injuries from falling objects are common.[3,10] The most important indicator is an altered loss of consciousness.

In general, guidelines for treatment of patients with a head injury are not different to those for head trauma casualties from other trauma scenarios. Still, treatment possibilities in an earthquake scenario are limited by the confined surroundings in which the casualties are trapped. Even though intubation and controlled ventilation are indicated as the initial treatment for the unresponsive or the combative victim with a head injury, the use of orotracheal intubation under rubble should be restricted because of the high rate of failure to intubate the trachea. Medical treatment in the field should focus on avoiding hypoxemia and hypotension, which may increase mortality rates by more than 50%. If the victim is combative, ketamine, benzodiazepines, or fentanyl may be used for sedation. Administration of Pentobarbital is limited to normotensive victims. However, the victim under rubble should be assumed to be severely dehydrated. During volume resuscitation, glucose-containing solutions should be avoided. As stated above, NaCl is preferred to Ringer's Lactate. The victim's spinal column is stabilized with the aid of a backboard and a cervical collar. The goals of prehospital therapy in the victim with severe head injury are: (1) mean blood pressure (BP) 90–110 mmHg; and (2) SaO_2 = 100%.

In the prehospital setting, intracranial pressure usually is monitored using clinical signs. These include: (1) deteriorating level of consciousness; (2) lateralizing signs; and (3) convulsions. Of these, development of convulsions is the only reliable clinical sign of an acute head injury. If the level of consciousness in a casualty under rubble deteriorates, and the casualty is suspected of having suffered a head injury, the sequence of treatment is: (1) sedation using a narcotic agent; (2) oxygenation; and (3) intubation and hyperventilation to pCO_2 levels of 28-30 mmHg only if the surroundings allow relatively safe intubation. If seizures occur, these are treated with the administration of intravenous diazepam, 10 mg intravenously. Further measures to reduce intracranial pressure once seizures appear are: (1) intravenous mannitol, 25–50 g every 4 hours; and (2) intravenous furosemide, 20–40 mg every 4 hours. If the trapped victim convulses several times, the administration of intravenous pentobarbital (up to 10 mg/kg) over 1 hour, and then 1 mg/kg/hr is administered to the well-hydrated victim. Anticonvulsant prophylaxis with phenytoin should not be given to a victim still under rubble.

Head trauma casualties also should be suspected of having sustained spine injuries. Spine injuries also are common in earthquake victims without head injuries. Spine injury has been reported both in casualties who were standing up and those lying down when the collapse occurred.[3] Spine injuries most often have been associated with structures built with rigid floors and roof slabs.

Exposure and Hypothermia

The primary survey ends with exposure. Expose body parts only if deemed absolutely necessary for saving life. A secondary survey then is conducted that is aimed at identifying possible life-threatening injuries. Once the immediate medical treatment allows, the entrapped victim should be covered to avoid the development of hypothermia.

Hypothermia is a major factor threatening trauma patients in all scenarios. Hypothermia has both protective and harmful effects. At temperatures of 32–33°C,[67] it may limit neuronal damage after head injury, but it has a negative effect on both metabolic and hemostatic functions. Oxygen consumption may increase, platelet activation is inhibited, and the actions of clotting enzymes are inhibited.[68–70] Both platelet function and clotting enzyme activity diminish considerably at body temperatures below 34°C.[71] Overall, hypothermia is an independent risk factor for early mortality following major trauma.[72] Hypothermia is a frequent event in trauma patients and may occur despite high environmental temperatures, and entrapment is a major risk factor for developing hypothermia.[73,74] Age is another risk factor for the development of hypothermia. Once hypothermia develops, it is difficult to correct. Efforts to prevent and treat hypothermia should be started as early as possible.[75] The relative effectiveness of different methods of active rewarming during the prehospital stages of treatment has been studied.[76] In this study, only patients treated with hot packs showed an increase in body temperature. This increase has been demonstrated in all patients treated with this modality. The use of passive rewarming, reflective blankets, and warmed intravenous fluids has not prevented a decrease in body temperature.

Other Injuries

In 1906, many of the houses in San Francisco affected by the earthquake actually were destroyed by fire. Fires are considered secondary events and may be caused by explosions triggered by damaged gas pipes and electrical lines. The actual incidence of burns in earthquake casualties during the last two decades has been less than expected. In the earthquake that struck Kobe in 1995, 504 deaths were listed as fire-related.[77] Most of the victims probably were crushed or suffocated before they were burned. Only 1.9% of those hospitalized had burns and most involved <20% of total body surface area. No relationship was noted between the number of burned houses and the number of burn patients hospitalized.

