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НЕОТЛОЖНАЯ ХИРУРГИЯ БОЕВЫХ ПОВРЕЖДЕНИЙ

Учебно-методическое пособие по военно-полевой хирургии для студентов 5 курса факультета по подготовке специалистов для зарубежных стран медицинских вузов

EMERGENCY SURGERY OF MILITARY TRAUMAS

Teaching workbook on Combat Surgery for 5th year students of the faculty on preparation of experts for foreign countries of medical highest educational institutions

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I. ORGANIZATION OF SURGICAL CARE IN ARMY AND IN EMERGENCY SITUATIONS

1. Definition and content of military surgery

The ultimate goals of combat medicine are the return of the greatest possible number of soldiers to combat and the preservation of life, limb, and eyesight in those who must be evacuated. In consideration of the factors related to organization for care of solitary urgent trauma patients where every available resource may be utilized to preserve life and function, to the mass situation, where the high numbers of injured overcome available resources.

2. Main stages of military surgery development

Ambulances or dedicated vehicles for the purpose of carrying injured persons were first used by Spanish soldiers in 1487 during the siege of Malaga.

French military surgeon Ambroise Paré (1510–1590) pioneered modern battlefield wound treatment. His two main contributions to battlefield medicine are the use of dressing to treat wounds and the use of ligature to stop bleeding during amputation. While running out of boiling oil, which was used at that time for wound cauterization and sterilization. Pare learned that cold, not heat, is more beneficial for the control of wound bleeding. Pare is also credited with inventing artery forceps. In 1536, Ambroise Pare rediscovered Celsus principles: amputation through viable tissue and the use of the ligatures.

In 1588 William Cloves described the first successful above-knee amputation and, in 1593. Fabry, in his monograph on gangrene, reported the first amputation through the thigh. The introduction of Morel's tourniquet in 1674 (old name — the Spanish windlass) and Petil's tourniquet in 1718 were significant steps in the control of hemorrhage.

The Napoleonic Wars led to further improvements in battlefield surgery. Jean Dominique de Larrey, of France, is considered one of the founders of military medicine. Napoleon's surgeon, Baron Dominique-Jean Larrey, is often credited with establishing the ambulance team, as well as, the modern battlefield triage system that set the priorities for evacuation. During Napoleonic warfare; the larger force was often victorious. Hence, the highest priority patients were the ones that could be returned to duty quickly. Often, the severely wounded were left on the battlefield until the conclusion of the battle. Larrey altered the established pattern of the times by going forward with his ambulances (horse-drawn carts called «flying ambulances») to evacuate the wounded who could potentially survive with surgical care (often amputations) and medical supplies into the field of battle.

The introduction of ether anesthesia in 1846 and the subsequent development of antiseptics led to more precise surgery and a lower risk of wound infection.

3. N. Pirogov — the founder of scientific military surgery

Nikolai Ivanovich Pirogov (1801–1881), the most renowned military surgeon in Russian history, performed hundreds of amputations during the Crimean War (1853–1856), in which France, the UK, the Kingdom of Sardinia, and the Ottoman Empire fought against Russia. In 1847, Nikolai Pirogov was one of the first surgeons to use ether as an anaesthetic, as well as the very first surgeon to use anaesthesia in a field operation. The «Pirogov amputation» is a surgical salvage procedure for the complex injuries of the forefoot. Pirogov also introduced nurses on the battlefield and published the Atlas of Cross-sectional Anatomy based on sawed frozen sections. He noted that «war is an epidemic of injuries».

4. Role of surgeons in a modern system organization of evacuation and levels of medical care

During the U. S. Civil War, Charles Tripler and Jonathan Letterman further developed Larrey's principles by formally establishing a military ambulance service and forward aid stations. Also during the American Civil War general anesthesia was available for the surgeons and became common.

Early in the twentieth century, technological revolutions in warfare (machine guns, chemical and trench warfare) speeded evolution in medicine — as well as the ability to care for casualties (patients). World War I (1914–1918) brought artillery into the battlefield, which caused over seven million deaths, 19 million wounded, half a million amputations, and additional experience in the care of wounded soldiers.

The first practical method for transporting blood was introduced by Norman Bethune during the Spanish Civil War.

During WWI, thousands casualties quickly overcame the capabilities of hospitals designed for hundreds. The establishment of fully equipped and mobile field hospitals such as the Mobile Army Surgical Hospital was first practiced by the United States in World War II. It was succeeded in 2006 by the Combat Support Hospital.

WWII with its global nature and warfare advancements such as tanks and air support stretched the capabilities of the medical system, especially in transporting the wounded. World War II (1939–1945) was plagued with heavy civilian casualties from massive aerial bombardments. The use of helicopters as ambulances, or MEDEVACs was first practiced in Burma in 1944. The first MEDEVAC under fire was done in Manila in 1945 where over 70 troops were extracted by five helicopters, one and two at a time. With the advent of aeromedical transportation during the Korean War, the transportation times decreased from 12 hours in WWII to 4 hours in Korea.

5. Development of military surgery in postwar (WW II) period

The use of modern medical support (blood and plasma transfusions and antibiotics) as well as surgical advances (arterial repair), early evacuation, and better splinting made the salvage of many limbs possible. While the mortality rates of the subsequent wars have significantly decreased, the amputation rate has remained high (13 %), which is quite likely due to more destructive weapons. This allows medicine and treatment to be administered as soon as possible in the field and during extraction.

Unlike the Persian Gulf War of 1991, in which prewar casualty estimates far exceeded the actual number of casualties sustained, Operations Iraqi Freedom have generated casualties in the largest numbers the United States military has sustained since the Vietnam War.

6. Perspectives and future of combat surgical care

The more efficient evacuation system required a change in phylosophy of battlefield casualty care — from buddy care (in WWII) to standardized prehospital battlefield trauma care in the current Global War on Terror (such as Operation Iraqi Freedom). In low-intensity urban conflict, it is difficult to identify a casualty and get immediate qualified care. Thus, there is increasing reliance on self- and buddy aid for point-of-wounding care. Dispersed, low-density conflict also creates problems with access to and exit from the tactical environment. When a combat medic can get to the casualty, interventions must be focused and effective.

The era of global terrorism and asymmetric warfare proclaimed by the September 11, 2001 attacks on the United States has continued with the Bali bombings and the Madrid and London train bombings (and other smaller terrorist events too numerous to list here). These types of incidents blur the traditional lines between civilian and military trauma victims. In addition, national and international natural disasters, such as Hurricane Katrina and the Asian tsunami in December 2004, have created intense focus on the medical community preparation for such events. The lessons learned by physicians in the theaters of war, particularly regarding the response to mass casualties, blast and fragmentation injuries, and resuscitation of casualties in poore environments. It is critical impotent to share these valuable lessons with civilian colleagues.

7. Modern combat trauma

The mechanisms of injury in conventional land warfare: penetrating, blast, blunt, and thermal.

Weapons of conventional war can be divided in to ***explosive munitions and small arms.***

Explosive munitions: artillery, grenades, mortars, bombs, and hand grenades.

Small arms: pistols, rifles, and machine guns.

These are the result of the high energy of wounding agents, multiple causes of wounding, (fragment, bullet, blast injury, penetrating and blunt, primary and multiple, combined injury).

While a blunt mechanism is the most important source of injury for civilians, trauma inflicted during combat is overwhelmingly penetrating in nature.

8. General characteristics of modern weapons

Wound ballistics is the same and often injuries are due to the same projectiles used 35 years ago in Vietnam. The ghastly penetrating wounds, blast trauma, and burns produced by present day conventional and improvised weapons are essentially unchanged from those produced in the last half of the 20th century. The automatic rifle, rocket-propelled grenade, mortar, and improvised explosive item are widely available, easy to obtain, simple to use and fatal, and not confined to the arsenals of disciplined soldiers. Bearers of these arms today include suicidal fanatics, women, and children.

Another characteristic to modern explosive munitions design is that the individual munitions are frequently clustered (i.e., packaged together) in a carrier (a bomb, shell, or rocket) for delivery to the enemy position. The individual submunitions are disseminated from their carrier before being detonated. Such cluster munitions greatly increase the casualty-generating potential of power.

Latest trends in military surgery are to deal with the large number of horrendous lower limb and pelvic injuries resulting from the increased use of Improvised Explosive Devices (IEDs) by the Insurgents in Iraq and Afghanistan. These produce a new type of high energy injury characterized by massive contaminated bone and soft tissue destruction combined with major blood loss.

9. Influence of modern gunshot wounds on medical care organization

The traditional approach to combat injury care is surgical exploration with definitive repair of all injuries. This approach is successful when there are a limited number of injuries. Prolonged operative times and persistent bleeding lead to the lethal triad of coagulopathy, acidosis, and hypothermia, resulted in a mortality of 90 %.

Damage control concepts might increase the life and limb salvage rate in troops in the field setting. Damage control is defined as the rapid initial control of hemorrhage and contamination, temporary closure, resuscitation to normal physiology, and subsequent re-exploration and definitive repair. This approach reduces mortality to 50 % in some civilian settings. Damage control techniques should be considered in all multi-system casualties.

Tactical Abbreviated Surgical Control (TASC) is damage control techniques in a tactical environment. Abbreviated, focused operative interventions for peripheral vascular injuries, extensive bone and soft tissue injuries, and thoracoabdominal penetrations in patients expected to survive, instead of definitive

surgery for every casualty. This may save precious resources, such as time, operating table space, and blood.

The goal of damage control is to restore normal physiology rather than normal anatomy. It is used for the multiple injured casualties with combinations of abdominal, vascular, genitourinary, neurologic, orthopedic, and/or thoracic injury in three separate and distinct phases:

1. ***Primary Operation and Hemorrhage Control*** – surgical control of hemorrhage and removal of contamination.

Includes 5 distinct steps:

1. Control of hemorrhage/Vascular injury repair.
2. Exploration to determine extent of injury
3. Control of contamination.
4. Therapeutic packing.
5. Abdominal closure.

2. ***Critical Care Considerations*** — restoration normal physiology by core rewarming, correction of coagulopathy and hemodynamic normalization. Physiologic support in the post-op patient control is paramount to survival:

- *Core rewarming*: warmed resuscitative fluids, blankets, ventilator air, and environment.
- *Reversal of acidosis*: appropriate/ aggressive resuscitation with crystalloid, colloid, and blood products.
- *Reversal of coagulopathy:* only ultra-fresh whole blood is available to correct coagulopathy.

3. ***Planned Reoperation*** — re-exploration to complete the definitive surgical management or evacuation.

Packs should be left in place until the patient's hemodynamics is stable and all major sites of hemorrhage have had time to clot.

Reoperation should be scheduled when the probability of achieving definitive organ repair and complete fascial closure are highest.

Timing must coincide with reversal of hypotension, acidosis, hypothermia, and coagulopathy. It typically occurs 24–48 hours following the primary insult.

General Considerations

Philosophy of damage control is «alive patient above all else».

- Avoid hypothermia.
- Rapidly achieve hemostasis.
- Perform only essential bowel resections.
- Close or divert all hollow viscus injuries, only performing reconstruction at the second operation after the patient has stabilized and can survive a prolonged operation.

Conclusions

Trust no one's examination before your own because the patient's condition may have changed, or prior examinations may be inaccurate or incomplete. Provide necessary available *monitoring* of physiology, with periodic assessment of pain control, level of consciousness, and intake and output.

- *Resuscitate* from shock, using appropriate endpoints.
- *Provide organ-specific support*, as it is done for CNS injury, pulmonary failure, cardiovascular collapse, and renal dysfunction.
- *Ensure adequate pain control*. Use IV (not IM) narcotic agents in sufficient doses to reduse pain. Patients on mechanical ventilation require *both* narcotics (morphine, fentanyl) and sedatives (propofol, lorazepam, midazolam).
- Prepare the patient for *transportation*.

10. Possible structure of mortality in modern warfare and its variability

It is discouraging that salvage rates from severe battlefield trauma sustained in conflict are similar to previous wars despite improvements in armor, surgery, critical care and evacuation. Died of wounds (% DOW) rate during the American campaign in northwestern Europe of 1944–1945 (approximately 3 %) was noticeable better than that of the American Civil War (14 %) nearly a century earlier. But enormous advances in medicine and surgery have not been reflected in substantial improvement in lives saved in forward combat surgical facilities since then (in World War П and Vietnam the rates were 3.5 and 3.4 %).

11. Medical care in evacuation echelon

Military doctrine supports an integrated health services support system to triage, treat, evacuate, and return soldiers to duty in the most time efficient manner. It begins with the soldier on the battlefield and ends in base hospitals located within the middle of the country (out of battlefield).

Echelon of Care

Level I

Immediate first aid delivered at the scene.

First aid and immediate life-saving measures provided by self-aid, buddy aid.

Level II

Immediate first aid delivered behind the scene.

Immediate life-saving measures provided by *a combat lifesaver* (medical team/squad member trained in enhanced first aid called Combat Medic).

Level III

Immediate physician care increased medical capability and limited inpatient bed space, 100 % mobile. Each service has a slightly different unit at this level.

Includes basic primary care, combat operational stress control and mental health, surgical (when required) care.

Level IV

It includes medical and surgical care outside the combat zone, within the communication zone of the theater of operations. Patients require more intensive rehabilitation or special needs.

It represents the highest level of medical care available within the combat zone with the bulk of inpatient beds. Most deployable hospitals are modular, allowing the commander to give the medical response to expected or actual demand. Traditionally consists of the Field Hospital (FH) or General Hospital (GH). It includes laboratory, intensive care, surgical and X-ray capability.

Level V

This level of care is provided inside the country. Hospitals in the sustaining base will provide the ultimate treatment capability for patients generated within the theater. Department of Defense (DoD) hospitals (military hospitals) will be specifically designated to provide the soldier with maximum return to function through a combination of medical, surgical, rehabilitative, and convalescent care. Under the National Disaster Medical System, patients overflowing DoD hospitals will be cared in civilian hospitals.

12. Role of triage in medical care organization

A mass casualty situation occurs when the number of casualties exceeds the available medical treatment capabilities (medical personnel, supplies, or transportation/evacuation assets). Triage — from the French «trier» (to sort) — is a casualty management system/process for sorting a large number of injured personnel on the basis of where resources can be best used, are most needed, and/or are most likely to achieve success.

Triage is the dynamic process of sorting casualties to identify the priority of treatment and evacuation of the wounded, given the limitations of the current situation, the mission, and available resources (time, equipment, supplies, personnel, and evacuation capabilities) in the evacuation chain.

Triage occurs at every level of care and the evacuation system.

Triage Categories

It is anticipated that triage will be performed at many levels, ranging from the battlefield to the field hospital. Traditional categories of triage are ***Immediate, Urgent, Delayed, Minimal,*** **and Expectant.** This classification scheme is useful for mass casualties involving both surgical and medical patients.

Immediate: This group includes those soldiers requiring life-saving surgery. The surgical procedures in this category should not be time consuming and should concern only those patients with high chances of survival (e.g. respiratory obstruction, unstable casualties with chest or abdominal injuries, or emergency amputation).

Urgent: This group includes those wounded who are badly in need of time-consuming surgery, but whose general condition permits delay in surgical treatment without extremely endangering life. Sustaining treatment will be required (e.g. stabilizing IV fluids, splinting and administration of antibiotics, catheterization, gastric decompression, and relief of pain). The types of injuries include

large muscle wounds, fractures of major bones, intra-abdominal and/or thoracic wounds, and burns less than 50 % of total body surface area (TBSA).

Delayed*:** This category was historically divided between ***urgent (would require intervention, however, could stand significant delay) and ***minimal*.** This is the group of patients that although injured and may require surgery, does not require the attention of the emergent group and lacks significant potential for loss of life, limb, or eyesight. Examples include: walking wounded, single long-bone fractures, closed fractures, soft tissue injuries without significant bleeding and facial fractures without airway compromise.

Minimal: These casualties have relatively minor injuries (e.g. minor lacerations, abrasions, fractures of small bones, and minor burns) and can effectively care for themselves or can be helped by nonmedical personnel.

Expectant: Casualties in this category have wounds that are so extensive that even if they had the benefit of optimal medical resource application, their survival would be unlikely. The expectant casualty should not be abandoned, but should be separated from the view of other casualties. Expectant casualties are unresponsive patients with penetrating head wounds, high spinal cord injuries, mutilating explosive wounds involving multiple anatomical sites and organs, second and third degree burns in excess of 60 % TBSA, profound shock with multiple injuries, and agonal respiration. Using a minimal but professional staff, provide comfort measures for these casualties.

Standard military triage categories into the table 1.

Table 1 — Standard military triage categories

Triage category	Characteristics	Example
Immediate	Unstable and requiting attention in the next hour Threat to life, limb or eyesight without prompt intervention	Tension pneumothorax, Dysvascular limb
Urgent	Temporarily stable but requiring care within the next few hours Patient is at risk if treatment or transportation is delayed unreasonably	Penetrating abdominal wound in a hemodynamically stable
Delayed	No risk to life or complication if more definitive care is not rendered quickly	Large muscle wounds Long bone fractures
Minimal	Relatively minor injuries that can be helped by non-medical personnel	Lacerations with controlled bleeding, fractures of small bones
Expectant	Wounds so extensive, chances of survival is unlikely even with optimal medical resources	Penetrating head wounds Mutilating explosive wounds involving multiple anatomic sites 3rd degree burn > 60 % TBSA

Military evacuation priorities

Once casualties have been categorized according to emergent/non-emergent, they are then re-categorized for evacuation to the next appropriate echelon of care.

Military evacuation priority categories into the table 2.

Table 2 — Military evacuation priority categories

Priority 1	Requires evacuation within 2 h
Priority 1a	Requires evacuation to the nearest facility with surgical capability within 2 h

Priority 2	Requires evacuation within 4 h
Priority 3	Routine evacuation within 24 h
Priority 4	Appropriate occasions

13. Organization of surgical care in an emergency is an urgent problem of military surgery nova days

The rapid expansion of knowledge regarding of combat casualty care since the start of the Global War on Terror has required constant updating of training courses for prehospital providers, physicians, surgeons, and deploying units.

II. GUNSHOT WOUND AND PRINCIPLES OF ITS TREATMENT ON MEDICAL CARE LEVELS

1. Modern types of firearms

The ghastly penetrating wounds, blast trauma, and burns produced by present day conventional and improvised weapons are essentially unchanged from those produced in the last half of the 20th Century. The automatic rifle, rocket-propelled grenade, mortar, and improvised explosive devices are widely available, easy to obtain, simple to use, lethal, and not confined to the arsenals of disciplined soldiers. Modern explosive devices are designed to spread more uniform fragments in a regular pattern over a given area.

2. Wound ballistics and mechanisms of projectile impact

The amount of tissue damage and the injury severity of gunshot injuries are due to the energy transmitted by the bullets or projectiles, depending mainly on their velocity. Therefore, the injuries are not divided any more, as in the past, to «high- and low-velocity injury» but to «a high- or low-energy injury».

Projectile or bullet injuries may be classified as «low-energy» or «high-energy», which describe the amount of damage to the tissues. The factor that most affects energy transfer, which is mostly related to kinetic energy that is presented by the equation:

$$\text{Energy transferred} = \text{Vi Ч M Ч } [(\text{V entering})^2 - (\text{V exiting})^2]$$

where M — mass; V — velocity.

in a bullet that does not «waste» energy on deforming.

The energy transfer is also affected by the tissue involved in the projectiles tract, and is related to the density and rigidity of the tissue. More rigid tissue such as bone resists deformation, and offers a greater resistance, resulting in greater energy transfer. The higher bone rigidity compared to skin and muscle produces a greater resistance and results in greater energy transfer, and commonly results in fracture of the bone.

The direction of the projectile is described as its rotation axis, and the deviation in a yaw. If the bullet remains parallel with its line of Might, the energy loss is proportional to the difference of velocity squares and hence the energy decreases over longer distances strikes the target. If a bullet wobbles and then comes to 90° to its initial direction, maximal energy transfer is achieved. The highly complex action of spin on a yawing bullet (precession), combined with a second complicated motion of higher frequency and lower amplitude (nutation), will cause the projectile to rotate in a rosette pattern of motion, imparting stability analogous to a spinning top.

Once the bullet strikes the target, it may start to rotate and tumble due to energy and speed reduction and by that cause the tissue damage.

3. Mechanism of gunshot injury. Morphological and functional changes in tissues

The energy transfer may cause tissue damage by direct laceration by the projectile. Energy lost due to the resistance of the tissue results in the development of compressive waves that radiate away from the projectile tract and can damage tissues (with the formation of a temporary cavity) by accelerating energy transfer to anything in contact with the projectile as it passes through the tissue (cavitation), which is be the most significant factor in tissue injury from high-energy projectiles.

4. Features of harmful effect of modern firearms, mine and other explosive weapons

Ballistic

Fragments from explosive munitions cause ballistic injuries. Fragments are most commonly produced by mortars, artillery, and grenades. Fragments produced by these weapons vary in size, shape, composition, and initial velocity. Modern explosive devices are designed to spread more uniform fragments in a regular pattern over a given area.

Blast

The blast effects take place relatively close to the exploding munition relative to the ballistic injury. Blast overpressure waves, or sonic shock waves, are clinically important when a patient is close to the exploding munition, such as a land mine. By increasing the pressure or its duration, the severity of injury will also increase. The ears are most often affected by the overpressure, followed by lungs and the gastrointestinal (GI) tract hollow organs. GI injuries may present 24 hours later.

Thermobaric devices work by increasing the duration of a blast wave to maximize this mechanism of injury. The device initially explodes and puts a volatile substance into the air (fuel vapor). A second explosion then ignites the aerosolized material producing an explosion of long duration. The effects from this weapon are magnified when detonated in an enclosed space such as a bunker.

5. Zones of tissue damage in gunshot wound. Morphology of wound canal (entrance or exit wounds, zone characteristics)

For missile injuries, there are two areas of projectile-tissue interaction: ***permanent cavity*** (the *central primary track*) and ***temporary cavity*** **(**includes 2 zones: *primary necrosis zone*, *secondary necrosis zone*).

Permanent cavity is localized area of cell necrosis, proportional to the size of the projectile as it passes through.

Temporary cavity (concussion area) is a transient lateral displacement of tissue, which occurs after passage of the projectile. It inversely depends on the transferred energy. Elastic tissues, such as skeletal muscle, blood vessels and skin, may be pushed aside after passage of the projectile, but then rebound. Inelastic tissues, such as bone or liver, may be fractured in this area. *Temporary* cavity includes 2 zones: primary necrosis zone and secondary necrosis zone.

As energy increases, the area of the injured muscles increases too and usually can be easily diagnosed by direct vision. The direct range of damage is energy dependent and may reach a radius of a few centimeters from the primary tract due to deformation of the penetrating projectile.

Gunshot injuries are classified as either entrance or exit wounds. Atypical wounds (grazing) may also be present. Physical findings in and around these wounds may offer evidence as to the actual mechanism, supporting or refuting the initial history given to the provider. As these findings may be transient, the emergency physician must be diligent in recognizing and documenting them at the time of presentation.

Entrance Wounds

Gunshot wounds of entrance are divided into four categories based on their range of fire: distant, intermediate, close, and contact. Range-of-fire is the distance from the gun's muzzle to the victim.

The size of the entrance wound bears no relation to the caliber of the inflicting bullet. Entrance wounds over elastic tissue will contract around the tissue defect and have a diameter much less than the caliber of the bullet.

Distant Wounds: The distant wound is inflicted from a range sufficiently distant that the bullet is the only projectile expelled from the muzzle that reaches the skin. As the bullet penetrates the skin, friction between it and the epithelium results in the creation of an «abrasion collar». Most entrance wounds will have an abrasion collar; however, gunshot wounds to the palms and soles are exceptions — there entrance wounds appear slit-like.

Intermediate-Range Wounds: tattooing is pathognomonic for an intermediate-range gunshot wound and presents as punctate abrasions from contact with partially burned or unburned grains of gunpowder. Clothing and hair, as intermediate objects, may prevent the gunpowder grains from making contact with the skin.

Close-Range (Near Contact) Wounds: «Close range» is defined as the maximum range at which soot is deposited on the wound or clothing and typically is a muzzle-to-victim distance of 6 in (15 cm) or less.

Contact Wounds: A contact wound occurs when the barrel or muzzle is in contact with the skin or clothing as the weapon is discharged. Contact wounds can be described as tight, where the muzzle is pushed hard against the skin, or loose, where the muzzle is incompletely or loosely in contact with the skin or clothing. Wounds sustained from tight contact with the barrel can vary in appearance from a small hole with dry, blackened edges (from the discharge of hot gases and an actual flame), to a gaping, stellate wound (from the expansion of the skin from gases). Large stellate wounds are often misinterpreted as exit wounds based solely upon their size and without adequate examination of the wound.

Exit wounds

The exit wounds can appear cone shaped; the base at the entry site, stellate, slit-like, crescenlic, circular, or completely irregular and depends on the tissue density and the bullet behavior in different tissues or organ, the weapon and bullet characters, and it is not necessarily larger than the entrance wound. Exit wounds assume a variety of shapes and appearances and are not consistently larger than their corresponding entrance wounds. The size of an exit wound is determined primarily by the amount of energy possessed by the bullet as it exits the skin and by the bullet's size, shape, and attitude.

At high velocities, mainly over 2,000 fps (700 mps) the bullet deformity and tumbling in the body usually causes a larger and more irregular exit wound than the entrance. These high velocities may allow the cavity formation at the exit site to suck into the wound foreign materials or debris. Energy transferred to bone, with resultant ballistic fracture, may also result in an exit wound larger than the entrance wound.

Graze Wounds

Graze wounds are considered atypical and result from tangential contact with a passing bullet. The direction of the bullet's path is determined by careful wound examination.

6. Concept of primary and secondary wounds microbial contamination

Material contamination caused by a combination of soil, clothing and skin, carried by the bullet or projectile is a major complication. This happens in all kinds of projectile wounds. Weapon fragments have been shown experimentally to cut clothing and skin and to transport these into the wound.

In high-energy wounds the fragments tend to shred clothing into pieces, these accompanied with the high-energy projectile drive these particles throughout the large temporary cavity so that contamination is spread widely away from the wound track.

The battlefield environment is conducive to wound infection due to:

• Absence of «sterile» wounding agents on the battlefield. All foreign bodies (wounding projectile fragments, clothing, dirt) are contaminated with bacteria.

• High-energy projectile wounding (devitalized tissue, hematoma, tissue ischemia).

• Delay in casualty evacuation.

7. Early prevention of infectious complications

All wounds obtained on the battlefield are grossly contaminated with bacteria. Most will become infected unless appropriate treatment is initiated quickly. This explains why at least 24 h of intravenous antibiotic treatment is required in fractures caused by high-velocity weapons in conjunction with the appropriate wound and fracture care.

Prompt surgical debridement is the cornerstone of prophylaxis/treatment of wound infections.

8. Medical care in gunshot wounds on battlefield and in evacuation echelons. Wound Care. Recent views on surgical debridement

Debridement is the medical removal of dead, damaged, or infected tissue to improve the healing potential of the remaining healthy tissue. Removal may be surgical, mechanical, chemical, autolytic (self-digestion), and by maggot therapy, where certain species of live maggots selectively eat only necrotic tissue.

Primary Surgical Wound Care:

• Limited longitudinal incisions for revision.

• Excision of foreign material and devitalized tissue.

• Irrigation, hemostasis, drainage.

• Leave Wound Open — No Primary Closure.

• Antibiotics and tetanus prophylaxis.

• Splint for transport (improves pain control).

Longitudinal incisions

Wounds are extended with incisions parallel to the long axis of the extremity, to expose the entire deep zone of injury. At the flexion side of joints, the incisions are made obliquely to the long axis to prevent the development of flexion contractures. The use of longitudinal incisions, rather than transverse ones, allows for proximal and distal extension, as needed, for more careful visualization and debridement.

Wound excision (current use of the term ***debridement***):

Skin

Conservative excision of 1–2 mm of damaged skin edges. Excessive skin excision is avoided; questionable areas can be assessed at the next debridement.

