

# З ДОСВІДУ ВИЩОЇ ШКОЛИ

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## DEGENERATIVE DYSTROPHIC DISEASES OF JOINTS AND SPINE

### DEGENERATIVE DYSTROPHIC DISEASES OF JOINTS

Degenerative-dystrophic diseases of joints are characterized by the chronic progressive impairment of the articular cartilage, epiphyseal bone structures metabolism with the following involvement of other articular structures into the pathologic process. They are accompanied by the development of joint deformations, incongruency, loss of functional activity, development of the secondary inflammatory process in the joint of different intensity, disability and decreasing of quality of life in such patients.

According to clinical and morphological data of NS Kosinskaia and AG Rohlin, three forms of Degenerative-dystrophic lesions of joints are distinguished (Kosinskaia NS, Rohlin AG, 1961), which get their place in group XIII of ICD-10 "Diseases of the musculoskeletal system and connective tissue".

### OSTEOARTHRITIS

Osteoarthritis – heterogenic group of diseases with different etiology and similar biological, morphological and clinical manifestations and consequences, in the background of which lies damage of all the articular elements, articular cartilage in the first turn, and also subchondral parts of the bone, synovial membrane, ligaments, capsule, paraarticular muscles (Fig. 1). Mentioned changes occur gradually as the injury of the articular cartilage during loads exceeds its recovery speed (physiological regeneration).

These diseases compound up to 80 % in the general structure of joint pathology in people above 60 years, osteoarthritis leads to the disability of different degree in 10–30 % of cases. Growth of the osteoarthritis morbidity is fixed during the last 30–60 years in 5–9 times. Medicodemographic indicators of Europe and USA indicates the continuation of the population "aging" and increase of the amount of people elder than 60 years; this age group population will increase twice by 2020. Increase of the agerelated morbidity of osteoarthritis is predicted, especially in employable age, and also morbidity increase in childhood and adolescence – "osteoarthritis rejuvenation". All these components predetermine further morbidity growth of this pathology in absolute as well as in relative numbers.

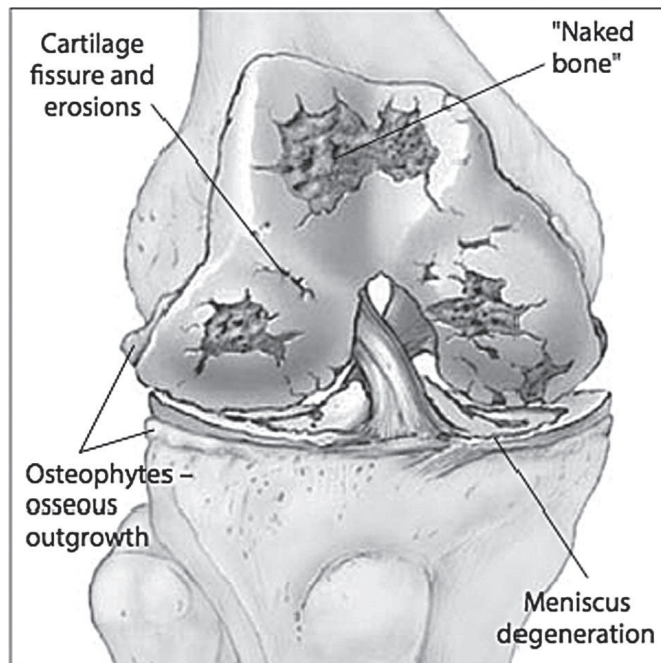


Figure 1. Osteoarthritis.

Terminological definitions – osteoarthritis, arthrosis, osteoarthrosis, degenerative arthritis – are currently represented as synonyms in ICD-10.

Joints of the wrist, first metatarsophalangeal, joints of the cervical and lumbar parts of the spine, knee and hip joints are more often affected by osteoarthritis. By the severity of the musculoskeletal system function impairment, first place takes the hip, knee and ankle joints, and also shoulder joint.

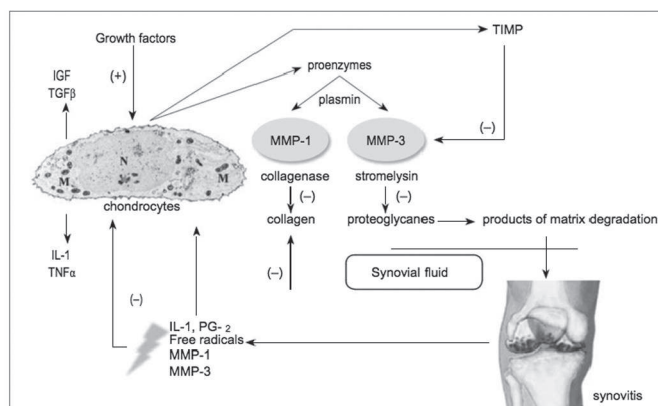
Osteoarthritis treatment is complex and continuous. Presence of multiple comorbid pathologies in elderly patients requires strict approach in the choice of the effective and safe therapy, based on the reliable evidential data.

### ETIOLOGY AND PATHOGENESIS

Osteoarthritis (OA) – one of the most widespread degenerative-dystrophic disease of joints. From 6.4 % to 12 % of world population suffers from it.

This is polyetiological disease, which occurrence and development are connected with a series of genetic, endogenic (hormonal disbalance, immune disturbances, oxidative stress) and exogenic factors (trauma, obesity). Most scientists consider that cartilage tissue is initially damaged in OA. Not only the impairment of the cartilage matrix structure and function are observed

in the pathogenesis of this disease, but also its metabolism. Main pathologic osteoarthritis manifestation is the articular cartilage destruction, the main function of which is to adapt joint to the mechanical load and providing of free movements of the articular surfaces.



**Figure 2.** Osteoarthritis pathogenesis scheme; (-) - catabolic influence on the articular cartilage; (+) - anabolic influence on the articular cartilage.

Cartilage consists of two main elements: intercellular substance (matrix), which consists 98 % of the cartilaginous tissue volume, and cells: chondrocyte and chondroblast (2 %). Important components of the cartilaginous matrix are macromolecules of different types of collagen (mainly type II) and proteoglycans (PG). PG provides unique adaptive qualities of the cartilage. 90 % of cartilage refers to aggrecans. This molecule consists of the protein nucleus with binded chains of chondroitin sulfate (ChS), keratan sulfate (KS) hyaluronic acid (HA).

Typical signs of cartilage destruction in OA is a loss of glycosaminoglycan (GAG) with matrix – ChS, KS, HA in superficial, intermediate and deep zones. Decrease of the PG molecule is also observed, which lose the ability to going out from the cartilage matrix. PG (changed and small) are able to absorb water, but are not able to keep it. Water excess is absorbed with collagen, which distends and lose fibrous structure, that leads to the cartilage resistance decrease.

Chondrocytes – cells, that regulate the cartilage tissue metabolism, its synthesis (anabolism) and degradation (catabolism) of the aggrecan and other components of the cartilage matrix. These processes are normally balanced, but impairment of the normal cartilage tissue metabolism is observed in OA towards the prevalence of catabolic processes over the anabolic.

Significant role in the catabolic processes development in OA plays proinflammatory cytokines, especially IL-1. Chondrocytes synthesize proteolytic enzymes under its influence, as called matrix metalloproteinases, that cause the collagen and cartilage PG degradation. Chondrocytes synthesize PG, are not able to aggregate and produce collagen types I, IX, X, which does not form fibers, instead of normal (Fig. 2).

Typical chondrocyte feature in OA is cyclooxygenase-2 (COX2) hypersecretion (enzyme, which induces prostaglandins synthesis, that participate in the inflammation development) and induced form of nitric oxide synthase, that provides toxic influence on the cartilage and induce chondrocytes apoptosis.

Release of active biologic substances promotes the inflammation maintenance in articular tissues. As the result, further destruction of the synovial layer with the development of reactive synovitis and increased production of proinflammatory cytokines occur. Released PG, products of chondrocytes and collagen breakup, as antigens can induce antibodies generation with formation of the local inflammatory process.

Vascular genesis of degenerative diseases consists in changes of the subchondral bone blood supply, hypertension, swelling and microfractures development with following remodeling and sclerosis, that changes the conditions of load on the articular cartilage with OA development

Data about the role of freeradical peroxidase reaction in osteoarthritis pathogenesis showed, that excessive activation of the FRLP is one of the key initial mechanisms in osteoarthritis development. High capability of free radicals to oxidative modification of highest and intracellular proteins, proteins of the cartilage matrix, that is accompanied by tearing of proteoglycans macromolecular bands, is an important direct factor of the cartilage destruction initiation and progressing in osteoarthritis.

## CLASSIFICATION

Osteoarthritis is conditionally divided into primary and secondary. Clinical manifestation of osteoarthritis occurs in the load increasing in the joint, that leads to the articular cartilage damage. Usual load causes osteoarthritis symptoms occurrence, when there are pathologic changes of cartilages, bones, synovial membrane, ligaments, muscles, which are conditioned by the certain primary process (diseases). Secondary osteoarthritis does not differ from primary by clinical manifestations, with the exception of the fact, that the certain etiologic factor is a cause of the secondary osteoarthritis. So, information mentioned above allows considering, that in the basis of the primary osteoarthritis lies natural aging, degeneration of the articular cartilage and paraarticular tissues – tendons, ligaments, articular capsule, muscles. Agerelated degeneration occurs in the connective tissue, which is the basis of all these structures. In the case of secondary osteoarthritis development, an influence of different exogenic and endogenic factors on the articular tissues is combined with involuntary processes, that occurs in them.

The Ukrainian Association of Rheumatologists (UAR) osteoarthritis classification adopted at 2000 is currently used as the main working classification.

#### CLINICAL DIAGNOSTICS

Clinical symptoms in different pathologic processes, that take place in the joint, is rather similar – clinical signs do not correspond to certain anatomical changes. Significant difference between the clinical and radiological investigations attract attention: significant radiological changes can be find in the lack of clinical data and vice versa. Primary changes in osteoarthritis occur in the articular cartilage, so symptoms are not expressed at the early disease stages, and is often conditioned only by the inflammatory process presence – reactive synovitis. Main clinical manifestations of osteoarthritis are: pain (Table 1), joint deformations, their function impairment – have different intensity degree depending on the process stage. Fourstaged radiological classification according to J. H. Kellgren and J. Lawrence is more widely used among the orthopedisttraumatologists. The following clinical changes usually correspond to the radiologic changes in the joint.

Table 1.

#### Characteristics of the pain syndrome is osteoarthritis.

PAIN CHARACTER	CLINICAL FEATURES
Nocturnal pain	Connected with venous hyperemia, blood stasis in subchondral bone parts, intraosseal hypertension. Pain intensity decreases in the morning during walking (in affection of lower limbs)
Initial pain	Occur in the beginning of walking, then quickly disappears, occurs again continuously, during movements
Initial pain	Occur in loads on the joints, gradually increase to the evening, disappears after night rest. Pain, that occurs during movements, is often conditioned by the tendo-bursitis, periarthrititis, tendons affection presence. Pain can also be connected with synovial membrane irritation and osteophytes.
Blocking pain	Occurs in the presence of the articular foreign body - cartilage fragment, that is compressed between articular surfaces
Reflex pain	Conditioned by the reactive synovitis
Reflected pain	Is explained by the involvement of the articular capsule in the inflamative process

First stage is characterized by discomfort or insignificant pain presence in the joint during or just after the intensive load. These symptoms disappear quickly after the rest. Tenderness in palpation, limitation of the active and passive movements are absent during clinical examination. Limitations of the move-

ments, that has minimal amplitude can be found in the reactive synovitis presence: extension in the ankle joint, overextension in the knee joint, internal rotation in the hip joint, etc. Functional ability is practically preserved in these patients, disorders occur only in significant physical loads.

In the second stage of the disease, pain in the joint is characterized by greater duration and intensity, disappears only after long rest. Limitation of active and passive movements, pain in palpation are determined constantly, but patients can do everyday work. Muscular force decrease is determined. Limping can occur sometimes. Flexion (knee joint), adduction (hip joint) contractures are often observed, which have extraarticular character and can be corrected with conservative treatment. Functional capacity is impaired, especially in patients with the significant physical load.

Clinical symptoms in the third stage are conditioned by articular as well as extraarticular lesions. Pain syndrome intensity increases significantly during physical activity and decrease at rest. Morning stiffness is observed in patients. Palpation of the affected joint is painful for the patient. Movements in the joint are limited, crepitus can be observed in movements; contracture, forced limb position occur. Joint function is stably impaired. Working capacity of the patient is limited or lost depending on the character of performed work.

Fourth stage of osteoarthritis is characterized by constant pain syndrome, which intensity increases significantly in physical activities. Expressed morning stiffness, crepitus, atrophy of periarticular muscles are determined. Movements in the joint are significantly limited, and sometimes are characterized only as oscillating. In the big joint of lower limb affection, gait impairment is expressed in this patients, need of additional support occurs (crunches, orthopedic cane). Functional capacity of the joint is significantly reduced or lost. Reactive inflammatory process often occurs at this stage, with moderately or significantly expressed synovitis.

#### RADIOLOGIC DIAGNOSTICS

The most widespread instrumental method of diagnostics is radiography. OA has typical radiologic signs – joint space narrowing, subchondral osteoporosis and osteosclerosis, osteophytes and subchondral cysts formation. J.H. Kellgren and J. S. Lawrence in the 1957 year suggested osteoarthritis classification according to radiologic diseases stages, which is currently used with clinicomorphological supplements and is generally accepted.

It should be mentioned that all radiologic OA signs reflect changes of bone structures, but provide

no direct picture of hyaline cartilage, and is only indirect sign of its change. Absence of correlation between the progress of clinical and radiologic osteoarthritis manifestations is reflected in articles of various authors. E. Bagge study showed, that 57 % of patients with clinical OA signs were detected no radiological changes. So, the radiologic method is not sufficiently effective in determining early OA stages.

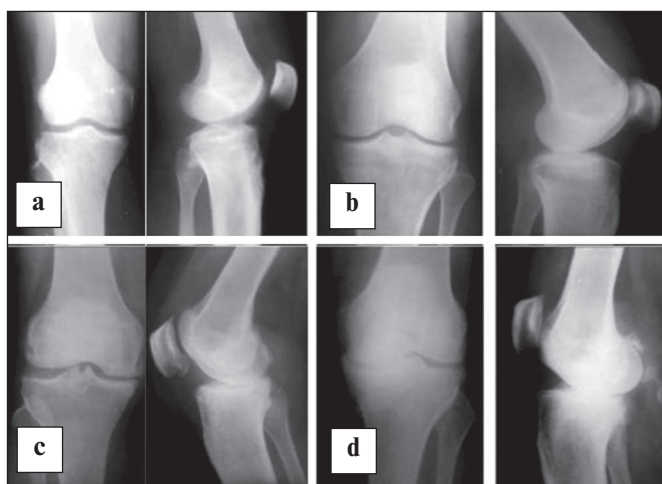
Radiologic diagnostics of osteoarthritis is based on the following radiologic signs determination according to J. H. Kellgren and J. S. Lawrence classification (1957), Figure 3.:

- **0 stage** – absence of radiologic signs;
- **I stage (questionable)** – insignificant narrowing of the joint fissure, irregularity of the joint fissure;
- **II stage (minimal)** – joint fissure narrowing is less than 50 %, its irregularity, areas of subchondral osteosclerosis, single focus of osteoporosis, single osteophytes (marginal osseous overgrowth);
- **III stage (medium)** – joint fissure narrowness of more than 50 %, its expressed irregularity, expressed subchondral osteosclerosis, multiple foci of osteoporosis, multiple but small osteophytes, presence of the insignificant epiphysis deformation;
- **IV stage (expressed)** – significant joint fissure narrowness up to its disappearing, large areas of osteosclerosis in loaded areas, diffuse osteoporosis, cystic cavities presence, massive osteophytes and significant epiphysis deformation.

Computed tomography (CT), arthrosonography, magnetic-resonance imaging (MRI) and diagnostic arthroscopy obtain bigger importance in OA diagnostics during last years.

#### ARTHROSONOGRAPHY

Arthrosonography – ultrasound investigation of the joint – allows determining anatomical structures,

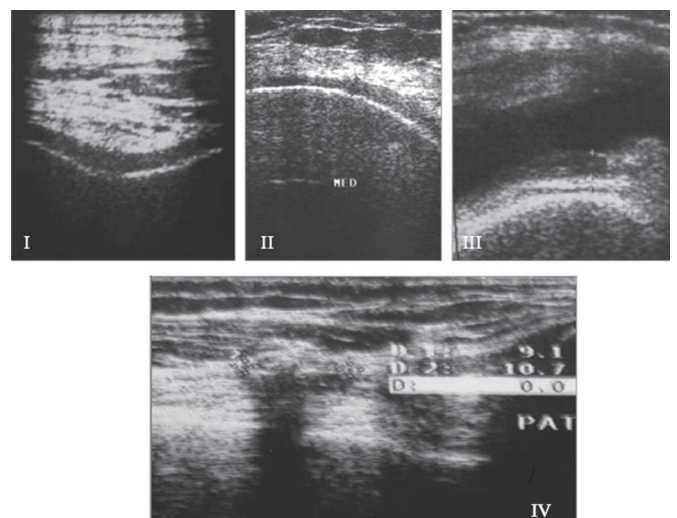


**Figure 3.** X-ray patterns of different osteoarthritis stages of knee (A - correspond to I radiologic stage according to Kellgren&Lawrence, 1957, B - II stage, C - III stage, D - IV stage)

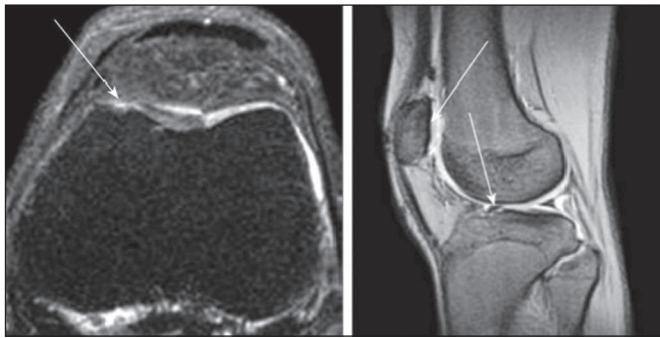
that are poorly determined during other methods of investigation. This method gives a possibility to determine fluid accumulation in the joint even in the insignificant amount of it. Fluid character can be preliminarily assessed by the echo signal character (serous, hemorrhagic, purulent). Ultrasound investigation also gives a possibility to determine cysts presence, changes of tendons and tendon sheaths, ruptures of ligaments and tendons. This method allows assessing the degree of degenerative changes of the articular cartilage, its thickness, joint fissure parameters, subchondral bone condition (Fig. 4).

