**МИНИСТЕРСТВО ЗДРАВООХРАНЕНИЯ РЕСПУБЛИКИ БЕЛАРУСЬ**

**УЧРЕЖДЕНИЕ ОБРАЗОВАНИЯ**
**«ГОМЕЛЬСКИЙ ГОСУДАРСТВЕННЫЙ МЕДИЦИНСКИЙ УНИВЕРСИТЕТ»**

**Кафедра травматологии, ортопедии и ВПХ**

**А. А. ТРЕТЬЯКОВ, В. И. НИКОЛАЕВ**

# ОСНОВЫ ТРАВМАТОЛОГИИ И ОРТОПЕДИИ

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

# BASIC PRINCIPLES OF TRAUMA AND ORTHOPEDICS

**Teaching workbook**
**for 5$^{th}$ year students of the Faculty on preparation of experts**
**for foreign countries of medical highest educational institutions**

**Гомель**
**ГомГМУ**
**2017**

УДК 616-001 + 617.3 (072)=111
ББК 54.58 + 54.582 я73Анг
Т 66

**Рецензенты:**
кандидат медицинских наук, доцент,
заместитель директора по науке Республиканского научно-практического центра радиационной медицины и экологии человека
***Э. А. Надыров;***
доктор медицинских наук, профессор,
профессор кафедры травматологии, ортопедии и ВПХ
Витебского государственного ордена Дружбы народов медицинского университета
***В. П. Дейкало***

**Третьяков, А. А.**
Т 66 Основы травматологии и ортопедии: учеб.-метод. пособие для студентов 5 курса факультета по подготовке специалистов для зарубежных стран медицинских вузов = Basic principles of trauma and orthopedics: teaching workbook for 5th year students of the Faculty on preparation of experts for foreign countries of medical highest educational institutions / А. А. Третьяков, В. И. Николаев; пер. на англ. яз. А. А. Третьякова. — Гомель: ГомГМУ, 2017. — 28 с.
ISBN 978-985-506-905-9

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

Предназначено для студентов 5 курса факультета по подготовке специалистов для зарубежных стран медицинских вузов.

Утверждено и рекомендовано к изданию научно-методическим советом учреждения образования «Гомельский государственный медицинский университет» 29 декабря 2016 г., протокол № 6.

**УДК 616-001 + 617.3 (072)=111**
**ББК 54.58 + 54.582 я73Анг**

**ISBN 978-985-506-905-9**

# CONTENTS

# I. INTRODUCTION TO TRAUMA AND ORTHOPEDICS

## *1. Introduction*

Orthopedics has come a long way since the days of Nicholas Andry, a French physician, who is credited for coining the term, orthopedics from two words, Ortho = straight and Pedics = child, in 1741.

The development of orthopedics as a specialty was very slow till 19th century. The discovery of anesthesia and aseptic surgical techniques opened-up new roads of treatment like open reduction, debridement, etc. The discovery of X-rays by Roentgen in 1895 and the introduction of the usage of Plaster of Paris by Antonius Mathijsen (Mathysen) in 1852 revolutionized the diagnosis and management of orthopedic disorders.

What was a primitive branch then restricted to correcting deformities in children, has developed into a full-fledged specialty from simple treatment, as done by traditional bonesetters to highly advanced joint, spine and hand surgeries.

## *2. The history of Trauma and Orthopedics*

The earliest examples of the active management of fractures in humans were discovered at Naga-ed-Der (about 100 miles north of Luxor in Egypt). Certainly, both Hippocrates and Celsus described in detail the splintage of fractures using wooden appliances. Longitudinal traction of the limb to overcome the overriding of fracture fragments had been described as early as the writings of Galen (ad 130 to 200).

But a fascinating account of external splintage of fractures is to be found in the work of Abu'l-Quasim Khalas Ibn'Abbas El Zahrawi from Cordoba in Spain (commonly shortened to Albucasis ad 936 to 1013). The technique of pouring a plaster-of-Paris mixture around an injured limb would appear to have been used in Arabia for many centuries and was brought to the attention of European practitioners by Eaton, a British diplomat in Bassora, Turkey, in 1798.

In Holland in 1852, Antonius Mathijsen (1805 to 1878) published a new method for the application of plaster in fractures. Mathijsen introduced his plaster bandage in 1876 at the Centennial Exhibition in Philadelphia. The use of plaster-of-Paris bandages for the formation of fracture casts became widespread after Mathijsen's death and replaced most other forms of splintage.

The man who coined the term osteosynthesis was Albin Lambotte (1866 to 1955). It is believed, however, that by osteosynthesis Lambotte meant stable bone fixation rather than simply suture. Lambotte is generally regarded as the father of modern internal fixation. The use of intramedullary rods to treat fractures was pioneered by Gerhard Küntscher of Germany.

Robert Danis (1880 to 1962) must be regarded as the father of modern osteosynthesis. Graduating from Danis devised numerous techniques of osteosynthesis based principally on interfragmentary compression, using screws and a plate designed to produce axial compression between two main bone fragments.

In the 1950s, Gavriil A. Ilizarov, a Soviet surgeon working in the Siberian city of Kurgan, made a serendipitous discovery: slow, steady distraction of a recently cut bone (securely stabilized in an external fixator) leads to the formation of new bone within the widening gap. Extending his observations, Ilizarov after that time developed an entire system of orthopedics and traumatology based on his axially stable tensioned wire circular external fixator and the bone's ability to form a «regenerate» of newly formed osseous tissue within a widening distraction gap.

Dr. Maurice Muller, who had read Darns s work, determined to embrace and develop the principles of the ideal osteosynthesis, as outlined by Danis, and to investigate the scientific basis for his observations. Over the next few years, Muller inspired a number of close colleagues to share his passion for the improvement of techniques for the internal fixation of fractures, gathering around himself particularly Hans Willenegger, Robert Schneider and subsequently Martin Allgower, who together laid the intellectual and indeed practical groundwork on March 15 to 17, 1958. Asembled surgeons formed a study group to look into all aspects of internal fixation — Arbeitsgemeinschaft fur Osteosynthesefragen, or AO. This was indeed a very active group that built on Danis's work in an industrious and productive way. No other single group of surgeons, coming together to pursue a common scientific and clinical aim, has had such an influence on the management of fractures.