Fat

Damaged, contaminated fat should be generously removed.

Fascia

Damage to the fascia is often minimal relative to the magnitude of destruction beneath it. Shredded, torn portions of fascia are excised, and the fascia is widely opened through longitudinal incision to expose the entire zone of injury beneath. Complete fasciotomy is often required.

Muscle

Removal of dead muscle is important to prevent infection. Accurate initial assessment of muscle viability is difficult. Tissue sparing debridement is acceptable if follow-on wound surgery will occur within 24 hours. More aggressive debridement is required if subsequent surgery will be delayed for more than 24 hours.

Bone

Fragments of bone with soft-tissue attachments and large free articular fragments are preserved. Remove all devitalized, avascular pieces of bone smaller than thumbnail size that have no soft-tissue attachment. Deliver each of the bone ends of any fracture independently, clean the surface and clean out the ends of the medullary canal.

Nerves and tendons

Do not require debridement, except for trimming frayed edges and grossly destroyed portions. Primary repair is not performed. To prevent desiccation, use soft-tissue or moist dressings for coverage.

Vessels

Only minimal debridement of vessel is required for a successful repair.

Irrigation

Following surgical removal of debris and nonviable tissue, irrigation is performed until clean. While sterile physiologic fluid is preferred, do not deplete resuscitation fluid resources, may use potable water as an alternative. The last liter of irrigate fluid should be a sterile solution with antibiotics.

At least ***hemostasis and drainage*** are performed. Drainage is generally employed in the wound to prevent accumulation of blood and exudation fluids, and its next contamination.

Leaving the wound open allows the egress of fluids, avoids ischemia, allows for unrestricted edema, and avoids the creation of an anaerobic environment (No Primary Closure of War Wounds). Do not plug the wound with pack-

ing as this prevents wound drainage. Place a nonconstricting, nonocclusive dry dressing over the wound.

9. Indications and contraindications for debridement

Indications for Surgical Debridement:
- Wound size lager than 2 cm.
- Extensive devitalized tissue.
- Associated fracture.
- Fascial defects.
- Penetration of pleura, peritoneum, vascular structures, joint capsule.

Contraindications for Surgical Debridement

Debridement is contraindicated for untreated bleeding disorders, arterial insufficiency or when underlying conditions have not been managed.

10. Wound Management after Initial Surgery. Early, delayed and late debridement

The wound undergoes a planned second debridement and irrigation in 24–72 hours, and subsequent procedures until a clean wound is achieved. Between procedures there may be better demarcation of nonviable tissue or the development of local infection. Early soft-tissue coverage is desirable within 3–5 days; when the wound is clean, to prevent secondary infection.

11. Primary and secondary wound healing. Ways to close gunshot wounds after debridement. Primary, primary delayed and secondary (early and late) sutures. Conditions for use of a primary suture

Primary Closure of War Wounds could be performing only when they localized at good vascularized and functionally important areas, such as: head, face, neck, palm, external genitalia, thorax etc.

Delayed primary closure (3–5 days) requires a clean wound that can be closed without excessive tension. This state may be difficult to achieve in war wounds.

Soft-tissue war wounds heal well without significant loss of function through secondary intention. This is especially true for simple soft-tissue wounds.

Definitive closure with skin grafts and muscle flaps should not be done in theater when evacuation is possible. The development and rotation of flaps for this purpose should not be done during primary surgical wound care. These techniques may be required, however, for injured civilians or prisoners of war.

After the elimination of local inflammatory changes in order to accelerate the healing wound defects impose a primary delayed (after 5–7 days, before to the development of granulation) and secondary sutures («early» — within 7–14 days after injury on the granulating wound and «late» — after 21 days on the wound covered with scar tissue with its further excising).

12. The most common errors in surgical debridement

Experience of military surgeons in Afghanistan and Chechnya revealed the following defects debridement:

- not enough high-quality primary debridement;
- suturing the wound before the optimum date (most surgeonss, educated in the principles of peace-time surgery, complete surgical debridmend used to overlay deaf suture wounds);
- avoid principles of delayed primary suture (i.e. waiting for the appearance of granulation);
- a very wide application of intramedullary structures.

The total number of suppurations even with all the requirements of field surgery is about 20 %.

B. COMPLICATIONS IN MILITARY TRAUMA

III. INFECTION COMPLICATIONS IN GUNSHOT WOUNDS. TETANUS

1. Concept of wound infection

Battlefield casualties are at high risk for infection. In particular, war wounds are predisposed to infection due to environmental conditions on the battlefield, devitalized tissue, and foreign bodies in the wound.

Wound infection — the infectious process developing in the walls of the wound channel and the tissues surrounding the wound, under the influence of micro-organisms belonging to a living tissue. It is accompanied by damage and loss of cellular structures, complicates the reparative process in the wound and is accompanied by clinically significant systemic reaction.

During the infection process there is an active interaction between microbes and the host response to the formation of local or systemic inflammatory response. Primary purulent-inflammatory processes account for 30–40 % of the total number of patients with surgical and 32–75 % of all postoperative complications, they also cause more than 40–50 % of mortality in the postoperative period.

2. Factors contributing to development of wound infection

The battlefield environment is conducive to wound infection due to:

• Absence of «sterile» wounding agents on the battlefield. All foreign bodies (wounding projectile fragments, clothing, dirt) are contaminated with bacteria.

• High-energy projectile wounding (devitalized tissue, hematoma and tissue ischemia).

• Delay in casualty evacuation.

3. The Wound Microenvironment

The epidemiology of combat-related wounds has changed significantly since Alexander Fleming characterized bacteria-infecting wounds during World War I. This includes a shift from Clostridia species in World War I to Streptococcus pyogenes and Staphylococcus aureus in World War II, to gram-negative bacilli (Pseudomonas aeruginosa, Enterobacter species, Escherichia coli, and Klebsiella species) since the Vietnam War. During the Korean conflict, it was noted that the bacteria responsible for infecting wounds varied with the seasons, with more staphylococci, streptococci, and Clostridium in winter months and fecal pathogens in summer months. Typically, an even representation of gram-positive bacteria such as Staphylococcus spp. and gram-negalive bacteria such as Pseudomonas aeruginosa, Enterobacter aerogenes, Proteus spp., and E. coli existed at the time of injury but, over the course of therapy, resistant gram-negative bacteria were responsible for the majority of infections, especially further back in the evacuation chain. During Operation Iraqi Freedom, samples from war wounds were obtained shortly after injury for aerobic culture. Approximately one-half of the culture results were positive, with most cultures yielding gram-positive skin flora.

One of the primary lessons learned during World War II was the role of nosocomial transmission of disease, with up to 86% of patients having hospital-associated infections.

Common Microorganisms Causing Battlefield Infections:

- Gram-positive cocci: staphylococci, streptococci, and enterococci.
- Gram-negative rods: Escherichia Coli, Proteus, and Klebsiella.
- Pseudomonas, Enterdbacter, Acinetobacter, and Serratia are common nosocomial pathogens usually expected among casualties who have been hospitalized for an extended period, not those fresh off the battlefield.
- Salmonella, Shigella, and Vibrio should be suspected in cases of bacterial dysentery.
- Anaerobic Gram-positive and Gram-negative rods: Clostridia, Bacteroides, and Prevotella species.
- Fungal species: Candida species should be suspected in casualties hospitalized for prolonged periods, those malnourished or immunosuppressed, or those who have received broad spectrum antibiotics, adrenocortical steroids, or parenteral nutrition. Empiric therapy should be considered in appropriate patients with presumptive evidence of fungal infection.

4. Local purulent infection

In 1980, the Foothills Hospital classified set of wound infection rates:

I — clean;
II — clean contaminated;
III — contaminated;
IV — dirty.

Diagnosis of a Wound Infection

The four «-ors»: dolor, rubor, calor, and tumor — ***pain*** and ***tenderness, redness, warmth***, and ***swelling***.

Clinical signs of wound infection

If the wound is not healing, it may be a sign of infection. In the wound, the following symptoms indicate infection: odour, increased exudates (drainage or discharge, ranging from frank pus to the foul «dishwater» discharge of clostridial infection), increased pain, absent or abnormal granulation tissue, systemic effects such as fever, leukocytosis, unexplained tachycardia, or hypotension.

Severity: a wound infection is described as minor if there is discharge without cellulitis or deep tissue destruction, and major if the discharge of pus is associated with tissue breakdown, partial or total dehiscence of the deep fascial layers of the wound, or if systemic illness is present.

Local infectious complications of combat injuries are a group of various clinical and morphological suppurative infectious processes, which are localized within the primary focus of the wound (wound gate), and due to the various aerobic and anaerobic pathogens.

They are following their clinical and morphological forms:
— suppuration of the wound (cellulitis without infection or with it);
— an abscess of the wound cannel (of the wound cavity or organ);
— wound phlegmon;
— burrowing pus;
— purulent fistulas;
— post-traumatic and gunshot osteomyelitis;
— an acute thrombophlebitis;
— specific processes (erysipelas, tetanus, diphtheria of wounds);
— anaerobic (clostridial and non-clostridial) infection;
— post-traumatic gangrene;
— wound sepsis.

Systemic sepsis*:* A life-threatening syndrome caused by a bloodborne or severe regional infection resulting in a SIRS — Systemic Inflammatory Response Syndrome that includes two or more of the following conditions:

- Tachycardia.
- Fever or hypothermia.
- Tachypnea or hyperventilation.
- Leukocytosis or acute leukopenia.

Progression to septic shock is manifest by systemic hypoperfusion: pro-

found hypotension, mental obtundation, or lactic acidosis. Asimilar inflammatory response without infection can be caused by a focus of retained necrotic tissue, or severe trauma itself.

5. Prophylactic and therapeutic value of antibiotics

Antibiotics have advanced the successful management of war wounds. Since 1943, when systemic penicillin was introduced onto the battlefield, the risk for wound myonecrosis and gas gangrene has decreased dramatically. Although a useful adjunct, antibiotic treatment cannot replace adequate debridement of devitalized and dead tissue from a war wound. Antibiotics play an adjunctive role in the prophylaxis of wound and other infections in the battlefield. Knowledge of likely pathogens for particular infections and sites, as well as optimal antibiotics to eradicate those pathogens, will aid the battlefield clinician in preventing and treating infections.

The recently published guidelines for the prevention of infection after combat-related injuries recommend the early use of cefazolin or another intravenous first-generation cephalosporin at Level I/II medical care in the combat zone for all extremity injuries.

6. Treatment of infectious complications in gunshot wounds on medical evacuation levels

Prophylaxis

The key to avoiding wound infection is prompt and adequate wound exploration, removal of all foreign material, and excision of all dead tissue. All battlefield wounds and incisions should have the skin left open.

Treatment

General Principles

Once a battlefield wound has become infected, treatment is two-fold—surgical and medical.

Optimally, surgical debridement should be achieved within 6 hours of injury. Following initial exploration and debridement, the wound should be sufficiently irrigated to ensure all dead material, bacterial contamination, also foreign material has been washed from the wound. Excessive irrigation, especially under pressure, should be avoided, because this can dilute the body's natural immune cellular defense and contribute to bacteremia.

Drainage is generally employed in abscess cavities to prevent premature closure and deformation.

The skin is left open, and a lightly moistened sterile gauze dressing is applied.

Antibiotics should be started as soon as possible after wounding, and then

continued for 24 hours, depending on the size, extent of destruction, and degree of contamination of the wound.

Empiric broad-spectrum antibiotic therapy is initiated against likely pathogens and continued for 7 to 10 days, ideally, obtain cultures and tailor therapy to cover the actual pathogens recovered on Gram stain and culture. Routine bacteriology is often not available in forward medical facilities. Because Bacteroides and Clostridia are difficult to culture, tailor antibiotic therapy should cover these organisms.

Systemic Sepsis treatment

Treatment is a three-pronged direction:

Identify and eradicate the source. It is often difficult to identify the source of sepsis, but it is the *most important factor* in determining the outcome.

Broad-spectrum intravenous antibiotics used for the most pathogens. Until the source for sepsis is identified and actual pathogens isolated, empiric therapy with broad-spectrum intravenous antibiotics is warranted.

Support for failing organ systems, such as cardiovascular collapse, acute renal failure, and respiratory failure. Intensive care support for sepsis involves vigorous resuscitation to restore perfusion for preventing multiple organ dysfunctions. This requires optimization of hemodynamic parameters (pulmonary artery occlusion pressure, cardiac output, and oxygen delivery) to reverse anaerobic metabolism and lactic acidosis. Endpoints of resuscitation, such as urine output, base deficit, and blood lactate levels guide successful treatment.

7. Anaerobic infection of wounds

The greatest threat of infection to the wounded battlefield casualty is the development of **clostridial myonecrosis (gas gangrene**) a bacterial infection that produces gas in tissues and gangrene, commonly due to Clostridium perfringens. These organisms create a rapidly advancing infection within the **subcutaneous tissues** and/or **muscle** by producing exotoxins that lead to bacteremia, toxemia, and septic shock. Gas gangrene (also known as «Clostridial myonecrosis» and «Myonecrosis») usually results from deep trauma or surgery, although minor procedures, such as intramuscular injection, have been associated with gas gangrene.

8. Terms of the appearance, symptoms and signs

Pathomorphology:

Organisms in the spore-forming clostridial species, including Clostridium perfringens, Clostridium septicum, Clostridium histolyticum and Clostridium novyi (oedematiens), cause most of the cases.

The incubation period is usually less than 24 hours but has been described in some cases from 7 hours to 6 weeks, though when symptoms start, clinical

deterioration can occur within hours.

With Cl. perfringens, the local and systemic manifestations of infection are due to the production of potent extracellular protein toxins by the bacteria. These are most notably alpha-toxin (a phospholipase C) and theta-toxin (a thiol-activated cytolysin). These toxins hydrolyze cell membranes, cause abnormal coagulation leading to microvascular thrombosis (further extending the borders of devascularized and thus anaerobic tissue), generating gas at the same time and have direct cardiodepressive effects. Furthermore, the products of tissue breakdown, including creatine phosphokinase, myoglobin, and potassium, may cause secondary toxicity and renal impairment. Systemic signs include moderate to high fever, increased heart rate (tachycardia), leukocytosis, mental obtundation, hemolytic anemia, and hypotension, progressing rapidly to multiple organ failure and death in untreated or under-treated cases.

All layers of soft tissue can be involved, including skin (blistering — vesicle formation which turn into large blisters and necrosis), subcutaneous tissue (panniculitis), fascia (fasciitis), and muscle. Clinical manifestations begin locally with severe pain, subcutaneous crepitus, progressive swelling around a skin injury and a thin, brown, serosanguineous, foul smelling discharge («rotten apple or eggs» stench). The skin may be tense and shiny.

Radiographs reveal fine gas bubbles within the soft tissues, dissecting into the intramuscular fascias and muscles.

9. Characteristics of different clinical forms

Not all wounds contaminated with clostridia develop gas gangrene; the myonecrosis seems to only develop when sufficient devitalized tissue is present to support anaerobic metabolism. Traumatic gas gangrene and surgical gas gangrene occur through direct inoculation of a wound. With a compromised blood supply, the wound has an anaerobic environment that is ideal for Cl. perfringens.

Spontaneous gas gangrene is most often caused by hematogenous spread of Cl. septicum from the gastrointestinal tract in patients with colon cancer or other portals of entry. Neutropenic and immunocompromised patients are also at risk. The organism enters the blood via a small break in the gastrointestinal mucosa and subsequently seeds muscle tissue. Unlike Cl. perfringens, Cl. septicum is aerotolerant and can infect normal tissues.

Necrotizing fasciitis is a progressive, rapidly spreading, inflammatory infection located in the deep fascia, with secondary necrosis of the subcutaneous tissues. Because of the presence of gas-forming organisms, subcutaneous air is classically described in necrotizing fasciitis. This may be seen only on radiographs or not at all. The speed of spread is directly proportional to the thickness of the subcutaneous layer. Necrotizing fasciitis moves along the deep fascial layer.

10. Prevention of anaerobic infections

The best weapon against gangrene is prevention.

Keep wounds clean and sterile by cleaning all wounds thoroughly with antiseptic solution. Watch for signs of infection, such as pus, redness, swelling, ***edema*** or ***unusual severe pain***. People with diabetes should control their blood-sugar levels with proper medication.

No vaccine can prevent clostridial infection. There should be no need to use this in most wounds. If you give it, do a skin sensitivity test first. Then give pentavalent gas gangrene antiserum intravenously and repeat it after 4 to 6 hours.

11. Specific and nonspecific treatment at medical care echelon

This condition needs to be treated aggressively because of the threat of the infection rapidly spreading via the bloodstream and damaging vital organs. The wound requires immediate debridement.

Antibiotics alone are not effective because they do not penetrate ischaemic muscles enough to be effective. However, penicillin is given as an additional treatment to surgery. Clostridial species are exquisitely sensitive to a combination of penicillin G and clindamycin. However, because it is difficult initially to distinguish gas gangrene from other soft tissue infections, such as necrotizing fasciitis, which is caused by a broad spectrum of pathogens, empiric first-line antibiotic therapy should be broad. Clindamycin, tetracycline, and other inhibitors of bacterial protein synthesis may, have some increased utility as they halt the production of bacterial toxin.

In addition to surgery and antibiotics, hyperbaric oxygen therapy (HBOT) is used and acts to inhibit the growth of and kill the anaerobic Cl. perfringens. Hyperbaric oxygen, if available, is specifically indicated in gas gangrene, as an adjuvant to radical excision of dead muscle, antibiotics, transfusion, and general intensive care support.

12. Outcomes of treatment

Gas gangrene complications:

- shock;
- renal failure;
- delirium;
- liver damage;
- spread of infection through the body;
- disseminated intravascular coagulation;
- acute respiratory distress syndrome;
- coma;
- mental confusion.

Gas gangrene usually has a bad prognosis and is often deadly. It usually begins

suddenly and quickly gets worse. Mortality from traumatic gas gangrene is greater than 25 %. Mortality from nontraumatic gas gangrene caused by Cl. septicum ranges from 67 to 100 %. Prognosis is good if incubation period is less than 30 hours. Advanced age and presence of comorbid symptoms are associated with poor prognosis.

13. Anaerobic non-clostridial (decay) wounds infection

Decay (Polymicrobal necrotizing soft tissue) infection is the most dreaded infection resulting from battlefield wounding. These include ***polymicrobial infections caused by Streptococcus, Staphylococcus, Enterococcus, Enterobacteriaceae, Bacteroides, and Clostridia.***

Polymicrobal infections are mixed infections with anaerobes. Organisms can include, for example, Bacteroides or Peptostreptococcus with a facultative anaerobe such as the Enterobacteriaceae Escherichia coli, Enterobacter, Klebsiella, or Proteus; or non-group A streptococci. It occurs most frequently after surgical procedures, and in patients with diabetes, alcoholism, immunosuppression, intravenous drug use, or peripheral vascular disease. These organisms create a slightly advancing infection within the subcutaneous tissues and/or muscle by producing exotoxins that lead to bacteremia, toxemia, and death.

Fournier's gangrene is a necrotising fasciitis of the perineal and genital region, resulting from synergistic polymicrobic infection. Men are affected more often than women.

Pathomorphology:

Soil-borne anaerobes are particularly well adapted to surviving harsh conditions. Often, there is a leak of nutrition and the presence of numerous other species competing for resources. Changes in pH and temperature are often significant also. Competing bacteria often also possess the ability to create exotoxins that assist them in competing with other microbes in their natural environment. When such bacteria are able to enter a living host, they encounter a vast supply of nutrients, warm conditions, and an abundance of water. This enables the microbes to rapidly proliferate, far in excess of the immune system's capability to defend. The combination of bacterial load and ability to multiply is the basis for the microbes' ability to cause massive infection. Alongside such rapid proliferation is a corresponding mass-production of exotoxin that causes severe damage to local tissue. Drainage or discharge smells decayed flash.

Massive infection, gross injury, and depletion of the host immune capability result in system-wide sepsis.

Treatment is surgical, including early, radical, and repeated (every 24–48 hours) debridement of all dead and infected tissue, combined with antibiotics and antiseptics. Excision of affected tissue must be as radical as necessary (including amputation or disarticulation) to remove all muscle that is discolored, noncontractile, nonbleeding, or suspicious.

Antibiotic treatment must be aimed at all possible organisms after identification of causative organisms (intravenous antibiotic therapy). For mixed infections, the Infectious Diseases Society of America (IDSA) recommends combination therapy with ampicillin/sulbactam plus clindamycin plus ciprofloxacin. A beta-lactam/beta-lactamase combination such as piperacillin/tazobactam or ticarcillin/clavulanate, or a carbapenem such as meropenem, may also be a reasonable initial empirical treatment. For patients allergic to penicillin, metronidazole and an aminoglycoside may be used. Vancomycin may need to be added to empiric antibiotic therapy until methicillin-resistant Staphylococcus aureus is shown not to be part of the infectious process. Further antibiotic treatment should be based upon Gram stain, culture, and sensitivity data when available.

14. Tetanus, its etiology and pathogenesis

Tetanus

The disease in humans is the result of infection of a wound with the spores of the bacteria Clostridium tetani. Bacteria grow anaerobically and release a CNS toxin that results in muscle spasm, trismus, neck rigidity, and back arching.

Clostridium tetani can enter through any wound — even minor burns and corneal abrasions. Prophylaxis is required to prevent tetanus toxemia.

These bacteria produce the toxin (poison) tetanospasmin, which is responsible for causing tetanus. Tetanospasmin binds to motor nerves that control muscles, enters the axons, and travels in the axon until it reaches the motor nerve in the spinal cord or brainstem (a process termed retrograde intraneuronal transport). Then the toxin migrates into the synapse (space between nerve cells critical for transmission of signals among nerve cells) where it binds to presynaptic nerve terminals and inhibits or stops the release of certain inhibitory neurotransmitters (glycine and gamma-aminobutyric acid). Because the motor nerve has no inhibitory signals from other nerves, the chemical signal to the motor nerve of the muscle intensifies, causing the muscle to tighten up in a huge continuous contraction or spasm. If tetanospasmin reaches the bloodstream or lymphatic vessels from the wound site, it can be deposited in many different presynaptic terminals resulting in the same effect on other muscles.

15. Tetanus symptoms and signs

The hallmark feature of tetanus is muscle rigidity and spasms.

In generalized tetanus, the initial complaints may include any of the following:

- Irritability, muscle cramps, sore muscles, weakness, or difficulty swallowing are commonly seen.
- Facial muscles are often affected first. Trismus or lockjaw is most common. This condition results from spasms of the jaw muscles that are responsible for chewing. A sardonic smile — medically termed «risus sardonicus» — is a

characteristic feature that results from facial muscle spasms.

• Muscle spasms are progressive and may include a characteristic arching of the back known as opisthotonus. Muscle spasms may be intense enough to cause bones to break and joints to dislocate.

• Severe cases can involve spasms of the vocal cords or muscles involved in breathing. If this happens, death is likely, unless medical help (mechanical ventilation with a respirator) is readily available.

The disease can show four possible types:

• Generalized tetanus can affect all skeletal muscles. It is the most common as well as the most severe form of the four types.

• Local tetanus manifests with muscle spasms at or near the wound that has been infected with the bacteria.

• Cephalic tetanus primarily affects one or several muscles in the face rapidly (in one to two days) after a head injury or ear infection. Trismus («lockjaw») may occur. The disease can easily progress to generalized tetanus.

• Neonatal tetanus is similar to generalized tetanus except that it affects an infant that is less than 1 month old (called a neonate). This condition is rare in developed countries.

16. Diagnosis, prevention and treatment guidelines for tetanus

The diagnosis of generalized tetanus is usually made by observing the clinical presentation and a combination of the following:

• History of a recent injury resulting in skin breakage (but this is not universal; only 70 % of cases have an identified injury).

• Incomplete tetanus immunizations.

• Progressive muscle spasms (starting in the facial region, especially lockjaw and progressing outward from the face to include all muscles of the body).

• Fever.

• Changes in blood pressure (especially high blood pressure).

• Irregular heartbeat.

• In localized tetanus, pain, cramps, or muscle spasms occur at or near a recent skin injury.

• Neonates show signs of being generally irritable, muscle spasms, and poor ability to take in liquids (poor sucking response), usually seen in neonates about 7–10 days old.

Laboratory tests are rarely used to diagnose tetanus. However, some reference labs can determine if the patient has serum antitoxin levels that are protective, and thus a positive test detecting these levels suggests that the diagnosis of tetanus is unlikely.

Treatment for established tetanus includes:

• IV antibiotics (penicillin G, 24 million U/d; or doxycycline, 100 mg bid; or metronidazole, 500 mg q6h for 7 days).

• Tetanus immune globulin.

• Wound debridement as needed.

• IV diazepam to ameliorate the muscle spasm.

• Place patient in a dark, quiet room free of extraneous stimulation.

• May warrant endotracheal intubation, mechanical ventilation, and neuromuscular blockade.

Prevention (profilaxis):

• In addition to surgical debridement of war wounds, additional prophylactic measures for tetanus-prone wounds include:

• Administration of 0.5ml IM of ***tetanus toxoid*** if prior tetanus immunization is more than five years since last dose.

• Administration of 250–500 units IM of ***tetanus immune globulin*** in a separate syringe and at a separate site from the toxoid if prior tetanus immunization is uncertain or less than three doses. or more than ten years since last dose.

17. Complications and outcomes

Overall, about 25–50 % of people with generalized tetanus will die. The disease is more serious when the symptoms come on quickly. Older people and very young children tend to have more severe cases; those over 65 years are more likely to die from the infection. Death is usually due to respiratory failure or disturbance of heart rhythm.

Intensive medical care improves the prognosis in severe cases.

IV. BLEEDING, BLOOD LOSS

Hemorrhage is the acute loss of circulating blood volume. An element of hypovolemia is present in nearly all polytraumalized patients. Hemorrhage is the most common cause of shock and of preventable death on the battlefield.

Sites of Hemorrhage

• *External*.

Extremity injury usually associated with an open fracture or amputation (most common cause of massive external blood loss in combat), scalp, and torso wounds.

• *Internal*.

Chest, abdomen, pelvis, and closed extremity fractures. Blood loss into the abdomen or chest cannot be controlled in the field and requires immediate evacuation for salvage or definitive surgery. Stabilization of pelvic fracture with garment, or by wrapping the pelvis tightly with a wide strap (such as a folded sheet), may reduce pelvic bleeding.

Class of hemorrhage:

1. Class I hemorrhage is characterized by no measurable change in physiologic parameters (heart rate, blood pressure, urine output, etc.) despite a < 15 % blood loss (< 750 ml).

2. Class II hemorrhage is characterized by mild tachycardia (> 100), a moderate decrease in blood pressure, and low normal urine output (20–30 ml/h). It represents 15–30 % blood loss (750–1,500 ml).

3. Class III hemorrhage is characterized by moderate tachycardia (>120>, a decrease in blood pressure, and a decrease in urine output (5–15 ml/h). The pa-

tient is typically confused. It represents a 30–40 % blood loss (1,500–2,000 ml).

4. Class IV hemorrhage is characterized by a severe tachycardia (>140), decreased blood pressure, and negligible urine output. The patient is lethargic. It represents > 40 % blood loss (> 2,000 ml).

1. Calculating methods for determining volume of blood loss

Existing methods of determining the volume of blood loss can be divided into two groups: direct and indirect effects.

Direct methods are divided into:

- colorimetric;
- weight;
- volume.

These methods estimate the amount of blood loss during the bleeding.

Indirect methods are the following groups:

- clinical;
- laboratory;
- hardware;
- mathematical.

They can be used to estimate blood loss in all phases of treatment.

If the bleeding was less than 12 hours should be guided by clinical criteria. One of the clinical signs indicating a decrease TBV (total blood volume), and as a consequence, the development of centralization of circulation, is to reduce the minimum hourly urine output of 0.5–1 ml / kg body weight.