#### MAGNETIC RESONANCE IMAGING AND COMPUTED TOMOGRAPHY

Magnetic resonance imaging (MRI) is considered as the best noninvasive method of the hyaline articular cartilage investigation. Early MRI investigations showed relative homogeneity of the unchanged articular hyaline cartilage, then the presence of layers was determined. MRI reliably demonstrates focal as well as generalized lesions of the cartilage (Fig. 5). MRI allows assessing the water content in the hyaline cartilage using standard impulse sequences. There is a method with contrast substance use – MR-arthrography. Increasing of methods sensitivity to 93 %, specificity – to 97,6 %, diagnostic value – to 91,5 % are observed after the contrast substance introduction. MRI use for assessment of the articular hyaline cartilage condition is a reliable and perspective method in OA diagnostics, especially at early stages. D. Rappeport et al. in the 1996 year suggested to perform MRI before arthroscopy in the investigation algorithm of the patient with osteoarthritis.



**Figure 4.** US-pattern of the osteoarthritis stages of knee (I-IV stages).



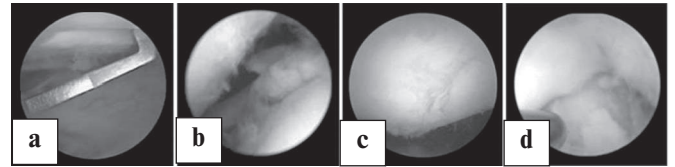
**Figure 5.** Knee MRI. Osteoarthritis patterns of the knee. Areas of cartilage lesions are indicated with arrows.

Computed tomography (CT) gives the possibility to assess objectively the articular surfaces interposition and bone structure character of the epiphysis. Joint investigation provides the tridimensional view of the lesion focus, its localization, sizes, extension, connection with other articular elements. Advantage of this method is the possibility of detailed assessment of osseous articular elements structure, that has significance in posttraumatic osteoarthritis, when it is important to determine the bone defects presence, changes of the articular surfaces form, character of the bone structural changes (osteosclerosis, osteonecrosis, osteoporosis, cystic formations, etc.).

#### DIAGNOSTIC-MEDICAL ARTHROSCOPY

Diagnostic arthroscopy – one of the modern widespread method of osteoarthritis diagnostics and treatment, especially at early stages. Arthroscopy allows determining of pathologic lesions or injuries of practically all intraarticular elements and fix the determined changes at the film or digital storage. All intraarticular elements are visualized during investigation – articular cartilage, synovial membrane, intraarticular bodies, menisci, foreign bodies, lipid bodies, etc. Changes in the articular cartilages such as softening, garrnetting and erosions are clearly determined in osteoarthritis (Fig. 6). Hyperemia and moderate swelling of the synovial membrane are determined, its villous hypertrophy often with areas of fibrous degeneration close to the area of the articular cartilage destruction. Typical changes of the synovial fluid are also determined – it obtains intensive yellow color, turbid, opalescent (contains a large amount of fibrin, remainings of collagen fibrils and fragments of the cartilage matrix). Arthroscopic investigation can be accompanied by the significant lavage of the articular cavity with removing of free small fragments of the degenerative changed articular tissues, part of the hypertrophic synovial villi. Debridement and articular cartilage smoothing can be conducted if necessary, that allows improving significantly the joint function for the long period of time.

It is possible to sample biologic material (cartilage) during diagnosticmedical arthroscopy – biopsy for the following histomorphologic investigation, that provides the possibility of the final reliable diagnosis verification. Diagnostic arthroscopy is currently considered the "gold standard" in osteoarthritis diagnostics, especially at early stages, as it allows to clearly determine the degree of degenerative changes of the articular cartilage, extension and spread to other articular elements, and also helps to choose optimal treatment method for the certain patient.



**Figure 6.** Arthroscopic patterns of the articular cartilage condition in osteoarthritis of knee (A – corresponds to stage according to J. H. Kellgren & J. S. Lawrence; 1957 B – II stage; C – III stage; D – IV stage)

#### LABORATORY DIAGNOSTICS

To serologic OA markers refer products of PG degradation (keratan sulfate, chondroitin sulfate, glycosaminoglycan (GAG), PG fragments), stromelysin, procollagen type II, osteocalcin, products of synoviocytes (cytokines, proteins), crystals (calcium, apatites, pyrophosphates). Catabolic processes prevail in osteoarthritis, macromolecules (predominantly of the articular cartilage matrix) or their fragments get to the synovial fluid, blood and urine, where they can be determined. Special cell enzymes and cytokines, that get to the interstitial fluid in inflammation, can also be metabolic markers of the articular cartilage and synovial membrane.

Physical, cellular and molecular content of the synovial fluid changes in osteoarthritis, that is determined in laboratory investigation. Synovial fluid is transparent or slightly turbid, of a high or medium viscosity, mucin clot is dense. Number of cells in 1 mcl of the synovial fluid is from 500 to 5000, neutrophiles compounds less than 50 %, fragments of the cartilaginous tissue can appear.

According to modern data about molecular markers of articular tissues metabolism in synovial fluid and serum. Aggrecan, proteins of the cartilage matrix, metalloproteinases are considered the most objective markers. Also increased levels of keratan sulfate, proteoglycans, protease, C-propeptide, collagen type II, fibronectin, cytokines, hyaluronidase and others can be determined in the synovial fluid.

#### HYSTOMORPHOLOGIC DIAGNOSTICS

Hystomorphologic method of the biopsy material assessment of the articular cartilage or synovial mem-

brane, that can be obtained during the arthroscopy, gives the possibility of reliable diagnosis, and also specifying its stage. During the biopsy material of synovial membrane investigation the following is determined: covering cells are located in one layer, villi are atrophic, lack of vessels, significant zones of fibrosis are determined, fatty degeneration. Investigation of the cartilage tissue biopsy material gives the possibility to determine the decrease of the perichondrocytic lacunae area in superficial layers, a decrease of the cellular density in deep layers, decrease of the nuclei amount in the medium layers, increase of the calcified cartilage layer thickness. Morphologic signs of osteoarthritis in modern literature are described by V. Scoft.

#### TREATMENT OF PATIENTS WITH OSTEOARTHRITIS

Modern principles of the osteoarthritis treatment is based on staging, continuity and succession. Recommendations on the knee and hip joints osteoarthritis treatment were developed and approved basing on the evidential medicine data of European League Against Rheumatism (EULAR). These recommendations consider four main groups of medical measures in osteoarthritis: nonpharmacologic treatment, pharmacologic treatment, intraarticular treatment and surgical treatment.

#### I. NONPHARMACOLOGIC TREATMENT

Nonpharmacologic treatment provides the following measures: educative programmes for patients to study the basic principles of the osteoarthritis prevention and treatment, excessive weight decrease by the diet correction and creation of the optimal motion regime, development of the individual exercise complex, orthopedic unloading regime, a complex of physiotherapeutical treatment, therapy with vitamins and minerals, phytotherapy.

Exercise therapy is one of the most important methods of rehabilitation and joint function improvement in patients with osteoarthritis.

##### **Exercise therapy in osteoarthritis promotes:**

- prevention and removal of periarticular muscles atrophy;
- prevention and removal of the joint instability;
- decreasing of arthralgias intensity, function improvement of the affected joints;
- slowdown of the further osteoarthritis progression;
- body weight decrease.

##### **Scheme of osteoarthritis diagnostics:**

###### *I. Clinical criteria*

1. Articular pain, that occurs at the end of the day in the first half of the night.

2. Articular pain, that occurs after the mechanical load and decreases during rest.
3. Joint deformations due to the marginal outgrowth (osteophytes), increase of the axial joint deformations.
4. Limitation of the active and passive movements range in the joint.

#### **II. Instrumental diagnostics:**

1. Radiology, main signs:
  - narrowing of the articular fissure;
  - subchondral thickening – zones of subchondral osteosclerosis;
  - marginal osseous overgrowth – osteophytes;
  - subchondral osteoporosis.
  - Radiologic stages of osteoarthritis according to J. H. Kellgren and J. Lawrence (1957): 0, I, II, III, IV.
2. Arthroscopy – direct visual intraarticular investigation with the possibility of biopsy, Now is considered as the "gold standard" in the osteoarthritis diagnostics.
3. Arthrosonography – ultrasound investigation of the joint.
4. Computed tomography.
5. MRI: noninvasive, highly informative, without radiation exposure.
6. Histologic method: biopsy.

#### **III. Laboratory diagnostics**

1. Assessment of the physical, biochemical, cellular and molecular condition of the synovial fluid.
2. determining of the biologic molecular markers in the biologic mediums (serum, synovial fluid, etc.).

#### **Purposes of the osteoarthritis treatment are:**

- Slowdown of the osteoarthritis progression.
- Decreasing of the pain syndrome intensity.
- Increasing of the functional activity and quality of life of the patients.

There are following treatment methods for performing these tasks in osteoarthritis patients:

- Education of the patient (explaining the basics of this disease) and social support.
- Decreasing of the excessive body weight.
- Orthopedic regime and auxiliary orthopedic devices (insole, stick, crutches and other).
- Exercise therapy, massage and physical activity.
- Physiotherapeutical treatment.
- Sanatorium and spa rehabilitation.
- Medications.
- Operative treatment.

Physiotherapeutical treatment decreases the pain syndrome and synovitis manifestations. Influence of electromagnetic fields of high and superhigh frequencies, ultrasound therapy and phonophoresis of antiinflammatory and other drugs, shortwave diathermy in synovitis absence, electrophoresis, laser therapy, balneotherapy (radon, hydrosulfuric, sodium chloride, turpentine, iodidebromine), hydrotherapy (multiple procedures) are used for reaching the mentioned above effects.

## II. PHARMACOLOGICAL TREATMENT

The main purpose of pharmacological (medicamentous) treatment of osteoarthritis are the correction of the intraarticular lesions, pain syndrome decrease, improvement of intraosseous and regional blood flow, metabolism stimulation throughout the organism in general and in the articular cartilage in particular, treatment of accompanying pathology. Correction of the intraarticular lesions is in the protective action on the articular cartilage, normalization of the biosynthetic processes in chondrocytes, catabolic processes inhibition in bone and cartilage tissues, normalization of the synovial fluid secretion in synoviocytes and synovitis depression.

The task of the pain syndrome intensity decreasing is aimed at the overcoming of the inflammatory process in the joint and normalization of the periarticular muscles tone. Metabolic processes stimulation in the organism consists in the metabolism correction, improvement of the bone tissue quality, vitamin supplements and mineral metabolism correction, psychotropic therapy.

**Antiosteoarthritis drugs.** Broadening and deepening of conception about the disease nature and mechanisms of its development lead to the revision of the application point and assessment of the pathogenetic importance of most drugs, that are used in OA therapy.

The classification of antiosteoarthritis drugs, that include two classes of drugs, according to their pharmacologic action is currently accepted:

### I. Symptoms modifying antiosteoarthritis drugs – SMOADs):

- *Symptomatic drugs of fast action* (to them refer nonsteroid antiinflammatory drugs (NSAIDs, paracetamol, opioid analgesics, glucocorticoids).
- *SYSADOA – symptomatic slow acting drugs for osteoarthritis* (chondroitin sulfate, glycosamin sulfate, hyaluronic acid, nonsaponifying compounds of avocado/soya, diacerein).

### II. Structure modifying or disease modifying antiosteoarthritis drugs – DMOADs).

It should be mentioned that practically all drugs, that are used for osteoarthritis treatment (nonsteroid antiinflammatory drugs, analgesics, glucocorticoids,

hyaluronic acid, glucosamine and chondroitin sulfate) have symptomatic (symptommodifying) effect, that is characterized by the different onset of action. As to the pathogenetic action (structuremodifying or diseasemodifying), there is no 100 % for any of the pharmacologic drugs. But the fact, that all mentioned above symptom-modifying drugs have the influence on some links of the osteoarthritis pathogenesis, is undoubtful. It is impossible to take the effect of these drugs only to the symptomatic action. The possibility of SYSADOA (symptomatic slow acting drugs for osteoarthritis) group drugs to influence positively the metabolism of the articular cartilage in osteoarthritis was shown in series of experimental and clinical studies. So drugs of this group (chondroitin sulfate, hyaluronic acid and others) are currently called as chondromodifying or such, that structurally modifies the cartilage. It was also demonstrated the possibility of pharmacological correction of the articular cartilage reparation in its injuries and posttraumatic osteoarthritis with these drugs use.

So, symptom-modifying drugs can provide the pathogenetic action and vice versa, pharmacologic drugs of pathogenetic action can have the symptomatic effect.

Nonsteroid antiinflammatory drugs (NSAIDs) has wide practical use in medicine. Around one hundred of NSAIDs of different classes are currently developed and produced. Development of new, more safe and effective drugs of this groups continues. The main mechanism of NSAIDs action – inhibition of prostoglandins (PG) biosynthesis, that are formed from the arachidonic acid, by the cyclooxygenase (COG) activity inhibition.

There is NSAIDs classification according to the character of their influence of the articular cartilage metabolism, suggested by J. Dingle and M. Parker in the 1997 year, which was then specified and supplemented (Table. 2).

Table 2

#### Classification of some NSAIDs action on the articular cartilage metabolism

Inhibits glucosaminoglicans biosynthesis and articular cartilage metabolism	Acetsalicylic acid Indometacin Ibuprofen Fenoprofen Phenolbutason
Do not influence the glucosaminoglycans biosynthesis and articular cartilage metabolism	Meloxicam Piroxicam Diclofenac Sulindac Celexocib
Stimulate glucosaminoglycans biosynthesis and articular cartilage metabolism	Benoxaprofen Tiaprofenic Paracetamol Nimesulide (10 mcg/ml)

Depending on the action duration, NSAIDs are divided into the short-acting, medium-acting and long-acting.

- I. Short-acting NSAIDs has the half-period from 2 to 8 hours. To them refer: ibuprofen, ketoprofen, indometacin, fenoprofen, diclofenac, fenamates, tolmetin.
- II. Medium-acting NSAIDs are characterized by the half-period from 10 to 20 hours. representatives of this group are naproxen, diflunisal, sulindac.
- III. Long-acting NSAIDs has 24 and more half-period. To these drugs refer oxicams (piroxicam, meloxicam, lornoxicam), phenylbutazone.

**Steroid anti-inflammatory drugs.** Systemic use of glucocorticoids is not indicated in osteoarthritis, and periarticular and intraarticular injections of prolonged glucocorticoid forms exclusively according to indications and with strict maintenance of injection rules. This provides significant but temporary, symptomatic effect. Glucocorticoids action feature is that they are able to rather freely get through the plasmatic cell membranes unlikely the most hormones. The leading path of the steroid hormones influence on cellular processes – influence on the gene expression with inhibition of prostaglandins and leukotrienes synthesis, that play significant role in the inflammatory process development. So, leading mechanism of corticosteroid action is inhibition of the cyclooxygenase and lipoxygenase ways of the arachidonic acid metabolism.

Injections of glucocorticoids are not recommended to perform more often than 3–4 times a year, and the interval between the injections in the same joint should be more than 3 months. Main indications for glucocorticoids use in osteoarthritis – persistent synovitis on the background of the conservative treatment, and also persistent inflammation of periarticular tissues (tendovaginitis, bursitis, etc.).

Glucocorticoid injections are contraindicated in infectious arthritis of different etiology, infection of skin and subcutaneous fat tissue or muscles in the area of injection, sepsis, hemarthrosis in patients with hemophilia or traumas, in intraarticular fractures. Absolute contraindication to the glucocorticoid use is the Cushing's syndrome or disease. These drugs should be administered with caution in patients with arterial hypertension, peptic ulcer disease of the stomach and duodenum, diabetes mellitus, predisposition to the clot formation, cachexia. In persistent pain syndrome and synovitis absence, that does not pass after conservative treatment, intraarticular injection of glucocorticoids is contraindicated. Glucocorticoids injection into tendons is contraindicated. According to H. J. Kreder et al. (1994) data, negative influence of intraarticular glucocorticoid injections in animals was

increased by their physical activity. After glucocorticoid drugs injection it is necessary to provide rest to the joint for 1–2 days, that promotes more expressive and long effect.

