МИНИСТЕРСТВО ЗДРАВООХРАНЕНИЯ РЕСПУБЛИКИ БЕЛАРУСЬ

УЧРЕЖДЕНИЕ ОБРАЗОВАНИЯ
«ГОМЕЛЬСКИЙ ГОСУДАРСТВЕННЫЙ МЕДИЦИНСКИЙ УНИВЕРСИТЕТ»

Кафедра травматологии, ортопедии и ВПХ

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НЕОТЛОЖНАЯ ХИРУРГИЯ ВОЕННОЙ ТРАВМЫ

Рекомендовано учебно-методическим объединением по высшему медицинскому, фармацевтическому образованию в качестве учебно-методического пособия для студентов учреждений высшего образования, обучающихся по специальности 1-79 01 01 «Лечебное дело»

EMERGENCY SURGERY OF MILITARY TRAUMA

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В данном учебно-методическом пособии отражены вводные вопросы военно-полевой хирургии, касающиеся терминологии, классификации, принципов диагностики и лечения. Материал изложен в соответствии с современными представлениями по данным отечественной и зарубежной литературы.

Учебно-методическое пособие составлено в соответствии с типовой программой по военно-полевой хирургии для студентов 5 курса факультета иностранных студентов учреждений высшего медицинского образования.

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EDUCATIONAL AND BEHAVIORAL GOALS, MOTIVATION FOR LEARNING OF THE TOPIC, REQUIREMENTS FOR THE INITIAL LEVEL OF KNOWLEDGE

The academic purpose of the classes: To teach students how to diagnose combat injuries and their complications, modern approaches to the tactics of treating these injuries. Teach to carry out the right choice of treatment for patients with injuries in the army and emergency situations.

Educational purpose of the classes: Formation of students' academic, social, personal and professional competences of the doctor.

Motivation for learning of the theme: Combat damage is characterized by a relatively greater severity, plurality, vastness and depth of damage to the tissues and organs, and this will create great difficulties during the stages of medical evacuation of the victims.

Requirements for the baseline: The study of topics is carried out on the basis of the knowledge and skills acquired by the student in the sections of previously studied academic disciplines.

As a result of the class:

1) The student must know:

The organization of surgical care in wartime.

The basics of medical sorting and the order of surgical care on medical evacuation levels.

The maintenance, volume and principles of the organization of medical care on medical evacuation levels.

Types and volume of surgical care on medical evacuation levels.

Principles of stage treatment of the wounded and injured.

Classification, clinical features and diagnosis of gunshot wounds.

Features of modern gunshot wounds.

The content, volume and principles of the organization of medical care on medical evacuation levels.

Classification, clinical picture and diagnosis of infectious wound complications.

Principles for the prevention and treatment of infectious complications of wounds.

Stage treatment of the wounded with infectious complications.

Classification of bleeding depending on its source, time of its occurrence and the degree of blood loss.

A clinical picture of bleeding and acute blood loss.

Determining the extent of blood loss in the field.

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Indication for blood transfusions.

Types of transfusion and infusion solutions, indications for their use in the field.

Complications in blood and plasma transfusion and their treatment.

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Medical sorting and anti-shock therapy during evacuation.

Comprehensive pathogenetic therapy for shock at the stage of first medical and qualified care.

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Pathogenesis of MMCS (crush-syndrome).

Periods of MMCS (crush-syndrome) and their symptoms.

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Treatment for head injuries, neck, spine and spinal cord injuries on medical evacuation levels.

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The technique of primary surgical care in the open penetrating wound of the spine and spinal cord.

Indications for emergency surgery for injuries to the skull, neck, spine and spinal cord.

Management of the wounded after surgery on the skull and brain.

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Clinical picture and diagnosis of closed chest injuries (rib fractures, rib valve fractures, fractures of sternum, etc.

Classification, clinical picture and diagnosis of pneumothorax and hemothorax.

Signs of penetrating chest wounds, including heart injury.

The amount of care for victims with chest injuries on medical evacuation levels.

Indications for pleural puncture.

Indications for basic methods of specialized treatment of the wounded in the chest.

Classification and frequency of fractures.

Diagnosis of fractures.

Principles of medical sorting for limb injuries.

Principles of medical care on the battlefield and stages of medical evacuation for injuries to the limbs.

Indications for different types of fracture treatments.

Diagnosis and medical care in vessels and nerves injuries of the limbs.

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Indications and contraindication, and the technique of primary surgical treatment of bone wound.

2) The student must be able:

To ground the choice of the right method for treatment of combat injuries in different localization.

To organize and conduct medical sorting of the wounded at their mass arrival.

To organize and provide the first, pre-medical and first physician care of combat surgical trauma.

To organize and provide treatment during the stages of medical evacuation.

To rehabilitate the wounded with injuries of different localizations.

To bandage the wounded.

Take off the stitches.

To carry out the whole range of first medical care in case of gunshot wounds.

Organize and provide treatment on the stages of medical evacuation.

To diagnose the most common infectious complications of wounds.

To determine the blood type by standard serums.

To identify rhesus compatibility.

To make a temporary bleeding arrest.

To fill the blood transfusion system.

To puncture the peripheral vein.

To make and correctly formulate a diagnosis of traumatic shock and MMCS (crush syndrome).

To carry out medical sorting of the injured.

To provide primary medical care, primary physician and qualified physician care to the wounded in shock and with MMCS (crush syndrome).

To diagnose a closed, open traumatic brain injury on the stages of medical evacuation.

To differentiate penetrating and non-penetrating traumatic brain injury based on clinical data.

To diagnose progressive brain compression.

To assess the severity of the injury in various types of traumatic brain injury.

To organize the sorting of the wounded on the stages of medical evacuation (the wounded in the head to be evacuated to a specialized hospital; the wounded requiring emergency surgery and agonizing ones).

To provide primary pre-medical care on the stages of medical evacuation (transportation, applying of bandages).

To provide primary pre-medical assistance for breathing disorders associated with mechanical obstacles in the trachea (aspiration, tongue retraction).

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To determine the indications and the place for the tracheostomy applying, the use of the air-passage and tongue holder.

To diagnose a complicated and uncomplicated, penetrating and non-penetrating injury to the spine.

To diagnose progressive compression of the spinal cord.

To assess the severity of the condition in various types of spinal and spinal cord injuries.

To organize the sorting and transportation of the wounded on the stages of medical evacuation.

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To diagnose and formulate a diagnosis of limb damage of different origins, localization and accompanying pathology.

To apply transport immobilization, protective bandage, tourniquet.

To make a local anesthesia in fractures.

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CONTROL QUESTIONS FROM RELATED DISCIPLINES

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Biological chemistry: biochemical indicators in normal and in pathology conditions.

History of medicine: the main stages of medicine and military field surgery development.

Radiation diagnostics and radiotherapy: X-ray semiotics of traumatic injuries to the skull, chest, abdomen and musculoskeletal system.

Neurology and Neurosurgery: diagnosis and treatment of closed brain and spinal cord injuries.

General surgery: desmurgy, transport immobilization, bleeding and blood loss, blood transfusion and blood substitutes, wounds, surgical infection.

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Pathological physiology: traumatic, hemorrhagic, infectious-toxic shock.

Topographic anatomy and surgery: topography of the skull, chest, abdomen, projection of vessels and nerves of limbs on the skin.

Pharmacology: drugs and medicines used in the treatment of surgical diseases, infusion therapy, acute renal failure, principles of rational pharmacotherapy.

CONTROL QUESTIONS ON THE CLASSES TOPICS

Practical classes:

1. Organization of surgical care in army and in emergency situations.

Definition and content of military surgery.

Main stages of military surgery development.

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

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

Perspectives and future of combat surgical care.

Modern combat trauma. General characteristics of modern weapons.

Influence of modern gunshot wounds on medical care organization.

Possible structure of mortality in modern warfare and its variability.

Medical care in evacuation echelon.

Role of the triage in medical care organization.

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

2. Gunshot wound and principles of its treatment on medical care levels.

Modern types of firearms.

Wound ballistics and mechanisms of projectile impact.

Mechanism of gunshot injury.

Morphological and functional changes in tissues.

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

Zones of tissue damage in gunshot wound.

Morphology of wound canal (entrance or exit wounds, zone characteristics).

Concept of primary and secondary wounds microbial contamination. Early prevention of infectious complications.

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Medical care in gunshot wounds on the battlefield and in evacuation echelons. Wound Care.

Recent views on surgical debridement. Indications and contraindications for debridement.

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

Primary and secondary wound healing. Ways to close gunshot wounds after debridement.

Primary, primary delayed and secondary (early and late) sutures. Conditions for the use of a primary suture.

The most common errors in the surgical debridement.

3. Infection complications in gunshot wounds. Tetanus.

Concept of wound infection.

Factors contributing to the development of wound infection.

Wound Microenvironment.

Local purulent infection.

Prophylactic and therapeutic value of antibiotics.

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

Anaerobic infection of wounds. Terms of the appearance, symptoms and signs. Characteristics of different clinical forms.

Prevention of anaerobic infections. Specific and nonspecific treatment at medical care echelon. Outcomes of treatment.

Anaerobic non-clostridial (decay) wounds infection.

Tetanus, its etiology and pathogenesis.

Tetanus symptoms and signs.

Diagnosis, prevention and treatment guidelines for tetanus. Complications and outcomes.

4. Bleeding, blood loss.

Calculating methods for determining the volume of blood loss.

Vascular injury.

Treatment on medical care levels.

Methods of temporary and final bleeding arrest. Indications for blood transfusion.

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

Possible complications of blood transfusion, prevention and treatment.

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Traumatic shock: Definition of traumatic shock.

Frequency and severity of shock state in war conditions.

Resent views on traumatic shock etiology and pathogenesis.

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Clinical shock manifestations in different locations of wounds.

Value of modern anesthesiology achievements and resuscitation management in treating shock state. Modern corrective methods of hemodynamic and respiratory disorders.

Content of antishock measures at medical care echelon. Early prevention of shock state.

Crush syndrome: Crush syndrome pathophysiology and classification. Dependence of clinical manifestations from the volume of damaged tissue, compression strength and duration.

Stage, clinical picture of crush syndrome, treatment at medical care echelon.

6. Injuries of the head. Injuries of the spine.

Injuries of the head: Frequency of open and closed injuries. Classification by injury nature and clinical manifestations. Clinical picture, diagnostics and complications.

Primary care in head injuries on the battlefield. Content of primary medical and surgical care. Organization of specialized surgical care.

Triage of the wounded in a skull.

Injuries of the spine: Classification of open and closed spine and spinal cord injuries. Symptoms and Diagnostics. Primary care on the battlefield. Content of primary medical and surgical care. Organization of specialized surgical care.

7. Gunshot wounds and closed chest injuries

Frequency and classification of chest injuries.

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

Hemothorax.

Pneumothorax (closed, open, tension).

Subcutaneous and mediastinal emphysema.

Pathophysiology of chest trauma complications.

Acute respiratory distress syndrome or acute lung injury.

Late complications of chest gunshot wounds.

Closed chest injuries.

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Limb injuries classification.

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Symptoms of damaging of major vessels and limb nerves.

Methods of final bleeding arrest.

Gunshot fractures of extremities.

Signs of bones fracture.

Medical care organization of injured limbs at medical care echelon, first aid.

Preventive measures of shock, bleeding and infection.

Surgical management of injured limbs. Principles of specific medical care.

Blast trauma: General characteristics and frequency of blast injuries.

Classification of blast trauma.

Pathogenesis and pathomorphology of damage in blast injury.

Clinical features of blast injury.

Blast injury diagnostics. Stages of examination.

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

Management in blast injuries at medical care echelon.

9. Thermal injuries

Burns: classification of burns according to severity.

Degree of burns.

Determining the burn size.

Local and systemic manifestations of thermal burns.

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

Light radiation lesions of a nuclear explosion, incendiary weapon.

Burns triage and treatment at medical care echelon.

Frostbite and Hypothermia: Frostbite. Classification, clinical features, diagnosis and treatment at echelon of care.

Preventing frostbite.

Hypothermia. Classification, clinical features, diagnostics and treatment at medical care echelon.

PRACTICAL PART OF THE CLASS

Guidelines for the class:

Teachers will select thematic patients for educational curation and case history formulation.

Each of you will be offered X-rays of patients with studied pathology.

The educational material will be illustrated with tables, diagrams and illustrations/photographs.

Tests will be carried out on the basic and final levels of knowledge.

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GENERAL QUESTION OF MILITARY TRAUMA

I. Organization of surgical care in army and in emergency situation

Definition and content of military surgery

War (or Combat) surgery is the branch of surgery which research and organize surgical care providing and treatment of combat casualties during military conflicts and extreme situations.

The content of the subject of military field surgery is:

1. Study, analysis and development of new directions of organization of all types of surgical care for wounded and affected in military conditions of various scales.
2. Analysis and study of modern combat surgical pathology.
3. Develop medical kits and equipment for field surgery.
4. Analysis, study and implementation of military field surgery of advanced advances in modern medical science and technology.

The goals of military field (combat) medicine are the return of the greatest possible number of injured soldiers to duty and the preservation of life and health during evacuation (in medical evacuation chain).

For all this goals achieving medical care in emergency situation must be:

Continues and uninterrupted (medical care are providing at all levels of evacuation chain).

Specific (all casualties needs to be divide to the particular group for different type of medical care).

Successive (to the tasks of the next levels).

There is great difference of factors in administering care for solitary urgent trauma patients, compared to the situation of mass of casualties, where the high numbers of injured exceed available resources.

One of the most dramatic ways in which military surgery differs from civilian trauma management is the staged provision of care: emergency surgery is carried out at one locale, while definitive and reconstructive surgeries take place at different sites.

Main stages of military surgery development

In ***the first historical period*** of development (until the 19th century) military field surgery accumulated information about the

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pathology and treatment of non-fire (until the 14th century) and gunshot wounds (except the issues of the organization of medical care for the wounded).

First time "ambulance" vehicles were used for carrying of injured solders during the siege of Malaga in 1487 in Spain.

Ambroise Paré (1510–1590) the French military surgeon pioneered modern military-field medicine. Ambroise Paré refused to use boiling oil, which was the method of treatment for wound at that time. In 1536, he rediscovered Celsus principles: amputation through viable tissue and the use of the ligatures. Paré taught that cold, not heat, is more effective for stopping of wound bleeding [1].

In 1588 William Cloves described the first successful above-knee amputation and, in 1593 Fabry reported the first amputation through the thigh to prevent gangrene. Control of hemorrhage in combat was achieved with introduction of Morel's tourniquet in 1674 and Petil's tourniquet in 1718 [1].

The second historical period of the military field surgery development was marked by the development of scientific foundations for the medical and evacuation support of the combat operations.

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 [1]. This Napoleon's surgeon is often credited with establishing the ambulance team, and modern battlefield triage system that set the priorities for evacuation. During Napoleonic warfare, the highest priority patients were the ones that could be returned to duty quickly [1]. Often, the severely wounded were left on the battlefield until the end of the battle with consequence in mass death. Larrey changed the established pattern of delayed medical care by going forward with his horse-drawn ambulances (called "flying ambulances") to evacuate the wounded who could potentially survive with surgical care and medical supplies into the field of battle [1].

The introduction of ether anesthesia in 1846 and the subsequent development of antiseptics led to more effective surgery and a lower risk of wound infection [1].

N. Pirogov — the founder of scientific military surgery

Nikolai Ivanovich Pirogov (1801−1881) is the most renowned military surgeon in Russian history, performed hundreds of amputations during the Crimean War (1853−1856). In 1847, Nikolai Pirogov was the very first surgeon to use ether anesthesia in a field operation. He noted that "war is an epidemic of injuries". Pirogov also introduced nurses on the battlefield and published the Atlas of Cross-sectional Anatomy based on sawed frozen sections [1].

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

The third historical period of the development of military field surgery is the development and implementation of the system of stage treatment of the wounded in the war. The essence of the stage treatment is that the treatment of the wounded is closely linked to the evacuation, with medical and surgical assistance being provided as early as possible after the injury. The stage treatment system determines the amount of surgical care for each stage of medical evacuation, as well as the means and methods of evacuating the wounded. The medical sorting of the wounded was recognized as the main element of the stage treatment system [1].

Early in the twentieth century, technological revolutions in warfare (machine guns, chemical and trench warfare) speeded evolution in medicine and ability to care for casualties. World War I (1914–1918) brought artillery into the battlefield, which caused millions of deaths and additional experience in the care of wounded soldiers [1].

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

World War II (1939–1945) with its global scale and warfare advancements such as tanks and air support increased the capabilities of the medical system, especially in transporting the wounded. During WWI, the number of casualties quickly exceeded the capabilities of civilian hospitals. In World War II the United States starts to use fully equipped mobile field hospitals such as the Mobile Army Surgical Hospital (MASH) for the first time. It was succeeded by the Combat Support Hospital [1].

Helicopters were first used as ambulances during military operations in Burma in 1944. With the advent of air medical transport during the Korean War, transportation time was reduced from 12 hours during World War II to 4 hours in Korea.

Development of military surgery in postwar (World War II) period

The organization of medical care for the wounded in the Vietnam War was so effective that the U.S. was completely rebuilt the system of emergency medical care for peaceful victims. 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, this 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 [1].

Perspectives and future of combat surgical care

The content of the modern — ***the fourth historical period*** of the hystory of military field surgery is the development of the organization of surgical assistance to the wounded in local wars and armed conflicts, the introduction of the concept of early specialized surgical care.

The more efficient evacuation system required a change in philosophy of battlefield casualty care — from "buddy" care (in World War II) to standardized prehospital battlefield trauma care in the current wars (such as Military operation in Iraq) [1]. In low-intensity urban conflict, it is difficult to identify a casualty and get immediate qualified care and it 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 asymmetric hybrid wars, global terrorism and other small terrorist events blur the traditional lines between civilian and combat trauma victims. In addition, national and international natural disasters, such as Hurricane Katrina, Japan earthquake and the Asian tsunami, have created intense focus on the medical community preparation for such events.

Modern combat trauma

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

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

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

Small arms: pistols, rifles, and machine guns.

While a blunt mechanism is the most common source of injury for civilians, trauma appeared during combat is in general penetrating in nature. These are the result of the high energy wounding agents (fragment, bullet, blast injury, penetrating and blunt, primary and multiple, combined injury).

General characteristics of modern weapons

Wound ballistics and injuries are due to the same projectiles used many years ago in Vietnam and Afghanistan. The gunshot wounds, blast trauma, and burns produced by modern 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, fatal

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and not confined to the arsenals of disciplined soldiers. Bearers of these arms today include suicidal fanatics, women, and children.

Another characteristics of 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 and disseminated by detonation. Such cluster munitions greatly increase the casualty-generating potential of power.

Latest trends in military surgery are to deal with the large number of severe lower limb and pelvic injuries resulting from the increased use of improvised explosive devices in Iraq, Afghanistan and Syria. They produce a new type of high energy injury characterized by massive contaminated bone and soft tissue destruction combined with major blood loss.

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 time and persistent bleeding lead to the lethal triad of coagulopathy, acidosis, and hypothermia, resulting in a mortality of 90% [2].

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. Damage control concepts might increase the life and limb salvage rate in troops in the field setting. This approach reduces mortality to 50% in some civilian settings. Damage control techniques should be considered in all multi-system casualties [2].

Tactical Abbreviated Surgical Control (TASC) is damage control techniques in a combat 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 may save precious resources, such as time, operating table space, and blood products reserve [2].

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 [2]:

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

It includes 5 main steps:

Control of hemorrhage and Vascular injury repair.

Exploration to determine extent of injury.

Control of contamination.

Therapeutic packing.

Abdominal closure.

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

Core rewarming includes warmed resuscitative fluids, blankets, ventilator air, and environment.

Reversal of acidosis includes appropriate aggressive resuscitation with crystalloid, colloid, and blood products.

Reversal of coagulopathy includes use only ultra-fresh whole blood if 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 hemodynamic is stable and all major sites of hemorrhage have had time to clot.

Reoperation should be done when the probability of achieving definite organ repair and complete fascial closure are optimal [2].

It should prevent reversal of hypotension, acidosis, hypothermia, and coagulopathy, which typically occurs 24–48 hours following the primary insult.

General Considerations

Philosophy of damage control is "alive patient is above all else" [2].

Avoid hypothermia

Rapidly achieve hemostasis

Perform only essential bowel resections

Close or divert all hollow organs injuries, only performing reconstruction at the second operation after the patient has stabilized and can survive a prolonged operation.

Provide necessary available monitoring of physiology, with periodic assessment of pain control, level of consciousness, and intake and output.

Resuscitate from shock, using appropriate tactics.

Provide organ-specific support, as it is done for CNS injury, pulmonary failure, cardiovascular collapse, and renal dysfunction.

Ensure adequate pain control. Use intravenous (not intramuscular) narcotic agents to reduce pain.

Prepare the patient for transportation.

NB! Shouldn`t trust no one's examination before your own because the patient's condition may have changed, or prior examinations may be inaccurate or incomplete.

NB! Patients don't often suddenly deteriorate; healthcare providers suddenly notice! [2]

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Possible structure of mortality in modern warfare and its variability

It is sad that salvage rates from severe battlefield trauma are similar to previous wars despite improvements in armor, surgery, critical care and evacuation. Died of wounds (% DOW) rate during the WWII American campaign in Europe of 1944–1945 (approximately 3%) was noticeable better than that of the American Civil War (14%) nearly a century earlier. But next big advances in medicine and surgery doesn`t increase in lives saved in forward combat surgical facilities since then (the same rates of 3% in World War II and Vietnam).