In the military surgery can be guided by the clinical method of assessing the relative value matching the localization of bleeding injuries and the volume of damaged tissue: in severe chest trauma — 1.5–2.5 l, belly — up to 2 l, multiple fractures of the pelvis, 2.5–3.5 l, open fractures of the hip — 1.5–1.8 l, closed femur fracture — 2 l, calf — up to 0.8 l, the shoulder — 0.6 l, forearm — 0.3 l, with extensive superficial wounds on the size of the wound surface (open palm of the wounded being 0.5 l).

The simplest and most frequently used method of determining the amount of blood loss before the start of infusion therapy is the calculation of the quotient of heart rate on the value of systolic blood pressure, so called «shock index» (SI) by Algover-Burri. The normal value is equal to the SI 0,54 ± 0,021 units. Each subsequent to an increase of 0.1 units corresponds to a loss of 0.2 l of blood, or 4 % of TBV.

The level of central venous pressure (CVP), as an integral indicator reflects the level of venous pressure, characterized by right ventricular pump function and directly correlates with the TBV. Reduction of central venous pressure is below normal (6-12 cm of water column) indicates the development of hypovolemia. Deficiency of the TBV to 25% of predicted value corresponds to the central venous pressure equal to 0.

After 12–18 hours of onset of bleeding the most informative for estimating the volume of blood loss are indicators of laboratory hematology.

2. Vascular injury

The large physical forces and thermal effects applied to the vascular wall during the traumatic event may cause a spectrum of injuries. These may range from a mild contusion of the adventitia or a minor intimal damage to a complete transaction of the vessel.

Arterial injuries of the extremities are manifested by life-threatening hemorrhage or by ischemia resulting in limb loss. Hard signs for vascular injuries are active arterial bleeding, proximity to a named large artery, expanding or pulsating hematoma, ischemia (pallor, pulselessness, paresis, or paralysis), and a thrill/bruit. Presence of a hard sign with a single entry and/or exit wound lead to surgery after the primary assessment in the emergency room. With multiple injuries or the presence of soft signs for a possible vascular injury, US imaging is necessary for both the correct diagnosis and the choice of the preferred treatment. A high index of suspicion in high-energy trauma is mandatory especially in periarticular knee and elbow injuries or a gross displacement fracture.

In sharp contrast, severe ***venous trauma*** is manifested by hemorrhage, not ischemia. Bleeding may be internal or external and rarely may lead to hypovolemic shock.

Spontaneous thrombosis is frequently seen in the injured segment, usually on both sides of the injury up to the nearest uninvolved branch.

A pseudoaneurysm or a pulsating hematoma may develop if the arterial bleeding is partially contained by nearest tissues and fasciae.

An arteriovenous fistula may develop when both an artery and an adjacent vein are lacerated simultaneously.

3. Treatment on medical care levels

In vascular trauma, the goal of treatment is twofold: First to stop continued bleeding, and then to revascularize ischemic limbs. Injured patients with limb vascular trauma are frequently saved from exsanguinations by liberal use of rubber tourniquets that are available to almost every soldier in the battlefield nowadays.

4. Methods of temporary and final bleeding arrest

Methods of temporary bleeding arrest:

- ***Direct pressure*** at site of injury is the most effective and preferred method of hemorrhage control.
- ***Hemostatic bandages*** currently being developed may stop bleeding.
- ***Elevation*** of the extremity will decrease most bleeding — this is an under-appreciated technique.

• ***Clamping vessels***: If there is continued bleeding and a damaged vessel can be readily identified, a hemostat may be used to clamp the vessel.

• ***Limb splints*** will decrease bleeding associated with fractures and soft tissue injury by stabilizing and returning the limb to length.

• ***Military Anti-Shock Trousers (***MAST) possible use.

• ***Tourniquet*** may be first choice in combat. A tourniquet should be applied if previous techniques fail. Rapid method to secure hemorrhage control. Does not require constant attention; allows first responder to care for others — extends resources. Use a tourniquet early, rather than allow ongoing blood loss.

Substitutes for issued tourniquet include: belt, torn cloth, gauze, and rope, among others.

Tourniquets should not be removed until the hemorrhage can be reliably controlled by advanced haemostatic agents or until arrival at surgery.

Tourniquet placement on the forearm or leg may not compress the vessels, which lie between the double long bones. Tourniquets on the upper extremity should be placed on the upper arm and if bleeding from the lower extremity is not controlled by a tourniquet on the leg, it should be moved to the thigh where the vessel maybe more easily compressed.

Application for more than 2 hours may increase limb loss.

Don't avoid a tourniquet in order to save a limb, and then lose a life!

• The ***temporary vascular shunt*** serves for temporary revascularization by restoring blood flow to the leg, thereby reducing the ischemic time until the definitive vascular procedure is performed. Recent studies in animal model have confirmed a physiologic beneficial effect of temporary vascular shunts. Early shunting protects the extremity from further ischemic insult and reduces circulating markers of tissue injury.

Methods for final bleeding arrest

After control of bleeding, the vessel is inspected and the best method of repair is decided. Simple repairs of injured blood vessels are preferred. A segment of the injured vessel is resected if required.

• ***Ligation*** of a major vessel should be used only in dying patients.

• ***Lateral suture*** of lacerations and debridement of short segments with end-to-end anastomosis when possible are rapid and effective with few complications.

• ***Reversed venous bypasses*** are used when required, usually if the gap is of more than 3 cm long. Multiple segments of injured vessels may be repaired by short venous segments.

• ***Synthetic grafts should*** be avoided if possible because of a higher risk of thrombosis and infection.

The current teaching is to avoid ***venous repair*** in an unstable or multi-trauma patient. Repair of a vein is of special importance when this vein is the only venous drainage route, as in the popliteal vein.

All vascular repairs should be well covered by viable clean tissues to pre-

vent late infection and bleeding.

5. Indications for blood transfusion

Transfusion Therapy

Blood transfusion

Blood should be added to the resuscitation of patients who have lost 30–40 % of their blood volume. Blood may also be necessary in patients who have not reached this threshold but have ongoing blood loss. Whole blood has a greater risk for immunologic reactions than packed cells. Blood products fielded with forward medical units are predominantly group Packed Red Cells and Fresh Frozen Plazma. Upon reaching a stabilization phase of operations, type-specific packed cells and platelets will be supplied through theater specific cannels.

6. Classification of transfusion solutions and blood products, indications for their use

Blood components and their storage conditions into the table 3.

Table 3 — Blood components and their storage conditions

Product	Storage temperature	Storage time	Indication	Dosage
Packed red blood cells (PRBC)	1–6 °C	35–42 days	Impaired oxygen transfer capacity. Addition of each unit roughly increases hemoglobin by 1 g	According to bleeding
Fresh frozen plasma (FFP)	-18 °C or lower	1 year	Impaired coagulation as results of massive bleeding	2–4 units
Cryoprecipitate	-18 °C or lower	1 year	Low fibrinogen and massive bleeding	10 units for adult
Random donor platelets Single donor platelets (SDP)	20–24 °C 20–24 °C	5 days 5 days	Thrombocytopenia less than 10^5/μL and massive bleeding	4-8 units 1 unit
Deep frozen platelets	-80 °C	2 years	Massive bleeding with no other platelet product available Can be stored for up to 6 h after melting	According to bleeding
Frozen leukoreduced PRBC	-80 °C	10 years	Bleeding Can be stored for up to 14 days after melting	According to bleeding
Deep frozen plasma	-80 °C	7 years	Bleeding	According to bleeding

Storage, self-life, and availability of these products are outlined in Table 4.

Table 4 — Blood Products Available to the Theater

Product	Unit of Issue	Storage	Self Life for Transfusion
Liquid PRBCs	-250mL	35d	35d
Frozen deglycerolized RBCs	-250mL	10y	3d (postwash)
Fresh Frozen Plasma (FFP)	-250mL	ly	24h (postmelt)
Platelet concentrate	-60mL	5d	5d

Crystalloid Solutions

Crystalloids are salt solutions constituted of small molecules that diffuse easily from the intravascular to the interstitial space, which stands for more than 70 % of the extracellular volume. Rapid diffusion of the salts results in a relatively small intravascular effect, only 20–30 % of the infused crystalloid remains intravascular. This dictates a rough 3:1 ratio for blood loss compensation when using crystalloids only.

• ***Lactated ringer*** [(LR) or Hartman's solution] is a slightly hypotonic solution containing near-normal concentrations of sodium (130 meq L), chloride (109 meq L), potassium (4 meq L), and calcium (2.7 meq L). It also contains lactate 20 meq L, which makes it unsuitable for treatment in a situation of lactic acidosis. The calcium in the LR solution binds to the citrate in banked blood and might cause coagulation, thus LR solution should not be used for dilution of banked blood.

• ***Isotonic saline*** [or normal saline (NS)] is a solution of 0.9 % NaCI in water. It contains 154 meq L sodium and 154 meq L chlorine, and has an osmolarity of 308 mOsm L. The high chloride concentration may induce hyperchloremic metabolic acidosis when used in large volumes (i.e. > 3 L).

• ***Hypertonic saline***, both 3 % and 7.5 % NaCl. Resuscitation with small volumes of hypertonic saline is effective for the trauma patient with traumatic brain injury.

• ***Glucose-containing solutions*** — 5 % glucose in water. Following administration, glucose is rapidly metabolized causing unwanted hyperglycemia and, leaving only the free water, it may induce cellular edema and injury. Other than in the treatment of hypoglycemia, dextrose-containing solutions have supported place in fluid resuscitation of the trauma patient.

Colloid Solutions

Colloid solutions contain large molecules that stay inside the capillaries, increasing the oncotic pressure and by altering the balance between intravascular hydrostatic pressure and exlravascular oncotic pressure, attract fluids from the interstitium into the capillaries. Colloid solutions are the most effective means of increasing cardiac output.

• ***Albumin*** — 5 and 25 % heat-treated human albumin solutions are commercially available, with oncotic pressure of 20 and 70 mmHg, respectively. Having a half-life of 16 h, the intravascular volume expansion is about 1:1 using

the 5 % solution, and almost 4:1 when using the 25 % solution. The 25 % solution should be avoided in patients with true hypovolemia.

• ***Dextran*** — 10 % dextran 40 and 6 % dextran 70 solutions, both with an oncotic pressure of 40 mmHg are commercially available. Dextran has the strongest intravascular volume effect. Dextran causes doserelated coagulopathy in doses > 20 mL kg and rarely may induce renal failure due to reduced filtration pressure.

• ***Hetastarch*** — 6 % hydroxyelhyl starch in 0.9 % NaCI solution has an oncotic pressure of 30 mmHg, and hence has an excellent intravascular volume expansion effect of more than 1:1, which lasts for approximately 10 h. The hydroxyethyl starch causes a bleeding tendency caused by impaired platelet function and inhibition of clotting factors; thus its use should be limited to 1,000 ml/24 h.

7. Possible complications of blood transfusion, prevention and treatment

Transfusion reactions may be difficult to recognize in severely or multiply injured casualties. Hemolytic (ABO mismatch) reactions present acutely (< 24 hours) with fever, chills, back pain, dyspnea, and renal failure. Delayed reactions may occur. Transfusion should be halted immediately in all cases, except minor allergic reactions (urticaria, fever, +/- mild bronchospasm), which are treated with diphenhydramine (25–50 mg IV or PO), H-2 blocker, methylprednisolone, +/- epinephrine.

V. TRAUMATIC SHOCK

1. Definition of traumatic shock. Frequency and severity of shock state in the war

Shock is an abnormality of the circulatory system that results in inadequate organ perfusion and tissue oxygenation (end organ hypoperfusion). Shock state is defined as an inability to supply adequate tissue perfusion and oxygenation.

Shock states may be divided by originating etiology to preload, contractility, obstruction, and afterload. It can be divided into hemorrhagic and non-hemorrhagic shock. Non-hemorrhagic shock includes cadiogenic (obstructive) and distributive (septic) shock.

2. Resent views on traumatic shock etiology and pathogenesis

In the immediate, acute phase of the armed conflict trauma patient's treatment, a shock slate is considered hypovolemic unless strong evidence suggests otherwise. **Hypovolemic** shock is further classified by the estimated blood loss. Reversal of hypovolemic shock will be achieved almost always by intravascular volume expansion, e.g., intravenous fluids and/or blood products.

Cardiogenic shock: Pump failure from intrinsic cardiac failure or obstructive cardiac dysfunction from a tension pneumothorax, or cardiac tamponade with distended neck veins, or unilateral absence of breath sounds.

Shock originating from cardiac pump failure may be a result of exacerbation of congestive heart failure, ischemic injury or direct damage to the heart. Hypoxia, acidosis, hypothermia, and electrolyte abnormalities (hypocalcaemia, hyperkalemia) may also contribute to the cardiac depression. Treatment of cardiogenic shock will be achieved by optimization of electrolyte imbalances and heart preload and afterload and, in certain circumstances, by inotropic and chronotropic pharmacologic agents. In unresponsive cases, an intraaortic balloon pump is used to support the failing heart while maintaining coronary perfusion.

Obstructive shock (it`s a kind of cardiogenic shock). Obstruction of the blood flow to or from the heart may be caused by damage to the great vessels, tamponade, tension pneumothorax, pulmonary emboli, or mass effect in the mediastinum. Treatment of obstructive shock is almost always directed towards the underlying cause — pericardial tamponade will need to be evacuated, either by percutaneous or open technique, tension pneumothorax must be drained. Emergent decompression is necessary. When obstruction of the great vessels occurs, specific surgical procedures (pulmonary artery thrombectomy, repair of aortic dissection) may be the only solution.

Distributive (Vasodilatative) shock

Distributive shock originating from inadequate decrease in cardiac afterload (resulted in massive vasodilatation) may be due to metabolic state (e.g., acidosis, electrolyte disturbances, hypoxia, or hypercarbia), loss of sympathetic tone due to nerve or spinal cord injury, or from capillary leak origination from systemic inflammatory response syndrome (SIRS) or sepsis. Treatment of distributive shock is based on pharmacologic vasoconstrictive agents.

3. Clinical shock manifestations in different locations of wounds

Shock is a clinical condition marked by inadequate organ perfusion and tissue oxygenation, manifested by poor skin turgor, pallor, cool extremities, capillary refill greater than 2 seconds, anxiety/obtundation, tachycardia, weak or thready pulse, and hypotension.

Severe extremity trauma has local and systemic, immediate and delayed effects. Local effects are usually immediate and it is easy to recognize and plan treatment. Systemic complications may be occult, delayed in their development, and difficult to diagnose and treat. Obvious local events causing systemic and potentially life-threatening consequences are bleeding and infection that may lead to massive blood transfusion, ARDS, sepsis, septic shock, and multiorgan dysfunction syndrome. Occult events that may start locally and turn into a systemic problem with potentially detrimental complications are deep vein throm-

bosis and pulmonary embolism, rhabdomyolysis and severe renal failure, fat embolism and fat embolism syndrome.

In addition there are systemic problems related to the general severity of the injury, especially in the setting of armed conflict multitrauma. These include aspiration and pneumonia, acute lung injury and ARDS, systemic inflammatory response, sepsis and septic shock, and critical illness polyneuropathy.

4. Value of modern anesthesiology achievements and resuscitation management in treating shock state

5. Modern corrective methods of hemodynamic and respiratory disorders

If the patients develop systemic complications, they may need intensive monitoring and advanced treatment by an intensive care team experienced in the care of severe trauma cases. This may be part of the decision to transfer the patients to a Level 4.

Central nervous system: in comatose or obtunded patients, brain CT or MRI may need to be performed to evaluate events such as edema, hematoma, infection, or infarction.

Respiratory: if severe ARDS develops, the patient may require advanced ventilation devices. Mechanical ventilation should employ lung protective principles according to ARDS protocols.

Cardiovascular: invasive hemodynamic monitoring, such as central venous pressure and invasive blood pressure, should be performed and blood volume should be optimized to maintain adequate organ perfusion. Oxygen delivery should be supported so that central venous saturation will be above 70 %, according to the therapy protocol. Fluid over-load should be avoided to minimize lung and gut edema.

Renal: the goal of urine output should be no more than 0.5–1 cc kg·1h·L If the patient develops acute renal failure renal replacement therapies such as hemodialysis or hemofiltration may need to be performed.

DVT and PE prophylaxis: deep vein thrombosis and pulmonary embolism present a constant threat to the immobile traumatized patient. Preventive measures include anticoagulant medication and mechanical devices, such as pneumatic compression devices and inferior vena cava filters.

6. Content of antishock measures on medical care echelon

Level I and II Immediate first aid delivered at and behind the scene: temporary stop external bleeding, excessive drinking, except for the wounded in the abdomen, head, in the absence of consciousness.

Infusion therapy: at the stage of first aid is necessary to establish a system for intravenous administration of plasma substitutes (0.9 % sodium chloride)

and its continuation during the evacuation, immobilization of the affected area and evacuation at the next stage.

Level III Immediate physician care: triage of casualties with severe blood loss and bleeding (they are referred to the first place in the dressing room), temporary stop external bleeding and tourniquet control, infusion therapy with crystalloids and colloids, bolus of crystalloid solution into the vein (0.8 L 0.9% sodium chloride solution) and colloid (0.4 liters polyglyukin solution), urgent measures to eliminate breathing disorders, evacuation of the next stage.

Level IV and V Definitive medical and surgical care outside the combat zone: complete treatment of traumatic shock is carried out during the division of qualified surgical care in hospital anesthesiology and resuscitation department.

Comprehensive differentiated therapy consists of the following events:

— Measures for the final stop of external and internal bleeding (primary debridement, laparotomy, thoracotomy, etc.);

— Combined therapy of blood loss and hypovolemic shock (infusion-transfusion therapy);

— The stabilization of hemodynamics with glucocorticoids, the normalization of fluid and electrolyte balance are indicated;

— Addressing the causes of asphyxia and control of acute respiratory failure is need.

Performing operation in a state of shock is dangerous, but the shock is not an absolute contraindication for urgent surgery. In some cases emergency surgery is the only thing that can save the life.

7. Early prevention of shock state

Nearly all the wounded with severe concomitant injuries need to conduct transfusion therapy in the prehospital phase and during the evacuation (early antishock measures), and they need primary evacuation.

Compliance with the principles of pre-emptive therapy is that by knowing the sequence of events, begin treatment before detection of clinical signs of complications.

VI. CRUSH SYNDROME

A high-energy injury to a limb not only tears, disrupts, and causes tissue loss but often hide an occult crushed muscle tissue mass, especially when a powerful blast force has acted on the wound. In this open crush wound (OCW) it is difficult to define the border between living and dead muscle.

Another type of crush injury of a limb is the closed crush, typical of casualties crushed under masonry, vehicles, or victims lying unconscious without movement for many hours (mechanical muscle-crush injury — MMCI).

Compartment syndrome may occur with an injury to any fascial compartment. The fascial defect caused by the injury may not be adequate to fully decompress the compartment, and compartment syndrome may still occur.

Reperfusion injury can cause up to 10 l of third-space fluid loss per limb that can precipitate hypovolemic shock.

1. Crush syndrome pathophysiology

When a victim is crushed or trapped with compression on the extremities for a prolonged time, there is the possibility for the crush syndrome (CS), characterized by ischemia and muscle damage or death (rhabdomyolysis). With rhabdomyolysis there is an efflux of potassium, nephrotoxic metabolites, myoglobin, purines, and phosphorous into the circulation, resulting in cardiac and renal dysfunction.

Limb after crush injury

Muscle subjected to weight crushed, bleeding and swelling, appearing of muscle tissue necrosis, and release a large number of metabolites, myoglobin, potassium, creatine, creatinine. Muscle ischemia and hypoxia, acidosis can contribute to potassium ions escape from the cell outward, so that the serum potassium concentration rose rapidly high. Hypovolemic shock appear to peripheral vasoconstriction and hipovolemia, manifested as ischemic kidney, renal blood flow and reduced glomerular filtration (depending mainly on the glomerular efferent artery, renal arteries) can increase the degree of renal ischemia, or necrosis. After the release of necrotic muscle tissue myoglobin appears in a large number of tubular filtration. In acidosis (acidic urine),cases can be deposited in the renal tubules with formation of myoglobin casts, that may increased kidney damage, and finally lead to acute renal failure (ARF).

The systemic causes of death in MMCI are: hypovolemic shock, hyperkalemia, hypocalcemia, metabolic acidosis, disseminated intravascular coagulation and acute myoglobinuric renal failure. This series of events begins with dehydration and is followed by the dangers of the reperfusion of the limb crushed tissues.

Rhabdomyolysis

Definition: Traumatic rhabdomyolysis is caused by the destruction of skeletal muscle mass. This may be caused by direct crush of the muscles or ischemia caused by vascular injury or development of compartment syndrome.

Muscle can survive circulatory ischemia for up to 4 h. Violent crushing destroys muscle immediately; even if the force is insufficient to mangle the muscle tissue, the combination of mechanical force and ischemia will cause muscle death within an hour.

Critical tissue ischemic times into the table 5.

Table 5 — Critical tissue ischemic times

Tissue	Time

Muscle	4 hours
Nerve	8 hours
Fat	13 hours
Skin	24 hours
Bone	4 days

External mechanical pressure destroys the volume regulation of myocytes, whose cytoplasm is negatively charged and is hyperosmotic compared with the extracellular fluid. By disrupting the impermeability of the sarcolemma, extracellular cations and fluid flow down the electrochemical gradient into sarcoplasm. Overhelming the capacity of the cationic extrusion pumps and leading to swelling of the myocytes. Consequently, MMCI causes such gross edema that it may incarcerate much of the extracellular fluid and cause ***hypovolemic shock*** within hours of injury.

At the cellular level depletion of the energy source ATP leads to failure of membrane transporters, influx of calcium, and disruption of cellular membranes. As a result, muscle cell proteins and electrolytes leak into the blood stream. Creatine phosphokinase enzyme (CPK) and myoglobin are the most known and measured. Potassium is the main cellular electrolyte of interest due to its potential harm if renal failure develops.

Acute renal failure (ARF) is the most common and life-threatening complication of rhabdomyolysis, with an incidence of 13–50 % and a reported mortality of up to 59 %. For ARF to develop, two factors have to be present in addition to myoglobinemia: hypovolemia and aciduria. Myoglobin is filtered by the glomerulus into tubular fluid and, in an acidic environment, forms casts together with Tamm-Horsfall proteins that occlude the tubule. In addition, myoglobin contains hemoproteins that generate toxic ferrum radicals that attack tubular cells. If anuria does develop, it may continue for 4-8 weeks before kidney function recovers.

Disseminated Intravascular Coagulation (DIC)

By consensus, DIC is defined as «an acquired syndrome» characterized by the intravascular activation of coagulation with loss of localization arising from different causes.

- ***I Phase***

The coagulation cascade is triggered by the exposure of blood to excessive amounts of thrombotic factors (due to either mechanical tissue injury or endothelial and monocyte activation), which leads to thrombin generation. Thrombin converts fibrinogen to fibrin monomers. Additionally, thrombin is a potent agonist for platelet activation and aggregation. The above-mentioned processes produce either large-vessel thrombosis or, more commonly, microvessel fibrin deposition, which can result in tissue ischemia and organ dysfunction (sludge syndrome).

- ***II Phase***

Thrombin accelerates the proteolysis and depletion of coagulation factors, including fibrinogen, and factors II, V, VIII, and X. The depletion of these factors is a

function of their relatively short plasma half-lives and the rate of synthesis by the liver. Furthermore, thrombin induces endothelial cells to release t-PA, which converts plasminogen to plasmin in the presence of the newly formed fibrin monomer. This production of plasmin results in fibrinolysis, which may lead to further consumption of coagulation factors, thus worsening bleeding (consumption coagulopathy).

- ***III Phase***

Finally, plasma levels of natural anticoagulants, including protein C and antithrombin III, are depleted during DIC and create a long term hypercoagulation (to 3–4 weeks).

2. Classification. Dependence of clinical manifestations from volume of damaged tissue, compression strength and duration

Clinical classification of Crush syndrome, according to the severity of injury, the capacity of muscles involved and the corresponding results of various laboratory tests, is divided into three grade.

- First grade — mild: myoglobinuria test positive, CPK greater than 1 million units (normal 130 units), without acute renal failure and other systemic reactions. It appears in case of defeat small areas of the body — the forearm or shin, with compression of less than 2–3 hours. If you do not perform fasciotomy early after injury reducing tension, systemic reactions may occur.
- Second grade — moderate: myoglobinuria test positive, CPK greater than 2 million units, serum creatinine and urea nitrogen levels without oliguria, effective blood volume loss, hypotension. It appears in case of defeating lager areas of the body — the hip, shoulder or upper limb, with compression of less than 6 hours.
- Third grade — severe: myoglobinuria test positive, CPK increased significantly, oliguria or urinary retention, shock, metabolic acidosis and hyperkalemia. It appears in case of one or two limbs defeated, with compression more than 6 hours.

3. Stage

Stage of crush syndrome development.

1. Stage I — Initial (early) stage (shock, I Phase of the disseminated intravascular coagulation (DIC), I phase of acute renal failure (ARF)).
2. Stage II — Intermediate (rhabdomyolysis, II phase of ARF and DIC).
3. Stage III — Late (advanced) stage of crush syndrome (secondary immunodeficiency, septic disorder, III phase of ARF and DIC).
4. Stage IV — Recovery.

4. Clinic

Signs and Symptoms

The local manifestation is acute muscle-crush compartment syndrome which develops rapidly in and around the crushed muscle as a reperfusion syndrome, and which appears immediately after the extrication of a trapped victim and the consequent removal of the crushing force. An ominous chain of events then unfolds as the crushed vasculature allows the rapid seepage of fluid and plasma proteins into the dead muscle that is sheathed within its inelastic fascial compartment.

Depending on the nature of injury, local signs will reflect severity of damage to muscle tissue. In direct crush of a limb as well as in blunt or penetrating injury, all components of tissue should be considered: bone, blood vessels, nerves, and muscles. Bleeding, hematoma, edema, ischemia, pain, paresthesia, and paralysis may all coexist. Severe edema may lead to compartment syndrome that further aggravates muscle damage.

Systemic: Muscle disruption leads to spillage of potassium, phosphate, urates, creatine phosphokinase enzyme (CPK), and myoglobin into the blood stream. Severe hyperkalemia may lead to cardiac arrhythmia and even cardiac arrest. Myoglobinemia may lead to renal tubular obstruction and acute renal failure. Typically, dark red urine is produced followed by oliguria and anuria. Blood creatinine level rises sharply.

5. Treatment at medical care echelon

Prevention

Once severe muscle injury has occurred, it is of paramount importance to prevent secondary injury. Early reperfusion and early recognition and treatment of compartment syndrome may prevent the development rhabdomyolysis. Treatment of hemorrhagic shock and hypovolemia are of crucial priority. Once rhabdomyolysis has developed, prevention of renal failure becomes the focus. Again, normovolemia in addition to forced diuresis and urine alkalinization may prevent the development of acute kidney injury.

On scene while still trapped

The primary goal of therapy is to prevent acute renal failure in crush syndrome. Suspect, recognize, and treat rhabdomyolysis early in victims of entrapment.

Therapy should be initiated as soon as possible, preferably in the field, while the casualty is still trapped. The injured limb should be cooling with cold water or exposure to cool air. *N. B. Prohibit massage and heat, so as not to aggravate tissue hypoxia.*

In order to counter both the life-threatening hyperkalemia and hypocalcemia, and to prevent myoglobinemia from causing acute renal failure, massive fluid transfusion and alkalinization of the urine must be done as early as possible.

Drinking alkaline beverages (per 8g sodium bicarbonate dissolved in 1000 ~ 2000ml water, plus the amount of sugar and salt), both diuretic, but also alkaline urine to avoid myoglobin deposition in the renal tubules.

Massive infusion must be started as soon as intravenous access has been obtained, even while the victim is still trapped. The sooner fluid replacement is established, the better the chance of avoiding renal failure.

Ideally it is recommended to establish intravenous (IV) access in a free arm or leg vein.

Avoid potassium and lactate containing IV solutions. At least 1 L should be given prior to extrication and up to 1 L/h (for short extrication times) to a maximum of 6–10 L dayly in prolonged entrapments.