**Symptomatic slow acting drugs for osteoarthritis (SYSADOA).** As was mentioned above, drugs from SYSADOA groups is currently named as chondro-modifying, structurally modifying the cartilage, diseases-modifying, chondroprotectors. To these drugs in the first turn refer the structural analogs of the cartilaginous tissue: chondroitin sulfate, glucosamin sulfate, hyaluronic acid drugs, diacerein, nonsaponifying compounds of avocado and soy, complex drugs (alflutop), combined drugs.

Practically in all conducted studies, drugs of SYSADOA group showed efficacy and safety in osteoarthritis treatment.

Glycosamine (GA) – amino sugar, that is the component of practically all human tissues, including cartilage. It was established, that it compounds GAG, that forms the matrix of all connective tissues. GA is a substrate for proteoglycans biosynthesis of the articular cartilage and also their components. It was established, that the primary biological role of the glucosamine is directly conditioned by its ability to stimulate the glucosaminoglycans biosynthesis and hyaluronic acid, which are necessary for the proteoglycans formation.

Chondroitinsulfate (ChS) together with keratn-sulfate (KS), dermatansulfate (DS), heparansulfate and heparin refer to the group of compounds, that has common name – sulphated glucosaminoglycane (GAG). ChS in formed from the glucosamin in the organism. It compounds the aggrecan– macromolecule, that creates the microenvironment for the chondrocytes functioning in the articular cartilage, influence their metabolism in such a way. An important value of ChS is it ability to influence the proliferation and metabolism of chondrocytes.

**Hyaluronic acid (HA)** – natural polysaccharide, that is present in many tissues of the organism. OA is accompanied by changes of the cartilage metabolism. One of the consequences of this processes is the synthesis impairment, and also changes of HA properties, natural cartilage component, that determines viscoelastic properties of the synovial fluid, which are lost in osteoarthritis, as depends on the HA molecular weight and concentration. Antiinflammatory effect of glucocorticoids is connected with their ability to interact with specific cell receptors (chondrocytes, synoviocytes), such as CD44, RHAMM, CAM. HA inhibits the enzyme activity, that destroys articular cartilage. Analgesic effect of HAs is connected with their lubricating properties, as well as with the possible direct influence on the nerve endings of the sy-



novial membrane. Exogenous HA inhibits synthesis of endogenous HA. Multiple clinical studies showed the high efficacy of HA in OA.

Antioxidant drugs inhibits processes of lipids peroxidation, prevent and decrease the free radicals formation and inhibits cartilage destruction due to this.

Drugs for microcirculation improvement are used in a complex treatment of osteoarthritis during the remission stage for decreasing the venous congestion, improvement of hemocirculation in the synovial layer and subchondral areas, trophics optimization of the articular cartilage, other articular and extraarticular structures. The following medications are currently used for this purpose: kurantil, trental, nicotinic acid, detralex, cyclo3fort and others.

Metabolic therapy is directed at the metabolic processes improvement in the articular cartilage, periarticular tissues and in the organism in general, To them refer drugs, that influence the bone tissue metabolism (osteotropic drugs); metabolism and functional condition of the muscular tissue (drugs, that normalize muscular tone), energetic state of tissues and organs, anabolism and catabolism activity. To this groups also refer the drugs for enzymotherapy, vitamin therapy, macro and microelements.

### III. INTRAARTICULAR TREATMENT

Local peri and intraarticular injection of drugs in osteoarthritis is directed in the first turn at the inhibition of the local inflammatory process intensity, and also prevention of degenerative, destructive process in the joint. Reaching of this purpose is realized by the intra and periarticular drugs injection, action of which is directed at the cellular membranes stabilizing, synthesis inhibition and activity of a series of proteolytic and biological active substances, that promotes the cartilage destruction, hemocirculation improvement, activity inhibition of the freeradical lipid peroxidation; immunologic correction, etc.

**Among drugs, that are more often used for periarticular and intraarticular injection is osteoarthritis, the following should be mentioned:**

- local analgesics (novocaine, lidocaine, bupivacaine, etc.);
- glucocorticoids (Diprosan, Flosteron, Depomedrol, etc.);
- Protease inhibitors (Contrycal, Gordox, Trasilol);
- chondroprotectors (complex drugs: Alflutop, hyaluronic acid drugs: Hyalualarthro, Sinocrom, Syngyal, etc.);
- synovial fluid substitute (Polyvinylpirrolidon, Noltrex, Synvix, etc.);
- antihomotoxic therapy drugs (Traumel C, Cell T, Discus compositum, etc.);
- antioxidant drugs (Orgotein).

It should be mentioned, that method of local intra and periarticular injection of pharmacological agents in osteoarthritis is used in complex with systemic pharmacological, nonpharmacological and operative treatment.

### IV. OPERATIVE TREATMENT

As the disease has progressing course, the issue of the possible operative treatment should be decided in early terms for preservation of the intact cartilage areas, providing the even load at all joint areas, congruency renovation, hemocirculation activation in subchondral areas and others. In patients with late disease stages (III–IV) an issue of prosthetic replacement or stabilizing (arthrodesis) operations performance.

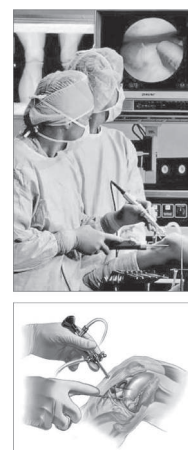
**Surgical interventions in Degenerative-dystrophic diseases of joints are represented by the following types:**

- Arthroscopic treatment of joints, lavage
- Corrective osteotomy
- Arthroplasty
- Arthrodesis

#### **Arthroscopic operative treatment of joints.**

Arthroscopy development originates from 20–30s years of the XX century. This method was initially used only as the experiment, but with the occurrence of the optic device in the 1931 year owing to the professor Kenji Takagi, that has a diameter of 4.0 mm, it became possible not only to examine the joint, but also to perform biopsy; the special device was in the set for this.

Arthroscopy method allows performing operative interventions on the joints with minimal injuries of the surrounding tissues (Figure 7). At the articular fissure projection (knee, shoulder, ankle, elbow, and other joints) several, usually twothree, small sections (punctures) are made. Thin optic device is introduced in one of them – arthroscope, that has a diameter from 2 to 5.5 mm, length of 12–14 cm, that is connected with a digital camera. Special probe or thin devices (manipulators) are introduced through another port. Surgeon controls the operation progress at the monitor, that provides image zooming in 6–8 times in comparison with the real size of all intraarticular structures. This method provides high accuracy of the manipulation in the joint and their carefulness, This type of surgery allows avoiding large sections and opening of the patients joint, that has the principal value for the enhanced recovery of the patient after the operation.



**Figure 7.** Arthroscopy of the knee performing

Arthroscopic operative treatment of the knee and elbow joints are most widespread nowadays. Arthroscopy of the elbow joint, wrist joints, hip, ankle joints have mainly diagnostic character, but indications and possibilities of the arthroscopic operation performance become wider with the arthroscopic technique development.

Arthroscopic surgery of osteoarthritis is characterized by the following possibilities. First of all, it gives a great diagnostic opportunity to determine the treatment tactics: for example, if the cartilage is absent in external and internal parts of the knee joint – its total arthroplasty is indicated, if one of the part is preserved, then the indications for correction osteotomy or monocondylar arthroplasty are determined, that are performed immediately after arthroscopic operation or later.

Chondromatous and other bodies, free as well as a(ached a removed during arthroscopy. Injured menisci are removed, as well as hypertrophic and fibrous changed parts of the synovial membrane and lipid body in the anterior part of the joint, which usually impacts the complete extension. Sometimes these allow removing flexion contracture. Lateral release of the patella is performed with use of the electric device. Contracture is sometimes conditioned by the osseous deformations of the femoral condyles; arthroplasty is not performed in such cases, because it can lead to the articular surfaces trauma and will not reach the clinical effect ion osseous outgrowths or osteophytes presence in the intercondylar area, which causes discomfort, their removal is indicated. If the area of damaged cartilage is found, which is characterized by its softening, gar netting and irregularity; its polishing is performed using shaver. Mentioned above operations in "arthroscopic" literature are called arthrolysis, debridement or abrasive arthroplasty.

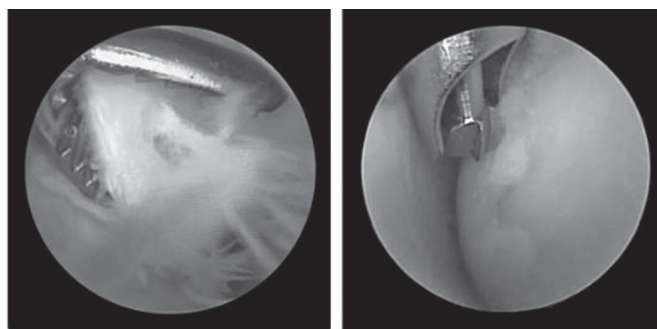
Performing of arthroscopic operations gives a possibility to use efficiently correction osteotomies, as the joint examination allows assessing condition and localization of the preserved cartilage areas and accurately determined the indications presence and directly the osteotomy type.

Synovectomy is indicated in pigmentovillous synovitis and in cases of productive inflammatory processes, with chondromatosis in particular. In chronic recurrent synovitis, that are not treated conservatively, total synovectomy is recommended to be performed, complete radicalism is necessary for such situations, because a high level of recurrence exists. Anterior as well as posterior approaches are recommended if necessary for reaching the radicalism of the arthroscopic intervention.

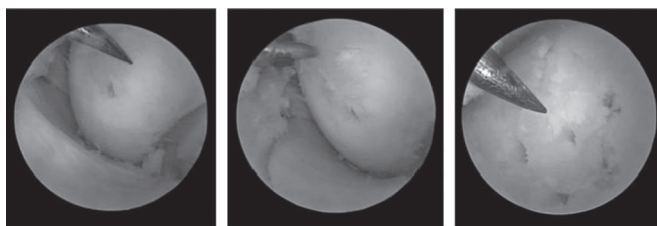
For the prevention of the articular cartilage destruction progressing in osteoarthritis in arthroscopy

performance, the series of operative methods are used, that are directed at the replacement of the articular cartilage defect with the regenerants. Regenerants properties depend directly on the operative intervention technique. Regenerant, that has properties close to the hyaline cartilage, is formed in certain conditions of the operative treatment performance. Among these methods the following are used more often: abrasive chondroplasty, microfractures and forage of the articular cartilage defect bottom, osteoarticular autografting (OATS, mosaicplasty) and osteoarticular allografting.

*Abrasive chondroplasty* (Fig. 8). Advantages of its use are the technical simplicity and satisfactory clinical results of the treatment. This technique the use excludes use of other methods. To disadvantages refer, that defects are filled exclusively with the fibrocartilage. P. Angermann with colleagues assessed clinical results of the treatment during 6 years. All patients reported about the positive effect of the abrasive chondroplasty: 69 % assessed the knee joint condition as good or verygood, 77 % assessed the treatment effect as constantly positive. Technique of the arthroscopy performance with abrasive chondroplasty provides for the damaged cartilage parts removal and careful polishing of defect margins.



**Figure 8.** Technique of the abrasive chondroplasty performance (damaged cartilage fragments removal and margins polishing)



**Figure 9.** Arthroscopic microfracturing

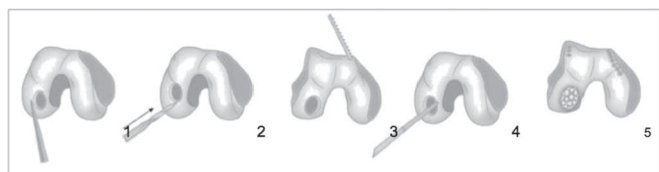
Microfracturing – microfractures, forage of the articular cartilage defect bottom performance (Fig. 9). Advantages of this method are economic efficiency, technical simplicity, good clinical results of the treatment. Use of this method excludes using of other methods.

Method of arthroscopic microfracturing provides for primary processing (polishing) of the cartilage defect. Bottom of the defect should be processed for the

removing of the calcified cartilage layer. Then 3–4 perforations at the square centimeter are performed, that is performed from the periphery to the center. Early movements in the knee joint with limited for 6 weeks loads are indicated to the patient in the postoperational period.

Osteoarticular autografting (OAS, mosaicplasty). Osteoarticular autografting was initially described by H. Wagner in 1964. Technique of this method under the arthroscopic control was developed by Y. Matsusue in 1993. Following developments of the arthroscopic technique and devices were introduced in studies of L. Hangody and V. Bobic.

Mosaic chondroplasty – single stage procedure, that provide relatively fast recovery, can be an alternative in the treatment of small and medium defects (Fig. 10). It is recommended for the treatment of cartilaginous and osseocartilaginous defects of the knee joint as the safe procedure for recovery of the injured articular surface and providing its properties similar to the hyaline cartilage. This method preserves integrity and function of the injured joint, providing promising results in plane of prevention of the early osteoarthritis development in young people. Good clinical results, low expenses on treatment and short recovery time are main advantages of this method.



**Figure 10.** Scheme of mosaic chondroplasty technique: 1 – polishing of defect margins; 2 – channels forming for grafts; 3 – taking of osteoarticular grafts, 4 – moving of osteoarticular blocks, 5 – view after the grafting

Also to advantages refer potentially high survival degree of grafted chondrocytes: recreated tissues is similar by its characteristics to hyaline cartilage. Disadvantages are defect formation of the grafted areas, treatment result dependence on the surgical technique, the limited size of the work defects, the risk of the surface congruency trauma with the bone cartilage block in its incorrect placement. Operation technique consists in the grafting of cylindrical osteoarticular blocks, taken from the articular surface, that does not bear loads (usually from the anterior surface of the lateral condyle).

These blocks are mechanically fixed like a mosaic, in order to cover 80–90 % of the defect. This method is recommended for cartilage defects with the area from 1.5 to 3.0 square centimeters.

Corrective osteotomy. Purpose of osteotomy is the renovation of the limb axis and joint biomechanics, load normalization at articular surfaces, renovation of

the supporting ability and due to these, decreasing the dystrophic process progression in the joint.

Till recent times, correction osteotomy has the leading role in the surgical treatment of osteoarthritis even at the 3rd stage of the disease. Histological facts of the cartilage tissue formation in the area of cartilage defects, at the area of femoral and tibial condyles in particular after the sustained operation, indicates the possibility of osteoarthritis progressing slowdown. Positive influence of osteotomy is also confirmed by the facts of intraosseous pressure decrease and microcirculation renovation.

Two main types of osteotomies are defined: correction and medicinal (osteotomia medicata). Among correction osteotomy, the following are defined: angular (wedged and osteoplastic) and arched. V-shaped osteotomy is their combination. There are a big amount of surgical approaches in this intervention and fixation methods.

Correcting osteotomies of the knee, hip and ankle joints are performed more often in osteoarthritis. Indication to performance of such an operation are I, II and even the beginning of third osteoarthritis stage on the background of the biomechanical joint axis impairment. Use of the correctional osteotomies is especially actual in presence of the mentioned above indications in young patients. This is conditioned by the fact, that endoprosthetic replacement is limited in young age, because it connected with risk of series of complications and the possible necessity of further multiple revision of endoprosthesis.

Knee joint osteoarthritis is mainly (up to 90 %) accompanied by varus deformation of the limb axis, so antivarus and valgus osteotomies of the femoral and tibial bones. Supracondylar Vshape antivarus osteotomy is performed for the correction of the varus deformation that is localized at the level of the femoral bone. Antivarus osteotomies of the femoral bone is not used frequently. Antivalgus osteotomy of the femoral or tibial bones are biomechanically grounded in the knee joint osteoarthritis with valgus deformation. Antivalgus osteotomy of the femoral bone is more often performed at the deformation localization at the hip, which is indicated in valgus deformations less than 25°, movements range in the joint of up to 90° and absence of significant flexion contracture of the knee joint. The main osteotomy type is V-shaped. Antivalgus osteotomy of the tibial bone is performed in the deformation localization at the shin. V-shaped osteotomy is more common. Indications to the osteotomy performance are valgus deformation less than 15°, medial incline of the articular surface up to 12°.

For good postoperational result, osteotomy should be combined with other interventions, that are directed to the liquidation of other morphological (for

example, cartilage or meniscus injury) or functional (for example, instability) impairments in the joint.

Arthroplasty – surgical operation, during which destructed by the disease parts of the joint are replaced by artificial, that reduplicate the normal joint form and reproduce the joint function. Replacement of the affected parts of the joint with new leads to the complete pain elimination or its significant decreasing and recovery of the mobile limb function with preservation of its support ability. Endoprosthetic replacement is often the only method, that can recover lost joint mobility and eliminate pain in it. In one to two months after endoprosthetic joint replacement patients can return to active life.

Modern endoprosthesis have long exploitation period and can serve during 15–20 years, in some endoprosthesis parts wear they can be replaced. Around one billion of hip and more than half of a billion of knee are replaced now each year. Endoprosthesis for shoulder, elbow, ankle, fingers interphalangeal joints also exist and are used.

Construction of endoprosthesis underwent significant changes during its 30 year history. Modern endoprosthesis consists of high strength and bioinert metallic and polymeric parts (sometimes ceramics), the form of which reduplicate the joint form, where the endoprosthesis should be placed.