Arthroscopy was pioneered in the early 1950s by Dr. Masaki Watanabe of Japan to perform minimally invasive cartilage surgery and reconstructions of torn ligaments. The modern total hip replacement was pioneered by Sir John Charnley in England in the 1960s. Since Charnley, W. H. Harris and his team at Harvard pioneered uncemented arthroplasty techniques with the bone bonding directly to the implant. Knee replacements using similar technology were started by McIntosh in rheumatoid arthritis patients and later by Gunston and Marmor for osteoarthritis in the 1970s developed by Dr. John Insall, Dr. Frederick Buechel and Dr. Michael Pappas utilizing a fixed and mobile bearing system.

Needless to say many pioneers both at the international and national level have contributed enormously for the development of this branch to the present what is today.

## II. INITIAL DEFINITIONS

***1. Sprain***: A sprain is an incomplete tear of a ligament or complex of ligaments responsible for the stability of a joint: e.g. a sprain of the ankle is a partial tear of the external ligament and is not associated with instability (as distinct from a complete tear).

***2.*** The term ***strain*** is applied to incomplete tears of muscles and tendons.

***3. Subluxation***: In a subluxation, the articulating surfaces of a joint arc no longer congruous, but loss of contact is incomplete. The term is often used to

describe the early stages in a condition which may proceed to complete dislocation (e.g. in a joint infection or in rheumatoid arthritis).

***4. Dislocation***: In a dislocation there is complete loss of congruity between the articulating surfaces of a joint in one or more planes. The bones taking part in the articulation are displaced relative to one another. Traditionally, displacement refers to the position of the distal fragment in relation to the assumed stationary proximal fragment.

**5.** A ***fracture-dislocation*** is a fracture occurring in or near a joint that results in a subluxation or dislocation of the joint.

***6. Fracture***

A fracture is present when there is loss of continuity of cortical and/or cancellous bone. The term covers all bony disruptions, ranging from one end of the scale when a bone is broken into many fragments (multifragmental, or comminuted fracture) to hair-line and even microscopic fractures at the other. All fractures are divided as open and closed. An open fracture can be defined as a broken bone that is in communication through the skin with the environment. Intraarticular fractures enter a joint and disrupt the joint surface and its articular cartilage. An incomplete fracture, typical in a child, is one that affects only a portion of the bone.

*Causes of fracture:*

— Direct violence.

— Indirect violence.

— Fatigue fractures.

— Pathological fractures.

***7. Fatigue (stress) fractures***

Stress fractures are fractures resulting from repetitive loading, each load being below the endurance limit but summated to produce a level of force that indeed causes a fracture. These injuries are typical in the proximal tibia, the second metatarsal, and the femoral neck. They may heal well if the cause of the force ceases soon enough, that is, if the patient stops running for a period of time.

*Progression of stress fractures:*

— stage I — crack initiation: areas of stress concentration;

— stage II — crack propagation: no repair or more damage than repair;

— stage III — final fracture: cracks coalesce, enlarge, ultimate failure.

Stresses, repeated excessive frequency to a bone, may result in fracture. This mechanism is often compared with fatigue in metals which break after repeated bending beyond their elastic limit.

***8. Pathological fracture***

Pathological fracture is one which occurs in an abnormal or diseased bone.

***9. Pediatric fractures***

The outer covering of the bone is a dense fibrous layer, which in the child is significantly thicker than that of the adult. Because of this thickened periosteum, fractures do not tend to displace to the degree seen in adults, and the intact periosteum can be used as an aid in fracture reduction and maintenance. Child

fractures will heal significantly faster than similar injuries in adults because all the cellular precursors are already present.

The defining component of the immature skeleton — is the growth plate, or the «physis». The physis is a cartilage plate interposed between the epiphysis (the secondary ossification center) and the metaphysic, thus a point of mechanical weakness. In children, a fracture through the cartilaginous growth plate is a common event. The Salter-Harris classification for physeal fractures system has allowed such injuries to be more precisely characterized. It is important to remember that physeal fractures heal very rapidly, but they may be complicated by complete or incomplete growth arrest, producing shortening or angular deformity of the limb.

Although a child's bone is lamellar in pattern, there remains enough flexibility in the skeleton to permit what has been called «biological plasticity», a phenomena not nearly as extensive in adult bone. Essentially, this allows a bone to «bend without breaking». It is responsible for some of the unique types of fractures seen in the pediatric age groups, specifically, torus and greenstick fractures.

1. Greenstick fracture: This occurs on the tension side of the bone and involves the diaphysis or cortical bone.

2. Torus or buckle fracture: Known by either name, this occurs on the compression side of bending and involves cancellous bone.

## III. EXAMINATION AND TREATMENT IN TRAUMA AND ORTHOPEDICS

***1. Muscle power***

Muscle power is usually graded on the Medical Research Council scale:

Grade 0 No movement;

Grade 1 Only a flicker of movement;

Grade 2 Movement with gravity eliminated (incomplete movement);

Grade 3 Movement against gravity (complete movement without resistance);

Grade 4 Movement against resistance with some limitation;

Grade 5 Normal power (complete movement without any limitation).

***2. Limb axis diagnostics tests and disorders***

*Normally limb axis goes straight across:*

— for upper limb: acromion — **shoulder bone head** — **radial bone head** — distal tendon of biceps — **ulnar bone head** — ulnar styloid;

— for lower limb: spina iliaca anterior superior — patellar middlpoint — 1 inter toes space (2 toe base).

Alignment means that the axes of the proximal and distal fragments are parallel to each other and the joint above and below are in the normal (parallel) relationship. Angulation is typically defined by the direction in which the apex of the angle points — medial, lateral, dorsal, volar, etc.

*Limb axis disorders:*

— varus deformation — the angle is open medially;

— valgus deformation — the angle is open laterally;

— antecurvation — the angle is open backward (but apex of the angle points is turned forward);

— recurvation — the angle is open forward (but apex of the angle points is turned backward);

— torsion (antetorsion — forward and retrotorsion — backward) — segment rotation according the direction.

