What is rheumatoid arthritis?

Rheumatoid arthritis, also known as chronic polyarthritis, is a complex, chronic inflammatory autoimmune disease with an intermittent course that attacks mainly the synovial joints, tendons, synovial sheaths and synovial bursae, destroying them by means of chronic inflammatory processes, potentially accompanied by development of deformities in the extremities and spinal column instabilities.

The disease may affect the synovial facet joints of the cervical spine between the atlas and axis (1st and 2nd cervical vertebrae), as well as the eyes, heart, lungs, nervous system and skin.

Rheumatoid arthritis is not curable. About 0.5-2% of the population suffer from this disease and 3 times as many women as men are affected. The typical onset age is between the ages of 35 and 45, although the special form known as “juvenile rheumatoid arthritis” has its onset in childhood.

How does this disease develop?

The exact cause of rheumatoid arthritis is not yet completely understood. Our understanding of rheumatoid arthritis as an autoimmune disease considers various causative factors such as genetic disposition, viruses and bacteria, along with special hormonal factors as potential initiators of a pathological reaction by the body’s immune system.

Normally, the immune system can differentiate between the body’s own substances (e.g. body cells) and foreign matter (antigens, such as bacteria and viruses).

During an immune reaction to foreign antigens, immune defense substances called antibodies (also known as immunoglobulins) are produced. The various immunoglobulins are proteins and can be classified in different groups: immunoglobulin classes G, A, M, D and E. But the immunoglobulins can also function as antigens to which new antibodies are produced. These anti-antibodies include the rheumatoid factors, which, together with the immunoglobulins, form immune complexes. These are then integrated into the synovial membrane (internal joint membrane) and can be detected by blood analysis.

75-90% of patients suffering from rheumatoid arthritis are seropositive for rheumatoid factors. Immunoglobulin M rheumatoid factors may be detectable well before disease symptoms occur.

What causes the pathological immune defense reaction in the joints?

The potential causal factors described above confuse the immune system so that it loses the ability to differentiate, and its “defense function” turns against the body’s own tissues as well, classifying them as foreign matter and destroying them.

Pathologically activated immune cells cause the production of messenger substances or cytokines in the affected joint. These substances normally function as messengers for the communication among individual cells. The main cytokines involved in the pathogenesis of rheumatoid arthritis are interleukin-1, which causes the destruction of cartilage tissue and activation of cells that breakdown the bones, the osteoclasts, and TNF-alpha, the tumor necrosis factor, mainly responsible for initiating and influencing the inflammatory process. Most of these cytokines are found in the synovial tissues of the joints. The synovial membrane forms the inner layer of each joint capsule and is responsible for production of synovia, the “joint fluid” that lubricates the joints and supplies the joint cartilage with nutrients. The synovial tissue is the primary target. It is from there that the joint destruction by means of pannus formation is initiated in rheumatoid arthritis.
Rheumatoid arthritis · Inflammatory diseases

Pannus formation using the knee joint as an example

• Normal knee from the front
  - Femoral condyles (thigh)
  - Kneecap
  - Joint cartilage
  - Head of tibia
  - Patellar tendon

• Normal knee from the side
  - Femoral condyles (thigh)
  - Kneecap
  - Joint cartilage
  - Head of tibia
  - Patellar tendon

• Normal knee from the side with bursa and joint capsule, in which the synovial membrane is located
  - Prepatellar bursa
  - Joint capsule
  - Head of tibia
Pannusbildung und fortschreitende Gelenkzerstörung

- Healthy synovial membrane
- Inflamed synovial membrane begins to proliferate and produces aggressive cell groups (pannus)
- Pannus covers the cartilaginous layer between the insertion of the synovial membrane and the cartilage-bone border.

Initiating factors in rheumatoid arthritis

- Inflammation is exacerbated, grows to include the bursa and the tendoligamentous apparatus.
- Cytokines are released, causing the destruction and disintegration of bones.
- Pannus inhibits circulation in the joint cartilage.

Advanced stage with joint stiffening (ankylosis) and development of deformities.

- Cross-section of a knee joint showing pannus formation
  - Pannus tissue
  - Joint cartilage
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• Cross-section of a knee joint showing advanced pannus formation and cartilage destruction

On what criteria is the diagnosis based?

