Orthopedics Must Knows

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This book is dedicated to my wife Nadja and my daughters Saena and Dena.

Preface to the First Edition

Musculoskeletal disorders are the most common reason for hospitalizations and, after cardiovascular diseases, the second largest cost driver in healthcare. Every physician will encounter patients with orthopedic pathologies during their career. This necessitates a basic knowledge of musculoskeletal diseases, not least because overlooking these can lead to significant consequences. The challenge for the medical student, the general practitioner or even the specialist in orthopedic surgery is keeping the overview, as orthopedic surgery is a very broad field. This book is a summary of the basic knowledge, the so-called "must-knows" of orthopedic surgery. It serves to maintain an overview for those who are or will become physicians, and as a base for aspiring orthopedic surgeons. Therefore, the focus is on presenting a concise content.

The chapters are written by renowned specialists from the Balgrist University Hospital, who are at the forefront of their specialized field. They are lecturers at the University of Zurich and experienced in teaching and didactics. Special thanks go to them. Thanks also go to Mr. Noah Gieriet, a medical student from Zurich, who brought in the perspective of a medical student and learner, under the supervision of PD Dr. S. Bouaicha. The informative illustrations are thanks to Mrs. Dalkowski, and the creation of the book would not have been so efficient without the constant support of Mrs. Scheddin and the Springer-Verlag.

The first edition of this book is dedicated to Prof. em. Dr. Christian Gerber. He has dedicated his life to orthopedic surgery and has greatly contributed to its evolution.

I wish you an inspiring read and that your curiosity will be ignited for this exciting field of medicine.

Zurich, Switzerland Mazda Farshad

Preface to the Second Edition

In the second updated edition, the basic knowledge for students and non-orthopedic physicians is supplemented with the respective specifically marked "advanced knowledge," specifically aimed as preparation for the Swiss board examination in Orthopedics and Traumatology of the Musculoskeletal System. The book is written in a concise form and includes numerous illustrations.

Special thanks to Dr. Andreas Flury, who has also brought in the perspective of a candidate for the board exams.

I wish you an efficient and inspiring read.

Zurich, Switzerland Mazda Farshad

Preface to the Current Edition

The third edition of this book is the result of a collective national effort. Members of the Swiss Orthopaedic Society (Swiss Orthopaedics) have made significant contributions to this updated version. The content has been translated into English, revised with the latest information, and enhanced with additional illustrations.

I would like to extend special thanks to Dr. Andreas Flury, whose extensive contributions were instrumental in the creation of this third edition. Our gratitude also goes to the members of Swiss Orthopaedics for their valuable input and to the board of Swiss Orthopaedics for their ongoing support.

This book can be seen as a reflection of the "Swiss way" of practicing orthopedic surgery, grounded in a rich and successful tradition.

I hope you find inspiration in its pages, and that the insights within may assist you in providing the best care for your patients.

Zurich, Switzerland Mazda Farshad

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Basic "Must Knows"

1

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Anamnesis

The 90-second rule: Let the patient speak 90 seconds at least, uninterrupted → otherwise, you risk losing important information. Rarely do patients need more than 90 seconds to provide the most important facts (Marvel JAMA 1999 and Imran Ir J Med Sci. 2019).

Orthopedic Examination

1. **Inspection** (Deformities, gait, Varus/Valgus etc.)
2. **Palpation**
3. **Functional** tests (ROM [Neutral-Zero-Method], Joint stability etc.)
4. Specific **neurological** examination

Treatment Concepts

Conservative

- Pharmaceutical/chemical (painkillers, Corticosteroid infiltration etc.)
- Physio-, Occupational therapy
- Physical therapy (cold, heat, TENS etc.)
- Immobilization (cast > splint > bandage) CAUTION: Thrombosis prophylaxis
- Orthosis, corsets: Functional support and guidance (see Chap. 10)

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Surgical

Joint Preservation

Osteotomy, arthroscopy, cartilage reconstructions, ligament reconstructions.

Joint Replacement

Prosthesis (cemented, uncemented: depending on anatomical region and bone density).

Materials in Orthopedics

(decreasing modulus of elasticity): Ceramic > Co-Cr > Surgical steel > Titanium > Cortical bone > Polymers > PMMA (Cement) > Polyethylene > Spongious bone > Tendons/Ligaments > Cartilage.

Wear Behavior of Prostheses

Ceramic-Ceramic 0.0005 mm/year < Metal-Metal 0.005 mm/year (historical) < Polyethylene (highly crosslinked) 0.1 mm/year.

Infections of Prostheses (Parvizi, J Arthroplasty 2018)

“Major criteria”: (one enough for diagnosis)

- 2 positive cultures of the same germ
- fistula

“Minor criteria”:

- Serum: increased CRP, D-Dimer, ESR
- Synovial: **increased leukocytes** (**> 3000** cells/microL) or leukocyte esterase, **positive alpha-defensin**, proportion **polymorphic cells (PMN > 80%)**, increased CRP

Arthrodesis

Arthrodesis angles:

- Shoulder: 15–20° abduction, 20–25° flexion, 40–50° internal rotation
- Elbow: 90° flexion, slight pronation
- Hand: MCP 20–30°, PIP: 40–50°, DIP 15–20°, Dig 1: each approx. 20° flexion
- Hip: 25–30° flexion, 0–5° adduction, 0° rotation.
- Knee: 0–5° valgus, 10–15° flexion
- Ankle: 0° flexion, 5–10° external rotation, 5° valgus

Amputation

See Chap. 10.

Trauma

Osteosynthesis

- Kirschner wire (see pediatric ortho)
- Plate: stable (total stability) or bridging (anatomical stability)
- Cerclage: Conversion of muscle pull force into compression force on fracture gap
- Nailing: intramedullary (static and dynamically locked), usually directly loadable

External Fixator

Construct is more stable if: large pin diameters, pins in different planes, large distance of the pins (2 pins near the fracture and 2 pins as far away as possible ["near-near, far-far"]) and smallest possible distance between bone and rod.

Internal Fixator

For example, in the case of vertebral body fracture, material removal after fracture healing.

Bone Remodeling

Wolff's Law Bone remodeling is dependent on the load: load increase → compaction of the trabeculae.

Hueter-Volkmann Law chronically excessive compression force decreases and tensile force stimulates enchondral growth (important for understanding of scoliosis).

Osteoarthritis

Radiologically: Joint **space narrowing**, **osteophytes**, **subchondral cysts** and **sclerosis**.

Osteoarthritis of a right knee

Aseptic Osteonecrosis

Pathogenesis

Unknown, most likely micro-ischemia (RF: smoking, sickle cell anemia, micro-trauma etc.); Details: see respective chapters.

Eponym	Location	“Typical”
M. Köhler 1	Os naviculare pedis	Mainly boys
M. Köhler 2	Caput metatarsale II > III	Mainly girls
M. Perthes	Femoral head	Mainly boys
M. Schlatter	Tiabial apophysis	Mainly boys
M. Kienböck	Os lunatum	Mainly young men
Osteochondrosis dissecans	Femoral condyle, medial > lateral	Mainly young men
M. Kümmell	Vertebra	Old people

Spine

2

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Anatomy

Bones

33 vertebral bodies	7 cervical (C), 12 thoracic (Th), 5 lumbar (L), 5 sacral (S) (fused) and 4 coccygeal (Co) (fused)
Sagittal Alignment	C: lordosis, Th: kyphosis, L: lordosis, S: kyphosis

Cervical Spine

C1/2	50% of C-spine rotation; stabilizer = Ligamentum transversum
Rheumatoid Arthritis (RA)	CAUTION: Pannus formation in the atlantoaxial (C1/2) joint → may lead to instability (*C-spine X-ray recomended before elective orthopedic surgery in confirmed RA*)

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C2–7	Foramina transversaria (*intraforaminal course of the A. vertebralis from C1 to C6; unilateral dominance of the A. vertebralis: 50 % of the population left, 25 % right, 25 % with similar caliber on both sides*), bifid Proc. spinosi (*except C7*) Placement of pedicle screws from C3-6 is challenging due to small pedicles; an alternative is massa lateralis screws or placement using patient-specific guides or navigation Orientation of facet joint surfaces in the cervical region: 0° coronal, 45° sagittal

Thoracic Spine

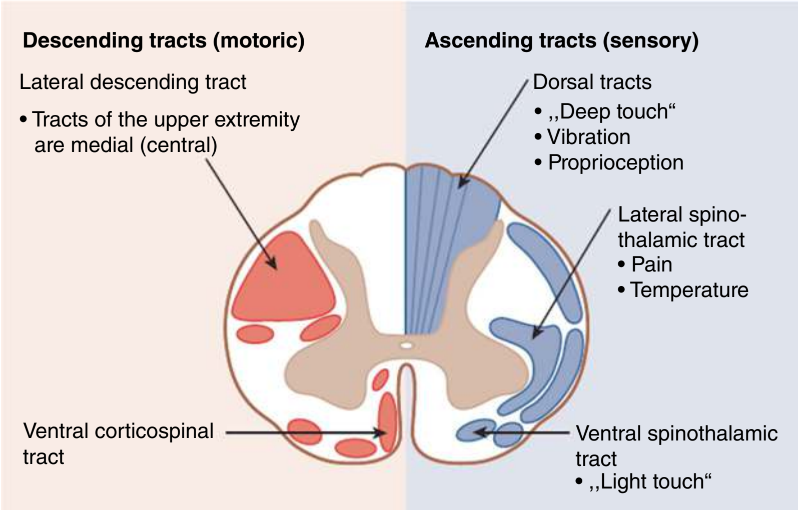
General info	Stiffest area due to rib cage
Thoracic kyphosis	35–45°
Pedicle walls	Twice as thick medially as laterally
Th4	Smallest pedicle diameter at thoracolumbar level
Th12	Has a larger pedicle diameter than L1
Facet joints	Articular surfaces: 20° coronal, 55° sagittal

Lumbar and Sacral Spine

Lumbar lordosis	Approximately 60% of this lordosis is formed in the L4-sacrum segments
Spondylolysis	Defect in the pars interarticularis: Most common cause of back pain in children and adolescents
Ligament structures	Anterior longitudinal ligament (ALL) Posterior longitudinal ligament (*PLL*) Ligamentum flavum (consist mainly of elastin compared to all others, which consist of collagen type I) Interspinous ligament Supraspinous ligament

Facet joints	Articular surfaces: 50° coronal, 90° sagittal
Intervertebral disc	Anulus fibrosus = collagen type I, nucleus pulposus = collagen type II and high amount of polysaccharides and water
Spinal cord (SC)	Extends from the brainstem to approximately L1, where it terminates as the conus medullaris. Below L1: Cauda equina
Ascending pathways (sensory)	*Posterior funiculi* (*dorsal tract*) conduct touch, vibration, and proprioception *Lateral spinothalamic tract* conducts pain and temperature *Ventral spinothalamic tract* conducts fine touch
Descending pathways (motor)	The *lateral and ventral corticospinal tract*s conduct voluntary muscle contraction. The motor function of the upper extremity is more central/medial than that of the lower extremity, therefore the “central cord syndrome” affects the upper extremities more than the lower extremities

Special Features



Sectional organization of the spinal cord with spinal tracts and pathways

Nerve roots	8× C, 12× Th, 5× L, 5× S, 1× Co (= 31 in total). Can be compressed in the recess and/or foraminal/extraforaminal
Cervical	Nerve root exits **above** the corresponding vertebral body (*C6 exits through foramen C5/6*). Nerve root runs horizontally until it exits ➔ *C5/6 disc herniation can compress C6 nerve root both foraminal and recessally*. Nerve root C8 has no corresponding vertebral body (*exits through foramen C7/Th1*)
Lumbar	The nerve roots run vertically, pass the disc compartment above the corresponding vertebral body and exit the intervertebral foramen **below** the pedicle of the corresponding vertebra ➔ *L4/5 disc herniation with recess compression = affected nerve root L5; L4/5 disc herniation with foraminal compression = L4 nerve root affected*

Sagittal Balance

Components are cervical lordosis, thoracic kyphosis, and lumbar lordosis

- Normal alignment: The vertical axis (*C7 sagittal plumb line*) on lateral radiograph runs from the center of C7 within 2 cm of the posterior edge of the S1 endplate. Normal sagittal balance is associated with a better postoperative outcome
- *Positive imbalance*: The C7 sagittal plumb line passes >2 cm anterior to the posterior edge of the superior endplate of S1
- *Negative imbalance*: The C7 sagittal plumb line passes >2 cm posterior to the posterior edge of the superior endplate of S1.

Spinopelvic Parameters

Pelvic Incidence (fixed individual value between 35–85°) = **Pelvic Tilt** (variable) **+ Sacral Slope** (variable), determines the relative position of the sacrum to the femoral heads

- Pelvic Incidence = Angle on the lateral radiograph between the line connecting the center of the upper endplate S1 to the center of the femoral heads and the perpendicular to the center of the endplate S1
- Pelvic Tilt = Angle on the lateral radiograph between the connecting line from the center of endplate S1 to the center of the femoral heads and the vertical
- Sacral Slope = Angle on the lateral X-ray between the endplate S1 and the horizontal
- Pelvic Incidence = Lumbar lordosis ±10°. Important for sagittal correction, where lumbar lordosis ±10° should be adjusted to the individual's pelvic incidence

Back Pain

Table: Epidemiology

Lifetime prevalence >80%	
Age of the patient	Most common pathology
Children	Congenital or developmental disorders Infections
Young patients	Disc herniation Spondylolysis/spondylolisthesis Fractures Scheuermann's disease
Older patients	Spinal stenosis Osteochondrosis Facet joint arthrosis Osteoporotic fractures Bone metastases

Special Features

- > 50% of all patients who seak medical care for back pain recover within a week, 90% within 1–3 months
- 50% of all patients with radicular pain recover within 4–6 weeks. If symptoms persist after 6 weeks of conservative treatment, further evaluation (*e.g., MRI*) is indicated. Earlier if motor deficits are present.
- Red flags: Infection (*i.v. drug abuse, fever, chills*), tumor (*B symptoms*), trauma, cauda equina syndrome

Differential Diagnoses

Urological (e.g. nephrolithiasis), gynecological, gastrointestinal, vascular (e.g. aortic aneurysm), other orthopedic (e.g. coxarthrosis, trochanteric bursitis) disorders.

Spinal Stenosis

Epidemiology

Prevalence:

- > 40 years = 4%
- 60–69 years = 19.4%
- > 80 years = > 80%

Mostly affects L4/5. Risk factors: Caucasians, obesity, congenital spinal anomalies.

Etiology

Central stenosis: hypertrophied ligamentum flavum, facet joint osteoarthritis or cysts, herniated disc, degenerative spondylolisthesis.

Pathogenesis

Dural sac compression.

Clinical Presentation

Causes lumbar pain or bilateral buttock pain +/− paresthesia with prolonged walking/standing (*especially in the buttocks and thighs*). Inclination leads to pain relief (*spinal canal volume increases as ligamenta flava and dorsal disc portions are stretched*).

Characteristics of neurogenic claudication to distinguish it from intermittent claudication (*vascular claudication*): Pain radiates from proximal to distal. Foot pulses are palpable. No pain with cycling.

Classification

According to Schizas (on axial MRI)

- **Grade A:** no or minimal stenosis
  - A1: Nerves are dorsal, filling < 50% of the dural sac
  - A2: Nerves are dorsal in a horseshoe shape
  - A3: Nerves are dorsal, filling > 50% of the dural sac
  - A4: Nerves are central, filling nearly the entire dural sac
- **Grade B:** moderate stenosis, nerves fill the entire dural sac, but can still be individually demarcated
- **Grade C:** severe stenosis, nerves cannot be delineated, no cerebrospinal fluid visible, posterior epidural fat present
- **Grade D:** extreme stenosis, as grade C, but without posterior epidural fat

Diagnosis

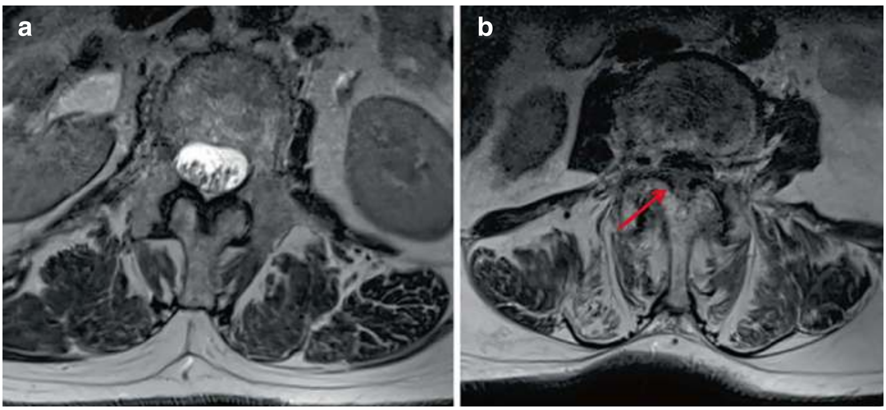
Medical history and MRI or Myelo-CT (*if MRI not possible due to pacemaker or neurostimulator*).

Therapy

Conservative Epidural infiltration (corticosteroids).

Operative

- Decompression
  - Indication: not conservatively treatable or neurological deficits
  - Procedure: decompression, additional fusion for segment instability (instable spondylolisthesis, resection > 50% of the facet joints)
  - Alternatively, vertebropexy as a semi-rigid ligamentous fixation for selected indications



78 year old patient with spinal claudication, caused by a severe spinal stenosis L3/4 (**b**), compared to a normal segment at L2/3 (**a**)

Recessal Stenosis

The recess is defined by the following anatomical boundaries

- Posterior = Proc. superior of the facet joint (SAP)
- Medial = dural sac
- Lateral = pedicle and foramen
- Anterior = posterolateral vertebral body

Recessal (or posterolateral) stenoses result in compression of a single nerve root (e.g., L5 at L4/5).

Therapy

Conservative Epidural infiltration or nerve root injection.

Operative

- Decompression by laminotomy and recessotomy
  - Indication: conservatively unmanageable or neurological deficits
  - Alternatively, endoscopic decompression
  - Complications: dural tear (about 4%). No long-term negative consequences for the patient when treated with a watertight closure

Patient with a recessal stenosis at L4/5

Foraminal Stenosis

The foramen is defined by the following boundaries

- Superior/inferior = pedicle
- Posterior = facet joint
- Anterior = vertebral body and disc

Foraminal stenoses result in compression of a single nerve root (e.g., L4 at L4/5).

Therapy

Conservative Nerve root infiltration.

Operative
- Fusion with implantation of an intervertebral cage to expand the neuroforamen if not controllable by extraforaminal decompression
  - Complication: Risk of adjacent segment degeneration >30% within 10 years
  - Alternatively, endoscopic techniques

Spondylolisthesis

Pathogenesis/Definition

Sliding of the cranial vertebral body over the caudal vertebral body.