Medical care at evacuation echelon

Integrated health services support system to triage, treat, evacuate, and return soldiers to duty in the most efficient manner is the main goal of medical care in combat. 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 (medical care levels)

Level I (self-aid and buddy-aid). It is immediate first aid delivered on the scene. First aid and immediate life-saving measures provided by self-aid, buddy aid.

Level II (self-aid and buddy-aid). It is 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 (first physician care). Immediate physician care delivered behind the scene, 100% mobile. It includes basic primary care, combat stress control, surgical (when required) care. Each service has a slightly different unit at this level, which increases medical capability.

Level IV (qualified physician care). It includes medical and surgical care (qualified surgical care) outside the combat zone, within the communication zone of the theater of operations. Patients require more intensive rehabilitation or special needs. Level IV represents the highest level of medical care available within the combat zone with the bulk of hospital beds. Most of mobile hospitals are modular, allowing the commander to give the medical response to expected or actual demand. Traditionally it consists of the Field Hospital (FH) or General Hospital (GH). It includes laboratory, intensive care, surgical and X-ray capability.

Level V (specialized physician care). Hospitals inside the country will provide the ultimate treatment for patients from the theater (specialized surgical care). Military hospitals are specifically designated

for providing the soldier with maximum return to function through a combination of medical, surgical, rehabilitative, and convalescent care. Under the National Medical System, patients overflowing military hospitals will be treated in civilian hospitals.

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 facialities)[1].

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 to achieve success [1]. 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 and available resources (time, equipment, supplies, personnel, and evacuation capabilities) in the evacuation chain delivered behind the scene. Triage occurs at every level of care and the evacuation system [2].

Sorting (triage) must be:

Continues and uninterrupted (sorting/triage are providing at all levels of medical care).

Specific (all casualties needs to be divide to the particular group).

Successive (to the tasks of the next levels).

Triage Categories

Sorting (triage) is performed at many levels, ranging from the battlefield to the field hospital. Traditional categories of triage are ***Immediate, Urgent, Delayed, Minimal***, ***and Expectant*** (table 1). This classification scheme is useful for mass casualties involving both surgical and medical patients [2].

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) [2].

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) [2].

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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 emergency 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 [2].

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 [2].

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 [2].

Table 1 — Standard military triage categories [1]

Triage category	Characteristics	Example
Immediate	Unstable and requiring 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 patient.
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 are unlikely even with optimal medical resources	Penetrating head wounds Mutilating explosive wounds involving multiple anatomic sites 3rd degree burn > 60% TBSA

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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 [1].(table 2).

Table 2 — Military evacuation priority categories

Military evacuation priority categories	Evacuation actions
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	Evacuation in appropriate occasions

Organization of surgical care in an emergency is an urgent problem of military surgery nowadays.

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

II. Gunshot wound and principles of its treatment on medical care levels

Modern types of firearms

The 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.

Weapons of conventional war can be divided into explosive munitions and small arms.

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

Small arms: pistols, rifles, and machine guns.

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

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soldiers. Modern explosive devices are designed to spread more uniform fragments in a regular pattern over a given area.

Wound ballistics and mechanisms of projectile impact

Combat mechanical damage could be:

In relation to the cover tissues: closed and open.

By the appearance of a wounding agent: bullet, shrapnel, from secondary shrapnel, from cold weapons, wounds with balls, arrows and other firearms.

In the course of the wound canal: blind, end-to-end, tangential, ricocheted.

In relation to the cavities of the body: penetrating and non-penetrating.

In the number of injuries: single, multiple, concomitant, combined.

On the form of tissue damage: damage to soft tissues, with the injury of bones and joints, nerves, vessels, internal organs.

Anatomical: head, neck, chest, etc. [3].

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. In this way, 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" [1].

Low-energy injury:

- superficial injury;
- isolated injury;
- high-energy injury:
- multiple injuries;
- concomitant injuries;
- combined injury.

Last three types of injuries are also known as *"**polytrauma" or "multi-trauma**"*.

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 is mostly related to kinetic energy. 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. The higher bone rigidity in compare to skin and muscle produces a greater resistance and results in greater energy transfer, and commonly results in fracture of the bone [1].

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 flight, the energy loss is proportional to the difference of velocity squares and hence, the energy decreases over longer distance. 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, which improve bullet stability analogous to a spinning top [1].

Once the bullet strikes the target, it may start to rotate and tumble due to energy and speed reduction and by that causes the tissue damage [1].

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 the most significant factor in tissue injury from high-energy projectiles [1]. There are 4 factors in mechanism of gunshot wound: a) compressive ballistic wave, b) missile injury, c) permanent and temporary cavity, d) vortex wake of the bullet or projectile.

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 [1].

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. 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 [1].

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

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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.

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: wound canal and primary necrosis zone*) and ***temporary cavity*** (*secondary necrosis zone*).

Permanent cavity is localized area of defects and cell necrosis, proportional to the size of the projectile as it passes through (wound channel and primary necrosis zone). ***Wound canal (or crush zone***) is filled with torn and necrotic tissues. It 2–4 times exceeds the bullet diameter. ***Primary necrosis zone (contusion zone)*** is wound canal wall of the dead muscle adjacent to the missile tract. The thickness of this zone is variable and averages about 0.5 cm.

Temporary cavity (concussion area or secondary necrosis zone) 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. Stretch in this zone is not great enough to tear tissues, but sufficient to injure capillaries. Inelastic tissues, such as bone, may be fractured in this area. 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 (secondary necrosis zone) [1].

Temporary cavity is the cause of secondary necrosis zone in the next hours and days after the injury. The demarcation between the concussion zone and viable tissues is not always well defined.

Gunshot injuries are classified as either entrance or exit wounds.

Entrance Wounds

Gunshot wounds of entrance are divided into four categories based on their range of fire (the distance from the gun to the victim): distant, intermediate, close, and contact. Entrance wounds over elastic tissue will contract around the tissue defect and have a diameter much less than the caliber of the bullet.

Exit wounds.

The exit wounds can appear cone shaped [1]. The size of an exit wound is determined primarily by the amount of energy possessed

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by the bullet as it exits the skin and by the bullet's size, shape, and position. 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.

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 [1].

In high-energy wounds the projectile may allow the large cavity formation at the exit site to suck into the wound foreign materials or debris, so that contamination is spread widely away from the wound track [1].

The battlefield environment is conducive to wound infection due to [2]:

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.

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 [2]. This explains why at least 24 h of intravenous antibiotic treatment is required in injury caused by high-energy weapons in conjunction with the appropriate surgical care [1].

NB! Prompt surgical debridement is the cornerstone of prophylaxis and treatment of wound infections.

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

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tissue. Removal may be surgical, mechanical, chemical, autolytic (self-digestion), and by maggot therapy (where certain maggots selectively eat only necrotic tissue).

Primary Surgical Wound Care includes:

Limited longitudinal incisions for revision (examination).

Excision of foreign material and devitalized tissue.

Irrigation, bleeding arrest, drainage.

Leaving the wound open (no primary closure of gunshot wound).

Antibiotics and tetanus prophylaxis.

Splint for transporting (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 [2].

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

Skin

Conservative excision of 1–2 mm of damaged skin edges may be required. Excessive skin excision should be avoided. Questionable areas can be assessed at the next debridement [2].

Fat

Damaged, contaminated fat should be removed.

Fascia

Damage to the fascia is often minimal relative to the level 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. Wide fasciotomy is often required to prevent further compartment syndrome development [2].

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 [2].

Bone

Fragments of bone with soft-tissue attachments and large free articular fragments are preserved. It`s need to remove all small devitalized, avascular pieces of bone that have no soft-tissue attachment. Bone ends of any fracture should be exam independently with next cleaning of the bone surface and the ends of the medullary canal [2].

Nerves and tendons

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Do not require debridement, except for trimming frayed edges and grossly destroyed portions. Primary repair is not performed.

Vessels

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

Irrigation.

After surgical removal of debris and non-viable tissues, irrigation is performed before cleansing. If there is a lack of sterile saline solution for washing wounds, to predict the depletion of resuscitation resources, drinking water can be used as an alternative. The last liter of irrigating fluid should be a sterile solution with antibiotics [2].

Bleeding arrest (hemostasis) must be performed at the end of debridement.

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: It`s better not to plug the wound with packing as this prevents wound drainage. Best solution in the end is to cover the wound with a nonconstricting, nonocclusive dry dressing over the wound [2].

NB! No primary closure of gunshot wounds.

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.

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 [2]. Depending on the timing of the initial surgical care is called:

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Early, if performed in the first day after the injury;

Delayed if performed within a second day; late, if performed on the third day and later;

Repeated surgical care of the wound (according to primary indications) is performed when the progression of secondary necrosis in the wound (in the absence of signs of wound infection) is detected. The purpose of the operation is to remove necrosis, diagnose and eliminate the cause of its development.

Primary and secondary wound healing

Primary healing of wounds occurs in conditions of good preservation of tissues and skin, lack of tension and infection, before the development of granulation. It goes without the formation of a hard (keloid) scar. Primary healing could be difficult to achieve in war wounds because their high energy nature. Soft-tissue war wounds heal well without significant loss of function through secondary intention. This is especially true for simple soft-tissue wounds [2].

Ways to close gunshot wounds after debridement

Early soft-tissue coverage is desirable within 3–5 days; when the wound is clean, to prevent secondary infection.

Definitive closure with skin grafts and muscle flaps should not be done in theater when evacuation is possible. The formation of flaps should not be performed during primary surgical wound care [2].

Conditions for use of a primary suture

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

Primary, primary delayed and secondary (early and late) sutures

Early primary closure (1 days) requires a clean wound without injury to the surrounding tissue. 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 [2].

After the elimination of local inflammatory changes in order to accelerate the healing wound defects impose a primary late closure (after 5–7 days, before to the development of granulation) and secondary sutures (“early” — within 7–14 days after injury on the granulating

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wound and “late” — after 21 days on the wound covered with scar tissue with its further excising).

The most common errors in surgical debridement.

Experience of military surgeons in Afghanistan, Syria and Ukraine revealed the following defects debridement:

not enough high-quality primary debridement;

suturing the wound before the optimum date (most surgeons, educated in the principles of peace-time surgery, complete surgical debridement 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 devises.

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

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COMPLICATIONS IN MILITARY TRAUMA

III. Infection complications in gunshot wounds, tetanus

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 [2].

Wound infection is an infectious process that develops in the walls of the wound canal and the tissues surrounding the wound, under the influence of microorganisms that enter the wound from the surface of the skin and from the surrounding space. 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.

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, color, 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 (from frank pus to the foul "dishwater" discharge);

Increased pain;

Absent or abnormal granulation tissue;

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Systemic effects such as fever, leukocytosis, unexplained tachycardia, or hypotension [1].

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 opening of the deep fascial layers of the wound, or if systemic illness is present.

Nonspecific purulent infection:

- local;
- regional;
- generalized;
- specific wound infection;
- anaerobic and aerobic non-clostridial (decay) wounds infection;
- anaerobic (clostridial gas gangrene) infection;
- tetanus;
- specific processes (erysipelas, diphtheria of wounds).

Factors contributing to development of wound infection

The battlefield environment is conducive to wound infection due to:

Not enough high-quality primary debridement.

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

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

Delay in casualty evacuation [2].

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 [1]. Typically, over the course of therapy, resistant gram-negative bacteria were responsible for the majority of infections. It becomes possible because Bacterial biofilms formation. This biofilms protect bacteria from systemic antibiotics, topical agents; make host defenses unable to clear infections and impaired migration and proliferation of immune cells. In this way Bacterial biofilm is the major barrier to wound healing.

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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 [1].

Common Microorganisms Causing Battlefield Infections:

Gram-positive cocci: staphylococci, streptococci, and enterococci.

Gram-negative rods: Escherichia Coli, Proteus, and Klebsiella.

Pseudomonas, Enterobacter, 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 (decay infection).

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. Empiric therapy should be considered in case with evidence of fungal infection [1].

Nonspecific Local and Generalized purulent infection

Wound 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 pathogens.

Local infectious complications of combat injuries are the suppurative infectious processes, which are localized and bordered near the primary focus of the wound.

Regional infectious complications of combat injuries are a group of various clinical and morphological suppurative infectious processes, which are spread out from the primary focus of infection in different direction.

Generalized infectious complications of combat injuries are the infectious processes, when infection agents invade the bloodstream (because of absence of immune protection) and be carried throughout the body, producing generalized bloodstream infection (septicemia).

They are following their clinical and morphological forms:

Local (localized form: prevalence of immune system on infection agents):

Suppuration of the wound;

An abscess of the wound canal (of the wound cavity or organ).

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Regional (spreaded form: immune system and infection agents forces are relatively equal):

- wound phlegmon;
- burrowing pus;
- purulent fistulas;
- post-traumatic and gunshot osteomyelitis;
- an acute thrombophlebitis.

Generalized (immunodeficiency and domination of infection agents):

Post-traumatic gangrene;

Wound sepsis.

Systemic sepsis*:* Sepsis is a condition observed in the presence of an infectious (surgical) hearth. Sepsis (from the Greek word sepsis — "decomposition," "rotting") is understood as a non-specific life-threatening reaction of the body to the generalized infectious process, arising from the constant or periodic intake of microbes (toxins) from the primary hearth and with the inability of the immune system to their localization and suppression (outside the infectious hearth) due to the developed immunodeficiency and disturbance of homeostasis (poly-organic insufficiency due to systemic inflammatory response syndrome), and lost the initial dependence of the general process on the primary hearth.

The end of 20th century was marked by the evolution of the representation of the pathogenesis of sepsis as the result of a "cascade" of cellular and humoral reactions associated with the release of inflammatory mediators from the cells of the body, stimulated by the toxins of microorganisms. It occurs with the inability of anti-inflammatory factors to support homeostasis.

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 manifested by systemic hypoperfusion: profound hypotension, mental obtundation, or lactic acidosis [1].

Prophylactic and therapeutic value of antibiotics

Antibiotics have advanced the successful management of war wounds. Since 1943, when systemic penicillin was introduced onto the

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battlefield, the risk for wound infection 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 [2].

The recently published guidelines for the prevention of infection after combat-related injuries recommend the early use of intravenous first-generation cephalosporin at Level I and II medical care in the combat zone for all extremity injuries [1].

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. Almost all battlefield wounds and incisions should have the skin left open [2].

Treatment

General Principles of wound infection treatment

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 [2]. Excessive irrigation should be avoided.

Drainage is generally employed in abscess cavities to prevent infected fluids accumulation. The skin is left open, and a lightly moistened sterile gauze dressing is applied [2].

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 [2].

The tailor therapy must be administered to cover the actual pathogens recovered on Gram stain and culture. Because Bacteroides and Clostridia are difficult to culture, tailor antibiotic therapy should cover these organisms [2].

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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 [2].

Broad-spectrum intravenous antibiotics are 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 [2].

Support for failing organ systems, such as cardiovascular collapse, acute renal failure, and respiratory failure. Intensive care support for sepsis involves intensive resuscitation to restore perfusion for preventing multiple organ dysfunctions. This requires optimization of hemodynamic parameters (pulmonary artery pressure, cardiac output, and oxygen delivery) to reverse anaerobic metabolism and lactic acidosis. Normalization of such marks as urine output, base deficit, and blood lactate levels guide successful treatment and resuscitation [2].

Anaerobic and aerobic 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 aerobic infections with anaerobes. Organisms can include, for example, Bacteroides or Peptostreptococcus with a facultative anaerobe such as the Escherichia coli, Enterobacter, Klebsiella, Pseudomonas aeruginisa, Acinetobacter baumanii or Proteus; or non-group A streptococci. It occurs most frequently in patients with immunosuppression, diabetes, peripheral vascular disease, alcoholism or intravenous drug use. Commonly it is antibiotic resistant hospital infection. These organisms create a slightly advancing infection within the subcutaneous tissues and/or muscle by producing exotoxins that lead to bacteremia, toxemia, and death [2].

Pathomorphology:

Soil-borne anaerobes are particularly well adapted to surviving harsh conditions (such as changes in pH and temperature). Often, there is a lack of nutrition and the presence of numerous other species competing for resources. 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

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to enter a living host, they start rapidly proliferate, far in excess of the affected immune system's capability to defend. The combination of bacterial load and ability to multiply is corresponding with mass-production of exotoxin that causes severe damage to local tissue. Drainage or discharge smells of decayed flesh or fish.

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

Clinical picture

Characteristic features presence of foul-smelling, the release of abundant hemorrhagic exudate. Granulations are faded, white-grayish. Due to the affection of vessels, there is a high risk of secondary bleeding. Decay infection is characterized by the formation of extremely wide zones, in which the area of detritus (liquefied zone) passes into the zone of decay necrosis.

Specific signs of the decay wound infection:

The rotten (foul) nature of the exudate, which is a dirty gray color with droplets of fat.

Wounds and cavities have been colored in black, gray, brown, blue-green, emerald.

Rotten nature of detritus (nonstructural, gray, gray or brown, without white blood cells (no pus!).

Characteristic tissue necrosis with the predominance of alteration over exsudation.

Antibacterial resistance (a frequent source of intra-hospital infection).

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 [2].

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, 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 conjunct the infectious process. Further antibiotic treatment should be based upon Gram stain, culture, and sensitivity data when available.

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Anaerobic infection of wounds (Gas gangrene)

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*** destruction 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 even surgery, although minor procedures [2].

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 Clostridium perfringens, the local and systemic manifestations of infection are due to the production of potent extracellular protein toxins by the bacteria. These toxins hydrolyze cell membranes, provoke protein decomposition with hiperhidratation, cause abnormal coagulation leading to microvascular thrombosis (further extending the borders of devascularized and thus anaerobic tissue), generating gas at the same time. 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 cases [2].

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 "rotten eggs" stench — H2S, as result of protein decomposition). The skin may be tense and shiny [2].

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

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Characteristics of anaerobic infections 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 def closed wound has an anaerobic environment that is ideal for Clostridium perfringens.

Spontaneous gas gangrene is commonly caused by hematogenous spread of Clostridium septicum from the gastrointestinal tract with 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 Clostridium perfringens, Clostridium 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.

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, make a skin sensitivity test first. Then give pentavalent gas gangrene antiserum intravenously and repeat it after 4 to 6 hours.

Specific and nonspecific treatment of anaerobic infections 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 with decompression fasciotomy or Guillotine amputation.

Antibiotics alone are not effective because they do not penetrate ischemic muscles enough to be effective and they will be given as an

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additional treatment to surgery. Clostridial species are exquisitely sensitive to a combination of penicillin G and clindamycin. 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 too. 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 Clostridium 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 [1].

Outcomes of anaerobic infections 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 Clostridium 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.

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***. Clostridium tetani can enter through any wound, even minor burns and corneal abrasions, colon ulcers, skin abrasions in newborn, after appendectomy etc. [2]

Bacteria grow anaerobically and release a CNS toxin (tetanospasmin) that results in muscle spasm, trismus, neck rigidity, and back arching [2]. Tetanospasmin binds to spinal cord 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 and

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inhibits or stops the release of certain inhibitory neurotransmitters. 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 and lymphatic vessels from the wound, it can affect many different presynaptic terminals resulting in spasm effect on other muscles.

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 bone fractures and joints dislocation.

In severe cases spasms of the vocal cords and muscles involved in breathing appear. It`s life threatening condition that needs immediate medical help (mechanical ventilation with miorelaxants administration).

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 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. This condition is rare in developed countries.

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 break, or abdominal injury, surgery or ulcer colon disease.

Incomplete tetanus immunizations.

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.

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

Laboratory tests are rarely used to diagnose tetanus.

Treatment

Life saving measures at tetanus

Neuromuscular blockade (miorelaxants administration);

Endotracheal intubation, mechanical ventilation [2].

NB! Intensive medical care improves the prognosis in severe cases!

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 for sedation.

Place patient in a dark, quiet room free of extraneous stimulation [2].

Prevention (prophylaxis)

In addition to surgical debridement of wounds, additional prophylactic measures for tetanus-prone 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 [2].

Tetanus 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. Death is usually due to respiratory failure or disturbance of heart rhythm. Intensive medical care improves the prognosis in severe cases.

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IV. Bleeding, blood loss

Hemorrhage is the acute loss of circulating blood volume. An element of hypovolemia is present in nearly all polytraumatized patients. Hemorrhage is the most common cause of shock and of preventable death on the battlefield [1].

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]:

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 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 patient 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;

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• hardware;
• mathematical.

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

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

In military surgery blood loss volume can be estimated 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 liter, belly — up to 2 liters, multiple fractures of the pelvis, 2.5−3.5 liter, open fractures of the hip — 1,5−1.8-liter, closed femur fracture - 2 liters, calf — up to 0.8 liters, the shoulder — 0.6 liter, forearm — 0.3 liter, 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 liters of blood, or 4% of TBV [4].

The level of central venous pressure (CVP), as an integral indicator reflects right ventricular pump function and directly correlates with the TBV. Reduction of central venous pressure 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.

Indicators of laboratory hematology are the most informative method for estimating the volume of blood loss after 12−18 hours of the bleeding onset.

Vascular injury

The large physical forces and thermal effects damage the vascular wall during the traumatic event. These injuries may range from a mild contusion of the adventitia or a minor intimal damage to a complete transaction of the vessel [1].

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 (3Ps: pallor, pulselessness, paresis, or paralysis), and a thrill. Presence of a hard sign with a single wound indicates primary surgery intervention in

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the emergency room. High-energy traumas such as periarticular knee and elbow injuries, or a gross displacement fracture are associated with high rate of vascular trauma [1].

In contrast, ***venous trauma*** is manifested by hemorrhage, not ischemia. Venous 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 [1].

A pseudoaneurysm or a pulsating hematoma may develop if the arterial bleeding is partially contained by nearest tissues and fasciae [1].