To ensure uniform diagnostic principles, the 7 criteria defined by the American College of Rheumatology are normally used, where 4 of the 7 must be fulfilled for a diagnosis of rheumatoid arthritis.

1. Morning joint stiffness (persisting for at least 1 hour, over a period exceeding 6 weeks)
2. Arthritis with palpable swelling in 3 or more joint regions, persisting for longer than 6 weeks
3. Arthritis in hand or finger joints, persisting for longer than 6 weeks
4. Symmetrical arthritis (simultaneous, same joint region on both sides), persisting for longer than 6 weeks
5. Rheumatoid nodules
6. Rheumatoid factor detected in blood
7. Typical radiological changes (osteoporosis and/or erosions in vicinity of joints)

What examinations are done?

The “rheumatoid arthritis” diagnosis is based on the overall result of typical findings from clinical, laboratory chemistry and radiological examinations.

Rheumatological case history and examination

A review of a specifically rheumatological medical history can provide valuable information as a basis for an initial tentative diagnosis with a few specific questions:

- Where is the source of the pain?
  - In the joints?
  - In soft tissues?
  - In the spinal column?

- How has the disease developed over time?
  - Acute start or gradual worsening?
  - Intermittent episodes with phases of remission and worsening?
  - Does the pain exhibit a typical pattern over the day?

- Is there a specific pattern of affected joints?
  - Are large or small joints more typically affected?
  - Is only one joint affected, or several?
  - Do the symptoms move from one joint to another?
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Since rheumatic conditions also frequently affect organ systems, it makes sense to note any abnormalities in the following areas:
- Kidneys and efferent urinary tract (cystourethral inflammations, balanitis, previous STDs)?
- Gastrointestinal tract (diarrhea, admixture of blood)?
- Cardiovascular system
- Lungs
- Skin (psoriasis, nodular changes, swellings, ulcers)?
- Eye symptoms (burning, vision problems, foreign-body sensation, dryness, redness)?
- Fever (bouts of fever, chills)?
- Neurological symptoms

Every physical examination should cover all organ systems. We will limit our focus in what follows to the most important clinical examinations in cases of suspected rheumatic disease.

Inspection
- Skeletal malpositions, asymmetries or deformities?
- Normal leg axes?
- Joints (mobility, joint effusions, redness, swelling?)
- Muscles (too little/too much, (=atrophy/hypertrophy), normal strength development?
- Normal posture and movement?
- Skin and mucosa (skin changes, psoriasis, nodular changes, fluid accumulation)?

Palpation
- Muscles: muscle tone (tonus) raised or reduced, myogeloses (hardened muscles), trigger points (pressure points from where a spreading pain can be induced)?
- Pain induction (pressure, percussion, tension, compression pain)?
- Pain induction around the insertions of tendons, ligaments and joint capsules (enthesopathy)?
- Tendons and bursae (inflammatory changes)?
- Joints (effusion, mobility, capsule thickening, grating noises)?
- Thorax (respiratory movements normal, can compression pain be stimulated)?

Functional tests - specific spinal column functional tests:
- Active and passive mobility tests of the spinal column (flexion, extension, stretching, lateral inclination)
- Finger-floor distance (measurement of overall spinal mobility)
- Schober’s sign (measurement of mobility of the lumbar spine)
- Ott’s sign (measurement of mobility of the thoracic spine)
- Occiput-wall distance (measurement of extent of thoracic kyphosis)
- Chin-sternum distance (measurement of cervical spine mobility)
- Tests for sacroiliac joint involvement:
  - Three-phase test
  - Forward flexion phenomenon
  - Mennell’s sign

Other functional tests include:
- Measurement of joint mobility with the neutral zero method
- Measurement of crude strength in arms and legs
- Joint stability testing
Laboratory diagnostics

- Nonspecific inflammatory signs such as elevated white blood cell count (leukocytes), blood sedimentation rate (BSR) and C-reactive protein (CRP). Elevated BSR and CRP indicate the presence of an inflammatory process. These parameters are often elevated in patients suffering an acute episode.
- Specific confirmation of a diagnosis is supported by the detection of rheumatoid factors, which are in fact autoantibodies to a specific structure in the body’s own immunoglobulins and are mainly found in the immunoglobulin classes A, M and G.
- Detection of antibodies to CCP (cyclic citrullinated peptide) is possible in a very early stage of the disease and relates to the severity of the disease. Citrulline is produced from the amino acid arginine and is a component of a number of proteins held responsible for development of rheumatoid arthritis.
- Other autoantibodies can also be detected, e.g. to type II collagen and histone.