In healthy human joint friction occurs between articular cartilage. In artificial joints rubbing surfaces are often made from:

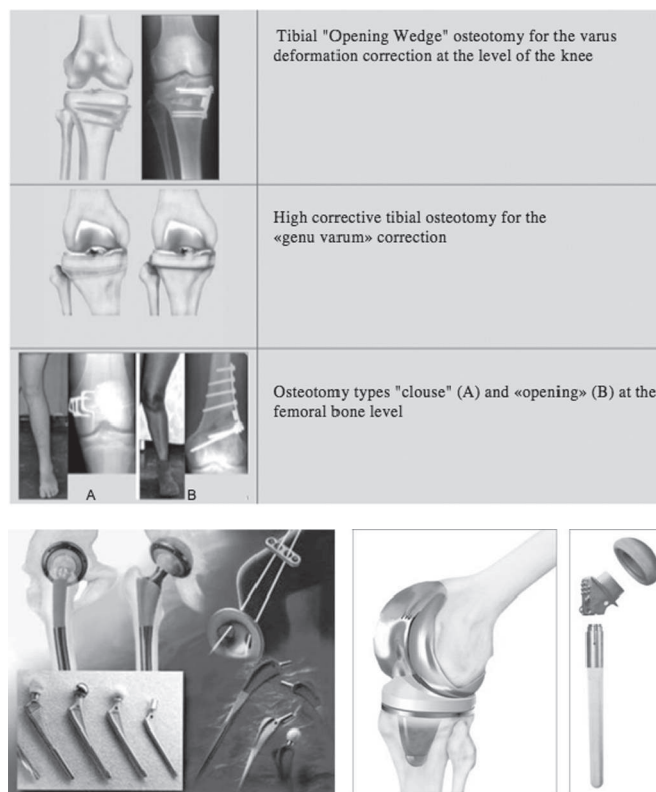
- metallic compositions and high strength polymer, which is called high density polyethylene (rubbing couple "metalplastic");
- ceramics (rubbing couple "ceramicsceramics");
- metallic compositions (rubbing couple "metal-metal").

The most widespread rubbing couple is "metalplastic". Such combination of materials provides the long joint functioning, but has disadvantages: plastic wear. Plastic microparticles, get to the surrounding tissues and promotes loosening of endoprosthesis components. This leads to the necessity of new operation – artificial joint replacement.

Rubbing couple "ceramics – ceramics" is void of these disadvantages, but has its own: insufficient mechanical strength and complexity of production. That's why these endoprostheses are used significantly rarer.

In rubbing couple "metalmetal" high strength is combined with minimal wear, that guarantees the greatest service period of such endoprosthesis (up to 20 years and more). Metal type, from which the prosthesis is made, is the most significant, because metal microparticles in prosthesis wear can provide negative impact, when getting to the organism.

There are two types of endoprosthesis fixation: cemented and uncemented. In the first case, joint components are fixed to bones with polymethylmethacrylate (PMMA), that is called bone cement. In the second case, ceramic is sprayed on the endoprosthesis surface (more often hydroxyapatite), to which adhere surrounding bones. Both fixation methods provide reliable adherence of endoprosthesis. But it is considered that uncemented endoprosthesis pass better to young physically active patient with strong bone tissue, whereas cemented pass better to elderly people, that have osteoporotic changes in bones. A lot of other factors also impact the choice of the endoprosthesis, so only doctor can choose endoprosthesis type correctly (Fig. 11).



**Figure 11.** Construction of the hip, knee and shoulder endoprosthesis

Endoprosthesis, as well as any mechanical construction, has a predisposition to wear. Service period depends on the load in some extent, that occurs during the exploitation period. It is obvious, that the younger patient is and the more active way of his life, the more intensive will be the endoprosthesis wear. And in the opposite, in elderly patients, when physical activity decreases, exploitation term of the endoprosthesis increase.

Usually, if the doctors recommendations are respected, then more than 95 % of endoprosthesis function normally during 15 years, in some cases – more than 20 years. After this period the possibility of mechanical destruction or endoprosthesis loosening in

the bone significantly increase. Usually, it manifests with the articular pain. Repeated endoprosthesis replacement is needed in such case (as called revision), when the instable endoprosthesis is replaced with new.

Somelderly patients can avoid repeated revision endoprosthesis replacement. Necessity of the revision endoprosthesis replacement is practically unavoidable in middle age and especially in young patients, that should be remembered in individual treatment methods selection.

#### Indications for endoprosthetic joint replacement:

- severe forms of degenerative-dystrophic and inflammatory diseases of the joints;
- posttraumatic, dysplastic osteoarthritis of III–IV stage;
- joints affection in StrumpellMari disease, rheumatoid, psoriatic and other nonspecific arthritis;
- false joint of the femoral, humeral necks;
- irregularly healed fractures with the joint function impairment;
- bone tumors.

Following accompanying diseases can be referred to contraindications to such types of operative interventions:

- acute and chronic osteomyelitis;
- tuberculosis of bones and joints;
- severe cardiovascular diseases;
- psychoneurological diseases.

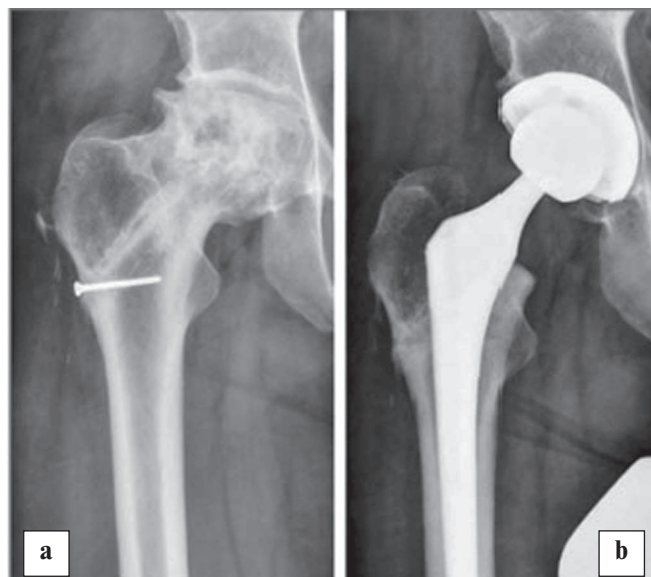
Patients age is not a contraindication in the absence of severe accompanying diseases.

**Hip arthroplasty.** The biggest part of endoprosthetic replacement operations is related to the hip joint. Destroyed by the disease joint is removed irreversibly from the organism. Endoprosthesis is implanted at its place after this (Fig. 12).

**Indications to the hip joint endoprosthetic replacement:**

- hip joint osteoarthritis (coxarthrosis) of III, IV stages;
- Strumpell-Mari disease with main affection of the hip joint;
- hip joints affection in psoriasis;
- femoral head avascular necrosis;
- ununited fractures and false joints after femoral head fractures;
- fresh fractures of the acetabulum and femoral neck in patients older than 65 years;
- rheumatoid arthritis with the hip joints affection;
- tumors of the femoral head, femoral neck in patients of any age.

Durable capsule around the endoprosthesis is formed during 4–6 weeks after the operation, that does not allow the endoprosthesis dislocation.



**Figure 12.** Patient A., 1964. X-rays of the affected hip joint before (A) and after (B) the arthroplasty.

#### Risk of endoprosthesis dislocation.

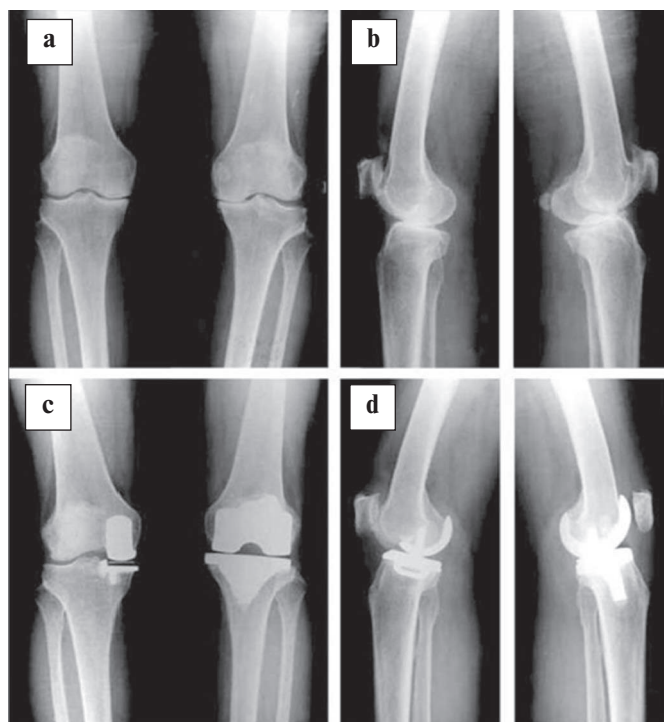
For this complication prevention patient should follow some limitations during 6 weeks after operation: do not flex operated leg in the hip joint more than 90 degrees; lay in bed only on the back or health side with the pillow between legs, do not rotate the operated leg inside. These limitations are usually taken off after 6 weeks and the patient returns to normal life.

Movements in the joint are started on the first day after the operation. Respiratory gymnastics, exercise therapy for limb muscles with isometric exercises are indicated on the second day. Flexion movements in the hip joint with small amplitude are performed. Walking with crutches is recommended on the third day after the operation. Special attention is paid to the leg positioning during walking for the luxation prevention. Patient should limit loads on the joint after discharge (walking with crutches) up to 6–8 weeks after the operation. Then it is recommended to walk with the stick. It is recommended to walk with crutches for up to 3 months in uncemented joint fixation. It is recommended to limit lifting of significant weight, running, squatting after the hip joint endoprosthetic replacement.

**Knee arthroplastics.** Knee joint endoprosthetic replacement is technically more complicated operation than the hip joint endoprosthetic replacement. During operation destroyed parts of the knee joint are removed with the use of special devices, lower limb axis is recovered.

Then endoprosthesis is implanted, that reduplicate the normal knee joint form (Fig. 13). Endoprosthesis components are fixed with the bone cement. Insertion of a special polymer material is placed between them in the articular surface. Function of this insertion – to improve slipping of the articular surfaces. It is also

a damper between the endoprosthesis surfaces. Then the plastic regulation of the articular ligaments tension is performed. Own articular ligamentous apparatus is usually managed to be preserved with correction of the ligaments tension. In case, when own patients ligaments are injured or degenerative changes, then their prosthetic replacement is performed.



**Figure 13.** Patient M., 1947. Radiographs of the affected knee before (A,B) and after (C, D) arthroplasty

#### Indications for the knee arthroplastics:

- knee osteoarthritis of III, IV stage;
- severe lesions after intraarticular fractures;
- persistent lesions after knee osteonecrosis;
- severe joint affections in rheumatoid arthritis, podagra, psoriasis, Strumpell-Mari diseases;
- tumors at the knee area.

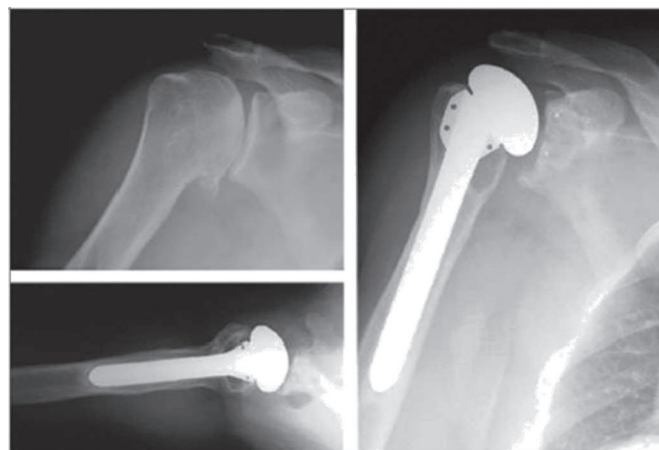
Main differences of the recovery period in knee joint endoprosthesis replacement are early regular movements in the operated joint. This procedure starts in the first days after the endoprosthesis replacement and lasts for at least 3–4 weeks after it. Respiratory gymnastics, exercise therapy for the lower limb muscles with isometric exercises are indicated on the second day. Slight flexion movements in the replaced joint are performed. Patient can sit up in the bed. Walking with crutches is recommended on the third day. Patient should limit the load on the joint after the discharge (walking with crutches) for 4–6 weeks after the operative treatment depending on the endoprosthesis features. Then walking with stick is recommended for up to three months, Full loads on the joint are possible after this.

**Shoulder arthroplasty.** Shoulder joint endoprosthesis replacement is effective and often the only method of the lost limb function recovery. Two types of the shoulder joint endoprosthesis are suggested nowadays – humeral head endoprosthesis and total endoprosthesis, ie replacement of both joint components. Almost all movements, that are typical for the shoulder joint can be performed in the artificial joint. Endoprosthesis is chosen individually, taking into account patient's features, character and stage of the pathologic process, etc.

Indications for the shoulder joint endoprosthesis replacement:

- shoulder osteoarthritis of III, IV stage;
- consequences of the glenoid cavity of scapula and/or humeral head;
- congenital dysplasia of the shoulder joint;
- tumors of the proximal part of the humeral bone.

Healthy bone tissue is tried to be maximally preserved in the shoulder joint endoprosthesis replacement. Endoprosthesis components, that reduplicate the articular surfaces form are placed instead of articular surfaces destroyed with the pathologic process, that are removed during operation. Implants are fixed to the bone with bone cement (Fig. 14).



**Figure 14.** X-rays of affected shoulder joints before and after the arthroplasty

Movements in the joint are started on the first day after the operation. Respiratory gymnastics, exercise therapy for the limb muscles with isometric exercises are indicated on the second day. Slight movements in all directions are performed in the replaced joint. Patient should limit loads on the joint for the terms, that are indicated by a doctor, after discharge. Then dosed loads are recommended with the transition to the full load on the joint.

#### Rehabilitation after the arthroplasty

Patient usually starts to walk with the help of special frame («go-cart») on the next day after the en-

doprosthesis joint replacement. Patients walk only in the nit during first 2–3 days and then gradually increases the walking duration. Approximately in 5–7 days crunches are recommended instead of a gocart. Patient usually stay in the department for 10–14 days after the endoprosthesis replacement. Postoperational wounds heal in this period and patient is discharged home. Till the dishcharge patient should walk confidently with crunches.

Patient continues to do exercises, which he/she was educated by the doctor or physical therapist, every day at home. Most of the patient also continue to intake anticoagulants, because the risk of thrombosis is preserved during 3–4 weeks after the operation.

Possibility to walk with the full load on the lower limb is allowed in 1–1.5 months after the cemented endoprosthesis fixation and in 2–2.5 months after the uncemented. General terms of rehabilitation depend on the patients age and features of the operative intervention. It usually consists around 3–4 months. Most of the patients return to the normal life during this period.

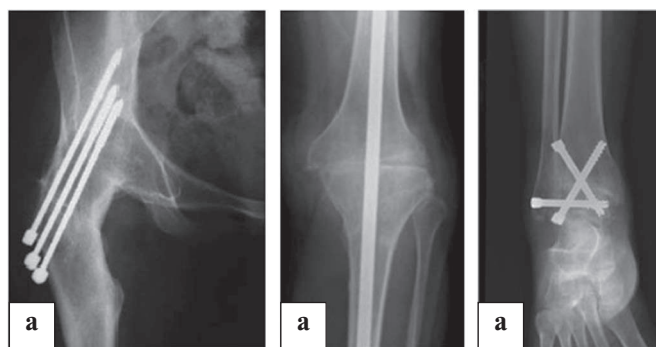
It should be mentioned, that physiotherapeutical procedures after endoprosthesis replacement are contraindicated in the area of operative treatment: electrophoresis, magnet, UHF, heating, pelotherapy, etc. These methods are not able to improve the artificial joint functioning and should not be used in any term after the operation.

**Arthrodesis.** Arthrodesis – surgical operation, that consists in the bones fixation in the joint and leads to the complete immobilization of this joint (ankylosis). Arthrodesis is performed in the case, when the joint is rather painful, instable, significantly deformed or is affected by the chronic infectious disease, and if it is impossible to perform athroplastics or endoprosthesis replacement due to some reasons.

This type of operative treatment is the oldest from a historical point of view. A lot of experience of the arthrodesis use and significant amount of different methods of its performance is accumulated nowadays. Different fixation methods are used in arthrodesis operations: plates with screws, rods, pins, stirrups, osseous auto and allografts, intramedullary rod, external fixation devices, plaster casts. Arthrodesis of any joint can be performed if necessary. some advantage of this type of the operative treatment is that, it does not require significant economic and technical resources, but the most significant aspect of the successful arthrodesis performance is surgeon qualification, that performs this operation.

Arthrodesis provides for preservation or recovery of the limb supporting ability in the affected joint or significant decrease or elimination of the pain syn-

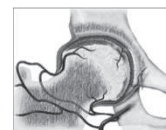
drome, but the function of this joint is lost irreversibly. So currently this type of operative treatment is last and forced intervention and indications for its performance should be maximally grounded and justified. Arthrodesis of the ankle, rare knee, hip joints are performed for osteoarthritis treatment (Fig. 15).



**Figure 15.** Radiologic pattern of the hip arthrodesis with screws (A), knee with intramedullar nail (B), ankle with screws (C)

### Avascular necrosis

Aseptic necrosis (avascular necrosis – according to ICD10) – form of the degenerative-dystrophic affection of joints, when the subchondral bones tissue is initially affected with the avascular necrosis focus formation with the following involvement into the degenerative-dystrophic process of all joint elements – articular cartilage, synovial membrane, capsule, periarticular muscles and others (Fig. 16.)