Treatment on medical care levels

In vascular trauma, the goal of treatment is to stop continuing bleeding, and then to revascularize ischemic limbs. Injured patients with limb vascular trauma are frequently saved from exsanguinations by use of rubber tourniquets that are available to every soldier in the battlefield nowadays [1]. Final repairs of injured blood vessels are performed on IV (qualified) and V (specialized) medical care levels.

Methods of temporary and final bleeding arrest

Methods of temporary bleeding arrest

Direct pressure at site of injury is the most effective and simple method of hemorrhage control.

Elevation of the extremity will decrease most.

Hemostatic bandages may stop bleeding.

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

Military Anti-Shock Trousers will decrease bleeding by vessels and soft tissue circular compression.

Clamping vessels may be used if there is continued bleeding and a damaged vessel can be easily identified.

The temporary vascular shunt serves for temporary revascularization by restoring blood flow to the limbs. It reduces the ischemic time until the definitive vascular restoration will be done. Early shunting protects the extremity from further ischemic necrosis and reduces levels of circulating tissue injury markers [1].

Tourniquet may be first choice in combat. A tourniquet should be applied if previous techniques were ineffective. It is a most rapid and effective method of hemorrhage control. Tourniquet does not require constant attention.

NB! Tourniquet should be used early, before severe blood loss appear.

Rules of tourniquet applying:

1 step — indication for tourniquet applying;
2 step — choosing of tourniquet applying level;
3 step — underlining stuff;
4 step — applying in tension;
5 step — overlying bends;
6 step — strong fixation;
7 step — tourniquet efficiency checking;
8 step — notation of time of tourniquet applying;
9 step — transport immobilization;
10 step — pain relief drugs administration;
11 step — duration less than 2 hours;
12 step — relaxation every 30 minutes.

Tourniquets should not be removed until the hemorrhage can be controlled by advanced haemostatic agents or until arrival at surgery. Substitutes for issued tourniquet include: belt, torn cloth, gauze, and rope, among others.

Tourniquets on the upper extremity should be placed on the upper arm if bleeding is not controlled by a tourniquet on the forearm. 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.

NB! Tourniquets application for more than 2 hours may increase limb loss.

NB! Never avoid using a tourniquet in order to save a limb, and then lose patients life.

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 [1].

Ligation of an injured vessel should be used if there is adequate collateral blood flow. Major vessel ligation should be used only in dying patients.

Lateral suture of lacerations with end-to-end anastomosis when possible are rapid and effective with low rate of complications.

Reversed venous bypasses are used usually when the gap is of more than 3 cm long. Segments of injured vessels may be repaired by auto-venous segments.

Synthetic grafts should be avoided if possible because of a higher risk of thrombosis, infection and late obliteration.

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The current teaching is to avoid ***venous repair*** in an unstable or multi-trauma patient. Repair of a vein is important when this vein is the only venous drainage route, as in the popliteal vein [1].

All vascular repairs should be well covered by viable clean tissues to prevent late infection and bleeding [1].

Indications for blood transfusion and infusion therapy

Transfusion and Infusion Therapy.

Infusion therapy. If patients have lost 10–30% of their blood, infusion solution (colloid and crystalloid) should be added to restore circulating blood volume.

Blood transfusion. If patients have lost 30–40% of their blood or have ongoing blood loss volume, blood or blood products should be added to the resuscitation. 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 (to restore transport function and oxygenation) and Fresh Frozen Plasma (to restore haemostatic function).

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

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 stays intravascular. This dictates a 3:1 ratio for blood loss compensation when crystalloids are using only [1].

Lactated ringer (LR) or Hartman's solution is a slightly hypotonic solution containing near-normal concentrations of sodium, chloride, potassium, and calcium. It also contains lactate, 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 [1].

Isotonic saline or normal saline (NS), is a solution of 0.9% NaCI in water. The high chloride concentration may induce hyperchloremic metabolic acidosis when used in large volumes (i.e. >3 L) [1].

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 as prevention of brain tissue swelling [1].

Glucose-containing solutions is 5% or 10% glucose solution in water. Following administration, glucose is rapidly metabolized causing unwanted hyperglycemia and, leaving only the free water, it may induce cellular edema [1].

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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 in case of increasing cardiac output [1].

Albumin — 5% and 25% heat-treated human albumin solutions with oncotic pressure of 20 and 70 mmHg are available. The 25% solution should be avoided in patients with true hypovolemia [1].

Dextran — 10% dextran 40 and 6% dextran 70 solutions, both with an oncotic pressure of 40 mmHg are available. Dextran has the strongest intravascular volume effect. It causes dose related coagulopathy in high doses and rarely may induce renal failure due to reduced filtration pressure [1].

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 [1].

Blood components and indication for their use (table 3).

Table 3 — Blood components and their storage conditions [1]

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 a result 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

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Storage, self-life, and availability of these products (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	1y	24h (postmelt)
Platelet concentrate	−60mL	5d	5d

Possible complications of blood transfusion, prevention and *treatment of these complications*

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 stopped 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

Definition of traumatic shock

Shock is an abnormality of the circulatory system that results in inadequate organ perfusion and tissue oxygenation (end organ hypoperfusion) [1].

Shock states may be divided by originating into hemorrhagic (preload) and non-hemorrhagic shock. Non-hemorrhagic shock includes cadiogenic (obstructive ore by contractility dysfunction) and distributive (afterload) shock (i.e. septic) [1].

Recent views on traumatic shock etiology and pathogenesis

Hypovolemic shock is further classified by the estimated blood loss. In the immediate, acute phase of the war conflict trauma treatment, a shock state is considered hypovolemic unless strong evidence suggests otherwise. Reversal of hypovolemic shock will be achieved almost always by intravascular volume expansion, e.g., intravenous fluids and blood products [1].

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Cardiogenic shock is pump failure from intrinsic cardiac failure or obstructive cardiac dysfunction from a tension pneumothorax, or cardiac tamponade.

Contractive shock originating from cardiac pump failure (contractility dysfunction) may be a result of 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 [1].

Obstructive shock. Obstruction of the blood flow to or from the heart may be caused by damage to the great vessels, tension pneumothorax, pulmonary emboli, pericardial tamponade, or mass effect in the mediastinum (i.e. mediastinum emphysema). 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 etc. Emergency 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 [1].

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 [1].

The mechanism for the shock development is schematically as follows. Blood loss and hypovolemia as a result of severe mechanical trauma, or gunshot wound, with accompanied damage to vessels of various calibers provoke a sharp discrepancy between the amount of circulating blood and the capacity of the vascular bed. As result the vegetative centers in the hypothalamus sharply increases the secretion of stress hormones and stimulation of the adrenal glands, which produce almost 10 times higher than the norm of catecholamines (adrenaline, noradrenalin). Catecholamines have a vasoconstrative effect, by stimulation the alpha-adrenal receptors of the vascular wall. The same effect has angiotensin enzyme, which starts to work a little bit later [5].

The general vascular reaction does not affect only the arteries of the heart, lungs, liver and brain, as they are practically devoid of alpha-

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adrenal receptors. The described vascular response has a strategic goal of preserving the circulation of the heart and brain at the cost of periphery ischemia. This protective and adaptive mechanism is called "circulatory centralization". It is the ***compensated stage of shock*** [5].

All these compensatory mechanisms in shock conditions work only a few hours and with insufficient force of adaptive reactions and lack of adequate medical care leads in to development of ***decompensated stage*** of shock. It divided in to 2 stages:

reversible decompensation — exhaustion of sympatic-adrenal and renin-angiotensin systems, acidosis, peripheral vasodilation — drop in blood pressure ;

irreversible decompensation — complete depletion of sympatic-adrenal and renin-angiotensin systems, refractory peripheral vasodilation, polyorganic failure — irreversible loss of blood pressure.

As a result of the disturbance of microcirculation due to blood bypassing the capillary bed develops tissue hypoxia and acidosis with toxic metabolic products accumulation. Hypoxia and acidosis lead to the loss of sensitivity of the vascular wall to vasopressors. The so-called phenomenon of "decentralization of circulation" is developing, accompanied by gross disorders of central hemodynamics with reduced venous blood flow to the heart and reduced cardiac output. These pathological complex of functional and morphological changes of vital organs and systems leads to a "vicious circle" from which the body is not able to get out on its own. There comes an irreversible (refractory) shock and transition to a terminal state with an inevitable fatal outcome [5].

The terminal state, depending on the degree of loss of the vital functions of the body, is divided into preagony state, agony state and clinical death. These conditions are reversible if they are not associated with life-threatening injuries, and immediate resuscitation can save the life of the injured person [5].

Shock clinical manifestations in different locations of wounds, frequency and severity of shock state in war conditions

Shock is marked by inadequate organ perfusion and tissue oxygenation, manifested by poor skin turgor, pallor, cool extremities, anxiety/obtundation, tachycardia, weak or thread 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 treat. 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 polyorganic dysfunction syndrome.

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Occult events that may start locally and turn into a systemic problem with deep vein thrombosis and pulmonary embolism, rhabdomyolysis and severe renal failure, fat embolism and fat embolism syndrome [1].

In addition, there are systemic problems related to the general severity of the injury, especially in the setting of armed conflict polytrauma. These include aspiration and pneumonia, acute lung injury and ARDS, systemic inflammatory response, sepsis and septic shock [1].

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

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 in ICU [1].

Early prevention of shock state

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

Modern corrective methods of shock hemodynamic and respiratory disorders

Respiratory: if severe ARDS develops, the patient may require advanced ventilation devices. Mechanical ventilation should employ lung protective principles according to ARDS protocols [1].

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 [1].

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 [1].

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 [1].

Content of antishock measures at medical care echelon

Level I and II Immediate first aid delivered at and behind the scene: temporary stop of 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 it 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 arrest of external bleeding and tourniquet control, infusion therapy with crystalloids and colloids, bolus of crystalloid solution into the vein (0.8L 0.9% sodium chloride solution) and colloid (0.4 liters), urgent measures to eliminate breathing disorders, evacuation on 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).

Stabilization of hemodynamic with glucocorticoids and normalization of fluid and electrolyte balance.

Revealing the causes of asphyxia and control of acute respiratory failure.

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 life [4].

VI. Muscle mechanical compression syndrome (crush syndrome)

Severe extremity trauma has local and systemic, immediate and delayed effects. Systemic complications may delayed in their development, and difficult to diagnose and treat [1].

The amount of tissue damage and the injury severity are due to the energy transmitted by the traumatic agent. In this way the injuries are divided in to “a high- or low-energy injury” [1].

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Low-energy injury:
• superficial injury;
• isolated injury.
• high-energy injury:
• multiple injuries;
• concomitant injuries;
• combined injury.

Last three types of injuries are also known as *"**polytrauma" or "multi-trauma**"*. Therefore polytrauma is the pathoanatomical issue of ***High energy injury***.

Pathophysiology of high-energy trauma is characterized by muscle and soft tissue damage or death (***rhabdomyolysis***). With rhabdomyolysis there is an efflux of potassium, nephrotoxic metabolites, myoglobin, purines, calcium, potassium and phosphorus into the circulation, resulting in cardiac and renal dysfunction, coagulopathy (traumatic shock, acute renal failure, and disseminative intravessel coagulation) [2].

Main causes of a high-energy trauma are:
• car incident;
• falling from height;
• natural disaster;
• technological disaster;
• gunshot injury;
• blast injury;
• criminal trauma;
• industrial trauma;
• large surgery.

Types of crush injury of a limb are:

Crush syndrome — a high-energy injury to a limb with tears, disrupts, tissue loss and occult crushed muscle tissue mass, especially when a powerful blast force has acted on the wound [1], large surgery (for example: total hip replacement, massive oncologic surgical intervention).

Mechanical muscle-crush injury (MMCI) — a closed crush, typical of casualties crushed under masonry, vehicles, or victims lying in compression for many hours [1].

Prolonged compression injury — a closed ischemic necrosis in victims lying unconscious without movement for many hours [1].

Compartment syndrome. It may occur in case of 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 [1].

Reperfusion injury. It may occur after late restoration of blood circulation (later than 6 hours).

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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 Muscle Mechanical Compression Syndrome (MMCS) ore 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 phosphorus into the circulation, resulting in cardiac and renal dysfunction, coagulopathy. It can cause up to 10L of third-space fluid loss per limb that can precipitate hypovolemic shock [1].

Limb after crush injury.

Crushed, bleeding and swollen muscle leads to tissue necrosis which releases 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 rise rapidly high. Hypovolemic shock is manifested by peripheral vasoconstriction and hypovolemia. It leads to ischemic kidney, decreased renal blood flow and reduced glomerular filtration, or even renal necrosis. After the release of necrotic muscle tissue myoglobin appears in a large number of tubular filtration. In acidosis (acidic urine), myoglobin can be deposited in the renal tubules with formation of myoglobin casts, that increase kidney damage, and finally lead to acute renal failure (ARF).

The systemic causes of death in crush injury 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 [1].

Rhabdomyolysis.

Definition: Traumatic rhabdomyolysis is caused by the destruction of skeletal muscle mass. This may be caused by one or both of direct crush of the muscles or ischemia caused by vascular injury or development of compartment syndrome. Violent crushing destroys muscle immediately; even if the force is insufficient to destroy the muscle tissue, the combination of mechanical force and ischemia will cause muscle death within an hour [1]. Muscle can survive circulatory ischemia for up to 4h (table 5).

Table 5 — Critical tissue ischemic times [6]

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

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External mechanical pressure destroys the regulation of myocytes. Consequently, MMCS causes such gross edema that it may include much of the extracellular fluid and cause *hypovolemic shock* within hours of injury [1].

Cellular destruction 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 (CPK) enzyme and myoglobin are the most known and measured. Potassium is the main cellular electrolyte of interest due to its potential harm to the heart [1].

Acute renal failure (ARF) is the most common and life-threatening complication of rhabdomyolysis, with a high rate of incidence and mortality. Hypovolemia and aciduria have to be present in addition to myoglobinemia for ARF to develop [1]. The most frequent causes for ARF in crush injury are hypovolemia and acute tubular necrosis due to hypovolemia, massive crush soft-tissue injury or compartment syndrome, with resultant rhabdomyolysis and myoglobinuria.

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 iron radicals that attack tubular cells. ARF is manifested by oliguria (< 0.5 cc/kg/h) and a rise in creatinine. If anuria does develop, it may continue for 4–8 weeks before kidney function recovers [1].

Disseminated Intravascular Coagulation (DIC)

DIC is 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. This process produces both incomplete large-vessel thrombosis and, 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. Furthermore, thrombin induces conversations from plasminogen to plasmin in the presence of the newly formed fibrin monomer. This production of plasmin results in fibrinolysis, which may lead to

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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 (to 3–6 weeks) hypercoagulation (late tromboembolism).

Classification in dependence of clinical manifestations from volume of damaged tissue, compression strength and duration

Clinical classification of MMCS and Crush syndrome, according to the severity of injury, the capacity of muscles involved, compression strength, duration and the corresponding results of laboratory tests, is divided into three grades.

First grade v 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 injury small areas — the forearm or shin, with compression of less than 2–3 hours. If fasciotomy will not perform early after injury reducing tension, systemic reactions may occur.

Second grade v moderate: myoglobinuria test positive, CPK greater than 2 million units, serum creatinine and urea nitrogen levels without oliguria, significant blood volume loss, hypotension. It appears in case of injury larger areas — the hip, shoulder or upper limb, with compression of less than 6 hours.

Third grade v severe: myoglobinuria test positive, increased CPK significantly, oliguria, shock, metabolic acidosis and hyperkalemia. It appears in case of one or two limbs injuries, with compression of more than 6 hours.

Stage

Stage of crush syndrome development:

Stage I — Initial (early) stage (shock, I Phase of the disseminated intravascular coagulation (DIC), I phase of acute renal failure (ARF)).

Stage II — Intermediate (rhabdomyolysis, II phase of ARF and DIC).

Stage III — Late (advanced) stage of crush syndrome (secondary immunodeficiency, septic disorder, III phase of ARF and DIC).

Stage IV — Recovery.

Clinical manifestations

Signs and Symptoms

The local manifestation is acute muscle-crush compartment syndrome which develops rapidly in and around the crushed muscle as a

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reperfusion syndrome, and which appears immediately after the liberation of a trapped victim and the consequent removal of the crushing force [1].

Local signs will reflect severity of damage to muscle tissue depending on the nature of injury. In crush of a limb all components of tissue should be considered: bone, muscles, blood vessels, and nerves. Bleeding, hematoma, ischemia, edema, pain, paresthesia, and paralysis may all exist in a same time. Severe edema may lead to compartment syndrome that further aggravates muscle damage [1].

Systemic: potassium, phosphate, urates, creatine phosphokinase enzyme (CPK), and myoglobin go into the blood stream after muscle disruption. 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 [1].

Treatment at medical care echelon

Prevention.

It is important to prevent secondary injury if severe muscle injury has occurred. Treatment of hemorrhagic shock and hypovolemia are of crucial priority. Early reperfusion and early recognition of compartment syndrome may prevent the development of rhabdomyolysis. Once rhabdomyolysis has developed, prevention of renal failure becomes the main task. Therefore, normovolemia in addition to forced diuresis and urine alkalinization may prevent the development of acute kidney injury [1].

On the scene while patient is still trapped

Therapy should be initiated as soon as possible, preferably in the field, while the casualty is still trapped [2]. The injured limb should be cooled with cold water or exposure to cool environment.

NB! Prohibit massage and heat, so as not to aggravate tissue hypoxia.

The primary goal of therapy is to prevent acute renal failure in crush syndrome. Suspect, recognize, and treat rhabdomyolysis early in victims of entrapment [2].

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 [1].

Drinking alkaline beverages (per 8g sodium bicarbonate dissolved in 1000–2000ml water, plus the amount of sugar and salt) change urine pH into alkaline to avoid deposition of myoglobin in the renal tubules.

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Massive infusion must be started as soon as intravenous access has been obtained, even while the victim is still trapped [1]. The sooner fluid replacement is established, the better the chance of avoiding renal failure. It is recommended to establish intravenous (IV) access in a free arm or leg vein [2].

Potassium and lactate containing IV solutions should be avoided. 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 daily in prolonged entrapments [2].

Amputation may be necessary for rescue of entrapped casualties as final attempt (ketamine 2 mg/kg IV for anesthesia and use of proximal tourniquet).

Hospital care:

Foley catheter for urine output monitoring, central venous ppreassure monitoring, serum creatinine, and serum electrolytes may be needed, until necessary urine output are achieved.

Systemic therapy.

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

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 and renal vasodilator can be added to relieve spasm and increase renal blood flow [1].

3. Alkalinization of urine is necessary because acidic urine promotes myoglobin cast formation. Large volume crystalloid infusion may be enough to prevent urine acidification. If this fails, alkalinization of urine with IV sodium bicarbonate to keep pH >6.5 may be necessary. Urine pH should be monitored to titrate bicarbonate dosing [1].

4. Electrolyte abnormalities: Hyperkalemia, hyperphosphatemia, hypocalcemia, hyperuricemia must be corrected. Blood calcium level should be followed and hypocalcemia treated if it is symptomatic [1].

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 are controlled by daily or continuous renal replacement therapies [1].

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 [1].

Peritoneal dialysis is simple, for most patients also received good results. Hemoftitration techniques have also been evaluated for the

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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 [1].

Local therapy

The classical management of MMCS and crush-syndrome includes an immediate fasciotomy, which is performed to achieve decompression, thereby improving both local and distal blood supply. Early fascia cutting reduces tissue tension. The decrease in tissue pressure compartment area prevents or reduces the incidence of crush syndrome and 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 [1].

Open wound: radical debridement, repeated 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 are frequently necessary. The wound is inspected regularly. Repeated bacterial testing for direct microscopy and culture are taken at this time [1].

Amputation: consider in casualties with irreversible muscle necrosis ore necrotic extremity [2].

Amputation indications:

No limb blood flow or severe blood circulation disorder (acute ischemia more than 6 h.) leading to later non-functional limb;

Severe systemic symptoms that endanger the patient's life;

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

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 [1].

Orthopedic treatment should be primarily conservative. Joints are splinted in a functional position. 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 [1].

Outcomes

Most patients with rhabdomyolysis who survive the complexity of their injury will recover also from its complications. The development of acute renal failure adds 20–30% to mortality of critically injured. In the end, rhabdomyolysis-induced renal failure is reversible and most patients achieve normal renal function even after 4–8 weeks of anuria and dialysis [1].

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SPECIAL TYPES OF MYLITARY TRAUMAS

VII. Injuries of a head

Frequency of open and closed injuries

Head injury is one of the most common life-threatening conditions encountered in combat care. Approximately 50% of all deaths from trauma are associated with a significant head injury. In the USA, a patient dies of a head injury every 12 minutes.

Classification by injury nature and clinical manifestations

Traditional Classification of Head Injuries:

Traumatic brain injury is traditionally classified as resulting from penetrating (open) or closed head injury.

Closed injuries are seen more often in civilian settings and may have a higher frequency in military operations.

Open injuries are the most commonly encountered brain injuries in combat.

Scalp injuries may be closed (e.g. contusion) or open (e.g. 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 [2].

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 [2].

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) [2].

Closed head injury (CHI) refers to injury where the cranium remains intact and where the dura has not been breached. This is also

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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 from motor vehicle crashes. In CHI, the disruption in brain function is due to the brain motion and deformation, 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 shock wave, ischemia and hemorrhage. The events most commonly associated with PHI involved bullet, knife, shrapnel, etc.

Penetrating wounds of the skull and brain are characterized by the periodisation of the course of ***traumatic brain disease***. Each period is distinguished by its own neurological and surgical features.