Imaging methods

- Conventional x-rays of hands and forefeet show the typical change patterns in the small joints of the fingers and toes.
  Instability of the upper cervical spine can be diagnosed on the basis of images of the cervical spine in 2 planes and sagittal functional images in dorsal and ventral inclination, as well as transoral dens images.
- Computer tomography (CT) and magnetic resonance tomography (MRT) can be used to confirm instabilities of the upper cervical spine and to evaluate inflammatory changes in bony and soft-tissue structures.
- A whole-body scintigraphic scan reveals the areas altered by inflammatory activity.
- Joint sonography can reveal joint effusions and can be used to assess soft tissue swelling.

What are the possible symptoms of rheumatoid arthritis?

Early stage rheumatoid arthritis:

- Diffuse, temporary joint pain
- Synovial sheath inflammations
- Malaise
- Patient tires easily
- Symmetrical polyarthritis in finger joints
- Morning stiffness

Full-blown rheumatoid arthritis:

The full-blow stage of all the pathological changes caused by rheumatoid arthritis is characterized by wide variations in the destruction of large and small joints that rapidly inhibit normal activities. The painful restriction of movement and deformation of the hands and feet results in a clear pattern of severe symptoms. Over the course of the disease, the spinal column is also affected in many rheumatoid arthritis patients, in particular the cervical spine. This can result in life-threatening complications in some cases. Since the symptoms of rheumatoid arthritis symptoms in the cervical spine may over longer periods “hide behind” the symptoms of degenerative processes that can affect this part of the spine, it is important to detect rheumatic destruction of the spinal column at an early stage.
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Early diagnosis of rheumatoid processes in the cervical spine can facilitate surgery at a stage in which the local destruction of bony and ligamentous structures, and complications such as spinal cord and nerve compression, are still so minor that a high level of surgical success can be achieved.

· Spinal column symptoms
Rheumatoid arthritis frequently affects the cervical spine, whereby the upper cervical spine (occipital bone, 1st and 2nd cervical vertebrae - C0-C2) is most frequently affected and instability is often observed between the 1st and 2nd cervical vertebrae (atlantoaxial instability). Rheumatoid processes below the 2nd cervical vertebra (C2) cause subaxial instabilities.
The thoracic and lumbar spinal sections are only rarely affected.

What are the effects of rheumatic processes in the upper cervical spine?

The instability in the upper cervical spine is caused by inflammatory rheumatic processes in the atlantooccipital and atlantoaxial joints along with the bony and ligamentous structures that ensure a stable connection between the head and the upper cervical spine.

Anatomy of the cervical spine:

· Cervical spine from the rear

![Cervical spine from the rear](image)

· Dens axis
· Atlas (C1)
· Axis (C2)
· 3rd cervical vertebra (C3)

· 7th cervical vertebra (C7)

· Atlas (C1) from above

· Axis (C2) from above

![Atlas (C1) from above](image)

![Axis (C2) from above](image)
The atlantooccipital joint (C0/C1) connects the occiput (occipital bone, os occipitale, C0) to the joint facets of the 1st cervical vertebra (atlas, C1). The atlantoaxial joint (C1/C2) connects the 1st and 2nd cervical vertebrae.

This section of the cervical spine “carries” the head and is responsible for the high level of mobility of the cervical spine. This is a considerable load that requires a stable connection between the occipital bone and the 1st and 2nd cervical vertebrae that is provided for by a taut and complex ligamentous apparatus.

Ligamentous apparatus of the atlantooccipital and atlantoaxial joints, upper cervical spine from the rear, membrana tectoria removed, the cruciform ligament connects the atlas, axis and occiput, the alar ligaments connect the dens axis with the occiput and the atlas.