As a last resort, amputation maybe necessary for rescue of entrapped casualties (ketamine 2 mg/kg IV for anesthesia and use of proximal tourniquet).

Hospital care:

— foley catheter for urine output monitoring, monitor urine output, blood urea nitrogen (BUN), serum creatinine, and serum electrolytes, establish and maintain urine output > 100 cc/h until pigments have cleared from the urine;

— central venous monitoring may be needed with the larger volumes (may exceed 12 L/d to achieve necessary urine output) of fluid given.

Systemic therapy

1. Correction of hypovolemia/dehydration — fast and aggressive fluid resuscitation to recover normovolemia is the first and most important mode of treatment. Normal saline is the preferred solution although 0.45 % saline or lactated ringer's solution may be used as well.

2. Forced diuresis — maintenance of brisk urine flow of 1–2 mL kg·1h·L may reduce cast formation and tubular obstruction. Loop diuretics such as Furosemide may be used to «push» urine output and prevent oliguric renal failure. It should be noted that loop diuretics may acidify urine and urine pH should be closely monitored. Early use of mannitol at the same time can be added renal vasodilator to relieve spasm and increase renal blood flow.

3. Alkalinization of urine: acidic urine promotes myoglobin cast formation. Large volume crystalloid resuscitation may be enough to prevent urine acidification. If this fails, alkalinization of urine with IV sodium bicarbonate to keep pH > 6.5 has been advocated. Urine pH should be monitored to titrate bicarbonate dosing.

4. Electrolyte abnormalities: Hyperkalemia, hyperphosphatemia, hypocalcemia, hyperuricemia must be corrected. Blood calcium level should be followed and hypocalcemia treated if it is symptomatic.

5. Renal replacement therapies: hemodialysis or hemofiltration are necessary to maintain balance in anuric patients. Fluid balance, acid base status, electrolyte level, and uremic material control are all enabled by daily or continuous

renal replacement therapies. Full nutrition to caloric and protein goals should not be prevented by anuria and are keys to recovery and survival. Dialysis enables nutrition by maintaining fluid and metabolic balance.

Peritoneal dialysis is simple, for most patients also received good results.

Hemoftitration techniques with high flux fillers have also been evaluated for the purpose of removal of myoglobin from the blood. This needs to be done, however, very early after injury as the half-life of myoglobin in blood is very short, about 2–3h. Therefore it is usually ineffective.

Renal System and Electrolytes

Acute renal failure (ARF) is manifested by oliguria (< 0.5 cc/kg/h) and a rise in BUN and creatinine.

The most frequent causes for ARF are:

- Hypovolemia.
- Acute tubular necrosis (ATN) due to hypovolemia.
- Sepsis.
- IV contrast agents.
- Aminoglycoside antibiotics.
- Bilateral renal or ureteral trauma.
- Massive crush soft-tissue injury or compartment syndrome, with resultant rhabdomyolysis and myoglobinuria.

Local therapy

When the skin is torn, laying open the MMCI or when faced with a high-energy wound, especially a blast wound, the treatment is the same as for any severe traumas.

Open wound: radical debridement, repealed as often as necessary (performed under general anesthesia whenever possible); the opening of fascia and extension of the wound in order to remove all dead tissues and achieve adequate drainage is frequently necessary.

Early cut to reduce tension: the decline in tissue pressure compartment area prevents or reduces the incidence of crush syndrome. Even if the muscle is necrotic, drainage sheets by reducing harmful substances can also invade the bloodstream to prevent, reduce body symptoms.

Hence the wound is inspected regularly. Repeated bacterial swabs for direct microscopy and culture are taken at this time.

Amputation: consider in casualties with irreversible muscle necrosis/necrotic extremity.

Amputation indications:

1 — No limb blood flow or severe blood circulation disorder, estimating to keep the latter non-functional.

2 — Severe systemic symptoms that endanger the patient's life.

3 — Limbs concurrent specific infections, such as gas gangrene and so on.

The classical managemenl of an acute compartment syndrome has recently been reviewed; an immediate fasciotomy is performed in order to achieve decompression, thereby improving both local and distal blood supply. The purpose of the fasciotomy is to prevent ischemic muscle death. By converting the closed crushed limb segment into an open wound, profuse bleeding may occur, aggravating coagulopathy and complicating dialysis for myoglobinuric acute renal failure. The only indication for fasciotomy is when the distal pulse is absent and when both direct, major arterial injury and systemic hypotension have been excluded.

A further form of conservative treatment is hyperbaric oxygenation (HBO). This specifically reduces edema and floods the tissues with oxygen dissolved in the extracellular fluid. This oxygen is available to the compromised cells without the energy expenditure otherwise required for its transfer from hemoglobin.

Orthopedic treatment should be primarily conservative. Joints are splinted in a functional position, while active and passive movements are encouraged as soon as pain allows. Finally, ischemic muscle contractures and paralysis caused by the destruction of muscle are corrected by late reconstructive surgery.

Outcomes

Most patients with rhabdomyolysis who survive the complexity of their injury will recover also from its complications. The development of acute anuric renal failure in critically ill patients adds 20–30 % to their chances of over-all mortality. However, rhabdomyolysis-induced renal failure is reversible and most patients regain normal renal function even after 4–8 weeks of anuria and dialysis.

C. SPECIAL TYPES OF MILITARY TRAUMA

VII. INJURIES OF A HEAD

1. Frequency of open and closed injuries

Head injury is one of the most common life-threatening conditions encountered in critical care. Approximately 50 % of all deaths from trauma are associated with a significant head injury, and over 60 % of deaths from motor vehicle accidents are caused by head injury. In the USA, a patient dies of a head injury every 12 minutes.

2. Classification by injury nature and clinical manifestations

Traditional Classification of Head Injuries:

- ***Open*** injuries are the most commonly encountered brain injuries in combat.
- ***Closed*** injuries are seen more often in civilian settings and may have a

higher frequency in military operations.

• ***Scalp*** injuries may be closed (eg, contusion) or open (eg, puncture, laceration, or avulsion). Any scalp injury may be associated with a skull fracture and/or underlying brain injury. Open scalp injuries bleed profusely, even to the point of lethal blood loss, but usually heal well when properly repaired.

• ***Skull fractures*** may be open or closed, and are described as linear, comminuted or depressed. Skull fractures are usually associated with some degree of brain injury, varying from mild concussion to devastating diffuse brain injury and intracranial hematomas. Open skull fractures are prone to infection if they are not properly treated.

Traumatic brain injury- is traditionally classified as resulting from penetrated or closed head injury.

Combat Head Injury Types:

• Blunt (closed head injury).

• Penetrating:

— penetrating with retained fragments;

— perforating;

— guttering (grooving the skull);

— tangential;

— cranial facial degloving (lateral temple, bifrontal).

Closed head injury (CHI) refers to injury where the cranium remains intact and where the dura has not been breached. This is also referred to as blunt head injury. Concussion is the classic example of CHI. Typically CHI is caused by head impact from falls, violence, sports, and more commonly from motor vehicle crashes. In CHI, the disruption in brain function is due to the brain motion and deformation within the cranium, resulting in the classically observed injuries to the brain parenchyma, blood vessels, and fiber tracts.

Penetrating head injury (PHI) involves disruption of the cranium with concomitant dural breach. Typically, this injury results from a projectile that violates the bony skull and then passes through the brain parenchyma.

Pathology is a result of physical disruption of neuronal cells and fiber tracts exacerbated by ischemia and hemorrhage. The events most commonly associated with PHI involved bullet, knife, shrapnel, and the like.

Emerging evidence suggests that there may be another class of head injury, Blast TBI (bTBI). Blast TBI involves disruption of brain function following exposure to an explosion. Patients who suffer bTBI may have an intact calvarium, but not always. Typically, their injuries result from explosive forces transmitted transcalvarially into brain parenchyma. The forces responsible for bTBI are overpressure, electromagnetic energy, acoustics, and others. The agent most commonly associated with bTBI is high explosive ordnance.

3. Clinical course and diagnostics

Traumatic Brain Injury (TBI) is defined as traumatically induced physiological disruption in brain function such as: loss of consciousness (LOC), loss of memory preceding or following injury (amnesia), alteration in mental status at time of injury, and/or focal neurological deficit. TBI severity can be classified as mild, moderate, or severe.

Mild TBI is a brief (< 5 min.) loss of consciousness or awareness. Typically, patients also complain of headache, confusion, and amnesia. Other symptoms that may occur include difficulty concentrating, mood alteration, sleep disturbance, and anxiety. These often resolve within a few hours or days.

A postconcussive syndrome or delayed symptoms may develop. This delayed syndrome can be treated with nonnarcotic analgesics, antidepressants, and conservative therapy. Typically, it will last a few weeks but, in some cases, can persist up to a year or more.

Moderate TBI is associated with a presenting Glasgow Coma Score (GCS) from 9 to 13, often with prolonged loss of consciousness and focal neurological deficit. Patients suffering from moderate TBI will require hospitalization and may need neurosurgical care. They too may develop postconcussive syndrome.

Severe TBI occurs when a patient is obtunded or comatose. They suffer from significant neurological injury to the extent that their presenting GCS is 8 or less, often with abnormal neuroimaging, like a CT scan revealing fracture or hemorrhage. These patients require advanced medical care, which commonly includes airway protection, mechanical ventilation, neurosurgical intervention, intracranial pressure monitoring, and treatment in an intensive care unit setting. Recovery is prolonged and usually incomplete if at all. A significant percentage of severe TBI patients will not survive to 1 year.

Concussion, a subtype of CHI, can also be classified as mild (Grade I), moderate (Grade 2), or severe (Grade 3). Mild concussion is defined as a brief confusion lasting less than 15 minutes but without loss of consciousness. Moderate is when a patient has a period of confusion that lasts longer than 15 minutes but still does not experience loss of consciousness. Severe concussion is whenever there is any loss of consciousness.

4. Complications

The prognosis of brain injuries is good in patients who respond to simple commands, are not deeply unconscious, and do not deteriorate. The prognosis is grave in patients who are rendered immediately comatose (particularly those sustaining penetrating injury) and remain unconscious for a long period of time. Any subsequent neurologic improvement may indicate salvage ability and should prompt reevaluation.

5. Primary care in head injuries on battlefield

Primary principals are basic but vital: to clear the airway, to ensure ade-

quate ventilation, and to assess and treat for shock (excessive fluid administration should be avoided).

Supportive medical therapy is usually sufficient. Cranial decompression is necessary only in rare cases. In the absence of hematomas the use of magnesium has been beneficial. Structures particularly sensitive include optic apparatus, hippocampus, and basal ganglia. Delayed intracranial hemorrhages have been reported. Additionally, these patients have a higher susceptibility to subsequent injury and should be evaluated at a level 4 or 5 facility. Repetitive injury and exposure to blast over-pressure may result in irreversible cognitive deficits.

6. Content of primary medical and surgical care

7. Triage of wounded in a skull

Patient Assessment and Triage

During the primary and secondary assessment, attention should be placed on a complete examination of the scalp and neck. Fragments that enter the cranial vault with a transtemple, transorbital, or cross midline trajectory should be suspected as having associated with neurovascular injuries. Wounds are typically contaminated by hair, dirt, and debris and should be copiously irrigated clean with control of scalp hemorrhage but not at the expense of delaying definitive neurosurgical treatment. Scalp hemorrhage can be controlled with a head wrap, scalp clips, or surgical staples.

The most important assessment is the ***vital signs*****.** Next is the level of consciousness, best measured and recorded by the Glasgow Coma Scale (GCS).

Neurosurgical damage control includes early intracranial pressure (ICP) control; cerebral blood flow (CBF) preservation; and prevention of secondary cerebral injury from hypoxia, hypotension, and hyperthermia.

8. Organization of specialized surgical care

Surgical management

Goals: to prevent infection and to relieve/prevent intracranial hypertension.

Indications for emergent exploration:

- Space-occupying lesions with neurological changes (eg, acute subdural/epidural hematoma, abscess).
- Intracranial hematoma producing > 5 mm midline shift or similar depression of cortex.
- Compound depressed fracture with neurological changes.
- Penetrating injuries with neurological deterioration.

Surgery:

The hematoma should then be gently evacuated with a combination of suction, irrigation, and mechanical removal. Approach to penetrating injury with neurologic changes is aimed at removal of devitalized brain and easily accessible foreign bodies.

Careful hemostasis should be achieved and the dura closed. Excellent results can be achieved with cranioplasty after evacuation out of the theater and a sufficient delay to minimize risk of infection. If a duraplasty is required, pericranium, temporalis fascia, or tensor fascia lata may be used. Tack-up sutures should be placed around the periphery and in the center of the dural exposure to close the dead space and discourage post-operative epidural hematoma formation. You may replace bone flap and be secure with wire or heavy suture.

The galea of the scalp should generally be closed separately with an absorbable suture, and with staples used to close the skin. A subgaleal or epidural drain may be used at the discretion of the surgeon.

Identifying patients with linear ***skull fractures*** also specifies a patient population at greater risk for a major intracranial disaster. As a general principle, all patients with skull fractures should undergo CT scanning and close observation.

An open, depressed skull fracture always requires surgical management. This type of injury represents a potential for bacterial contamination of the CSF pathways and the brain. It requires prompt and meticulous debridement, repair of any dural lacerations, and removal of contaminated skull fragments. Depending on the location, underlying structures, and depth of the depressed fragments, closed depressed skull fractures may be treated conservatively.

A basilar skull fracture is generally a clinical diagnosis rather than a radiographic one. Occasionally, a basilar fracture may be identified on a CT scan, but such a fracture is most commonly diagnosed through physical findings (e.g., hemotympanum, ecchymosis in mastoid region (Battle's sign), periorbital ecchymoses («raccoon eyes»), or a CSF leak from ear or nose). Fractures that cross the cribriform plate, paranasal sinuses, or mastoid cells of the petrous temporal bone and that breach the underlying dura may produce CSF leaks. Patients with suspected basilar skull fracture should be questioned closely for signs or symptoms of CSF otorrhea or rhinorrhea and examined serially to detect a leak or facial nerve paralysis.

VIII. INJURIES OF THE SPINE

1. Classification of open and closed spine and spinal cord injuries

Combat injuries of the spinal column, with or without associated spinal cord injury, differ from those encountered in civilian practice. These injuries are often open, contaminated, and usually associated with other organ injuries.

Classification

Four questions must be considered in the classification and treatment of spinal injuries.

- Is injury open or closed?
- Neurologic status: complete or incomplete or intact.

Complete injury demonstrates no neurologic function ***below the level of injury*** after the period of spinal shock (usually 24–48 h, evidenced by return of the bulbocavernosus reflex).

- Location of the injury: cervical, thoracic, lumbar, or sacral.
- Degree of bony and ligamentous disruption: stable or unstable.

Loss of integrity of two of the three columns results in instability of the spine.

Instability is common following blunt injury of the vertebral column, but is not usually the case with gunshot or fragment wounds of the vertebral column.

2. Symptoms and Diagnostics

Motor Examination

The motor examination is of primary importance. All major muscle groups in each of the four extremities should be serially examined, and the strength of muscle contraction carefully documented. Special care should be taken to determine whether the muscles examined have any contractile function, including that which does not exceed the threshold for movement.

Sensory Examination

The sensory examination, performed next, should be a careful, systematic examination with a variety of modalities, including nociception, light touch, and proprioception. Multimodality testing is essential, because the structures within the spinal cord that conduct sensation decussate at different levels in the central nervous system and travel in different quadrants of the spinal cord. Incomplete spinal cord lesions may produce a combination of sensory findings that depend on the tracts involved. Below the level of a complete lesion, all sensory modalities will be absent. Radicular pain or paresthesias may be present at the level of the injury and may have localizing value.

Patterns of spinal cord injury may be divided into complete and incomplete syndromes. With complete spinal cord injury, no motor or sensory function is detectable below the affected level. Before designating a lesion as complete, the patient should be examined carefully for evidence of preserved perianal sensation or sphincter tone. All sensory modalities, including pain, light touch, proprioception, and vibration should be tested before determining that a patient has a complete spinal cord injury.

In acute spinal cord lesions, the muscles innervated below the level of a complete injury are flaccid and areflexic. In male patients, priapism accompanies spinal cord injury. With more chronic spinal cord injury, muscle tone is increased or spastic below the injury level. Hyperreflexia and up-going toes with plantar stimulation are found on examination. Common radiographic correlates of complete spinal cord injury are bilaterally locked facets or compression fractures of cervical vertebrae suffered in flexion or axial loading injuries. Auto-

nomic dysreflexia may be precipitated by bladder distention, skin stimulation, or bowel distention. Gastric atony and ileus are common.

Incomplete spinal cord injuries require a greater understanding of spinal cord anatomy. Three incomplete cord injury syndromes deserve special attention. Incomplete spinal cord syndromes include Brown-Sequard syndrome, central cord syndrome, and anterior cord syndrome. Rarely are these syndromes present in the pure form; more commonly an incomplete spinal cord injury shares elements of these syndromes.

The Brown-Sequard syndrome follows hemisection of the spinal cord. Because of the different levels of decussation of the anterior spinothalamic tract and the posterior sensory columns, dissociation is noted between the sides of the sensory impairment occurring after this unilateral cord lesion.

A central cord syndrome is produced when the deepest regions of the cervical spinal cord are injured. Characteristically, distal weakness or paralysis of the upper extremities with relative sparing of motor function in the lower extremities is observed.

An anterior cord syndrome may occur after trauma when disc or bone fragments are driven into the spinal canal and compress the ventral spinal cord. On examination, these patients have suffered profound motor loss, as well as loss of pain and temperature sensation below the affected level (ventral spinothalamic tracts). Only posterior column function, light touch, and proprioceptive sensation may remain intact.

Cervical spine

A spinal cord injury above C4 disrupts function of the diaphragm and of the accessory muscles of respiration.Patients with lesions at C5 or below, who have lost accessory and intercostals muscles, have a functional flail chest.

Thoracic and Lumbar Spine

Although the thoracic rib cage contributes considerable rotatory stability, it does not protect completely against injuries. The vascular supply of the spinal cord is most vulnerable between Th-4 and Th-6 where the canal is most narrow. Even minor deformity may result in cord injury. The most common place for compression injuries is at the thoracolumbar junction between Th-10 and L-2.

3. Primary care on battlefield

4. Content of primary medical and surgical care

Optimal management of patients with spinal cord injury requires immediate stabilization of the unstable spinal segment; accurate, well-documented neurologic assessment; and timely recognition and management of medical complications that may limit the patient's recovery.

All potentially unstable cervical spine injuries should be immobilized in a rigid collar (Philadelphia collar), unless «halo» immobilization is required. A

cervical foam collar does not provide any measure of immobilization or security for patients with known or suspected spinal cord injury. They should never be used due to transport immobilization.

Initially, indwelling urinary catheters are recommended. When intermittent catheterization is feasible, urinary bladder volumes should not exceed 400–500 ml.

Closed spinal cord injuries may be treated with an IV corticosteroid if started within 8 hours of injury. ***Penetrating injuries of the spine should NOT receive corticosteroid treatment.***

General Management Considerations

• *Neurogenic shock*

Traumatically induced sympathectomy with spinal cord injury symptoms includes bradycardia and hypotension.

Treatment: Volume resuscitation to maintain systolic BP > 90 mm Hg. May use phenylephrine (50–300 ug/min) or dopamine (2–10 ug/kg/min) to maintain BP.

• *Bladder Dysfunction*

Failure to decompress the bladder may lead to autonomic dysreflexia and a hypertensive crisis. The bladder is emptied by intermittent or indwelling catheterization.

• *Decubitus ulcers*

Skin breakdown begins within 30 minutes in the immobilized hypotensive patient. ***For prolonged transport, the casualty should be removed from the hard spine board and placed on a litter.***

5. Organization of specialized surgical care

Nursing orders also should include frequent neurologic checks. Initially these should be performed every hour in a patient with acute spinal cord injury and no less than twice in an 8 h period in any patient with spinal cord compromise.

Patients who are in cervical traction or those patients with a known unstable spinal fracture or dislocation should have daily lateral cervical spine films to detect changes in alignment. Additionally, plain films should be repeated after any major event in the patients hospital care, for example, an extended transport elsewhere in the hospital, a change in traction alignment or weights, placement in a «halo» vest, or adjustment of any orthotic device.

Patients with spinal cord injury also should be routinely monitored with oxygen saturation monitors, careful fluid balance measurements, and daily weighs. In the acute setting, all patients with spinal cord injury should have a nasogastric tube placed at low continuous suction, and gastric aspirate should be monitored for pH levels. EKG monitoring, arterial pressure monitoring, and Swan-Ganz catheterization also may be indicated.

Physical therapy should be initiated as soon as the patient is medically stable. Early goals are passive range-of-motion exercises, avoiding contractures and establishing a relationship with the providers who may be responsible for

later substantive rehabilitation. The psychological effects of this therapy are of great benefit to the patient.

IX. GUNSHOT WOUNDS AND CLOSED CHEST INJURIES

1. Frequency and classification of chest injuries

About 15 % of war injuries involve the chest. Of those, 10 % are superficial (soft tissue only) and requiring only basic wound treatment. The remaining 90 % of chest injuries are almost all penetrating.

Those injuries involving the central column of the chest (heart, great vessels, pulmonary hilum) are generally fatal on the battlefield. Injuries of the lung parenchyma (the vast majority) can be managed by the insertion of a chest tube and basic wound treatment. Although penetrating injuries are most common, blunt chest trauma may occur and can result in disruption of the contents of the thorax as well as injury to the chest wall itself. Blast injuries can result in the rupture of air-filled structures (the lung) as well as penetrating injuries from fragments.

2. Clinical manifestations of different kinds in chest injuries, their diagnostics

Chest trauma can be classified as blunt or penetrating. Blunt and penetrating injuries have different pathophysiology and clinical courses.

Specific types of chest trauma include:

Injuries to the chest wall:

- Chest wall contusions or hematomas.
- Rib fractures.
- Flail chest.
- Sternal fractures.
- Fractures of the shoulder girdle.

Pulmonary injury (injury to the lung) and injuries involving the pleural space:

- Pulmonary contusion.
- Pulmonary laceration.
- Pneumothorax.
- Hemothorax.
- Hemopneumothorax.

Injury to the airways:

— Tracheobronchial tear

Cardiac injury:

- Pericardial tamponade.
- Myocardial contusion.

• Traumatic arrest.

Blood vessel injuries:

• Traumatic aortic rupture, thoracic aorta injury, aortic dissection.

And injuries to other structures within the torso:

• Esophageal injury (Boerhaave syndrome).

• Diaphragm injury.

3. Hemothorax

The most common causes of chest pain related to hemothorax are blunt and penetrating traumas:

• Blunt trauma can result in broken ribs that slice into lung tissue and cause bleeding. Falls, car accidents and contact sports injuries can all cause blunt trauma.

• Penetrating trauma describes such injuries as knife wounds, gunshot wounds or wounds caused by other sharp objects. Penetrating trauma can cut into the lung tissue, causing bleeding and hemothorax.

Common hemothorax symptoms include:

• anxiety;

• respiratory failure;

• restlessness;

• shortness of breath;

• tachycardia (rapid heart rate);

• varying degrees of chest pain.

Hemothorax treatment focuses on stabilizing the patient, locating the source of chest cavity bleeding and stopping the bleeding:

• treatment of the underlying cause;

• pulmonary toilet;

• needle aspiration;

• chest tubes — tube thoracostomy drainage;

• underwater seal suction.

Hemothorax treatment aims to drain existing blood out of the pleural cavity by a chest tube or needle aspiration. The return of blood may indicate a significant intrathoracic injury. Generally, if the wound was sustained within the past hour, *the immediate return of 1,500 cc of blood mandates thoracotomy*. With less blood initially, but a continued loss *of 200 cc/hour for over 4 hours*, thoracotomy is indicated. Casualties with massive thoracic hemorrhage require damage control techniques.

4. Pneumothorax (closed, open, tension)

Close pneumothorax. Traumatic pneumothorax have been found to occur in up to half of all cases of chest traumas, with only rib fractures being a more common problem in this group. On physical examination, breath sounds (audi-

ble using a stethoscope) may be diminished on the affected side, partly because air in the pleural space dampens the transmission of sound. Percussion of the chest may be perceived as hyperresonant (like a booming drum), and vocal resonance and tactile fremitus can both be noticeably decreased. Importantly, the volume of the pneumothorax can show limited correlation with the intensity of the symptoms experienced by the injured, and physical signs may not be apparent if the pneumothorax is relatively small.

Open traumatic pneumothorax (hole in chest wall) occurs most commonly when the chest wall is pierced, such as when a stab wound or gunshot wound allows air to enter the pleural space. Open pneumothorax is treated by placing a chest tube and sealing the hole. Alternatives include one-way valve chest dressings or a square piece of plastic dressing taped to the chest on three sides.

Tension Pneumothorax

A patient with a known chest injury presenting with an open airway and difficulty breathing has a tension pneumothorax until proven otherwise and requires rapid decompression and the insertion of a chest tube.

Associated Clinical Features

A ***tension pneumothorax*** results when air is able to enter but not exit the pleural space. Air in the pleural space accumulates and compresses the ipsilateral lung and vena cava, with a rapid decrease in cardiac output. The contralateral lung may suffer ventilation/perfusion mismatch.

Subcutaneous air, tracheal deviation, jugulovenous distention (JVD), and diminished or hyperresonant ipsilateral breath sounds can be clues. Subcutaneous emphysema may be visible on the neck and chest and is easily diagnosed by palpation.

The immediate recognition and treatment of tension pneumothorax is the single most important and life-saving intervention in the treatment of chest injuries in combat. Distended neck veins, tracheal shift, decreased breath sounds, and hyperresonance in the affected hemithorax, and hypotension are the cardinal signs. None or all may be present.

In cases of tension pneumothorax, *immediate decompression with a large bore needle is lifesaving.* The released air from a tension pneumothorax can be heard escaping from a needle thoracostomy**.** Video-assisted thoracoscopy or thoracostomy may be necessary to eliminate the site of the air leak. A haemothorax may accompany and/or complicate a traumatic pneumothorax. The presence of a haemothorax necessitates chest tube placement. If bleeding continues, exploration of the thoracic cavity may be necessary to achieve haemostasis.

5. Subcutaneous and mediastinal emphysema

6. Pathophysiology features of these complications

Subcutaneous emphysema usually occurs on the chest, neck and face, where it is able to travel from the chest cavity along the fascia. Particularly in the chest and neck, air may become trapped as a result of penetrating trauma (e.g. gunshot or stab wounds) or blunt trauma. Its most common causes are pneumothorax. Signs and symptoms of subcutaneous emphysema vary based on the cause, but it is often associated with swelling of the neck and chest pain, and may also involve sore throat, neck pain, difficulty swallowing, wheezing and difficulty breathing. Subcutaneous emphysema has a characteristic crackling feel to the touch, a sensation that has been described as similar to touching Rice Crisps; this sensation of air under the skin is known as subcutaneous crepitation.

It is possible to identify the following forms of subcutaneous emphysema:

- Local (within the limits of chest).
- Spread,(within the limits of neck, chest and abdomen).
- Massive subcutaneous emphysema (if it spread on face, leg and scrotum).

When the amount of air pushed out of the airways or lung becomes massive, the eyes will be obscured by the swollen eyelids making the patient sightless.

Air in subcutaneous tissue does not usually pose a lethal threat; small amounts of air are reabsorbed by the body. In severe cases of subcutaneous emphysema, catheters can be placed in the subcutaneous tissue to release the air. Small cuts, or «blow holes», may be made in the skin to release the gas.

Pneumomediastinum (also known as ***mediastinal emphysema***) is a condition in which air is present in the mediastinum. It is uncommon and occurs when air leaks into the mediastinum due to chest injury (esophageal rupture, injury of a lungs, airways or bowel).

Signs and symptoms of the mediastinal emphysema

The main symptom is usually severe central chest pain. Other symptoms include laboured breathing, voice distortion (as with helium) and subcutaneous emphysema. It is often recognized on auscultation by a «crunching» sound timed with the cardiac cycle (Hamman's crunch).