1st — The initial (acute) period of penetrating wounds of the skull and brain is characterized by a severe condition of the wounded, the predominance of general brain symptoms over the local. There are different degrees of consciousness disorder, vegetative reactions, exacerbated by the transportation of the wounded, inevitably occur during this period of wounds. The initial period lasts 3 days.

2nd — The period of early reactions and complications begins on the 3–4th day after the injury and is characterized most often by the build-up of traumatic swelling and swelling of the brain, most pronounced around the area of its damage. The clinical picture of this period is characterized by a more distinct manifestation of focal neurological symptoms against the background of general and focal neurological symptoms. The persistence of traumatic swelling at the same time to a certain extent prevents the spread of infection from the area of the wound canal to the brain and shell.

3rd — The period of elimination of early complications, during which there is a tendency to delineate and cleanse the infectious locus, begins three to four weeks after the injury. In this period, the self-cleaning of the damage centers and the replacement of tissue defects is completed. In the case of an unfavorable clinical course, there may be secondary inflammatory processes - encephalitis and meningitis.

4th — The period of late complications lasts for an average of 2–3 years after injury and is characterized by exacerbations of the inflammatory process, which in some cases leads to the formation of brain abscess.

5th — The period of long-term consequences reflects the persistent end result of trauma and complications, morphologically characterized by the presence of a formed scar at the site of the former injury.

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.

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.

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

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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.

Brain compression is caused by many factors, which include compression of bone breaks, sub- or epidural hematomas, intraventricular hemorrhage, pneumocephaly, expressed swelling — swelling of the brain [4].

The compression of the brain is characterized by an increase in neurological symptoms in the dynamics: aggravation of the disorder of consciousness, the severity of focal lesions and general health symptoms. The presence of a "light gap" is pathological for intracranial hematoma, and the shorter it is, the faster the clinical picture of brain compression develops, the worse the prognosis of the disease [4]. Depending on the background (concussion, brain injury of varying degrees), on which traumatic compression develops, the "light gap" can be deployed, erased or absent. On the second or third day during the formation of intracranial hematoma on the eye bottom can be found signs of stagnation of discs of optic nerves [4]. In the untimely delivery of assistance develops a terminal state characterized by gross dislocation phenomena with a sharp violation of vital functions.

Complications

The prognosis of brain injuries is good in patients who respond to 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 [2].

Following TBI, regardless of injury mechanism, there are significant issues that may affect recovery and cause significant distress. These include somatic symptoms, such as headache, sleep disturbance, and dizziness; emotional difficulties, such as irritability and mood change; and cognitive difficulties, such as attention dysfunction, memory problems, or difficulties in communication. Typically, the frequency and duration of such symptoms is related to injury severity. Continued expression of symptoms may, in part, be linked to emotional factors.

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Late complications:

Infections via the meninges.

Nerve damage — paralysis of facial muscles, double vision, problems with eye movement, and a loss of the sense of smell.

Problems with the senses*:* Tinnitus (ringing in the ears); Recognizing objects may become difficult; Clumsiness, due to poor hand-eye coordination; Double vision; Blind spots; Sensing bad smells; Sensing a bitter taste.

Seizures — TBIs do not increase an individual's risk of developing epilepsy, unless there have been major structural brain injuries.

Alzheimer's disease — risk is linked to severity of the TBI (as well as how many TBIs the patient had).

Parkinson's disease — risk is linked to severity of the TBI (as well as how many TBIs the patient had).

Coma — a percentage of patients with TBI become comatose, go into a vegetative state, and/or eventually die without ever waking up.

Cognitive problems — people with moderate to severe TBI, especially severe TBI, may experience some cognitive problems, including their ability to:

Communicate properly — some patients may have problems with written and spoken language.

Ability to process non-verbal signals is worse than before their injury occurred.

Judge situations.

Pay attention.

Remember things v especially the short-term memory.

Solve problems.

Think, organizing thoughts and ideas properly.

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 [2].

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

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Primary care in head injuries on battlefield

Primary principals are basic but vital:

• clear the airway;

• ensure adequate ventilation;

• assess and treatment for shock (but excessive fluid administration should be avoided).

Content of primary medical and surgical care

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. Delayed intracranial hemorrhages have been reported. These patients should be evacuated at a level 4 or 5 facility. Repetitive injury and exposure to blast over-pressure may result in irreversible cognitive deficits [2].

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 [2].

Organization of specialized surgical care

Surgical management

Goals are to prevent infection and to relieve intracranial hypertension.

Indications for urgent exploration [2]:

Space-occupying lesions with neurological changes (e.g. 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.

Guidelines for penetrated head injuries:

Remove foreign bodies only if it is safe for patient.

Evacuate the hematoma.

Always remove brain debris and nonviable brain tissue.

Perform a complete hemostasis.

Close the dura mater defects.

Avoid hard fixation of a bone flap/fragment.

Always close the galea and skin.

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Surgery:

The hematoma should be gently evacuated with a combination of suction, irrigation, and mechanical removal [2]. Devitalized brain and easily accessible foreign bodies should be removed off.

Careful hemostasis should be achieved and the dura closed. If a duraplasty is required, pericranium, temporalis fascia, or tensor fascia lata may be used. 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 [2].

The galea of the scalp should generally be closed separately with an absorbable suture, and with nonabsorbable suture used to close the skin. A subgaleal or epidural drain may be used at the discretion of the surgeon [2].

Good results can be achieved with cranioplasty after evacuation out of the theater and a sufficient delay to minimize risk of infection [2].

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 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.

VIII. Injuries of the spine

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 [2].

Classification

Four questions must be considered in the classification and treatment of spinal injuries [2]:

Is injury open or closed?

Neurologic status: complete or incomplete rapture 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) [2].

Location of the injury: cervical, thoracic, lumbar, or sacral?

Degree of bony and ligamentous disruption: is it stable or unstable?

Loss of integrity of two of the three vertebral columns results in instability of the spine. Instability is common following blunt injury of the vertebral column, but is not usual in case of gunshot or fragment wounds of the vertebral column [2].

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Symptoms and Diagnostics

As a response to the initial mechanical insult, hemorrhage, edema, and ischemia rapidly follow, extending to contiguous areas of neural tissue. A subsequent biochemical cascade of events that involves a variety of complex chemical pathways leads to delayed or secondary cell death that evolves over a period of days to weeks.

Motor and Sensory 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.

The sensory examination, performed next, should be a careful, systematic examination with a variety of modalities, including nociception, light touch, and proprioception.

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. Incomplete spinal cord lesions may produce a combination of sensory findings that depend on the tracts involved. Radicular pain or paresthesias may be present at the level of the injury and may have localizing value.

Before determining that a patient has a ***complete spinal cord injury***, the patient should be examined carefully with all modalities and vibration for evidence of preserved perianal sensation or sphincter tone. 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. Autonomic dysreflexia may be precipitated by bladder distention, skin stimulation, or bowel distention. Gastric and ileus atony are common.

Bilaterally locked facets or compression fractures of cervical vertebrae suffered in flexion or axial loading injuries are common radiographic changes correlated with complete spinal cord injury.

Incomplete spinal cord injuries require 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***. These syndromes are rarely 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.

Personal Copy

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 proprioceptive sensation may remain intact.

Traumatic spinal cord disease

1st period (acute) — for 2–4 weeks against the background of the general severe condition of the wounded in the clinical picture of trauma is dominated by the phenomenon of impaired nervous conductivity of the spinal cord, which manifests itself in the form of paralysis, anesthesia below the area of damage, impaired function of pelvic organs, etc. In the acute period of spinal cord injury, the neurological symptom complex does not correspond to the level of spinal cord injury. This is due not only to the anatomical mismatch between the length of the spinal cord and the spinal column, but also to the spread of spinal cord swelling in combination with spilled inhibition in its cells and synapses in response to trauma. This phenomenon is called spinal shock. Spinal shock is caused by trauma passing the depression of the reflex activity of the spinal cord outside the zone of the damage.

The 2nd period (early) — lasts 2–3 following weeks, characterized by the same clinical pattern of spinal shock, which by the end of the 3-4th week is gradually smoothed out [4] with particular injury clinical picture development.

The 3rd period (intermediate) — lasts for 2–3 months. During this time, the phenomena of spinal shock are eliminated; it becomes possible to clarify the nature and extent of spinal cord injury. With minor injuries, the spinal cord function begins to gradually recover. In more severe injuries in the wound are scars, sharply disrupting liquor dynamics and circulation, arachnoiditis, meningitis, epidural abscesses [4].

The 4th period (late) — begins with a favorable course of the wound process after 3–4 months after the injury and lasts 2–5 years or more. In this period, the nervous conduction of the preserved elements of the spinal cord is restored [4].

Classification of closed spinal cord injuries

The following types of injuries are highlighted:

Spinal concussion is a functionally reversible form with no signs of morphological disorders of the spinal cord substance, lumbal liquor

without blood. Clinically manifested syndrome partial or complete impairment of conduction, called spinal shock. Recovery occurs with conservative treatment for 5–7 days [4].

A spinal cord contusion is an injury accompanied by morphological damage to the spinal cord substance, the cells of the segment device and the conductive pathways. The lumbar liquor is stained with blood. Brain damage occurs primarily, and it is joined by secondary changes in the form of locus of softening. Regardless of the severity of spinal cord injury, immediately after the injury there is a complete violation of conductivity below the level of trauma, manifested by complete immobility (tetraplegia, paraplegia), loss of all kinds of sensuality and disorder of pelvic organ function on the tin of delay. The prognosis is extremely unfavorable [4].

The compression of the spinal cord is the result of damage to bone structures in fractures and dislocations of the vertebrae, which leads to deformation of the spinal canal by a destroyed intervertebral disc, epidural hematoma, traumatic hydroma, swelling of the spinal cord, a combination of these factors [4].

Anatomical rupture of the spinal cord accompanied by a complete impairment of conductivity below the level of trauma, manifested by complete immobility (tetraplegia, paraplegia), loss of all kinds of sensuality and disorder of pelvic organs [4]. The prognosis is extremely unfavorable.

Hematomyelia is an intravertebral hematoma formed in the central canal cavity of the spinal cord. Clinically manifested segmental and conductor disorders.

Hematarrahis is a hemorrhage under the membrane of the spinal cord, usually subarachnoid. It occurs when blood vessels are damaged, accompanied by meningeal symptoms without damage to the segmentation apparatus and conductor disorders [4].

Radicular (root) syndrome — compression of the spinal roots, romping in the intervertebral hole, which changes in size and shape as a result of trauma damaged intervertebral disc, vertebrae dislocation or bone fractures. The roots of the cauda equina can be stretched in the gross deformity of the spine and are injured by the mechanism of traction. Root syndrome is manifested by pain in the corresponding dermatom, impaired motor function [4].

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 intercostal muscles, have a functional flail chest [2].

Thoracic and Lumbar Spine

Although the thoracic rib cage contributes considerable rotatory stability, it does not protect completely against injuries. The vascular

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supply of the spinal cord is most vulnerable between Th4 and Th6 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 Th10 and L2 [2].

Primary care on battlefield

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.

Field evaluation of patients with suspected spinal injury begins with the primary and secondary surveys. The primary survey begins with evaluation of the airway, breathing, and circulation, followed by assessment of disability and exposure (ABCDE).

All potentially unstable cervical spine injuries should be immobilized in a rigid collar (Philadelphia collar), unless "halo" immobilization is required [2]. 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.

Urgent transport to centers with the appropriate resources should follow initial stabilization in the field. Ideally, patients with SCI should be transferred directly to a facility experienced in the care of these patients.

Content of primary medical and surgical care

In patients with a suspected SCI maintenance of oxygenation and hemodynamic stability with supplemental oxygen, blood pressure support, and early use of blood products may minimize the potential for secondary ischemic injury. Patients who present with hypotension and shock usually have hypovolemia from hemorrhage and should be aggressively treated with fluid resuscitation and blood products. In patients with an injury to the spinal cord, however, hypotension may result from neurogenic shock, which is due to disruption of sympathetic output to the heart and peripheral vasculature. Neurogenic shock is distinguished by bradycardia, instead of tachycardia, in the presence of hypotension. These patients typically require the use of inotropic and chronotropic support to maintain adequate systolic blood pressures. Aggressive fluid resuscitation in patients with neurogenic shock leads fluid overload, pulmonary edema, and heart failure.

Initially, indwelling urinary catheters are recommended.

Closed spinal cord injuries may be treated with an IV corticosteroid if started within 8 hours of injury.

NB! Penetrating injuries of the spine should NOT receive corticosteroid treatment [2].

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Initially, frequent neurologic checks t should be performed every hour in a patient with acute spinal cord injury and no less than twice in an 8h 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 bone fragments position. 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 [2].

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. Phenylephrine (50–300 ug/min) or dopamine (2–10 ug/kg/min) may be used to maintain blood pressure [2].

Bladder Dysfunction.

Failure to decompress the bladder may lead to autonomic dysreflexia and a hypertensive crisis. The bladder is emptied by repeated or permanent catheterization [2].

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 soft stretcher [2].

Organization of specialized surgical care

There is no significant difference in outcomes between early and late surgery in acute SCI. "Early surgery" have significant benefits in terms of more rapid rehabilitation, decreased incidence of pneumonia, decreased length of stay in the intensive care unit. Only spinal injuries that are unstable with or without neurologic involvement require surgical treatment.

Surgical objectives include the correction of spinal alignment, the restoration and maintenance of spinal stability with instrumentation, and decompression of compromised neural elements to permit maximal functional recovery. The indications for immediate surgery are

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progressive neurologic deterioration and fracture–dislocations associated with incomplete or without neurological deficit. In the absence of a neurologic deficit, it is reasonable to delay surgery. Furthermore, organization of a hematoma occurs at about 48 hours after the injury and decreases intraoperative blood loss. Recently, with the development of less invasive spinal interventions, there is suitable in the application of "damage control" principles to spinal trauma.

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 are 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

Frequency and classification of chest injuries

About 15% of war injuries involve the chest. Only 10% are superficial with soft tissue injury only and require basic wound treatment. Other 90% of chest injuries are almost all penetrating. Penetrating injuries of the chest central column (heart, great vessels and 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. 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].

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.

Clinical manifestations of different kinds in chest injuries, their diagnostics

The main etiological factors of functional disorders in the wounded in the chest is acute respiratory failure and severe blood loss with impaired vital organs. Acute respiratory failure is the leading cause of death for up to 50% of chest-injured people at the scene.

Acute respiratory failure is divided into three degrees:

I degree — breathing rate (BR) to 25 per minute, tachycardia 100–110 per minute, reduction of blood oxygen saturation to 92–90%, pH within 7.35–7.30, may be observed minor cyanosis.

II degree — BR 30–35 per minute, oxygen saturation to 81–90%, pH to 7.25, cyanosis [4].

III degree — low intensive breathing, BR more than 35 per minute, pulse 140–180 per minute, blood oxygen saturation 75–80%, pH 7.15–7.20, pronounced cyanosis, loss of consciousness [4].

Preagonal stage is characterized by hypoxic coma.

Hemothorax

The most common causes of ***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 injuries can all cause blunt trauma [3].

Penetrating trauma are such injuries as gunshot wounds or wounds caused by other sharp objects. Penetrating trauma can cut into the lung tissue, causing bleeding and hemothorax [3].

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Common hemothorax symptoms include:
anxiety;
respiratory failure;
restlessness;
shortness of breath;
tachycardia (rapid heart rate);
differant degrees of chest pain.

X-rays in hemotorax determine the oblique fluid level and the displacement of the lungs up and the mediastinum organs in the opposite direction.

Degrees and management:

Minimal (within the rib-diaphragmal sinus — less than 500ml) — thoracentesis is rarely necessary.

Moderate (at the corner of the scapula — 500–1000ml) — thoracentesis and drainage with tube suction.

Massive (at the middle of the scapula — more than 1000ml) — two drainage tubes with suction and early thoracotomy is necessary to arrest bleeding.

Total (above the second rib — more than 1500ml) — early thoracotomy is necessary to arrest bleeding.

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. With continued bleeding, the blood received form blood clots (Ruvilua-Gregoire test). 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 [2].

Pneumothorax (closed, open, tension)

Closed pneumothorax. Rib fractures being are more common cause traumatic pneumothorax. It has been found to occur in up to half of all cases of chest traumas. On physical examination, breath sounds (audible

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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 hyperresonant (like a booming drum), and vocal resonance can be noticeably decreased. The volume of the pneumothorax has limited correlation with the intensity of the injury, and clinical 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 injured, like in case of a stab wound or gunshot wound allows air to enter the pleural space. If the wound of the thoracic wall gapes, there is always a chain of adverse anatomical and functional changes. When inhaling, a portion of air, entering the pleura cavity, compresses the lung, malleable parts of the heart and hollow veins, pushes the mediastinum to a healthy side, and the diaphragm — downwards. When exhaling, the air is pushed out of the pleural cavity; lung partially straightened. As a result, cardiopulmonary syndrome develops [6].

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 [2].

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.

Associated clinical features: subcutaneous air, tracheal deviation, jugulovenous distention (JVD), and diminished or hyperresonant ipsilateral breath sounds can be presents. Subcutaneous emphysema may be visible on the neck and chest and is easily diagnosed by palpation. 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 [2].

The immediate recognition and treatment of tension pneumothorax is the 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. Some or all signs may be present [2].

In cases of tension pneumothorax, immediate decompression with a large bore needle is lifesaving. Video-assisted thoracoscopy or thoracostomy may be necessary to eliminate the site of the air leak [2].

Traumatic pneumothorax may be complicated by hemothorax. X-rays in hemopneumotorax determine the collapse of the lung,

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horizontal fluid level and the displacement of the mediastinum organs in the opposite direction. The presence of a hemopneumothorax needs 2 chest tube placements. If bleeding continues, exploration of the thoracic cavity may be necessary to achieve bleeding arrest.

Subcutaneous and mediastinal emphysema, cardiac tamponade, 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. The most common causes are pneumothorax. Signs and symptoms of subcutaneous emphysema 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. 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.

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).

Since subcutaneous emphysema is an important diagnostic sign, it is necessary to monitor its dynamics. The regression of subcutaneous emphysema is a favorable sign. Its rapid spreading is a sign of severe damage. Rapidly spreading subcutaneous emphysema, combined with the deterioration of the injured, should be regarded as an absolute sign of the tension pneumothorax requiring urgent action.

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 bronchi).

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 through jugular space, releasing the air. Surgery may be needed to repair the hole in the trachea, esophagus or bowel.

Cardiac Tamponade

Cardiac tamponade is accompanied by accumulation of blood in the inelastic pericardial cavity (more than 150ml of blood).

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 [2]. 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 appearance of all three physical signs in one time is a late manifestation of tamponade and usually seen just prior to cardiac arrest. Other symptoms include shortness of breath, dyspnea and symptoms of inadequate perfusion.

Fluid resuscitation may temporarily stabilize a patient in tamponade [2].

Pericardiocentesis is only a stop gap measure on the way to definitive surgical repair (any pericardial blood mandates median sternotomy/ thoracotomy) [2]. Extradermal (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 of penetrating chest injuries

NB! Most penetrating chest injuries reaching medical attention are adequately treated with tube thoracostomy (chest tube) alone [2].

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Tube thoracostomy (chest tube) ***indications:***
Known or suspected tension pneumothorax.
Pneumothorax (including open).
Hemothorax.
Pneumohemothorax.
Any penetrating chest injury requiring transport [2].

Surgical Management of Specific Injuries
Vascular.

Initially, holes in vessels should be occluded by finger pressure. 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.

NB! Surgery is the definitive treatment.

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 [4]. Acute respiratory distress syndrome (ARDS) is a more severe subset of ALI characterized by dyspnea, refractory hypoxemia, decreased lung compliance, and diffuses 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. 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. In same time type II alveolar cells undergo hyperplasia. Type I alveolar cells damage increases entry of

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fluid into the alveoli and decreases clearance of fluid from the alveoli. Type II alveolar cells damage is associated with decreased production of surfactant with resultant decreased compliance and alveolar collapse. The result is diffuse alveolar damage in 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. 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.

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.

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 (fractures of the ribs, damage to lung tissue). It includes restoration of patency of the airways, oxygen inhalation continuous artificial ventilation.

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 [2].

It occurs when multiple adjacent ribs are broken in multiple places, separating a segment, so a part of the chest wall moves independently. The number of ribs that must be broken varies by differing definitions: some sources say at least two adjacent ribs are broken in at least two places, some require three or more ribs in two or more places. The flail segment moves in the opposite direction to the rest of the chest wall: because of the ambient pressure in comparison to the pressure inside the lungs, it goes in while the rest of the chest is moving out, and vice versa. This so-called "paradoxical breathing" is painful and increases the work involved in breathing.

Flail chest will requires 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 [2].

Treatment of the flail chest initially follows the principles of advanced trauma life support. Further treatment includes:

Good pain management includes intercostal blocks and avoiding opioid pain medication as much as possible. This allows much better ventilation, with improved tidal volume, and increased blood oxygenation.

Positive pressure ventilation, meticulously adjusting the ventilator settings to avoid pulmonary barotraumas.

Chest tubes as required.

Adjustment of position to make the person most comfortable and provide relief of pain.

Aggressive pulmonary toilet.

Surgical fixation, that can help in significantly reducing the duration of ventilatory support and save pulmonary function.

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X. Abdomen gunshot wounds and closed injuries

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 possible.

Classification:

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 [5].

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

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

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.

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. 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. Gunshot wounds and other projectiles have a higher degree of energy and produce fragmentation and cavitations, resulting in greater morbidity. However, occult injuries can be overlooked, resulting in devastating complications.

There are of two types damaging factors of mine-explosive wounds of abdominal injuries:

1) shrapnel;

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

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.