- Ligamentous apparatus of the atlantooccipital and atlantoaxial joints

- Occiput, os occipitale
- Cruciform ligaments, crus superius
- Alar ligaments
- Atlas
- Cruciform ligament of the atlas
- Axis
- Vertebral joint capsule

Ligamentous apparatus of the atlantooccipital and atlantoaxial joints, upper cervical spine from the rear, cruciform ligament removed, the ligaments that hold the dens axis (alar ligaments and apical odontoid ligament) are exposed.

- Occiput
- Apical odontoid ligament
- Alar ligaments
- Atlas
- Dens axis
- Axis

Die entzündliche Wucherung des Synovialgewebes mit Pannusbildung führt an der Halswirbelsäule zur Zerstörung von Wirbelbestandteilen, Wirbelgelenken und Bandscheiben, sowie zur Zersetzung des komplexen Bandapparats, wodurch es zur Instabilität kommt.

Im Bereich der Halswirbelsäule unterscheidet man folgende Instabilitätsformen:

- Ventral (anterior) atlantoaxial instability, in which the aggressive effect of the inflammatory pannus tissue can destroy the ligaments between the 1st and 2nd cervical vertebrae (alar ligaments and transverse ligament of the atlas) and the atlantoaxial joint capsules and cause the erosion of the dens axis. This inflammatory destruction causes a shift of the 1st cervical vertebra (atlas) in relation to the 2nd cervical vertebra (dens axis).
Rheumatoid arthritis · Inflammatory diseases

- **Atlantoaxial kyphosis**, a special form of ventral atlantoaxial instability. In an advanced stage this condition can lead to the destruction of the front parts of the 2nd cervical vertebra, whereupon, in the presence of ligamentous and joint capsule instability, the 1st cervical vertebra slips forward and downward over the 2nd cervical vertebra. This slippage causes the 1st cervical vertebra to “upright” itself in the rear vertebral area.

- **Vertical atlantoaxial instability** is where a one-sided destruction of the atlantoaxial joint connections results in instability with rotation of the upper cervical spine. If the atlantoaxial joints are destroyed on both sides, the dens axis may project into the occipital foramen (foramen occipitale magnum), which is referred to as a basilar impression.

- **Subaxial instability**, all rheumatic instabilities of the cervical spine below C2 are called subaxial instabilities. Destruction of the capsuloligamentous apparatus, the facet joints, and the intervertebral discs causes instabilities in the vertebral segments, accompanied by rotational or translational malpositions.

What symptoms result from these cervical spine instabilities?

- Neck pain/headaches
- Sense of instability
- Painful crepitations (“grinding and popping”)
- Blocked mobility
- Saltatory conduction
- Paresthesia (burning, prickling, tingling) and even complete neurological failure in arms and legs as a sign of cervical myelopathy (pressure on the spinal cord)
- Vertebrobasilar insufficiency with vertigo, nausea and nystagmus (rapid involuntary eye movements). This can be considered a sign of insufficient blood supply through the basilar artery and vertebral arteries potentially due to pressure on these vessels arising from shifts in the upper cervical spine.
- Drop attacks, a sudden fall while retaining consciousness, due to basilar artery insufficiency and incipient gait disorder.
- Lhermitte’s sign, a nerve extension pain: When the cervical spine is moved, an electrifying sensation is felt along the entire spine, potentially reaching as far as the buttocks.
- Dyspnea (shortness of breath), dysphagia (swallowing difficulties) and dysphonia (vocal dysfunctions) are signs of bulbar involvement (pressure on the extended spinal cord).
- Bladder and colonic dysfunctions.
- Respiratory paralysis, sudden death, due to acute constriction of the upper cervical medulla.

What diagnostic measures are done if cervical spine instability is suspected?

- Conventional x-ray images:
  - Cervical spine in 2 planes, oblique images, the cervical spine extended and flexed, transoral dens image to assess shifts in the upper cervical spine.
  - With computer tomography (CT) and magnetic resonance tomography (MRT), excellent images are obtainable of bony changes in the vertebrae, destroyed ligamentous structures in the upper cervical joints, the extent of pannus formation, and any compression damage in spinal cord and nerves. CT and MRT can also be used for so-called functional images in overextension and flexion positions of the cervical spine that improve documentation of an instability.
  - Neurophysiological examinations such as evoked potentials, electromyography and transcranial magnetic stimulation (TMS) make it possible to differentiate any nerve or spinal cord damage present.
When is surgical treatment of cervical spine instability indicated?