Table: Classification

Wiltse-Newman	
Type I:	dysplastic = congenital defect of the pars
Type IIA:	isthmic = fatigue fracture of the pars (mainly affecting **L5**), with **spondylolysis** = stress fracture of the pars interarticularis (*often associated with repetitive hyperextension, e.g. ballet*)
Type IIB:	isthmic = elongation of the pars
Type IIC:	isthmic = acute fracture of the pars
Type III:	degenerative = facet joint instability without fracture of the pars (mainly affects **L4/5**)
Type IV:	traumatic = acute fracture of the posterior arch
Type V:	neoplastic = pathological destruction of the pars

Meyerding	
Grade I:	< 25%
Grade II: – 25	50%
Grade III: – 50	75%
Grade IV: – 75	100 %
Grade V:	**Spondyloptosis** (*complete slippage of the upper vertebral body without contact to the lower vertebral body*)

- Progression occurs primarily in adolescence, rarely after achieving skeletal maturity

Clinical Presentation

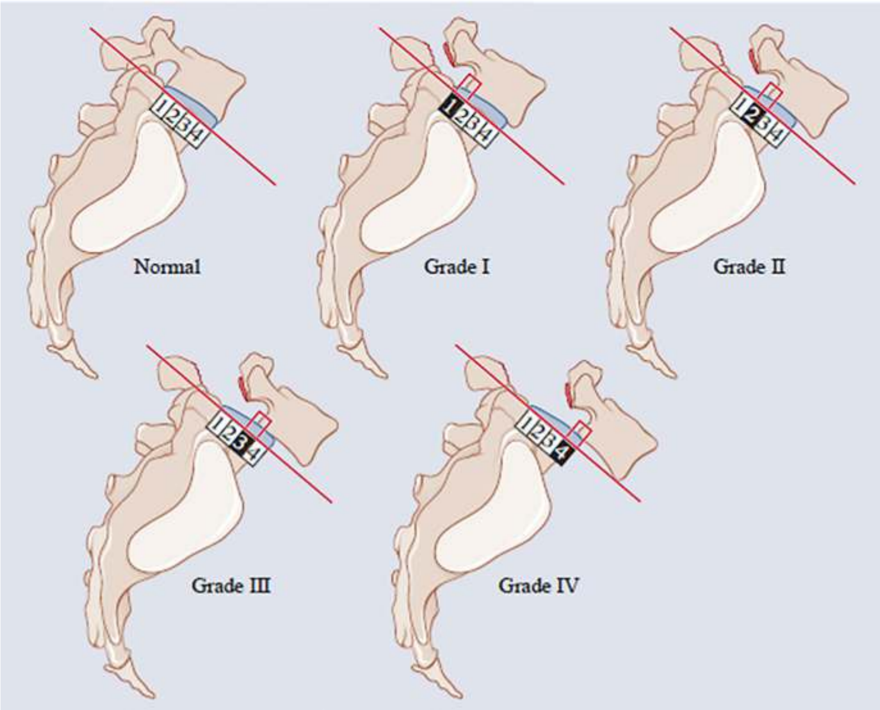
The most common symptom is mechanical back pain (typically *when standing up from a bent position*)**,** associated with dynamic compression of neural structures.

Diagnosis

- In the oblique view on X-ray: “*Scotty-Dog*” appearance in spondylolysis cases
- *Instability* = lumbar flexion−/extension X-rays with translation of >3 mm and/or bilateral facet joint effusions >2 mm on MRI

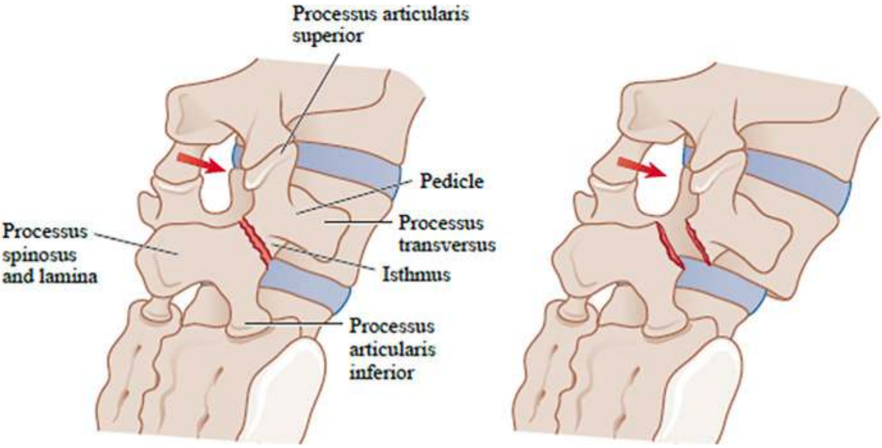
Table: Therapy

Classification according to Meyerding	Therapy	Indications for surgery
I/II	**Conservative:** Activity modification and physiotherapy **Operative:** “Pars repair” with tension screw and bone grafting (*Buck procedure*) in adolescents without degeneration of the disc (up to 20 years), then fusion	(1) Persistent pain despite exhaustion of conservative therapy (2) Neurological deficits
III/IV	**Conservative:** Activity modification and physiotherapy **Operative:** Spinal fusion with neuromonitoring	See above



Meyerding classification of spondylolisthesis with sliding of the cranial vertebral body over the caudal vertebral body

- In spinal fusion with sagittal correction, it is important to restore global and spinopelvic balance (see ▶ Section "Sagittal Balance"). The *C7 sagittal plumb line* should be within the normal range and the lumbar lordosis should be within ±10° of the individual pelvic incidence
- Complications of spinal fusion:
  - Risk of adjacent segment degeneration >30% within 10 years
  - Pseudarthrosis rate: 5–35% (risk factor #1: smoking)



Pathogenesis of isthmic spondylolysis and spondylolisthesis: fracture of the pars interarticularis with subsequent sliding of the cranial vertebral body

Cervical Pathologies

Degenerative Changes

Pathogenesis

Intervertebral disc degeneration, uncovertebral osteoarthritis and facet joint arthrosis.

Clinical Presentation

- Discogenic neck pain; radiculopathy due to nerve root compression; myelopathy due to spinal cord compression
- Most common site of degenerative changes: **C5/6**
- Acute radiculopathies are typically caused by herniated discs
- Ossification of the PLL results in cervical spinal canal stenosis and myelopathy (*OPLL*), more common in Asian populations, unclear etiology: i.e., multifactorial (associated with diabetes, obesity, low calcium intake)

Therapy

Conservative for minimal symptoms

- NSAIDs, physiotherapy (isometric strengthening)
- Radiculopathy: Indirect extraforaminal nerve root block
- Cervicalgia: Facet joint infiltrations

Operative for myelopathy or nerve compression

- Indication: radiculopathy with <M4, conservatively exhausted, painful radiculopathies, cervical myelopathy
- Procedure: Anterior cervical discectomy and fusion or prosthesis (= ACDF/ACDP) or for acute (<3 months) lateral disc herniation: dorsal foraminotomy according to Frykholm or endoscopic sequestrectomy
- Multisegmental laminectomy (± fusion) for multisegmental spinal canal stenosis
- Risks:
  - With anterior approaches to the cervical spine: Injury to the recurrent laryngeal nerve ➔ hoarseness and raspy voice due to unilateral vocal cord paralysis. Because the recurrent laryngeal nerve originates at the level of the subclavian artery on the right and the aortic arch on the left, left anterior approaches to the cervical spine are preferred. If a contralateral anterior approach must be used for revision surgery, a preliminary ENT evaluation is indicated
  - With anterior approaches to the cervical spine: injury to the vertebral artery, esophagus (CAUTION: immediate repair or risk of mediastinitis), sympathetic trunk (Horner's triad with ptosis, miosis, enophthalmos), temporary dysphagia
  - A particular complication of laminectomy is a transient C5 motor radiculopathy of the M. deltoideus (*"C5 palsy"*), which occurs in about 5%. This is most likely due to traction on the C5 nerve root due to dorsal migration of the spinal cord as a result of removal of the posterior structures

Cervical Myelopathy

Pathogenesis

- Spinal cord compression (degenerative, congenital, OPLL, tumor, abscess, trauma) with gradual worsening of symptoms and also stable intervals
- *Snake eye myelopathy* is the result of a longstanding cervical spinal canal stenosis and is characterized by cystic myelomalacia due to ischemic conditions in the central myelon due to the compression in the spinal canal
- Tandem stenosis in 20% with additional lumbar spinal canal stenosis

Clinical Presentation

- Triad: *Sensory disturbances* in the hands. *Deterioration of fine motor skills* (buttoning, " *clumsy hands* "), *gait instability* (broad-based unsteady gait), often also bladder dysfunction
- Examination: Hyperreflexia, clonus, positive Babinski sign, positive Lhermitte sign (sudden shooting sensation with forced neck flexion)

Classification

Japanese Orthopaedic Association (JOA) classification: Point score (max. 17 = normal) for upper extremity motor function, lower extremity motor function, and sensory bladder function.

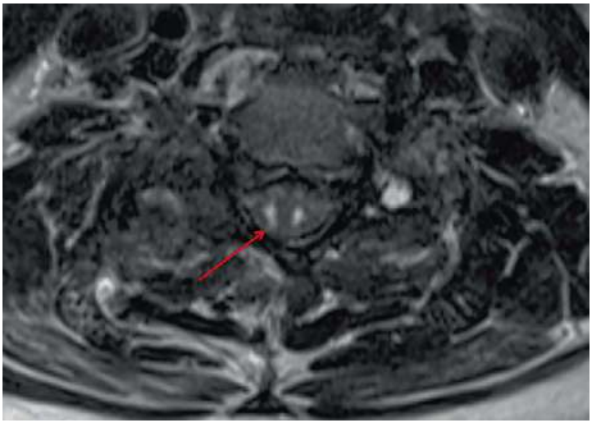
Diagnosis

MRI to evaluate spinal canal stenosis, neurophysiologic testing to evaluate conduction disorders.

Therapy

Conservative in patients without functional limitations, prevention of trauma/falls with possible plegia.

Operative with functional limitation, progression of clinical symptoms, or neurophysiologic confirmation of myelopathy, to prevent neurologic deterioration.



Snake-Eye myelopathy at C4/5

Thoracolumbar Pathologies

Disc Herniation

Disc protrusion	Protrusion of the nucleus pulposus with an intact anulus fibrosus
Disc extrusion	Protrusion of the nucleus pulposus with a ruptured anulus fibrosus
Sequester	No contact of herniated disc material with the disc

- Typically at **L4/5** or **L5/S1** levels (95%) and often posterolateral herniation (*recessal*). This affects the lower nerve root, therefore L5 is affected by a herniated disc at the L4/5 level

Prognosis

90% of patients show a spontaneous improvement of symptoms within 3 months. Less likely if symptoms persist > 6 weeks. Sequestered discs have the highest potential for spontaneous reabsorption. The risk of recurrence is 15%.

Therapy

Conservative For painful radiculopathy

- Corticosteroid infiltration with nerve root infiltration, analgesia, and physical therapy

Operative for radiculopathy with sensorimotoric component (<M4)

- Microsurgical or endoscopic sequestrectomy

Osteochondrosis

Pathoanatomy

Segmental degeneration of the endplates adjacent to the disc with clearly visible "***Modic***" changes (= inflammation of the bone marrow) on MRI.

Therapy

Conservative NSAIDs, physiotherapy (isometric strengthening), chiropractic. For radicular pain: nerve root infiltration. For back pain due to facet syndrome: facet joint infiltrations.

Operative if resistant to conservative treatment or associated with motoric radiculopathy < M4 and or myelopathy

- Surgical technique: disc replacement vs. spinal fusion vs alternative semi-rigid stabilization techniques

Cauda Equina Syndrome

Pathogenesis

Overall rare, mainly affecting **L4/5** due to massive space occupying lesion in the spinal canal *(central large disc herniation, epidural hematoma, tumor, facet joint cyst, abscess, trauma, spondylolisthesis with cauda equina compression).*

Clinical Presentation

- Bilateral gluteal and leg pain with bladder and rectal dysfunction, saddle anesthesia and, varying degrees of motor and sensory dysfunction

Diagnosis

- Clinical examination
  - Sensomotoric function
  - Digital rectal examination and perianal sensation testing
  - Post-void residual urine volume measurement (normal post-void residual volume 50–100 ml)
- Radiographic examination
  - MRI

Therapy

Immediate placement of a catheter for residual urine, **emergency** spinal decompression surgery.

Prognosis

Delayed diagnosis and therapy can lead to lifelong limitations (*motor function of the lower extremities, bladder, and rectal dysfunction*), even early surgery does not guarantee complete recovery of neurological deficits. The presence of saddle anesthesia or bladder dysfunction is associated with a worse outcome.

CAVE
- Cauda equina syndrome = surgical emergency! → immediate surgery
- Saddle anesthesia, bladder and rectal dysfunction
- In case of residual urine, insert permanent catheter immediately to avoid neurogenic bladder dysfunction

Fractures

Approach and Management

There are different classifications and therapeutic algorithms depending on the type of fracture.

Hereafter the AO Spine Injury Classification Systems is summarized.

The AO Spine Injury Classification Systems were developed and funded by AO Spine through the AO Spine Knowledge Forum Trauma, a focused group of international spine trauma experts. AO Spine is a clinical division of the AO Foundation, which is an independent medically-guided not-for-profit organization. Study support was provided directly through AO Network Clinical Research and AO ITC, Clinical Evidence.

Cervical Spine Fractures

AO Spine Upper Cervical Injury Classification System

- Type A injuries: purely bony lesions
- Type B injuries: bony and ligamentous injuries
- Type C injuries: injuries with displacement of the spine in any direction.

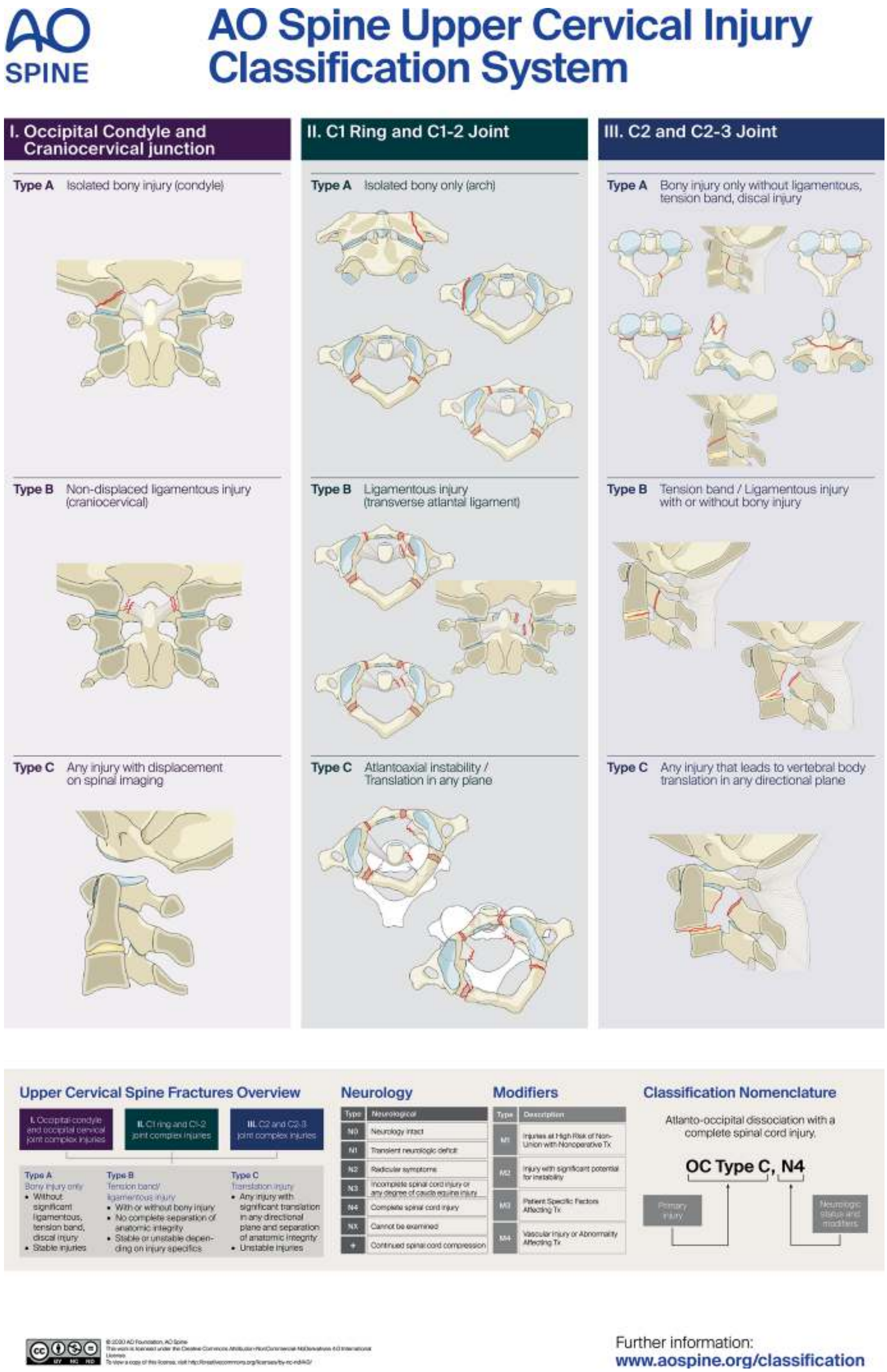
Modifiers:

- M1 Injuries at high risk of Non-union with nonoperative Tx
- M2 Injury with significant potential for instability
- M3 Patient-specific factors affecting treatment
- M4 Vascular injury or abnormality affecting treatment

Neurological status:

- N0 Neurology intact
- N1 Transient neurologic deficit
- N2 Radicular Symptoms
- N3 Incomplete spinal cord injury or any degree of cauda equina
- N4 Complete spinal cord injury
- Nx Cannot be examined (ie intubated patient)

+ Persistent Spinal Cord compression.



Classification of upper cervical injuries according to the AO Classification System

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Data based on bibliographic list available at the end of the chapter*.

Following paragraphs include additional information for specific fractures:

C1 Fractures

Symptoms may include C2 radiculopathy (*numbness and pain radiating occipitally*) and dizziness, doubled vision, ataxia, weakness, dysphagia, and nausea (*vertebral artery injury*).