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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 organs (i.e. stomach, ileum, jejunum, transverse colon). Retroperitoneal organs include the solid organs (pancreas, kidneys), the hollow organs (duodenum, ascending and descending colon, and rectum), ureters, urinary bladder, major abdominal vessels.

Clinical manifestations of open abdominal injury

One or more of the intra-abdominal organs may be injured in abdominal trauma. The characteristics of the injury are determined by what organ or organs are injured. Intra-abdominal trauma can be associated with rib fractures, vertebral fractures, pelvic fractures, and injuries to the abdominal wall [7].

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 [7].

Symptoms of abdominal penetrating wounds

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 [7].

Wounds of the hollow organs are characterized by symptoms of rapidly evolving peritonitis. Injuries of the hollow organs (the liver, spleen, blood vessels) are characterized 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 of urine from the wound.

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 [7].

Abdominal trauma can be life threatening because solid abdominal organs, especially those in the retroperitoneal space, can bleed profusely, and the space can hold a great deal of blood. Solid abdominal organs,

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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 [7].

Hollow organs such as the stomach 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. Massive one-stage microbial contaminations of the abdominal cavity take place in gunshot wounds of the abdomen with hollow organ injury [7].

Clinical picture of closed abdominal injuries

Abdominal guarding is a tension of the abdominal wall muscles to guard inflamed organs within the abdomen. Pneumoperitoneum (air or gas in the abdominal cavity), leukocytosis and fever may be an indication of a hollow organ rupture because of peritonitis development. A ruptured solid (parenchymal) abdomen organ results in hemorrhagic shock (low blood pressure, tachycardia), because of profuse bleeding [7].

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. Proctoscopy and sigmoidoscopy should be performed.

Wound Exploration

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

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

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Diagnostic Peritoneal Lavage.

It 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.

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 [7].

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 with cardiac arrest or near cardiac arrest. 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 an ultrasound as a confirmatory test for hemoperitoneum may be useful. They must be performed immediately not to 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 [7].

Surgical exploration is necessary for people with penetrating injuries and signs of peritonitis or shock. Laparotomy is often performed in blunt abdominal trauma, and is urgently required if an abdominal injury causes a large, potentially deadly bleeding. Surgery may be needed to repair injured organs. Patients with penetrating trauma who are haemodynamically unstable require immediate operation. These patients should be taken immediately to the operating room, without further unnecessary investigations or interventions. Patients with clinical signs of peritonitis or with evisceration of bowel should be taken immediately to the operating room [5].

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.

Penetrating thoracoabdominal wounds provide a serious challenge to the trauma surgeon as they imply potential injuries to the chest,

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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 [7].

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 with resection of clearly nonviable pancreatic body/tail tissue [7].

Liver Injuries.

Liver injuries present a serious risk for shock because the liver tissue is delicate and has a large blood supply and capacity [5]. 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 [7]. 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 because they are not completely protected by the ribs. Kidney lacerations and contusions may 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 [7].

Small-Bowel Injuries.

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Gas within the abdominal cavity is a diagnostic sign of bowel perforation. Wound edges should be debride to freshly bleeding tissue. Enterotomies should be close 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, single resection with primary anastomosis should be performed. Should be avoided multiple resections.

Colon Injury.

Simple, isolated colon injuries are uncommon. The presence of any of the complicating factors mandates colostomy. Simple, isolated colon injuries should be repaired primarily. For complex injuries, ***strongly consider colostomy or diversion.***

In summary, gunshot wounds to the anterior and posterior abdomen wall can be safely managed nonoperatively. Diagnostic tests can be ordered as indicated, but clinical examination is the most accurate tool to identify patients requiring an operation or not. The key to success with selective nonoperative management is continuous monitoring and frequent clinical examination by physicians familiar with trauma. Negative laparotomy rate reduced to approximately 10% with a tactic of selective nonoperative management.

XI. Pelvis elvis gunshot wounds and closed injuries

Classification of pelvis injuries

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

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 clinical gunshot injuries of the pelvis should be considered for 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.

Wounds and closed injuries of pelvis and pelvic organs

Transpelvic wounds are defined as a missile trajectory between the iliac crest and the perineum. Penetrating wounds that traverse

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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 [7].

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 [7].

Complications in gunshot pelvis injuries

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

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 fractures.
Acetabulum fractures, solitary.

Palpation and percussion allow 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 in rectal or perineal laceration. In a female, a vaginal examination is also important. Female urethral lacerations

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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 is 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.

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.

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.

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 3l. 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 [1].

Rectal Injuries.

Rectal injuries can be difficult to diagnose. Any question of an injury needs rectal examination, plain abdominal film radiography and proctoscopy. All abnormal findings should have corrective intervention. It consider the 4 “Ds” of rectal injury: Diversion, Debridement, Distal washout, and Drainage.

First of all the most important is diversion with transabdominal sigmoid colostomy. If the peritoneum has not injured, exploration of the

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extraperitoneal rectum should not be done at laparotomy. This avoids contaminating the abdominal cavity with stool. Fecal contamination of the perirectal space mandates presacral drainage.

Genitourinary injuries (except injuries of the external genitalia, are typically associated with serious visceral trauma).

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. Gross hematuria in the combat patient is concerning for a significant injury. Hematuria is usually present in patients with renal trauma, but the absence of hematuria does not exclude renal trauma.

Renal trauma is categorized by the range of kidney damage:

Minor injuries. Include renal contusions and cortical lacerations. Nonoperative management contents hydration, antibiotics, and bed rest.

Major injuries. Include shattered kidneys, deep cortical lacerations (with or without urinary extravasation), renal vascular pedicle injuries, and total avulsion of the kidney. Its require a laparotomy for evaluation and repair. Kidney preservation should be done in all possible cases. For the severely damaged kidney total nephrectomy may be required in the unstable patient.

Vast majority of blunt renal injuries will heal with observation and conservative therapy. Renal injuries from penetrating wounds require exploration.

Ureteral Injuries.

Isolated ureteral injuries are rare and usually occur in cases of retroperitoneal hematoma and injuries of the colon, duodenum, and spleen.

Ureteral injuries can represent a difficult diagnostic challenge and are frequently overlooked when not appropriately considered. If they are initially missed, urinary diversion with a nephrostomy tube and delayed repair at 3-6 months is a safe intraoperative and postoperative approach.

Bladder Injuries.

Bladder injuries can occur as the result of abdominal gunshot wounds, pelvic fractures, and laceration by bony fragments from a pelvic fracture on the intraperitoneal or extraperitoneal surface. The localization of bladder injury defines the clinical features, management, and complications of this trauma.

Evaluation of the bladder injuries is performed radiographically with a cystography. Cystography shows extravasation of a radiographic contrast medium in the pelvis on the X-ray film.

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The bladder injury usually heals with 10–14 days of Foley catheter drainage without primary repair surgery. Primary repair and drainage are necessary in cases of abdominal exploration for other injuries.

Urethral Injuries.

The urethra is divided by the urogenital diaphragm into anterior and posterior segments. Anterior urethral injuries may result from blunt trauma or from penetrating injuries. Posterior urethral disruption usually occurs as result of pelvic fracture injuries.

Retrograde urethrography is performed to exam the anatomical integrity of the urethra. If difficulty in urine passing the catheter or urethral disruption is detected, a suprapubic tube cystostomy and surgery is performed. Catheterization is indicated if urethral integrity is not violated.

External Genitalia Injuries

Injuries to the external genitalia usually result from direct trauma. This injury is usually quite painful. Blunt genitalia trauma may result in a significant hematoma formation. External genitalia injuries result in severe bleeding.

The management should consist of hemorrhage control, debridement, and early repair. The external genitalia are highly vascularized, and extensive debridement is usually not necessary for genitalia wounds. When external genitalia skin is lost, these organs may be placed in a skin tunnel until a plastic repair can be performed. Testicle should be resected if is severe damaged and its blood supply destroyed.

Genitourinary injuries treatment adheres to established surgical principles of hemostasis, debridement, and drainage. Proper radiographic evaluation prior to surgery may be useful in the diagnosis of serious genitourinary injuries.

Transporting immobilization in pelvis injures

Patient with pelvic injuries are transported to the “frog position” on a shield or stretcher. Tying a sheet or placing a binder around the pelvis at the greater trochanters level for hemorrhage control are necessary in case of hemorrhagic shock.

Treatment at medical care echelon

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

Resuscitation.

Infusion and transfusion therapy alone (treatment for shock).

Angiography (extremely useful in identifying and controlling pelvic arterial hemorrhage).

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External fixation (opposition of the bony elements for venous hemorrhage control). Definitive internal pelvic stabilization (plates, screws, etc) is done outside of the combat zone.

External tamponade.

Penetrating Injuries

Treatment:

Immediate hemorrhage control by packing.

Aggressive and thorough debridement of wounds and fractures;

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

Diagnosis of associated injuries may require exploratory laparotomy.

Fractures should be diagnosed with radiographs and CT (if it available).

Pelvic stabilization.

Bleeding from main vessels injury should be controlled surgically. Retroperitoneal packing is extremely useful with profuse hemorrhage. Local packing is effective enough for venous bleeding arrest.

The stabilization of pelvic injuries, fractures, and dislocations is the emergency orthopedic procedure, especially when it was complicated by significant internal hemorrhage. Life saving pelvic external fixators placement reduces the volume of the pelvic cavity, stabilize the pelvic ring with closing the “open book” pelvic fractures, and leads to bleeding arrest. In case of severe pelvic injury, the fixators should be placed before exploration of the abdominal cavity. External fixators can be placed in the emergency department.

Stable pelvic bone injuries (stable pelvic, acetabulum, and spine fractures) are generally treated between 24 and 48 h after injuries because of theirs complexities. This interval allows time for specialized studies and for soft tissue injury and bleeding management prior to orthopedic surgery.

XII. Gunshot wounds and closed injuries of limbs and joints

General characteristics and frequency of limb injuries

The extremities are the most common site to be injured in combat environment. Limbs soft-tissue injures are caused by several mechanisms: cold weapon, blast injuries, gunshot wounds and burns. The most common combination is blast with fragmentation injuries. Frag-

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ments from explosive munitions are responsible for 70–80% of combat injuries. The worst prognosis has injury located below the knees [1].

Limb injuries classification: closed and open injuries of bones, nerves and blood vessels

Injuries to the extremities due to traumatic agent are mechanical or caused by gunshot. Gunshot injuries are divided by type of wounding projectile (high-speed, low speed bullets), fragmentation (big and small fragments), explosive (including mine blasting), other (shot, arrow-shaped elements, balls etc.).

Injuries of limbs are divided into two groups according to the type of damaged tissue:

- soft tissue damage only;
- damage to bones and joints (open and closed fractures).
- wounds of the extremities due to type of the wound canal are divided into:
- blind;
- cross-cutting;
- shearing.
- additional injury:
- peripheral nerve injuries;
- vascular injuries;
- crush injuries;
- compartment syndrome;
- fat embolism syndrome.
- according to multiplicity injuries are divided into:
- 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 of 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 are head, neck, chest, abdomen, pelvis, spine and limbs. In the diagnosis of concomitant trauma damaged areas are listed in order “from top to down”.

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

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Last three types of injuries are also known as “polytrauma” or “multi-trauma”.

Frequency and characteristics of major limb nerves damaging

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

Peripheral nerve injuries represent 10% of all injuries and 30% of extremity injuries. These injuries could be mild or severe.

Nerves are made of axons, which are surrounded by tissues that act like insulation. If in a peripheral nerve injury, either the fibers or the insulation could be damaged. In more-severe peripheral nerve injuries both the fibers and the insulation are damaged and the nerve may be completely cut. First types of injuries are more likely to heal, and the second types of injuries are very difficult to treat and recovery may not be possible. Because these nerve injuries are ultimately determine the functionality of the injured limb, it is of major importance to perform initial neurological assessment and medical care as soon as possible at the levels of medical facility [1].

There are next ***types of peripheral nerves injury*** (Seddon, 1942):
Neuropraxia (conduction block).
Axonotmesis (axons divided).
Neurotmesis (nerve divided).

Neuropraxia.

This is the least severe form of nerve injury, with complete recovery. In this case, the axon remains intact, but there is myelin damage causing an interruption in conduction of the impulse down the nerve fiber. Most commonly, this involves compression of the nerve or disruption to the blood supply (ischemia). There is a temporary loss of function which is reversible within hours to months of the injury (the average is 6–9 weeks). Wallerian degeneration does not occur, so recovery does not involve actual regeneration. In electrodiagnostic testing with nerve conduction studies, there is a normal compound motor action potential amplitude distal to the lesion at day 10, and this indicates a diagnosis of mild neuropraxia instead of axonotmesis or neurotmesis.

Axonotmesis.

This is nerve injury with disruption of the neuronal axon, but with maintenance of the epineurium. This type of nerve damage may cause paralysis of the motor, sensory, and autonomic. Its frequently

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seen in crush injury. If the force creating the nerve damage is removed in a timely fashion, the axon may regenerate, leading to recovery. Electrically, the nerve shows rapid and complete degeneration, with loss of voluntary motor units. Regeneration of the motor end plates will occur, as long as the endoneural tubules are intact. Because axonal continuity is lost, Wallerian degeneration occurs. Recovery occurs only through regenerations of the axons, a process requiring time over weeks to years.

Neurotmesis.

Neurotmesis is the most severe lesion with no potential of full recovery. It occurs on severe contusion, stretch, laceration, or complitr rapture. The axon and encapsulating connective tissue lose their continuity. The last (extreme) degree of neurotmesis is transsection, but most neurotmetic injuries do not produce gross loss of continuity of the nerve but rather internal disruption of the architecture of the nerve sufficient to involve perineurium and endoneurium as well as axons and their covering. Denervation changes recorded by EMG are the same as those seen with axonotmetic injury. There is a complete loss of motor, sensory and autonomic function. If the nerve has been completely divided, axonal regeneration causes a neuroma to form in the proximal stump.

For neurotmesis, it is better to use a new more complete classification called the Sunderland System. In 1951, Sunderland expanded Seddon's classification to ***five degrees of peripheral nerve injury***:

First-degree (Class I).

Seddon's neurapraxia and first-degree are the same.

Second-degree (Class II).

Seddon's axonotmesis and second-degree are the same.

Third-degree (Class III).

Third-degree is included within Seddon's Neurotmesis. Sunderland's third-degree is a nerve fiber interruption. In third-degree injury, there is a lesion of the endoneurium, but the epineurium and perineurium remain intact. Recovery from a third-degree injury is possible, but surgical intervention may be required.

Fourth-degree (Class III).

Fourth-degree is included within Seddon's Neurotmesis. In fourth-degree injury, only the epineurium remain intact. In this case, surgical repair is required.

Fifth-degree (Class III).

Fifth-degree is included within Seddon's Neurotmesis. Fifth-degree lesion is a complete transection of the nerve. Recovery is not possible without an appropriate surgical treatment.

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Symptoms of major limb nerves damaging

The signs of nerve damage:
Numbness in the hands or feet.
Muscle weakness, especially in arms or legs.
Sharp pains in hands, arms, legs, or feet.
A buzzing sensation.
Clinical picture of extremity nerve injuries (table 6 and table 7).

Table 6 — Upper limbs nerve injuries

Injured nerve	Common traumatic causes	Motor deficits	Sensory deficits
Axillary nerve injury	Anterior shoulder dislocation Fracture of surgical neck of the humerus Iatrogenic (rotator cuff and shoulder reconstruction surgery	Paralysis of the deltoid muscle present in impaired arm abduction Paralysis of the teres minor muscle present in impaired external rotation of the arm Muscle atrophy present in flattened deltoid	Lateral shoulder (lower half of the deltoid region)
Musculocutaneous nerve injury	Shoulder joint trauma Upper trunk compression (e.g., Erb palsy)	Paralysis of brachialis and coracobrachialis muscles present in impaired elbow flexion Paralysis of biceps brachii present in impaired forearm supination	Lateral forearm, from the elbow to the base of the thumb
Median nerve injury	Proximal: supracondylar fracture of humerus Distal: wrist laceratio and fractures	Proximal injury present in ulnar deviation upon wrist flexion Distal injury present in median claw Both injuries present in ape hand (inability to make OK sign) and muscle atrophy of thenar muscles	Palmar aspect of thumb, index and middle fingers, lateral ring finger
Radial nerve injury	Mid-arm: midshaft fracture of the humerus bone Wrist: radial fracture	Axillary injury present in impaired forearm extension at elbow and wrist drop Mid-arm injury present in wrist drop	Dorsal aspect of thumb, index, and middle fingers, lateral ring finger
Ulnar nerve injury	Proximal: fracture of medial epicondyle of humerus Distal: distal ulnar, hook of hamate bones fracture	Ulnar claw	Palmar and dorsal aspects of lateral half of ring finger, little finger

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Table 7 — Lower limbs nerve injuries

Injured nerve	Common traumatic causes	Motor deficits	Sensory deficits
Superior gluteal nerve injury	Iatrogenic injury due to intramuscular injections in the superomedial region	Paralysis of gluteus medius and minimus, tensor fascia lata present in impaired hip abduction Positive Trendelenburg sign: lateral pelvic tilt towards the opposite side	None
Inferior gluteal nerve injury	Posterior hip dislocation	Paralysis of gluteus maximus present in impaired thigh extension (difficulty standing from a sitting position and climbing stairs) Forward pelvic tilt	None
Femoral nerve injury	Direct injury (trauma) Iatrogenic: pelvic, abdominal, or spinal surgery, total hip arthroplasty	Paralysis of iliopsoas, pectineus, rectus femoris, and sartorius muscles present in impaired hip flexion Paralysis of quadriceps femoris muscle present in impaired knee extension and decreased patellar tendon reflex	Anterior cutaneous branches: anteromedial thigh Saphenous nerve lesion : medial lower leg, medial edge of the foot
Lateral femoral cutaneous nerve injury (meralgia paresthetica)	Compression at the level of the inguinal ligament, caused by: direct impact or hematomas	None	Pain and paresthesias on the lateral surface of the anterior thigh
Obturator nerve injury	Pelvic ring fractures	Paralysis of hip adductors	Medial thigh
Sciatic nerve injury	Direct trauma (gun and/or stab wounds) Hip dislocation Iatrogenic: Total hip arthroplasty	Paralysis of hamstring muscles → impaired knee flexion Motor deficits of tibial nerve injury and common peroneal nerve injury	Lower leg and foot
Tibial nerve injury	Tibial fracture Tarsal fracture	Paralysis of foot flexors present in inability to walk on the toes or balls of the feet; inability to invert foot	Sensory loss over sole of the foot
Common peroneal nerve injury	Fracture of the fibular head Iatrogenic: compression with tight casts	Deep peroneal nerve: paralysis of foot and toe extensors (dorsiflexors) present in foot drop present in high-stepping gait Superficial peroneal nerve: paralysis of peroneus longus and peroneus brevis present in impaired pronation of the foot	Deep peroneal nerve: area between the first and second toes Superficial peroneal nerve: lateral surface of the lower leg, dorsum of the feet and toes, except for the space between the first and second toe

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Nerve Disruption Management

If the nerve is injured but not cut in half, it may not need surgical repair, but only conservative treatment. The risk of leaving a nerve lesion untreated is unacceptable and justifies a more aggressive early approach.

Surgery.

Sometimes a section of a nerve is cut completely. When it possible, reconnect healthy nerve ends an end-to-end suture of the nerve (*nerve repair*) performed during the initial emergency procedure gives the repair the highest chance of success. If an end-to-end suture of the nerve cannot be performed, a delayed graft (*nerve graft* from another part of the body) will be performed. This nerve repair may be done during the replacement of the external fixation for an internal device [1]. These procedures can help injured nerves to regrow. Sometimes surgeon can borrow another working nerve to make an injured nerve work (*nerve transfer*).

Rehabilitation.

If the nerve is healing properly, it may need to rest the affected area until it's healed. Nerves recover slowly and maximal recovery may take many months or several years. Physical therapy may prevent stiffness and restore function. In some cases, it is needed to address anti-inflammatory drugs and/or corticosteroid injections to relieve pain.

A number of treatments can help restore function to the affected muscles.

Braces or splints. These devices help hold the affected limb, fingers, hand or foot in the proper position to improve muscle function.

Electrical stimulator. Stimulators can activate muscle served by an injured nerve while the nerve regrows. However, this treatment may not be effective for everyone.

Physical and occupational therapy. Therapy involves specific movements or exercises to keep an affected muscles and joints active. Physical therapy can prevent stiffness and help restore function and feeling.

Exercise. Exercise can help improve muscle strength, maintain range of motion and reduce muscle contracture.

Frequency and characteristics of major limb vessels damaging

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.

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Vascular limb injury. It`s signs and diagnostics (symptoms of major limb vessels damaging)

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. Presence of a major sign with a single wound leads to primary surgery intervention in the emergency room.

Mild signs 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 [1].

Arterial injuries of the extremities are manifested by life-threatening hemorrhage or by ischemia resulting in limb loss. *Hard signs of arterial injury* should lead to immediate surgical exploration, without further preoperative studies. *Mild signs of arterial injury* that require additional diagnostic evaluation include proximity of wound to major vessels, history of hemorrhage, hematoma, diminished pulse, and anatomically related nerve injury [2].

In contrast, ***venous injury*** is manifested by hemorrhage without ischemia. Bleeding may be internal or external and rarely may lead to hypovolemic shock. In difference to arterial injury, repair of major veins should be avoiding in an unstable or multi-trauma patient.

The **6 P**s of acute ischemia are: 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 [8].

Simple classification of acute limb ischemia:
according to adequacy of collateral flow:
compensated ischemia;
subcompensated ischemia;
decompensated ischemia;
according to duration of the ischemia:
mild ischemia;
moderate ischemia;
severe ischemia.