**Figure 16.** Avascular necrosis.

Avascular necrosis of the femoral head, that is the consequence of the blood flow impairment and bone marrow elements necrosis in the femoral head. Disease usually develops in the background of corticosteroids use, joint trauma, alcohol abuse, pancreatitis, sickle cell anemia, ionizing radiation, etc. In absence of the clear reason, the diagnosis of idiopathic femoral head avascular necrosis is made, but the number of such diagnosis recently decrease due to the widening of medicine diagnostic capabilities. Radiologic diagnostics allows to mainly diagnose late stages of the disease in femoral head fragmentation or deformation. Orthopedic prognosis is unfavorable in most of the cases – deforming osteoarthritis of the hip joint, when arthroplasty, arthrodesis or correction osteotomies are often used as methods of choice in surgical treatment. Early diagnostics is possible only using (computer tomography) and magnetic resonance imaging (MRI) of the hip joint and gives hope for favourable consequences of the diseases with using of conservative or surgical treatment.

### Pathogenesis

Two points of view are widely represented in the literature: traumatic and vascular. It is known, that

avascular necrosis of the bone tissue can be caused by different reasons: impairment of the arteries integrity by their compression or rotation, embolism, continuous arterial spasm, venous stasis and other mechanical impacts. AVN of the femoral head due to the trauma (luxation, metaphyseal zone fracture) or surgical intervention are studied completely, and its occurrence mechanism is conditioned by the arterial and venous blood flow area impairment.

Issue of the pathogenesis of the as called non-traumatic avascular necrosis of the femoral head. Gracianskiy V.P. in the 1955 year has shown, that due to the microtrauma, joint overload and other unfavorable factors, processes of the "overstrain" occurs in the bone tissue. Impulses from the focus go to the cerebral cortex and cause corresponding return signals, that provoke the vascular spasm and blood and lymph congestion, metabolism, impairment, accumulation of the catabolic products in the bone. These lead to changes of physicochemical and structural dynamic properties of the bone, leads to slow destruction of the bone trabeculae, further complication of the local blood flow and progress progressing. his theory of the "chronic microtrauma" is currently supported by many scientists.

J. Welfling in 1967 made a conclusion, that all femoral head necrosis have the vascular genesis, occur due to the arterial embolism. Impairments of the venous circulation in the affected femoral diaphysis in patients with avascular necrosis allowed some authors (Shumada IV., 1990; Ayrolles Chr., 1962) to suggest, that the initial change is the venous insufficiency with following process transmitting to the arterial system.

Blood flow impairment manifests with the increased intraosseous pressure, enhancement of the ischemic disorders. According to Arlet J. and Float opinion, that one of the reasons of the femoral head avascular necrosis is an intraosseous pressure increase in the proximal femoral end.

Experimental studies, compared to the histologic investigation of the operation material (Stecula V.I., Moroz N.F., 1988), showed, that osteonecrosis foci are formed in 3–5 days in the blood flow impairment in the epiphyseal area, that are replaced with osteogenic tissue during the revascularization process and during the differentiation process are transformed into the normal bone tissue. In unfavorable conditions (joint overload), perifocal necrosis area develops at the border of the osteonecrosis focus. and then on the background of secondary circulation disorders the perifocal osteosclerosis zone is formed. Bone trabeculae are without osteocytes during this period, space between them are filled by protein masses, osteonecrosis focus is limited by the fibrous tissue.

Common cause, that unites all nontraumatic cases of osteonecrosis – osteopenia. Fractures occur in

the anterosuperior part of the femoral head, where the most part of pressure is applied to, due to the "weariness" of the osteopenic trabeculae. Accumulation of the fractured trabeculae more probably can lead to the arterial blood flow closure and cause bone necrosis.

Osteoporosis combination, mechanical pressure, frequent use of the osteo and chondroprotective medications, and also inflammation lead to the subchondral trabecular fracture and further destructive and degenerative processes in the hip joint. Expressed movement function impairment is observed in all patients, caused by the pain syndrome, movements limitation in the joint, muscular hypertrophy and their function. impairment. laboratory investigation in patient indicates the capillary blood flow impairment, intraosseous pressure increase, hypercoagulation syndrome, and also vegetotrophic regulation in these patients.

### *AVN of the femoral head*

**The following are defined according to etiology:**

#### **I. Primary**

- idiopathic

#### **II. Secondary:**

- neuroendocrine;
- posttraumatic;
- postarthritic;
- dysmetabolic;
- other

Choose of the treatment method in AVN of the femoral head depends in the first turn on the diseases stage on the moment of the diagnosis formulation.

Different classifications are currently suggested by the orthopedics, in which different stages are defined. Reinber S.A., 1964, and Ritz I.A., 1981, developed 5-stage classification. Mankin H. J., 1992, define 6 stages. Serre H. and Simon L. define only three stages of the process, Korzh et al adhere to the same classification (1982).

Most acknowledged classification among the Ukrainian specialists is Kulish-Filippenko classification (1986), where 4 clinicoradiologic stages of the AVN of the femoral head are defined (Fig. 17):

- 1 stage – initial (radiologic lighting, sequestration). It is clinically the stage of the initial manifestations, where nonintensive pain syndrome can be observed, that occur during loads. The only limitation of the internal rotation is defined. Impairment of the femoral head bone structure like the osteosclerosis and osteoporosis foci can be defined.
- 2 stage – stage of the impressive fracture (impression fracture and demarcation of the avascular necrosis focus). Transition into the second stage is accompanied by the intensive pain occurrence due to the capital impression fracture. Then pain



becomes less intensive and its intensity increase during activities. External and internal rotations, abduction are significantly limited. Radiologic signs: articular space widening; femoral head – irregular contours due to the necrotic focus impression in the anterosuperior part, which is more loaded, necrotic focus is clearly differentiated due to the increased radiologic density; osteolysis and reactive sclerosis zones are located around it.

- 3 stage – necrotic focus punching in the articular area with the secondary osteoarthritis development, or the stage of secondary osteoarthritis. Pain is intensive, constant, its intensity decrease during rest. Movements are limited in all three dimensions, adduction-flexion contracture occurs. Patients are forced to use additional support during walking (crutches, orthopedic stick). Marginal osseous overgrowth, irregular narrowing of the articular space, changes of the capital contours are determined during X-ray investigation. Necrotic focus is clearly determined, is surrounded by the osteolysis and sclerosis zones.
- 4 stage – final stage. Intensive, constant pain, which can even increase at night, is typical for this stage. Movements in the joint are absent or significantly limited, expressed adduction flexion contracture.

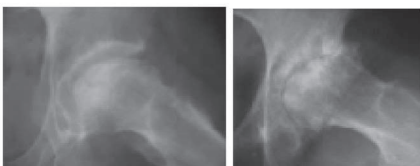
I stage



II stage



III stage



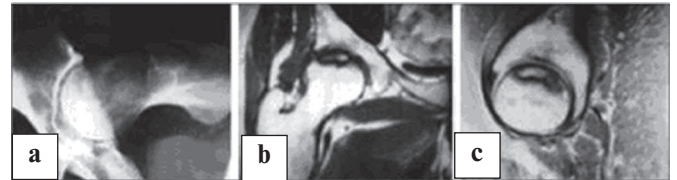
IV stage



**Figure 17.** Clinical and radiological stages of the AVN of the femoral head.

Division into stages is relative, because the process develops dynamically, one stage transit into another and has no distinctive limits. But such a division is necessary from the practical point of view. Each stage characterizes the stage and depth of the pathologic process, indicates in which direction it develops, ie provides the possibility to predict further process development. Question of the treatment tactics and methods depends on this. Plain X-ray has relative diagnostic value at the stage of the femoral head destruction (especially taking into account the image quality). Information value of the radiologic investigation increases with use of the special positioning according to Launstein.

Clinical manifestations and special method of investigation have an important value in the initial disease stages; magnetic resonance imaging and computed tomography (CT), allows to determine the avascular necrosis presence in the epiphysis, its sizes, localization and spread. MRI sensitivity reaches 90–100 % in the initial disease stages (Fig. 18).



**Figure 18.** AVN of the femoral head, I stage.

A - Radiograph according to Launstein

B - MRI: frontal plane

C - MRI: horizontal plane

### Treatment

AVN of the femoral head treatment should be complex and differentiated depending on the process stages and clinical manifestations, prognosis depends on the early diseases diagnostics, osteonecrosis spread and proper treatment tactics. Possibility of the bilateral hip joints affection should be taken into account in more than 50 % of patients.

In spite of success of the surgical treatment: flexion-antvalgus osteotomy, which prevents the femoral head collapse in 95 % of cases; subchondral autoplastics of the femoral head, arthroplasty, and sometimes arthrodesis – see in osteoarthritis treatment section). AVN of the femoral head is the problem of the outpatient orthopedy. Disease duration (from 1.5 to 2 years) require a lot of patience from doctor and patient and performance of the necessary medical rehabilitation complex adjusted for the process stage and orthopedic situation. Complete recovery of the joint usually does not occur. But rather acceptable disease outcome can be reached in most of the cases: prevention of the collateral joint affection, decrease of destructive processes in the femoral head and sec-

ondary coxarthrosis; pathological hip positioning in the position of flexion, adduction or extensive rotation; minimal movement range limitation in the hip joint; good functional condition of muscles and mildly expressed pain syndrome.

**Orthopedic regime.** Special importance has the adherence to the orthopedic regime. According to the university biomechanical laboratory at the Berlin orthopedic hospital, and also according to IETC (Russia, Moscow), the regime of the maximal joint unloading is not reasonable and not justified (crunches) for the continuous period of time and adherence to the bed rest at the initial period of the disease. Studies that were conducted in the IETC biomechanical laboratory, proved, that walking with crunches for more than 2–3 months leads to the progressive hypotrophy and function impairment of the main muscular groups, persistent pain syndrome and vegetotrophic impairments formation, impairment of the motor stereotype. These complicates orthopedic status of patients and lead to severe impairment of the lower limbs function, that in its turn complicate the AVN of the femoral head course, provokes pathologic process development in the collateral joint, causes problems in adjacent joints and spine. Patient's walking should not be limited, it is enough to exclude inertial loads on the joint (running, jumping, lifting weight); orthopedic stick should be used only during first 3–4 weeks after the pain syndrome onset and during walking on the long distances. In the opposite, dosed walking (15–20 minutes) in the medium pace, climbing the stairs, riding the exercise bicycle, swimming facilitates course and reduce term of the first disease stages. Measures directed at the excessive body weight reduce are also necessary.

There are interesting studies, that were conducted in the university biomechanical laboratory at the Berlin orthopedic hospital in patients after the bilateral endoprosthetic replacement of hip joints using the telemetric probe built in the prosthesis neck. Loads on the joint, force moments and temperature were studied during 18 months after the endoprosthetic replacement in different walking modes. In walking with impaired locomotion, stumbling, loads increased in 46 times. Additional support does not solve the problem of the joint unloading, Support with 2 crunches reliably unload the joint during first 2–3 weeks (25–60 %), in 1 month unloading effect decreases to 10–15 %.

**Walking up/down the stairs.** According to the Bergmann et al., 1995 data, walking up the stairs with support in the handrail unloads the joint significantly more than walking with support on crunches. Big load was determined during walking down the stairs.

Stumbling leads to the significantly bigger load on the joint (720–870 %), then in other studied activities.

The most optimal load on the joint is on the exercise bicycle: medium loads on the joint are always lower than 60 % of the normal load, when the combined muscular force was not lower than 80 % of the normal value (during walking). Carrying weight of 10 % of the body weight in one hand leads to the load increase in the collateral joint by 22 % (in decreasing on the same side by 3 %), and with equal weight in both hands – by 8.8 %.

Besides this, temperature change in the joint was studied. Articular temperature starts to grow up in walking for more than 15 minutes, and in 45 minutes articular temperature reaches 45 °C.

**Medicamentous treatment.** Vascular drugs use is pathogenetically grounded at the initial disease stages for the ischemic changes reducing in the femoral head, normalization of the blood rheological properties.

Decompression of the metaepiphyseal area is undoubtedly pathogenetically grounded and rather effective method for early decompression of the femoral metaepiphyseal area. Elimination of the increased intraosseous pressure can "break" ischemic cycle of the diseases. Success of this method use varies from 40 to 90 % (Hopson CN, 1998). According to Bluemke D. A., Petri M., Zerhouni . A. (1995) data, femoral head collapse was prevented in 25 % of patients. here are many methods of influence on the bone issue – from osteotomy to cryodestruction of the spongy substance. IETC actually recommends lowtraumatic and available in the outpatient condition method of metaepiphyseal zone forage with the bone decompression and blockades conducting. Unlike the blockades, suggested by Poliakov VA (1975), autoblood is used as prolongator, adding 2 % novocaine solution and vascular drugs in it (curantyl). in some cases (in patients with diabetes mellitus and nonclostridial cellulitis, and also in patients who sustained infection – gonorrhoea, chlamydiosis, etc), use of the intravenous antibiotics is appropriate.

Pain syndrome elimination at the initial stages of the AVN of the femoral head is an important issue in patient's treatment, allows to influence on multiple cause effect mechanisms of the disease. The most effective method of the pain elimination is the iliopsoas muscle blockade, which performance in combination with other medical measures allows to eliminate or decrease manifestations of the pain syndrome to minimal.

**Physiotherapeutical treatment.** EHF-therapy, laser therapy, magnet therapy, pelotherapy and balneotherapy are widely used in patients with AVN of the femoral head.

## CYSTIC RESTRUCTURING OF THE ARTICULAR BONES

Cystic restructuring – form of the degenerative-dystrophic articular lesions, when the subchondral bone tissue is initially affected by the formation of multiple or singular cysts in it, their merging, burst into the articular cavity and secondary osteoarthritis development with the following involvement into the degenerative-dystrophic process of all articular elements (articular cartilage, synovial membrane, capsule, periarticular muscles, etc.).

This pathology is observed significantly rarely than osteoarthritis and avascular necrosis. Degenerative-dystrophic changes with the cystic restructuring of the articular bone differ from osteoarthritis with the clarification presence in the articular bone ends. In the primary form, they are more often found in the small bones of the hand and are consequences of the continuous, permanent microtrauma. But the predisposition to such changes occurrence usually exists.

### Clinical picture

Pain intensity increases gradually, slowly, it increase during movements and after loads. But then in pathologic fractures with the burst of the cystic formations into the articular cavity, intraarticular hemorrhages, degenerative articular lesions, secondary osteoarthritis can occur. Normal or slightly reduced height of the radiological articular space, slightly visible marginal bone outgrowth and expressed cystic restructuring of the bones are determined and the X-ray film. Cysts are more often multiple, small, rarer – solitary and large. Areas of the cystic restructuring have not distinct borders during formation.

Formed cystic formations have clear contours due to the sclerotic endplates. In lasting physical load (overload) fracture of the cystic formation wall can occur, that is clearly observed at computed tomography. Described changes are primary manifestations, that are typical for the vibration disease, decompression disease, permanent microtrauma and overload, and also for some other professional diseases.

Secondary cystic restructuring is often observed as a consequence of the primary diseases: dysplastic and other types of osteoarthritis, after sustained arthritis, after endocrine diseases, in systemic metabolism impairment (dismetabolic), etc.

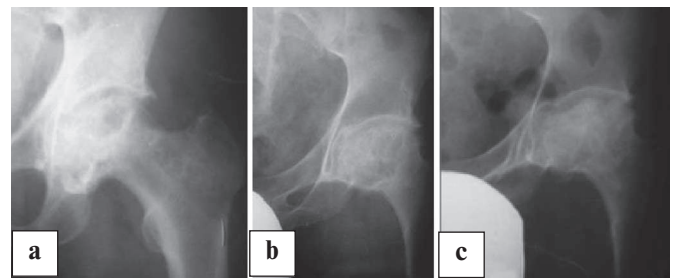
In the background of the cystic restructuring occurrence mechanics, lies impairment of hemocirculation in certain epiphysis part in the subchondral bone tissue, that leads to it hypoxia, dystrophy and lysis of the local subchondral part with cyst formation.

**The following clinico-radiological changes are determined during the clinical investigation depending on the pathologic process stage:**

- clinical symptoms are practically absent in the first stage, patients notice insignificant discomfort after the continuous physical load in the area of the affected joint. Overextension limitation in the joint is observed sometimes. Clarification with irregular contours is determined at X-ray, often with regular form – onset of the cyst formation;
- pain syndrome intensity and discomfort in the joint increase in the second stage, sometimes gait impairment, limping, insignificant movements limitations in the joint occur. Cyst is of the irregular form, but clearly limited at X-ray. Then cyst enlargement occur, sometimes its restructuring and its wall fracture is visible till it burst into the joint; articular surfaces and space are not changed;
- third stage is characterized by the presence of constant pain syndrome in the area of the affected joint, pain irradiation into the adjacent parts of the musculoskeletal system, movements range decrease, occurrence and increase of contractures, muscular hypotrophy. Then, with the development and progressing of secondary osteoarthritis, intensity of these symptoms increase. Flattening of the articular surface at the limited area is observed at X-ray (due to the bone wall punching), its structure is heterogenous due to the restructuring and reactive changes around the cyst, marginal bone outgrowth occur. As the consequence, deformation increases and secondary osteoarthritis deforms progresses.

Following radiologic stages of the cystic restructuring of the articular bones are defined (Fig. 19):

- I stage – occurrence of singular subchondral cysts – round form clarification in the epiphyseal bone area;
- II stage – widening, generalization of certain cysts;
- III stage – cysts burst into the articular cavity, articular surfaces deformation, secondary osteoarthritis deforms development.