Much more common is the vulnerability of the entrapped casualty to noise and dust. The latter may damage the eyesight of the casualty. Casualties should be given occular and aural protection as early as possible.[16] Open wounds should be cleaned and dressed to prevent further wound contamination. Intravenous antibiotic coverage with a second generation cephalosporin and intramuscular tetanus toxoid, 0.5 ml should be administered once all other life-saving treatments have been completed.

Extrication

Ideally, extrication should follow initiation of the primary survey and resuscitation. However, both the victim and the rescue team remain exposed to an unstable environment that endangers them. This includes unstable construction debris that at any moment may collapse and bury both the survivors and rescuers. It also may include fires and an atmospheric methane content of 5% to 15% that may lead to an explosion. In these circumstances, removing the casualty and the rescue team from immediate danger assumes the highest priority.

If the environment is stable, airway control, proper ventilation, and rehydration should be carried out (Figure 3). After proper oxygenation and primary volume replacement, careful extrication is carried out. Whenever possible, cervical spine stabilization and maintaining in-line position of the entire spine on a backboard also should be undertaken before extrication. Long-bone fractures should be stabilized for pain control during extrication when the victim is not immediately life-threatened.

Hyperkalemia is an early cause of death in casualties just extricated from under rubble. Once circulation to previously crushed limbs is restored, potassium is released by dead tissues into the circulation. Identification and treatment of hyperkalemia should be part of any protocol of treatment during and immediately after extrication. Retrospective analysis of casualties suffering from crush injuries shows that serum potassium concentration >6.5 mEq/l is common immediately after extrication and affects up to 22.7% of those victims who eventually demonstrate deterioration in renal function.[78] Increased potassium serum levels are found mainly in adult male victims with severe soft-tissue trauma.[78,79] Hyperkalemia in children recently extricated from under rubble is rare, affecting relatively few of those with established renal failure.[80]

Monitoring of serum potassium levels during extrication may be improvised by using a portable cardiac monitor. Though not as sensitive as a complete electrocardiogram, cardiac monitoring allows comparison between pre-extrication to post-extrication rhythm strips and follow-up thereafter. The first evidence of elevated serum potassium levels is the appearance of a peaked T-wave usually present in precordial leads. This pattern is characteristic of mild to moderate hyperkalemia (<8 mmol/L), but also may be seen in normal slender individuals without hyperkalemia. With further elevation of potassium levels, the atrial complex disappears and the QRS complex widens until it blends with the peaked T-wave. With widening, the configuration of the QRS complex becomes bizarre, simulating a sine wave. At this stage, one must assume the presence of severe hyperkalemia, which can procede to ventricular fibrillation or asystole.

If hyperkalemia is suspected, it should be treated immediately and vigorously. If the cardiac monitor is the only method of monitoring potassium levels, treatment should be considered once peaked T-waves are recorded. As discussed above, 100-150 mEq of bicarbonate can be administered intravenously, and 1 liter of 4.3% dextrose in saline can be administered peripherally to non-diabetic patients. Alkalemia and glucose, together with endogenously produced insulin, will cause potassium to shift back into the cells. This treatment may reduce serum potassium levels as much as 2 mmol/L for several hours.[61]

Another possible alternative for the treatment of hyperkalemia is the administration of inhaled antiasthmatic, beta-2 adrenoceptor agonists, such as ventolin and salbutamol.[81] Serum potassium should decrease up to 0.5 mmol/L within 75 to 90 minutes following one inhalation.

If the atrial complex disappears and the QRS complex widens, hyperkalemia is severe and should be treated immediately. In such a case, appropriate treatment consists of intravenous 50 ml dextrose, 50%, with 10 units of short-acting insulin added.

Although calcium infusion is advocated to prevent cardiac toxicity secondary to hyperkalemia, delaying calcium infusion until ectopy appears is suggested.[61] There is no proof that calcium is beneficial in a casualty suffering from hyperkalmia and extensive muscle damage. Infused calcium rapidly deposits in injured muscle tissue and calcium has been hypothesized to be a mediator of cell injury.[82,83] It is unclear how much of the infused calcium will stay within the circulation to counteract the cardiotoxicity of hyperkalemia. Still, one should weigh the potential benefits of calcium upon survival against possible adverse effects on damaged muscle tissue. Though the casualty under rubble may suffer from muscle damage, changes in potassium levels may be extreme once the patient is extricated. Proponents of calcium treatment may argue that waiting for ectopic ventricular complexes to appear before administering calcium may prove to be too late. If deemed necessary, 10 to 30 ml of 10% calcium gluconate are infused intravenously within 1 to 5 minutes under constant cardiac monitoring.[61] Calcium almost immediately counteracts the adverse effects of potassium on neuromuscular membranes. When the calcium infusion is stopped, serum calcium rapidly falls to its previous level.[82] Since the effect of calcium infusion is transient, alternative strategies meant to lower serum potassium levels should be instituted.