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Compensated ischemia: with adequate collateral flow (present blood flow is sufficient for all types of tissue).

Subcompensated ischemia: with partial collateral flow (present blood flow is sufficient for connective tissue and insufficient for neural and muscular tissue).

Decompensated ischemia: with absence of collateral flow (present blood flow is insufficient for all types of tissue).

Decompensated ischemia:

Mild ischemia can be defined as a cool, pale limb in which there is no impairment of muscle function or loss of sensation (***reversible changes***). Duration is up to 3–4 hours from injury [8].

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. The skin capillaries will be pink (***irreversible changes***). Duration is between 4 to 6 hours from injury [8].

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 *necrotic changes*. Duration is longer than 6–8 hours from injury [8].

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 [8].

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 operative revascularization) and relates to the systemic risk. If muscle has not yet been devitalized, anticoagulation sufficient to stop clot formation in the limb will be associated 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 if restoration of limb circulation is concerned, but will associate with risk of systemic complications [8].

In case of severe ischemia, the risk of reperfusion is probably acceptable only if the limb can be revascularized within the critical 6 h prior to muscle death. Later than this, surgery will be unsuccessful and will be associated with high mortality. Amputation done after sufficient time will provide a much better chance for successful healing at whatever the level selected [8].

NB! Ischemia of striated muscle for more then 4–6 hours (irreversible muscle spasm) will lead to myonecrosis and major amputation.

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Methods of final bleeding arrest

NB! Save Life over Limb.

Initial management includes:

Immediate external bleeding control with direct pressure to the bleeding wound is preferable.

Temporary tourniquet (blood pressure cuff) placed proximal to the injury site and inflated above systolic blood pressure may be helpful.

If all conservative techniques are unsuccessful, temporary prolonged shunting with different modifications can be used.

Indications for operation in a suspected vascular injury:

Major signs of a vascular injury.

Minor signs of a vascular injury confirmed by Ultra Sound or angiography.

Ligation is possible in case of compensated ischemia.*Ligation of major veins* is acceptable in life-threatening situations, but in a stable patient and proper medical envirmoment, venous repair should be performed and may enforce arterial repair [2]. *Ligation of artery:* only small arterial vessels with good collateral blood flow can be ligated.

Type of repair will depend on the size of injury.

In case of vessels injury with subcompensated ore decompensated ischemia if gap is less than 1–2 cm:

Lateral suture repair: Required for minimal injuries without flow compromise more than 25%.

Patch angioplasty*:* Needed for larger, tangential wounds to prevent stenosis of the vessels.

End-to-end anastomosis: Could be done if it is possible to mobilize vessels ends (generally less than 2 cm gap) without tension. An oblique anastomosis is less likely to stenose [2].

In case of vessels injury with subcompensated ore decompensated ischemia if gap is more than 2–3 cm:

Interposition graft (Reversed venous bypasses): Required if the vessel cannot be primarily repaired without strong tension, usually if the gap is more than 3 cm long [2].

Autogenous vein grafts is 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

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injury), contralateral lesser saphenous vein (LSV), ipsilateral LSV (if no deep venous injury)/ cephalic vein, and basilic vein [2].

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 [2].

***Venous repair*:** Repair of a vein is of special importance when this vein is the only venous drainage route, as in the popliteal vein. In this case options are similar to arterial repairs. The experience of recent war shows preference of venous repair over ligation [1].

Gunshot fractures of the extremities

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

Another kind of fracture occurs when a high-velocity bullet passes near the bone without direct contact. This has been called an "indirect gunshot fracture". Such injuries are caused by the high pressures 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. All open fractures are considered contaminated.

A closed fracture is one that is without damaging the skin, and usually can be considered clean if the skin is not devitalized.

Open fractures are classified according to their severity (table 8).

First degree open fractures have very small wounds and simple bone fractures. First degree open fractures needs following surgical debridement and can be treated as a closed fractures.

Second-degree open fractures have larger wound that communicates with the fracture and are more extensive than first degree open fractures. These fractures represent a middle ground between first degree and third degree fractures.

Third-degree open fractures present combination of the fracture and massive soft tissue damage. It is the most severe form of open fracture. Generally third degree open fractures in the combat are the result of high-velocity bullet wounds.

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Table 8 — Classification of open fracture by Gustillo-Anderson [9]

Gustillo 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 fractured 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 rotational flap)
IIIC	Type III fracture associated with an arterial injury requiring repair, irrespective of degree of soft-tissue injury.

Signs of bone fractures

Hard signs of fractures:

1. Crepitus (bony grating) may be heard or felt.
2. Unnatural movement at the site of the fracture.
3. Bone`s fragment in the wound.
4. Fat drops in the joint punctuate.
5. Loss of sound conductivity.
6. Pain under the pressure at long axis of bone.

Mild signs and symptoms of fractures:

1. Pain at or near the location of fracture.
2. Tenderness or discomfort on gentle pressure over the affected area.
3. Swelling about the fracture area.
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.

Medical care organization of injured limbs at medical care echelon: first aid (preventive measures of the shock, bleeding and infection)

General considerations of initial management.

Antibiotics, tetanus toxoid, analgesia and infusion therapy could be administered as preventive measures of the shock and infection development.

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Tourniquet could be used for temporary bleeding arrest and hemorrhagic shock prevention.

Intravenous antibiotics should be used as soon as possible at the evacuation chain. Use a broad spectrum cephalosporin (cefazolin etc.) — the two most harmful bacteria (Clostridia and streptococci) are covered by a first generation cephalosporin.

Irrigation and debridement should be used as soon as possible to prevent infection.

Neurovascular status of the extremity should be checked repeatedly.

Transportation casts ore external fixators should be used to immobilize a fracture along the evacuation chain. The cast must meet the dimensions of the standard litter. Internal fixation is contraindicated as initial management of bone fractures. 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 [1].

Principles of specific medical care: Surgical management of injured limbs

Wound incision/excision (debridement):

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

Irrigation of the wound;

All foreign material in the operative field must be removed.

Bone fragments should be leaved if they have a soft tissue attachment. Small detached bone fragments should be removed. Larger fragments that are necessary to the structural integrity of the bone should be retained.

Closure of wounds.

Primary closure limb gunshot wound is never indicated (except on palm). Loose approximation of tissues with retention sutures is appropriate to cover nerves, vessels, and tendons, but there must be a provision for free drainage. Skin grafts, local flaps, and relaxing incisions are contraindicated in the initial management.

Delayed primary closure should be accomplished in a stable environment.

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. After identification, goals of treatment are prevention of infection and preservation and restoration of normal joint function. Open-joint injuries always require surgery. Any time joint infection is suspected, the joint should be immediately explored or re-explored with subsequent synovium closening. Delayed primary closure can be done 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.

For examination - biplanar radiographs should be obtained.

Both ***fixatation casts (Plaster of Paris) and external fixators*** are equally acceptable methods for the initial management of nondisplaced 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 [1].

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 time prior to evacuation; load large casualty.

Operative management (Open reduction and internal fixation).

One of the principal is staged protocols, including provisional external fixation followed by a delay definitive internal fixation (days to weeks after initial treatment). This form of treatment used in any one of three scenarios: (1) a critically injured patient, (2) the presence of a wound with a significantly damaged soft-tissue, or (3) a severely contaminated open fracture [1].

In general, good indications for external fixator use is when the soft tissues need to be constantly evaluated, such as with a vascular injury, such as with a femur fracture and abdominal injury; or when the patients have extensive burns. 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 [1]. *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 [1].

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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) [1].

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. Primary temporary unilateral tubular stabilization followed by definitive circular Ilizarov/Taylor/hybrid fixation, is an effective method for the treatment of patients with severe damage to the limbs caused by high-energy war injuries [1].

Compartment Syndrome

Compartment syndrome may occur with an injury to any fascial compartment. If fascial defect caused by the injury may not be adequate to fully decompress the compartment, and compartment syndrome may still be present [2].

Mechanisms of injuries associated with compartment syndrome [2]:

- high-energy wounds;
- open fractures;
- closed fractures;
- penetrating wounds;
- crush injuries;
- circumferential dressings or casts;
- vascular injuries.

The diagnosis of a compartment syndrome is made on clinical pictures [2]:

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

Late clinical signss: **4 Ps (**Paresthesia; Pulselessness; Pallor; Paralysis) [2].

Prophylactic fasciotomy is recommended in the combat zone.

Indications for fasciotomy in the levels of medical care [2]:

4–6 hour delay after vessel injury.

Combined vein and artery injury.

Arterial or main venous ligation.

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Concomitant fracture/crush, severe soft-tissue injury, muscle edema or local necrosis;

Crush injuries.

Vascular repair.

Tense compartments.

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

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. Fat globules enter via venous sinuses in the bones and travel to the lungs and into the systemic circulation via pulmonary capillaries [1].

While usually asymptomatic, fat embolism (FE) may give rise to the fat embolism syndrome (FES), a critical and often fatal disease [1].

Two main mechanisms are considered in the pathogenesis of fat embolism syndrome: the "***mechanical***" and 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 fatty 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 [1].

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 [1].

The pathognomonic triad of symptoms includes [1]:

1. Petechial rash of conjunctiva.
2. Progressive hypoxemic respiratory failure.
3. Altered level of consciousness, seizures, focal deficits.

Up to one half 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, reduction of interstitial edema, inhibition of the inflammatory response, and inhibition of delayed platelet aggregation [1].

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XIII. Blast trauma

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. 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 [1].

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. 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 [1].

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 normal pressure determines the blast strength and its potential to produce primary blast injuries [1].

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

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previously unaffected areas. However, blast effects are magnified if the explosion is in an enclosed space and causes amplification of the wave as it's reflected off the enclosure walls [1].

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 [1]:

Direct compressive effects of the blast wave, in which denser tissue fragments pass 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 ***secondary*** 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 [1].

In ***tertiary*** mechanism, the blast wave torns the subject into another causing injury. Injuries such as soft crash or fractures are caused by tertiary mechanism when the victims are thrown to the ground or other objects [1].

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 [1].

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

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these blast injury mechanisms [1] (table 9).

Table 9 — Overview of Explosive-related Injuries [2]

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, aspiration 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 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 separate petechae to confluent hemorrhages. A blast lung should be suspected in 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 Prophylactic chest tube (thoracostomy) is recommended before general anesthesia or air transport is indicated if blast lung is suspected [2].

Abdominal Injury.

Gas-containing sections of the GI tract are 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

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rupture. Blast abdominal injury should be suspected in anyone exposed to an explosion with abdominal pain, nausea, vomiting, 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 [2].

Brain Injury.

Primary blast waves can cause concussions or mild traumatic brain injury 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 [2].

Blast injury diagnostics: Stages of examination

At first step, medical personnel should carefully examine the casualty for signs of contusion or penetrating wounds. Sometimes, a relatively minor injury 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 like respiratory damage or by blood loss from gastrointestinal hemorrhage require immediate stabilization in ICU.

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. Serial monitoring of hematological and biochemical parameters may be useful in following the complicated medical course of any seriously injured patient.

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 selfpoisonings [2].

Stages of traumatic disease development:

Stage I — Initial (early) stage (shock, I Phase of the disseminated intravascular coagulation (DIC), I phase of acute renal failure (ARF));

Stage II — Intermediate (rhabdomyolysis, II phase of ARF and DIC);

Stage III — Late (advanced or resulting) stage of crush syndrome (secondary immunodeficiency, septic disorder, III phase of ARF and DIC);

Stage IV — Recovery.

Systemic therapy of traumatic disease:

Correction of hypovolemia/dehydration — fast and aggressive fluid resuscitation to recover normovolemia;

Forced diuresis — maintenance of brisk urine flow of 1-2 mL kg·1h·L may reduce mioglobin cast formation and tubular obstruction;

Alkalinization of urine: acidic urine promotes myoglobin cast formation;

Correction of electrolyte abnormalities: hyperkalemia, hyperphosphatemia, hypocalcemia, hyperuricemia;

Renal replacement therapies: hemodialysis or hemofiltration are necessary to maintain balance in anuric patients [1].

Extremely contaminated wounds 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.

Management in blast injuries at medical care echelon

Some manifestations of blast injury will resolve spontaneously 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 the same time, severe manifestations of blast injury may have long-term sequelae.

Casualties who have extreme respiratory disturbance 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 insufficiency, 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

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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 every new generation of military surgeons at the beginning of all war. They are the key components in managing high energy injuries. The use of external fixators in military practice for open fracture stabilization has not been as successful as hoped. This has resulted in the use of plaster combined with other methods, even including the Thomas splint.

Battle casualties who sustain amputations have the most severe extremity injuries. In history, 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:

Guillotine amputations (one level amputation);

Flap amputations (multi-layer amputation):

Myoplastic technique (soft-tissue flaps);

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 combat setting should be performed as distally as possible, and revised later in stable conditions. This procedure was called a “Guillotine” amputation, because the muscle and bone were cut at the same level [1].

It was subsequently replaced with shortening of the bone and formation of the long flaps which close the wound and form a stump [1].

Reconstructive. Bone cutting is carried out in consideration of the soft-tissue coverage (multi-layer flap amputation), so after wound

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closuring, the skin is not under tension. It leads to final healing of the amputated limb with formation of the optimal prosthetic stump [1].

General Principles of Amputation Technique:

The skin flaps must be made with enough length to avoid closure under the tension.

The skin should not adhere to the underlying bone.

Avoid scar formation in the areas of the prosthesis contact [1].

The "Pirogoff amputation" (kind of ***osteomyoplastic amputation***) is a surgical for the complex injuries of the forefoot, where there is loss of the bones and soft tissues. Part of calcaneal bone together with the skin and fat pad is rotated and fused to the partially resected tibial plato, which allowed for a longer stump, eliminated the need for below-knee amputations, and allowed for effective weight bearing [1].

Final amputation and definitive treatment should occur in the stable environment of a base hospital, not in the combat zone medical facilities.

XIV. Burns

Burns constitute between 5% and 20% of combat casualties during conventional warfare. Even relatively small burns can be dangerous, and can block 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 present the vast majority of the other.

Classification of burns according to severity: 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 could be 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. Fat layer are under the dermis.

Classification of Burn Injuries

Simple anatomical classification of burn degree is used for the definition of burns:

First degree (injures to the epidermis) — reddening, swelling due to reactive edema.

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Second degree (epidermal detachment of the cutis) — development of blisters.

Third degree:

3A — partial destruction of the epithelium at the middle of papillary layer of dermis — remaining small pieces of epithelium, possible source of regeneration.

3B — total destruction of epithelium and papillary layer of dermis — no regeneration because of absence of small pieces of epithelium

Fourth degree (damage to far-reaching tissue formations, possible partial charring) — fat layer, muscles, and tendons are 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 scar formation.

A *"second degree"* burns involve the whole epidermis. This type of burn is erythematous with a blister formation. It heals in 7–10 days without scar formation. Superficial second-degree burns will heal in children under 10 years in 2 weeks; and in adults, healing takes 3 weeks. Patients with superficial burns less than 10% TBSA are usually treated as outpatients. Superficial first-degree and second-degree burns are quiet painful.

Partial-Thickness Burns

"3-A degree" burns. A superficial 3-A degree burns involve whole epidermis and superficial portions of the dermis. The skin is blistered with a moist and erythematous base. Superficial 3-A degree burns produce minor color changes with different range of scarring.

Full-thickness burns.

"3-B degree" burns. Burns can involve epidermis and all layers of the dermis with death of all germinal epidermal elements. It is usually dry and demonstrates an ivory or yellowish base. It appears like leather, totally anesthetic, cannot heal spontaneously, as all layers of the dermis are gone.

"4-degree" burns. "4-degree" burns involve deeper structures such as fat layer, tendon, muscle, bone etc. It appears like hard dark covering (rust), totally anesthetic, cannot heal spontaneously, often need amputation of the injured limbs.

Determining the burn size

The initial description of the burn includes an estimation of the percent of the total body surface area (TBSA). 1, 2 and 3-A degree

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burns are included in superficial burn size calculations. 3-B and 4 degree burns are included in burn size calculations as deep burns.

Three methods can be used to calculate burn size:

The "rule of palm". For evaluation of small or middle sized burns, the rule of the palm meanes that the patient's palm, including the fingers and the thumb, equals ~1% of the patient's TBSA.

The "rule of nines" [10]. 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% for right and 9% for left. The anterior trunk are 18–9% for chest and 9% for abdomen region. The posterior trunk are 18–9% for upper back and 9% for low back and buttocks. Ieach leg is 18–9% for hip, and 9% for leg and foot. The perineum comprises 1%.

Lund and Browder chart with ready templates.

Local and systemic manifestations of thermal burns

Minor superficial burns are painful but doesn`t lead to general problem if burned body surface area (BSA) is less than 10% in children and 20% in adults. If burned BSA is lager, it provokes burn disease development with drastic and extremely crucial clinical picture.

Circumferential burns of the chest may prevent effective chest motion and breathing. If that happens, immediate thoracic escharotomy is a life-saving procedure to permit adequate chest excursion and prevent breathing arrest.

Thermal inhalation injury is more common in patients with extensive open fire burns, especially facial burns and a history of injury in a closed space (e.g., building or vehicle).

Inhalation injury manifests by stridor, hoarseness, cough, carbonaceous sputum, dyspnea. 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. Patients who have symptomatic inhalation injury are prophylactically intubated.

Carbon monoxide (CO) poisoning causes cardiac and neurologic symptoms and requires moist 100% oxygen inhalation for at least 3 hours or until symptoms resolve.

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 agent, the more serious consequences could be. Also the basic functional state of the organism is very important.

General reactions occurring in burns are due to absorption and accumulation of tissue decomposition toxic products in the organism,

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which cause intoxication and ***thermal rhabdomyolisis*** (also known as ***burn disease***).

Prognosis in burn diseases depends on the size and depth of the burned BSA. In cases of vast and severe burns death occurs rapidly or within 2–3 days. Early death is due to the burn shock (depression and subsequent paralysis of the circulatory and respiratory centers). The clinical picture of a burn disease shows, in first a rise and then a drop in blood pressure, different range of respiratory disorders, hemoconcentration due to the passage of plasma through the capillaries into the injured tissue with relative increase in the erythrocyte rate, hemolysis, accumulation of toxic products of tissue decomposition, a rise in body temperature, and development of infection which has entrance into the wound. In severe cases the kidneys are affected, urination decrease and oliguria (sometimes anuria) develops.

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

Extensive burns cause disorders in general functions, which always lead to irreversible collapse of all vital functions.

Burn disease.

Burn disease is a constellation of clinical signs that result from superficial burns (degrees 2–3-A) 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 [8].

The four periods of the disease are identified as [8]:

1. Burn shock.
2. Acute burn toxemia.
3. Septicemia.
4. Recovery.

Burn (hypovolemic) shock.

Loss of blood plasma and hypovolemia are the major pathogenic mechanism of burn shock. Hypovolemia is the result of loss of liquid through injured capillaries and cell membranes, and evaporation from extensive wound areas. Hypovolemia (low blood volume) occurs, leading to a loss of circulating blood of up to 15–30% after only 1–2h. Hypovolemia leads to an increase of liquid accumulation in the burned tissue and, as a result — sequestration with a possibility of edema development in the liver, kidney, and brain (cerebral edema). This causes conditions of confusion and restlessness.

Hypovolemia results in an increase in blood viscosity and local accumulation of vasoactive substances (e.g. histamine, serotonin),

which impairs microcirculation. Tissue hypoxia causes an increase in lactates, leading to intracellular acidosis with imbalances in the microcirculation too. Disturbances in microcirculation and blood clotting can promote the formation of microclots that can cause consumption coagulopathy and pulmonary embolisms.

The formation of "shock lung" is a next difficult problem. Reabsorbtion of sequestered liquids and excessive amount of parenteral liquid affects the alveolar wall in edematous manner with difficulties in oxygen restoration, and next hypoxia development. Lung edema caused by efflux of secretions into the alveoli with suffocation of the patient.

Renal functions are reduced because filtration pressure is limited in the vessels owing to shock. If fluid intake is too low, renal functions will be damaged. The parallel contraction of the renal vessels makes the oxygen uptake insufficient and it leads to permanent tubulonecrosis (shock kidney).

With reduced renal function there is an increase in potassium against the sodium intracellular calcium (witch flow directly from the cells via cell substructures and exchange for extracellular sodium), and the sodium level in the serum is reduced. This electrolyte disbalance leads to disorders in the liquid exchange and cardiac function (arrithmia).

The manifestations of burn shock in depend of severity of circulatory defects may have duration for 2–72 hours.

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 rhabdomyolisis). Burn toxemia manifests by fever, marked tachycardia, anemia, hypo- and dysproteinemia, abnormal hepatic and renal functions [10].

Acute renal failure (ARF) in burn disease 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.

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 from the region affected by burns. Spasm of afferent glomerular arteriolae produces

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sudden decreasing of glomerular filtration. In 20–30% of the patients with burn disease anuria is absent.

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

Development of ARF in burn disease is a very unfavorable prognostic sign necessitating a complex evaluation and treatment. Achieving of high percentage of survival among patients with burn disease 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 extracorporal hemodialysis with early surgical management. Hemodialysis is the most effective therapeutic procedure in the treatment of ARF.

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 active evaporation and healing process (the regeneration process also necessitates additional energy and protein). The burn areas must therefore be covered as soon as possible. Only then it will be possible to reduce the supply of calories.

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. In this period, patients with deep and extensive burns show signs of general exhaustion, which in severe cases may present as weight loss, skin dryness and pallor, pronounced muscular atrophy, bed sores and contractures of joints. This period being characterized by proliferation of microbes and development of varied septic conditions (e.g. wound suppuration, pneumonia, and sepsis) [10].