***3. Limb length evaluation***

| Extremity | Length types, segments | Proximal point | Distal point |
|---|---|---|---|
| Upper E. | Relative length | Acromion | Top of the 3-ed finger |
| | Anatomical / absolute length | Grater tubercle | Ulnar styloid processus |
| | Shoulder | Grater tubercle | External epycondylus |
| | Forearm | Olecranone | Ulnar styloid processus |
| Lower E. | Relative length | Spina Iliaca anterior superior | Top of the medial ankle |
| | Anatomical / absolute length | Trochanter major | Top of the lateral ankle |
| | Thigh | Trochanter major | Knee joint lane |
| | Shin | Knee joint lane | Top of the lateral ankle |

***4. Types of limb length changing (shortening or lengthening)***

— *true* (absolute or bony) — fracture, acquired and congenital bone deformity, etc;

— *relative* (dislocation or joint) — as result of joint dislocation, without real bone shortening;

— *apparent* (projection) — as result of muscle shortening and rigidity (contracture), without real bone shortening and joint dislocation;

— *functional* (summary) — sums of all limb disorders presenting in this particular case.

***5. Range of motion and joint flexibility tests***

This 0- to 180-degree system of notation, also called the neutral zero method, is widely used throughout the world. First described by Silver 3 in 1923, its use has been supported by many authorities, including Cave and Roberts, Moore, the American Academy of Orthopedic Surgeons, and the American Medical Association.

***6. Range of joint motion according neutral zero method***

| Joint | Flexion-Extension | Adduction-Abduction | Internal-External Rotation (pronation – supination) |
|---|---|---|---|
| Shoulder J. | 180° – 0° – 40° | 40° – 0° – 180° | 90° – 0° – 10° |
| Elbow J. | 140° – 0° – 0° (5°) | — | 90° – 0° – 90° |
| Wrist J. | Back Flexion — Palmar Flexion: 70° – 0° – 70° | Radial Abduction — Ulnar Abduction: 20° – 0° – 70° | — |
| Hip J. | 180° – 0° – 10° | 45° – 0° – 45° | Straight joint: 20° – 0° – 70° Flexed joint: 70° – 0° – 20° |

| Joint | Flexion-Extension | Adduction-Abduction | Internal-External Rotation (pronation – supination) |
|---|---|---|---|
| Knee J. | 140° – 0° – 0° | — | Straight joint: 0<br>Flexed joint: 5° – 0° – 5° |
| Ankle J. | Back Flexion — Plantar Flexion: 30° – 0° – 60° | 40° – 0° – 15° | — |

***7. Types of joint movement volume changing***

— *Ankhilosis* — absolute absence of movement — hard joint fusion;

— *Rigidity* — joint movement limitation to 3° – 5° – 7° — fibrous ankhilosis;

— *Contracture* — more or less significant limitation of joint movement. It could be I, II, III degree of severity. According to main causes we can divide it into desmogenic, dermatogenic, tendogenic, miogenic, arthrogenic, neurogenic etc.

— *Normal (physiological) volume of* joint movement;

— Excessive *movement*— more than normal volume of joint movement without functional disorders (training flexibility, congenital features);

— *Pathological movement* — more than normal volume of joint movement with functional disorders (congenital disease, neurological disorders, ligament rupture);

— *Luxated o*r *unstable joint (articulatio fluctuans*) — unlimited movement without any control (full destruction of joint surfaces and ligaments after severe injury, join resection, endoprothesis removing).

## IV. CLINICAL FEATURES AND CORDIAL SIGNS OF BONE FRACTURES AND DISLOCATION

***1. General signs and symptoms of fractures and dislocation***

1. Hard pain at or near the location of fracture;
2. Swelling about the fracture area;
3. Loss of power: the injured part cannot be moved normally;
4. Deformity of the limb the limb may assume an unnatural position and be misshapen;
5. Irregularity of the bone. If the fracture is near the skin the irregularity of the bone may be felt.

***2. Hard signs of Fractures***

1. Crepitus (bony grating) may be heard or felt;
2. Unnatural movement at the seat 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.

***3. Hard signs of Dislocations***

1. Hollow joint deformity;
2. Spring joint movement limitation.

## V. RADIOGRAPHIC FEATURES OF THE FRACTURES AND DISLOCATION

***1. Musculoskeletal Radiography***

Radiographs have been used for imaging since their discovery by Roentgen in 1895, and are usually the initial method performed for investigating musculoskeletal pathology. The most-frequently requested diagnostic studies, they are obtained relatively quickly and cheaply. Radiography continues to be a vital imaging technique in all spheres of orthopedic practice.

***2. Radiographic sign's of musculoskeletal pathology***

a) Fractures lane patterns:

— for the long bones: transverse, oblique, spiral, T, Y and V-shaped, longitudinal;

— for the cancellous bones: transverse, marginal, avulsion, impacted or compressed;

— for the plane bones: transverse, marginal, avulsion, stellar.

b) Fracture dislocation types:

— transverse dislocation where the two fragments are moved at width;

— vertical dislocation with shortening in relation to one another, and distraction, where essentially the bone ends are pulled apart;

— angulations' dislocation occurs when the two fracture fragments are not aligned and an angular deformity is present. Angulations are typically defined by the direction in which the apex of the angle points — medial, lateral, dorsal, volar, etc;

— rotational dislocation present when there is a torsional relationship between the two fracture fragments: antetorsion, retrotorsion.

c) Sign`s of comminuted (multi fragmental) fractures characterized by the presence of one or more intermediate fragments.

d) Sign`s of subperiosteal fractures:

— greenstick fracture: cortical layer deformation occurs on the tension side of the bone and involves the diaphysis or cortical bone;

— torus or buckle fracture: cortical layer deformation occurs on the compression side of bending and involves cancellous bone.

e) Sign`s of incomplete fractures: loss of continuity that involves only one cortical layer of a bone.

f) Radiographic findings supporting non union:

— persistent fracture lines with absence of bone crossing the fracture site (bridging trabeculas);

— sclerotic fracture edges with obliteration of bone canal, failing or broken implants.

g) The radiographic sign`s of chronic osteomyelitis may reveal osteolysis, periosteal reaction and sequestra (segments of necrotic bone separated from living bone by granulation tissue) in the same time — coral-shaped bone transformation;

Subacute (primary chronic) osteomyelitis:

— Garre`s — thickening (bulge) of the diaphisal cortical layer with or without bone canal obliteration;

— Brodie's — ovoid/spherical metaphisal bone abscess;

— Ollye`s – surface abnormalities of bone (e.g., periostitis) — small areas of osteolysis in cortical bone.

h) Periosteal reaction in malignant bone tumors (cortical destruction and periosteal elevation):

— triangle periosteal elevation (Codman's sign);

— periosteal accretion (onion shell sign);

— spicular (needle) periosteal reaction;

— cauliflower (festoon, lace-shaped) shaped reaction.

i) Bone tumors signs:

— local bone destruction with clear or poorly defined borders (minus-tissue);

— extraosseous bone extension (plus-tissue) with soft tissue ossification;

— abnormal trabecular bone structure.

j) Additional bones (sesamoid);

k) Congenital bone fragmentation;

l) Synostosis (congenital and pathological joint union).

m) Osteoarthritis sign`s:

— marginal osteophyte formation;

— subchondral sclerosis;

— localized joint space narrowing;

— and subchondral cysts.

n) Signs of avascular bone necrosis: osteosclerosis (so-called relative radiodensity) with bone compaction (impression).

***3. Computed tomography (CT)***

Like plain tomography, CT produces sectional images through selected tissue planes — but with much greater resolution. A further advance over conventional tomography is that the images are trans-axial (like transverse anatomical sections), thus exposing anatomical planes that are never viewed in plain film x-rays.

***4. Magnetic resonance imaging (MRI)***

Magnetic resonance imaging produces cross-sectional images of any body part in any plane. It yields superb soft-tissue contrast, allowing different soft tissues to be clearly distinguished, e.g. ligaments, tendons, muscle and hyaline cartilage. Another big advantage of MRI is that it does not use ionizing radiation. It is, however, contra-indicated in patients with pacemakers and possible metallic foreign bodies in the eye or brain, as these could potentially move when the patient is introduced into the scanner's strong magnetic field.

***5. Radionuclide imaging***

Photon emission by radionuclides taken up in specific tissues can be recorded by a gamma camera to produce an image which reflects physiological activity in that tissue or organ. The radiopharmaceutical used for radionuclide im-

aging has two components: a chemical compound that is chosen for its metabolic uptake in the target tissue or organ, and a radioisotope tracer that will emit photons for detection.

***6. Diagnostic ultrasound***

High-frequency sound waves, generated by a transducer, can penetrate several centimetres into the soft tissues; as they pass through the tissue interfaces some of these waves are reflected back (like echoes) to the transducer, where they are registered as electrical signals and displayed as images on a screen. Unlike x-rays, the image does not depend on tissue density but rather on reflective surfaces and soft-tissue interfaces. Real-time display on a monitor gives a dynamic image, which is more useful than the usual static images. A big advantage of this technique is that the equipment is simple and portable and can be used almost anywhere; another is that it is entirely harmless.

## VI. FRACTURE HEALING

***1. Normal Bone Growth and Development***

Bone is a biphasic connective tissue consisting of an inorganic mineral phase and an organic matrix phase. The hardness of bone allows it to provide several specialized mechanical functions: the protection of internal organs, the scaffold that provides points of attachment for other structural elements, and the levers needed to improve the efficiency of muscle action. In addition, bone serves two biologic functions: a site for hematopoietic activity and a reservoir of minerals needed for metabolic interchange.

***2. Etiology of skeletal disease***

As one considers the etiology of skeletal disease, it is helpful to evaluation of possible diagnoses that may explain the findings manifested by the skeleton. The seven disease categories are best remembered using the acronym «VITAMIN»:

**V**, vascular disease
**I**, infection
**T**, tumor
**A**, arthritis
**M**, metabolic bone disease
**I**, injury
**N**, neurodevelopmental causes

***3. The biology of fracture healing***

The biology of fracture healing is not particularly complex and parallels that of any nonossified tissue. Essentially, fracture healing occurs in three phases:

1. *Vascular phase*. This phase begins at the time of the insult and proceeds through the development of a hematoma (*period of inflammation*). This hematoma is then infiltrated by cellular elements, which in turn lay down collagen and cause hematoma organization (*period of proliferation*). A vascularization

step follows when the organized hematoma is vascularized by small arterial extensions. The end result of the vascular phase is the development of a soft callus.

2. *Metabolic phase (period of regeneration)*. This stage begins about 4 to 6 weeks after the injury. During this period, the soft callus is reworked by a number of specific cellular elements to produce a firm, hard callus satisfactory for meeting the mechanical demands placed upon the fracture in the early phase.

3. *Mechanical phase (period of remodeling)*. This phase begins once a hard callus is present, which is then manipulated according to the rules of Wolff's law. Essentially, mechanical stress is required to produce skeletal remodeling during this phase and ultimately to produce a solid, mechanically strong bone with normal structure).

***4. Fracture healing complications***

A number of complications can occur following fractures and joint dislocations.

*Chronic regional pain syndrome* (reflex sympathetic dystrophy, Zudec-syndrom): This unusual and disastrous complication is typically seen following trivial trauma after prolonged immobilization. The patients develop an exquisitely painful tender extremity and present a management disaster. Prognosis depends on early recognition. Early aggressive physical therapy and return to normal function are important to the rehabilitation of patients with these difficult complications.

*Problems of union*

a. Malunion: defined as a healing in poor position for function.

b. Delayed union: a fracture that has not healed in the usual statistical time frame.

c. Nonunion: A fracture that has not healed and will not heal because it has lost its «biological drive» (a pseudarthrosis, or «false joint», develops).

## VII. NON UNION AND PSEUDOARTHROSIS

***1. Non union etiology, diagnostic, treatment and prophylaxis***

A number of reasons can be found why fractures do not heal. Excessive motion, infection, steroids, radiation, age, nutritional status, and devascularization locally have all been implicated in the delay of healing. The worst case scenario typically involves skeletal nonunion.

Radiographic findings supporting non union:

— persistent fracture lines with absence of bone crossing the fracture site (bridging trabeculas);

— sclerotic fracture edges with obliteration of bone canal;

— lack of evidence of progressive change toward union on serial x-ray;

— failing or broken implants;

— lack of callus (most unreliable finding).