Pain control is an important aspect that facilitates extrication.[16] Different methods to control pain include the use of narcotics, ketamine, and local blocks. Morphine is very efficacious in achieving pain control. It can be titrated according to the victim's needs. In this setting, morphine should be administered intravenously only. The response to intramuscular morphine is unpredictable especially in the hypovolemic victim whose intravascular volume has been diverted from the periphery to essential organs. Once the intravascular volume is replenished, large amounts of morphine, repeatedly given intramuscularly to allow proper pain control, may be rapidly absorbed into the systemic circulation, leading to both central nervous system and respiratory depression. Respiratory depression may be difficult to monitor in the confined environment in which the victim is trapped. Morphine provided intravenously will control pain for 1 to 2 hours. In this setting, the major drawback with the administration of morphine is its tendency to cause hypotension, which may be very severe in the volume-depleted victim. Naloxone, an opiate antagonist, administered intravenously will promptly reverse morphine-induced hypotension.

In the prehospital setting, Ketamine is a very efficient analgesic. Ketamine provides profound sedation, analgesia, and amnesia while maintaining spontaneous ventilation and preserving the gag reflex. Similar levels of analgesia would necessitate intubation and ventilation with most other classes of agents. Repeated intravenous doses of 0.2 mg/kg are administered for maintenance of analgesia.

Another option for pain control is the use of local blocks. Injection of local blocks requires previous expertise. The appropriate technique of administrating local blocks

in different areas of the body is beyond the scope of this paper and the reader is referred to textbooks specializing in this topic. Local blocks will facilitate the extraction of the entrapped limb.

Evacuation

On 17 January 1995, the city of Kobe and its vicinity experienced an earthquake with a magnitude of 7.2 on the Richter scale.[49] A total of 6,308 people were killed and >35,000 were injured. More than 400,000 houses and buildings were damaged. Many roads, highways, bridges, and railways were destroyed. Telephone lines either were disconnected or overloaded.

Very few casualties were transported to hospitals outside of the disaster area on the first day of the disaster.[8] Most of the casualties were transported to hospitals that were damaged by the earthquake. Mortality rates for those hospitalized in affected hospitals was considerably higher than they were for those hospitalized in non-affected hospitals, 8% vs. 3% respectively.[84] Damage to hospitals within the disaster area should be expected and considered within the medical evacuation plan.[25]

What to Expect

The medical team on-site will encounter two waves of injured victims. The first wave will represent the "walking wounded". These include a large number of people who have been hurt by falling objects or victims trapped by light debris and who have been promptly rescued by their family members and neighbors. Most often, they will suffer from contusions, lacerations, fractures, strains, mild head injury, and/or soft tissue foreign bodies such as broken glass. Wound care, tetanus toxoid, antibiotics, and analgesics probably will be the most commonly needed treatment. Still, "walking wounded" is a misnomer, and the term "first wave of casualties" should be used. A few of the casualties encountered in this wave will suffer from potentially life-threatening injuries such as crush injury, pneumothorax, abdominal injury, and/or pelvic injury. A few others may be threatened by conditions that are unrelated to the earthquake itself, such as loss of important medication, acute coronary syndrome, or labor. Any medical team deployed following a catastrophic earthquake should take these possibilities into consideration in their preparations. The second wave of casualties includes those who are buried deeply under structural debris. These casualties will be extricated usually within hours to days of the primary injury. The primary concern with the second wave is the complexity of their medical treatment and not their numbers, since unlike those in the first wave, these casualties will be rescued one-by-one.

Appropriate and dignified handling of the dead is another issue.[85] Identification and establishing the cause of death most readily are done at the disaster site. This information regarding a dead victim should be meticulously documented. The exact site from which the body is removed is one of the most important clues to the dead victim's identification. The victim should have some form of tag attached with identifying information. Whenever possible, transportation of the dead to temporary storage facilities should be planned in advance to avoid an unnecessary burden on the site of the search and rescue operations.

Members of the search and rescue team also are the responsibility of the medical team.[16,17] Responsibility includes prophylactic immunizations and medical treatment as indicated by the setting of the disaster. Search and rescue personnel should be monitored for adequate rest, hydration, caloric intake, and signs of excessive stress. Rescue workers, particularly those with more catastrophic exposure, are at risk for symptomatic chronic stress reactions.[86] Blisters, abrasions, or other minor problems that may incapacitate the team workers must be treated. Rescue dogs are an integral part of some teams, and knowledge with basic veterinary care is a prerequisite.

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