The tissues in the mediastinum will slowly resorb the air in the cavity so most pneumomediastinums are treated conservatively. Breathing high flow oxygen will increase the absorption of the air. If the air is under pressure and compressing the heart, a needle may be inserted into the cavity, releasing the air. Surgery may be needed to repair the hole in the trachea, esophagus or bowel.

Cardiac Tamponade

Beck's triad of acute cardiac tamponade includes jugulovenous distention (JVD) from an elevated central venous pressure (CVP), hypotension, and muffled heart sounds. In trauma, only one-third of patients with cardiac tamponade demonstrate this classic triad, although 90 % have at least one of the signs. The simultaneous appearance of all three physical signs is a late manifestation of tamponade and

usually seen just prior to cardiac arrest. Other symptoms include shortness of breath, orthopnea, dyspnea on exertion, syncope, and symptoms of inadequate perfusion.

Distended neck veins (may be absent with significant blood loss) in the presence of clear breath sounds and hypotension indicate the possibility of life-threatening cardiac tamponade.

Fluid resuscitation may temporarily stabilize a patient in tamponade.

Pericardiocentesis is only a stop gap measure on the way to definitive surgical repair (any pericardial blood mandates median sternotomy/thoracotomy). Extra dermal (ED) pericardiocentesis is a diagnostic and resuscitative procedure in patients with suspected cardiac tamponade. Goals of ED pericardiocentesis include identification of pericardial effusion and removal of blood from the pericardial space to relieve the tamponade.

Surgical Management

Most penetrating chest injuries reaching medical attention are adequately treated with tube thoracostomy (chest tube) alone.

Tube thoracostomy (chest tube) indications:

- Known or suspected tension pneumothorax.
- Pneumothorax (including open).
- Hemothorax.
- Any penetrating chest injury requiring transport.

Surgical Management of Specific Injuries

Vascular

Initially, holes in vessels should be digitally occluded. Stopgap measures include placing Fogarty or Foley catheters, side-biting clamps, or in the case of venous injuries, sponge sticks.

Heart

The usual result of high-velocity injuries to the heart is irreparable destruction of the muscle. Isolated punctures of the heart should be exposed (opening the pericardium) and occluded by finger pressure. Other methods include the use of a Foley catheter or skin staples.

Lung

Tube thoracostomy alone is adequate treatment for most simple lung parenchymal injuries.

Large air leaks not responding to chest tubes or that do not allow adequate ventilation will require open repair (see tracheobronchial tree below). Posterolateral thoracotomy is preferred for isolated lung injuries. Anterior thoracotomy may also be used.

Tracheobronchial tree

Suspect the diagnosis with massive air leak, frothy hemoptysis, and pneumomediastinum. Confirm by bronchoscopy. Airway control is paramount. Median sternotomy is best approach. Repair over endotracheal tube with absorbable suture — may require segmental resection. Bolster with pleural or intercostal muscle flap.

Esophagus

Isolated thoracic esophageal injuries are exceedingly rare. They will usually be diagnosed incidentally associated with other intrathoracic injuries.

Surgery is the definitive treatment.

For stable patients in a forward location, chest tube drainage and a nasogastric tube placed above the level of injury is a temporizing measure.

Diaphragm

All injuries of the diaphragm should be closed.

7. Acute respiratory distress syndrome or acute lung injury

Acute lung injury (ALI), first described by Ashbaugh in 1967, is a disorder with varying degrees of pulmonary cellular damage (parenchymal and vascular) that alters alveolar capillary membrane permeability, produces accumulation of noncardiogenic extravascular lung water, and results in hypoxic respiratory failure. Patients with this syndrome have dyspnea, refractory hypoxia, reduced lung compliance, and diffuse radiologic changes. Acute respiratory distress syndrome (ARDS) is a more severe subset of ALI characterized by dyspnea, refractory hypoxemia, decreased lung compliance, and diffuse radiologic changes that occur in the absence of cardiac failure or chronic lung disease.

A stimulus, such as trauma or sepsis, causes the release of inflammatory cytokines, which is a strong chemoattractant for neutrophils and which triggers the acute inflammation within the lungs.

The neutrophils that are attracted by interleukin-8 (IL-8) and other stimuli accumulate in the air spaces and interstitium of the lungs. When activated- they release toxic mediators, such as reactive oxygen species, platelet aggregation factors, metabolites of arachidonic acid, and proteases. These substances cause damage to the capillary endothelium and alveolar epithelium. As normal barriers are lost, protein escapes through the alveolar capillary membrane into the airspace of the lung. The resultant pulmonary edema causes surfactant dysfunction and necrosis of type I alveolar cells. Type II alveolar cells undergo hyperplasia. Damage to type I alveolar cells increases entry of fluid into the alveoli and de-

creases clearance of fluid from the alveoli. Damage to type II alveolar cells is associated with decreased production of surfactant with resultant decreased compliance and alveolar collapse. The result is diffuse alveolar damage, which is seen histologically during the early stages of ALI/ARDS.

The management of ALI and ARDS is mainly supportive. Treatment of the underlying cause is the initial step in the management of this syndrome. At this time there is no known treatment exists to reverse increased vascular permeability changes or fibrosis occurring with ALI.

The need to assess volume status and proceed with fluid resuscitation is an essential basic step in managing ARDS. Intravascular volume must be adequate to maintain gas exchange, oxygen delivery, and hemodynamic stability.

8. Late complications of chest gunshot wounds

After prolonged exposure of blood in the pleural cavity, fibrin clots are gradually organizing in the connective tissue with formation of massive fibrous tissue. Fibrothorax (developed irreversible process) contain infectious foci, supporting intoxication. In the absence of a well-organized triage and medical evacuation, such processes as abscess, gangrene of the lung, pneumonia and sepsis determine high level of lethality.

9. Closed chest injuries

Traumatic asphyxia

Traumatic asphyxia, or Perte's syndrome, is a medical emergency caused by an intense compression of the thoracic cavity, causing venous back-flow. Traumatic asphyxia occurs when a powerful compressive force is applied to the thoracic cavity. This is most often seen in motor vehicle accidents, as well as industrial and blast accidents, a crush by an object that compresses the chest or upper abdomen.

Traumatic asphyxia is due to a sudden increase in intrathoracic pressure against a closed glottis. The elevated pressure is transmitted to the veins, venules, and capillaries of the head, neck, upper extremities, and upper torso, resulting in capillary rupture (craniocervical cyanosis). Survivors demonstrate plethora, ecchymoses, petechiae, and subconjunctival hemorrhages. Severe cases may produce CNS injury with seizures, posturing, and paraplegia. It’s important to keep in mind that many of the classic signs and symptoms of traumatic asphyxia may not be initially present at the scene.

Traumatic asphyxia has a good prognosis. Supportive treatment such as oxygenation and elevation of the head to 30° is usually sufficient in the management of these patients. However, specific treatments may be needed for the associated injuries.

10. Multiple rib fractures with formation of «costal valve»

Rib fractures

A rib fracture is a break or fracture in one or more of the bones making up the rib cage. Fractures of the first and second ribs may be associated with head and facial injuries. Rib fractures are usually quite painful because the ribs have to move to allow for breathing. There is no specific treatment for rib fractures, but various supportive measures can be taken. In simple rib fractures, pain can lead to reduced movement and cough suppression; this can contribute to formation of secondary chest infection. Adequate analgesia can avoid this.

Flail chest (entire segment of the chest wall floating due to fractures of a block of ribs, with double fractures on each rib) will require treatment (either airway intubation or observation) based on the severity of the underlying lung injury. In cases where intubation is not required, repeated intercostals nerve blocks with a long-acting local anesthetic may be very helpful in relieving pain and limiting atelectasis and other pulmonary complications.

X. ABDOMEN GUNSHOT WOUNDS AND CLOSED INJURIES

1. Frequency and classification of abdominal injuries

Abdominal traumas, both blunt and penetrating, can lead to occult injury that can be devastating or fatal if not treated. In the unstable patient with abdominal injury, the decision to operate is usually straight forward and should be acted on as soon as it is made.

Abdominal gunshot wounds can be divided into five discrete anatomical regions: anterior abdomen, back, buttocks, transpelvic and thoracoabdominal, all with different implications in terms of injury and management.

Abdomen gunshot wound, depending on the wound channel can be blind, cross-cutting and tangents; and depending on the damage to the parietal peritoneum can be penetrating and non-penetrative.

Blunt abdominal injuries can be divided into three main groups:

I — damage to the anterior abdominal wall (hematoma, rupture of the muscles);

II — damage to internal organs of the abdomen (hollow and solid);

III — damage to the organs of the retroperitoneal space.

In penetrating abdominal trauma due to gunshot wounds, the most commonly injured organs are as follows:

- Small bowel (50 %).
- Colon (40 %).
- Liver (30 %).
- Abdominal vascular structures (25 %).

Penetrating abdominal trauma typically involves the violation of the abdominal cavity by a gunshot wound or stab wound.

Stab wounds are caused by penetration of the abdominal wall by a sharp object. This type of wound generally has a more predictable pattern of organ injury. However, occult injuries can be overlooked, resulting in devastating complications.

Injury patterns differ depending on the weapon. Low-velocity stab wounds are generally less destructive and have a lower degree of morbidity and mortality than gunshot wounds and shotgun blasts. Gunshot wounds and other projectiles have a higher degree of energy and produce fragmentation and cavitations, resulting in greater morbidity.

In mine-explosive wounds of abdominal injuries are of two types, corresponding to two damaging factors of mine-explosive wounds:

a) shrapnel;

b) closed injuries of the abdominal cavity as a result of the shock wave.

Anatomic classification is important in guiding the clinician's suspicion for specific organ injury. Intraperitoneal abdominal organs include the solid organs (i.e. spleen, liver) and the hollow viscus organs (i.e. stomach, ileum, jejunum, transverse colon).

Retroperitoneal organs include the duodenum, pancreas, kidneys, ureters, urinary bladder, ascending and descending colon, major abdominal vessels, and rectum.

2. Clinical manifestations of open abdominal injury

3. Symptoms of abdominal penetrating wounds

One or more of the intra-abdominal organs may be injured in abdominal trauma. The characteristics of the injury are determined which organ or organs are injured. Intra-abdominal trauma can be associated with rib fractures, vertebral fractures, pelvic fractures, and injuries to the abdominal wall.

Early indications of blunt abdominal trauma include nausea, vomiting, and fever. Blood in the urine is another sign. The injury may present with abdominal pain, tenderness, distension, or rigidity to the touch, and bowel sounds may be diminished or absent. Abdominal guarding is a tensing of the abdominal wall muscles to guard inflamed organs within the abdomen. Pneumoperitoneum (air or gas in the abdominal cavity) may be an indication of rupture of a hollow organ.

The clinical picture of abdominal penetrating wounds depends on which organ (hollow or solid) is damaged or there is a combination of these injuries. Isolated damage to solid organs is rare, more common in the combination of damaged hollow and solid organs. In penetrating injuries, an evisceration (protrusion of internal organs out of a wound) may be present.

Wounds of the hollow organs are characterized by symptoms of rapidly evolving peritonitis, and injuries of the liver, spleen, blood vessels due to intensive intraperitoneal bleeding accompanied by a symptoms clinic of acute blood loss.

The main symptoms of kidney damage include: hematuria, perinephric hematoma (urohematoma), excretion urine from the wound.

4. Shock, hemorrhage and peritonitis, and their roles in penetrating wound outcomes

Hemorrhage and systemic infection are the main causes of deaths that result from abdominal trauma.

Abdominal trauma can be life threatening because abdominal organs, especially those in the retroperitoneal space, can bleed profusely, and the space can hold a great deal of blood. Solid abdominal organs, such as the liver and kidneys, bleed profusely when cut or torn, as do major blood vessels such as the aorta and vena cava. If it does not stop in the next few hours it leads to the death from hemorrhagic shock.

Hollow organs such as the stomach, while not as likely to result in shock from profuse bleeding, present a serious risk of infection (peritonitis), especially if such an injury is not treated properly. Gastrointestinal organs such as the bowel can spill their contents into the abdominal cavity. In gunshot wounds of the abdomen with hollow organ injury take place a massive one-stage microbial contamination of the abdominal cavity.

5. Clinical picture of closed abdominal injuries

Stomach Injuries

The stomach is a vascular organ and will do well after almost any repair. Always enter the lesser sac to determine posterior wall injuries. Encircle the distal esophagus with a Penrose drain to provide traction and improve visibility in high midline injuries. Minimally debride and primarily close stomach defects.

Duodenum Injuries

Injuries to the Duodenum are associated with massive upper abdominal trauma. Early consideration for damage control surgery should be considered. Missed injuries of the duodenum have devastating morbidity.

Pancreas Injuries

The pancreas may be injured in abdominal trauma, for example by laceration or contusion. Indications that the pancreas is injured include enlargement and the presence of fluid around the pancreas. Any injury to the pancreas/duct requires drainage. Even if ductal injury is not identified, it should be presumed and drained. Resect clearly nonviable pancreatic body/tail tissue.

Liver Injuries

Liver injuries present a serious risk for shock because the liver tissue is delicate and has a large blood supply and capacity. Most liver injuries can be successfully treated with direct pressure and packing followed by aggressive resuscitation and correction of coagulopathy. Generous exposure is required and

should be gained early and aggressively. Avoidance of coagulopathy, hypothermia and acidosis is essential in successful management of major liver injuries.

Spleen Injuries

Spleen is the most common damaged organ in blunt abdominal trauma. Because of the spleen's ability to bleed profusely, a ruptured spleen can be life threatening, resulting in shock. Spleen salvage has no place in combat surgery. Spleen injury should prompt exploration for associated diaphragm, stomach, pancreatic, and renal injuries.

Kidneys Injuries

The kidneys may also be injured; they are somewhat but not completely protected by the ribs. Kidney lacerations and contusions may also occur. Blunt abdominal trauma, may be associated with bloody urine. Kidney lacerations may be associated with urinoma, leakage of urine into the abdomen. A shattered kidney is one with multiple lacerations and an associated fragmentation of the kidney tissue.

Small-Bowel Injuries

The bowel may be perforated. Gas within the abdominal cavity is understood to be a diagnostic sign of bowel perforation. Debride wound edges to freshly bleeding tissue. Close enterotomies in one or two layers (skin stapler is a rapid alternative). With multiple enterotomies to one segment of less than 50 % of small-bowel length, perform single resection with primary anastomosis. Avoid multiple resections.

Colon Injury

Simple, isolated colon injuries are uncommon. In indigenous populations and enemy combatants (e.g. patients who cannot be evacuated), diversion with colostomy should be the procedure of choice, especially at Level 2. The often poor nutritional status of these populations does not support primary repair. The presence of any of the complicating factors mandates colostomy. Simple, isolated colon injuries should be repaired primarily. For complex injuries, ***strongly consider colostomy/diversion.***

6. Diagnostics of penetrating wounds and closed abdominal injuries

Initial evaluation of intra-peritoneal trauma:

- Chest X-ray. An erect chest radiograph may identify sub-diaphragmatic air. This must be interpreted with some caution in the absence of peritonitis, as air may be entrained into the peritoneal cavity with a stab or gunshot wound. However it certainly signals of peritoneal penetration and warrants further investigation.
- Nasogastric Tube. Blood drained from the stomach will identify gastric injury.
- Urinary catheter. Macroscopic hematuria indicates a renal or bladder injury. Microscopic injury suggests but is not pathognomonic of ureteric injury.
- Rectal examination. Rectal blood indicates a rectal or sigmoid penetration. Protoscopy & sigmoidoscopy should be performed.

Wound Exploration

Blast injuries and improvised explosive devices (IEDs) create many low-velocity fragments that may penetrate the skin but not the abdominal cavity. Operative wound exploration in the stable patient with precious careful examination can help determine the need for formal exploratory laparotomy.

When possible wound exploration should be performed in the operating room with adequate instruments and lighting.

Diagnostic Peritoneal Lavage

DPL has been a mainstay of blunt abdominal trauma diagnosis for many years. The only reliable information obtained from DPL is the aspiration of 10 cc of gross blood. Gross blood aspiration is the most infrequently positive criterion of DPL.

7. Treatment of abdominal injuries at medical care echelon

Initial treatment involves stabilizing the patient enough to ensure adequate airway, breathing, and circulation, and identifying other injuries (control hemorrhage).Giving broad-spectrum antibiotic pre-op, continue for 24 hours. Redosing short half-life antibiotics intraoperatively and consider redosing antibiotics with large amounts of blood loss.

The goal of any algorithm for penetrating abdominal trauma should be to identify injuries requiring surgical repair, and avoid unnecessary laparotomy with its associated morbidity.

The presentation of patients who sustain abdominal gunshot wounds can be separated into three distinct groups. The first group includes patients who present in cardiac arrest or near cardiac arrest. Though the mortality in these patients is higher than 90 %, prompt emergency department thoracotomy may lead to successful outcomes. The second group includes patients who present with severe hypotension or frank peritonitis. These patients require immediate exploratory laparotomy, usually without any investigations. A plain abdominal radiograph to locate missiles or a trauma ultrasound as a confirmatory test for hemoperitoneum may be useful. They are immediately available and do not delay laparotomy. Once the decision has been made to go to the operating room, time becomes a critical factor. The third group consists of patients who present to the emergency room with hemodynamic stability and absence of diffuse abdominal tenderness. Such patients are candidates for selective nonoperative management.

Surgical exploration is necessary for people with penetrating injuries and signs of peritonitis or shock. Surgery may be needed to repair injured organs. Laparotomy is often performed in blunt abdominal trauma, and is urgently required if an abdominal injury causes a large, potentially deadly bleeding.

Patients with clinical signs of peritonitis, or with evisceration of bowel should be taken immediately to the operating room.

Patients with penetrating trauma who are haemodynamically unstable require immediate operation. Haemodynamically unstable includes non-responders and transient-responders to initial small-volume fluid bolus administration. Patients should be taken immediately to the operating room, without further unnecessary investigations or interventions.

Penetrating thoracoabdominal wounds provide a serious challenge to the trauma surgeon as they imply potential injuries to the chest, the abdomen or both. Besides identifying the need for operation, the dilemma is which body cavity should be explored. If both cavities require exploration, the sequence in which they are explored becomes a critical decision. Thoracoabdominal injuries traditionally have a high negative laparotomy rate and often, incorrect sequencing occurs.

Laparotomy is performed through a midline incision. When wide exposure is needed, the incision extended superiorly just lateral to the xiphoid process and inferior to the symphysis pubis.

However, intra-abdominal injuries are also frequently successfully treated nonoperatively. In summary, gunshot wounds to the anterior and posterior abdomen can be safely managed nonoperatively. In the absence of abdominal tenderness, hemodynamic instability, or factors clouding the abdominal exam (head injury, spinal cord injury, heavy intoxication, anesthesia for other operations), such patients can be selected for careful observation. The key to successful selective nonoperative management is continuous monitoring and frequent clinical examination by physicians familiar with trauma. Diagnostic tests can be ordered as indicated, but clinical examination is the most accurate tool to identify patients requiring an operation or not. By a policy of selective nonoperative management, the negative laparotomy rate can be reduced to approximately 10 %.

XI. PELVIS AND ABDOMEN GUNSHOT WOUNDS AND CLOSED INJURIES

1. Wounds and closed injuries of pelvis and pelvic organs

2. Classification of pelvis injuries

3. Complications in gunshot pelvis injuries

Injury of the pelvis can be divided into open and blunt. ***Blunt injuries*** may be associated with major hemorrhage and early mortality.

Among the blind gunshot wounds of the pelvis are more common than cross-cutting and shearing. ***Penetrating injuries*** to the skeletal pelvis are usually associated with abdominopelvic organ injury.

The clinic gunshot injuries of the pelvis should consider the possibility of damage to all six systems involved in the formation of the pelvis:

- bones;
- cardio-vascular system;
- nervous system;
- digestive system;
- urinary system;
- the reproductive system.

Vascular injury may result from laceration of a vessel by a bony fragment, laceration of a vessel as a direct consequence of external traumatic forces, and sudden distortion with shearing of vessels at their point of origin.

Pelvic ring injuries can cause large retroperitoneal hematoma, which may mimic abdominal injuries, and are particularly dangerous due to the enormous potential space of more than 4l. Leading to lack of auto-tamponade; exsanguinating hemorrhage is the major cause of death in multiply injured patients with pelvic ring disruptions. Immediate recognition of hemorrhage and effective control of bleeding are vital to survival.

Arterial bleeding is known to occur in pelvic fracture patients both with and without initial hypotension. The arteries most frequently injured, in descending order, are the superior gluteal, internal pudendal, obturator, and lateral sacral arteries. An open surgical exploration to gain access to the retroperitoneum with an attempt to ligate the injured arteries is almost always ineffective. It may, on the other hand, aggravate the condition by disrupting the retroperitoneal tamponade and place the patient at a greater risk of dying from exsanguination.

Transpelvic wounds are defined as a missile trajectory between the iliac crest and the perineum. Penetrating wounds that traverse this region provide a serious challenge in management. The complex pelvic anatomy, combined with the retroperitoneal and extraperitoneal location of its structures, makes the evaluation of this region quite difficult. Gunshot wounds in this area are thought to have a high serious abdominal injury. Carefully performed physical exam is the cornerstone to the management of gunshot wounds to this area. Based on clinical findings, patients can be appropriately triaged to either operation or close observation.

In case of ***buttocks injury*** although local adipose tissue and muscle may act as a protective shield, significant intra-abdominal injury may occur. Organs such as the rectum, bladder and ureters are at particular risk, which may not manifest clinically upon presentation.

4. Symptoms and diagnostics of pelvis gunshot fractures with or without damage to pelvic organs

Fractures of the pelvis

The pelvic fractures are classified according to the Young classification.
Type of pelvic fracture:

- Pelvic ring injuries

— antero-posterior compression I, II, III;
— lateral compression I, II, III;
— vertical shear.

- Iliac bone fractures, solitary.
- Ramus pubis fractures solitary.
- Ramus pubis + acetabulum.
- Acetabulum.

Palpation and percussion allowed determining the fracture site. Evaluation should consist of:

1. Feeling the pelvic brim for a step off.
2. Palpating the iliac wings.
3. Examining the rectum for the position of the prostate and for the presence of blood rectal or perineal laceration. In a female, a vaginal examination is also important. Female urethral lacerations most commonly occur with an anterior vaginal laceration. Posterior vaginal lacerations are commonly associated with rectal tears.
4. Urinalysis for hematuria.
5. X-ray of the pelvis (if clinical diagnosis difficult).

Pelvic fractures are encountered especially in blunt trauma and are often complicated by massive hemorrhage and urologic injury.

The hypotension in the patient with pelvic fractures represents a difficult problem. This hypotension in the vast majority of cases is due to bleeding from the fracture site, adjacent soft tissue or vessels injuries mostly venous vessels. This bleeding is controlled by stabilizing the fractures (external pelvic fixation) especially in the unstable patient, allowing the retroperitoneum to tamponade. If bleeding persists and other sources of bleeding have been ruled out in the chest and in the abdomen, angiography with possible embolisation should be considered.

Injuries to organs within the pelvic cavity can occur with or without pelvic fractures. Injury of the hollow organs is characterized by symptoms of feces peritonitis rapidly evolving, and for the injury of the uterus, blood vessels due to intraperitoneal bleeding — clinic of acute blood loss.

Genitourinary (GU) injuries constitute approximately 5 % of the total injuries encountered in combat. Their treatment adheres to established surgical principles of hemostasis, debridement, and drainage. Proper radiographic evaluation prior to surgery may replace extensive retroperitoneal exploration at the time of laparotomy in the diagnosis of serious GU injuries.

GU wounds, aside from injuries of the external genitalia, are typically associated with serious visceral injury.

Renal Injuries

Most renal injuries, except for those of the renal pedicle, are not acutely life threatening. Undiagnosed or improperly treated injuries, however, may cause significant morbidity.

While the vast majority of blunt renal injuries will heal uneventfully with observation and conservative therapy, a significant number of renal injuries in combat will come from penetrating wounds and require exploration.

Hematuria is usually present in patients with renal trauma, and gross hematuria in the adult patient is concerning for a significant injury. *The absence of hematuria, however, does not exclude renal trauma.*

Renal trauma is categorized by the extent of kidney damage.

Minor injuries

Consist of renal contusions or shallow cortical lacerations. Hydration, antibiotics, and bed rest are the cornerstones of successful nonoperative management.

Major injuries

Consist of deep cortical lacerations (with or without urinary extravasation), shattered kidneys, renal vascular pedicle injuries, or total avulsion of the renal pelvis. Most cases will require a laparotomy for evaluation and repair of concurrent intraperitoneal injuries. Kidney preservation should be considered if at all possible, although total nephrectomy may be required for the severely damaged kidney or the unstable patient.

Ureteral Injuries

Ureteral injuries are rare but are frequently overlooked when not appropriately considered. They are more likely in cases of retroperitoneal hematoma and injuries of the fixed portions of the colon, duodenum, and spleen.

Isolated ureteral injuries are rare and usually occur in conjunction with other significant injuries. They can represent a difficult diagnostic challenge in both the preoperative and intraoperative settings. Hematuria is frequently absent.

If a ureteral injury is initially missed and presents in a delayed fashion, urinary diversion with a nephrostomy tube and delayed repair at 3–6 months is a safe approach.

Bladder Injuries

Bladder wounds should be considered in patients with lower abdominal gunshot wounds, pelvic fractures with gross hematuria, or those patients unable to void following abdominal or pelvic trauma. Bladder disruptions can occur on the intraperitoneal or extraperitoneal surface of the bladder. The location may change the symptoms, complications, and management of this injury. In extraperitoneal injuries Bladder laceration is most often the result of laceration by bony fragments from a pelvic fracture.

After ensuring urethral integrity in appropriate cases (see Urethral Injuries, below), evaluation of the bladder is performed radiographically with a cystogram. Cystography reveals a dense, flame-like extravasation of contrast medium in the pelvis on the film.

The bladder usually heals with 10–14 days of Foley catheter drainage without the need for primary repair. If the urine is clear, catheter drainage alone is preferred for treatment of most extraperitoneal ruptures. In cases of abdominal exploration for other injuries, primary repair and drainage are necessary if the extra-peritoneal space is entered.

Urethral Injuries

A urethral injury should be suspected in patients with a scrotal hematoma, blood at the meatus, or a floating/high-riding prostate. The urethra is divided into anterior and posterior (prostatic) segments by the urogenital diaphragm:

- Anterior urethral injuries may result from blunt trauma, such as results from falls when astride an object (straddle) or from penetrating injuries.
- Posterior urethral disruption commonly occurs following pelvic fracture injuries.

Catheterization is contraindicated until urethral integrity is confirmed by retrograde urethrography. Retrograde urethrography is performed to evaluate the anatomy of the urethra.

If any difficulty in passing the catheter is encountered, the urethra should not be instrumented and a suprapubic tube cystostomy is performed.

External Genitalia Injuries

Injuries to the female genitalia usually result from direct trauma to the area. Injuries to the male genitalia are more common. These structures are highly vascular and have many nerve endings so injuries to this area are usually quite painful. Lacerations may result in severe bleeding and blunt trauma may result in a significant hematoma.

The management of wounds to the penis, scrotum, testes, or spermatic cord should be as conservative as possible and consists of hemorrhage control, debridement, and early repair to prevent deformity.

Injuries to the penis that disrupt Buck's fascia should be sutured to prevent further bleeding and avoid future penile curvature with erection. When extensive penile skin is lost, the penis may be placed in a scrotal tunnel until a plastic repair can be performed.

The scrotum is highly vascularized, and extensive debridement is usually not necessary for scrotal wounds. Most penetrating scrotal injuries should be explored to evaluate the testicle for injury and reduce the risk of hematoma formation.

The testicle is placed in the scrotum or in a protective pocket in the medial thigh. A testicle should never be resected unless it is hopelessly damaged and its blood supply destroyed.