If a bone fails to heal, surgical intervention for stabilization is frequently required. In addition to stabilization, biologic stimulation is necessary to make the fracture heal. Usually, this is accomplished through the application of bone graft material, with or without some type of external bone stimulation.

***2. Hypervascular or Hypertrophic (horse hoof) nonunion***

Hypertrophic non unions are rich in callus and have a rich blood supply in the ends of the fragments. They result from insecure fixation (inadequate stability) or premature weight bearing in a reduced fracture whose fragments are viable. Hypertrophic nonunion displays exuberant callus on radiographs. There is increased uptake on radionuclide scans. Management: may have high incidence of union after rigid ORIF compression plates or medullary nails, and cancellous bone grafts are optional.

***3. Oligotrophic nonunion***

These are not hypertrophic, and callus is absent. They typically occur after major displacement of fractures, distraction of fragments, or internal fixation without off accurate apposition of fragments. Has an intact blood supply and demonstrates radiotracer uptake on radionuclide scans. It has inadequate healing response.

***4. Avascular or Atrophic nonunion***

Ends of the fragments have become osteoporotic and atrophic. The nonunion is inert and incapable of biologic reaction. It will have cold bone scan. There is poor blood supply to the ends of the fragments. These are usually final result when intermediate fragments are missing and scar tissue that lacks osteogenic potential is left in their place. Radiographs may show eburnated, osteopenic, and/or sclerotic bone ends. Management: open decortication must be carried out, and cancellous bone grafts should be added.

***5. Comminuted nonunion***

Typically these nonunions result in the breakage of any plate used in stabilizing the acute fracture. Comminuted nonunions are characterized by the presence of one or more intermediate fragments that are necrotic.

***6. Defect nonunion***

Defect nonunions are characterized by the loss of a fragment of the diaphysis of a bone. These non unions occur after open fractures, sequestration in osteomyelitis, and resection of tumors. Ends of the fragments are viable, but union across the defect is impossible. As time passes the ends of the fragments become atrophic. Management: Ilizarov bifocal bone transport or cage plastic surgery is optional.

## VIII. TREATMENT FOR FRACTURES

***1. Indications for different types***

All fractures require that two basic goals be accomplished in their treatment: (1) reduction and (2) maintenance of that reduction. Different techniques may be used for achieving these two goals. First, the reduction of a fracture can be accomplished by closed manipulative methods, by surgical open reduction, or through the application of traction. Following reduction, the fracture site must be immobilized so that the fracture will heal in the optimum position. Immobilization can be achieved with external methods such as casts, splints, and external

fixators: with internal methods, using various devices such as screws, plates, and intramedullary rods; or by the maintenance of the patient in traction. Fracture healing is promoted by physiological loading of the bone, so muscle activity and early weight bearing are encouraged.

These objectives are covered by three simple injunctions:

- Reduce.
- Hold.
- Exercise.

*Orthopedic Emergencies*

Relatively few orthopedic problems mandate immediate intervention. However, those that do exist truly represent emergent situations: these are open fractures, dislocations of major joints, and fractures associated with vascular injury, including compartment syndrome.

***2. Fixation treatment of fractures***

***Transporting immobilization***

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

***Cast splintage***

Plaster of Paris is still widely used as a splint, especially for distal limb fractures and for most children's fractures. It is safe enough, so long as the practitioner is alert to the danger of a tight cast and provided pressure sores are prevented. After the fracture has been reduced, stockinet is threaded over the limb and the bony points are protected with wool. Plaster is then applied. While it is setting the surgeon moulds it away from bony prominences; with shaft fractures three-point pressure can be applied to keep the intact periostal hinge under tension and thereby maintain reduction.

In general, cast splintage is used for all undisplaced fractures, for most fractures in children and for fractures that are stable after reduction

***3. Traction treatment of fractures***

***Closed reduction***

Reduction should aim for adequate apposition and normal alignment of the bone fragments. The greater the contact surface area between fragments the more likely healing is to occur. A gap between the fragment ends is a common cause of delayed union or nonunion. On the other hand, so long as there is contact and the fragments are properly aligned, some overlap at the fracture surfaces is permissible. The exception is a fracture involving an articular surface; this

should be reduced as near to perfection as possible because any irregularity will cause abnormal load distribution between the surfaces and predispose to degenerative changes in the articular cartilage (posttraumatic osteoarthritis).

In general, closed reduction is used for all minimally displaced fractures, for most fractures in children and for fractures that are not unstable after reduction and can be held in some form of splint or cast.

***Skeletal tension***

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

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

Traction is applied to the limb distal to the fracture, so as to exert a continuous pull in the long axis of the bone, with a counterforce in the opposite direction (to prevent the patient being merely dragged along the bed). This is particularly useful for shaft fractures that are oblique or spiral and easily displaced by muscle contraction. Traction is safe enough, provided it is not excessive and care is taken when inserting the traction pin. The problem is speed: not because the fracture unites slowly (it does not) but because lower limb traction keeps the patient in hospital.

Traction includes:

*Traction by gravity* — This applies only to upper limb injuries. Thus, with a wrist sling the weight of the arm provides continuous traction to the humerus.

*Skin traction* — Adhesive plaster is stuck to the shaved skin and held on with a bandage, and cords or tapes are used for traction. Skin traction will sustain a pull of no more than 4 or 5 kg.

*Skeletal traction* — A stiff wire or pin is inserted - usually behind the tibia tubercle for hip, thigh and knee injuries, or through the calcaneum for tibia fractures — and cords tied to them for applying traction. Whether by skin or skeletal traction, the fracture is reduced and held in one of three ways: fixed traction, balanced traction or a combination of the two.

***4. Surgical treatment of fractures***

Stabilizing two segments or fragments of bone is usually by internal or external fixation methods. In internal fixation, this may involve screws, wires, plates or intramedullary rods (nails). External fixators come in a variety of types.

***External fixation***

External fixators are useful for open fractures and for reconstruction of limbs using the Ilizarov method. They can also be used as temporary fracture stabilization devices when the local soft tissue conditions need improving before open surgery, or during emergency fixation of multiple long-bone fractures. The fixators functions as an exoskeleton through which the patient's own skeleton can be supported and adjusted. The basic components are wires or pins inserted into bone to which rods or rings are attached and interconnected. Pin- or wire-related problems have limited widespread adoption of this method.

