

# Bone growth and healing

Bone is formed by the remodeling of connective tissues (ligaments and cartilage) in a process called **ossification** = **ossification** .

**Bones grow in width** and **length** during life.

## Bone growth to length

The bone grows in length at a place called the epiphysis or **growth cartilage** (plate). It is located between the epiphysis and the diaphysis and is maintained throughout bone growth. There are typically two growth cartilages in long bones, one in flat and short bones.

The activity of growth cartilage is mainly influenced **by hormones** . It is activated by growth hormone and suppressed by gonadal hormones. The earlier onset of puberty in girls leads to an earlier braking of the activity of the growth cartilage, which ossifies and its function is terminated. The growth cartilage closes between the ages of 14 and 24, leaving only the epiphyseal line, which is visible throughout life.

**Proximal and distal epiphyseal cartilages** of long bones do not participate in growth to the same extent. As a rule, one of them is **more active** in growth and the bone grows faster and more with it than with the other cartilage. Proximal growth cartilage is more active in the humerus and calf bones. In the bones of the forearm and femur, the distal cartilage is more active.

## Bone growth to width

The bone grows in width from the periosteum, sometimes also from the endosteum, by the mechanism of **apposition** (increasing the volume by adding or attaching new layers). However, only this mechanism would preserve shapes and proportions during growth. Therefore, in some places, apposition is accompanied by bone breakdown, **resorption** (provided by **osteoclasts** ), both from the periosteum and from the endosteum .

In flat cranial bones, during growth (proceeds from the suture sites), the shape and curvature (changing as the radius of curvature increases) are modified by apposition from the periosteum on the outer side of the bone and resorption by the endosteum on the inner side.

## Growth regulation

Bone growth is influenced by genetic, hormonal, neural, mechanical and nutritional factors.

### Hormonal regulation

**Thyroxine** stimulates bone growth early in life. **Parathyroid hormone** affects the activity of osteoclasts, mobilizes calcium and phosphates. **Calcitonin** inhibits the resorption of bone mass (matrix). It is interesting that the female sex hormone **estradiol** is indispensable for growth acceleration in puberty in both sexes, it is responsible for maintaining growth over time (it affects the disappearance of growth cartilages).

### Regulation by mechanical activity

**The axial pressure** caused by the pull of the periosteum, the pull of the muscles passing through the joints, the pull of the joint capsules, static load (body weight) is of fundamental importance. **An increase in pressure** can lead to a limitation or even a stoppage of bone growth in length. Abnormal pressure (caused, for example, by a tumor) leads to bone erosion. **A decrease in pressure** leads to an acceleration of growth, but a very significant decrease causes a slowdown. Loss of normal load (e.g. in long-term immobilized patients) leads to demineralization.

## Bone healing

### Secondary healing

If the strength of the bone tissue cannot withstand it, the bone breaks. This healing is also called natural fracture healing. During healing, a scar is first formed from ligaments and cartilage, later the **fracture gap** is bridged by a bone scar (bone callus). The callus is first composed of laminae of fibrous bone and is broad and rough. Subsequently, it is rebuilt and within a few months the original bone shape and lamellar architecture are restored.

### Primary healing

In the fracture gap , lamellar bone can be created immediately without intermediate steps with the help of surgical **osteosynthesis (using plates and screws)**. Optimal adaptation (spacing less than 1 mm) and stable fixation of the fracture ends must be achieved. The lamellae are first arranged parallel to the cleft (perpendicular to the

longitudinal axis of the bone) - cleft healing. In the course of remodeling, osteoclasts drill through these transversely constructed lamellae, and the ends of the fractures are joined (ostemelia) by new osteons.

## Links

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### Refereneces

- ČIHÁK, Radomír – GRIM, Miloš. *Anatomie*. 2., uprav. a dopl edition. Grada Publishing, 2002. 470 pp. vol. 1. ISBN 80-7169-970-5.
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- LÜLLMANN-RAUCH, Renate. *Histologie*. 1. edition. Grada, 2012. 576 pp. ISBN 978-80-247-3729-4.