The aggressive inflammatory destruction of the bony and ligamentous structures of the upper cervical joints increases over the course of the disease, increasing both the occurrence of neurological failures due to compression of nerves and spinal cord and worsening instability. The pannus tissue leads to a fixation of the structures, further complicating surgical procedures.

Indications:
- Persistent strong pains despite adequate therapy
- Confirmed increase of instability
- Confirmed increase of destructive changes in bones and ligamentous apparatus, with increasing deformity.
- Signs of cervical myelopathy (changes in spinal column due to continuous pressure)
- Confirmation of a basilar impression
- Neurological dysfunctions
- Diameter of spinal cord < 6 mm, confirmed by MRT of cervical spine during flexion.

What surgical methods are used to treat the instability of the cervical spine?

Excellent surgical results are obtainable in cases of instability of the cervical spine if a diagnosis is reached early on and surgical intervention is timely as well.

In the presence of existing extensive vertebral, ligamentous and spinal cord damage, surgery can often only stabilize existing damage and prevent the spread of further complications.

There are a number of different approaches available for surgery on the rheumatic cervical spine, which must always be selected based on the specific individual situation.

Here we will describe 3 surgical methods used frequently in our department for the treatment of instabilities of the cervical spine:
- Transoral dens resection with dorsal spondylodesis
- Dorsal decompression with cervical fusion
- Ventral corpectomy with cervical spondylodesis

Other symptoms of rheumatoid arthritis:

- Symptoms in the hands
  - Normal right hand
    - Distal finger joint
    - Proximal finger joint
    - Basal joint
    - Metacarpal bones
    - Wrist bone
Rheumatoid arthritis · Inflammatory diseases

- Arthrosis in a finger joint
  - Normal joint cartilage
  - Destroyed joint cartilage

- Polyarthritis in the basal joints
  - Joint destruction

- Capsule swelling with joint effusions
- Tendovaginitis with swelling of the tendons and tendon sheaths

- Normal tendons, flexor side
  - Normal flexor tendons
  - Normal tendon sheath
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- **Normal tendons, extensor side**
  - Normal extensor tendons
  - Normal tendon sheath

- **Tendovaginitis, flexor side**
  - Inflamed flexor tendons
  - Inflamed tendon sheath

- **Tendovaginitis, extensor side**
  - Inflamed extensor tendons
**Rheumatoid arthritis**  ·  **Inflammatory diseases**

- **Tendovaginitis**
  - Normal tendon with tendon sheath
  - Inflamed tendon and tendon sheath

- **Arthritic destruction of the carpus (wrist joint)**
  - Normal structure of the carpus (wrist)
    - Normal wrist bone
    - Intact joint cartilage

- **Inflammatory destruction of the wrist bone**
  - Destroyed wrist bone
  - Joint surface dissolution
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- Gaenslen’s test induces pain due to inflammation of the basal joints.
- Carpal tunnel syndrome caused by pressure of inflamed tissue on nerves
- Incomplete fist closure due to increasing destruction of joints
- Muscle atrophy in thenar (ball of the thumb)
- Reduction of crude strength
- Arthroses in the proximal interphalangeal joints (Bouchard) and distal interphalangeal joints (Heberden) with distensions around the joints.

• Proximal interphalangeal joint arthrosis (Bouchard)

• Distal interphalangeal joint arthrosis (Heberden)
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· Ulnar deviation of fingers 2-5

Due to the joint destruction and shrinkage of the joint capsules and tendons, the 2nd to 5th fingers are pulled outward (to the ulnar side). This phenomenon is known as ulnar drift.

· Ulnar drift

· Other hand deformities:
  · Swan neck deformity, in which destruction of the joints and tendons results in a fixed overextension of the proximal interphalangeal joints with flexion of the distal interphalangeal joints.
  · Buttonhole deformity, in which the proximal interphalangeal joints are fixed in flexion with the distal interphalangeal joints fixed in overextension.
  · So-called 90°/90° deformation of the thumbs with flexion in the basal joint and overextension in the distal joint.