***Internal fixation by intramedullary devices***

Two major design types are used: those with and those without interlocking capabilities (*Interlocked* and *Unlocked* intramedullary nails).

*Interlocked intramedullary nails* offer better control of length and torsion than the unlocked varieties of this device. Stability from these nails is due to a combination of an interference (frictional) fit within the medullary canal and the capture of bone to nail by means of the interlocking screws. Interlocking nails have become a standard fixation method tor most shaft fractures of the tibia and femur in adults.

*Unlocked intramedullary nails* are increasingly used in the treatment of long-bone shaft fractures in children. These flexible rods are inserted so as not to damage the physes at either end of the long bone and function as internal splints until callus formation takes over.

***Internal fixation by plates with screws***

Plates of varying design may be incorporated:

1) simple straight compression plates, which will allow compression along the axis of the plate;

2) contoured plates to fit specific bones;

3) low-profile plates that reduce the "footprint" on the bone so as to preserve local vascularity;

4) locked plates where the screw also engages the plate by a secure mechanism so as to create a stable construct and prevent toggling.

***Internal fixation by screws***

Screws can be used simply by holding two fragments in close proximity or to fix a plate to the bone. They may also be used to compress two fragments together through what is called the "Lag principle". Some form of external splintage (usually a cast) is applied as support.

***Internal fixation by transfixing wires***, often passed percutaneously, can hold major fracture fragments together. They are used in situations where fracture healing is predictably quick (e.g. in children or for distal radius fractures). Some form of external splintage (usually a cast) is applied as support.

***Internal fixation by cerclage and tension-band wires (TBW)*** are essentially loops of wire passed around two bone fragments and then tightened to compress the fragments together. During the usage of cerclage wires, it`s need to be sure that the wires hug the bone and do not embrace any of the nearest nerves or vessels.

## IX. OPEN FRACTURES

***1. Definition***

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

By definition, an open fracture involves exposure of fractured bone to the extracorporeal environment, thus increasing the risk of bone contamination from foreign debris and bacteria.

Open fractures are classified according to their severity.

***2. Classification of open fracture by Gustillo-Anderson***

| 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 fracture bone despite the extensive soft-tissue laceration or damage |
| IIIB | Type III fracture with extensive soft-tissue loss and periosteal stripping and bone damage. Usually associated with massive contamination. Will often need further soft-tissue coverage procedure (i.e. free or rotational flap) |
| IIIC | Type III fracture associated with an arterial injury requiring repair, irrespective of degree of soft-tissue injury |

In a first degree open fracture, the bone fragment penetrates the skin. First degree open fractures have very small wounds and following surgical debridement can be treated in the same manner as a closed fracture.

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

Third-degree open fractures represent the most severe form of open fracture with combination of the fracture and massive soft tissue damage. Soft tissue and bony avulsion may be part of the fracture, and often these injuries are caused by outer forces. Most commonly third degree open fractures in the combat are the result of high-velocity bullet wounds.

***3. Treatment of the open fractures***

Open fractures are considered operative emergencies and need to be taken to the operating room as soon as the patient is considered medically stable enough to tolerate surgical intervention. Wounds should not be explored in the emergency room as further soft tissue damage may be incurred. Active bleeding can almost always be controlled with local compression before surgical exploration. Reduction of severely contaminated fractures should be avoided in the emergency room to prevent the drawing of foreign debris into the wound. In addition, intravenous antibiotics should be given immediately upon admittance to the emergency room. A general rule with regard to antibiotic therapy is that first-generation cephalosporins are given for grade I and grade II fractures. For

patients with grade III fractures, a first-generation cephalosporin is given in conjunction with an aminoglycoside.

Another important concept is «the zone of injury» which refers to the area around the wound that has been traumatized but can recover with appropriate management of the soft tissues and bone. Although an initial debridement should aggressively remove necrotic or devitalized tissues, marginal tissue may be preserved to permit potential recovery. Tissues that have demonstrated local recovery can be preserved, whereas other tissues that have continued to remain ischemic and demonstrate evidence of necrosis should be widely excised. One cardinal rule is to never close the soft tissue laceration of an open fracture. This procedure is potentially disastrous in that an open drainage system is converted to a closed cavity, with a greater susceptibility to develop an abscess or, in the worst case scenario, frank gangrene. All open fractures should be covered with moist sterile dressings that prevent bone and soft tissue desiccation.

The principles of wound management are undisputed when dealing with open fractures, with the majority of controversies surrounding when and what to use for bone stabilization. Early bone stabilization via intramedullary nailing, plate and screw fixation, or external fixation optimizes soft tissue healing and access to the extremity for examination and treatment. By preventing continued soft tissue shearing forces, with a resultant further devascularization of soft tissue, further extension of the zone of injury can be minimized. Options for soft tissue coverage should be individualized for the patient and the degree of injury.

***4. Complication***

Open fractures increase the risk of subsequent sepsis. Closed fractures that have been treated operatively are also at risk. The use of implants increases the risk of infection simply because they provide a substrate for the microcolonization of certain bacteria. The presence of necrotic bone also contributes to infection risk.

## X. POSTTRAUMATIC CHRONIC OSTEOMYELITIS

Generally, these bone infections are secondary to an open wound, most often an open injury to bone and surrounding soft tissue. Localized bone pain, erythema and drainage around the affected area are frequently present. The cardinal signs of chronic posttraumatic osteomyelitis include draining sinus tracts, deformity, instability and local signs of impaired vascularity, range of motion and neurologic status. Patient factors, such as altered neutrophil defense, humoral immunity and cell-mediated immunity, can increase the risk of osteomyelitis.

Staphylococcus epidermidis, S. aureus, Pseudomonas aeruginosa, Serratia marcescens and Escherichia coli are commonly isolated in patients with chronic osteomyelitis.

***1. Diagnosis***

The diagnosis of osteomyelitis is based primarily on the clinical findings, with data from the initial history, physical examination and laboratory tests serv-

ing primarily as benchmarks against which treatment response is measured. Leukocytosis and elevations in the erythrocyte sedimentation rate and C-reactive protein level may be noted.