**Figure 19.** Stages of the cystic restructuring of the articular bones. A – stage; B – II stage; C – III stage

**Treatment of the cystic restructuring of articular bones.** Treatment depends on the pathologic process form (primary or secondary), and also the stage of course. Taking into account the similarity of this form of the degeneratedistrophic articular lesion with avascular necrosis, which is characterized by the primary affection of the subchondral bone tissue and more often has vascular disorders in its genesis, treatment of these forms is similar. In early stages, it is directed at the unloading of the affected joint and use of drugs and surgical methods for the blood circulation improvement, decrease of the inflammatory process if it is present, pain syndrome decrease, osteotropic and chondroprotective drugs. At the final stages, when drugs are usually ineffective, endoprosthetic replacement of the affected joint is used, and in case of impossibility or contraindications presence – arthrodesis. General principles of the nonmedicamentous, medicamentous and surgical treatment (forage, correction osteotomy, endoprosthetic replacement, arthrodesis) stated in detail in previous sections.



## OSTEOCHONDROSIS

Osteochondrosis – disease of the spine, in the background of which lies the primary degenerative-dystrophic process in the intervertebral disk (IVD), with the following development of reactive and compensatory changes in intervertebral joints and ligamentous apparatus, and then in the bodies of adjacent vertebrae, and as the result total affection of all elements of the vertebral motor segment.

People after 45 years nowadays limit their activity due to the constant lowback and neck pain. Prevalence of the chronic lowback pain consists 26–32 % of the adult population. Among diseases with the working capacity loss in the adult population, more than 50 % consist diseases of the peripheral nervous system.

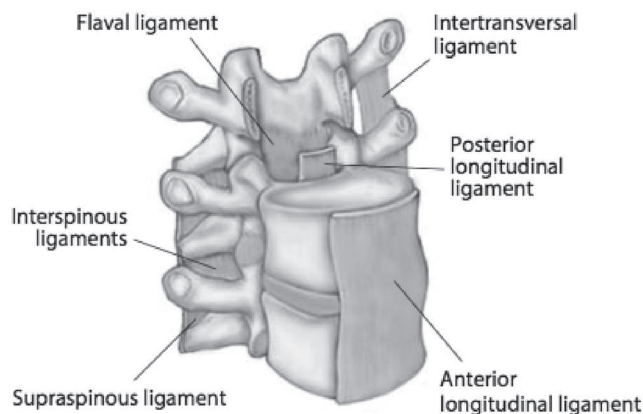
**Etiology.** According to modern conceptions, osteochondrosis refers to the polyetiological group of diseases. It is chronic systemic affection of the connective (cartilage) tissue, that develops on the background of its existing congenital or acquired functional (predominantly metabolic) insufficiency.

The most spread are involutinal and microtraumatic theories of the osteochondrosis development. According to the involutinal theory, aging and intervertebral dicks involution are the diseases background.

Microtraumatic theory assumes, that trauma of the spine can have etiological, as well as the provocative character in the diseases development. In osteochondrosis development importance is attached to the hereditary predisposed biochemical, hormonal, neuromuscular and immuno logic disorders, and also

spine development anomalies, which can impact the clinical course of the diseases. Certain role is given to the exogenic factors, supercooling in particular, which influence can provoke autoimmune disorders or reflex arterial spasm development, that supply nervous root and vertebral segments.

**Biomechanics and physiology of vertebral motor segment (VMS)** . The spinal column consists of vertebrae connected by IVD, massive ligamentous apparatus and paired zygapophysial joints. IVD together with adjacent vertebrae forms structuralfunctional unit – vertebralmotor segment (Fig. 20).



**Figure 20.** Schemes of the spine and intervertebral disk structures

Leading role in the VMS and spine in general plays IVD, which perform the following functions: vertebral bodies connection, vertebral bodies protection in overloads and trauma (amortization).

IVD consists of the nucleus pulposus, which is located in the center, and fibrous ring, that surrounds nucleus pulposus. Nucleus pulposus – ellipsoid avascular formation, that consists of the intercellular substance. Proteins, mucopolysaccharides (glucosaminoglycans), hyaluronic acid and water compound the intercellular matrix. Depolymerization of the polysaccharides occur with age, nucleus loses its elasticity.

Fibrous ring consists of very dense connective tissue interlacing plates, which are located around the nucleus pulposus. The fibrous ring incorporates vertebral bodies and IVD into the whole.

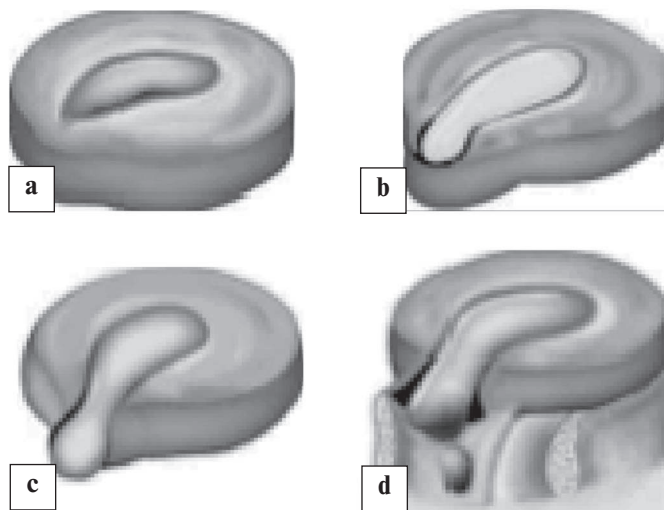
Movements of the spine are performed in the following planes: flexion and extension – in the sagittal, lateral body inclination – in the frontal, rotation – around the spine axis.

**Osteochondrosis pathogenesis.** depending on the process prevalence in the segment and segment function impairment, stable and unstable form of the diseases are defined.

**Staging of the dystrophicdegenerative changes in the IVD is important in the pathogenesis of osteochondrosis clinical manifestations. Three stages of osteo-**

**chondrosis are defined (Hvysiuk N.I., Prodan A.I.):**

- stage of the intradisk dislocation of the nucleus pulposus within the undamaged fibrous ring;
- protrusion of the nucleus pulposus in absence of the fibrous ring ruptures – disk protrusion (more often towards the posterior longitudinal ligament, where the fibrous ring structure is thinner);
- stage of the nucleus pulposus prolapse through the destructed fibrous ring – hernia (a disk hernia).



**Figure 21 A-C.** Stages of degenerative-dystrophic changes in intervertebral disks:

- A – initial disk degeneration;  
B – protrusion (prolapse) of the disk;  
C – disk hernia formation (disk sequestration)

Spine osteochondrosis development starts with the nucleus pulposus degeneration – poly saccharides depolymerization. Nucleus dehydrates and loses fibrous structure, elasticity and break up into separate fragments. Fibrous ring becomes fragile, radial ruptures and delamination occur at different distance. If the nucleus elasticity was preserved in some degree, weakened fibrous ring can not resist the nucleus tendency to widening during the axial load. Nuclear fragments penetrate through the fibrous ring fissure, stretch and stick out its external layers. Fibrous ring protrusion can be ventral, into the spongious substance of the vertebral body, through the hyaline membrane ruptures, forming Schmorl's nodules or hernia, and also towards the spinal canal, causing compression of the neurovascular formations. Mentioned structural changes are typical for the stable osteochondrosis form (Fig. 21).

Process can spread through several ways in the final stage: substitution of the degenerative changed IVD elements with the connective tissue and fibrous ankylosis formation and immobilization of the vertebral motor segment; involuntary reduction and autolysis of the IVD tissues, that fall out into the spinal canal; cicatricial commissural process formation in the spinal canal around the IVD fragment; the diffuse af-

fection of the fibrous ring and instability occurrence in the VMS (unstable form of osteochondrosis). Spinal instability – is a clinicoradiological syndrome of osteochondrosis, which manifests with the functional inability of the spine, especially during static-dynamic loads, and is characterized by excessive pathologic mobility in the horizontal plane with the following dislocation of one vertebra according to another, that leads to its resistance decrease.

**Three stages of instability are defined (Hvysiuk N.I., Prodan A.I.):**

- I - stage – diskogenic, when pathologic process is diffusely spread only in disk tissues and pathologic mobility of the vertebra only in horizontal plane is observed;
- II stage – disko-arthrogenic, when zygapophysial joint of the spine, ligaments and muscles are also involved in the pathologic process besides the disk;
- III stage – disk-arthro-osteogenic, when all mentioned above structures and vertebral arc are affected with its resorption, anterior dislocation of the vertebra – degenerative spondylolisthesis.

Clinical symptoms of osteochondrosis usually manifest, when the pathologic process affects posterior parts of the fibrous ring and posterior longitudinal ligament, that are well supplied with the meningeal nerve endings, that consist from sympathetic and somatic fibers. Pain syndrome is especially expressed in a disk hernia, that cause compression of the nerve roots and (rare) spinal cord. Diskogenic (hernial) compression causes changes in the root. Three stages of changes in the root are defined: irritation (are characterized by paresthesia and pain), compression (sensitivity impairments occurs) and rupture or radicular paralysis (paresis or paralysis and anesthesia, pain syndrome disappear).

Vascular impairments in osteochondrosis occur due to the vasomotor innervation impairment and rarer due to the mechanical compression of the vessels. Visceral disorders are also conditioned by the irritation or dropout of the viscerosensitive or visceroefferent fibers. Presence of a big amount of sympathetic fibers in roots (especially thoracic) can be a reason of visceral pain or dyskinesia.

Mechanical stability of the VMS and all spine recover with years due to the marginal outgrowth (osteophytes), disk and capsule fibrosis, facet joint ankylosis, ligaments thickening. These changes finish the "degenerative cascade" in the spine and sometimes lead to the spontaneous decline of pain. But in the same time they can cause the spinal canal stenosis. Besides, osteophytes, that are directed towards the spinal canal can injure roots and cause persistent pain syndrome and neurologic disorders.

Clinical picture and diagnostics of osteochondrosis. Formation of the osteochondrosis clinical manifestation depends on the process localization, disease form (stable or unstable) and process stage, and also from the nervous, immune, endocrine, cardiovascular and muscular systems condition.

Clinical manifestations of the spinal osteochondrosis are multiform: from severe lowback pain in acute hernia of the dystrophic changed disk to the discomfort feeling. Provocative factors of the lowback pain are more often muscular overstrain, weight lifting and awkward movement, continuous uncomfortable position, overcooling, straining effort, etc. Vertebral (connected with the impairment of one or two VMS functioning) and the extravertebral (connected with pathologic outflow from the affected vertebral segment) manifestations (syndromes) are defined.

Extravertebral manifestations occur due to the pathologic outflow from the affected vertebral part, that spread to the corresponding sclerotomes to certain body parts. These syndromes obtain names according to localization: cranialgia, toracoalgia, brachialgia, ischialgia, cruralgia, calcaneo, achill, coccygodynia (pain in the area tailbone). Painful feelings can also irradiate across the wide vegetative net to the visceral sphere (heart, lungs, pleura, liver, pancreas, intestine) – visceral symptoms. Pain syndromes, that occur in the compression of roots, cauda equina and other parts of the peripheral nervous system, consist a special group. All mentioned types of "irradiated" and reflected pains form the variety of extravertebral neurovascular, musculotonic, neurodystrophic, vertebrovisceral and neural syndromes.

#### **Vertebral manifestations of osteochondrosis:**

- change of the spinal configuration (lordosis smoothening or increase, kyphosis, scoliosis, kiph-lordoscoliosis);
- spinal mobility impairment (mobility limitation due to the miofixation or pseudospondylolistesis);
- local pain and tenderness in active and passive movements. These symptoms are conditioned by the recurrent (meningeal) nerve receptors irritation;
- feeling of the "spine fatigue" and discomfort in the back – sign of the VMS absorbing function loss, decrease of the spine ability to resist usual loads;
- paravertebral back and neck muscles spasm, tenderness during palpation at the paravertebral soft tissues, and also tenderness of the spinous process, interspinous ligaments, facet joints area, sacroiliac junctions.

#### **Extravertebral vertebrogenic syndromes of spinal osteochondrosis**

Neurologic manifestations of spinal osteochondrosis are considered as vertebrogenic:

1. **Reflex syndromes** – main their reasons are fissures and ruptures of the fibrous ring fibers, that manifest clinically with tension and dystrophic changes of various muscles and fascias, that is called miofascial syndromes. In reflex syndrome pain is distinguished by the diffuse spreading, has sclerotomic character, its intensity increase in weather changes, is accompanied by distressing painful paresthesiae, limbs cooling, formication. Dystrophic changes of joints and ligaments and multiple vegetovascular disorders also compose the reflex syndromes group. They manifest with the vascular spasm of limbs and heart. The last is called cardialgia and their differential diagnosis with vascular heart diseases often cause significant complications.

**Reflex syndrome** is divided into three big groups: musculotonic, neurodystrophic and vegetovascular.

**Musculotonic syndrome** of spinal osteochondrosis manifests with the tension of different paravertebral muscular groups.

**Neurodystrophic syndrome** is represented by the noninflammatory affection of the joints – periarthrititis and their combination with vegetative disorders of limbs, dystrophic changes in muscles and ligaments. Dystrophic changes in muscles at the place of their adjunction of the bone are determined by the tendons induration, tenderness and are called with the term «neuroosteofibrosis».

**Vegetovascular syndrome** manifests with vascular and vegetative disorders in arms and legs.

2. **Radicular syndrome** – diskogenic (vertebrogenic) affection of the spinal roots/ affection of the root is conditioned by not only its mechanical compression, but also inflammation, swelling and demyelination. Immunologic processes have the certain role in their development. Changes in sensitivity, reflex sphere (reflex decrease or absence) or motor (paresis, paralysis) are typical for diskogenic radiculitis. Main reason of diskogenic radiculitis is usually the rupture of the fibrous ring and secondary compression of the spinal root with the disk hernia. Pain in radicular syndrome has nagging, burning, stabbing, sharp character, is accompanied by the feeling of numbness, formication, electric current passing. Pain increases in bendings, mild physical activity, cough, irradiate to the one or both legs. Affection of the motor root leads to the muscular contracture occurrence, and in more severe cases – to muscular hypertrophy.

3. **Radiculovascular syndrome** (radiculischemia, myeloischemia – is determined by the acute occurrence of motor and sensitive disorders of radicular type with the muscular paresis and paralysis occurrence at the upper and lower limbs on the background of the pain syndrome disappearing.

**Cervicalgia** – cervical pain in osteochondrosis – can be instant or like attacks (lightning pain). Pain in attacks can be very intensive, aching, but is always sensed deeply in the neck. It is accompanied by the cervical muscles tension, stiffness, its intensity increase in attempts to turn over in the bed.

**Cervicocranialgia** – characterized by the pain in the cervicooccipital area with irradiation (more often unilateral) to the parietal, temporal, frontoorbital and auricular area. Pain is of throbbing, darting, aching and burning character, that occurs and increases at head movements.

**Cervicobrachialgia** – cervical vertebrogenic syndromes in the area of the arm and muscles of the anterior chest wall. They are conditioned by the reflex dystrophic changes in muscles of the proximal part of the upper limb, shoulder girdle and chest, and also tendoperiarticular tissues of the shoulder and elbow joints. Local manifestations – tenderness and muscular tension induration presence in them, nodules – are often accompanied by the tenderness in the area of muscular tendons adjunction to the bone prominences (coracoid process of scapula, external epicondyle of shoulder, etc.).

Osteochondrosis of the cervical and thoracic spine are often accompanied by the cardialgic syndrome (pain in the area of the heart, sternum or beside sternum of noncoronary genesis can be continuous, aching, stabbing), which should be distinguished stenocardia or myocardial infarction.

Vertebral artery syndrome which includes the complex of the cerebral vegetative and vascular symptoms, that are conditioned by the sympathetic plexus of the vertebral artery irritation, its wall deformation and lumen narrowing (Fig. 22).

Vertebral artery syndrome manifest with pain, paresthesia in the cervicooccipital area with irradiation to the anterior part of the head, crown, temple, dizziness, that are accompanied by nausea, vomiting, stupefaction or noise in the ears, photopsia.

### **Osteochondrosis of the lumbar part of the spine**

*Clinical pattern of the stable forms of osteochondrosis.*

*I stage: intradiscal dislocation of the nucleus pulposus – diskogenic. Manifests with pain like lumbago, lumbodinia or lumbischialgia.*



**Figure 22.** Instability of the vertebral motor segment in the cervical part of the spine with the vertebral artery deformation

**Lumbago** – acute, like darting, lumbar pain. Occurs in the weight lifting, awkward movement, cough, sneezing. Movements in the lumbar part of the spine are sharply limited or absent.

**Lumbodinia** – subacute or chronic lumbar pain. Occurs gradually after physical activity, continuation of the lumbar lordosis, movements limitation, mild tenderness in paravertebral points in the lumbar area are observed.

**Lumboischialgia** – lowback pain with irradiation to one or both legs of sclerotomic character. The following forms of lumbischialgia are defined.

**Reflex-tonic** – muscular tension, changes of the vertebral configuration, sharp movements limitation in the lumbar area prevail.

**Vegetovascular** – combination of pain with the feeling of leg numbness, especially foot, feeling of "afflux", heat or chill in it. Unpleasant painful feelings occur in the transition from horizontal into the standing position.

**Neurodystrophic** – pain has burning character and usually increase during the night. trophic impairments, feet hyperkeratosis, sometimes ulcer are observed during the physical examination.