· Symptoms in feet and ankles

· Anatomy of the extensor tendons of the foot

· Achilles tendon

  Extensor tendons of toes:
  · Extensor digitorum longus
  · Extensor digitorum brevis
  · Extensor hallucis brevis
  · Extensor hallucis longus
**Rheumatoid arthritis · Inflammatory diseases**

- Anatomy of the flexor tendons of the foot
  - Achilles tendon
  - Flexor tendons of the toes:
    - Tendon of the m. tibialis posterior
    - Flexor digitorum longus
    - Flexor hallucis longus
    - Tendon of the m. tibialis anterior

- Normal bony anatomy of ankle joint and foot
  - Shin bone (tibia)
  - Calf bone (fibula)
  - Ankle bone (talus)
  - Scaphoid bone (os naviculare)
  - Heel bone (calcaneus)
  - Cuboid bone (os cuboideum)
  - Metatarsals (metatarsalia)
  - Metatarsophalangeal joints
  - Toes (phalanges)

- Normal bone anatomy of the ankle joint
  - Shin bone (tibia)
  - Ankle joint with joint cartilage
  - Calf bone (fibula)
  - Ankle bone (talus)
Rheumatoid arthritis - Inflammatory diseases

- Arthritis in the ankle joint
  - Osteoarthritis in the ankle joint
    - Inflammation of the ankle joint with destruction of cartilage and bone (osteoarthritis)

- Big toe deformities, hallux valgus and hallux rigidus
  - Hallux valgus, with lateral deviation of the big toe and arthrotic changes in the metatarsophalangeal joint of the big toe
    - Arthrosis in the metatarsophalangeal joint of the big toe with axial deviation of the big toe
  - Hallux rigidus with severe arthrotic changes in the metatarsophalangeal joint of the big toe with no axial malposition in the joint
    - Destruction of the metatarsophalangeal joint of the big toe
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- Deformities of the small toes, hammer and claw toe
  - Toe deformities (claw toe)
  - Toe deformities (hammer toe)

- Windmill vane foot deformity, in which all toes deviate laterally
- Capsule thickening, pain in toe joints
- Tendonitis

- Hip joint symptoms
  - Joint pain, restricted movements
  - Progressive joint destruction

- Normal hip joint
- Hip joint with arthrotic changes

- Trochanteric bursitis
  - Gluteal muscles
  - Head of the femur
  - Pubic bone (os pubis)
  - Ischial bone (os ischii)
  - Neck of the femur
  - Trochanter major
  - Inflamed bursa (trochanteric bursa)
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- Symptoms in the shoulder joint
  - Anatomy of the shoulder from the rear
    - Acromion
    - Coracoid process
    - Head of the humerus
    - Glenoid cavity (cavitas glenoidalis)
    - Upper arm (humerus)
    - Shoulder blade (scapula)
  - Anatomy of the shoulder from the front
    - Clavicle (clavicula)
    - Acromion
    - Coracoid process
    - Glenoid cavity (cavitas glenoidalis)
    - Shoulder blade (scapula)
  - Ligamentous apparatus and bursa, position in relation to one another in shoulder joint when arm is raised
    - Clavicle (clavicula)
    - Coracoclavicular ligament
    - Coracoacromial ligament
    - Subacromial bursa
    - Coracoid
Rheumatoid arthritis · Inflammatory diseases

- Muscle atrophy in rotator cuff

- Muscles in the rotator cuff of the shoulder as seen from the front
  - Clavicle (clavicula)
  - Supraspinatus muscle
  - Subscapularis muscle
  - Infrascapularis muscle

- Muscles in the rotator cuff of the shoulder as seen from the rear
  - Supraspinatus muscle
  - Infrascapularis muscle
  - Teres minor muscle

- Tendovaginitis and bursitis in subacromial bursa
- Painful inflammatory capsule swelling
- Joint destruction with restricted movement

- Inflammatory capsule swelling
  - Inflamed and swollen joint capsule
Rheumatoid arthritis - Inflammatory diseases