In osteomyelitis of the extremities, plainfilm radiography and bone scintigraphy remain the primary investigative tools. Radiographic evidence of bone destruction by osteomyelitis may not appear until approximately two weeks after the onset of infection. The radiographs may reveal osteolysis, periosteal reaction and sequestra (segments of necrotic bone separated from living bone by granulation tissue).

For nuclear imaging, technetium Tc-99m methylene diphosphonate is the radiopharmaceutical agent of choice. CT scan can reveal small areas of osteolysis in cortical bone, small foci of gas and minute foreign bodies. Magnetic resonance imaging (MRI) can be extremely helpful in unclear situations MRI also provides greater spatial resolution in delineating the anatomic extension of infection.

***2. Posttraumatic osteomyelitis management***

Management of posttraumatic osteomyelitis involves consideration of several patient variables: physiologic, anatomic, and psychosocial. After the initial evaluation, staging and establishment of microbial etiology and susceptibilities, treatment includes: antimicrobial therapy, debridement with management of resultant dead space and, if necessary, stabilization of bone. In most patients with osteomyelitis, early antibiotic therapy produces the best results. Antimicrobials must be administered for a minimum of four weeks (ideally, six weeks) to achieve an acceptable rate of cure.

***3. Cierny-Mader Staging System for Chronic Osteomyelitis***

The Cierny-Mader classification has been developed to assist surgeons in classifying and selecting various modalities of treatment and to assist in predicting outcomes.

*Anatomic type*

— Stage 1: medullary osteomyelitis.

— Stage 2: superficial osteomyelitis.

— Stage 3: localized osteomyelitis.

— Stage 4: diffuse osteomyelitis.

*Physiologic class* (Factors affecting immune surveillance, metabolism and local vascularity).

A host: healthy

B host:

Bs: systemic compromise. Systemic factors (Bs): malnutrition, renal or hepatic failure, diabetes mellitus, chronic hypoxia, immune disease, extremes of age, immunosuppression or immune deficiency

Bl: local compromise. Local factors (Bl): chronic lymphedema, venous stasis, major vessel compromise, arteritis, extensive scarring, radiation fibrosis, small-vessel disease, neuropathy, tobacco abuse

Bls: local and systemic compromise

C host: treatment worse than the disease

***4. Surgical treatment***

Surgical treatment of osteomyelitis involves three main facets:

1) extensive debridement

2) vascular soft tissue coverage, and

3) bone stabilization. Intravenous antibiotics should be administered and directed toward isolated organisms for at least 6 weeks.

Surgical debridement in patients with chronic osteomyelitis can be technically demanding. The quality of the debridement is the most critical factor in successful management. An aggressive debridement is crucial to achieving successful eradication of osteomyelitis. All nonviable tissue must be removed to prevent residual bacteria from persistently reinfecting the bone. Removal of all adherent scar tissue and skin grafts should be undertaken. Hardware removal and debridement of avascular outer cortex (down to bleeding bone — «paprika sign») must be done. Multiple cultures of all debrided material should be obtained before the initiation of antibiotic therapy.

After debridement with excision of bone, it is necessary to obliterate the dead space created by the removal of tissue. Dead-space management includes local myoplasty, free-tissue transfers and the use of antibiotic-impregnated beads. Soft tissue reconstitution may involve a simple skin graft, but it often requires a local transposition of muscular tissue or vascularized free tissue transfers to effectively cover the debrided bone segment.

Loose implants should be removed and either replaced or substituted by another implant type (i.e., an external fixator replacing a loose plate and screws). Once a fracture has healed, the implant can be removed and further debridement performed as necessary.

Finally, bone stability must be achieved with bone grafting being undertaken when necessary to bridge osseous gaps. Cancellous and cortical autografts are commonly used, with vascularized bone transfer (vascularized free fibular, iliac, and rib grafts) being occasionally necessary. Although vascularized bone grafts provide a fresh source of blood flow into previously devascularized areas of bone. Technically demanding, application of a small pin (Ilizarov method) or half-pin external fixator with bone distraction following a cortical osteotomy can produce columns of bone that fill segmental defects. As distraction is carried out, the soft tissues regenerate along with the bone to cover the newly generated tissue. Recent results seem encouraging, as these patients appear to achieve greater success rates for limb-sparing methods as compared to patients undergoing more conventional bone replacement techniques.

## XI. POLYTRAUMA (MULTIPLE TRAUMA)

Polytrauma, multiple fractures and high-velocity injuries severely exposed the limitations of the conventional treatment in orthopedics, as the fracture pat-

terns were bizarre and complicated. Thus newer modalities of treatment like improved methods of internal fixation, the AO systems, the interlocking nail system, Ilizarov's method, etc. were introduced into orthopedic management.

***1. Mechanical injuries classification***

*Injury Patterns*

According to multiplicity injury are divided to:

— *Isolated injury.*

— *Multiple injuries.*

— *Concomitant injuries.*

— *Combined injury.*

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

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

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

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

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

***2. High energy trauma***

According to traumatic energy following injury are divided to:

— *Superficial injury* (bruises, contusions, abrasions etc.);

— *Single low energy injury* (fractures, dislocations, wounds, tendon ruptures);

— *High energy injury* (polytrauma or multiple traumas).

Superficial injury associated with absence of anatomical disruption and accompanied with local inflammation. This injury doesn`t needs any special care — just prolonged rest, light cooling and administration of non-steroid anti-inflammatory drugs.

Isolated (low energy) injury associated with presence of single organ anatomical disruption and always needs injured organs restoration, or creation condition for this (casts, surgery, fixation and etc.). But this type of injury never accompanied with significant general dangerous pathological reaction.

A high-energy injury associated with multiple injuries (polytrauma) and accompanied with general life threatened pathological reaction (co called mechanical muscle-crush injury — MMCI, or traumatic disease).

It is typical of casualties crushed under masonry, vehicles, or victims lying unconscious without movement for many hours (mechanical muscle-crush injury — MMCI). Compartment syndrome, rhabdomyolysis, and acute renal fail-

ure are associated with structural collapse, prolonged extrication, severe burns, and some poisonings. In this type of injury it is difficult to define the border between living and dead muscle.