Rectal Injuries

Rectal injuries can be difficult to diagnose unless very dramatic. Any question of an injury raised by proximity of another injury, rectal examination, or plain abdominal film radiography mandates proctoscopy. Gentle distal washout with dilute Betadine solution is usually required to be able to perform rigid proctoscopy. Findings can be dramatic disruptions of the rectal wall but more commonly are subtle punctuate hemorrhages of the mucosa. All abnormal findings should prompt corrective intervention.

Consider the traditional 4 «Ds» of rectal injury: Diversion, Debridement, Distal washout, and Drainage.

Of these, diversion is the most important. Transabdominal sigmoid colostomy is easiest. If the injury has not violated the peritoneum, exploration of the extraperitoneal rectum should NOT be done at laparotomy unless indicated for an associated nonbowel injury. This avoids contaminating the abdominal cavity with stool.

Debridement and closure of small- to medium-sized wounds is unnecessary in patients who have been diverted and drained. In any but the lowest of wounds, debridement and closure are difficult and troublesome. Distal washout is usually necessary to assess the injury. Fecal contamination of the perirectal space mandates presacral drainage.

5. Transporting immobilization in pelvis injures

Mechanical stabilization for hemorrhage control: tying a sheet or placing a binder around the pelvis at the level of the greater trochanters.

6. Treatment at medical care echelon

The management of ***pelvic injuries*** may include:

1. Resuscitation.
2. Transfusion alone (treat for shock).
3. Angiography (this has been found to be very successful in identifying and controlling pelvic arterial hemorrhage).
4. External fixation (opposition of the bony elements controls venous hemorrhage). Definitive internal pelvic stabilization (plates, screws, among others) is done outside of the combat zone.
5. External tamponade.

Penetrating Injuries

• Evaluation:

— Diagnosis of associated injuries may require exploratory laparotomy.

— Fractures should be assessed with radiographs and CT scans, when available, to *rule out extension into the hip and acetabulum*.

• Treatment:

— Immediate hemorrhage control by packing.

— Diverting colostomy in the presence of wounds at risk for fecal soilage.

— Debride wounds and fractures. Aggressive and thorough debridement. Pelvic stabilization.

Retroperitoneal packing has been successfully used with extreme hemorrhage. Venous bleeding can only be controlled by local packing and bleeding from large bore vessels can only be controlled surgically.

The emergency orthopedic procedure is the stabilization of pelvic injuries, fractures, and/or dislocations that are complicated by significant hemorrhage into the pelvic cavity. The placement of a pelvic external fixator to close the «open book»

pelvic fractures, to reduce the volume of the pelvic cavity, and to stabilize the pelvic ring can be life saving. In the patient with severe pelvic injury, the fixator should be placed prior to exploration of the abdominal cavity. Although preferably done in the operating room, the fixator can be placed in the emergency department.

Stable pelvic, acetabulum, and spine fractures are generally treated between 24 and 48 h after injuries. This interval allows time for specialized studies such as CT scans and for soft tissue injury and bleeding to stabilize prior to orthopedic surgery. The complexities of these bone injuries are usually best managed when the operating team is rested and hospital resources are most available.

XII. GUNSHOT WOUNDS AND CLOSED INJURIES OF LIMBS AND JOINTS

1. General characteristics and frequency of limb injuries

The extremities are the most common site to be injured by war weapons. The location of the injury also has prognostic role, the worst being for those injuries located below the knees. Weapons cause soft-tissue damage by several mechanisms: blast injuries produced by rockets, grenades, mortar and land mines, gunshot wounds and burns. The most common combination is blast with fragmentation injuries. Secondary lesions caused by mobilization of the fragmented bone or foreign bodies should also be considered during the patient's examination. It was reported that 70–80 % of combat injuries are caused by fragments from explosive munitions.

2. Limb injuries classification

3. Closed and open injuries of bones and blood vessels

Injuries to the extremities due to traumatic agent are mechanical or causing by gunshot.

In turn, gunshot injuries are divided: by type of wounding projectile — the bullet (high-speed, low speed), fragmentation — (big and small fragments), explosive (including mine blasting), other (shot, arrow-shaped elements, balls etc.).

By type of damaged tissue injuries of limbs are divided into two groups:

1) with only soft tissue damage;

2) damage to bones and joints (open and closed fractures).

Wounds of the extremities due to type of the wound cannel are divided into blind, cross-cutting and shearing.

Additional injury:

- crush injuries;
- vascular injuries.

According to multiplicity injury are divided to:

- Isolated injury.
- Multiple injuries.
- Concomitant injuries.
- Combined injury.

In case of ***isolated injury*** to the extremities there is only one injury. Simultaneous failure in one place soft tissue, bone, major blood vessels or nerve trunk in the military surgery is considered as one injury and, therefore, an isolated injury.

Multiple injuries are the injuries in which there are two or more injuries within the same anatomical region (the upper and lower extremities comprise one anatomical region — «limb»).

Concomitant injuries are the injuries in which there is damage to two or more anatomic regions. The last seven: head, neck, chest, abdomen, pelvis, spine and limbs. In the diagnosis of concomitant trauma damaged areas are listed in order to «from top to down».

Combined injuries occur when the body exposed to two or more damaging factors: mechanical, thermal, chemical or radiation.

Last three types of injuries are also known as «polytrauma» or «multi-trauma».

4. Frequency and characteristic of damaging of major vessels and limb nerves

The extremities remain the most common sites of wounding in conflict, are associated with a significant incidence of vascular trauma and peripheral nerve injury.

Peripheral nerve injuries represent 10 % of all injuries and 30 % of extremity injuries. However, since these nerve injuries will ultimately determine the functionality of the injured limb, it is of major importance to perform an even superficial initial neurological assessment and to act as soon as the victim has reached a medical facility.

Peripheral injuries account for 80 % of all cases of ***vascular trauma***. Penetrating trauma accounts for 70–90 % of vascular injuries. The lower extremities are involved in two thirds of all patients with vascular injuries. Major venous injuries accompany 13–51 % of significant arterial injuries.

5. Vascular limb injury. It`s signs and diagnostics

6. Symptoms of damaging of major vessels and limb nerves

Types of vascular (artery or vein) injury:

- Contusion.
- Puncture.
- Laceration.
- Transection.
- Crush injury.

Hard signs for ***vascular injuries*** are active arterial bleeding, expanding or pulsating hematoma, ischemia (pallor, pulselessness, paresis, or paralysis), and a thrill/bruit. Presence of a hard sign with a single entry and/or exit wound lead to surgery after the primary assessment in the emergency room. Soft signs which call upon further investigation of a possible vascular injury include a history of a moderate hemorrhage, hypovolemic shock, decreased but present peripheral pulses, peripheral neurologic deficit, or proximity to a named large artery. With multiple injuries or the presence of soft signs for a possible vascular injury, imaging is necessary for both the correct diagnosis and the choice of the preferred treatment.

Hard signs of arterial injury should lead to immediate surgical exploration, without further preoperative studies.

Soft signs of arterial injury that require additional diagnostic evaluation include proximity of wound to major vessels, history of hemorrhage/shock, non-expanding hematoma, diminished pulse, and anatomically related nerve injury.

Arterial injuries of the extremities are manifested by life-threatening hemorrhage or by ischemia resulting in limb loss. In sharp contrast, severe *venous trauma* is manifested by hemorrhage, not ischemia. Bleeding may be internal or external and rarely may lead to hypovolemic shock. Unlike arterial injury, repair of major veins and the current teaching is to avoid venous repair in an unstable or multi-trauma patient.

Critical tissue ischemic times into the table 5.

Table 5 — Critical tissue ischemic times

Tissue	Time
Muscle	4 hours
Nerve	8 hours
Fat	13 hours
Skin	24 hours
Bone	4 days

The **6 P**s of acute ischemia is: pain, pallor, pulselessness, poikilothermia, paresthesia, and paralysis.

Degree of injury and adequacy of collateral flow will determine the severity of distal ischemia.

The risk of reperfusion relates to the severity and duration of the ischemia. In this regard a simple classification of ***mild, moderate, and severe*** ischemia is of value in clinical assessment. ***Mild*** ischemia can be defined as a cool, pale limb in which there is no impairment of muscle function or loss of sensation. ***Moderate*** ischemia can be defined as a limb that is painful and cold; the muscle is still soft or slightly firm and any impairment of sensation is limited to the toes and distal foot. On dependency, the skin capillaries that fill will be pink. ***Severe*** ischemia is associated with loss of sensation, severe pain, and muscle paralysis; muscle rigidity and bluish mottled skin. Changes will develop 6–8 h following the onset of symptoms. These are manifestations of irreversibility. Severe ischemia will most commonly be

associated with embolic or traumatic occlusion; that is, sudden occlusion of a major artery in which there is no collateral developed circulation.

In the first instance, mild ischemia, all tissues of the limb are viable, the consequences of reestablishment of normal blood flow will be minimal, and therapeutic options are multiple.

In the second instance, moderate ischemia, extensive tissue necrosis is unlikely and the morbidity associated with reperfusion relates to the mass of ischemic muscle involved and the duration of the ischemia. Moderate ischemia is associated with the potential for viability (the options for treatment consist of anticoagulation and/or operative revascularization) and relates to the systemic risk. If muscle has not yet been devitalized, anticoagulation sufficient to stop clot propagation in the limb will be associated inevitably with improvement in viability. This is because collateral flow will improve due to the reversal of the vascular spasm caused by the inflammatory mediators released by the clotting process. Operative revascularization should be successful in so far as restoration of limb circulation is concerned, but will carry risk of systemic complications.

As regards severe ischemia, the risk of reperfusion is probably acceptable only if the limb can be revascularized within the critical 4–6 h prior to muscle death. Later than this, it is not only likely that surgery will be unsuccessful but that it will also be associated with high mortality. Amputation, should it be necessary, is best done after sufficient time has been allowed for maximal collateral development, as this will provide a much better chance for successful healing at whatever the level selected.

Remember: Warm ischemia of striated muscle for > 4–6 hours will likely lead to myonecrosis and major amputation.

A high degree of suspicion must be maintained for established or evolving limb-threatening situations: severe injuries to arteries or nerves are easy to miss in the acute situation and compartment syndrome can be difficult to diagnose.

7. Methods of final bleeding arrest

Management Aspects

Initial management.

Control external bleeding immediately! Blind or imprecise placement of vascular clamps in a bloody held is discouraged. Direct pressure to the bleeding wound is preferable; temporary tourniquet (BP cuff) placed proximal to the injury site and inflated above systolic blood pressure may be useful.

Indications for operation in a suspected vascular injury:

— Hard signs as discussed above.

— Soft signs confirmed by duplex Ultra Sound and/or angiography.

Prolonged shunting*:* If the above techniques are unsuccessful or precluded by patient physiology or the tactical environment, shunting with the following modifications can be used for up to 72 hours.

Preparation and draping of injured extremity as well as contralateral uninjured lower or upper extremity requires autogenous vein graft.

Ligation

Ligation of major veins is acceptable in life-threatening situations, although in a stable patient and time permitting, venous repair should be performed and may enhance arterial repair patency.

Ligation of artery: If the above options for repair are unsuccessful or unavailable, vessels can be ligated. ***Emphasis is Save Life over Limb.***

Type of repair will depend on the extent of injury.

- ***Lateral suture repair***: Required for minimal injuries that, when repaired, will not compromise the lumen > 25 %, result in a thrill, nor decrease pulse or Doppler signal.
- ***Patch angioplasty****:* Needed for larger, tangential wounds; to prevent stenosis.
- ***End-to-end anastomosis***: Excise extensively damaged segments and performs anastomosis if able to mobilize ends (generally < 2 cm gap) without tension. An oblique anastomosis is less likely to stenose.
- ***Interposition graft (Reversed venous bypasses)***: Required if the vessel cannot be primarily repaired without undue tension, usually if the gap is more than 3 cm long.
- ***Autogenous vein*** grafts preferred, usually the contralateral greater saphenous vein (GSV). Harvest vein from the contralateral limb, if possible. The reason for this is in the injured limb, superficial veins may be an important source of venous outflow if deep veins are injured. Order of preference vein harvest for arterial conduit is contralateral GSV, ipsilateral GSV (if no concomitant deep venous injury), contralateral lesser saphenous vein (LSV), ipsilateral LSV (if no deep venous injury)/ cephalic vein, and basilic vein.
- ***Prosthetic*** grafts may be required when autogenous vein is inadequate or unavailable, expeditious repair is indicated, or for large vessels (aortoiliac system) for which there is a large size discrepancy. Polytetrafluoroethylene (PTFE) grafts are more resistant to infection than Dacron and have acceptable patency rates when used in the above-knee position. Prosthetics can also be used in areas of extensive soft-tissue debridement as a «prolonged shunt» where planned revision days to weeks later, out of theatre, will be expected.
- ***Venous repair*****:** Repair of a vein is of special importance when this vein is the only venous drainage route, as in the popliteal vein. The experience in the 2006 Lebanon War leads to preference of venous repair over ligation in such situation, even when an interposition graft, rather than simple repair is needed. Options are similar to arterial repairs outlined above.

Nerve Disruption Management

When it possible, an end-to-end suture of the nerve performed during the initial emergency procedure gives the repair the highest chance of success because of the perfect adaptation it allows.

If an end-to-end suture of the nerve cannot be performed as is often the case in high-velocity missiles, a tagging of the slumps and even a few loose stitches to bring them closer are recommended. As soon as local conditions allow, a delayed graft will be performed.

This nerve repair may be done in conjunction with the replacement of the external fixation for an internal device.

The risk of leaving a nerve lesion untreated is in our opinion unacceptable and justifies a more aggressive early approach.

If the nerve is found in continuity, the treatment will consist of a simple follow-up. A further neurolysis will be indicated if clinical or EMG improvement does not occur within 6 months or if the recovery is still insufficient after 9 months. At this point, the surgical procedure consists of releasing the injured nerve from the adjacent scar tissue and eventually from its anatomical tunnels.

8. Gunshot fractures of the extremities

9. Signs of bone fractures

Damage to bone may result from the bullet striking the bone directly. The effects of such contact depend on the type of bone structure that is struck, the surrounding and supporting structures, and the physical characteristics of the missile.

Another and less severe kind of fracture occurs when a high-velocity bullet passes near the bone but does not contact it directly. This has been called an «indirect fracture». Such injuries are caused by the high pressures suddenly launched against the bone by the leading particle edge of the rapidly expanding temporary cavity.

An ***open fracture*** can be defined as a broken bone that is in communication through the skin with the environment. The amount of communication can vary from a small puncture wound in the skin to a large avulsion of soft tissue that leaves bone exposed. In contrast, a closed fracture is one that is contained within the extremity without damaging the skin.

A closed fracture can usually be considered clean if the skin is not devitalized. All open fractures, on the contrary, are considered contaminated.

Open fractures are classified according to their severity. In a first degree open fracture, the bone fractures and penetrates the skin. First degree open fractures have very small wounds and following surgical debridement can be treated in the same manner as a closed fracture.

Second-degree open fractures are more extensive than first degree open fractures, and there is usually a larger wound that communicates with the fracture. Soft tissue may be injured to greater extent, and these fractures represent a middle ground between first degree and third degree fractures.

Third-degree open fractures represent the most severe form of open fracture with combination of the fracture and massive soft tissue damage. Soft tissue and

bony avulsion may be part of the fracture, and often these injuries are caused by outer forces. Most commonly third degree open fractures in the combat are the result of high-velocity bullet wounds.

Classification of open fracture by Gustilo-Anderson into the table 6.

Table 6 — Classification of open fracture by Gustilo-Anderson

Gustilo type	Definition
I	Open fracture, clean wound, wound <1 cm in length
II	Open fracture, wound > 1 cm in length without extensive soft-tissue damage, flaps, avulsions
III	Open fracture with extensive soft-tissue laceration, damage, or loss or an open segmental fracture. This type also includes open fractures caused by farm injuries, fractures requiring vascular repair, or fractures that have been open for 8 h prior to treatment
IIIA	Type III fracture with adequate periosteal coverage of the fracture bone despite the extensive soft-tissue laceration or damage
IIIB	Type III fracture with extensive soft-tissue loss and periosteal stripping and bone damage. Usually associated with massive contamination. Will often need further soft-tissue coverage procedure (i.e. free or ratational flap)
IIIC	Type III fracture associated with an arterial injury requiring repair, irrespective of degree of soft-tissue injury.

General signs and symptoms of fractures:

1. Pain at or near the location of fracture.
2. Tenderness of discomfort on gentle pressure over the affected area.
3. Swelling about the fracture area. Swelling frequently render it difficult to perceive other signs of fracture and care must be taken therefore not to treat the condition as a less serious injury.
4. Loss of power: the injured part cannot be moved normally
5. Deformity of the limb the limb may assume an unnatural position and be misshapen.
6. Irregularity of the bone. If the fracture is near the skin the irregularity of the bone may be felt.

Hard signs of fractures:

7. Crepitus (bony grating) may be heard or felt.
8. Unnatural movement at the seat of the fracture.
9. Bone`s fragment in the wound.
10. Fat drops in the joint punctate.
11. Loss of sound conductivity.
12. Pain under the pressure at long axis of bone.

Compartment Syndrome

Compartment syndrome may occur with an injury to any fascial compartment. The fascial defect caused by the injury may not be adequate to fully decompress the compartment, and compartment syndrome may still occur.

Early clinical diagnosis of compartment syndrome: pain out of proportion, pain with passive stretch, tense, swollen compartment.

Late clinical diagnosis: **4 P (**Paresthesia; Pulselessness; Pallor; Paralysis).

The diagnosis of a compartment syndrome is made on clinical grounds.

Measurement of compartment pressures is not recommended in the combat zone. Consider prophylactic fasciotomy.

Mechanisms of injuries associated with compartment syndrome:

- High-energy wounds.
- Open fractures.
- Closed fractures.
- Penetrating wounds.
- Crush injuries.
- Circumferential dressings or casts.
- Vascular injuries.
- Reperfusion following vascular repairs.

Fat Embolism (FE) and Fat Embolism Syndrome (FES)

Definition: The embolization of fat globules into the systemic circulation occurs in nearly all patients with long tubular bone and pelvic fractures and significant orthopedic procedures such as hip and knee joint replacement. Fat globules enter via venous sinuses in the bones and travel to the lungs and into the systemic circulation via pulmonary capillaries and through anatomical or functionally patent atrial foramen ovale.

While usually asymptomatic, fat embolism (FE) may give rise to the fat embolism syndrome (FES), a critical and often fatal disease.

Two main mechanisms are considered in the pathogenesis of FES, the first being the ***«mechanical»*** and the second the **«*biochemical*»**. In the mechanical pathway, fat globules cause mechanical occlusion of pulmonary and systemic capillaries, thus causing local ischemia. The biochemical pathway is caused by serum lipases that release toxic free fatly acids from the small fat droplets that have penetrated the circulation and reached the lungs, brain, and other organs. Capillary endothelial cells are injured, followed by edema, and hemorrhage in affected organs.

Signs and symptoms: The clinical signs typically appear within 24–72 h of injury and their severity ranges from none or very mild to full blown life-threatening FES. The speed of development and severity of symptoms is relative to the load of embolized fat.

The pathognomonic triad of symptoms includes (% incidence):

1. Petechial rash of conjunctival runk and axillae — 40 %.
2. Progressive hypoxemic respiratory failure — 50 %.
3. Altered level of consciousness, seizures, focal deficits — 70 %.

Up to 50% of patients with FES will develop respiratory failure severe enough to require mechanical ventilation. Corticosteroids decrease the risk of

developing FES and hypoxemia after long bone fracture of the lower limbs. The effect of corticosteroids on FES may be related to stabilization of the alveolar-capillary membrane/complement system, reduction of interstitial edema, and inhibition of the inflammatory response, as well as to delayed platelet aggregation.

10. Medical care organization of injured limbs at medical care echelon

11. First aid

12. Preventive measures of the shock, bleeding and infection

13. Surgical management of injured limbs

14. Principles of specific medical care

General Considerations of Wound Management

Initial management

Administer antibiotics, tetanus toxoid, and analgesia.

Begin IV antibiotics as soon as possible and maintain throughout the evacuation chain. Use a broad spectrum cephalosporin (cefazolin 1g q 8h). An aminoglycoside may be harmful for someone in shock or dehydrated. The two most harmful bacteria — Clostridia and streptococci — are covered by a 1st generation cephalosporin.

Treat by irrigation and debridement as soon as feasible to prevent infection.

Neurovascular status of the extremity should be documented and checked repeatedly.

Wound incision/excision (debridement)

Longitudinal incisions to obtain exposure: fascia incised longitudinally to expose underlying structures and **compartment release**.

Irrigation is essential.

All foreign material in the operative field must be removed. Bone fragments should be retained if they have a soft tissue attachment. Detached bone fragments smaller than a thumbnail is discarded. Larger fragments that contribute to the structural integrity of the long bone should be retained.

Closure of wounds

Primary closure is never indicated (except on palm). Loose approximation of tissues with one or two retention sutures is appropriate to cover nerves, vessels, and tendons, but there must be a provision for substantial free drainage.

Skin grafts, local flaps, and relaxing incisions are contraindicated in the initial management.

Delayed primary closure may be attempted as described in the section on soft tissue wounds. This should be accomplished in a stable environment.

Compartment Syndrome *considers prophylactic fasciotomy. Indications for fasciotomy* in the combat setting:

- 4–6 hour delay after vessel injury.
- Combined vein and artery injury.
- Arterial or main venous ligation.
- Concomitant fracture/crush, severe soft-tissue injury, muscle edema or patchy necrosis.
- Crush injuries.
- Vascular repair.
- Tense compartments

All ***open-joint injuries*** must be explored and treated within 6 hours to prevent infection and joint destruction. The key to treating open-joint injures is recognition. Once identified, goals are prevention of infection and preservation/restoration of normal joint function. Open-joint injuries always require surgery. Joint aspiration/injection may be performed to confirm a suspected open joint. If in doubt, treat as an open-join injury to prevent missed injury sequelae. Any time joint infection is suspected, the joint should be *immediately* explored/re-explored.

Close synovium if possible without tension and without surgical tissue advancement. Delayed primary closure (DPC) can be undertaken in 4–7 days if there are no signs of infection.

Internal fixation is ***contraindicated*** with the ***exception of large articular fragments*** that may be stabilized with Kirschner wire (K-wire) or Steinmann pins.

Management of bone fracture

Transportation casts

The goal of transportation casts is to immobilize a fracture along the evacuation chain. The cast must meet the dimensions of the standard litter.

Biplanar radiographs should be obtained.

Advantages of transportation casts are that they preserve the maximum number of options for the receiving surgeon; the soft tissues are well supported, and the casts are relatively low tech. ***Disadvantages*** are that casts cover soft tissues, may not be suitable for polytrauma patients, and are more labor-intensive than external fixators.

Both transportation casts and external fixators are equally acceptable methods for the initial management of long bone fractures. In the end, the choice of initial fracture stabilization must be made individually by the treating surgeon. That decision should be based on the surgeon's experience, his assessment of the evacuation process, the available materials.

Internal fixation is contraindicated as initial management of bone fractures.

Skeletal traction

Skeletal traction provides a quick means to immobilize a large number of fracture cases with a minimum of technical support.

Indications: patients who are expected to have more than one procedure in the same forward hospital prior to evacuation; load large casualty.

Operative management

Open reduction and internal fixation. Therefore, one of the principal components of this evolution has been the introduction of staged protocols, including provisional external fixation followed by a delay of days to weeks before definitive internal fixation is performed. This form of treatment is generally utilized in one of three scenarios: (1) A critically injured patient, (2) the presence of a wound with a significantly damaged and tenuous soft-tissue envelope, or (3) a severely contaminated open fracture. In each of these cases, the use of temporary external fixation in order to bridge the treatment of the patient from the period of the acute injury to the time of definitive internal fixation.

In general, good indications for external fixator use include when the soft tissues need to be evaluated while en route, such as with a vascular injury; when other injuries make use of casting impractical, such as with a femur fracture and abdominal injury; or when the patients have extensive burns. ***Advantages of external fixation*** are that it allows for soft tissue access, can be used for polytrauma patients, and has a minimal physiologic impact on the patient. ***Disadvantages*** are the potential for pin site sepsis or colonization and less soft tissue support than casts.

In the management of diaphyseal fractures in the trauma patient, secondary ***intramedullary nailing*** after external fixation can be a safe treatment with high union rates (98 % in femur, 90 % in tibia) and with a low incidence of malunion and an acceptable risk of deep infection (3.6 % in femur, 9 % in tibia). For periarticular fractures treated in this way, various authors have demonstrated improved outcomes — particularly lower rates of wound complications, without increased infection rates related to the external fixator.

The extraordinary potential of the ***Ilizarov method*** for tissue neogenesis is a solution for almost any of bone damage, including massive bone loss. This method allows the performance of radical bone end debridement in the knowledge that future bone length restoration will be possible. Taking into consideration a staged protocol of external fixation, including primary temporary unilateral tubular stabilization followed by definitive circular Ilizarov/Taylor/hybrid fixation and reconstruction by the use of customized frames, is an effective method for the treatment of patients with severe damage to the limbs caused by high-energy war injuries.

XIII. BLAST TRAUMA

1. General characteristics and frequency of blast injuries

A ***blast injury*** is a complex type of physical trauma resulting from direct or indirect exposure to an explosion. Blast injuries occur with the detonation of explosives. These injuries are compounded when the explosion occurs in a con-

fined space. Blast injuries are also energy related and mainly dependent upon the distance from the blast, the energy released from the bombing device, the media (air or water) and the environment in which the blast takes place (close or open). Although the injury may look superficial, it might be much worse and the external wound is sometimes only the top of the iceberg.

2. Classification of blast trauma

The explosive device can be identified by the mechanism and intensity. Conventional weapons, for example, grenades, aerial bombs, and mortar bombs are characterized by predominance of penetrating injuries from multiple fragments. Terrorist devices vary by the explosive amount. Car bombs which contain typically 1–3 kg of commercial explosive positioned under the floor of directly beneath the driver's seat might cause bare charges, for example. Booby traps are small improvised devices (< 10 kg) detonated remotely by wire or radio signal are characterized by primary blast injuries and ballistic injuries due to secondary fragments. Lorry or van bombs are large devices (> 40 kg) in which the detonation results in the formation of large secondary projectiles (from the body of the vehicle) and crush injuries from secondary damage to buildings; Culvert bombs are very large devices designed to disrupt passing vehicles and the vehicle may be displaced and victims ejected and they may cause gross disruption and disintegration of the body Antipersonnel mines are characterized by a predominance of traumatic amputation of foot or leg due to standing on a buried «point detonating» mine and the damage might be increased due to shrapnel.

3. Pathogenesis and pathomorphology of damage in blast injury

As a bomb detonates, a chemical reaction converts the solid or fluid explosive into gas, which creates high-temperature thermal reaction accompanied with a blast wave at an initial speed of approximately 6,000–7,000 km/s. The difference between the blast «overpressure» and the surround's pressure determines the blast strength and its potential to produce primary blast injuries.

Blast waves of conventional explosives are pressure pulses, a few millimeters thick, which travel at supersonic speed outward from the point of the explosion. These waves are characteristically of short duration, a positive wave with a rapid upslope that is followed by a longer smaller negative wave. The pressure then drops below ambient air pressure, and the resultant vacuum effect can suck debris into previously unaffected areas. However, blast effects are magnified if the explosion is in an enclosed space that contains the blast and causes amplification of the wave as its reflected off the enclosure surfaces.

Blast injuries have been generally categorized as ***primary, secondary, tertiary, or miscellaneous***.

In ***primary*** mechanism, the blast wave causes the direct injury, typically to air-containing organs: the lungs, small and (particularly) large bowels, and the auditory system. The suggested mechanisms for primary blast effects include:

— Direct compressive effects of the blast wave: spilling, in which denser tissue fragments into less dense tissue as the blast wave advances through the tissues.

— Implosion, in which gas pockets momentarily contract and re-expand rapidly, injuring tissues.

— And stress and shear forces, which cause injury because of the differential response of tissues to these forces.