Septicemia starts approximately on day 10 and is characterized by severe infection [8]. Facultative Staphylococcus, Pseudomonas and Proteus spp. and E. coli are common causative agents. This may occur as a result of disorders of the immunological system and are related to the second immunodeficiency development. As the production of immunoglobulin is also reduced because of exhaustion, high doses of it must be delivered. Almost three quarters of patients with extensive burns die of the consequences of a severe infection.

The genesis of burn disease-associated anemia is multifactorial as follow: hemorrhage, hemolysis and decreased etrythropoesis level.

Separation of the necrotic eschar starts on days 7–10. Complete skin regeneration is indicative of the end of the septicemic period [10].

All the patients who have extensive and deep burns develop encephalopathy with vegetative-trophic disturbances and neurologic changes in the hand and feet.

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Recovery.

Recovery is characterized by restoration of functions, which have been affected during the earlier three periods of burn disease. Functional organ changes (e.g. the heart, liver, kidneys) can persist for as long as 2–4 years after the trauma [10].

Burns can cause scars and keloids — ridged areas caused by an overgrowth of scar tissue. Scar tissue can cause and form contractures, when skin, muscles and tendons shorten and tighten, permanently limit movement or even pulling joints out of position. All that conditions need specific treatment and collaboration between reconstructive and orthopaedic surgery.

Light radiation lesions of a nuclear explosion

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 main hazards are flash burns and eye injuries. Flash blindness is caused by the initial light produced by the nuclear detonation (bright flash).

Thermal radiation can cause burn injuries either directly by absorption of the infrared heat radiation energy by the skin, or indirectly by heating or ignition of clothing, or tertiary as a result of fires started by the explosion and radiation.

After explosion part of thermal radiation that strikes an object will be reflected, part transmitted, and the rest part absorbed. The part of radiation that is absorbed depends on the color and nature of the material. A light colored clothes may reflect much of the incident radiation and thus, protect from damage.

A 1 megaton explosion can cause first-degree burns (like sunburn) at a distance of about 11 km, second-degree burns (producing blisters and superficial scars) at distances of about 9 km, and third-degree burns (which destroy skin tissue) at distances up to 7 miles. The greatest number of deaths in the first 2 month after explosion is result of thermal injury (60–70%).

Flash burns.

One of the most serious consequences of the nuclear explosion is the large number of flash burns caused by the thermal radiation from the fireball. A specific feature of the flash burns was their sharp limitation to exposed areas of the skin facing the center of the explosion. It is result of the absorption of radiant energy by the skin of exposed victims and thus they are sometimes called “profile burns”. Another features, which appeared after the healing of flash burns, was the formation of keloids.

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Burns under clothing.

Burns beneath clothing which depend on the color, thickness, and nature of the fabric can arise from heat transfer for some time after the thermal pulse ends. Skin burns under clothing can be produced by:

Direct transmition through the fabric if is thin and doesn`t work as an protective screen.

Heating the fabric and causing steam that affects the skin.

Fabric ignition from longer heat application; next hot vapors and flames will cause burns which are more like to conventional burns.

These burns generally involve deeper tissues than the flash burns.

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 are caused by the ionization in the cells composing living tissue. As a result of ionization, cell structures which are essential to the normal functioning, are altered or destroyed.

"Beta" burns result from fallout particles shallow ionizing beta radiation. The largest particles in local fallout would be likely to have very high radioactivity and because of this would be able to cause a localized burn. But, these decay particles have a short range of penetrating.

"Gamma" burns appears from highly penetrating gamma radiation and result in whole body irradiation. After whole body gamma irradiation more than 10 Gy, some of the victims have developed injuries to their skin between the time of irradiation and death.

Incendiary weapon (Incendiary stuff or fire-mixes)

Napalm was formulated for use in bombs and flamethrowers by mixing a aluminium soap of ***na***phthenic and ***palm***itic acids (hence napalm) with gasoline. This soap turns gasoline into sticky syrup that burns more slowly but at a higher temperature.

There are two kinds of napalms. 'Standart' one produces a temperature of 800–1,200°C (1,450–2,150°F) and 'Super-napalm', enriched with polystyrene, sodium, magnesium or phosphorus, with which the temperatures reaches 1,500–2,000°C (2,700–3,600°F).

When used as an incendiary weapon, napalm can cause severe burns to the skin and body, asphyxiation, unconsciousness, and death. If explode it can create an atmosphere of more than 20% carbon monoxide and firestorms of up to 110 km/h. Every people in this condition can be killed by hyperthermia, radiant heat stroke, dehydration, suffocation, or carbon monoxide poisoning.

One of the main features of napalm is that it sticks to human skin, with no method for removal of the burning substance. Because of

napalm's adhesive quality, the burns it causes in general are of the 3-A and 4 degree. Napalm burns which affecting as little as five per cent of the body surface leads to burn disease development.

The second effect of napalm bombs is great amount of carbon monoxide produced after the explosion. Carbon monoxide poisoning leads to ineffective breathing and collapse, and victim get burned to death.

The third effect of napalm burns is a breath of heat fumes which affect lungs.

Pirogel is combination of napalm and thermite.

Thermite is a pyrotechnic composition of a metal powder and a metal oxide (e.g. iron (III) oxide) that produces an exothermic oxidation-reduction reaction known as a thermite reaction. The reactants are stable at room temperature, but they burn with an extremely intense exothermic reaction when they ignite. The products are burn with temperatures reaching up to 2,500°C (4500°F). Burns caused by thermite reaction are almost always of the 4 degree.

White Phosphorus.

Some incendiary weapons used in warfare contain white phosphorus. This element ignites on contact with air in every temperature. Most of the burns resulting from white phosphorus is due to the ignition of clothing, and is treated as a conventional burn. Fragments of this elements may be driven into the soft tissues provoke cell poisoning. Phosphorus in a wound will ignite as soon as the tissue dries out and it is very difficult to remove this stuff. Medical teams as well as patients are in danger. UV light can be helpful to locate phosphorus particle.

First aid treatment of casualties with white phosphorus burns includes intensive water irrigation, and wound dressing that must be kept continuously wet.

When surgical treatment is available, the wet wound can be irrigated with highly diluted solution of 1% copper sulphate (a palest blue color solution). This solution inactivates phosphorus with forming black copper sulphide, which should then be removed with forceps. After particals removing, the copper sulphate solution should be washed away, and the wound excised and dressed.

As general effects of white phosphorus injury has been described profound hypocalcemia and hyperphosphatemia. It should be treated with intravenous calcium.

Triage and treatment at medical care echelon

General therapy.

Pre-Hospital and Emergency Room Care for Burn Patients.

Should be started with fluid resuscitation.

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Calcium chloride administration to reverse potassium effects in cellular membrane (with 10 ml of 10% solution intravenously).

To transfer extracellular potassium into cells 10% Glucose solution (250–500 ml with 5-10 units of insulin) with sodium bicarbonate solution (50 mEq) should be used.

Prevention of hyperventilation to avoid respiratory alkalosis should be done.

Using of permanent bladder catheter to control urine output is necessary.

Hospital Care.

Specific shock therapy is always the basis of all further infusion therapy. Infusion administered through the central venous catheter. Fluid resuscitation should be started as soon as possible in the first 24h post-burn.

The quantities of infusion are calculated every day using the following formula for the first 24 hours. Estimate crystalloid and colloid solution needs:

Total Volume = (2 mL) • (% deep burn) • (kg weight). 50% for colloids and 50% for crystalloids.

Half of this total volume is programmed for the first 8 hours post-burn, and half for the second 16 hours post-burn. Burns less than 20% TBSA do not require colloid infusion. Fresh frozen plasma and synthetic colloid solutions can be used if albumin is not available. Infusion therapy is continued until the 48th hour post-burn with monitoring of urine output. Regular monitoring is necessary with regular laboratory tests of electrolytes and blood glucose, clotting time, renal and hepatic disfunction.

Adjuvant to resuscitation:

Low-dose dopamine.

Vasodilators.

Diuretics.

High doses of corticosteroids can be administered, as they are a useful prophylaxis against pharyngeal, pulmonary, and even cerebral edema.

Hemodialysis (continuous renal replacement therapy — CRRT).

Once the diagnosis of acute tubular necrosis has been made, it is indication to begin an immediate therapy with hemodialysis and hemofiltration.

The basic principle of CRRT is desintoxication (with the elimination of products of tissue decomposition, inflammatory mediators, urea, creatinine and uremic toxins) and normalisation of water and electrolytes balance.

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Indication for Hemodialysis and Hemofiltration are:
Oliguric renal failure;
Massive myoglobinuria (e.g. in electric burns).

Because of metabolic exhaustion development ***clinical nutrition*** should be administered.

The use of glucose only is not advisable in the presence of respiratory failure and in the case of patients in mechanical ventilation because it increase consumption of O_2 and production of CO_2.

In opposite the combined glucose-lipids system has many advantages because it reduces production of CO_2 and consumption of O_2. In same time it reduces frequency of hyperglycemia and hepatic steatosis because it is the supply of the essential fatty acids.

Local care.

In general, try to restore coverage and movement in dorsal injuries within 14 days. Palmar surface can tolerate longer periods of healing, but still attempt to have coverage and movement within 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 3Cs:
Cleaning.
Cooling.
Creaming (oil and fat should be avoid).

Second degree burning wound care includes:
Debridement of loose skin and pop blisters;;
Cleanse with mild anti-bacterial soap;
Application of an anti-bacterial cream or ointment twice daily (1% silver sulfadiazine burn creams should be used);
Large and middle sized wounds should be keep moist until they heal;
Small, superficial burns can be left dry after cleaning with antiseptics;
Systemic antibiotics are not indicated in absence of infection.

Following burn cream application and other, 2 degree 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. F.e.: hand burns must be kept moist but allowed to move. In this way loose-fitting surgical glove filled with antibiotic cream are indicated to use.

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Half-closed (wet-dried gauze). Burns dressed with fine-mesh gauze are treated open, with a heat lamp applied until the gauze is dry. Extremities are immobilized after dressing 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: safe, soft, effective, leave wound moist, easy to change, give good protection.

Local Care of Deep Dermal and Full-Thickness Burns

Escharotomy

It is used to improve perfusion by releasing restrictive burn eschar but it`s need to avoid tendons exposing to prevent further scar contracture.

Full-Thickness Burns should be managed by early excision and grafting. This is best performed in the first 5 days, before infection development under eschar. If this period is missed, more than 3 weeks should usually wait to surgery.

Skin Grafting

When the wound bed has good granulation tissue and no white underlying structure is exposed, skin grafts can be used for coverage and definitive closure of the wound. It can be taken using a sharp excision knife Weck-Goulian, Humby, Braithwaite. 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. Quiet good contact between the graft and the recipient bed is important for a successful take. The most common donor areas for STSG are the thighs and buttocks. These skin grafts are usually taken with a mechanical dermatome. It should be thinner in babies and older patients than in adults [1].

Donor sites dressed with fine-mesh gauze are treated open, with a heat lamp applied until the gauze is dry with immobilization for 4–5 days. Grafted sites are inspected 4–5 days after surgery and they should be inspected sooner in case of fever, pus, or other evidence of infection.

Physical and occupational therapy are begun as soon as graft take is sufficiently hard, usually 5–7 days after surgery. Extremities are splinted in the functional position at night. The dried gauze on the donor site is allowed to separate spontaneously. After time the donor site can be reused as necessary for further grafting. The patient is fitted for compression with elastic bandages when all wounds are closed and healed.

Full-Thickness Grafts

4th degree deep burns with exposure of bone or tendons post-debridement may require a flap. Usually it is used to delayed reconstruction. Hundreds of flaps are described for dealing with soft tissue lost from the extremities. There are no different general

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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. In general, pedicled flaps are more often used than free flaps for closing war wounds [1]. The most commonly used flaps are presented here:

Local Flaps. These can be axial or island and are usually used to cover small — to medium-sized defects. When a larger wound is present, these flaps have limited applicability because their blood supply may be compromised. The 1:1 to 2:1 proportions should be considered for local flaps in the limbs [1].

Pedicle Flaps. They are classified into fasciocutaneous, adipofascial, fascial, or neurovascular according to anatomy and distant pedicle and cross limb flaps according to surgical technic. These flaps have good applicability without microsurgical anastomosis formation. Medium to large size wounds can be covered in this way when the zone of injury is not extensive [1].

Fascial and Fasciocutaneous Regional Flaps are used more for upper extremity reconstruction or for areas where a thin tissue is required for coverage. Recently, it has been proved that fascial, adipofascial and fascioculaneous flaps are quiet successful for fractured bone coverage [1].

Muscle and Myocutaneous Pedicle Flaps have the advantage of filling the deep soft tissue loss to avoid dead space and there is no need for microsurgical anastomosis. The muscle and myocutaneous pedicle flaps are the most used flaps for calf wound reconstruction, for open fractured bone coverage and for dealing with osteomyelitis. The gastrocnemius and soleus muscle flaps are the most used for lower limb reconstruction [1].

Cross-Limb Flaps. Cross-limb flat is harvested from the contralateral limb and covers the wound on the other one. Connection is kept for a period of 3 weeks, what is enough for establishing the blood supply to the flap from the periphery and the wound bed. Next disconnection of the pedicle may be done. There are two main disadvantage of this method: continuous immobilization of the limbs for about 3 weeks to protect the flap pedicle and a skin graft for closure of the donor area. Advantage is that they have high rate of success when no other reconstructive method is available [1].

Distant Pedicle Flaps (axial flaps). These flaps have same disadvantages as cross-limb flaps. The reconstruction is done in stages. First the flap is harvested, fixed to cover the distant wound, and next kept connected for 2–3 weeks. In the second stage, the flap is detached with final closer of the wound. Periodic external compression for flap ischemia will short the period of limb immobilization [1]. The upper limb with fixation to the thorax and the abdomen is more favorable for these types

of flaps. The pedicled groin flap (lap or lower epigastric abdominal) is often the best choice to cover soft-tissue injuries to the hand and the distal forearms. Arm injuries can be closed by lateral thoracic flaps [1].

Flaps based on the Filatov-Crane principle can also be used, but it have low rate of success. The reconstruction is done in stages whith adiposo-skin "handle-flap" formation on the first step. After 3 weeks of this one of the flap paddle will be cut and fixed to the wound, thus leaving the donor skin paddles in place. The granulating tissue of the wound is subsequently covered by skin graft after the fixed flap paddle will be taken.

Free Flaps. Free flaps may be necessary, particularly in large 4 degree and electrical burns, when a local or regional flap would not be sufficient for local or regional flap coverage of exposed white structures, such as bones, tendons, nerves, and major blood vessels. Vascular anastomosis is necessary to get successful result. Functional reconstructions with sensate flaps are the most used for the plantar or palmar area injury [1].

Post-Operative Care.

In post-op period limb should be immobilized until graft has good primary take (at least for 5–10 days). Small areas of graft loss could be treated conservatively. Compressive garments should be used for a number of months after flaps taking.

After intensive medical treatment, the patients need further wound care, physiotherapy, kinetic therapy and psychological support for successful therapeutic treatment. Very resource intensive definitive burn care, including surgery and rehabilitation is not recommended in the combat zone.

To prevent scar contractures splinting with careful motion must be continued. If scar contractures and functional restriction will be recognize during follow-up care they may need a new operation with next intensive physical training.

After burns healing patients need a long-time period for complete rehabilitation combined with plastic surgery for aesthetically injured patients. All this will allow them return to associate with other people.

XV. Frostbite

Cold injury: frostbite and hypothermia

Cold injury 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

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loss or shock, nutrition, tobacco use, physical activity, drugs and medication, alcohol, duration and exposure, dehydration, environment (temperature, humidity, precipitation, and wind), and clothing [2].

Cold injury could be divided into:

A. Sharp local cold injury — ***Frostbite***:

1. Contact Frostbite;
2. Frostbite in Dry cold below 0 °C.
3. Frostbite in Wet cold above 0 °C.

B. Chronic local cold injury — ***Immersion-trench injury***:

1. Immersion Injury.
2. Trench Foot.

C. Sharp general cold injury — ***Hypothermia***.

D. Chronic general cold injury — ***Perniosis*** (cold neuropathy, dermatitis and vasculitis).

Preventing frostbite

Frostbite and immersion injury development depend on many factors (wind chill, altitude, duration, wetness, clothing, water immersion and prior exposure) and it is better to avoid them.

Frostbite classification, clinical features and diagnostics

Frostbite Associated Clinical Features.

Frostbite is true tissue freezing resulting from heat loss caused ice crystal formation in superficial or deep tissue (***Primary crionecrosis***). It is appiare 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. Shorter duration of tissue freezing at or near 0°C results in the same injury with microvascular vasospasm and tissue ischemia *(**Secondary crionecrosis***) as the second etiology of frostbite [2].

Frostbite may affect the extremities, nose, ears, scrotum and penis. Fingers, toes, and ears most commonly involved. Severity of symptoms is usually proportional to the severity of the injury.

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 [1].

Superficial frostbite involves only the skin with swelling, mild pain, and minor joint stiffness [1].

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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.

In ***Deep frostbite*** tissues are 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.

Blisters form and are clear, fluid-filled, or hemorrhagic (the latter indicates a more severe, deeper injury). Failure to form vesicles in an obviously deep-frozen extremity is a grave sign [1].

Frostbite period:

1. Hidden period (Pre-reactive) — before rewarming.

A sensation of numbness with accompanying sensory loss is the most common initial complaint. Frozen tissue may appear mottled blue, violaceous, yellowish-white, or waxy.

2. Reactive period — after rewarming:

Early reactive period — first 5 days after rewarming. 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 3h of rewarming; lack of edema is an unfavorable sign. Vesicles and bullae appear in 6 to 24 h after rewarming.

Late reactive period — after 5 days after rewarming (Phase of Demarcation). Dry necrosis occurs distally, but more proximal tissues aren`t compromised. Sharp line of demarcation of dead from viable tissue is presented.

Frostbite treatment at medical care echelon

Field treatment (first aid).

Patient should be protected from further cold exposure and next trauma.

Massage, rub with snow warm by an open fire or high-heat source are prohibited in threats of burns development.

Superficial (blanched cheeks, nose, ears, fingertips) frostbite.

It is better to warm with palm of hand.

Ointments may help prevent skin from drying or cracking.

Deep frostbite.

The body part and the patient as a whole should be protected from further cooling and traumas.

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Dry, sterile bandage should be applied and involved extremity elevated.

Protection from refreezing during evacuation should be organized.

Patient should be evacuate promptly to specialized medical care as soon as possible.

Hospital care.

Slow passive rewarming of frozen region should be organized. Limbs should be elevated. Immersion of frozen limbs in warm or hot water is unwanted.

Rehydratation with infusion therapy should be obtained.

Pain medication should be given with amitriptyline 50–150 mg at bedtime, because other analgesics are completely ineffective and do not relieve pain (even opioids).

Tetanus prophylaxis are indicated with any contaminated wound or nonviable tissues.

Systemic antibiotics should be given when there is evidence of infection with any contaminated wound, or when there are nonviable tissues.

Nonsteroid anti-inflammatory drugs (such as ibuprofen) should be given as systemic prostaglandin inhibitors.

Surgical management initially is conservative.

If vesicles develop they should be unroofed with antiseptics cleaning and sterile gauze application. In 2 degree frostbite they will heal in 7−10 days without consequence.

Early surgery (debridement of necrotic tissue) is indicated only in the most severe cases, where massive tissue destruction has taken place with severe infection development like in trench foot.

The best management of nonviable digits is amputation.

At least reconstructive surgery should be delayed for few months ("Freeze in January, operate in July") [2].

Rehabilitation

Recovery takes a lot of time and requires evacuation because deep frostbite may consequence in months of painful disability. Long-term consequences are very common and include high sensitivity to the cold (secondary Raynaud's phenomenon, perniosis), hyperhidrosis, peripheral neuropathy (paresthesias, loss of proprioception), chronic pain, and limping. Careful and consistent treatment of all of this complication could be effective.

Non-Freezing Cold Injury

Immersion Injury/ Trench foot.

Trench foot occurs from prolonged exposure to any cold, wet conditions at temperatures cooler than body temperature (as high as

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17 °C for more than 12 hours). Sometimes it results from intermittent exposure to temperatures above freezing, usually accompanied by high humidity and moisture; 1 to 6 hours of exposure each other. Shorter duration at or near O°C results in the same injury (occurs in nonfreezing temperatures 0 °–12 °C) [2]. Microvascular vasospasm with tissue ischemia ***(secondary crionecrosis)*** is the apparent etiology of trench foot and immersion injury. Nerve, muscle, and endothelial cells are the most affected by this long-term cooling.

Immersion injury is also known as shelter foot, sea boot foot, and foxhole foot. Immersion injury is a peripheral nonfreezing cold injury resulting from prolonged exposure to water (prolonged immersion of feet) usually at temperatures just above freezing.

It is different from tropical immersion foot or warm-water immersion foot as seen in the Vietnam War. Tropical immersion foot typically occurs after 3 to 7 days of exposure to water at 22 to 32 °C. The clinical picture are characterized by burning in the feet, pain on walking, edema, erythema and hyperhydration of the skin, which resolved completely after rest and removal from the wet environment.

Associated Clinical Features of the Trench Foot and Immersion Injury.

First symptoms are mildly painful, cold and numb feet or “walking on wood”. Mild joint stiffness may occur acutely. Next may appear swollen, with the mildly blue, red, or black skin (especially the fingers), foot are hot and often hyperhidrotic. After rewarming, pain becomes stronger and does not respond to pain medication, including opioids. Liquefaction necrosis occurs distally and may require many days of exposure as time progresses. Proximal tissue may also be compromised. There is no sharp line of demarcation of dead from viable tissue [2].