*II stage: disk protrusion; depending on the affected segment localisation, degree and place of the affection, diseases can manifest with several syndromes, where the lumbischialgia is a common symptom. Unlike the lumbischialgia in the I stage of the disease, pain at the protrusion irradiate to the corresponding dermatome, or combination of the sclerotomic and dermatomic irradiation are observed.*

Pain differs with duration, persistence, its intensity increase in loads on the spine, movements. Pain intensity decreases at rest, but not disappear completely. In patients with disks protrusions, reflex myotonic disorders are observed in practically all patients and are more expressed than in the diseases stage. Lumbar lordosis is smoothed, paravertebral muscles are tensed, movements in the lumbar part of the spine are sharply limited. Sensitivity disorders manifest with hypesthesia, paresthesia, numbness, formication. Reflex, muscular force decrease or absence, muscular hypotrophy are observed.

*III stage: disk hernia manifest with reflex pain and compressive radicular syndrome.*

Pain syndrome at lumbischialgia is more often occurs after the trauma or weight lifting, sometimes on the background of preceding moderate lumbodinia. Pain reaches bigger intensity only in the first 1–2 weeks, then its intensity decreases. Lumbodinia is mildly expressed initially and sometimes disappears completely till the visit of the doctor. Only ischialgia is preserved, which is characterized by the combination

of the dermatomic and sclerotomic irradiation as well as in the II stage. Tension symptoms (Lasegues symptom, Wasserman crisscross symptom). Myotonic reactions are sharply expressed: paravertebral muscles hypertone, kyphosis increase or lumbar lordosis smoothing, scoliosis. Forced antalgic postures (patient lies on the side with a ected leg fl exed in the knee and hip joints) and sharp limitation of all or some movements in the lumbar part of the spine (movements in the sagi(al plane and bending towards the affected side are more often absent) add to the clinical paernttof this stage of the disease. Radicular syndromes are observed in all patients with III stage of stable osteochondrosis form of the lumbar part of the spine. Patient has sensitivity disorders in the innervation area of the 1–2 roots.

Clinical picture of the instable forms of osteochondrosis. Clinical picture in instable forms of osteochondrosis of the lumbar part of the spine is characterized by the typical syndrome of instability and reflex myotonic syndrome in relatively rare expressed neurologic symptoms.

Three stages of instability of the lumbar osteochondrosis are defined depending on the affection of one or other structures of the vertebral motor segment.

Diskogenic instability (1 stage) – manifests mainly with lumbodynia, that pass in dynamic loads to lumbischialgia. Pain is more intensive in the morning at the beginning of the diseases; during the day due to the coordinated increase of the muscular activity and relative spine stabilization, but increases up to the evening, with the muscles fatigue. With increase of the disk destruction and protrusion, pain become so severe, that hamper the ability to be in a vertical position even for the short time. Refl ex syndrome prevails, that manifest with movements limitation in the lumbar part of the spine. In segmentar block, abrupt sharp lowback pain and movements absence are observed. Muscles are sharply tensed. Due to the swelling development radicular disorders can occur in several days.

Diskoarthrogenic instability (2 stage) – characterized by the expressed dependance of the painful feelings from the staticodynamic loads on the spine. Not only pain intensity depends on the staticodynamic load, but also paresthesiae, rare – antalgic scoliosis. These symptoms manifest during walking and movements, signifi cantly decrease or disappear in the recumbent position or spine unloading. Pain is localized in the lumbar part, usually irradiates at both lower limbs. Together with the expressed reflex syndrome (lordosis smoothing, spinal muscles tension, movements limitation in the lumbar part of the spibe) radicular disorders are determined in part of the patients. Cause of the radicular disorders is more often fragments of the ruptured disk, that dislocate into the

spinal canal. Expressiveness of neurologic disorders depends on their size – up to the gross function impairment of the lower limbs and small pelvis organs. Unlike the disk hernia, radicular disorders of such genesis are usually constant and do not regress.

Degenerative spondylolisthesis (3 stage) – manifests clinically with lumbischialgia and instability syndrome, that refl ects dependance of the clinical symptoms from the staticodynamic load on the spine. Reflex disorders are observed rarely in patients with degenerative spondylolisthesis. Sharp tension of spinal muscles, movements limitation of the lumbar part of the spine, radicular disorders are rarely observed. More often they manifest with hypesthesia in the area of 1–2 roots innervation, reflex asymmetry.

Instrumental diagnostics. Xray investigation specifies localization of the pathologic process and character of the functionstructural changes in VMS. Plain and functional (in the position of maximal flexion and extension in the lateral projction) Xray without contrast are performed. Mobility limitations (spine instability) in the VMS is determined at the functional spondylogramms in the lateral projection at maximal flexion and extension at the standing position. Vertebrae dislocation in the horizontal plane in maximal flexion and extension usually consists around 2 mm. Increase of the dislocation distance indicates the VMS instability or vertebra dislocation – spondylolisthesis. To radiologic signs of osteochondrosis refer: decrease of the intervertebral space height, subchondral sclerosis – configuration impairment and thickening of the endplate with the impression areas presence – Schmorl's nodule, marginal osseous outgrowth, arthrosis (of intervertebral joints, uncovertebral, vertebrocostal) (Fig. 23).

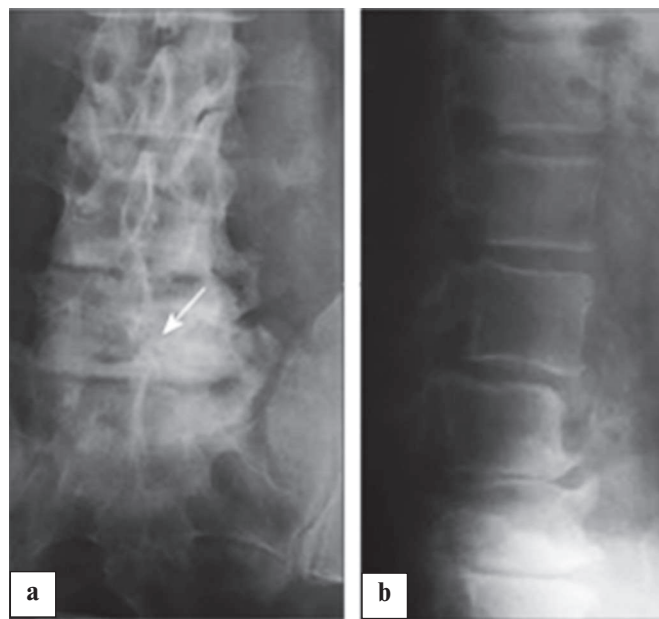


Figure 23.



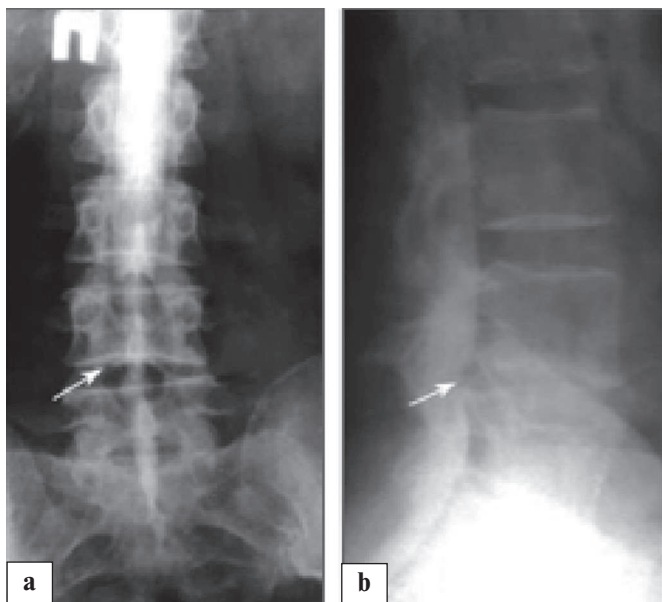


Figure 24.

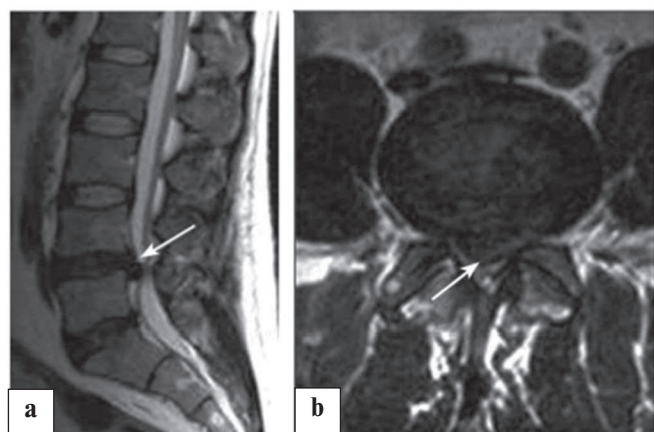


Figure 25. MRI (A – lateral and B – axial projections) of the lumbosacral part of the spine. L4–L5 intervertebral disk hernia.

Radiography with subarachnoid space contrasting (myelography) (Fig. 24) or epidural space (epidurography), magnetic resonance imaging (Fig. 25) or spiral computed tomography are performed according to indications.

**Treatment.** Treatment of spinal osteochondrosis should be directed at all links of the pathogenesis, should be conducted adjusted for the lesion localization, stage and form of the diseases, functional condition of the organism and separate systems. Such effectually provides complex use of different methods (drugs, exercise therapy, massage, traction, manual therapy, etc.) depending on indications. Plan of the medicorehabilitation interventions is made individually for every patient adjusting for the prevailing pathologic syndromes.

1. Pain syndrome – the following is used: analgesics, neuroleptics and sedatives, counter-attracting therapy, infiltration therapy, diadynamic therapy, UVI, anesthetics electrophoresis, spine immobilization.

2. Orthopedic disorders (VMS instability, functional block, stenosis manifestation) – spine immobilization and traction, exercise therapy, decompressive-stabilizing operative interventions.
3. Microcirculation disorders at the spine tissues and at the periphery (swelling, ischemia, hypoxia) – antiedematous, spasmolytic drugs (nicotinic acid, nicospan), ganglioblockers (gangleron, pachycarpinum, etc.), infiltrative therapy, ultrasound, segmentar massage, reflex therapy, local heat.
4. Musculotonic disorders – heating procedures, massage, infiltrative therapy are conducted, sedatives and muscle relaxant, ultrasound, reflex therapy, autogenic relaxation are recommended.
5. Neurodystrophic syndrome (neuroosteofibrosis) – segmentar nervepoint massage, hormone and enzyme therapy, biostimulators and resorptive drugs.
6. Nervous conduction disorders – cholinesterase inhibitors, vitamin therapy, massage, exercise therapy, anabolic hormones, ADP, biostimulators, resorptive drugs are administered.
7. Dyschemic disorders of the cerebral, spinal and peripheral blood circulation – bed rest in the acute period, immobilization of the affected part of the spine, spasmolytic, hypotensive, cardiotoxic, antiedematous drugs, neuroleptics and sedatives, antihistaminic drugs, decompression of the affected vessel.
8. Reactive cicatricial-commissural changes in the epidural tissue, nerves and spinal cord tunic – hormone and enzyme therapy are conducted (injection into epidural tissue, intramuscular, subcutaneously, using phonophoresis), resorptive drugs, biostimulators, surgical section of the commissures.

**Operative interventions is indicated in:**

- acute compression of the cauda equina, that cause the lower paraparesis and pelvic organs disorders;
- root compression, that causes progressing paresis;
- severe disabling pain syndrome, persistent after conservative treatment.

**Surgical interventions in the spinal osteochondrosis are divided into:**

- decompression – elimination of factors, that compress neurovascular formations of the spinal canal (discectomy, facetectomy, laminectomy, etc.);
- stabilizing (spondylodesis) – directed at stabilization of the affected VMS – anterior, posterior and anteroposterior spondylodesis;
- combined decompression-stabilizing operative interventions – single-stage performance of decompression with the following VMS stabilizing.

## DEFORMING SPONDYLOSIS AND SPONDYLOARTHRITIS

Spondylosis is characterized by the primary affection of the vertebral bodies with the following affection of the posterior supporting complex (facet joints and ligaments) with the occurrence and slow progression of degenerative changes in the IVD.

This pathology is more often observed in the elderly age. Among the spondylosis causes the following are defined: spinal injury of different genesis, continuous sitting work (continuous static load), posture impairment, osteochondrosis. Overcooling or excessive physical activity are often provocative factors. Process can be isolated or can have diffuse character.

Pathogenesis. The main sign of spondylosis – massive osteophytes formation in the area of vertebral bodies apophyses. Due to the degenerative-dystrophic changes, hyaline cartilage loses its fibrous structure and is replaced with the connective tissue, which decreases its ability to resist loads and traumas. Fibrous ring is also involved in the pathologic process, that is accompanied by tear of its fibers in the place of adjunction to the bone margins (apophysis) of the adjacent vertebrae (in front, aside, and back). Tissue, which loses strength and traumatizes the anterior or posterior longitudinal ligaments, of the peripheral disk part dislocate at the place of fibrous ring tear. Anterior longitudinal ligament delaminates from the adjunction place at the vertebral body limb, and then from the vertebral body. Anterior longitudinal ligament is a periosteum for the vertebral body. It reacts to constant trauma and irritation with the formation of the marginal osseous outgrowth at the anterior or lateral surfaces of one or two adjacent vertebrae. Typical feature of these ossification is their symmetry – direction towards each other. They often merge together forming a block of 2 or several adjacent vertebrae at the anterior or lateral surface. Depending on the main osteophytes localization, spondylosis is divided into ventral and dorsal (last can cause the spinal canal stenosis). Cervical (most common), thoracic and lumbar spondylosis are defined. Morphologic changes in the spine are confirmed by additional methods of investigation (Fig. 26).

**Figure 26.** X-ray of the cervical part of the spine (lateral projection). Cervical spondylosis in C4–C5, C5–C6 segments. Ventral and dorsal osteophytes with the spinal canal stenosis



## Clinical picture of spondylosis

Cervical spondylosis manifests with cervical pain and pain in the shoulder girdle, circulation disorders in the vertebrobasilar system (dizziness, noise in the ears, visual disorders, blood pressure changes). When the process is localized in the thoracic or lumbar part of the spine, patient complains of pain, that spread to the chest, buttocks and hips and is accompanied by movements limitation, stiffness, discomfort. Tender areas are determined at palpation along the spine with irradiation to the chest, abdominal wall, pelvic wings, buttocks and hips. Dorsal osteophytes can cause the spinal canal stenosis and radicular canals of the spinal cord with their irritation, rarer – roots compression, compressionischemic myelopathy in process localization at the cervical and thoracic parts of the spine. In patients with lumbar spondylosis, neurologic disorders can manifest with syndromes of radicular or caudal intermittent claudication (false intermittent claudication symptom).

## SPONDYLARTHROSIS

Spondylarthrosis (facet joints arthrosis, facet syndrome) – degenerative affection of the true synovial joints of the spine. Costovertebral (rib head and costotransversal) joints are also included in this group. Spondylarthrosis is often combined with spondylosis and osteochondrosis of the spine. Osteochondrosis development is accompanied by the hyaline covering cartilage destruction, subchondral sclerosis. marginal osteophytes formation, articular processes hyperplasia, dystrophic changes of the articular capsule with its weakening.

### Spondylarthrosis classification (Radchenko V.A., Prodan A.I.):

#### I. Dystrophic-destructive:

- dislocation (in osteochondrosis, scoliosis, hyperlordosis, osteochondropathy, posttraumatic);
- dysplastic;
- dyshormonal.

#### II. Inflammatory-destructive.

### Clinical picture of spondylarthrosis

In cervical spondylarthrosis patients complain of the cervical pain, that can irradiate to the shoulder girdle, interscapular area, upper limb, occipital area. Osteocartilaginous outgrowth from the joints can also lead to the intervertebral foramensnarrowing and irritation of the spinal cervical roots (radicular syndrome), impress into the vertebral artery canal and cause the vertebral artery syndrome development.

Thoracic and lumbar spondyloarthrosis manifest clinically manifest with bilateral pain, which is usually located in the paravertebral area, not at the mid-line, unlike the diskogenic pain. Pain often irradiates to the chest, sacroiliac junction, buttocks, hip, the more distal pain spread to the foot is possible. It usually has intermittent character and increase in continuous standing and extension, decrease in anterior flexion, sitting and walking, and also in dorsal recumbent position. Hyperplastic changes in the facet joints can lead to the spinal canal stenosis and canals of the spinal nerve roots, that manifests clinically with different neurologic disorders depending on the level of affection.

Additional methods of investigation in spondylosis and spondylarthrosis include: X-ray of the spine (narrowing of the intraarticular spaces, subchondral sclerosis, deformation and hypertrophy of the articular facets are observed), MRI and spiral computed tomography of the spine, in suspicion of the vertebral artery syndrome – doppler investigation of the head and neck arteries, MRI-angiography (Fig. 27).