Classification (acc. Landell)

- Type I: Isolated fracture of the anterior or posterior arch, stable fracture type
- Type II: **Jefferson burst fracture** (*anterior* and *posterior arch fractured due to axial loading*). Stability ensured with integrity of the transverse ligament.
  - If the transverse ligament is torn, the Dickman classification is used to make treatment decision

Type I	Intrasubstantial tear,
Type II	Bony avulsion at the lateral mass of C1

- Type III: Unilateral fracture of the lateral mass, stability provided by the integrity of the transverse ligament

(A combined dislocation of the two lateral masses **>7 mm** on conventional AP radiographs indicates **transverse ligament rupture**)

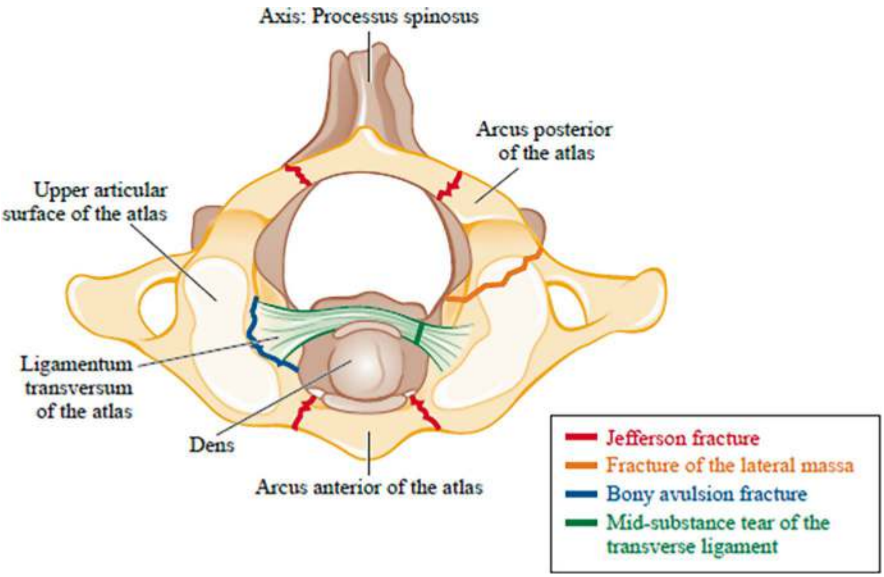
Therapy

Conservative Type I, type II with intact transverse ligament, stable type III.

- Hard neck collar

Operative

- Type II with Dickman type I or unstable type III: C1–2 fusion
- Dickman type II: Halo fixation



Different fracture types of C1

C2 Fractures

Traumatic Spondylolisthesis of Axis (*Hangman's Fracture*)

- Bilateral fracture of the pars interarticularis of C2
- Classification according to Levine and Edwards
  - **Type I:** < 3 mm horizontal dislocation, C2/3 disc intact
  - **Type II:** > 3 mm horizontal dislocation, vertical fracture line, C2/3 disc and PLL are injured
  - **Type IIA:** no horizontal dislocation but marked angulation (>10°), horizontal fracture line
  - **Type III:** Type II and bilateral C2/3 facet joint dislocation

Therapy

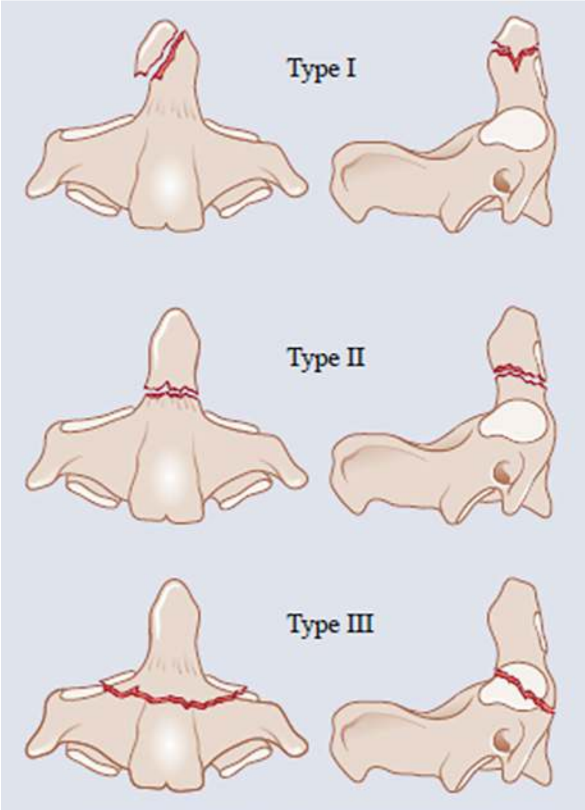
- Type I: hard neck collar for 6 weeks
- Type II: if <5 mm dislocation—reposition by traction and halo fixator for 6–12 weeks; if >5 mm dislocation: surgery
- Type IIA: Reduction with gentle axial compression and hyperextension, then halo fixator for 6–12 weeks
- Type III: Surgery with anterior and/or posterior fusion

Dens Fractures

- Most common fracture of the cervical spine in elderly patients
- Differential diagnosis: Os odontoideum
- Vascular watershed between the apex (A. carotis interna) and base (A. vertebralis) of the dens that interferes with healing (high pseudarthrosis rate)
- Classification according to Anderson-D'Alonzo:
  - **Type I:** Bony avulsion of the alar ligaments
  - **Type II:** Fracture of dens base (high pseudarthrosis rate)
  - **Type III:** fracture extending into the C2 vertebral body
- Grauer classification for "type II" fractures:
  - **Type IIA:** undisplaced
  - **Type IIB:** displaced with fracture line from anterosuperior to posteroinferior
  - **Type IIC:** displaced with fracture line from anteroinferior to posterosuperior

Therapy

- Type I, IIA in older people and III: hard neck collar for 6–12 weeks
- Type IIA in younger people (< 40 years): Halo fixation for 6–12 weeks
- Type IIB: anterior screw fixation
- Type IIC: posterior C1/2 fusion
- Type III: highly dependent on pattern, can often be treated conservatively if not disclocated



Classification of Dens fractures according to Anderson-D'Alonzo

AO Spine Subaxial Injury Classification System

- Type A injuries: purely bony lesions
- Type B injuries: bony and ligamentous injuries
- Type C injuries: injuries with displacement of the spine in any direction.

A special feature in assessing injuries of the subaxial spine is the evaluation of the facet joints. The severity of facet joint injuries is assessed in ascending order from F1 to F4.

Modifiers:

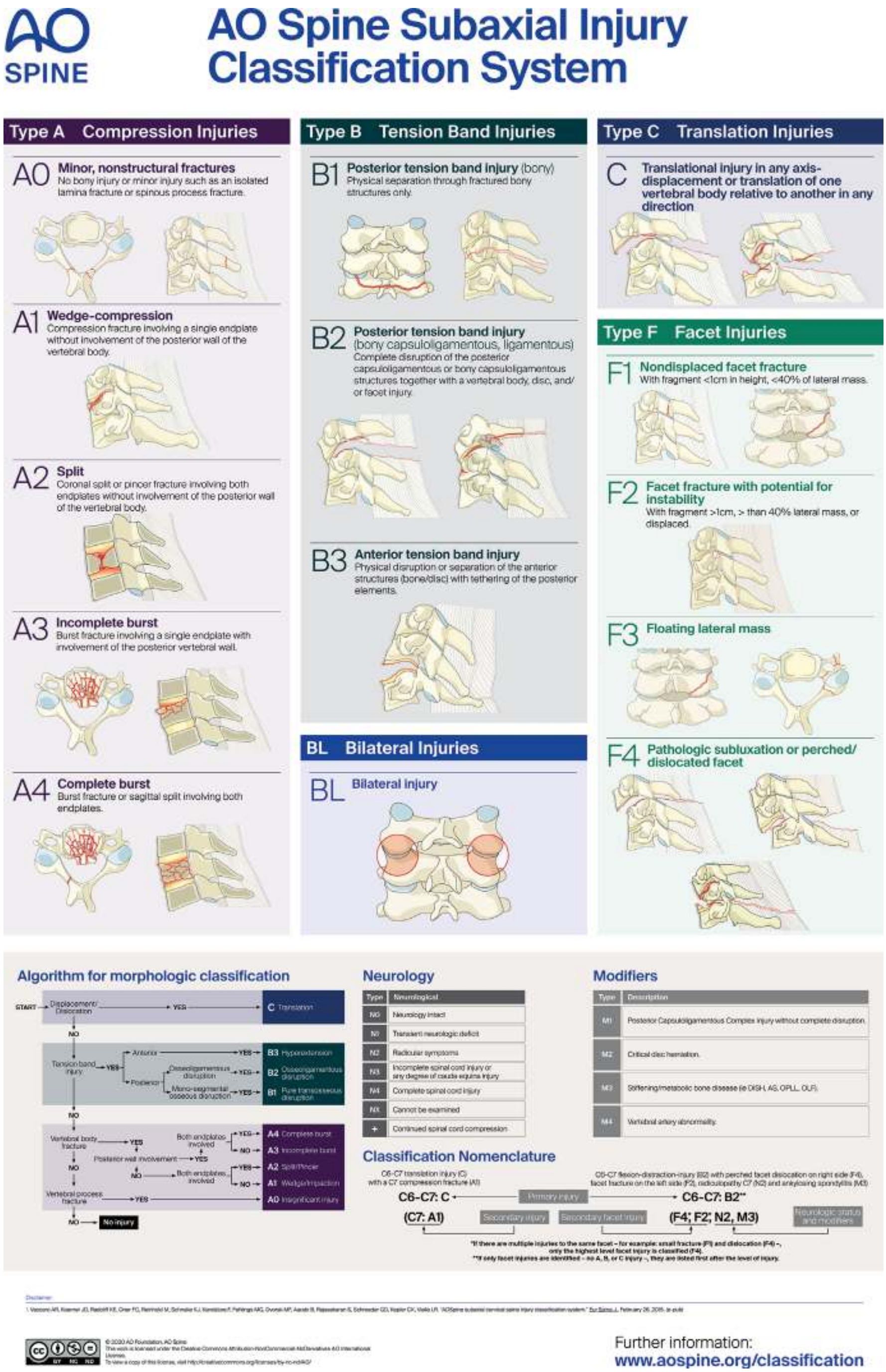
- M1: Posterior Capsuloligamentous Complex (PCC) injury without complete disruption.
- M2: Critical disc herniation.
- M3: Stiffening/metabolic bone disease (ie DISH, AS, OPLL, OLF).
- M4: Vertebral artery abnormality.

The assessment of the neurologic status is the same as previously described (N0-N4 with +).

Type A0, A1 and A2: usually treated conservatively, by close follow up within the first 6 weeks.

A3 and A4: are more likely to be treated surgically.

B/ C injuries: are considered unstable and therefore usually treated surgically. An exception poses the B3 injuries (hyperextension mechanism); if they occur without accompanying facet fractures and are stable on weight-bearing radiographs, conservative therapy with appropriate follow-up can be pursued.



Classification of subaxial injuries according to the AO Classification System

 Available at: https://www.aofoundation.org/spine/clinical-library-and-tools/aospine-classification-systems

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Data based on bibliographic list available at the end of the chapter*.

In addition, clinical decision making can be supported by the SLIC classification:

Table: Classification and surgical decision-making with the Subaxial Cervical Spine Injury Classification (*SLIC*) System

Description	Points
Morphology	
No anomaly	0
Compression	1
Burst	2
Distraction	3
Translation	4
Integrity of the disc-ligamentous complex	
Intact	0
Undetermined	1
Ruptured	2
Neurologic status	
Intact	0
Nerve injury	1
Complete spinal cord injury	2
Incomplete spinal cord injury	3
Persistent spinal cord compression	+1
Score	**Interpretation**
0–3	Conservative
4	Surgical vs. conservative
> 4	Surgical

CAUTION
- For facet joint fractures, imaging is indicated to rule out injury to the vertebral artery (Angio MR or CT)
- For vertebral artery dissection: Aspirin and neurological consultation

Thoracolumbar Fractures

AO Spine Thoracolumbar Injury Classification System

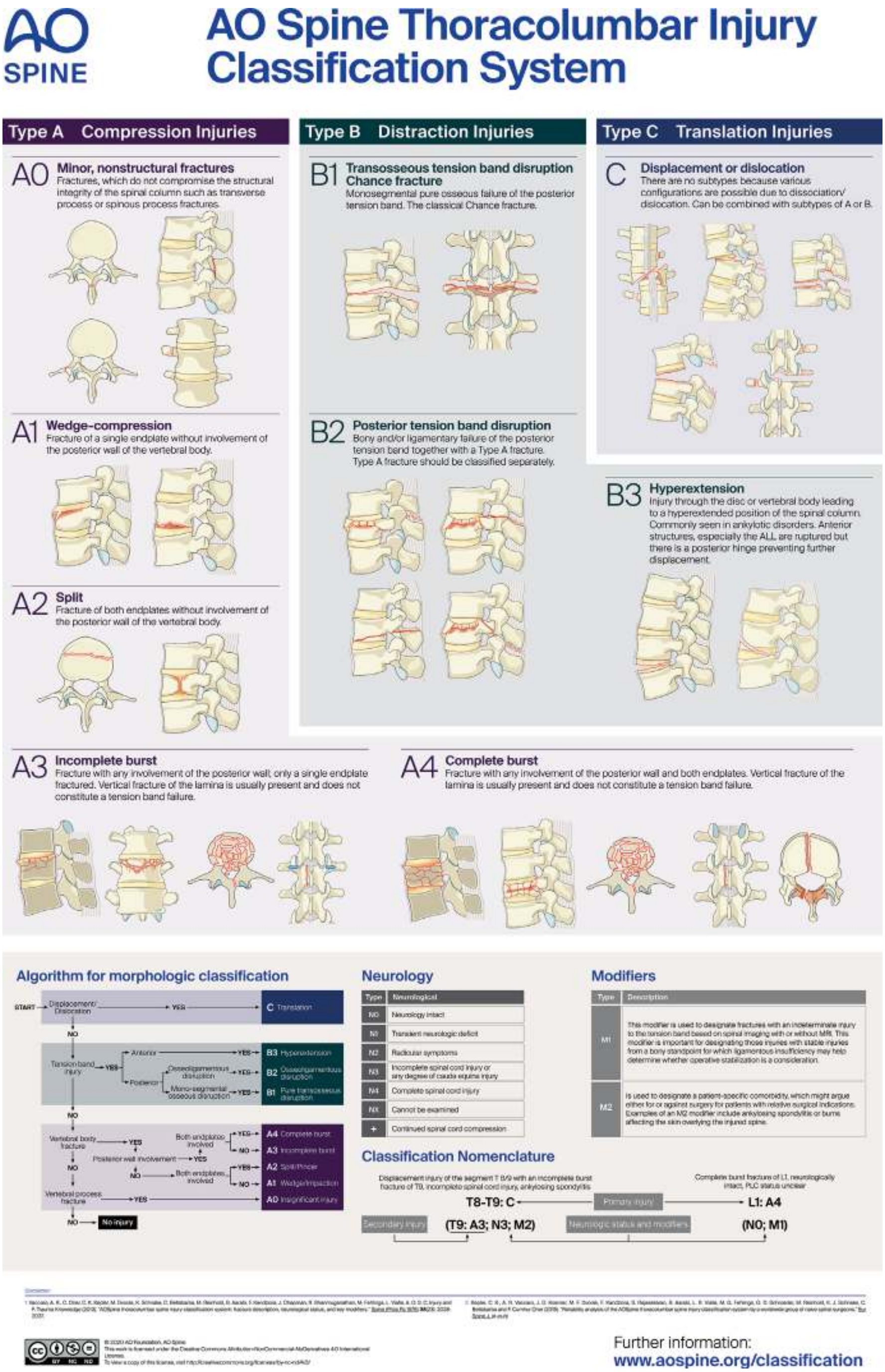
- **Type A** injuries: occur primarily due to axial compression and mainly cause compression fractures of the anterior vertebral body with increasing severity from A1 to A4
- **Type B** injuries: involve flexion or extension moments acting on the spine. This leads to involvement of the posterior structures and thus a higher degree of instability.
- **Type C** injuries: displaced and unstable in multiple planes. Neurological deficits frequently occur.

The transition from A3 and A4 injuries to Type B injuries is gradual, and detecting a possible injury of the posterior ligamentous complex (PLC) can be challenging (use MRI etc).

Injuries with neurological deficits should be treated surgically with decompression and stabilization of the spine. Type B and Type C injuries are often managed with posterior spinal fusion.

There are no internationally recognized guidelines for the treatment of A2, A3, and A4 injuries.

CAVE
In hyperossifying diseases of the skeletal system (ankylosing spondylitis or Forestier’s disease (DISH)), the likelihood of an unstable spinal injury is increased. A CT scan of the entire spine should be performed. Injuries of the rigid spine are usually unstable and should be surgically stabilized with a long segment stabilisation (three above and three below).