Post-injury sequelae include pain, numbness, loss of proprioception, and cold feet. Hyperhidrosis with subsequent paronychial fungal infections are common [2].

Perniosis, also known as chilblain cold sores, is the result of nonfreezing repeated cold exposure. Perniosis is development within 24 h of cold exposure with previous cold injury history. In general it frequently appears on the face, ears, hands, pretibial areas, and feet. A large range of lesions like localized erythema, cyanosis, edema, plaques, blue nodules and more severe lesions including vesicles, bullae, and ulcerations may be seen. Following rewarming the lesions persist for up to 2 weeks and may become more chronic in association with burning paresthesias, hyperhidrosis and hyperkeratosis as result of peripheral neuropathy, vasculitis and dermatitis.

XVI. Hypothermia

Hypothermia is defined as whole-body cooling below 35°C because the cold liver and tissue cannot metabolize lactate and then produce the warm. Degree of hypothermia is classified according to the body's core temperature and the clinical effects appeared subsequently in a different temperature range [2].

Causative factors and prevention

Water immersion.

Rain and wind.

Prolonged exposure to severe weather without adequate clothing, which insulation effect is decreased with wetness, what increases the conductive heat loss [2].

To prevent hypothermia one should stay dry and avoid windy exposure.

Classification, clinical features and diagnostics

1. ***Mild hypothermia*** from 35 °C to 33 °C (93–91 °F) — ***adinamic stage.*** Shivering, hyperreflexia, dysarthria, ataxia, poor judgment and apathy are present.

2. ***Moderate hypothermia*** from 33 °C to 28 °C (91–82 °F) — ***stupor stage.*** Stupor, loss of shivering, fibrillation and other arrhythmias, progressive decrease in level of consciousness, respiration, and pupillary reaction, eventual pupil dilation are present [2].

N.B. Standard hospital thermometers, (even digital) can`t measure temperatures below 34 ℃ (93 °F).

3. ***Severe hypothermia*** from 28 °C to 23 °C (82–72 °F) — ***convulsive (agony) stage.*** Ventricular fibrillation, which occurs spontaneously, loss of motion and reflexes (areflexic at 23 °C (72 °F), spontaneous convulsion, marked hypotension and bradycardia are present [2].

4. ***Profound hypothermia*** less than 20 °C (less than 68 °F) — ***clinical death.*** Asystole is present.

NB! The patient is not dead until he or she is warm.

Treatment at medical care echelon

Prehospital (field) treatment.

Awake patients.

Wet clothing should be removed; patient should be dry and insulate.

Warm sugar solutions (e.g. sweet tea) are given orally for rewarming and rehydratation.

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If patient has other injury it is better to transport them to next echelon.

Walking may deepen hypothermia, so it is better to limit unnecessary excessive movement to prevent the return of peripheral colder blood to the core [2].

Comatose patients.

Patient should remain horizontal position and be handled gently to avoid inducing arrhythmias [2].

Wet clothing should be removed; patient should be dry and insulate.

Patient should be wraped in multiple layers of insulation with creation of outer vapor barrier.

Massage is prohibited.

Warmed to 40–42°C intravenous fluids is used, if it is possible. The fluid of choice are 5% glucose or dextrose solutions (don`t use lactated Ringer's solution because the cold liver cannot metabolize lactate) [2].

NB! Continue resuscitation, if possible, until patient has been rewarmed [2].

Hospital care:

Treatment of mild stable hypothermia.

Mild stable hypothermia treated with warming (insulation, heat lamps, warmed intravenous fluids, warmed forced air, hair dryers) and vital function (breathing, cardiac function, urine output etc) monitoring and correction. Fast rewarming should be avoid to prevent collapse development.

Treatment of severe hypothermia with hemodynamic instability.

Cardiopulmonary bypass with rewarming, is the best solution because it provides core rewarming while ensuring circulatory stability [2].

Rewarming the periphery of the body rather than the core causes an inrush of cold acidotic blood from constricted peripheral vessels into the core, decrease the core temperature and provoke cardiac instability. Core rewarming needs external warm blankets application, heated and humidified oxygenation [2].

Ventilation;

Warmed intravenous fluids infusion;

Warm blankets application;

For ventricular fibrillation: core should rewarm to 32 °C (90 °F) with cardioversion;

Next rewarming should be continued with repeated defibrillation after every 1°C rise in temperature (if it is need);

Cardiopulmonary resuscitation (CPR) for asystolic patient: if cardiac monitor shows any electrical complexes, apical and carotid pulses should be check before initiating CPR. If any pulse (even thread) is present should do not initiate cardiopulmonary resuscitation.

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Lowest known accidental hypothermia with effective resuscitation of adult is 13.7 °C (56 °F).

PS.

Despite the changes in the conditions of practice, a military surgeon is far more likely to be deployed today than at any other time in the history since World War II. The era of global terrorism and asymmetric warfare has continued with the next wars and other terrorist events. These types of incidents blur the traditional lines between civilian and military trauma victims. In addition, natural disasters 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 poor environments is greatly important to share these valuable lessons with civilian colleagues. The physicians must know what to expect, and how to prepare the team in an austere and rapidly changing tactical environment with available and necessary equipment.

May the force be with you!

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THE COURSE OF THE CLASS

Introduction. Teacher's opening statement.
Basic level tests.
Basic level correction.
Tasks for class work.
Students` independent class-work.
Assessing the final level of topic knowledge.
Knowledge correction (solving situational cases, answering test questions).
Correction of the final level knowledge.
Summing up the lesson.
The assignment for the next class.

QUESTIONS FOR SELF-CONTROL OF KNOWLEDGE

QUESTIONS TO THE BASIC LEVEL OF KNOWLEDGE

1. Organization of surgical care in army and in emergency situations.

1. Name the distinctive features of military field surgery from other surgical disciplines.
2. Name the main periods of the military field surgery development and their features.
3. Name the first surgeon who used anesthesia to provide surgical care to the wounded in the war.
4. Name the features of modern military field surgery.
5. Principles of medical sorting.
6. Principles of medical care on the battlefield and evacuation levels.
7. What can be the source of radioactive radiation and pollution, chemical pollution in domestic, in industry, in the surrounding environment?

2. Classes: Gunshot wound and principles of its treatment on medical care levels.

1. Name the modern types of modern weapons.

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2. Describe the mechanism of action of the wounding projectile.

3. Describe the features of the harmful action of modern firearms, mine-explosives and other weapons.

4. Give a general classification of gunshot wounds.

5. What is wound ballistics; wound canal deviation, temporary pulsating cavity?

6. Name the zones of the gunshot wound.

7. What is the essence of the wounding projectile direct and side strikes theory?

8. What is the main difference between the entrance and exit holes of the gunshot wound?

9. What is the main difference between the wounds caused by low-speed and high-speed bullets?

10. What are the characteristic features of mine-explosive wounds?

11. Name the indications and contraindications for the wounds debridement.

12. What is the difference between microbial contamination of the wound and wound infection?

3. Classes: Infection complications in gunshot wounds. Tetanus.

1. Give the definition of a surgical infection in a gunshot wound.

2. Name of the main factors contributing to the development of wound infection.

3. What is the difference between microbial wound contamination and wound infection?

4. Describe the pathogenesis of the wound infection process.

5. What are the clinical criteria for diagnosing different phases of the wound infection process?

6. Give the classification of a nonspecific wound infection.

7. Identify different forms of nonspecific wound infection.

8. Give clinical characteristics of toxic-resorbative fever.

9. What is the sepsis? Give the definition.

10. What do the toxic-resorbative fever and sepsis have in common? What is their difference?

11. Name the methods for preventing wound infection during the stages of medical evacuation.

12. Set out the principles of local and general treatment for nonspecific wound infection.

13. Give the microbiological characteristics of gas gangrene anaerobic infection, tetanus, etc.

14. What factors of trauma and human body condition contribute to the development of infectious complications in wounds?

15. Name the principles of modern therapy for infectious wound complications.

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16. Name groups of drugs used for specific immunoprophylaxis of wound anaerobic infections.

17. What are the main differences between active and passive immunization?

18. List common contraindications for specific immunoprophylaxis in injuries and wound infections.

4. Classes: Bleeding, blood loss.

1. Name the types of bleeding depending on the cause.

2. Name the types of bleeding depending on the timing of the occurrence.

3. Name the main clinical symptoms of bleeding and acute blood loss.

4. Give clinical characteristics of blood loss severity and its corresponding indicators.

5. What are the ways to determine the extent of blood loss?

6. List the ways of temporary bleeding arrest.

7. Name the rules of tourniquet applying.

8. What are the ways for final bleeding arrest?

9. What indicates to inflammation in laboratory examination of peripheral blood?

10. What indicates to intoxication in laboratory examination of peripheral blood?

11. What indicates to anemia in laboratory examination of peripheral blood?

12. What indicates to haemoconcentration in laboratory examination of peripheral blood?

13. What are the signs of a circulatory disorder in the limbs in the first 4 hours after the injury and after 6 to 8 hours?

5. Classes: Traumatic shock. Crush syndrome.

1. Give the definition of shock.

2. What are the known causes of shock conditions?

3. Explain the basic theories of the traumatic shock pathogenesis.

4. Name the shock phases.

5. Name the classification of shock.

6. Give the characteristics of terminal states.

7. What signs for traumatic shock diagnosis in the field?

8. List the stages of acute renal failure and the possible causes of this condition.

6. Classes: Injuries of the head. Injuries of the spine

1. Frequency of closed injury to the skull and brain, their classification.

2. Clinical manifestations of concussion, contusion and compression of the brain.

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3. Skull fractures.

4. Gunshot wounds to the skull and brain, their classification and clinical features.

5. First medical and pre-medical care for skull injuries.

6. Medical sorting and the range of medical, qualified surgical and specialized care during the evacuation phases in the treatment of the skull and brain injuries.

7. Name the bones that form the facial and brain skull.

8. List features of cervical vertebrae anatomy, typical mechanisms of cervical spine injuries.

9. Define the concepts of “paresis” and “plegia”. Name and explain the possible causes of their occurrence.

10. What disorders of internal organs should be expected during the anatomical rupture of the spinal cord at the level of the lower cervical vertebrae? What is the prognosis for this damage?

11. Symptoms of concussion, contusion and compression of the spinal cord.

12. Gunshot wounds to the spine and spinal cord. Clinical periods.

13. First medical and pre-medical care for spinal and spinal cord injuries.

14. Medical sorting and the amount of medical, qualified surgical and specialized care for spine and spinal cord injuries.

15. The meaning of non-transportability.

16. Transport immobilization of skull and spine injured.

7. Classes: Gunshot wounds and closed chest injuries

1. Classification of chest injuries.

2. Clinical manifestations of various types of chest injuries, their diagnostics.

3. The most characteristic types and pathophysiological features of early complications of chest injuries (haemotorax, pneumothorax (closed, open, valve), subcutaneous and mediastinal emphysema).

4. Multiple rib fractures with the formation of a rib valve.

5. The pathogenesis of paradoxical breathing.

6. Late complications of gunshot wounds to the chest. Поздние осложнения огнестрельных ранений груди.

7. Name auscultative and percussion signs of accumulation of fluid, air in the pleural cavity.

8. Name X-ray signs of fluid and air accumulation in the pleural cavity.

9. In what position should a patient with a chest injury be X-rayed (vertically or horizontally), and why?

10. What will be the volume of blood loss in total one-sided hemotorax, with blood accumulation in the sinus or at the level of hemotorax up to fifth rib?

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8. Classes: Gunshot wounds and closed injuries of limbs and joints. Blast trauma.

1. What symptoms of closed fractures and bone dislocations do you know?
2. Frequency and classification of gunshot bone fractures.
3. Clinical features and diagnostics of gunshot fractures.
4. Name X-ray signs of gunshot fractures.
5. How will the wounding shells (shards, fractions, bullets, etc.) look like on the X-ray?
6. Clinical features and diagnostics of closed and open fractures.
7. Common and local clinical manifestations of joint damage.
8. Describe a clinical picture (symptoms) of penetrating and non-penetrating gunshot wounds of joints?
9. Gunshot wounds to the joints of the limbs, their classification.
10. Clinical picture and diagnostics of gunshot wounds of large blood vessels.
11. What are the clinical signs of limb ischemia.
12. Techniques and methods of bleeding arrest.
13. Symptoms and diagnostics of nerve damage.
14. Explain the symptoms of damage: radial, elbow, middle, femoral, fibula and tibia nerves.
15. Immobilization in nerve damage.
16. What are the characteristics of mine-explosive wounds?
17. Name the signs of tissues "viability" and "nonviability" (skin, muscles, blood vessels and nerves).
18. Transport immobilization in limb injuries.

9. Classes: Thermal injuries.

1. Which histological layers of human skin do you know?
2. What are the mechanisms of demage to physical factors (heat, cold, acids, alkaline, electrocution, ultraviolet and light radiation) on human tissue?
3. Name the basic safety rules when dealing with chemical reagents to prevent chemical lesions of the skin and respiratory tract.
4. What are the causes of burns in wartime and peacetime?
5. What are the burns in the depth of tissue damage and how to diagnose the degree of burn?
6. What are the ways to determine the area of the burn?
7. Name periods of burn disease.
8. Explain the main mechanisms of burn disease pathogenesis.
9. List the stages of acute renal failure and the possible causes of this condition.
10. What factors may be aggravating for patients with burn injuries?

РЕПОЗИТОРИЙ ГГМУ

QUESTIONS TO THE FINAL LEVEL OF KNOWLEDGE

1. Classes: Organization of surgical care in army and in emergency situations.

1. Set out the organizational structure and objectives of the evacuation levels.
2. Name the basic kits of medical equipment.
3. How is the sorting and evacuation unit organized?
4. What should be understood by the term "medical sorting"?
5. What is the purpose of medical sorting?
6. Name the types of medical sorting.
7. Name the groups of the wounded during medical sorting.

2. Classes: Gunshot wound and principles of its treatment on medical care levels.

1. Determine the repeated and secondary surgical treatment of wounds.
2. Give the name of the surgery, which is performed within 1 day of the injury; 2 days; after 48 hours.
3. What stages of wound debridement do you know?
4. Define primary, delayed primary and secondary (early and late) sutures.
5. List the conditions for the primary suturing of the wound.
6. What are the conditions and time for the secondary stitches applying?
7. Name the mistakes in the wound debridement performing.

3. Classes: Infection complications in gunshot wounds. Tetanus.

1. Give the definition to the repeated and secondary debridement of the wounds..
2. Describe the pathogens and clinical manifestations of decay infection.
3. What microbes cause anaerobic infection? Give their description.
4. Name the factors that contribute to the development of anaerobic infection.
5. Explain the clinical classification of anaerobic infection.
6. List early clinical symptoms of anaerobic infection..
7. What is the specific prevention and treatment of anaerobic infection?
8. What are the organizational and surgical principles for treating anaerobic infection?
9. Explain the pathogenesis of tetanus.
10. Name the early symptoms of tetanus.
11. Give the clinical classification of tetanus.

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12. How is emergency and planned tetanus prevention carried out?

13. Set out the principles of tetanus treatment.

14. List the anatomical pathways of the pus spreading in complications of upper and lower limb trauma.

4. Classes: Bleeding, blood loss.

1. Name the types of transfusion solutions and indications for their use.

2. List medical care for bleeding during the stages of medical evacuation.

3. Describe the basic rules for storing and transporting blood and blood substitutes.

4. Name the indications for blood transfusion and blood substitutes.

5. What tests should be done before blood transfusion?

6. How are the blood types determined?

7. Name the technique of performing samples for individual and rhesus compatibility.

8. How is the biological test performed?

9. Describe possible reactions during blood transfusions.

10. What complications after blood transfusion do you know?

11. What is the prevention and treatment of haemotransfusion shock?

5. Classes: Traumatic shock. Crush syndrome.

1. Define the term "shock" and characterize it as a universal reaction of the body in terms of pathogenetic mechanisms of development.

2. What bones fractures may lead to hypovolemic shock development?

3. Name the possible amount of blood loss in hip fracture, pelvic fractures, etc.

4. Is blood pressure an objective indicator when diagnosing shock and its degree?

5. Explain the basic principles of prevention and treatment of traumatic shock during the stages of medical evacuation.

6. Describe the anti-shock therapy at the stage of medical and qualified surgical care.

7. Explain the pathogenesis of the MMCS (crush-syndrome).

8. Describe the periods of MMCS (crush-syndrome).

9. Give MMCS (crush-syndrome) classification based on the range of damage and duration of limb tissue compression.

10. What factors can provoke complication in patients with combined injuries?

11. What are the signs of a circulatory disorder in the limbs in the first four hours after the injury and six to eight hours later?

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12. Describe the degree of limb ischemia in MMCS (crush-syndrome).

13. List the indications for MMCS (crush-syndrome) surgical treatment.

14. Explain the principles of pathogenetic MMCS (crush-syndrome) therapy at the stage of qualified surgical and specialized care.

6. Classes: Injuries of the head. Injuries of the spine

1. Give the classification of closed skull and brain injuries.

2. Name the main symptoms of closed brain injury.

3. What are the causes of general and focal symptoms in the closed brain injury?

4. Give the clinical characteristics of concussion, contusion and compression of the brain.

5. What types of damage are mild, moderate and severe in closed brain injury?

6. Give the classification of the skull and brain open (gunshot) wounds.

9. Range of medical care for damage to the skull and brain during the stages of medical evacuation.

10. What are the indications for urgent surgical interventions in closed and open injuries to the skull and brain.

11. How many functional support columns are there in the spine? What anatomical structures are the parts of each “column”.

12. Name all X-ray examinations used to diagnose spinal injuries.

13. Give the classification of closed injuries to the spine and spinal cord.

14. List the symptoms of the closed spinal cord injury.

15. Give the classification of gunshot wounds to the spine and spinal cord.

16. What periods are characteristic of the spinal cord injury.

17. Range of medical care for closed and open (gunshot) injuries to the spine and spinal cord during the evacuation phases.

7. Classes: Gunshot wounds and closed chest injuries

1. Name the main symptoms of closed chest injuries without damage to the internal organs (concussion, contusion, rib and sternum fractures).

2. Give the characteristics of double or ribs valve fractures, their diagnostics and treatment.

3. Explain the pathogenesis of paradox breathing.

4. Name the main symptoms of open penetrating and non-penetrating chest injury.

5. How is traumatic asphyxia clinically manifested?

6. How is the closed heart trauma clinically manifested (bruise, rupture of the heart muscle)?

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7. What types of pneumothorax do you know?

8. How is pneumothorax clinically diagnosed in the case of closed chest injury?

9. Range of medical care at the stages of medical evacuation in different types of pneumothorax.

10. Name the drainage points of the pleural cavity.

11. Name the methods of pleural cavity drainage

12. Describe the methods of anesthesia for chest injuries

13. Describe the technique of occlusion bandage applying.

14. List the indications for the surgical treatment of chest injuries.

15. Sorting and the amount of medical care for various chest injuries and their complications during the stages of medical evacuation.

17. Range of primary medical, pre-medical and first physician care for open chest injuries.

18. Name the possible late complications of chest injury and the principles of their treatment.

8. Classes: Gunshot wounds and closed injuries of limbs and joints. Blast trauma.

1. Give the classification of closed limb bone fractures.

2. Describe the classification of long bones open fractures by Gustillo-Anderson.

3. Describe the classification of long bones gunshot fractures.

4. Which pathomorphological features of gunshot fractures caused by modern high-speed firearms do you know?

5. Give the classification of gunshot injury to the joints?

6. Name and describe the most common complications typical in gunshot fractures and joints injury.

7. Range of the first medical, pre-medical and first physician care for various types (closed, open - gunshot and non-fire) injury to the bones and joints of the limbs.

8. Range of qualified surgical care for various types of bone and joint injury.

9. What are the features of surgical treatment of gunshot and open non-fire injuries to bones and joints?

10. What methods of fixing gunshot injury to the limb are most useful at the stage of qualified surgical care after primary debridement?

11. Range of specialized medical care for different types of limb injuries?

12. Give the classification of the limb vessels injury.

13. Range of first medical, pre-medical and first physician care for injuries to large vessels of the limbs.

14. Sorting and range of qualified surgical care for injuries to large limb vessels.

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15. Name the types of reconstructive surgery for injuries to the large limb vessels.

16. Give the classification of the large peripheral nerves injuries.

17. Range of medical care for peripheral nerves injury during the evacuation phases.

18. Name the types of reconstructive surgery for peripheral nerve damage.

9. Classes: Thermal injuries.

1. Name the existing types of incendiary stuff (fire-mixes) and their properties.

2. Describe burns caused by napalm, pirogel, termite, white phosphorus incendiary stuff (fire-mixes).

3. What are the causes of the burns caused by light radiation in a nuclear explosion?

4. Describe the first stage of burn disease.

5. What is the main difference between burn and traumatic shock?

6. What are the signs of the first period of burn disease end?

7. Describe the second, third and fourth period of burn disease.

8. What is the first medical care for the burns?

9. Range of pre-medical care to the burnt.

10. List the anti-shock measures carried out in burn shock during first aid.

11. List the groups of drugs used for intravenous infusions and determine their purpose in the treatment of different burn disease stages.

12. What formulae are used in infusion therapy for burn shock during the first day?

13. Name the criteria for efficiency of infusion therapy for burn shock.

14. What are the predictive signs of the victim's recovery from burn shock?

15. What can indicate changes in the color of urine, its specific weight, daily volume in case of extensive thermal burn?

16. Name the methods of local treatment for a burn wound.

17. List known types of skin plastics.

The questions for self-monitoring of the basic and final level are outlined in the methodical manual of test control for the course of war surgery for students of the 5th year.

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(на английском языке)

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