- Inflammatory joint destruction

- Symptoms in knee joints
  - Painful capsule swelling
  - Restricted movements due to muscle atrophy with flexion contraction and extension deficit
  - Instability due to inflammatory loss of function in lateral and cruciate ligaments
  - Increasing axial malposition
  - Joint destruction due to pannus formation

- Side view of knee, beginning joint destruction
  - Pronounced pannus formation on femoral condyle with advanced cartilage destruction
Rheumatoid arthritis · Inflammatory diseases

- Symptoms in the elbow joints

- Normal anatomy of the elbow, side view with humerus, ulna, radius and bursa

- Capsule swelling with inflammation of the joint mucosa
  - Inflammation of the olecranon bursa (bursa olecrani)

- Inflammation of the bursa olecrani

- Inhibited rotation and extension deficit of the lower arm
  - Joint destruction with movement restriction

- Elbow, extended, view from above

- Joint destruction
Rheumatoid arthritis - Inflammatory diseases

- Elbow, side view

- Joint destruction

- Symptoms in the skin

  - Rheumatoid nodules
    Rheumatoid nodules are normally observed only in patients with seropositive rheumatoid arthritis. They appear as subcutical nodules, taut and elastic, sometimes readily mobile and sometimes fixed, on the extensor surfaces of the lower arms and in the joints of the knees, elbows, ankles, toes and wrists. Rheumatoid nodules may, in rare cases, also be found in the lungs, heart or muscles.

- Symptoms in the organs

  - Heart: Inflammation of the pericardium (pericarditis) with effusion, rarely inflammation of the endocardium (endocarditis) and heart muscle (myocarditis).
  - Lungs: Pleuritis, changes in lung structure (interstitial pulmonary fibrosis)
  - Eyes: Reduced production of tear fluid, more rarely vascular inflammations (episcleritis)
  - Gastrointestinal tract: Ulcers and hemorrhages that may be caused by treatment with non-steroid antiarthmetics.
  - Nervous system: Nerve compression syndrome in peripheral nerves resulting from pressure exerted by inflammatory tissue.
  - Blood: Chronic inflammatory activity frequently results in low red blood corpuscle counts (anemia) resulting in reduced resilience to stress and shortness of breath.

  - Osteoporosis (bone atrophy), resulting from long-term cortisone therapy and inactivity. Osteoporosis can render patients susceptible to bone and vertebral fractures caused by even minor accident trauma.

How is rheumatoid arthritis treated?

The most important principles of treatment are:

- Improvement and preservation of quality of life
- Pain relief
- Slowing disease progression
- Control of inflammatory processes and episodes
- Preservation of joint mobility
- Early-stage spinal surgical intervention to avoid complications
Conservative and drug treatment

Objectives of conservative treatment:

- Physiotherapy and ergotherapy are used to treat kinetic and motor dysfunctions, prevent of malpositions, and to strengthen the muscles.
- Thermotherapy can be used to treat pain and inhibit inflammation.
- Classical massage, heat and cold therapy are used to loosen up muscles.
- Electrotherapy with low, medium and high frequencies to ease pain.
- Accompanying psychological support with relaxation and coping techniques.
- There are a number of other therapeutic approaches suitable for use in individual cases such as manual therapy, osteopathy, homeopathy, phytotherapy, traditional Chinese medicine, neurotherapy, etc.
- Orthopedic technology can be used in the form of individually fitted orthotic devices, special hand splints, orthopedic shoes, walking aids, and other therapeutic support aids for the treatment of existing or developing deformities and mobility restrictions.

Drug treatment of rheumatoid arthritis is complex, requiring a therapeutic schedule formulated to accommodate the individual patient’s condition. Each schedule also requires regular monitoring due to potentially severe side effects. Normally, the following medication groups are used in combination in individual therapy schedules for the treatment of specific disease phases:

- Basic therapeutics
  The objective of these drugs, which address the immune system, is to slow the strong inflammatory process of the disease, and thus the destructive processes. Use of these drugs should begin soon after diagnosis. Basic therapeutics include:

  - Immunosuppressants (e.g.: methotrexate, cyclosporine, leflunomide, azathioprine) are drugs that suppress the immune defense reaction. Possible side effects:
    - Altered blood counts
    - Inhibition of production of white blood cells
    - Renal and hepatic dysfunction
    - Methotrexate and leflunomide must be discontinued prior to pregnancy since they may cause damage to the fetus.
  - Gold compounds (e.g.: sodium aurothiomalate or auronofin) act to suppress the immune defense reaction. Possible side effects:
    - Inhibition of production of red and white blood cells
    - Inhibition of production of blood platelets
    - Tolerance of these drugs is often poor
  - Antimalarial drugs (e.g.: hydroxychloroquine, chloroquine) also suppress the immune defense reaction. The assumption is that these agents prevent the complexing of antibody and antigen, thus helping to interrupt the autoaggressive inflammatory process of the disease. Possible side effects include:
    - Disturbed vision and stomach discomfort
  - Other immunosuppressants that can be used include penicillinamine and sulfasalazine

- Drugs that block cytokines (messenger substances interleukin-1 and TNF alpha)
  - Tumor necrosis factor (TNF) alpha blockers (e.g.: infliximab, etanercept, adalimumab) inhibit the effect of cytokine, thus slowing the inflammatory tissue destruction process.
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- Interleukin-1 blockers (e.g.: anakinra), which block the effect of interleukin-1, thus slowing the destruction of cartilage and bones.

- Cortisone treatment (e.g.: prednisone)
  Cortisone is a strong antiphlogistic with an immunosuppressant effect.
  Main side effects:
  - Osteoporosis (bone atrophy)
  - Cataracts and glaucoma (diseases of the eyes)
  - Hemorrhages and ulcers in the gastrointestinal tract
  - Thinning of skin, acne
  - Visceral adiposity
  - Diabetes mellitus
  - Hypertension
  - Reduction of white blood cell count

- Non-steroid antirheumatics (NSAR) (e.g.: diclofenac, ibuprofen) have antiphlogistic and analgesic effects.

- Since many antirheumatics attack the stomach mucosa, medication to protect the mucosa is also generally prescribed.

- Modern pain therapies include a number of different approaches to the treatment of acute or chronic pain. An adequate therapeutic schedule is created based on the individual pain situation.

Treatment of rheumatoid arthritis with radioactive agents (radiosynoviorthesis)

This radiological method is used to treat the inflamed joint mucosa (synovia) directly with radiation by injecting radioactive drugs (radionuclides). The radionuclides implant themselves in the cells of the joint mucosa, where they cause sclerosing of the synovia, reducing the size of the proliferating joint mucosa and reducing the inflammation. The radionuclides used are erbium-169, rhenium-186 and yttrium-90, which are injected into the affected joints depending on their duration of action and penetration depth. Radiosynoviorthesis can be done in the small hand and foot joints, the knee and hip joints, and in the shoulder, elbow and wrist joints. Assessment of the efficacy of such a local radiation treatment is possible approximately 3 months after treatment. Most patients report a reduction of pain, depending on how severe the changes in the joint are.

Surgical therapy of rheumatoid arthritis

A number of surgical approaches are available to eliminate instabilities and compression of the spinal cord and nerve compressions in rheumatic disease of the cervical spine.
In our department, we frequently use the following surgical procedures to surgically treat rheumatic diseases of the spine:

- C1/C2 manifestations: dorsal C1/C2 fusion using the Harms technique
The following surgical approaches are used when operating on the large and small joints affected depending on the differing degrees of inflammatory process damage in specific cases:

- **Synovectomy:** The inflamed joint mucosa (synovia) is removed either by joint arthroscopy or in open surgery, thus slowing the process of inflammatory joint destruction.
- **Extensively destroyed joints are frequently replaced with artificial joints (endoprostheses). Both total and partial endoprostheses can be used to replace a joint.**
- **Resection arthroplasty is a procedure involving the partial removal of a joint followed by the plastic construction of a “pseudo-joint.” Such procedures are often used to correct foot deformities (hammer toes, claw toes).**

Surgical arthrodesis in joints is done in special cases. Arthrodesis (stiffening, rigidifying) of the joint always means a loss of mobility, but brings pain relief as well. The residual mobility of the other joints involved must always be considered when performing arthrodesis.