Classification of thoracolumbar injuries according to the AO Classification System

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Additionally, the TLICS Score can support in clinical decision making:

Table: Thoracolumbar Injury Classification System (*TLICS*)

Description	Points
Morphology	
Compression	1
Burst	2
Translation/rotation	3
Distraction	4
Integrity of the posterior ligamentous complex	
Intact	0
Possibly ruptured	2
Ruptured	3
Neurologic status	
Intact	0
Nerve root injury	2
Complete spinal cord injury	2
Incomplete spinal cord injury	3
Cauda equina	3
Score	**Interpretation**
0–3	Conservative
4	Surgical vs. conservative
> 4	Surgical

Therapy

Conservative For stable compression and burst fractures without neurological deficit and without focal kyphosis, intact posterior ligamentous complex and TLICS ≤ 3

- 3-point corset (for analgesia only, no effect on kyphosis) for 8 weeks

Operative For persistent pain despite conservative therapy, neurologic deficits, focal kyphosis, rupture of the posterior ligamentous complex, and unstable fractures (TLICS > 4)

- Surgical stabilization (instrumentation) +/− Fusion
  - Posterior approach: for injuries to the posterior ligament complex, especially below the conus
  - Anterior/lateral /transpedicular approach: in case of neurological deficits due to anterior compression, kyphosis > 30°, to reconstruct the anterior column

– *CAUTION: pay attention to the height of the diaphragm*
– Possibly combined anterior (corpectomy) and posterior (fusion)
– Vertebro−/kyphoplasty: Only for absolutely (pain-wise) uncontrollable compression fractures

For pathologic fractures, an osteoporosis evaluation or biopsy (for neoplastic fractures) should be performed. The *Spinal Instability Neoplastic Score* (*SINS*) is used to assess the stability of neoplastic pathologic fractures

Osteoporotic Fractures

AO Spine-DGOU Osteoporotic Fracture (OF) Classification System

- OF 1: No deformation of the vertebral body; diagnosis is made via MRI
- OF 2: Mild compression of an endplate with only slight involvement of the posterior wall (<20%)
- OF 3: Advanced compression with significant involvement of the posterior wall (>20%)
- OF 4: Loss of structure of the vertebral body with involvement of both endplates
- OF 5: Distraction/rotation injuries also involving the posterior structures of the spine

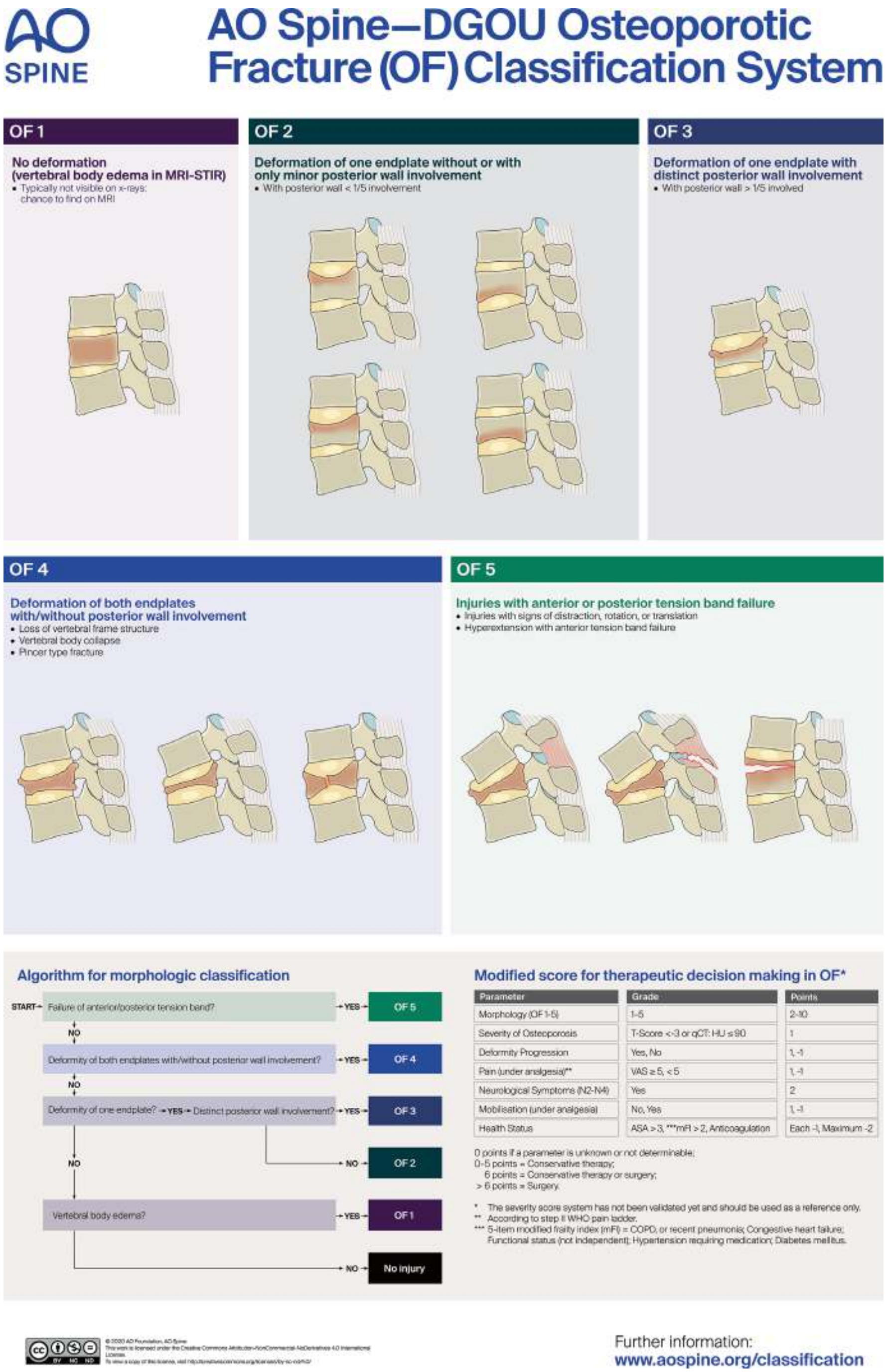
OF 1 injuries can be treated conservatively, and OF 5 injuries require surgical intervention.

For therapeutic decisions in OF 2 to OF 4 injuries, the OF Score offers additional guidance

- Score </=5: conservative
- Score 6: conservative
- Score </= 7: surgical

Conservative treatment: 1–2 clinical and radiological follow-ups within 6–8 weeks. At each new patient contact, the OF classification and the OF Score should be recorded accordingly, and the decision should be re-evaluated. There is little evidence so far for the benefit of using an orthosis. A mandatory assessment of bone metabolism should be conducted, and if possible, osteoanabolic therapy should be initiated.

Surgical treatment: If the posterior elements of the spine are involved, spinal fusion is usually recommended. For simple fractures (OF 2/OF 3), a vertebroplasty/kyphoplasty is often sufficient.



Classification of osteoporotic fractures (OF) according to the AO Spine—DGOU Classification System

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Data based on bibliographic list available at the end of the chapter*.

Spinal Cord Injuries

A thorough neurological examination to document the most distal remaining functional (= undamaged) level is critical and is performed according to the standards of the American Spine Association.

Classification of the *American Spinal Injury Association* (*ASIA*):

- **ASIA A:** 0/5 motor function, complete sensory deficit
- **ASIA B:** 0/5 motor function, incomplete sensory deficit
- **ASIA C:** > 50% of key muscles below neurological level have motor function M < 3, incomplete sensory deficit
- **ASIA D:** > 50% of key muscles below neurological level have motor function M ≥ 3, incomplete sensory deficit
- **ASIA E:** no motor deficit, no sensory deficit

Spinal Shock

- Begins immediately after spinal cord injury and lasts up to 3 weeks, consisting of flaccid paralysis, bradycardia/hypotension, and areflexia
- The return of the *bulbocavernosus reflex* indicates the end of spinal shock (*contraction of the anal sphincter when the glans penis is squeezed or the urinary catheter is pulled).* Only then the neurological level can be determined
- After the end of the spinal shock, spasticity, hyperreflexia, and clonus increase over days to weeks

Neurogenic Shock

- Loss of sympathetic tone (< *vasodilation leads to hypotension and bradycardia*), which can be fatal. This is distinct from hypovolemic shock, which is caused by hypotension and tachycardia. Treatment consists of careful fluid management and the administration of vasopressors

Complete Spinal Cord Injury

- No function below a certain level. Spinal cord injury above **C5** often requires intubation.
- Therapy: Consists of careful hemodynamic monitoring and avoidance of hypotension. The use of high-dose cortisone is controversial. Decompression may improve neurological deficits up to 2 levels
- Prognosis: Improvement +1 level in 80%, +2 levels in 20%. Conus medullaris syndrome has a better prognosis for recovery. The most important prognostic factor for neurological recovery is whether the injury is complete or incomplete

Incomplete Spinal Cord Injury

- Partially preserved motor or sensory function below a certain level, especially the anal sphincter contraction (*"sacral sparing" distinguishes the complete from the incomplete spinal cord injury*)

Central Cord Syndrome

- Most common incomplete spinal cord injury
- Greater motor and sensory loss in the upper extremity (*clumsy hands*) compared to the lower extremity (spasticity) due to its medial location in the corticospinal tract
- The distal musculature (*lateral corticospinal tract*) is more affected than the proximal musculature
- Common in patients with pre-existing cervical spinal canal stenosis and subsequent hyperextension injury
- Good prognosis especially in patients <50 years of age. Often leaves *clumsy hands*. Lower extremity and bladder function recover first

Anterior Cord Syndrome

- Patients have more loss of motor function in the legs than in the arms. Pain and temperature sensation are lost. Posterior pathways (*proprioception and vibration*) are preserved
- Typically, vascular etiology (*A. spinalis anterior syndrome*) due to flexion/distraction injury
- Worst prognosis of incomplete spinal cord injuries, 10–20% chance of recovery

Brown-Séquard Syndrome

- Injury to one half of the spinal cord cross section (*lateral*)
- Ipsilateral loss of motor function, proprioception, and vibratory sensation and contralateral loss of pain and temperature sensation (*typically 2 levels below the level of the lesion*)
- Usually penetrating injuries
- Best chance for motor recovery

Scoliosis

Definition

Three-dimensional deformity with lateral, coronal, and rotational deformity (> 10° Cobb angle).

Cobb angle: Measurement of the coronal angle between the upper and lower endplate of the most tilted vertebral bodies of a scoliotic curvature on a standing ap X-ray.

Classification

- **Idiopathic Scoliosis**
  - Infantile idiopathic scoliosis (age < 4 years)
  - Juvenile idiopathic scoliosis (age 4–10 years): high risk of progression
  - Adolescent idiopathic scoliosis (age > 10 years): most common (see ▶ Section "Adolescent Idiopathic Scoliosis (AIS)")
- **Congenital Scoliosis** (see ▶ Section "Congenital Scoliosis")
- **Neuromuscular Scoliosis** (in Duchenne muscular dystrophy, spina bifida, cerebral palsy, neurofibromatosis): often long C-shaped curve, associated with pelvic tilt, can progress rapidly

Early-onset scoliosis includes all spinal deformities diagnosed before the age of 10 years independent of the etiology (idiopathic, congenital, syndrome-related, neuromuscular). The main danger is the development of thoracic insufficiency syndrome (TIS): the inability of the thorax to allow normal breathing or lung growth.

Adolescent Idiopathic Scoliosis (AIS)

Etiology

Unknown, most likely multifactorial, hormonal, familial, females ≫ males.

Clinical Presentation

Patients often referred from general screening where a 7° curve on the scoliometer during the Adams forward flexion test is considered abnormal (7° on the scoliometer corresponds to a Cobb angle of 20°).

Diagnosis

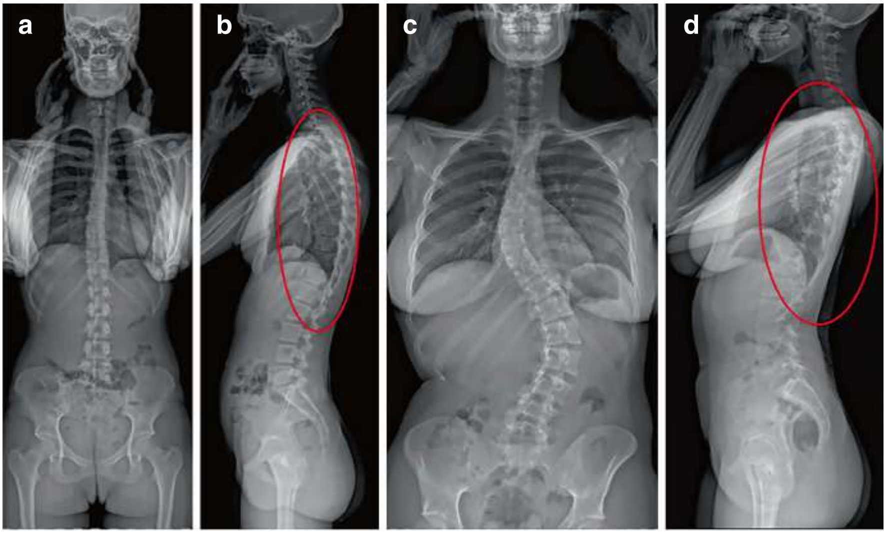
- Clinical examination
  - Levelled pelvis = prerequisite for adequate clinical assessment
  - Shoulder height imbalance
  - Asymmetry of the waist triangles
  - Rib hump or lumbar buldge (truncal shift)
  - Adams forward bending test
- Radiographic examination
  - Standing ap and lateral X-ray:
    Cobb angle measurement (usually right convex thoracic)
    Determination of the Risser stage to assess skeletal maturity (*ossification of the iliac crest apophysis; stage 0–5* [Fig. Risser Stages, p. 30])
    Hypokyphosis on lateral radiographs

Atypical Scoliosis	Left convex thoracic curve, painful scoliosis, early-onset scoliosis, rapid deformity progression, associated syndromes, neurological deficits, congenital anomalies, lack of hypokyphosis → MRI required to rule out intraspinal anomalies
Apical vertebra	Maximum deviation from the C7 plumbline (thoracic) or central sacral vertical line (CSVL) (thoracolumbar / lumbar)
End vertebra	Most tilted vertebra at the cephalad and caudal ends of a curve, used to determine cobb angle
Neutral vertebra	No rotation, i.e., most cephalad vertebra below the apex of the major curve whose pedicles are symmetric on ap X-ray
Stable vertebra	Most cephalad vertebra below the main thoracic curve that is most closely bisected by the CSVL

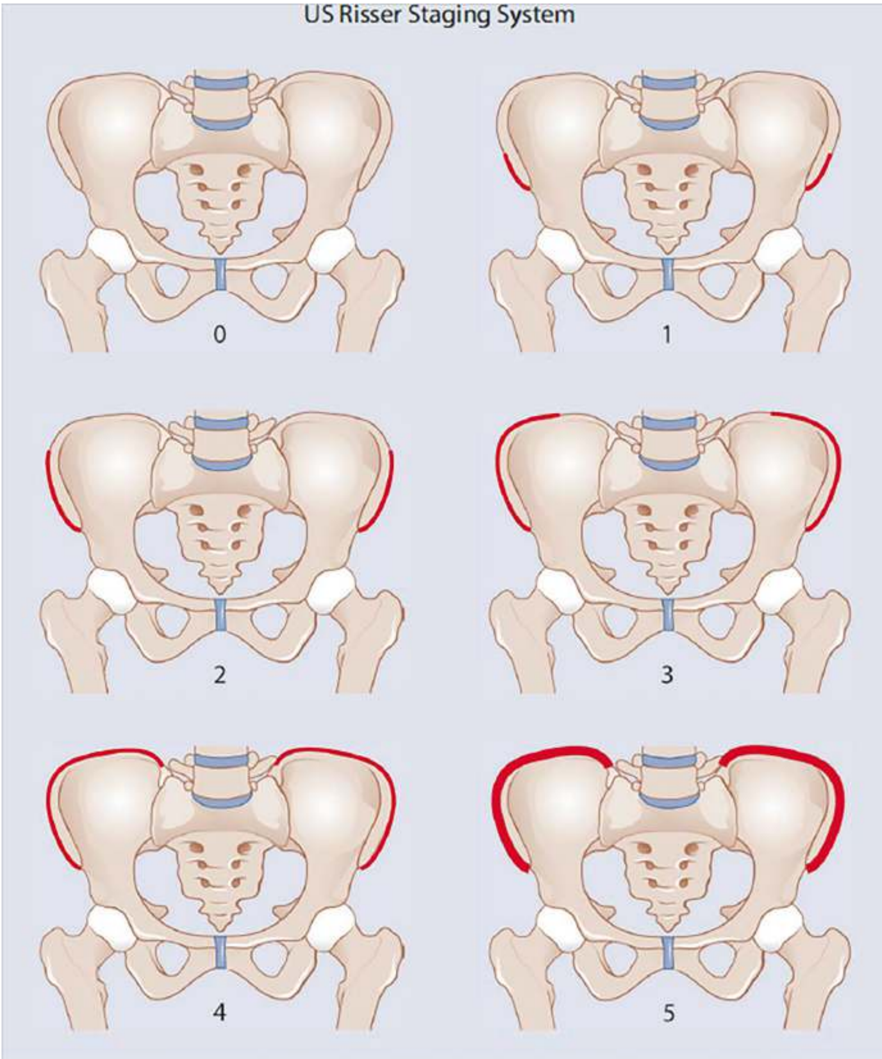
Therapy

➔ See treatment algorithm figure "Treatment Scheme for AIS"

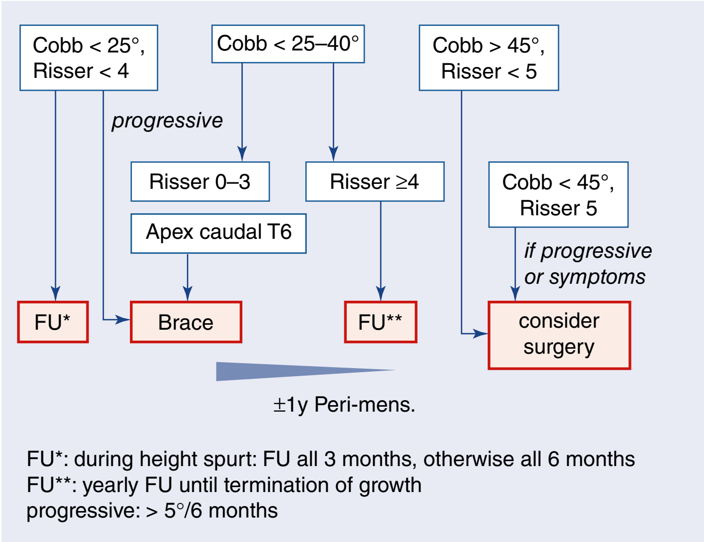
- Braces can only stop or slow the progression of scoliosis (< 5°), not correct it. Cobb angle 25°-40°. Recommended wearing time: 23 h/day until skeletal maturity (i.e. Risser stage 4, < 1 cm growth in 6 months, 2 years after menarche)
- Classification of AIS according to Lenke: Choice of levels to be fused in surgical treatment of AIS
- 3 risk factors for progression of the deformity: Cobb angle (> 25° before skeletal maturity, > 40° after skeletal maturity), residual growth (< 12 years of age, Risser stages 0 and 1, just before menarche), curve type (thoracic > lumbar, double curve > single curve)
- *Crankshaft phenomenon*: progressive rotational and angular deformity that may occur after posterior spinal fusion surgery, especially in very young patients due to continued growth of the anterior column
- Curves > 90° are associated with cardiopulmonary dysfunction and pain



(**a**, **b**) normal alignment of the spine (**c**, **d**) adolescent idiopathic scoliosis with hypokyphosis of the thoracic spine (compare areas within red cicles)



Classification of skeletal maturity according to the ossification of the iliac crest apophysis with the Risser Staging System



Treatment scheme for AIS. *FU* follow up

Congenital Scoliosis

Pathogenesis

Takes place during embryonic development:

- Most cases occur spontaneously
- *Failure of segmentation*: incomplete separation of adjacent vertebra
  - Posterior segmentation failure = fusion of the vertebral arches and joints
  - Anterior segmentation failure = fusion of the vertebral bodies without intervertebral disc space
  - Lateral segmentation failure = unilateral bar
  - Complete segmentation failure = block vertebra
- *Failure of formation*: incomplete vertebral body formation
  - Anterior malformation = wedge vertebra
  - Lateral malformation = hemivertebra
  - Middle malformation = butterfly vertebra

Associated with:

Syringomyelia	Formation of a cavity in the spinal cord usually due to a disturbance in the circulation of cerebrospinal fluid
Diastematomyelia	Parts of the spinal canal/spinal cord are divided longitudinally
Other anomalies	Genitourinary anomalies in 25% → ultrasound of the kidneys. Cardiac anomalies in 10% → echocardiogram

Prognosis

- *Block vertebra* has best prognosis regarding progression
- Unilateral failure of segmentation ("unilateral bar") combined with contralateral failure of formation (fully segmented hemivertebra) has the highest risk for progression

Scheuermann's Kyphosis

Definition

Increased, rigid thoracic (rarely thoracolumbar or lumbar) kyphosis (> 45°) with 3 or more consecutive wedged (>5°) vertebra.