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

## XII. TRAUMATIC DISEASE

***1. Stage of traumatic disease development***

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

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

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

4. Stage IV — Recovery.

***2. Signs and Symptoms***

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

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

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

***3. Rhabdomyolysis***

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

Muscle can survive circulatory ischemia for up to 4 h. Violent crushing destroys muscle immediately; even if the force is insufficient to mangle the muscle

tissue, the combination of mechanical force and ischemia will cause muscle death within an hour.

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

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

***4. Acute renal failure (ARF)***

Acute renal failure (ARF) is the most common and life-threatening complication of rhabdomyolysis, with an incidence of 13–50 % and a reported mortality of up to 59 %. For ARF to develop, two factors have to be present in addition to myoglobinemia: hypovolemia and aciduria. Myoglobin is filtered by the glomerulus into tubular fluid and, in an acidic environment, forms casts together with Tamm-Horsfall proteins that occlude the tubule. In addition, myoglobin contains hemoproteins that generate toxic ferrum radicals that attack tubular cells. If anuria does develop, it may continue for 4–8 weeks before kidney function recovers. The development of acute anuric renal failure in critically ill patients adds 20–30 % to their chances of over-all mortality. However, rhabdomyolysis-induced renal failure is reversible and most patients regain normal renal function even after 4–8 weeks of anuria and dialysis.

***5. Disseminated Intravascular Coagulation (DIC)***

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

- *I Phase*

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

- *II Phase*

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

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

• *III Phase*

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

***6. Prevention***

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

***7. Systemic therapy of traumatic disease***

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

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

3. Alkalinization of urine: acidic urine promotes myoglobin cast formation.

4. Correction of electrolyte abnormalities: hyperkalemia, hyperphosphatemia, hypocalcemia, hyperuricemia.

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

***8. Damage control (multiple trauma treatment) at medical care echelon***

In damage control for trauma patients, the main principles are to perform temporary operative procedures to provide time for physiologic stabilization before definitive surgical care. There are four distinct phases of the damage control philosophy: the first is recognition of the at risk patient, next, temporizing operative procedures to limit ongoing injury, third, intensive care unit (ICU) care for resuscitation, and finally definitive surgical procedures. Basic to this philosophy is the idea that the surgeon does anything to keep the patient alive and accepts the morbidity of the early procedures and later returns to reconstruct and repair.

In these settings, essential to have system of medical/trauma triage and to answer some key questions

1. If resources limited, who should receive priority?

2. Can care be safely rendered at battle site/place of disaster?

3. Is there land, sea, or aeromedical evacuation capability?

Always remember to focus team on saving life first, then limb-threatening injuries. Early involvement of orthopedic surgeon as part of a multi-disciplinary trauma team is essential to triage urgency of surgical interventions to multiple-injured patient.

*Resuscitation /Damage control*

A — Airway with cervical spine protection.

B — Breathing.

C — Circulation with haemorrhage control.

D — Disability or neurological status.

E — Exposure and Environment — remove clothing, keep warm.

Many early deaths can be prevented by:

1. Control of limb hemorrhage and aggressive management of shock.
2. Relief of airway obstruction.
3. Relief of tension pneumothorax.
4. Early medical evacuation to surgical treatment facility.

***9. Rehabilitation during multiple trauma treatment***

Systemic factors play a pivotal role in promoting multiple trauma healing. It has been estimated that 50% of medical and surgical patients have overt or subclinical protein and calorie malnutrition. Multiple injuries or even isolated fractures result in a large increase in a patient's metabolic demand needed to assist in healing. Systemic parameters that have been shown to impede soft tissue healing include a serum albumin less than 3.5mg/ml or a total lymphocyte count less than 1500 cells/ml. Patients presenting with or developing malnutrition following multiple traumatic injuries are at increased risk of infection, delayed union, or nonunion of open fractures. Aggressive nutritional resuscitation is necessary with either oral or feeding tube supplementation or, in extreme cases, parenteral nutrition.

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

Most patients with rhabdomyolysis who survive the complexity of their injury will recover also from its complications.

## LITERATURE

1. Louis Solomon, David Warwick and Selvadurai «Apley's System of Orthopaedics and Fractures, 9th edition» / Louis Solomon. — London: Hodder Arnold, 2010.

2. Miller`s Review of Orthopaedics, 7th edition / Edited by Mark D. Miller, MD and Stephen R. Thompson. — PA: Elsevier, 2016.

3. Essentials of Orthopedic Surgery, 4rt edition / Edited by Sam. W. Wiesel, John N. Delahay. — Springer Science + Business Media, 2011.

4. Ronald McRae, Max Esser «Practical Fracture Treatment, 5th edition» / Ronald McRae. — Churchill Livingstone: Elsevier, 2008.

5. Bruce D. Browner, Robert P. Fuller «Musculoskeletal Emergencies, 1st edition» / PA «Saunders Elsevier», 2012.

6. European Surgical Orthopaedics and Traumatology, The EFORT Textbook / Edited by George Bentley. — Springer, 2014.

7. Thomas P Rüedi, Richard E Buckley, Christopher G Moran «AO Principles of Fracture Management» / Thomas P Rüedi. — Thieme, 2007.

8. Suresh Sivananthan, Eugene Sherry, Patrick Warnke, Mark D Miller «Mercer's Textbook of Orthopaedics and Trauma, 10th edition» / Suresh Sivananthan. — CRC Press, 2012.

Учебное издание

**Третьяков** Александр Анатольевич
**Николаев** Владимир Иванович

**ОСНОВЫ ТРАВМАТОЛОГИИ
И ОРТОПЕДИИ
(на английском языке)**

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

Редактор ***Т. М. Кожемякина***
Компьютерная верстка ***С. Н. Козлович***

Подписано в печать 01.03.2017.
Формат 60×84$^{1}/_{16}$. Бумага офсетная 80 г/м$^{2}$. Гарнитура «Таймс».
Усл. печ. л. 1,63. Уч.-изд. л. 1,78. Тираж 50 экз. Заказ № 106.

Издатель и полиграфическое исполнение:
учреждение образования «Гомельский государственный медицинский университет».
Свидетельство о государственной регистрации издателя,
изготовителя, распространителя печатных изданий № 1/46 от 03.10.2013.
Ул. Ланге, 5, 246000, Гомель.