In s***econdary*** mechanism, the fragments from the bomb or the surrounding environment impact the subject causing blunt and penetrating injury. This may cause mild or severe injuries to many of the survivors from the primary blast effect and it can cause damage to up to two thirds of the survivors.

In ***tertiary*** mechanism, the blast wave propels the subject into another causing injury. Many injuries such as soft crash or fractures are caused by tertiary mechanism when the victims are thrown to the ground or other objects.

Miscellaneous orthopedic blast injuries are much less common than secondary blast injuries and may include burns from the thermal effects of explosions or from secondary fires. These bomb fragments are preplanned to aggravate the damage and they can be made from metal objects such as screws, pins, balls, or from other materials such as rubber, wood, plastics, and ceramics. Furthermore, these fragments may contain biologic, chemical, or even radioactive elements that are dispersed by the bomb blast causing other forms of injury.

4. Clinical features of blast injury

Common explosive blast injuries include pulmonary barotrauma, brain injury, abdominal hemorrhages, ocular injury, tympanic membrane rupture and middle ear damage, crush injuries, traumatic amputations, and burns. Blast injuries are the result of any of four basic mechanisms termed as primary, secondary, tertiary, and miscellaneous. Victims may have complex injury patterns involving multiple organ systems as a result of a combination of some or all of these blast injury mechanisms.

Overview of Explosive-related Injuries into the table 7.

Table 7 — Overview of Explosive-related Injuries

Auditory	TM rupture, ossicular disruption, cochlear damage, foreign body
Eye, Orbit, Face	Perforated globe, foreign body, air embolism, fractures
Respiratory	Blast lung, hemothorax, pneumothorax, pulmonary contusion and hemorrhage, A-V fistulas (source of air embolism), airway epithelial damage, a spiration pneumonitis, sepsis
Digestive	Bowel perforation, hemorrhage, ruptured liver or spleen, sepsis, mesenteric ischemia from air embolism
Circulatory	Cardiac contusion, myocardial infarction from air embolism, shock, vasovagal hypotension, peripheral vascular injury, air embolism-induced injury
CNS injury	Concussion, closed and open brain injury, stroke, spinal cord injury, air embolism-induced injury

Renal Injury	Renal contusion, laceration, acute renal failure due to rhabdomyolysis, hypotension, and hypovolemia
Extremity injury	Traumatic amputation, fractures, crush injuries, compartment syndrome, burns, cuts, lacerations, acute arterial occlusion, air embolism-induced injury

Selected Blast Injuries

Lung Injury

«Blast lung» is a direct consequence of the overpressure wave. It is the most common fatal primary blast injury among initial survivors. Signs of blast lung arc usually present at the time of initial evaluation, but they have been reported as late as 48 hours after the explosion. Blast lung is characterized by the clinical triad of apnea, bradycardia, and hypotension. Pulmonary injuries vary from scattered petechae to confluent hemorrhages. Blast lung should be suspected for anyone with dyspnea, cough, hemoptysis, or chest pain following blast exposure. Blast lung produces a characteristic «butterfly» pattern on chest X-ray. A chest X-ray is recommended for all exposed persons and a prophylactic chest tube (thoracostomy) is recommended before general anesthesia or air transport is indicated if blast lung is suspected.

Abdominal Injury

Gas-containing sections of the GI tract arc most vulnerable to primary blast effect. This can cause immediate bowel perforation, hemorrhage (ranging from small petechiae to large hematomas), mesenteric shear injuries, solid organ lacerations, and testicular rupture. Blast abdominal injury should be suspected in anyone exposed to an explosion with abdominal pain, nausea, vomiting, hematcmcsis, rectal pain, tenesmus, testicular pain, unexplained hypovolemia, or any findings suggestive of an acute abdomen. Clinical findings may be absent until the onset of complications.

Brain Injury

Primary blast waves can cause concussions or mild traumatic brain injury (MTBI) without a direct blow to the head. Consider the proximity of the victim to the blast especially when given complaints of headache, fatigue, poor concentration, lethargy, depression, anxiety, insomnia, or other constitutional symptoms are presented. The symptoms of concussion and post traumatic stress disorder can be similar.

5. Blast injury diagnostics. Stages of examination

If circumstances permit, medical personnel should carefully examine the casualty for signs of contusion or penetrating wounds. Sometimes, a sentinel (or associated) injury, which may be as dramatic as a traumatic amputation or as relatively minor as a temporary hearing loss, will indicate that the casualty may also have significant blast injury.

Medical personnel must ensure that the casualty is hemodynamically stable and that the airway is free. In the PBI casualty, the life-threatening injuries that

require immediate stabilization arc usually caused by respiratory damage or by blood loss from gastrointestinal hemorrhage.

Diagnostic Screening. As soon as the blast casualty is hemodynamically stable, medical personnel should take a chest X-ray examination, regardless of the casualty's symptoms. However, serial monitoring of hematological and biochemical parameters may be useful in following the complicated medical course of any seriously injured patient.

6. Traumatic disease in blast injuries. Its conception, peculiarities, stages, principles of medical care

Compartment syndrome, rhabdomyolysis, and acute renal failure are associated with structural collapse, prolonged extrication, severe burns, and some poisonings.

Stages of traumatic disease development

1. Stage I — Initial (early) stage (shock, I Phase of the disseminated intravascular coagulation (DIC), I phase of acute renal failure (ARF)).
2. Stage II — Intermediate (rhabdomyolysis, II phase of ARF and DIC).
3. Stage III — Late (advanced or resulting) stage of crush syndrome (secondary immunodeficiency, septic disorder, III phase of ARF and DIC).
4. Stage IV — Recovery.

Systemic therapy of traumatic disease

1. Correction of hypovolemia/dehydration — fast and aggressive fluid resuscitation to recover normovolemia
2. Forced diuresis — maintenance of brisk urine flow of 1–2 ml kg·1h·L may reduce cast formation and tubular obstruction.
3. Alkalinization of urine: acidic urine promotes myoglobin cast formation.
4. Correction of electrolyte abnormalities: hyperkalemia, hyperphosphatemia, hypocalcemia, hyperuricemia.
5. Renal replacement therapies: hemodialysis or hemofiltration are necessary to maintain balance in anuric patients.

Wounds can be grossly contaminated. Consider delayed primary closure and assess tetanus status.

In summary, patients injured in explosions may have both readily apparent and hidden wounds that are equally lethal if left untreated. Physicians should have a high clinical suspicion for occult hemorrhage or closed head injury. After control of the immediately life-threatening injuries in the operating room, it may be advisable to perform rapid diagnostic imaging to evaluate for occult hemorrhage before committing to a definitive operation. By the time a patient is determined to be unresponsive to resuscitation, the window of opportunity for life-saving intervention may have passed.

7. Management in blast injuries at medical care echelon

Some manifestations of blast injury will resolve on their own, or will require only a continuation of stabilization measures until the casualty is out of danger. Other manifestations will require immediate surgical intervention, or may call for sophisticated equipment that would not be available at the lower echelons of care. In addition, some manifestations of blast injury may have long-term sequelae.

Casualties who have extreme respiratory embarrassment should be intubated endotracheally to handle massive hemoptysis and in anticipation of mechanical ventilatory support.

For either a tension pneumothorax or a simple pneumothorax that has no accompanying contusion or evidence of cardiovascular insufficient, an immediate tube thoracostomy is the definitive treatment.

A hypotensive casualty must quickly receive sufficient volume replacement to bring the pulse and blood pressure back within normal limits. In casualties with combined burn and relatively mild pulmonary blast injuries, fluids can be replenished according to standard infusion formulae for burn casualties.

All medical personnel should be aware that excessive volume resuscitation may worsen the casualty's gas exchange, mechanical ventilation support may exacerbate both pulmonary barotrauma and air embolism, and general anesthesia for the blast casualty will be poorly tolerated.

Local care

The lessons of adequate wound debridement and secondary wound closure have to be relearned by new generations of military surgeons at the beginning of every war. They are the key components in managing high energy injuries and associating with them open fractures. The use of external fixators in military practice for fracture stabilisation has not so successful as hoped. This has resulted in the use of plaster combined with other methods, even including the 'old fashioned' Thomas splint.

Battle casualties who sustain amputations have the most severe extremity injuries. Historically, one in three patients with a major amputation (proximal to the wrist or ankle) will die, usually of exsanguinations.

The following indications for amputation:

- Partial or complete traumatic amputation.
- Irreparable vascular injury or failed vascular repair with an ischemic limb.
- Life-threatening sepsis due to severe local infection, including clostridial myonecrosis.
- Severe soft-tissue and bony injury to the extremity precluding functional recovery.

The surgeon must balance between ultimate reconstruction of a functional extremity and the risk of death associated with attempts to preserve a limb.

Types of amputation

1. Guillotine amputations (one level amputation)

2. Flaps amputations (multi-layer amputation)

2.1. Myoplastic technique (sofl-tissiue flaps).

2.2. Osteomyoplastic technique (the flexible bone graft).

The Open Length Preserving Amputation (formerly Open Circular Amputation, or Guillotine amputations) procedure has two stages: initial and reconstructive.

Initial. Complete the amputation at lowest possible level of bone and prepare the patient for evacuation to the next level of care. Guillotine amputations in a war setting should be performed as distally as possible, and revised later under calmer conditions. This procedure was called a «Guillotine» amputation, since the muscle and bone were cut at the same level.

It was subsequently replaced with the construction of the flap which, after being left for some time, facilitated closure of the wound.

Reconstructive. It involves final healing of the limb to obtain the optimal prosthetic stump. Bone cutting is carried out in consideration of the soft-tissue coverage, so that when the closure of the wound is performed, the skin is not under tension.

Final level of amputation and definitive treatment of the residual limb should occur in the stable environment of a base hospital*, not in the combat zone hospital.*

The *«Pirogoff amputation»* (kind of osteomyoplastic amputation) is a surgical salvage procedure for the complex injuries of the forefoot, where there is considerable loss of the osseous and soft tissues. Part of calcaneus together with the fat pad is rotated and fused to the tibial plafond, which allowed for a longer stump, eliminated the need for below-knee amputations, and allowed for weight bearing.

General Principles of Amputation Technique: No matter what type of amputation is performed, the skin flaps must be created with enough length to avoid closure under the tension. The skin should not adhere to the underlying bone, and there will preferably be no scar formation in the areas of the prosthesis contact.

XIV. BURNS

Burns constitute between 5 % and 20 % of combat casualties during conventional warfare. Even relatively small burns can be incapacitating, and can strain the logistical and manpower resources of military medical units.

Four types of energy cause burn wounds: thermal, electrical, chemical, and radiation. Thermal injury accounts for 85–90 % of burns. Chemical and electrical injuries comprise the vast majority of the remainder.

1. Classification of burns according to severity

2. Degree of burns

The severity of any burn is determined by the age of the patient, the depth of the burn, and the size of the wound measured as a percentage of total body

surface area (TBSA). Burn wounds are classified according to depth. Burns are described as first-, second-, third- or fourth-degree.

Understanding of skin biology and anatomy is important. Outer layer is called the epidermis. Deep layer is called the dermis, which has a superficial (papillary) layer and a deep (reticular) layer.

Classification of Burn Injuries

For the definition of burns a rough classification of burn degree is used, as follows:

1. First degree: injures to the epidermis — reddening, swelling due to reactive edema.

2. Second degree: epidermal detachment of the cutis — development of blisters.

3. Third degree:

3A — partial destruction of the epithelium — remaining small pieces of epithelium, possibly capable of regeneration;

3B — total destruction of epithelium — no regeneration because of absence of small pieces of epithelium

4. Fourth degree: damage to far-reaching tissue formations, partial charring — muscles, and tendons affected.

Superficial Burns

A «first-degree»

Epidermal burns — involve only the epidermis (such as Sunburn), cell damage without cell death.

Treatment is symptomatic (soothing lotion). Usually heals spontaneously in 2–3 days, without scarring.

A «second degree» burn involves whole the epidermis. This type of burn is erythematous with blister, and it heals in 7–10 days without sequelae. In children under10 years, a superficial second-degree burn will heal in — 2 weeks; in adults, healing takes < 3 weeks.

Patients with superficial first-degree and second-degree burns < 10 % TBSA are usually treated as outpatients. Both are very painful.

Partial-Thickness Burns

«3-A degree» burns

A superficial 3d-A degree burn involves the entire epidermis and superficial portions of the dermis. The skin is blistered with a moist and weeping base. Superficial 3d-A degree burns produce minor color changes but not hypertrophic scarring.

Deep partial thickness

A deep «3-A degree» burn extends through the epidermis and into deeper dermis. After blister removal, it is usually dry and demonstrates an ivory or mottled red base. This type of burn requires > 3 weeks to heal spontaneously and results in very significant scar formation.

Full-thickness burns

«3-B degree» burns

Burn goes through all layers of the dermis, death of all germinal epidermal elements. Appear like leather, totally anesthetic, cannot heal spontaneously, as all layers of the dermis are gone. A third-degree burn destroys the entire thickness of the epidermis and dermis.

«4-degree» Burn

Involves deeper structures (tendon, muscle, bone etc), usually due to severe electrical injury.

3. Determining the burn size

The initial description of the burn includes an estimation of the percent of the total body surface area (TBSA). Only 3B and 4 degree burns are included in deep burn size calculations.

Three methods can be used to calculate burn size:

- The «rule of palm». For evaluation of small or scattered burns, the rule of the palm states that the patient's palm, including the fingers and the thumb, equals ~1 % of the patient's TBSA.
- The «rule of nines». According to the rule of nines, the major body surfaces can be expressed as multiples of 9 % of TBSA in adults. The head and the neck comprise ~9 %, each arm and hand together represent 9 %, the anterior and the posterior trunk are 18 % each (two nines), each leg is 18 % (two nines), and the perineum comprises 1 %.
- and the Lund and Browder chart.

4. Local and systemic manifestations of thermal burns

Minor burns are no problem for either the physician or the patient, whereas a burned body surface area (BSA) of only 10 % in children and 20 % in adults presents a drastic and often extremely crucial clinical picture, as there are dramatic links between local skin injuries and the overall damage caused by the external heat effect in the organism.

Circumferential burns of the chest may prevent effective chest motion. If this occurs, perform immediate thoracic escharotomy as a life-saving procedure to permit adequate chest excursion.

Inhalation injury is more common in patients with extensive cutaneous burns, a history of injury in a closed space (e.g., building or vehicle) and facial burns. Carbon monoxide (CO) poisoning causes cardiac and neurologic symptoms. Patients with CO poisoning require 100 % oxygen for at least 3 hours or until symptoms resolve.

Inhalation injury may be manifested by stridor, hoarseness, cough, carbonaceous sputum, dyspnea, and so forth. It may cause airway obstruction at any time during the first 2 days postburn.

Patients who may have sustained inhalation injury should be closely observed in an intensive care unit, and may be monitored without intubation if minimally symptomatic. Prior to transport, prophylactically intubate patients who have symptomatic inhalation injury.

The general changes in the organism resulting from burns depend on the degree of the burn and the size of the burned area. The greater the burned surface of the body and the longer the action of the thermal stimulus, the more serious the consequences. The functional state of the organism is also very important.

General phenomena occurring in burns are due to accumulation in the organism of toxic products of tissue decomposition, which cause intoxication of the organism (so called thermal rabdomyolisis or ***burn disease***). However, the mechanism of development of general phenomena in burns must be ascribed primarily to reflex influences from the burned parts of the body. The reflex influences are followed by absorption of the products of tissue decomposition, especially when the burned tissues become infected. Prognosis in burn diseases depends on the extent and depth of the burned BSA.

Observations and experiments have shown that the organism perishes if one-third of the body surface is damaged (in second degree burns) and even less (in third and fourth degree burns). In cases of vast and severe burns death occurs instantaneously or within 2–3 days. Early death is due to the bum shock, i.e. sharp reflex depression and subsequent paralysis of the circulatory and respiratory centers. The general picture of a burn shows, in addition to disturbances in nervous activity, first a rise and then a drop in blood pressure, respiratory disorders, hemoconcentration due to the passage of plasma through the capillaries into the injured tissue, a relative increase in the erythrocyte count (sometimes 30–40 per cent), phenomena of hemolysis, accumulation of toxic products of tissue decomposition, a rise in body temperature, and development of infection which has gained- entrance into the wound. In protracted cases the kidneys are affected, urination is disturbed and anuria sometimes develops.

5. Stages of burn disease (burn shock, acute burn toxemia, septicemia, recovery)

Burn disease

Burn disease is a constellation of clinical signs that result from superficial burns (degrees 2–3a) with a burn area of above 15 % body surface and in deep burns of more than 10 % body surface. A serious burn progresses through successive stages: first of shock, second of poisoning; third of infection; and forth of healing.

The four periods of the disease are identified.

1. Burn shock.

2. Acute burn toxemia.
3. Septicemia.
4. Recovery.

Burn (hypovolemic) shock

Loss of blood plasma being the major pathogenic mechanism of burn shock, results in local accumulation of vasoactive substances (e.g. histamine, serotonin) and an increase in blood viscosity, which consequently impairs microcirculation.

Extensive burns cause disorders in general functions, which after some hours always lead to irreversible collapse of all vital functions. First of all hypovolemia (low blood volume) occurs, leading to a loss of circulating blood of up to 15–30 % after only 1–2 h. There is also an uncontrollable loss of liquids, lasting up to four days.

Hypovolemia leads to an increase of liquid accumulation in the burned tissue and, as a result — sequestration. The blood flowing back from this area is hypotonic to the entire organism, also making possible an increase in liquid receptivity. There is a possibility of edema in the liver, kidney, and brain, with the danger of cerebral edema. This causes conditions of confusion and restlessness.

There is also an increased loss of liquid through injured capillaries and cell membranes that continues throughout the healing process. Another loss of liquid develops owing to evaporation from extensive wound areas — this evaporation amounts to 1200 ml per day with a burned BSA of only 10 %. There is also an accumulation of sodium in the burn areas. The change in the liquid and electrolyte balance — intracellular calcium is exchanged for extracellular sodium — also changes the total capacity of extracellular liquids, and the sodium level in the serum is reduced. With reduced renal function there is an increase in potassium against the sodium losses by released calcium, flowing directly from the cells via cell substructures. If fluid intake is too low, renal functions will be damaged, as will eventually become manifest.

Tissue hypoxia causes an increase in lactates, leading to intracellular acidosis, the extent of which cannot be measured because of imbalances in the microcirculation. Burn patients always present an excessive catabolism. The high loss of secretions and the simultaneous onset of infection cause a negative protein balance, as there is an extremely high consumption of additional calories due to evaporation. For this reason severe infection may bring high fever. The regeneration process also necessitates additional energy demands. The burn areas must therefore be covered as soon as possible. Only then it will be possible to reduce the almost impossible supply of calories.

Renal functions are reduced by shock, and filtration pressure is limited in the vas afferens owing to shock. There is a parallel contraction of the vas afferens that makes the oxygen uptake insufficient, especially in the marking zone. This leads to permanent tubulonecrosis.

After a burn, blood circulation in the skin, intestine, and muscular system is reduced by vasoconstriction for the maintenance of blood circulation in all essential

organs (a.k.a. centralization). This causes an increase in the flow resistance of blood. The heart has to pump against increased resistance and may become insufficient because of the possibility of edema. An additional threat is the tendency for an aggregation of thrombocytes and erythrocytes. The additional hypoxia leads to the occurrence of permanent myocardial necroses, even in younger patients.

Disturbances in blood clotting have already been mentioned — they can promote the formation of microclots that can cause consumption coagulopathy and pulmonary embolisms.

The formation of «shock lung» poses a much more difficult problem. During the phase of reabsorbtion of sequestered liquids, the excessive parenteral amount of liquid inundates the alveolar wall in edematous manner, and oxygen restoration to the lung becomes more difficult or does not occur at all. Additionally, secretions will press into the alveoli, causing lung edema and suffocating the patient.

The manifestations of burn shock, which may last 2–72 hours, depend on its duration and severity of circulatory defects. Stabilisation of the latter may imply evolution to the further period of burn shock.

Acute burn toxemia

Once toxic products enter the blood stream, circulating blood volume returns to the basal levels; on the other hand, this leads to severe intoxication (thermal rabdomyolisis). Burn toxemia manifests by fever, pronounced tachycardia, and dullness of heart sounds, anemia, hypo- and dysproteinemia, abnormal hepatic and renal functions.

Acute renal failure (ARF) in burn disease results in a range of phenomena important not only from theoretical, but also from practical point of views. ARF is generally defined as a rapid renal failure resulting in accumulation of protein metabolism degradation products with formation of casts in acidic urine of the renal tubular system.

The most important factors influencing the renal function during the burns are: decreased cardiac output, respiratory failure with hypoxia and acidosis, toxemia and sepsis. ARF in burn disease may be early due to hypovolemia and hypoperfusion of the kidneys or late, occurring after a week as a consequence of infection and endotoxemia.

Development of ARF in burn disease is a very unfavorable prognostic sign necessitating a complex evaluation.

Anuria in an early phase of burn disease may indicate the development of ARF, particularly if urine findings are positive to hemoglobin, proteins, myoglobin. The immediate cause of anuria in burn disease may be a reflex transfer and penetration of the large quantities of toxic materials into the circulation form the region affected by burns leading to the spasm of afferent glomerular arteriolae producing sudden discontinuation of glomerular filtration. After burns,

sudden increase in the osmotic activity results in the affected tissue. In 20-30% of the patients with burn disease anuria is absent.

The prognosis for patients with early ARF was worse than for patients with late ARF. Rhabdomyolysis caused by flame injury was associated with high mortality. Flame burn with rhabdomyolysis and subsequent ARF predicts very poor survival. If a patient with severe ARF survives, the renal failure recovers over time.

Achieving of high percentage of survival among patients is based on an early diagnosis of ARF, understanding of pathophysiology of shock associated with burn disease, adequate therapeutic approaches, including both medicamentous treatment and extracorporeal hemodialysis along with early surgical management. For the time being, hemodialysis is the most effective therapeutical procedure in the treatment of ARF.

Acute burn toxemia continues for 7–8 days.

Septic Phase (Septicemia, toxemia & exhaustion)

In deep and extensive burns, suppuration of the burn wound may occur during toxemia. During this period, patients with deep and extensive burns show signs of general deterioration, which in severe cases may present as weight loss, skin dryness and pallor, pronounced muscular atrophy, bed sores and contractures of joints. The separation of the necrotic eschar starts on days 7–10, the period being characterized by proliferation of microbes and development of varied septic conditions (e.g. pneumonia, pressure sores, and sepsis).

Septicemia starts approximately on day 10 and is characterized by severe infection. Staphylococcus, Pseudomonas and Proteus spp. and E. coli are common causative agents. This may occur as a result of septic processes and activation of the RES system. Well-developed disorders of the immunological system are related to the specific and unspecific defense system. RES cells are damaged by hypoxia. Granulocytes are also disturbed in their enzyme system. The antigen-antibody reaction is injured by a considerable reduction in T-cells which reflects the cellular immune reaction. As the production of immunoglobulin is also reduced, high doses must be delivered. Almost three quarters of patients with extensive burns die of the consequences of a severe infection.

As a result of the long-lasting shock situation, the patient is threatened by stress ulcers. This may lead to anemia, which also develops because of the perishing of erythrocytes after burn injury.

The genesis of burn disease-associated anemia is therefore multifactorial. These factors are the following: hemorrhage, hemolysis and etrythropoesis level decrease.

Complete skin regeneration is indicative of the end of the septicemic period.

The changes in the neurologic state were characterized by pathological reflexes in the hand and feet, vegetative-trophic disturbances indicating that all the patients who had extensive and deep burns developed encephalopathy.

Recovery

Recovery is characterized by restoration of bodily functions, which have been affected previously, i.e. during the earlier three periods of the disease. As the functional organ changes (e.g. those of the heart, liver, kidneys) can persist for as long as 2–4 years after the trauma, the patients with a history of burn disease should be followed up regularly.

Burns can cause scars and keloids — ridged areas caused by an overgrowth of scar tissue. Deep burns can limit movement of the bones and joints. Scar tissue can cause and form contractures, when skin, muscles or tendons shorten and tighten, permanently pulling joints out of position. All that conditions need specific treatment and collaboration between patient and his physician.

6. Light radiation lesions of a nuclear explosion, incendiary weapon

Incendiary stuff (liquids)

Napalm was formulated for use in bombs and flamethrowers by mixing a powdered aluminium soap of naphthalene with palmitate (a 16-carbon saturated fatty acid) — also known as napthenic and palmitic acids — hence napalm. The aluminum soap of naphtenic and palmitic acids turns gasoline into sticky syrup that carries further from projectors and burns more slowly but at a higher temperature.

The two napalms are ‘ordinary’, which produces a temperature of 800–1,200 °C (1,472–2,152 °F) and ‘super-napalm’, enriched with polystyrene, sodium, magnesium or phosphorus, with which the temperatures reach 1,500–2,000 °C (2,732–3,632°F).

When used as a part of an incendiary weapon, napalm can cause severe burns (ranging from superficial to subdermal) to the skin and body, asphyxiation, unconsciousness, and death. In this implementation, explosions can create an atmosphere of greater than 20 % carbon monoxide and firestorms with self-perpetuating windstorms of up to 70 miles per hour (110 km/h). Even people in undamaged shelters can be killed by hyperthermia/heat stroke, radiant heat, dehydration, suffocation, smoke exposure, or carbon monoxide poisoning.

One of the main anti-personnel features of napalm is that it sticks to human skin, with no practical method for removal of the burning substance. Because of napalm’s adhesive quality, the burns it causes are almost always of the third degree. It is estimated that a napalm burn affecting as little as five per cent of the body surface is grave.

The second effect of napalm bombs is carbon monoxide poisoning. A great amount of carbon monoxide is produced right after the explosion of a napalm bomb. It makes it hard for people to breathe and they collapse, and get burned to death.

The third effect of napalm bombs is a burn in the upper part of the windpipe. A victim takes in a breath of heat fumes which burn his windpipe.

Pirogel is combination of napalm and thermite.

Thermite is a pyrotechnic composition of a metal powder and a metal oxide that produces an exothermic oxidation-reduction reaction known as a thermite reaction. Although the reactants are stable at room temperature, they burn with an extremely intense exothermic reaction when they are heated to ignition temperature. The products emerge as liquids due to the high temperatures reached (up to 2,500 °C (4,530 °F) with iron (III) oxide) — although the actual temperature reached depends on how quickly heat can escape to the surrounding environment. Because of thermite`s burning temperature, the burns it causes are almost always of the forth degree.

White Phosphorus

Many anti-personnel weapons used in warfare contain white phosphorus. This element ignites on contact with air, and fragments of phosphorus will be scattered throughout any wounds caused by such weapons. Most of the cutaneous injury resulting from phosphorus bums is due to the ignition of clothing, and is treated as a conventional burn. Fragments of this metal, which ignite upon contact with the air, may be driven into the soft tissues. It is a real problem to deal with a wound in which the embedded particles of phosphorus will ignite as soon as the tissue dries out. Medical teams as well as patients are in danger.

First aid treatment of casualties with imbedded phosphorus particles includes copious water irrigation, and placement of a saline-soaked dressing that must be kept continuously wet. Rapid surgical removal of the identifiable particles is often required. UV light can be used to help locate them.

When surgical treatment is available, the wet wound can be irrigated with a freshly prepared solution of 1 % copper sulphate. This is a very dilute solution; check that it is of palest blue colour. This solution combines with phosphorus to form black copper sulphide, which impedes violent oxidation and identifies the particles. The black particles can then be removed with forceps and placed in a dish filled with water. After completing the procedure, the copper sulphate solution must be washed away, and the wound excised and dressed.

Profound hypocalcemia, and hyperphosphatemia, have been described as effects of white phosphorus injury. Treat with IV calcium.

Light radiation lesions of a nuclear explosion

Nuclear weapons emit large amounts of thermal radiation as visible, infrared, and ultraviolet light. This is known as «Flash». The chief hazards are burns and eye injuries. Flash blindness is caused by the initial brilliant flash of light produced by the nuclear detonation. When thermal radiation strikes an object, part will be reflected, part transmitted, and the rest absorbed. The fraction that is absorbed depends on the nature and color of the material. A thin material may transmit a lot. A light colored object may reflect much of the incident radiation and thus escape damage like anti-flash white paint.