Pathogenesis

Unclear, but suspected are osteonecrosis of the anterior apophyseal ring, relative osteoporosis, disruption of collagen aggregation, and altered biomechanics leading to abnormal endplates, loss of anterior disc height, compression deformity, anterior wedging, and growth arrest

- Often associated with lumbar hyperlordosis, spondylolysis, scoliosis, and restrictive pulmonary disease in curves >100°

Clinical Presentation

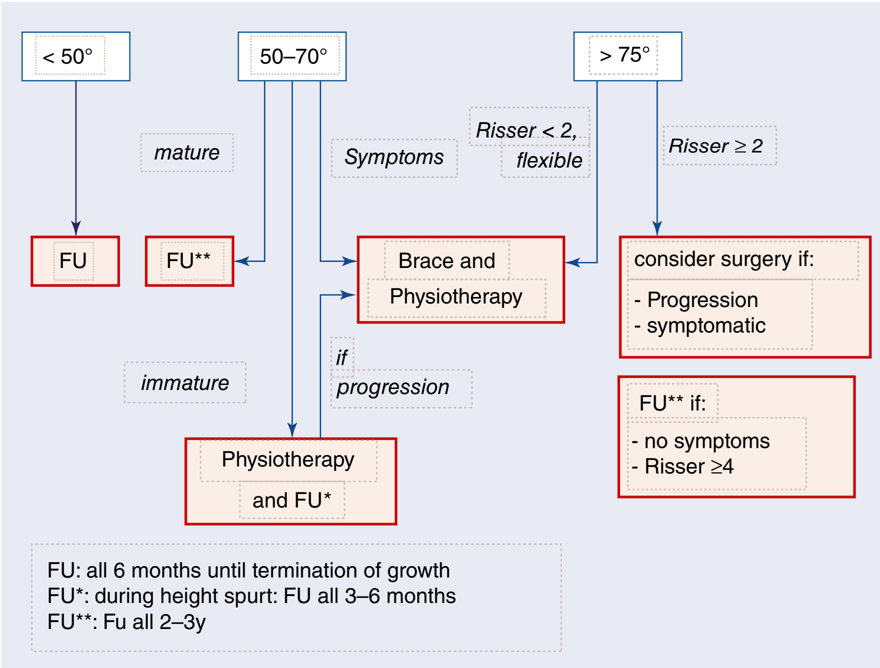
(Partially) fixed hyperkyphosis,, compensatory hyperlordosis, thoracic or lumbar back pain, relative shortening of hamstring muscles.

Diagnosis

- Clinical examination
  - Increased kyphosis (norm 20–45°) with greater angulation and forward flexion
- Radiographic examination
  - 3 consecutive vertebrae with more than 5° of anterior wedging per vertebra, endplate irregularities (= ***Schmorl's nodes, "Randleistenhernien"***), narrowing of the disc space

Therapy

See figure "Scheuermann's disease Therapy Scheme. Thoracic kyphosis is measured in degrees".



Treatment scheme for Scheuermann's Kyphosis. The thoracic kyphosis is measured in degrees. *FU* follow up

Prognosis

Benign natural history. Curves >75° are associated with increased risk for back pain. Indication for surgery (posterior instrumented spinal fusion) if kyphosis >75°–80°.

Tumors

Metastases vs. Primary Tumors

The spine is the most common site for osseous metastases, particularly of lung, breast, and prostate cancer. Metastases ≫ primary tumors (rare). These are rarely resected, but are treated with radiation or chemotherapy.

Radiographic Signs Suggestive of Tumor

- Absence of a pedicle on AP radiograph ("*winking owl sign*")
- Cortical erosion or expansion
- Atraumatic loss of height of a vertebra

Red Flags for Metastasis

- History of a tumor (especially breast, lung, prostate cancer)
- Unexplained/unintentional weight loss
- Pain at night

Most common primary tumors of the spine: Osteoid osteoma and osteoblastoma, aneurysmal bone cyst, hemangioma, eosinophilic granuloma, giant cell tumor, plasmacytoma/multiple myeloma.

Infections

Spondylodiscitis and Epidural Abscess

Pathogenesis

- Hematogenous infections can affect the intervertebral disc (**spondylodiscitis**) or lead to an accumulation of pus in the spinal canal (**epidural abscess**), especially in children, due to better blood supply to the disc space (*Staphylococcus aureus most common germ*)
- Risk factors include drug abuse, immunodeficiency or -suppression, HIV, previous spinal surgery
- The most common extrapulmonary manifestation of *tuberculosis* is the spine

Clinical Presentation

Back pain, neurological deficit (33% with epidural abscess), elevated inflammatory parameters and fever (50%).

Diagnosis

MRI = gold standard for diagnosis of spondylodiscitis (*with contrast medium of the entire spine for epidural abscess to diagnose skip-abscesses*).

Therapy

Conservative for small abscesses without neural compression. Identification of the pathogen (*CT-guided or open biopsy*), then i.v. antibiotics. Failure of conservative therapy associated with diabetes, CRP > 115 mg/L, positive blood cultures, age > 65 years, MRSA.

Operative indicated if neurological deficits, spinal cord compression, persistent infection despite antibiotic therapy, progressive deformity.

Prognosis

Neurological deficits are the main outcome parameter. Mortality = 5%.

Systemic Diseases

Diffuse Idiopathic Skeletal Hyperostosis (*Syn: DISH/ Morbus Forestier*)

Definition

Common disease of unclear etiology with enthesopathy and stiffness of the spine (*thoracic > cervical > lumbar, often mildly painful*) and extremities, particularly in gout, hyperlipidemia, and diabetes. Associated with lumbar spinal stenosis, cervical myelopathy, dysphagia, stridor, hoarseness and difficult intubation. Due to the long lever arm, even minor trauma can result in unstable fractures.

Diagnosis

Characterized by spondylophytes with horizontal growth pattern ("*flowing candle wax*") and subsequent bridging of the intervertebral disc compartments (*remaining intact*) of at least three adjacent levels. Also, calcification of the anulus fibrosus, ALL and PLL. No association with HLA-B27 (*but with HLA-B8*) and no involvement of the sacroiliac joint.

Even in minor trauma, a fracture should be excluded by CT (*CAVE: 67% mortality with conservative management of cervical trauma in DISH*).

Therapy

Mostly conservative with physiotherapy, NSAIDs, and bisphosphonate therapy.

Ankylosing Spondylarthropathy (*Syn: M. Bechterew*)

- 95% of patients are HLA-B27 positive. Typically males with back and groin pain in their third and fourth decade of life. Involvement of the sacroiliac joint
- "*Bamboo spine*" phenomenon on radiographs due to syndesmophytes (*vertical growth pattern*). May result in kyphotic deformities with sagittal imbalance

Definition

Chronic seronegative autoimmune spondyloarthropathy, more common in patients with HLA-B27, with enthesitis and ankylosis (*often painful contractures*). Associated with involvement of the eyes (*uveitis, iritis*), heart (*conduction abnormalities*), lung (*pulmonary fibrosis*), kidneys (*renal amyloidosis*), vessels (*aortitis, stenosis, regurgitation*), and joints (*mainly osteoarthritis of the hip and shoulder*). Due to the long lever arm, even minor trauma can lead to unstable fractures.

Diagnosis

Progressive kyphotic deformity with bridging spondylophytes ("bamboo spine"), square vertebral bodies with "shiny corners", ossification of the intervertebral disc space, bilateral SIJ involvement (sacroiliitis), possibly uveitis, HLA-B27 positive (in 90% of cases), < 2 cm chest expansion.

Even in the case of minor trauma, a fracture should be ruled out with a CT scan (WARNING: 75% neurological damage).

Therapy

Conservative NSAIDs, physiotherapy, TNF-alpha blockers.

Operative Osteotomy to restore sagittal balance and horizontal gaze

- *Smith-Petersen osteotomy*: for mild to moderate deformity, correction of 10° by resection of the facet joints bilaterally and spinal fusion; correction occurs at the level of the intervertebral disc
- *Pedicle subtraction osteotomy*: for moderate to severe deformity, correction of 30–40° by transpedicular resection of posterior structures; correction occurs at the level of the vertebral body
- *Vertebral body resection*: for severe deformity, correction of 45° by removal of an entire vertebral body and replacement with a cage; high surgical morbidity

Klippel-Feil Syndrome

Rare, congenital fusion of at least two cervical vertebrae. Fusion above C3 is more often symptomatic.

Associated with: Scoliosis, congenital cervical stenosis, atlantoaxial instability, brainstem invagination, hearing loss, kidney disease (aplasia), heart disease and Sprengel's deformity (= *small non-descended shoulder blade*). Contact sports should be avoided.

Classic triad:

- Low back hairline
- Short neck
- Limited cervical mobility

Clinical Examinations

"Polio" Strength Grades

5	Normal strength
4	Movement against resistance possible, but with reduced strength
3	Movement against gravity possible
2	Movement possible only when gravity is eliminated
1	Palpable muscle activity/fasciculations without movement effect
0	No muscle activity or fasciculations

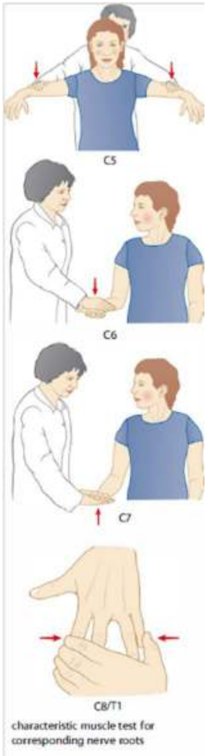
Sensory Testing

All dermatomes are tested for *pain* (with a sharp object) and *light touch* (feather), as well as for vibration, temperature, and 2-point discrimination.

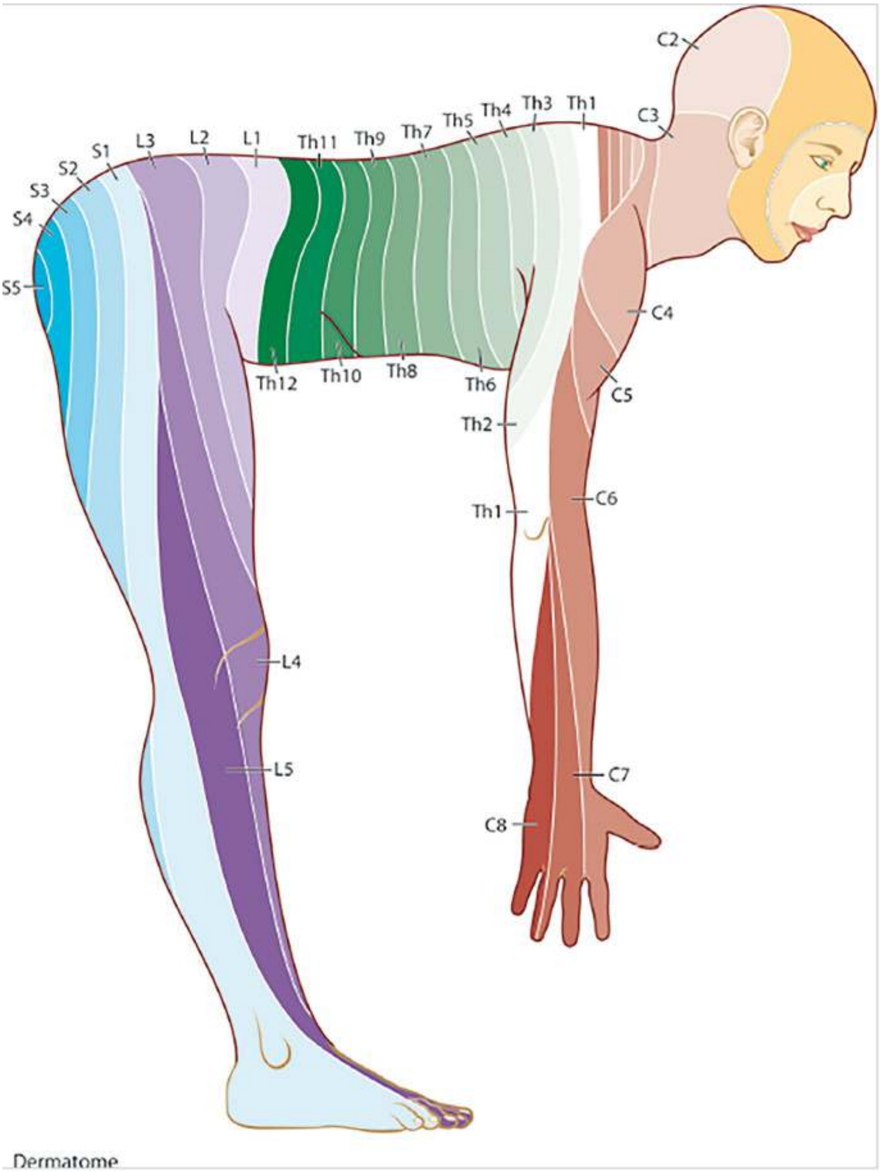
Key Muscles, Sensory Dermatomes and Muscle Stretch Reflexes

Upper Extremity

- M. deltoid C5 > 4
- M. biceps brachii C5 < 6, BSR (*biceps tendon reflex*)
- M. brachioradialis C6/7, BRR (*brachioradialis reflex*)
- M. triceps C7, TSR (*triceps tendon reflex*)
- M. interossei: C8/T1



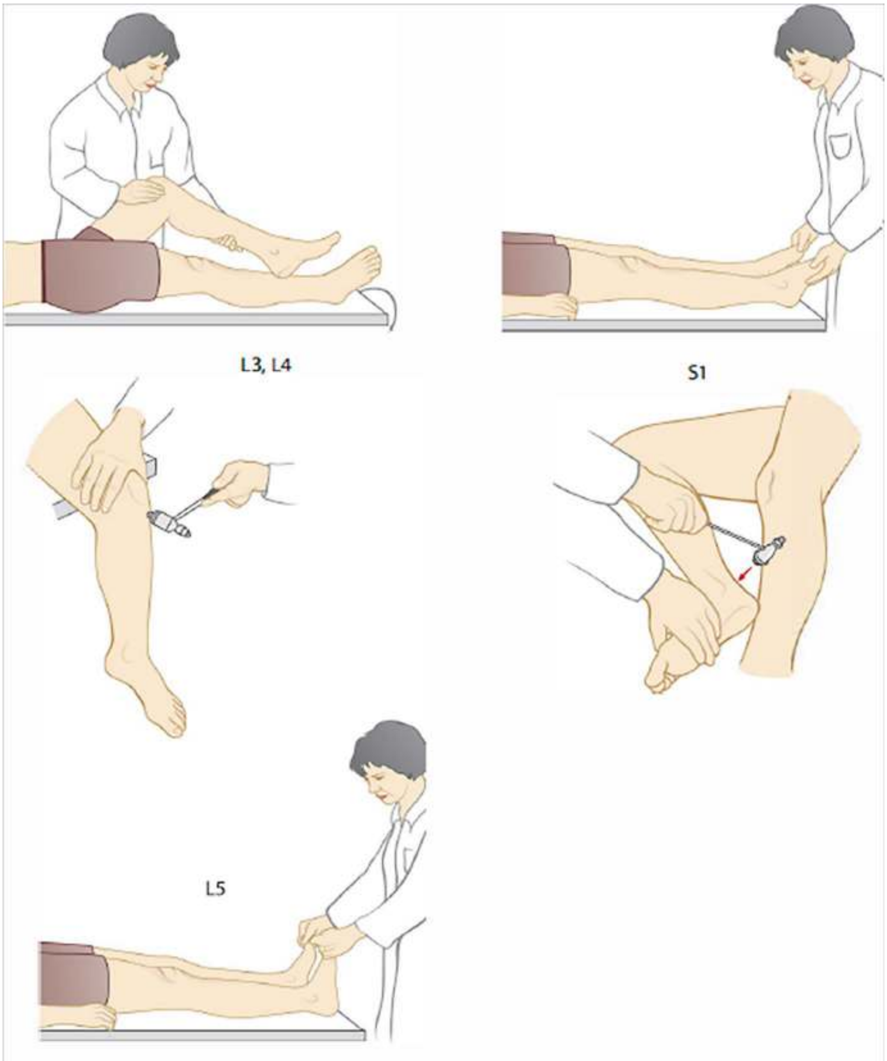
Clinical examination of the upper extremity key muscles



Sensory dermatomes

Lower Extremity

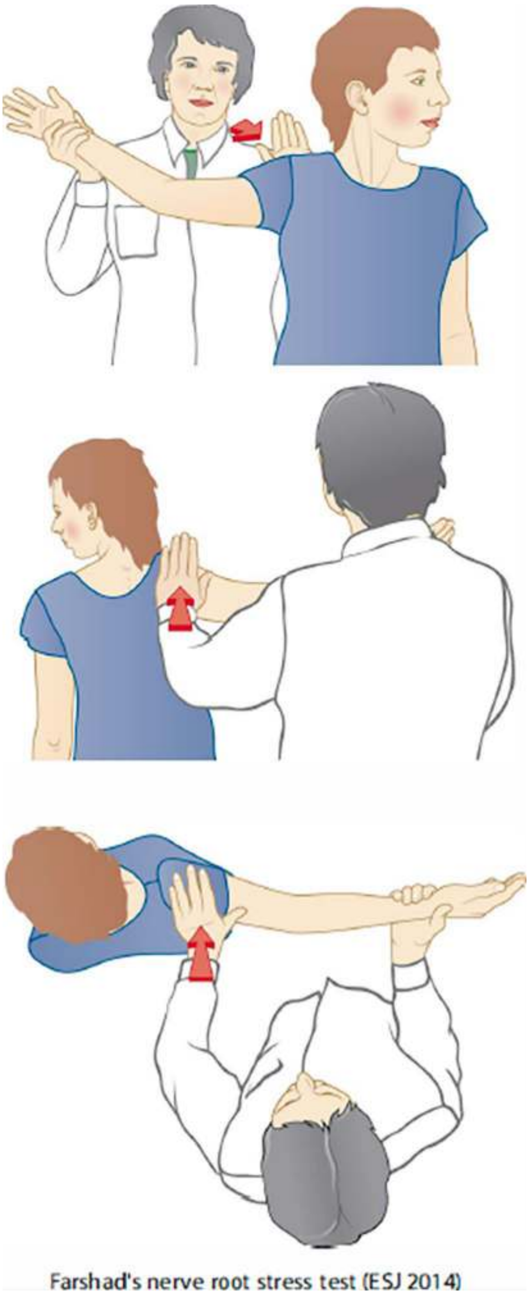
- Hip flexors L1/2
- Hip adductors L2 < 3
- Hip abductors L5
- M. quadriceps L2 < 3/4, PSR (*patellar tendon reflex*)
- Dorsal extension of foot L4 > 5
- Big toe extension L5
- Plantar flexion of foot S1



characteristic muscle tests for corresponding nerve roots. CAVE: L4: dorsal foot extension (Tib. ant.), L5: extension of greater toe (EHL)

