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## 8.3 Localized bone disorders

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### 8.3.1 Locally enhanced bone resorption

#### 8.3.1.1 Osteitis deformans (Morbus Paget)

Osteitis deformans is characterized by excessive anomalous remodelling affecting skeletal bones. The pathologic process is initiated by osteoclasts present in the given focus in great amount. The osteoclasts are functionally and structurally altered – they are large, polynuclear and contain plenty of cytoplasmatic and nuclear particles. These anomalous osteoclasts exert the bone resorption regardless of normal anatomical limitations. After the initial excessive bone resorption an increase in osteoblastic activity follows associated with bone formation. Medullar cavities in the focus are occupied with fibrous tissue extremely rich in blood vessels. Combination of a chaotic bone resorption with bone formation results in the typical mosaic pattern in the affected site. Defective structure of bone makes it more fragile. The most frequently affected bones are : the axial skeleton, lumbar part of vertebral column, bones of pelvis, the ribs, sternum, cranial bones, and femur. The non affected bones reveal normal structure even in severe forms of disease. That is why the condition is classified among the localized bone diseases.

Osteitis deformans occurs mainly in middle-aged men. Although even 3 per cent of population (mainly in Western Europe and USA) are involved. The progression of disease has slow onset and remains long time asymptomatic. Clinical manifestations are determined by combination of anomalous bone structure with enhanced blood flow in the affected bone. Anomalous bone structure is the cause of deformities and fractures. The enhanced blood flow through the affected bone acts as a powerful A-V shunt and may lead even to heart failure. Pathologic bone alterations may result in neurological complications e.g. by involvement of middle ear bones (ossicles) or by compression of n. acusticus deafness develops, the pressure of cervical vertebrae on the basis cranii (platybasia) leads to vertigo, ataxia eventually to hy-

drocephalus. In untreated patients secondary hyperparathyroidism may develop during the phase of high calcium utilization due to enhanced bone formation. Paget's disease is relatively frequently complicated by various types of malignant or benign bone tumours.

The underlying cause of Paget's disease is not well known. From the several existing hypotheses is at present the mostly accepted the hypothesis of viral origin of disease (slow viruses). Viral pathogenesis is supported by geographical and familial occurrence of disease, findings of inclusions in osteoclasts, and by identical stage of disease development if it occurs in more than one bones. This phenomenon might be caused by a single episode of viral infection of osteoclast precursors which have been transferred into one or more bones. Continual replication of virus in affected cells associated with transfer via cell fusion (normal property of osteoclasts) might explicate why is disease localized meanwhile the other bones are not affected.

### 8.3.2 Local disorders of blood supply

#### 8.3.2.1 Avascular necrosis

The bone responds to severe decrease in, or interruption of blood supply similarly as other tissues, i.e. by ischaemia and necrosis. The underlying cause of disorders of blood supply in certain part of bone may be trauma impairing the integrity of vessels, thrombosis or embolism. Bone infarction occurs with predilection in regions supplied via the end artery, hence the most exposed is the subchondral region at the ends of long bones. Most frequently affected are the collum femoris and the coxal joint. In patients with sickle cell anaemia necrosis of femoral head occurs due to artery occlusion by defective erythrocytes. Local ischaemia resulting from end artery occlusion may have as underlying cause hyperlipidaemia, arthritis urica, m. Gaucher or prolonged application of corticoids. The posttraumatic avascular necrosis of epiphysis in childhood may lead to growth retardation on the epiphyseal plate and to joint disorders. In adults becomes the healing process of fractures retarded and complicated and may result in irreparable joint impairment.

### 8.3.2.2 Sudeck's syndrome

Sudeck's syndrome – the posttraumatic atrophy of extremity – is known more than 200 years. It is an acute localized bone atrophy accompanied with inflammatory alterations in the surrounding soft tissues. The underlying cause of Sudeck's syndrome are circulatory disorders in the affected region of extremity induced by reflex action. The pain stimuli after the trauma induce by reflex action an increase in the tonus of adrenergic nervous system leading to local spasm of venules and resulting in blood stasis in capillaries. During the persistence of circulatory disorder dystrophy of all tissue layers is developing. At further stage local excessive bone resorption occurs owing to the osteoclast accumulation in the site of impairment which leads to the painful posttraumatic osteoporosis. The pain progressively rises, hence the circulus vitiosus closes sustaining the disorder of vasoregulation up to the last ischaemic stage. The affected extremity does not bear longer any loading and the following immobilization further impairs the tissue metabolism. Atrophy of the bone and soft tissues associated with contractures may lead to complete loss of functional ability.

Diabetic osteopathy seems to be similar to the Sudeck's syndrome. The underlying cause of this type of osteopathy are as well the disorders of circulation.

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## 8.4 Disorders of joints

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### 8.4.1 Some remarks on anatomy and physiology

Most of the permanent joints in the body are synovial. In **synovial joints** the ends of the bones are covered with smooth hyaline articular cartilage. The joint is lubricated by a thick synovial fluid and is enclosed by a flexible articular capsule. The four basic structures of a synovial joint are the synovial cavity, the articular cartilage, the articular capsule and ligaments. All the tissues of synovial joints receive

nutrients from blood vessels, except the articulating portions of the articular cartilages, disks and menisci.

**Synovial cavity** contains folds of **synovial membrane** that secretes the thick synovial fluid lubricating the synovial cavity. It is an adapted connective tissue, the cells are covered with small villi. The surface of synovial membrane is smooth, moisty and sheeny. Synovial membrane also plays an important role in exchange of substances between the joint and blood vessels and participates in the nutrition of cartilages. The cells on the surface of synovial membrane- the lining cells produce hyaluronic acid important for the exchange of proteins between synovial membrane and joint cavity. There exist two types of these cells.

- Type A – these cells are very similar to macrophages and contain a lot of cytoplasmatic lysosomes.
- Type B – they are very close in their structure to cells acting in proteosynthesis. The synovial membrane participates also in elimination of waste products of joint metabolism.

The metabolic activity of synovial membrane is very low, but it can be risen in pathologic conditions, e.g. in rheumatoid arthritis. The increase in metabolic activity is accompanied by proliferation of lining cells. It is assumed that the proliferation of lining cells is the determining factor of raised metabolism in inflammatory processes of synovial membrane.

The *synovial fluid* is a clear, viscous, amber-colored liquid present in human joints in small amounts. It has two essential functions:

1. the participation in the nutrition of cartilages,
2. lubrication of the synovial cavity.

The composition is the same as that of interstitial fluid except of the presence of hyaluronic acid. Hyaluronic acid is responsible for the viscosity rate in synovial fluid. Decrease in viscosity is typical for inflammatory processes of joints. In noninflammatory processes the viscosity increases and in haemorrhagic conditions the viscosity value varies. A part of calcium present in the synovial fluid is bound with hyaluronic acid in form of calcium hyaluronate which represents an important buffer system for maintenance of slightly alkalic pH in synovial fluid. The normal pH range in synovial fluid is 7.31 to 7.64. All