A 1-megaton explosion can cause first-degree burns (bad sunburn) at a distance of about 7 miles, second-degree burns (producing blisters and permanent scars) at distances of about 6 miles, and third-degree burns (which destroy skin tissue) at distances up to 5 miles. In the first 1–9 weeks, in which are the greatest number of deaths, with 60–70 % due to thermal injury.

Thermal radiation can cause burn injuries either directly, i.e., by absorption of the radiant energy (infrared heat radiation) by the skin, or indirectly by heating or ignition of clothing, or as a result of fires started by the radiation.

Flash burns

The direct burns are often called «flash burns», since they are produced by the flash of thermal radiation from the fireball. Flash burns are one of the serious consequences of a nuclear explosion: one of the most striking consequences of the nuclear bombings of Japan was the large number of casualties due to flash burns caused by the thermal radiation. Flash burns result from the absorption of radiant energy by the skin of exposed individuals. A distinctive feature of the thermal radiation (flash) burns was their sharp limitation to exposed areas of the skin facing the center of the explosion. For this reason they are sometimes called «profile burns». The phenomenon occurred because most of the radiation received had traveled in a straight line from the fireball and so only regions that were directly exposed were affected. Another phenomenon, which appeared in Japan after the healing of some of the more severe burns, was the formation of keloids.

Burns under clothing

Skin burns under clothing, which depend on the color, thickness, and nature of the fabric, can be produced in the following ways: by direct transmit lance through the fabric if the latter is thin and merely acts as an attenuating screen; by heating the fabric and causing steam or volatile products to impinge on the skin; by conduction from the hot fabric to the skin; or the fabric may ignite and hot vapors and flames will cause burns where they impinge on the skin. Burns beneath clothing can arise from heat transfer for some time after the thermal pulse ends. These burns generally involve deeper tissues than the flash burns produced by the direct thermal pulse on bare skin. Flame burns caused by ignited clothing also result from longer heat application, and thus will be more like burns due to conventional conflagrations.

Secondary burns

The indirect (or secondary) burns are referred to as «contact burns» or «flame burns»; they are identical with skin burns that result from touching a hot object or those that would accompany (or be caused by) any large fire no matter what its origin. In addition, individuals in buildings or tunnels close to ground zero may be burned from hot debris, gases, and dust.

It has long been known that exposure to radiations, such as X-rays, alpha and beta particles, gamma rays, and neutrons, which are capable of producing ionization, either directly or indirectly, can cause injury to living organisms. The harmful effects of nuclear radiations appear to be caused by the ionization (and excitation) produced in the cells composing living tissue. As a result of ionization, some of the constituents, which are essential to the normal functioning of the cells, arc altered or destroyed. In addition, the products formed may act as poisons.

Beta burns from shallow ionizing beta radiation (this would be from fallout particles; the largest particles in local fallout would be likely to have very high radioactivity because they would be deposited so soon after detonation; it is likely that one such particle upon the skin would be able to cause a localized burn). However, these decay particles are very weakly penetrating and have a short range.

Gamma burns from highly penetrating gamma radiation. This would likely cause deep gamma penetration within the body, which would result in uniform whole body irradiation rather than only a surface burn. In cases of whole body gamma irradiation (c. 10 Gy) due to accidents involving medical product irradiators, some of the human subjects have developed injuries to their skin between the time of irradiation and death.

7. Triage and treatment at medical care echelon

General therapy

Pre-Hospital and Emergency Room Care for Burn Patients

1. Fluid resuscitation.
2. Reverse potassium effects in cellular membrane with calcium chloride 10 % (10 ml intravenously over 10 min).
3. Transfer extracellular potassium into cells:
— Glucose (250–500 ml of 10 % solution) + insulin (5–10 U).
— Sodium bicarbonate (50–100 mEq over 5–10 min).
4. Remove potassium from the body by means of diuretics, potassium exchange resins or in serious cases, hemodialysis.
5. Care about:
— Urine output (use permanent bladder catheter).
— Hyperventilation to avoid respiratory alkalosis.

Hospital Care

The quantities of infusion are calculated every day and can be administered by means of a central venous catheter. Specific shock therapy will however always be the basis of all further infusion therapy.

Fluid Resuscitation should be started within the first 24h postburn:

Estimate crystalloid needs for the first 24 hours, using the following formula:

Total Volume = (2 ml) × (% deep burn) × (kg weight).

Half of this total volume is programmed for the first 8 hours postburn, and half for the second 16 hours postburn:

Burns < 30 % TBSA do not require colloid infusion.

If albumin is not available, fresh frozen plasma or synthetic colloid can be used at the same dose. If none of these is available, continue the LR until the 48th hour postburn, monitoring urine output, and so forth.

Adjuvant to resuscitation:

- Low-dose dopamine.
- Digitalis.
- Vasodilators.
- P-blocker, calcium channel blockers.
- Diuretics.
- High doses of corticosteroids can be administered, as they are a useful prophylaxis against pharyngeal, pulmonary, and even cerebral edema.

Throughout treatment regular monitoring is necessary, with essential laboratory tests of electrolytes, clotting time, hepatic impairment, and blood glucose.

Once the diagnosis of acute tubular necrosis has been made, it is clearly indispensable to begin immediately a therapy whose foundations are:

- Clinical nutrition.
- Hemodialysis and Hemofiltration.

Clinical nutrition:

Infusion with glucose only may be associated with:

- The inhibition of lipogenesis.
- An increase in the oxydization of the glucose and of the glycogen deposit.
- An increase of the catecholamines.
- Increased consumption of O_2 and increased production of CO_2.

So, the use of glucose only is not advisable in the presence of respiratory failure and in the case of patients in mechanical ventilation.

On the other hand, the combined glucose-lipids system has many advantages:

- Less metabolic overload compared to the infusion of a single substratum.
- The supply of the essential fatty acids.
- The diminished frequency of hyperglycemia and hepatic steatosis.
- A reduced production of CO_2 and consumption of O_2.

Hemodialysis (continuous renal replacement therapy — CRRT):

The basic principle of action of CRRT is the elimination of inflammatory mediators, urea, creatinine and uremic toxins with the maintenance of water and electrolytes balance.

- It depends on four physical principles: ultrafiltration, convection, diffusion and adsorption.

• CRRT has the capacity to eliminate inflammatory mediators, depending on the type of filter used, up to 30,000–50,000 Daltons (D).

Indication for Hemodialysis and Hemofiltration:

• Oliguric renal failure.

• Massive myoglobinuria (in electric burns).

The psychological guidance and care of burn victims is important for successful therapeutic treatment. After healing they need a protracted period of time for rehabilitation, often combined with plastic surgery, which in the end will allow these aesthetically injured patients to return to associate with other people.

After intensive medical treatment, the patient may be given further wound care, physiotherapy, ergotherapy, and the necessary psychiatric treatment in a special follow-up ward. The primary care physician providing follow-up care must be able to recognize scar contractures leading to functional restriction and may refer the patient to the Burn Center once again for a new operation.

Local care

In general, try to restore coverage and movement in dorsal injuries by 14 days. Palmar surface can tolerate longer periods of healing, but still attempt to have coverage and movement by 21 days. Wounds that will heel in this time period should not have surgical intervention (mostly partial thickness and superficial 3A degree).

Local Care of Superficial Burns

First degree burns care includes 3C:

• Cleaning.

• Cooling.

• Creaming (avoid oil and fat).

Second degree burning wound care:

• Debride loose skin, pop blisters.

• Cleanse with mild anti-bacterial soap.

• Place some type of anti-bacterial cream or ointment. Apply a topical antimicrobial cream twice daily after thorough cleansing with a surgical detergent such as chlorhexidine gluconate. One-percent silver sulfadiazine burn creams should be used.

• Burn creams are not really necessary in small, superficial burns.

• Keep wounds moist until they heal.

• Systemic antibiotics are not indicated early on.

Following burn cream application and other, burns may be treated as:

• Open (in case of single, small, superficial burns).

• Closed (wrapped in gauze). Extremity wounds can be wrapped in a thick layer of roller gauze that is changed twice daily with cream.

• Half-closed (wet-dried gauze). Dressed with fine-mesh gauze are treated open, with a heat lamp applied until the gauze is dry. After dressing extremities are immobilized for 4–5 days. The dried gauze on the wound is allowed to separate spontaneously.

• Moist wound cover (hydrogel cover). It combines value of closed and half-closed method

Hand Burns

Want to keep moist but allow movement. Glove filled with antibiotic cream (loose-fitting surgical glove) are indicated.

Local Care of Deep Dermal and Full-Thickness Burns

Deep Dermal and Full-Thickness Burns should be managed by early excision and grafting. This is best performed in the first 5 days, prior to the onset of infection and hyperemia under eschar. If this «golden period» is missed, one should usually wait at least 3 weeks prior to surgery.

Escharotomy

It used to improve perfusion by releasing restrictive burn eschar. Use electrocautery to do this. May add dorsal hand (avoid exposing tendons). Consider intrinsic release to avoid contracture. Try to avoid finger fasciotomies, but may be necessary (exposes structures, however).

Skin Grafting

When no white structure is exposed and the wound bed has good granulation tissue, skin grafts can be used for coverage and definitive closure of the wound. Split thickness skin grafts (STSG) are thinner and have a better chance to «take». They can be used as meshed or unmeshed. Meshed grafts have the advantage of allowing seroma and hematoma drainage. Maintaining good contact between the graft and the recipient bed is important for a successful «take» rate. Sheet grafts theoretically cause less scarring, and are arguably better cosmetically. The most common donor areas for STSG are the thighs and buttocks. However, the area can be adapted according to the wound distribution over the body areas.

Thickness skin grafts are usually taken with a mechanical dermatome. In adults, generally harvest at a level of 15/1000" thickness, thinner in babies and older patients.

Tangential Excision

Burns are excised using a sharp excision knife Weck-Goulian, Humby, Braithwaite. Burned skin is «tangentially» excised until multiple punctate bleeding points are seen. This can be an extremely bloody procedure. Some place STSG same day, others wait to get control of bleeding with epinephrine.

Donor sites dressed with fine-mesh gauze are treated open, with a heat lamp applied until the gauze is dry. Grafted extremities are immobilized for 4–5 days. Grafted sites are inspected 4–5 days after surgery. They should be inspected sooner in case of fever, malodor, or other evidence of infection.

Physical and occupational therapy are begun as soon as graft take is sufficient to discontinue immobilization, usually 5 days after surgery. Extremities are splinted in the position of function at night. The dried gauze on the donor site is allowed to separate spontaneously, at which time the donor site can be recropped as necessary for further grafting. After all wounds are closed, the patient is fitted for custom compression garments. If garments are not available, compression can be achieved with ace bandages.

Full-Thickness Grafts

Usually limited to delayed reconstruction. Sometimes is useful in acute burns, particularly for burns of the pulp of the fingers.

Initial Flap Coverage

Some deep burns (4th degree) with exposure of bone or tendons post-debridement may require a flap. The pedicled groin flap is often the best choice, particularly in women and/or children. Free flaps may be necessary, particularly in electrical burns. Options include muscle flaps, ALT, etc.

Flaps

Hundreds of flaps are described for dealing with soft tissue lost from the extremities. However, not all can be applied for war injuries. The most commonly used flaps in these situations are presented here. There are no different general principles of wound management for upper extremities as compared to lower extremities.

Choosing the ideal coverage method depends on the defect size, the patient's condition, the surgeon's preference, and the associated injuries. Principally, pedicled flaps are more often employed than free flaps for closing war wounds.

Local Flaps

These can be axial or island and are usually used to cover small- to medium-sized defects. Their applicability in war injuries is especially for small wounds such as those produced by gunshot. When a larger wound is present, these flaps have limited applicability because their blood supply may be compromised. While they are not widely accepted to cover wounds in the acute phase, they might be useful at a later stage, several weeks after the injury. During this time they behave like «trained» flaps with a greater chance of survival. The 1:1 to 2:1 proportions should be considered for random flaps in the limbs. When an axial flap is used, there are reports of using long and thin flaps in the noncompromised areas.

Cross-Limb Flaps

These types of flaps were widely used in the past but are rarely employed nowadays. As a principle, the flap is harvested from the contralateral limb, set into the wound, and kept connected for several weeks. Most often, a period of 3 weeks is enough for establishing the blood supply to the flap from the periphery and the wound bed, and disconnection of the pedicle may be accomplished.

There are two main drawbacks of this method: the need for continuous immobilization of the limbs for about 3 weeks in order not to shear the flap pedicle and the donor area deformity, which usually requires a skin graft for closure. Generally, these flaps are used when no other reconstructive method is available or when a free-flap failure was encountered. The method has a high rate of success.

Regional Flaps

These flaps have good applicability, and are also indicated when the zone of injury is not extensive. They are categorized into fasciocutaneous, adipofascial, fascial, or neurovascular. They are reliable flaps when good planning and judgment is used. Medium to even large size wounds can be covered in this way.

Distant Pedicle Flaps

These flaps are rarely employed for the lower limbs for the same reasons as for not often using cross-limb flaps. The upper limb, being close to the thorax and the abdomen, is more favorable for these types of flaps. Soft-tissue injuries to the hand and the distal forearms can be covered by a groin (lap or lower epigastric abdominal) flap. Arm injuries can also be closed by lateral thoracic flaps.

The reconstruction is done in stages. First the flap is harvested, insetted, and kept connected for 2–3 weeks. In the second stage, the flap is detached and trimmed for the final inset. The period of limb immobilization can be shortened if periodic flap ischemia is simulated by external compression. When white structures are exposed, they can be covered by the same type of flaps.

Flaps based on the Filatov-Crane principle can also be used. After 2–3 weeks of the flap inset, the limb segment is extracted only with granulating tissue, thus leaving the donor skin paddles in place. The granulating tissue is subsequently covered by skin graft.

Fascial and fasciocutaneous Flaps are used more for upper extremity reconstruction or for areas where a thin, pliable tissue is required for coverage.

In the past years, it has been proved that fascial, adipofascial. and fasciocutaneous flaps are not less successful for fractured bone coverage and for dealing with osteomyelitis then myocutaneous flaps.

Muscle and myocutaneous pedicle Flaps have more often been applied for lower limb reconstruction. There is no need for microsurgical anastomosis. The gastrocnemius and soleus muscle flaps are the most used flaps for calf wound reconstruction. These flaps have the advantage of filling the deep soft tissue loss to avoid dead space. There are numerous reports that muscle flaps are useful for open bone fracture coverage.

Free Flaps are indicated for:

- Large wounds, when a local or regional flap would not be sufficient for local or regional flap coverage.
- Vascular reconstruction; emergent flow-through free flaps are employed when soft-tissue defects and artery defects are present in an ischemic limb. When the arterial defect is small, a T-anastomosis can be used.
- Exposed white structures, such as bones, tendons, nerves, and major blood vessels.

• Functional reconstruction, when significant muscles have been destroyed; reconstruction of elbow flexion or ankle dorsiflexion is the problem most often encountered.

•When sensate flaps are needed, such as for the plantar or palmar area.

Post-Operative Care

• Need to keep immobilized for at least 5–10 days, or until graft has good primary take.

• Then begin careful motion with therapist.

• Must continue splinting.

• Small areas of graft loss are treated conservatively.

• May consider compressive garments and/or silicone sheeting for a number of months.

Definitive burn care, including surgery and rehabilitation, is manpower and resource intensive; therefore it is generally inadvisable to perform excision and grafting of burns in a theater of operations. However, under certain circumstances, this may be unavoidable. Definitive burn surgery in the combat zone is generally not recommended.

XV. FROSTBITE

Trench foot and frostbite together have accounted for over 1 million US casualties in WW I, WW II, and the Korean War. Influencing factors include: previous cold injury; fatigue; concomitant injury resulting in significant blood loss or shock; geographic origin; nutrition; tobacco use; activity; drugs and medication; alcohol; duration and exposure; dehydration; environment (temperature, humidity, precipitation, and wind); and clothing.

Freezing (frostnip)

Not true frostbite; freezing is limited to skin surface only, signals imminent likelihood of frostbite developing, resolves quickly with warming. Exposed skin appears red or minimally swollen, tissue is not actually damaged.

1. Frostbite

Associated Clinical Features

Frostbite is true tissue freezing resulting from heat loss sufficient to cause ice crystal formation in superficial or deep tissue (***Primary crionecrosis***). It caused by exposure to temperatures low enough to cause crystal formation in the tissues. Risk exists at temperatures below -2 ° C. Frostbite develops after exposure for 1 hour. Development of frostbite dependent on many factors: wind chill, altitude, duration, wetness, and prior exposure. Fingers, toes, and ears most commonly involved.

Frostbite may affect the extremities, nose, or ears (and the scrotum and penis in joggers). Severity of symptoms is usually proportional to the severity of the injury. A sensation of numbness with accompanying sensory loss is the most common initial complaint. Often, by the time the patient arrives in the ED, the frozen tissue has thawed. Frozen tissue may appear mottled blue, violaceous, yellowish-white, or waxy. Following rapid rewarming, there is early hyperemia even in severe cases. Favorable signs include return of normal sensation, color, and warmth. Edema should appear within 3 h of thawing; lack of edema is an unfavorable sign. Vesicles and bullae appear in 6 to 24 h. Early formation of large clear blebs that extend to the tips of affected digits is a good indicator. Small dark blebs that do not extend to the tips indicate damage to subdermal plexi and are a poor prognostic sign.

Non-Freezing Cold Injury

Chilblain

Results from intermittent exposure to temperatures above freezing, usually accompanied by high humidity and moisture; 1 to 6 hours of exposure. Swelling, tingling pain, and numbness with pink-to-red flushing of skin (especially the fingers). Extremities will be pruritic as they warm up. Symptoms usually subside overnight; some superficial scaling may occur. Mild joint stiffness may occur acutely but subsides in a few hours. No permanent damage occurs.

Perniosis, also known as chilblain cold sores, is the result of nonfreezing cold exposure. Pernio appears within 24 h of cold exposure, most frequently on the face, ears, hands, feet, and pretibial areas. A large range of lesions may be seen, with localized edema, erythema, cyanosis, plaques, and blue nodules occasionally progressing to more severe lesions including vesicles, bullae, and ulcerations. The lesions persist for up to 2 weeks and may become more chronic. They are typically very pruritic and associated with burning paresthesias. Following rewarming, pernio often takes the form of blue nodules, which are quite tender.

Immersion Injury / Trench foot

Trench foot occurs from prolonged exposure to cold, wet conditions or prolonged immersion of feet at temperatures as high as 17 °C for > 12 hours. Shorter duration at or near 0 °C results in the same injury (occurs in nonfreezing temperatures 0–12 °C). Tight footwear increases risk of trench foot. Microvascular vasospasm with tissue ischemia ***(secondary crionecrosis*)** is the apparent etiology of trench foot and immersion injury. Nerve, muscle, and endothelial cells are most susceptible to this long-term cooling.

Immersion injury is also known as peripheral vasoneuropathy, shelter foot, sea boot foot, and foxhole foot. Immersion injury is a peripheral nonfreezing cold injury resulting from exposure to water, usually at temperatures just above

freezing. However, it can occur during prolonged exposure (or water immersion) to any wet environment cooler than body temperature.

It is distinct from tropical immersion foot or warm-water immersion foot as seen in the Vietnam War. Tropical immersion foot was typically seen after 3 to 7 days of exposure to water at 22 to 32 °C. These syndromes were characterized by burning in the feet, pain on walking, pitting edema, and erythema, with wrinkling and hyperhydration of the skin. They resolved completely after rest and removal from the wet environment.

Associated Clinical Features of the Trench Foot and Immersion Injury

First symptom often is the feet becoming cold, mildly painful, and numb. Common symptoms are «cold and numb» or «walking on wood». Foot may appear swollen, with the skin mildly blue, red, or black. Limb is hot and often hyperhidrotic. On rewarming, pain is excruciating and does not respond to pain medication, including morphine. The first symptoms appear in hours; tissue loss may require many days of exposure**.** As time progresses, liquefaction necrosis occur distally, but more proximal tissue may also be compromised. No sharp line of demarcation of dead from viable tissue.

Postinjury sequelae include pain, numbness, loss of proprioception, and cold feet. Hyperhidrosis with subsequent paronychial fungal infections are common.

2. Classification, clinical features, diagnosis and treatment at the echelon of care

Frostbite grading **(**Frostbite classification).

Classification into degrees is primarily a retrospective evaluation and has little treatment value. A more clinically useful grading typically divides injuries into superficial or deep.

*Superficial frostbite i*nvolves only the skin with swelling, mild pain, and minor joint stiffness.

- 1st Degree (superficial). Erythema, edema, & hyperemia. No blisters or tissue loss. Nonmedical personnel can manage simply by rewarming.
- 2nd Degree. Erythema, vesicles, superficial skin slough.
- *Deep frostbite*. White-hard, anesthetic, blanched, and inflexible. Skin will not move over joints.
- 3rd Degree. (full-thickness skin) Edema and grayish blue discoloration followed by skin loss to subcutaneous level. On rewarming, there is great pain and a blue-gray- to-burgundy color change.
- 4th Degree. Cyanosis, no blisters or local edema. Necrosis of subcutaneous tissue down to muscle, tendon, and bone. Involves deeper tissues to include bone.

Blisters form and are clear, fluid-filled, or hemorrhagic (the latter indicates a more severe, deeper injury). They should be left in place; will slough in 7-10

days without consequence. Failure to form vesicles in an obviously deep-frozen extremity is a grave sign.

3. Preventing frostbite

As development of frostbite and immersion injury dependent on many factors: wind chill, altitude, duration, wetness, clothing, prior exposure and it can occur during prolonged exposure (or water immersion) to any wet environment cooler than body temperature. The best choice is avoid of them.

Treatment

Field treatment (first aid):

- Prevent further cold exposure.
- Do not massage, rub with snow, or warm part by an open fire or high-heat source.
- Avoid trauma.

Superficial (blanched cheeks, nose, ears, fingertips) frostbite

- Warm with palm of hand or warm, wet cloth; warm fingers in armpits.
- Emollients may help prevent skin from drying or cracking.
- Meticulous skin care is required.

Deep frostbite

- Prevent further cooling of body part as well as the patient as a whole.
- Apply dry, sterile bandage and elevate involved extremity.
- Protect from refreezing during evacuation.
- Evacuate promptly to definitive medical care. Frostbite cases will require prolonged hospital care (9 d on average); therefore, all but those with the most trivial injuries should be evacuated to more definitive care as soon as possible.

Hospital care:

- Dry extremity, warm torso, and allow slow passive rewarming of feet**.** Elevate feet. Never immerse feet in warm or hot water.
- Rehydrate.
- Pain medication. The only effective approach is amitriptyline 50–150 mg at bedtime. Other analgesics are either completely ineffective, or (as with narcotics) do not actually relieve pain.
- Systemic antibiotics and tetanus prophylaxis are indicated when there are nonviable tissues, as with any other contaminated wound, or when there is evidence of infection.
- Ibuprofen or ketorolac should be given as systemic thromboxane / prostaglandin inhibitors.

• Cigarette smoking and/or nicotine use is contraindicated during treatment due to its effect on the microvasculature.

Surgical management is conservative:

• Macerated or damaged skin requires topical antibacterial precautions.

• If vesicles develop do not debride. Blisters should be left intact; ruptured blisters require meticulous antiseptics after unroofing.

• Early surgery is indicated only in the most severe freeze- thaw-refreeze cases, where massive tissue destruction has taken place, and in some more severely infected cases. Debridement of necrotic tissue may be required in trench foot.

• Debride eschar late, amputation often best management of nonviable digits. Normally, surgery should be delayed for at least 6 months («Freeze in January, operate in July»).

***Rehabilitation*:** Early mobilization is vital to prevent long-term immobility. Recovery is protracted and may require evacuation because trench foot may lead to weeks-to-months of pain and disability. Long-term sequelae are very common and include sensitivity to the cold (secondary Raynaud's phenomenon), chronic pain, neurological impairment (paresthesias, loss of proprioception), cold, discolored feet; and gait modification and hyperhidrosis.

XVI. HYPOTHERMIA

Hypothermia is classically defined as whole-body cooling below 35°C. Degree of hypothermia is further defined according to the body's core temperature and the clinical effects seen in a given temperature range.

Causative factors and prevention

• Water immersion.

• Rain and wind.

• Prolonged exposure to severe weather without adequate clothing. The insulation effect of clothing is markedly decreased with wetness, which increases the conductive heat loss.

To prevent hypothermia should stay dry and avoid windy exposure.

1. Classification, clinical features, diagnostics and treatment at medical care echelon

1. ***Mild hypothermia*** > 33 °C (> 91 °F) — adinamic stage.

Shivering, hyperreflexia, amnesia, dysarthria, poor judgment, ataxia, apathy, cold dieresis are present.

2. ***Moderate hypothermia*** 28–33 °C (82–91 °F) — stupor stage.

Standard hospital thermometers, mercury as well as digital, cannot measure temperatures below 34 °C (93 °F). Stupor, loss of shivering, onset of atrial fibrillation and other arrhythmias, progressive decrease in level of consciousness, respiration, and pupillary reaction, eventual pupil dilation.

3. ***Severe hypothermia*** < 28 °C (< 82 °F) — convulsive (agony) stage.

Increased incidence of ventricular fibrillation, which often occurs spontaneously, loss of motion and reflexes (areflexic at approximated 23 °C (72 °F), marked hypotension/bradycardia are present.

4. ***Profound hypothermia*** < 20 °C (< 68 °F) — clinic death, asystole.

Lowest known adult survival from accidental hypothermia is 13.7 °C (56 °F).

Treatment

Prehospital (field) treatment

Awake patients

- Remove wet clothing; dry and insulate the patient.
- Give oral warm sugar solutions to hydrate.
- Walk out or transport to next echelon (this should be attempted if it is the only alternative because it is likely to worsen the condition).
- Although walking may deepen hypothermia due to the return of peripheral colder blood to the core, adequate prehydration decreases the postexposure cooling.

Comatose patients

- Patient should remain horizontal and be handled gently to avoid inducing arrhythmias; do not massage.
- Remove wet clothes, dry, insulate, and add an outer vapor barrier.
- Wrap patient in multiple layers of insulation.
- IV fluids, warmed to 40–42 °C, if possible. Do not use lactated Ringer's solution because the cold liver cannot metabolize lactate; warm (40–42 °C [104–107.5 °F]), 5 % Glucosae/dextrosae solution is the fluid of choice.

Remember: The patient is not dead until he is warm.

Continue resuscitation, if possible, until patient has been rewarmed.

Hospital care:

Ventilate; apply CPR if asystolic or in ventricular fibrillation.

As the body cools, the peripheral vasculature constricts, causing pooling of cold acidotic blood. Rewarming the periphery of the body rather than the core causes an inrush of this cold acidotic blood into the core, further dropping the core temperature and worsening cardiac instability.

Core rewarming needs heated and humidified oxygen, external warm blankets, and warm-water torso immersion.

For ventricular fibrillation: rewarm core to 32 °C (90 °F) and attempt cardioversion, continue rewarming and repeat, defibrillate after every 1 °C rise in temperature.

Cardiopulmonary resuscitation: if cardiac monitor shows any electrical complexes, check carefully for apical and carotid pulses before initiating CPR. If any pulse — however thread — is present do NOT initiate CPR.

Treatment of mild stable hypothermia

Insulation, heat lamps, warmed intravenous fluids, warmed forced air, hair dryers have been used for this purpose.

Treatment of severe hypothermia with hemodynamic instability

Cardiopulmonary bypass with rewarming, when available, is the ideal technique in this circumstance because it provides core rewarming while ensuring circulatory stability.

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(на английском языке)**

**Учебно-методическое пособие по военно-полевой хирургии
для студентов 5 курса факультета по подготовке специалистов
для зарубежных стран медицинских вузов**

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