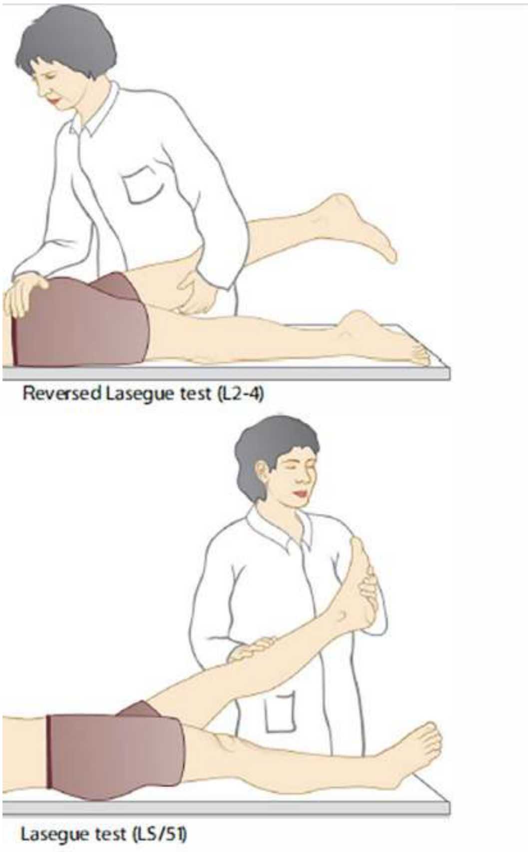
Root Stress Test

Cervical (Farshad)	Abduction and extension of the shoulder with Hypomochlion places stress on the brachial plexus and the cervical nerve roots



Farshad's nerve root stress test

Cervical (Spurling)	Rotation of the head to the affected side with extended neck with axial pressure on the head
Lumbar (Lasègue)	Supine position, lifting of the extended affected leg causes radicular pain (*L5/S1*) (*sensitive*). Note: The essential criterion for a positive test is the elicitation of **radicular pain**! All other sensations are called **pseudolasègue** and are usually due to a significant shortening of the hamstrings
Crossed Lasègue	Lifting of the contralateral leg causes radiculopathy in the affected leg (*specific*)
Reversed Lasègue	Prone position, lifting of the extended leg causes pain in the L2-L4 dermatome



Lasegue test

Reflexes

Babinski	Stroking the lateral sole of the foot with a sharp object
Bulbocavernosus reflex	Pinching the glans penis or the clitoris or pulling on the urinary catheter

Gait

Antalgic gait	due to pain in the affected extremity (hip, knee, radiculopathy)
Trendelenburg limp	Due to gluteal muscle weakness (*morphologic or motor L5 radiculopathy*)
Steppage gait	Compensation for foot and big toe extensor weakness
Ataxic gait	Unsteady swaying gait (DD: Spinal vs cerebral ataxia)

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Shoulder

3

Philipp Kriechling, Andreas Flury, Bettina Hochreiter, Samy Bouaicha, Silvan Beeler, Elias Ammann, Barbara Wirth, Alberto Schneeberger, Gregory Cunningham, and Karl Wieser

Anatomy

Bones	Humerus, scapula, and clavicle
Joints	Glenohumeral joint, acromioclavicular (AC) joint, sternoclavicular (SC) joint, scapulothoracic joint (ST)

Glenohumeral Joint

- Joint surface: → Glenoid: Humerus = 1:3
- Humeral head: Greater and lesser tuberosities as insertions for the rotator cuff (RC) tendons
- Blood supply to humeral head almost entirely through the anterior circumflex artery
- Planes of motion: Flexion-Extension/Adduction-Abduction/Internal and external rotation (IR, ER)

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M. Farshad (ed.), *Orthopedics Must Knows*,
https://doi.org/10.1007/978-3-662-70893-4_3

  - Maximum glenohumeral abduction of 90–120°. Global abduction/elevation = 170°, achieved only with **combined** glenohumeral and scapulothoracic movement (2:1)
- Predominantly ligamentous and musculotendinous stabilization (minimal bony stabilization), provides a wide range of motion (most mobile joint in the body) (CAVE: high risk of instability/dislocation)
  - Stabilizers: active: rotator cuff; passive: joint capsule including glenohumeral ligaments, labrum, and bony glenoid
  - Abduction and flexion require external rotation to prevent greater tuberosity impingement → loss of external rotation prevents full abduction and flexion
  - Glenohumeral ligaments:
    Superior glenohumeral ligament (SGHL): Origins at 12/1 o'clock on glenoid; prevents inferior translation in adduction
    Middle glenohumeral ligament (MGHL): Origins at 1/2 o'clock; prevents anterior translation in adduction-external rotation and 45° abduction
    Inferior glenohumeral ligament (IGHL): complex of structures consisting of an anterior band, a posterior band, and an interposed axillary pouch, origins at 3 and 8 o'clock; prevents anterior translation in 90° abduction and external rotation and posterior translation in 90° flexion and internal rotation
- Labral anatomic variations: Circumferentially intact and fixed to glenoid in 86%; detachment of anterosuperior labrum (= sublabral foramen or hole in 12%, sublabral recess in up to 70%); absent anterosuperior labrum with cord-like MGHL (= Buford complex, in 1.5%)

Acromioclavicular (AC) Joint

- Minimal motion, mainly rotation (about 10°)
- Intra-articular disk
- Stabilization:
  - Acromioclavicular ligaments—horizontal stability
    Posterior ligament is the strongest
  - Coracoclavicular ligaments—vertical stability
    Trapezoid ligament (lateral): Resistance to posterior
    Conoid ligament (medial): Resistance to superior

Sternoclavicular (SC) Joint

- Intra-articular disk
- Movement in the SC joint cranio-caudal approx. 35°, antero-posterior approx. 35°, rotation approx. 45°

Scapulothoracic Articulation

- Not a true joint, contributes about 60° (i.e., about 1/3) to global elevation/abduction
- Pathology: Abnormal scapulothoracic movement (functional problem of the periscapular muscles, insufficiency of the anterior and inferior trapezius muscles), medial scapula winging (Serratus anterior palsy)

The Rotator Cuff

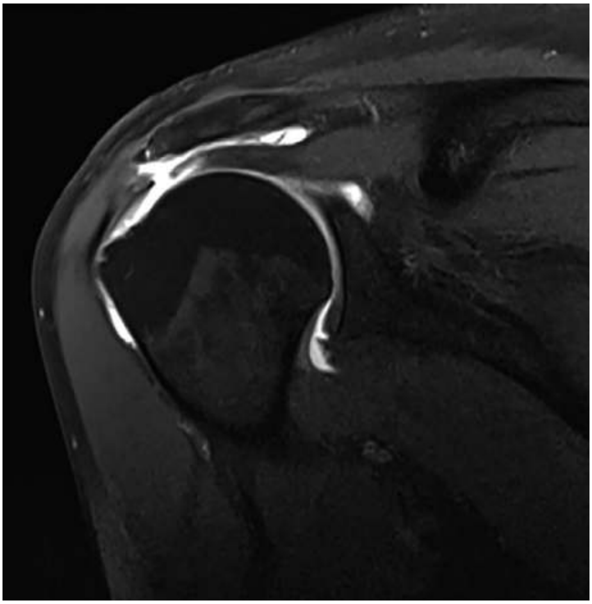
Muscle	Origin	Insertion	Primary function	Innervation
M. supraspinatus **(SSP)**	Supraspinous fossa	Gr. Tub.	Abduction	Suprascapular nerve
M. infraspinatus **(ISP)**	Infraspinous fossa	Gr. Tub.	External rotation	Suprascapular nerve
M. teres minor **(TM)**	Lateral border of the scapula	Gr. Tub	External rotation (in abduction)	Axillary nerve
M. subscapularis **(SSC)**	Subscapular fossa	Les. Tub	Internal rotation	Subscapular nerve

In addition, all muscle-tendon units of the rotator cuff have the secondary function of centering the humeral head in the glenoid cavity

The Long Head Biceps Tendon

- Passes from the supraglenoid tubercle through the glenohumeral joint and between the supraspinatus and subscapularis (rotator interval) into the intertubercular groove
- The biceps pulley (suspension of the long biceps tendon at the entrance to the groove) is composed of the coracohumeral ligament and cranial fibers of the SSC and SGHL

Rotator Cuff Tear (RC Tear)



Full-thickness tear of the supraspinatus tendon

Epidemiology

Increasing with age: < 10% at age 50/> 50% at age 80+

Etiology

- Chronic/degenerative → Older patients, often less symptomatic (wear and tear)
- Acute/traumatic → Younger patients, mechanism of accident: shoulder dislocation, fall on an outstretched arm (superior dislocation), eccentric load
  - > 40 years old: usually postero-superior RC tear (SSP, ISP, TM)
    RC tears after a dislocation occurs in up to 40% of dislocations in elderly patients
  - SSC more common in young patients after fall/dislocation
- Smoking
- Intrinsic factors (within the tendon) (age, decreased vascularity, chronic inflammation, genetics) versus extrinsic factors (overuse, bony impingement, altered shoulder kinematics, trauma)

Clinical Presentation

- Pain at night and on exertion (overhead movements), no specific area (around the deltoid, possibly radiating to the upper arm)
- Weakness: Varies by tear pattern, and acute versus chronic (compensation)

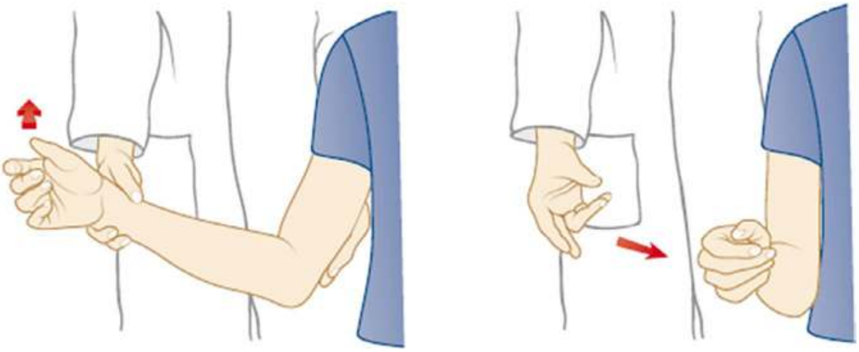
Diagnosis

- Clinical examination

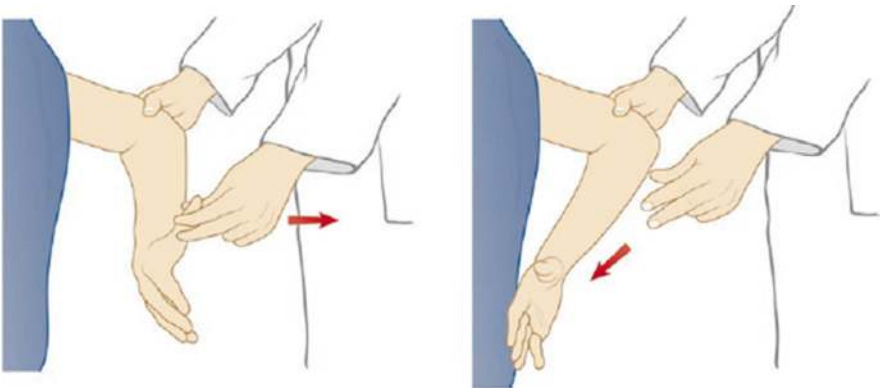
SSP	Flexion/abduction (*Jobe and Whipple tests*)
ISP	External rotation (*ER in neutral position, ER-Lag in abduction → M. teres minor involved*)
TM	Positive hornblower's sign, external rotation in abduction (*Patte's test)*
SSC	Internal rotation/behind-back function (l*ift-off Test, belly-press test*, *bear hug test*, excessive passive external rotation)

  – Lift-off has low sensitivity in painful or stiff shoulder
  – Pseudoparesis = ("pseudo": functional impairment due to rotator cuff lesion and not due to neurological deficit) Active elevation < 90° but > 45°; passive free glenohumeral mobility; the arm passively elevated to 90° can be actively held
  – Pseudoparalysis = Active elevation < 45°; the arm cannot be held in abduction and falls down

A positive Jobe- or Whipple Test is characterized by a (pain-free) weakening of abduction



Evaluation of external rotation strength (top). Passive evaluation of maximum external rotation (bottom left) and possible lag-sign (bottom right)



Gerber's Lift-off test: Loss of active internal rotation of the arm at the back of the body

- Radiographic examination
  - **X-ray**: glenohumeral centering, or superior migration of glenohumeral head (*acromiohumeral distance < 7 mm as a sign of a massive rotator cuff tear*)
    Critical shoulder angle (CSA): > 35° associated with increased risk of rotator cuff tear; < 30° associated with an increased risk of shoulder osteoarthritis
  - **MRI** (intra-articular contrast increases sensitivity for articular-sided partial RC tear detection): **Gold standard**, assessment of tear location and extent, muscle quality (atrophy and fatty infiltration), concomitant injuries
    Fatty infiltration according to Goutallier (originally described on CT): grade 1 = some fat streaks; grade 2 = more muscle than fat; grade 3 = muscle proportion equal to fat proportion; grade 4 = proportion of fat infiltration exceeds muscle proportion
    Muscle atrophy according to Zanetti: Tangent sign → to assess atrophy, which correlates with tendon irreparability
    Quantitative fat fraction
    Retraction: According to Patte: stage 1 = Retraction of the torn tendon stump to the cartilage-bone junction; stage 2 = up to the level of the humeral head; stage 3 = at the glenoid level or beyond
    Partial SSC tears can be difficult to detect on MRI. Medial subluxation of the long head biceps tendon ➔ sign of SSC tear
  - **Ultrasound**:
    Main disadvantage: highly user dependent, poor sensitivity especially for re-tear assessment after surgery

Therapy

Indications Conservative Versus Surgical Treatment

Conservative	Surgical
Older, rather inactive, non-sportive patient	Younger patient
Lower physical demand	Sportive
Muscle atrophy	Good muscle quality
Partial tears	Full-thickness tears Failed conservative treatment

Conservative: Physical therapy (at least 3–6 months), NSAIDs, corticosteroid injections

Operative: Relevant factors: limited tendon retraction (stages 1 or 2 according to Patte), fatty infiltration not more than grade II according to Goutallier, Zanetti sign negative

Repairable Tears

- RC reconstruction (mostly arthroscopic tendon to bone repair)
- Tear characteristics
  - Partial tears with less than 25–50% tendon involvement: debridement
  - Partial tears with >50% tendon involvement: repair

Irreparable Tears

- Tendon/muscle transfers (young patients)
  - Indication: Healthy and active patient (usually younger), no osteoarthritis
  - Techniques:

    Anterior RC tear (SSC): Pectoralis major or anterior latissimus dorsi transfer

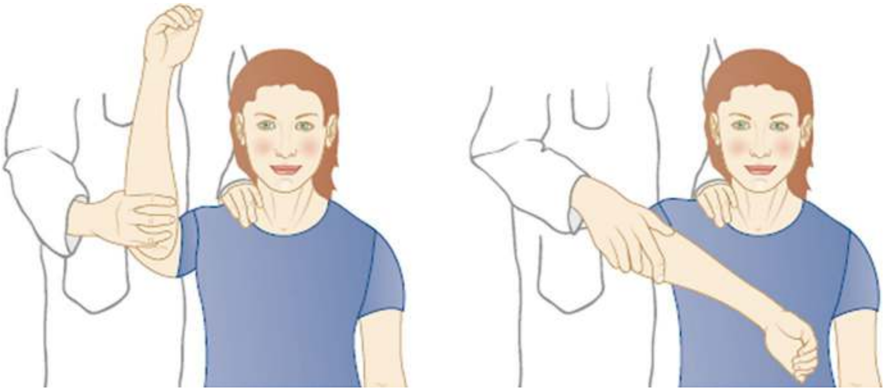
    Posterosuperior RC tear (SSP/ISP(+TM)): Latissimus dorsi or lower trapezius transfer
- Synthetic or biologic patches for RC augmentation (or bridging)
- Superior capsular reconstruction (fascia lata or biceps autograft, allograf, or xenograft)
- Subacromial balloon spacers (older patients without osteoarthritis, not recommended due to low evidence)
- Partial repair to restore force couple (SSC vs. ISP/TM)
- Reverse shoulder arthroplasty (semiconstrained arthroplasty with medialized and distalized center of rotation for better deltoid muscle recruitment, elderly patients or those with osteoarthritis)
- Biceps tenotomy: if the shoulder is functional but painful (anterior pain)

Differential Diagnoses

- Subacromial impingement/subacromial bursitis (modified Hawkins test)
- Symptomatic AC-joint osteoarthritis
- Calcific tendinitis
- Biceps tendinopathy
- Frozen shoulder

Special Feature Os acromiale

- Is an unmerged secondary ossification center of the acromion
- From proximal to distal: meta (base), meso (middle) and pre (tip) acromion
- Mesoacromion associated with rotator cuff tendinitis and tears (up to 50%)
- Therapy for persistent symptoms: Corticosteroid injection, Meso/Meta acromion fusion (resection results in weakness of the anterior deltoid)



Modified Hawkins test: Pain during forced internal rotation of the arm in 90° of flexion and horizontal adduction

Frozen Shoulder

Syn: *Adhesive capsulitis.*

Painful shoulder stiffness due to inflammatory thickening of the joint capsule

Etiology

Idiopathic (*primary*), post-traumatic, post-operative

- Risk factors: Diabetes mellitus, thyroid disorders (of the autoimmune type), Dupuytren's disease, atherosclerotic disease, cervical disc disease (known association, but rarely seen in clinical practice)
  - Regardless of the type of diabetes (types 1 or 2), but dependent on the duration of the disease

Clinical Presentation

Shoulder pain, impaired range of motion

Diagnosis

Clinical Examination

Frozen Shoulder is a clinical diagnosis

Painful active and especially passive movement restriction in all directions

Typically, painful end point pain in all directions with passive mobilization

Always compare to contralateral side or standard values (if both sides affected)

- Passive ER deficit **> 20**° compared to contralateral side
- Reduced glenohumeral abduction
- Inability to reach behind the back (limited internal rotation)

Night pain, almost all tests are painful for the patient (especially in stage 1)

Radiographic Examination

X-ray to exclude osteoarthrosis

MRI only if signs of rotator cuff tears

Pitfall: a normal X-ray including an axillary view is mandatory to rule out a chronic posterior dislocation, especially after a trauma (misdiagnosed as "post-traumatic frozen shoulder")

Prognosis

Time course: Average disease duration 18 months (6–60 months)

Stage 1	Freezing (*severe inflammation, severe pain at rest and movement*)
Stage 2	Frozen (*movement restriction with/without pain*)
Stage 3	Thawing (*decrease of pain and movement restriction*)

Therapy

Conservative: Successful in over 95% of cases. Patient education is very important

NSAIDs, Vitamin C, gentle mobilizing physical therapy, **intra-articular steroid injections**

Operative: In exceptional cases: arthroscopic capsulotomy, only in case of persistent shoulder stiffness (in stage 3) and failed conservative therapy

- Manipulation under anesthesia is controversial and has largely lost its value. The failure rate is high, especially in the inflammatory phase and in diabetics (up to 50%)

Shoulder Osteoarthritis

Etiology

Primary or secondary degeneration of the glenohumeral joint cartilage

Secondary etiologies:

- Cuff tear arthropathy: abnormal glenohumeral wear and superior migration of the humeral head, which is due to an insufficient rotator cuff (loss of the concavity-compression effect of the RC with simultaneous pull of the deltoid to cranial)
- Post-traumatic (proximal humerus or glenoid fractures, post-dislocation with incidence > 50% after 25 years, persistent posterior decentration of the humeral head on the glenoid)
- Rheumatoid Arthritis: Shoulder affected in 90% if untreated
- Avascular necrosis: Alcohol, steroids, sickle cell anemia, pancreatitis, trauma, idiopathic

Clinical Presentation

Motion- and load-dependent pain, limited mobility, crepitations

Diagnosis

Clinical Examination

Limited range of motion

Assess rotator cuff (➔ see section 'Rotator cuff tear') for tears

Radiographic Examination

Plain x-ray (radiological signs of osteoarthritis: typical inferior osteophyte on the humeral head, joint-space narrowing, subchondral sclerosis, subchondral cysts)

- Radiologically, there are two types of primary osteoarthritis: concentric and eccentric. In rheumatoid arthritis of the shoulder, a central erosion of the Glenoid (A2 according to Walch) is usually found
- In posterior subluxation of the humeral head with eccentric loading of the glenoid, eccentric osteoarthritic is usually found (B1-3 according to Walch)

Osteoarthritis of the left shoulder joint

CT & MRI for surgical planning. Assessment of accompanying pathologies (rotator cuff tear, humeral head necrosis) and glenoid bone stock (see indications for reverse shoulder arthroplasty)

Therapy

Conservative: NSAIDs, physical therapy, intra-articular steroid injections

Operative: In case of failure of conservative measures

- Anatomical total shoulder arthroplasty
  (a) Indication only with intact RC and sufficient glenoid bone stock (i.e. no significant posterior wear or posterior humeral head subluxation >80%)

- Arthroplasty for shoulder osteoarthritis with B2 Glenoid morphology:
  - Background: A (limited) correction by corrective reaming of the Glenoid is possible. However, this medializes the joint line, which is associated with loss of preload. In addition, the bone stock is reduced which might affect anchoring of the glenoid component
  - Anatomical shoulder replacement: associated with a high complication rate when the glenoid component is inserted at > 15° retroversion (Rocking horse phenomenon)

    Option 1: posterior augmentation with autograft or metallic wedge

    Option 2: Reverse total shoulder arthroplasty

    - Usually, an autologous bone block from the humeral head is augmented posteriorly or a wedge baseplate is used to prevent pronounced retroversion of the glenoid
- Reverse total shoulder arthroplasty:

  (a) Indications:

  (i) RC deficiency

  (ii) Insufficient/dysplastic glenoid bone stock

  (iii) Excessive glenoid retroversion

  (iv) Excessive static posterior subluxation of the humeral head (>80%)

  (v) Arthroplasty revision

  (vi) To be considered: inflammatory arthritis (ie rheumatoid), because of high risk of secondary rotator cuff failure

(b) Background

(i) Consider arthroplasty design: Lateralization of glenoid (baseplate/autograft) and humerus (onlay/semi-onlay design), lower neck-shaft-angle (135° or 145° instead of 155°) ➔ Lateralization might result in better range of motion, especially rotation, less scapular notching and possibly less acromial fractures (still unclear)

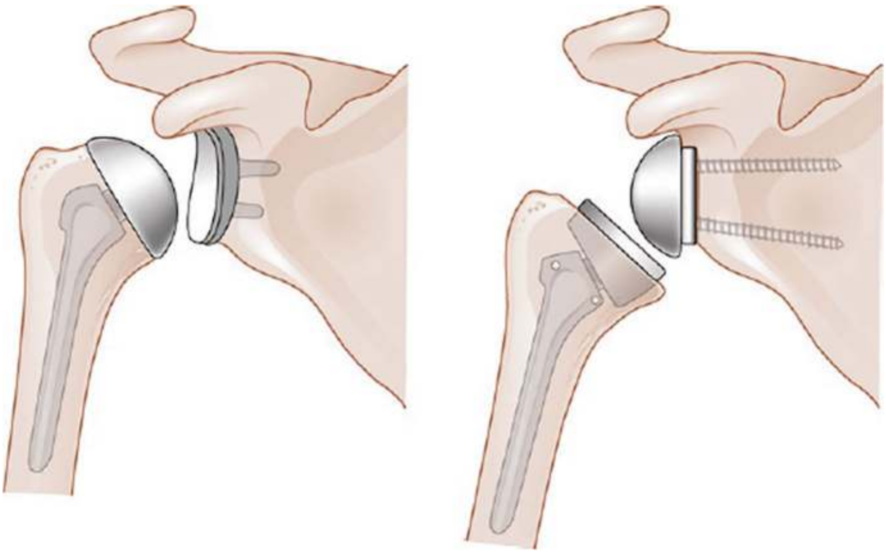
(ii) Acromial stress/scapular spine stress (-fracture): Common complication, 3–10% ➔ pain at acromion with or without fracture on CT (acromial stress reaction)

(iii) Approach: Deltopectoral (gold standard)

Deltoid split/anterosuperior –> positioning of components difficult (superior tilt of baseplate commonly seen)

Subscapularis management: subscapularis ON, peel off, tenotomy or lesser tuberosity osteotomy (the two latter more difficult to repair because of joint distalization/lateralization)

(iv) Intact axillary nerve is mandatory for postoperative function



Visualization of an anatomical total shoulder replacement (left) and a reverse total shoulder (right) replacement

Shoulder Dislocation/Instability

Anterior Shoulder Dislocation

Epidemiology

– M > F, 90% in the second decade of life
– In case of traumatic dislocation, 95% are anteroinferior

Etiology

Fall with abduction and external rotation resulting in anterior dislocation

Structural lesions:

- Bankart lesion: Avulsion of the antero-inferior labrum ± anterior band of the IGHL (in > 80% of cases) ± cartilage defect
  - + bony Glenoid defect corresponds to an osseous Bankart lesion
    Mostly seen in chronic cases
- Hill-Sachs lesion: Posterosuperior impression fracture of the humeral head due to glenoid impact
- Rotator cuff tendon tear (higher risk for older patients: 30% at > 40 years, 80% at > 60 years)
- Greater tuberosity fracture
- Brachial plexus lesion (up to 14% after traumatic dislocation, mostly transient)

Clinical Presentation

– Acute dislocation: severe pain and fixed malposition
– Chronic instability (recurrent dislocations): Feeling of instability, recurrent dislocations

Diagnosis

Clinical Examination (in recurrent chronic cases)

– Range of motion testing, neurovascular examination
– Apprehension test positive: Sensation of imminent shoulder dislocation, motor resistance, or discomfort, when the arm is brought in abduction and external rotation. Must be distinguished from pain

Radiographic Examination

– Plain x-ray → AP, Neer (Y) and axial images: The humeral head must be centered in all images
– Arthro-MRI and/or Arthro-CT scan to identify accompanying soft tissue (MRI) or bony (CT) lesions
  (a) Assess glenoid bone loss through reconstruction of CT scan ("en-face view"), over 13.5% is considerable bone loss that qualifies for a bone block procedure
  (b) *On-Track/Off-Track concept: Engagement of Hill-Sachs lesion with glenoid (➔* ***off-track lesion****) vs. no engagement* ***(➔ on-track lesion****). Off-track lesion through large bone defect at glenoid and large Hill-Sachs-lesion ➔ consider Bankart repair + remplissage (Fixation of Infrapinatus into Hill-Sachs defect) or bone block procedure for off-track lesion to convert off-track into on-track Hill-Sachs defect*

Therapy

Acute dislocation:

– Reduction (*Analgosedation and possibly intubation/relaxation needed*): Various methods described, the most effective: **axial traction + adduction + ER (for anteroinferior dislocation)**
– Mandatory (!) radiological control and neurological examination before and after reduction
– Immobilization in a sling (*Immobilization in external rotation is controversially discussed*)

- **Conservative:** For first-time dislocations almost exclusively. Physical therapy after 1–3 weeks
  (a) Recurrence risk increases with the number of dislocations and the size of the osseous glenoid defect; decreases with age (*first dislocation < 20 years → Approximately 70%, > 35 years → approx. 10% re-dislocations*)
  Based on ISIS (instability severity index score) by Boileau:
  Age < 20 years → 2 points
  Hyperlaxity (AR > 85°, hyperabduction = Gagey's sign, increased shift) → 1 point
  Hill Sachs lesion visible on AP radiograph yes/no → 2 points
  Glenoid bone loss (osseous Bankart) yes/no → 2 points
  Athletic activity: competitive → 2 points, contact sport → 1 point
  (→ < 6 points = 10% recurrence risk with Bankart Repair versus 70% at > 6 points)
- **Operative:** Depending on the individual recurrence risk
  (a) Arthroscopic: Refixation of the labrum with the capsular ligament apparatus (IGHL) = Bankart repair, and possibly additional remplissage (repair of ISP into bony humeral head defect)
    (i) Higher recurrence with soft-tissue repair (Bankart) compared to bone block procedure, careful discussion with patient. New data shows comparable risk for Bankart + Remplissage vs. Latarjet
  (b) Bone block procedures:
    (i) Bone block procedures are considered extra-anatomic but provide valid treatment options for patients with glenoid bone loss, recurrent instability after previous Bankart repair and high-risk patients (i.e. young, male, contact athletes). They are routinely performed in an open fashion, but arthroscopic techniques are described
    (ii) Common techniques:
      - Latarjet: Transfer of the coracoid process to the anterior glenoid to increase the bearing surface. Triple effect: Bone block increases glenoid track, sling effect of conjoint tendon and repair of CA ligament to capsule.
      - Eden-Hybinette or J-Span: Transfer of tricortical iliac crest bone graft

Antero-inferior shoulder dislocation in antero-lateral view (left) and Neer view (right)

Posterior Dislocation/Instability

Epidemiology

In 2–5% of cases (new data suggest that it might be up to 15% of cases)

Etiology

Acute: Almost always traumatic, caused by axial compression with shoulder in flexion/internal rotation (decreased tension in the posteroinferior glenohumeral ligament), epileptic seizure, electric shock

Chronic—recurrent: Associated with ligamentous hyperlaxity, congenital or due to microtrauma in sports with repetitive movements in high-risk positions (= flexion, internal rotation, e.g. in weightlifting or football) and osseous abnormality (e.g. glenoid retroversion, high and flat acromion)

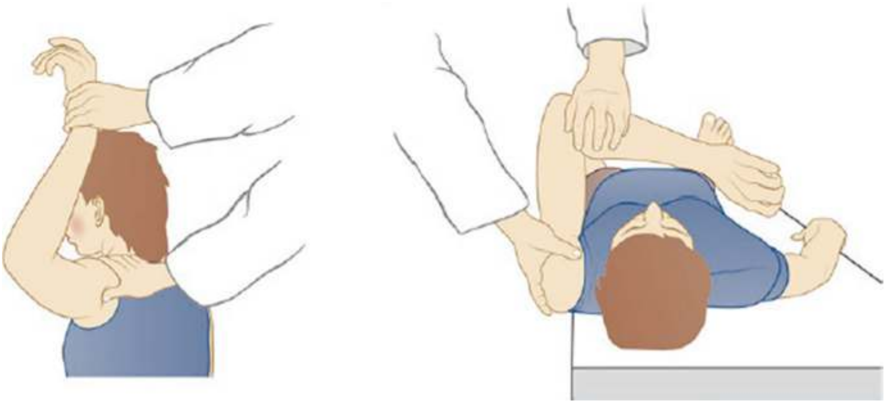
Clinical Presentation

– Acute dislocation: Severe pain and malposition following trauma/seizure
– Chronic instability (recurrent dislocations): Feeling of instability, recurrent dislocations, unclear pain

Diagnosis

Clinical Examination

Jerk test: 90° abduction, elbow bent, internal rotation ➔ axial force along humerus axis ➔ adduct arm; positive = subluxation ("clunk")



Jerk test: Posterior dislocation of the shoulder joint by the examiner

Kim test: 90° abduction, elbow bent, internal rotation ➔ axial force along humerus axis ➔ adduct and forward flex arm; positive = pain.

Radiographic Examination

Acute: Can be missed on plain x-ray AP image (pay attention to *"lightbulb sign"*, the glenohumeral joint line should be perfectly visible). An axillary view is essential, consider a traumatic axillary view in the acute setting if patient stiff or immobilized

Chronic: MRI → posterior labrum tear, reversed Hill Sachs lesion (McLaughlin)

Therapy

Acute dislocation:

– Reduction (analgesia and possibly intubation/relaxation necessary): **Axial pull and internal rotation**
– Mandatory (!) x-rays and neurological examination before and after reduction

– Immobilization in 10–20° external rotation (at least neutral position) for 6 weeks, then physical therapy (avoid combined adduction and internal rotation)

Conservative: Primary therapy (regardless of whether chronic or acute), satisfactory results, specialized physical therapy

Operative: To be considered after primary dislocation if (1) patient shows a persisting static posterior decentered humeral head (on MRI) and/or in case of significant structural lesion (posterior labrum tear) and clear positional instability (subluxation in clinical exam (Jerk test))

– Arthroscopic posterior Bankart repair (without bone defect)
– Open or arthroscopic posterior bone block (with bone defect)
– Rarely performed:
  (a) Posterior open-wedge osteotomy of the glenoid in case of excessive (congenital) retroversion; rarely indicated
  (b) Reversed Hill Sachs lesion < 40% → McLaughlin (SSC remplissage into the defect) or modified McLaughlin transfer (lesser tuberosity transfer into the defect)
  (c) Chronic dislocation > 6 months, arthritis, collapse of the Humeral head after reduction or size of the Hill Sachs lesion > 40% → Hemi arthroplasty (total arthroplasty in case of accompanying glenoid degeneration)

Posterior shoulder dislocation in a Neer view (left) and an anteroposterior view (right)

Multidirectional Instability

Etiology

No clear trigger, but usually atraumatic, generalized hyperlaxity (i.e., Ehlers-Danlos or Marfan) (But: Laxity does not necessarily mean instability)

Clinical Presentation

Typically pain, signs of instability or weakness, inability to elevate shoulder, apprehension

Diagnosis

Clinical Examination

– At least 2 directions of instability: Anterior (Apprehension/Relocation Test), posterior (Jerk test/Kim test) and/or inferior instability
– Hyperlaxity: Sulcus sign, increased external rotation, glenohumeral hyperabduction (Gagey Test), generalized hyperlaxity (Beighton score)
– Voluntary dislocations possible

Radiographic Examination

– X-ray: might be normal, exclude other pathologies
– MRI: patulous inferior capsule, most frequently no structural lesions (secondary lesions of the labrum and rotator cuff thinning may occur)

Therapy

Conservative: Physical therapy (primary and almost exclusively).

Operative: Associated with inferior results in cases of voluntary dislocation; capsular shift/plication possible treatment to reduce intraarticular volume, or IGHL graft reconstruction

– Voluntary dislocators are a contraindication for surgical treatment

Acromioclavicular (AC) Joint Injury

Epidemiology

M > F; young athletic patients

Etiology

Direct or indirect trauma

Clinical Presentation

Pain, hematoma

Diagnosis

Clinical Examination

- Piano key sign: Caudally positioned scapula can be elevated and the clavicle can be reduced (Rockwood types II—V, Type IV might not be reducible due to disruption and impingement of trapeziodeltoidal fascia) for assessment of vertical and horizontal instability
- Assess scapula secondary malrotation

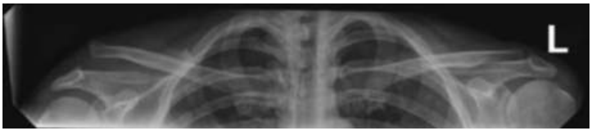
Radiographic Examination

- Rx:
  (a) Panoramic view AP **without weight**: comparison of the coraco-clavicular distance
  (b) Alexander view (bilateral): assessment of horizontal instability
- The CC distance is controversial as it is often underestimated and does not include the posterior dislocation component—therefore, assess horizontal stability clinically and with Alexander views (Table 3.1).

Table 3.1 Classification according to Rockwood (*Tossy classification is outdated and incomplete*)

Classification	Findings/classification criteria	Conservative	Operative
Type I	Strain of AC ligaments, no step formation	✓	
Type II	Tear of AC ligaments, CC intact/strained, step formation < shaft width	✓	
Type III	Tear of AC and CC ligaments, clavicle reducible, Step formation at least shaft width	✓	
Type IIIA	Vertical instability, no horizontal instability	✓	
Type IIIB	Vertical instability, horizontal instability	✓	✓[a]
Type IV	Tear of AC and CC ligaments + horizontal instability (trapezius fascia penetrated)		✓
Type V	= type III, but with penetration of the clavipectoral fascia (not reducible)		✓
Type VI	Dislocation of the clavicle under the coracoid (very rare)		✓

[a]Type IIIb operative—for active patients



Dislocation of the right AC joint

Therapy

Conservative: Immobilization with a sling for 1–3 weeks, Gladstone protocol

Operative: Arthroscopic or open CC-Stabilization possibly additional AC-stabilization with various suture anchor/button systems possible (*Hook plate obsolete!*)

Indication for surgery controversial, as most patients (90%) might have good clinical outcome after conservative treatment even with high grade (up to Rockwood V) injuries. Activity level of patient and amount of overhead activity important factors for surgical indication

CC stabilization and CC and AC ligamentous reconstruction in case of "chronic" (> 3 weeks after trauma) injuries with comparable outcome to primary repair

The use of a graft in is debatable in the acute setting but necessary in the chronic setting

Differential Diagnoses

Lateral clavicle fracture, coracoid fracture

Proximal Humerus Fracture

Epidemiology/Etiology

- Two peaks:
  - Common fracture in the elderly, F > M
  - Young high energy trauma patients

Clinical Presentation

Pain, possibly visible malposition, hematoma

Diagnosis

Clinical Examination

- Swelling, ecchymosis
- Crepitation
- Neurovascular exam, especially axillary nerve

Radiographic Examination

- X-ray: AP, Neer (Y), axial (if possible)
- CT: For a more precise assessment of the displacement, articular involvement (head split), posterior angulation, surgical planning; usually standard of care

Classification according to Neer:

- Number of fracture fragments, which are > 1 cm or > 45° displaced (*1–4 part fractures*). Fragments: shaft, head (*with joint surface*), greater and lesser tuberosity
- Special case: Fracture dislocation (*shoulder dislocation with fracture*)

Predictors of Humeral Head Ischemia (≠ necrosis!) following *Hertel criteria*

(a) *Fracture of the anatomical neck with residual length of the calcar hanging on the joint fragment of < 8 mm (metaphyseal head extension)*
(b) *Dislocated medial hinge (only relevant in the case of a residual calcar of < 8 mm)*
(c) *Angulation of the head fragment > 45° and 4-part fractures*
(d) *Head-split component*
   - In the *St. Gallen* algorithm (Spross et al. JBJS, 2021), (1) the fracture morphology, (2) the age, and (3) the bone quality are crucial for the therapy. While ORIF is generally recommended in young patients, conservative therapy is primarily used in older patients with limited bone quality (especially 1-part as well as valgus impacted 2- and 3-part fractures). 4-part fractures, fracture dislocations, and head-split fractures are usually treated with a reverse shoulder arthroplasty, unless the patient has a well-controlled pain and has low expectations