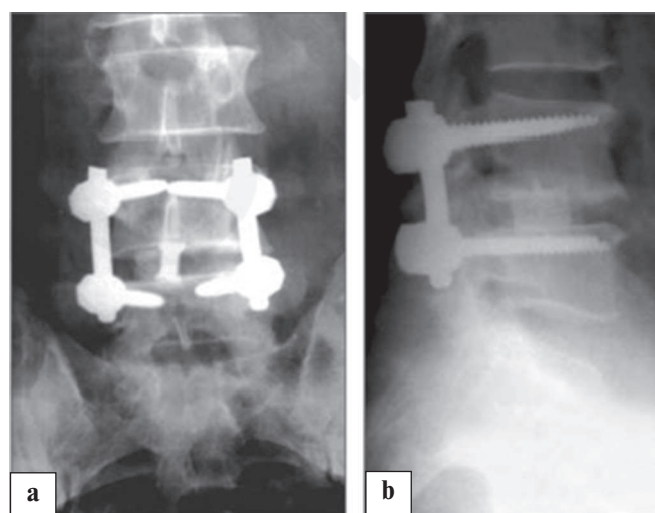
**Figure 27.** X-ray of the lumbosacral part of the spine (oblique projection). Decrease of the intervertebral space height of L3–L4, L4–L5, L5–S1, interarticular space narrowing, subchondral sclerosis, facet joints deformation and hypertrophy

### Treatment of spondylosis and spondylarthrosis

Spondylosis and spondylarthrosis treatment should be complex and pathogenetic. In expressed pain syndrome treatment should be directed at the pain and inflammation elimination. These can be reached by the following medicamentous agents: antiinflammatory drugs, in expressed muscular spasm – muscular relaxants of the central action, local use of the antiinflammatory ointments, plasters. Blockades of the facet joints are effective in persistent pains under the X-ray or CT fluoroscopy control – blockade of the joint or nerve, that innervates (supply with nerve endings) the corresponding joint is performed. Local anesthetics, for example lidocaine, and suspension of glucocorticoids, that has good antiinflammatory features, are used for the blockade. Blockade is not only medical, but also diagnostic procedure: if the positive effect was obtained

the doctor can state, that changes in the blocked joints were responsible for the pain syndrome development in this patient. Epidural injection of local anesthetics and glucocorticoids are used in the spinal canal stenosis, that have good analgesic and antiinflammatory effect. Massage, physiotherapeutical procedures (electrophoresis with novocaine at the affected area, diadynamic currents, ultrasound at the spine area), exercise therapy for the muscular corset strengthening of the spine, pelvic position correction, lumbar lordosis decrease are included in the treatment complex. If the conservative treatment is not effective, operative treatment is indicated. They are divided into decompression (neurovascular formations of the spinal canal decompression), stabilizing (anterior and posterior spondylodesis) and decompression-stabilizing (Fig. 28.A-B).

After the inpatient treatment patients require rehabilitation measures, that include sanatorium and spa treatment, exercise therapy, massage, rational organization of the working regime.



**Figure 28 (A-B).** X-ray (anteroposterior (A) and lateroposterior (B) projections) of the lumbosacral part of the spine before and after the operative intervention – discectomy of the L4–L5 intervertebral disk, interbody spondylodesis with ceramic implant and posterior spondylodesis with transpedicular fixation device

### SPONDYLOLISTHESIS

Spondylolisthesis – disease, that is characterized by the dislocation of the upper vertebra in relation to the lower (Fig. 29).

Spondylolisthesis etiology remains insufficiently studied. Impairment of the vertebropelvic balance, pars interarticularis pathology, etc., are considered as one of the spondylolisthesis development factors.

**Classification.** Depending on the leading etiological factor, that causes spondylolisthesis, the following are defined:

- isthmic (spondylolytic) – characterized by the isthmus pathology presence – pars interarticularis, which is divided into:

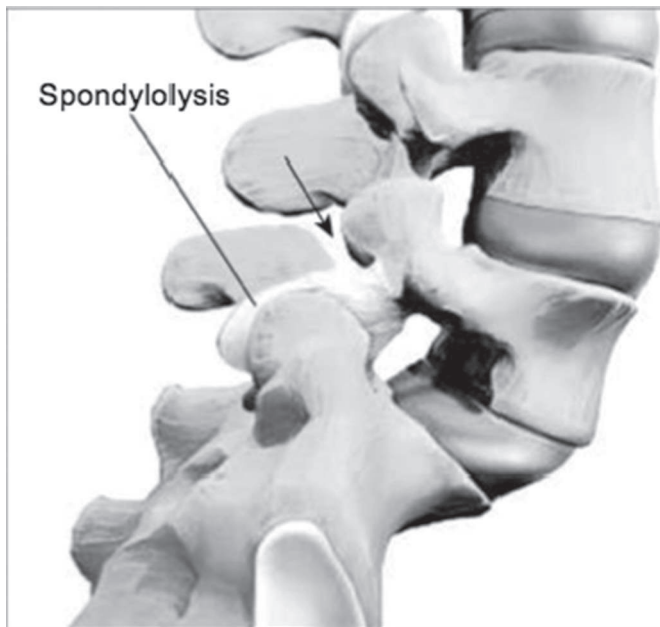


Figure 29. Spondylolysis (arrow)

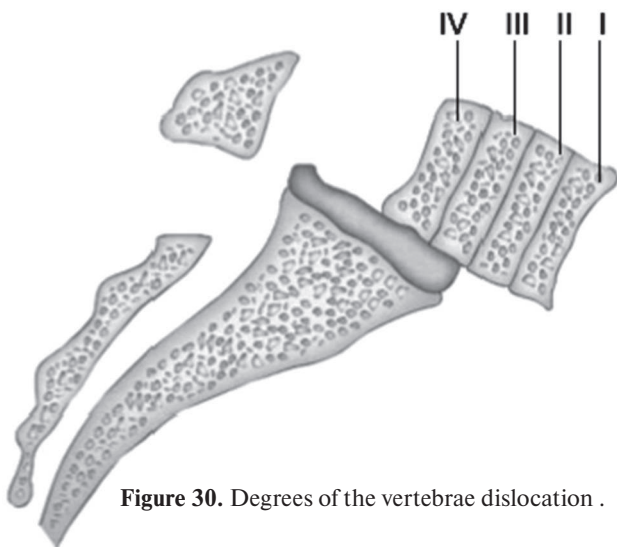


Figure 30. Degrees of the vertebrae dislocation .

- lysis of pars interarticularis;
- elongation of pars interarticularis;
- fracture of pars interarticularis.
- dysplastic or congenital;
- degenerative;
- traumatic;
- pathologic (generalized or local pathologic process in the bone tissue, tumor, spondylitis)

Depending on the upper vertebra dislocation direction the following are defined: anterolisthesis – towards, retrolisthesis – backwards, laterolisthesis – aside. Four degrees of the vertebrae dislocation relative to each other according to Meerding's classification:

I degree – dislocation for  $\frac{1}{4}$  of the vertebral body;  
 II degree – dislocation for  $\frac{1}{2}$  of the vertebral body;  
 III degree – dislocation for  $\frac{3}{4}$  of the vertebral body;

IV degree – dislocation for the entire vertebral body length or spondyloptosis (Fig. 30).

Also depending on the pathologic mobility presence between the vertebrae, stable and unstable forms of spondylolisthesis. This has an important value for the surgical intervention choice (to perform or not stabilizing interventions).

Clinical picture. Patients with the III–IV degree dislocation have typical telescopic body during the examination. Due to the center of gravity dislocation and vertebropelvic balance impairment, these patients have a gait like a "ropewalker". Palpation along the spinous processes determines retraction – stairs symptom. Patients have horizontal positioning in the sacral area.

Clinical picture of spondylolisthesis consists of two parts – back pain and neurologic disorders, that are conditioned by the compression of different nervous structures. There are many theories of the back pain occurrence. Pain syndrome in case of spondylolisthesis is conditioned by pathologic changes in the intervertebral disk, intervertebral joints and secondary – paravertebral muscles and ligaments.

Back pain has mechanical character, increase in physical activities, body bending. Also, complaints of posture and gait disorders occur.

Nervous structures compression (roots, spinal cord) occur in the spinal canal narrowing, due to the vertebrae dislocation relative to each other, pathologic outgrowth in pars interarticularis, sacral roots tension in the vertebrae slippage. Tension symptoms (Lasegue's symptom, reverse Lasegue's symptom or Wassermann's symptom) will be observed in case of nervous roots compression, decrease or absence of tendon reflexes, pain in the area of the corresponding dermatome, hypo- and anesthesia in the innervation area of the corresponding dermatome). "Intermittent neurogenic claudication syndrome" occurs in the cauda equina compression. Myelopathy occurs in the spinal cord compression. Disorders of the pelvic organs function can be observed.

Additional investigation includes X-ray (anteroposterior projection, lateral projection of functional tests, oblique projection), MRI, CT.

Isthmic spondylolisthesis – characterized by the pathology of pars interarticularis. It is characterized by the symptom of "Scottish dog" at oblique radiographs ( $B \frac{3}{4}$ ). This symptom can be observed in 5–20 % of healthy people. Cause compression of the spinal cord in the III–IV degree of the vertebrae dislocation.

Dysplastic spondylolisthesis is characterized by the dysplasia of the posterior supporting complex, which manifest with the impairment of space orientation (position in the frontal plane) of the articular processes, that form as called osseous uncus, which is formed by the upper articular processes of the subjacent vertebra and lower articular processes of the overlying vertebra. The more frontally articular pro-

cesses are located, the stronger is adhesion between the joints. This type of spondylolisthesis is the most complicated for the progressing prediction.

Degenerative spondylolisthesis develops as the result of continuous intersegmentar instability; isthmus pathology is absent in this case. It is observed in 6 % of men and 9 % of women and is characterized by the asymptomatic course.

Traumatic spondylolisthesis occurs as the result of pars interarticularis or posterior supporting complex fractures, which form are called osseous uncus. It is characterized by the sharp pain in the area of fracture, and neurologic disorders in presence of the nervous structures compression.

### **Treatment**

Conservative and operative treatment of spondylolisthesis are defined. Conservative treatment in spondylolisthesis is indicated in the pain syndrome presence in the lumbar part of the spine. Conservative treatment includes NSAIDs intake, therapy with corsets (Boston corset) with immobilization purpose, blockades. In continuous pain syndrome (more than 3–6 months), that does not react on conservative therapy, the issue of operation should be decided.

Conservative treatment is also indicated in 1, 2 degree according to Meerding's without neurologic deficit, in stable forms of spondylolisthesis. Spondylolisthesis progressing is an important issue for the treatment method choice.

Operative treatment is indicated in cases of neurologic deficit increasing: radiculopathies, radiculomyelopathies, myelopathies and neurogenic intermittent claudication. Main purposes of the operative treatment are the elimination of the nervous structures compression, renovation of the sagittal balance and stabilization. Stabilizing operations are directed at conditions for spondylodesis creation. Stabilization is performed with transpedicular fixatros, interbody cages, plates, that are placed on the ventral surface of vertebral bodies. Vertebra removing can be performed in case of spondyloptosis. An important problem of operative treatment is the necessity and reduction degree of the vertebra. Reduction can not be reached in some cases without the neurologic deficit increase.

### **DYSPLASTIC SPONDYLOLISTHESIS**

In degenerative spondylolisthesis with nervous roots and spinal canal content compression and progressing signs, decompression interventions (laminectomy, hemilaminectomy, spinal root canal decompression) and decompression-stabilizing interventions are performed.

Operative treatment is used for the traumatic spondylolisthesis, which consists in the nervous struc-

tures decompression and stabilization (using transpedicular fixation, cages, etc.), for creating the spondylodesis conditions.

### **SPINAL STENOSIS**

Spinal stenosis – decrease of the spinal canal lumen in relation to its content with specific clinical manifestations. Stenosis – structural, functional and clinical term. So, the following definition of the spinal stenosis can be given – disease, that is characterized by the expressed inconformity between the spinal canal size and its content, functions decompensation, loss of the protective functions reserve and occurrence of the specific neurologic disorders of the spinal cord elements and nervous roots (Prodan A.I.). Stenosis was described for the first time by Swedish neurosurgeon H. Verbiest.

#### **Classification**

Congenital and acquired stenosis are define by etiology (Verbiest). Congenital stenosis is observed rarely. Then Nelson modify and expand the stenosis classification.

#### **According to etiology:**

##### **1. Congenital:**

- idiopathic (is formed as the result of genetically determined impairment of the vertebral bone elements development in the postnatal period);
- chondrodystrophic.

##### **2. Acquired:**

- degenerative (central stenosis, lateral stenosis, degenerative spondylolisthesis);
- combined (combination of the congenital and degenerative stenosis elements);
- spondylolisthetic;
- iatrogenic (postlaminectomic, after dorsal and ventral spondylodesis).

In the institute named after Sitenko M.I., stenosis is divided into dysplastic, degenerative and combined. Dysplastic stenosis can be connected with congenital (genetically determined) dysplasia, as well as with the acquired in the postnatal period and connected with the vertebral morphogenesis impairment until the growth end. Degenerative stenosis occurs on the background of degenerative changes of the spinal canal. In their turn, each of these stenoses are divided into 3 types: dislocation, hyperplastic, combined. Depending on localization each of these types is divided into the central, lateral and foraminal.

Stenoses are defined depending on localization in modern classifications: central, lateral, foraminal. The term of "dynamic stenosis" is defined, which is characterized by spinal canal stenosis during movements (extension).

**The following mechanisms are defined in the stenosis pathogenesis:**

- mechanisms of the spinal canal remodeling, which lead to the spinal canal lumen narrowing;
- mechanisms of the nervous structures compression (spinal cord, nervous roots) and vascular structures compression, that cause the ischemia of nervous elements.

Degenerative changes in the IVD (height decrease), antelisthesis, retrolisthesis or laterolisthesis, facet joints hypertrophy, ossification of the flaval ligament, osteophytes from the facet joints cause the spinal canal lumen narrowing, that results in the nervous structures compression. Even in relatively wide spinal canal, significant ossification of the facet joints can cause the lateral recesses stenosis. Compression of primary central or lateral part of the spinal canal occurs depending on the hypertrophy or degeneration of different VMS structures. Flaval ligament, posterior longitudinal ligament (Forestier disease), lower facet joints hypertrophy and ossification causes primarily central stenosis; hypertrophy of upper facet joints – primarily lateral stenosis. So, spondyloarthrosis is more often accompanied by the lateral stenosis.

**Clinical picture.** Stenosis manifestations are mainly neurologic. Depending on the stenosis level different symptoms occur, Stenosis more often occur at the lumbar level (in decreasing order L4–L5, L3–L4, rarer L3–L4 and even more rare L5–S1), but also stenosis can occur at the cervical part. Morbidity is higher among elderly people. Stenosis in the cervical part of the spine is characterized by the myelopathy and/or radiculopathy syndromes with corresponding neurologic symptoms, depending on the compression of spinal and/or nervous roots.

"Anthropoid" posture attracts attention during the examination. It is characterized by the lumbar lordosis smoothing, –compensatory increase of the spinal canal lumen in flexion at the lumbar part. Main clinical symptom – "neurogenic intermittent claudication" – progressive fatigue and sensitivity disorders (along the dermatome) in legs during walking, that occurs in the insufficient blood supply of certain root with intermittent impairment of the nervous impulse passing. neurogenic intermittent claudication should be mandatory differentiated with intermittent claudication, that occur in vascular diseases – circulatory insufficiency of the lower limbs. Pulsation intensity decrease at the distal part of the limb, sensitivity decrease according to the peripheral type ("socks symptom"), normalization of the blood circulation occur during rest at US-investigation, etc. Patients say, that legs become feeble. Patient is asked to specify distance, that he can manage until the fatigue in the lower limbs occurs. Distance decreasing during observation indicates the progressing.

Depending on the level of compression and decompensation degree, different sensitive and motor disorders will occur, that accompany compression radicular syndromes. One, two or more roots can be compressed. Symptoms can be unilateral or bilateral.

Cauda equina compression can cause the pelvic organs function disorders.

Reflexes, sensitivity, muscular force can be preserved at rest during neurologic examination. Then the "march test" is performed with provocative purpose – patient is asked to walk outside.

**Diagnostics.** MRI, CT, Xray, different methods of spinal canal contrasting (myelography) are used in diagnostics. Narrowing of the anteroposterior sizes of the spinal canal, articular processes hypertrophy, osteophytes, possible manifestations of spondylolisthesis can be determined at radiographs. Use of the contrasting is possible (myelography).

Narrowing of the anteroposterior sizes, articular processes orientation impairment, lateral canal narrowing manifest with "trefoil" symptom, can be determined at scans.

Compression of nervous structures is determined at MRI. "Sand glass", "washboard" symptoms are typical.

Stenosis is diagnosed, when typical clinical data is present. MRI, CT, Xray specify only the spinal canal narrowing degree.

Electroneuromyography use is possible in addition.

Differential diagnosis is performed with vascular disorders of the lower limbs (peripheral artery disease with legs affection, obliterating endarteritis), IVD hernia, juxtafacet cysts, arachnoiditis, spinal cord tumors, diabetic neuropathy.

**Treatment** is divided into conservative and operative.

Conservative treatment is conducted with orthopedic braces (that decrease lumbar lordosis), NSAIDs, antiedematous therapy. Epidural blockades with glucocorticoids, epidural adhesiolysis are used.

Operative treatment is conducted in the effect absence or disease progressing. Operative treatment consists in decompression (laminectomy, hemilaminectomy, partial facetectomy, different laminoplastics) and decompressionstabilizing operations (stabilization with transpedicular fixators, cages, combined methods) operations. Decompressionstabilizing operations on the cervical part of the spine can be conducted from the anterior or posterior approaches, that depends on the compression presence with anterior or posterior structures. Expected results – neurologic symptoms regress. But this result, due to the cicatricial changes in the perineural membranes, liquor dynamics impairments, can not be reached. In case of myelopathy and sclerosis focus formation at the MRI, neurologic symptoms do not regress completely, the main purpose of the operation – decrease myelopathy progressing.