Therapy

Conservative: Very often possible with good results for mildly displaced fractures (especially in elderly patients)

Initial immobilization followed by the start of pendulum exercises and passive mobilization

Operative: Displaced fractures (in young patients: 2-, 3-, and 4-part fractures as well as 1-part with greater tuberosity dislocation > 5 mm), involvement of the joint surface (*"head-split"*) as well as dislocation fractures

- Open reduction and internal fixation (plate versus nail) (young patients, good bone)
- Reverse total shoulder arthroplasty (elderly patients)
- Hemiarthroplasty (young patients with severe articular involvement and risk for avascular necrosis, rare)
- Risk of AVN decreased by reducing time to operation and proper fracture reduction
- Carefully balance the risk of surgery versus conservative treatment also with regards to humeral head perfusion because the surgical procedure might damage blood supply in addition to the trauma. Critical blood supply of the humeral head through anterior circumflex artery

Complications

- Axillary nerve lesions (due to trauma or iatrogenic)
- Intra-articular screws (especially in case of humeral head collapse or necrosis)
  (a) **Cave:** regular x-ray follow-up necessary
- Secondary humeral head necrosis
- Non-healing of the tuberosities due to poor bone or improper fixation (Poor function after hemiarthroplasty due to non-healing of the tuberosities ➔ more predictable outcome after reverse shoulder arthroplasty even without tuberosity healing)

Fracture dislocation of a right proximal humerus

Humeral Shaft Fracture

Epidemiology

Trauma, low energy in elderly and high energy in young patients

Clinical Presentation

Pain, malposition, hematoma

Diagnosis

Clinical Examination

Neurovascular status due to possible radial nerve injuries (runs posteriorly in the spiral groove in the middle 2/3 of the shaft, diagonally around the humerus shaft)

In closed fractures, neuropraxia can be found in up to 15% of cases, but it is usually of a transient nature

In open fractures, it is often a case of neurotmesis. This requires surgical exploration

A secondary nerve lesion after manipulation (reduction) also requires surgical exploration

Radiographic Examination

Plain x-ray: AP and lateral with adjacent joints (*shoulder and elbow*)

Holstein-Lewis fractures (spiral fractures of the distal third of the humerus) are associated with a higher incidence of radial neuropraxia (up to 22%)

Therapy

Conservative: often possible, deformities often well tolerated

– Sarmiento Brace: Splinting of the fracture under compression through activation of the soft tissue envelope. Shoulder and elbow freely movable. Fracture reduction through gravity. Should be readapted frequently as the swelling of the arm decreases to maintain proper reduction
– **Not suitable** for obese arms or bedridden patients (*polytrauma, tetraplegia*, …)
– A primary radial nerve palsy is not a contraindication for conservative treatment (except: open fracture and palsy after manipulation)

Operative: Severe displacement, open fractures, nerve or vascular injuries

– Open reduction and internal plate fixation, or closed reduction and intramedullary nailing
– Surgical stabilization allows for early functional aftercare. The risk of non-union (15–20%) with conservative therapy should be weighed against the surgical risk including damage to nerves (e.g., radial nerve) and vessels or the risk of infection. However, current studies do not recommend surgical treatment over conservative treatment

Left humeral shaft fracture

Clavicle Fracture

Epidemiology

Young athletic patients

Clinical Presentation

Pain after fall, dislocation, crepitation, hematoma

Diagnosis

Clinical Examination

– Careful evaluation of skin and neurovascular compromise

Radiographic Examination

- X-ray: clavicle a-p, tangential
- Classification
  (a) Fracture location by third: **middle third (approx. 80%)** > lateral third > medial third
  (b) Simple or multifragmentary

Therapy

Conservative: Offset < shaft width (< 100%), shortening < 1 cm, immobilization in a sling and passive mobilization for 4–6 weeks, usually in children or frail/old patients

- Risk factors for non-union in midshaft fractures: Comminuted fracture, > 100% displacement, verticalized intermediary fragments (high energy)
  - The non-union rate is up to 15% after conservative and 2% after surgical therapy; in addition, there are malunions (up to 15%) after conservative therapy, but this does not necessarily mean that the patient is clinically disturbed
  - Shortening of the clavicle of > than 1.5 cm is poorly tolerated
- The risk of pseudoarthrosis in fractures in the lateral third is significantly higher. Fracture types II and IV (classified according to Neer) have a non-union rate of up to 50%

  Operative: displaced/shortened fractures and lateral fractures

- Middle third: Open reduction and internal plate fixation
- Lateral third: Open reduction and plate fixation
- Only CC-stabilization (*comparable to AC dislocation*) possible, occasionally in combination with lateral plating
- Stabilization of the clavicle in the event of a simultaneous fracture of ipsilateral ribs (rib 1–4) stabilizes the upper chest wall and reduces pain-related breathing difficulties
- Floating shoulder: A double disruption of the superior shoulder suspensory complex (typically combined fracture of the clavicle and the scapular neck) leads to an interruption of the upper shoulder suspensory complex. This is an unstable situation with an increased risk of non- or mal-union. The surgical management of the clavicle aligns the scapula, which is why the subsequent addressing of the scapula depends solely on the fracture pattern (e.g. joint involvement)

Left midshaft clavicle fracture

Elbow

4

Bettina Hochreiter, Philipp Kriechling, Andreas Flury, Samy Bouaicha, Silvan Beeler, Elias Ammann, Barbara Wirth, Alberto Schneeberger, Gregory Cunningham, and Karl Wieser

Anatomy

Joints	Ulno-humeral, radio-humeral and radio-ulnar
Movements	Flexion-extension (normal: 140–0-5°; but 130–30-0° is sufficient for daily activities), pronation-supination (*normal: 80–0-90°*)
Ulnar nerve	In the sulcus ulnaris posterior to medial epicondyle (*"=funny bone"*)
Ligaments	**Lateral**: The lateral collateral ligament complex consists of the lateral ulnar collateral ligament (= *LUCL*) and the lateral collateral ligament (=LCL or RCL radial collateral ligament). In addition, there is the annular ligament (*surrounds and stabilizes the radial head*) The lateral collateral ligament complex is a major varus stabilizer of the elbow. The LUCL extends from the lateral epicondyle to the supinator crest and is the primary posterolateral rotatory stabilizer. The LCL extends from the lateral epicondyle to the annular ligament. **Medial**: The medial collateral ligament has 2 parts. The anterior and the posterior bundle. The anterior bundle extends from the medial epicondyle to the *sublime tubercle* and is the primary valgus stabilizer of the elbow. The posterior bundle extends from the epicondyle to the olecranon.

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M. Farshad (ed.), *Orthopedics Must Knows*,
https://doi.org/10.1007/978-3-662-70893-4_4

Stability:	The ulnohumeral joint is a very congruent and constrained joint. Together with the collateral ligaments it provides the primary stability of the elbow. The coronoid is the major posterior, posteromedial and posterolateral stabilizer. The radial head is only a secondary stabilizer that gets very important in cases of defects of the coronoid or the collateral ligaments (especially the medial collateral ligament).

Epicondylopathia Humeri Radialis/Ulnaris ("Tennis and Golfer's Elbow")

Etiology

Insertion tendinopathy of the muscle/tendon origins (wrist extensors = "tennis elbow"/wrist flexors = "golfer's elbow") at the epicondyles, usually due to overuse

- Correctly, it is an Epicondylo-*pathy* rather than a Epicondyl-*itis*. Microscopically there are no inflammatory cells in the pathological tissue, but an angiofibroblastic dysplasia with disorganized collagen. In lateral epicondylopathy, microtears are found mainly in the M. extensor carpi radialis brevis and in medial epicondylopathy in the common flexor origin

Clinical Presentation

Pain during and after stress (wrist extension/-flexion) at the muscle origins (lateral/medial epicondyle)

Diagnosis

- Clinical examination
  - Tenderness on palpation (at the tendon origins at the epicondyles)
  - Stress testing
    Lateral epicondylopathy: pain at lateral epicondyle during resisted wrist extension with elbow in extension
    Medial epicondylopathy: pain at medial epicondyle during resisted wrist flexion and/or during resisted forearm pronation
- Radiographic examination
  - MRI (optional, not necessary for diagnosis): Tendinosis or (partial) tears of involved tendon origins at the epicondyles

Differential Diagnosis

- Nerve compression syndromes (PIN lateral = supinator syndrome, ulnar nerve medial = sulcus ulnaris syndrome)
- Intra-articular pathology (e.g., plica syndrome, early osteoarthritis, osteochondral lesions)
- Elbow instability
- Cervical radiculopathy

Therapy

Conservative: Mainstay of treatment, effective in 90%

NSAIDs, ergonomic workplace adjustments. PRP injections and shock wave therapy are possible (more evidence for lateral than for medial epicondylopathy). No evidence of beneficial effect for physical therapy, rest, splints/bandages

Cortisone injections **NOT** indicated (*iatrogenic tendon or ligament damage*)

Operative: Very rare, only in case of failure of conservative therapy after 6–12 months

- Tenotomy of involved tendons at epicondyles (=Hohmann procedure for lateral epicondylopathy)
- Debridement of the tendinopathic tissue and possibly tendon repair (*open or arthroscopically*)

Elbow Instability

Etiology

Acute Dislocation

Simple Dislocation	Defined as elbow dislocation without associated fracture. Testing of stability under anesthesia after reduction. Plain radiographs before and after reduction. Treatment: Mostly conservative with immobilization in a cast for 1 week, followed by a hinged brace up to 6 weeks for high-risk patients.
Complex dislocations	With associated fracture/s

Medial Instability

Medial collateral ligament rupture caused by trauma or chronic overuse (v*algus stress, e.g. throwers such as pitchers* [b*aseball*])

- After a dislocation, the MCL usually heals because the elbow is not exposed to valgus stress in daily use (except for pitchers)

Varus Instability

Insufficiency of lateral collateral ligament complex

Posterolateral Rotatory Instability (PLRI)

Lateral ulnar collateral ligament (*LUCL*) insufficiency after simple or complex elbow dislocation (e.g. fractures of coronoid and radial head)

- Instability with posterolateral subluxation or dislocation of the radiocapitellar and ulnohumeral joints in supination and valgus

Clinical Presentation

Feeling of loose and unstable elbow, recurrent subluxations, snapping

Medial pain for MCL lesions and lateral pain for LUCL lesions (mainly in supination)

Diagnosis

- Clinical examination
  - Instability assessment:

Medial Instability	Valgus stress testing in slight flexion (10–20°), painful milking maneuver and moving valgus stress test for detecting partial tears of the MCL
Posterolateral rotatory instability	Lateral pain, snapping phenomena during extension movements, loading tests (e.g. push-ups in supination and chair-rise test; drawer test and pivot shift test

- Radiographic examination
  - X-ray: Associated injuries, drop sign, ulnohumeral subluxation, posterior radial head subluxation
  - CT for assessment of associated fractures such as coronoid, radial head, distal humerus
  - MRI: Assessment of ligaments

Therapy

Conservative: After simple dislocation cast immobilization for 1 week in 90° of flexion. Thereafter X-ray control to exclude redislocation

Physical therapy mobilizes the elbow actively, not passively, as the muscles act as secondary stabilizers of the elbow. Overhead mobilization further uses gravity to prevent subluxation

Operative: After certain complex dislocations with fracture fixation and ligament repair and for persistent instability after conservative treatment

Prognosis

Recurrent dislocations are generally rare after simple dislocations (< 1–2%)

Radial Head/Neck Fracture

Etiology

Fall on wrist with extended elbow

Diagnosis

- Clinical examination
  - Swelling, hematoma, tenderness
  - range of motion (testing of stability, mechanical block for forearm rotation)
- Radiographic examination
  - X-ray: AP + lateral + radial head target image
  - CT: useful for quantifying the displacement and treatment planning

Mason Classification

Type I	Nondisplaced or mildly displaced (<2 mm)
Type II	Displaced ≥2 mm
Type III	Comminuted and displaced
Type IV	Radial head fracture with associated elbow dislocation

Therapy

Conservative:

- Mason type I and most type II (especially without mechanical block) → functional aftercare (i.e., full range of motion with no weight-bearing on the arm for 6 weeks). If immobilization in a cast is required for pain, for a maximum of 1 week
- Often associated with painful joint effusion. Can be aspirated through the lateral soft spot and injected with local anesthesia to assess if true mechanical blockage

Operative:

- Osteosynthesis with screws alone or with plate fixation, or radial head prosthesis for comminuted non-reconstructible fractures
  - 30% of the elbow's load is transmitted through the radius, making radial head resection an option only for patients with low functional demands. In case of concomitant elbow instability (Terrible triad, Essex-Lopresti, MCL deficiency, etc.), radial head resection is contraindicated

Mason type II fracture

Olecranon Fracture

Olecranon Fractures are common fractures of the elbow involving the extensor mechanism

Simple, transverse or special fracture combinations such as:

Monteggia **Fracture:**

- Definition: Fracture of the proximal third of the ulna with radial head dislocation, classified according to Bado

Transolecranon **Fracture:**

- Easily confused with a Monteggia fracture because the radiocapitellar dislocation is easily recognizable. However, the ulnohumeral joint is also dislocated → Radius and ulna are dislocated anteriorly together

Etiology

Direct blow after fall on elbow, or indirect blow after fall on outstretched upper extremity

Clinical Presentation

Posterior elbow pain, dislocation and hematoma

Diagnosis

- Radiographic examination
  - Anteroposterior and lateral radiographs
  - CT, to assess the fracture and for surgical planning

Therapy

Conservative: rather rare: fractures in elderly and frail patients (good functional outcome, avoids high risk of postoperative complication in this population)

Operative: For displaced fractures or in case of associated instability (transolecranon or Monteggia fractures)

- Anatomic reconstruction of the ulna in proximal fractures is important. The goal is to restore the proximal ulnar dorsal angulation (PUDA), the varus angulation (VA), the olecranon-diaphysis angle (ODA), and the trochlear notch
- Tension band wire osteosynthesis usually for transvers fractures with stable ulnohumeral joints (conversion of triceps traction into fracture compression)
- Plate osteosynthesis for oblique fractures, multifragmentary fractures, or fractures associated with ulnohumeral instability

Simple transverse olecranon fracture, treated conservatively

Terrible Triad

Combination of	Elbow dislocation (ligament lesion), radial head fracture, coronoid fracturerisk of persistent instability and early arthrosis

Etiology

- fall on outstretched arm
- If an isolated coronoid fracture is present, then it is not a terrible triad injury, but a posteromedial dislocation resulting from a varus deforming force. The coronoid fracture is typically anteromedial and the coronoid loses its function as a support pillar. This is always associated with a torn LCL

Epidemiology

Elbow dislocation is the second most common dislocation after shoulder dislocation. Terrible triad injuries are not that frequent

Clinical Presentation

Pain and swelling after fall on the outstretched arm

Diagnosis

- Clinical examination
  - Neurovascular examination
  - Shoulder and wrist evaluation
- Radiographic examination
  - X-ray: AP + lateral before and after reduction, carefully assess for congruent joint
  - CT to assess fracture pattern and associated fractures

Therapy

Conservative (rarely indicated): Immobilization after reduction for 7–14 days, then increase range of motion. Indicated when:

- Ulnohumeral joint concentrically reduced
- Small coronoid tip fractures with non-displaced radial head fracture

Operative: Osteosynthesis or radial head replacement, open reduction and screw or plate fixation of coronoid if fracture involves more than 50% of the height of the coronoid, and ligament repair or reconstruction (LCL/LUCL, if still unstable, also MCL)

Complications

Post-traumatic instability, stiffness, heterotopic ossification, secondary osteoarthritis

Elbow Osteoarthritis

Etiology

1. Primary (prevalence: 2%)
2. Secondary: post-traumatic > rheumatoid arthritis (affecting the elbow in 20–50% if untreated) > osteochondrosis dissecans, synovial osteochondromatosis, hemophilia

Clinical Presentation

Progressive, end-range pain for primary osteoarthrosis due to osteophytes and well-preserved joint space, mid-range pain typically for secondary osteoarthrosis with joint space narrowing due to cartilage damage, stiffness; blocking due to loose bodies

Diagnosis

- Clinical examination
  - Elbow ROM
  - Tenderness
  - Catching, cracking
- Radiographic examination
  - Osteophytes on the coronoid process, radial head and radial fossa/olecranon fossa, olecranon tip
  - Loose osteochondral bodies within the joint capsule (better seen on CT scan)

Therapy

Conservative: NSAIDs, immobilization, cortisone injection, lifestyle modification

Operative:

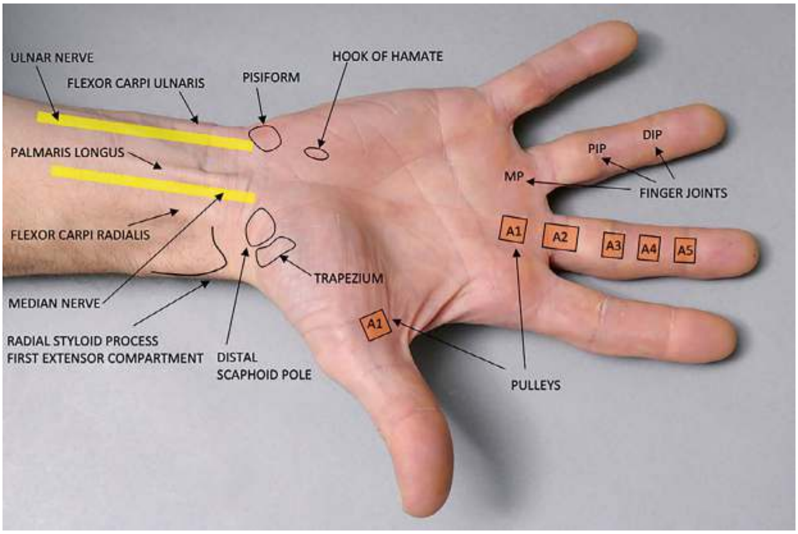
- Arthroscopic or open (column procedure) debridement with capsular release and osteophyte resection in young (or active) patients, usually for primary osteoarthrosis
- Total elbow arthroplasty:
  - In rheumatoid arthritis, the 10-year survival rate is reported to be 92%, and 65–72% for traumatic conditions
  - Non-reconstructable intra-articular distal humerus fracture with poor bone quality in elderly patients
  - Contraindicated in active patients <65 years of age due to high rate of mechanical complications (loosening, implant failure). The recommended weight loading of the total elbow prosthesis is 5 kg as a single event and only 1 kg for repetitive loadings

Hand

5

Andreas Weber, Lisa Reissner, Lea Estermann, Ladislav Nagy, Philipp Honigmann, and Andreas Schweizer

Anatomy



Landmarks of the hand, palmar

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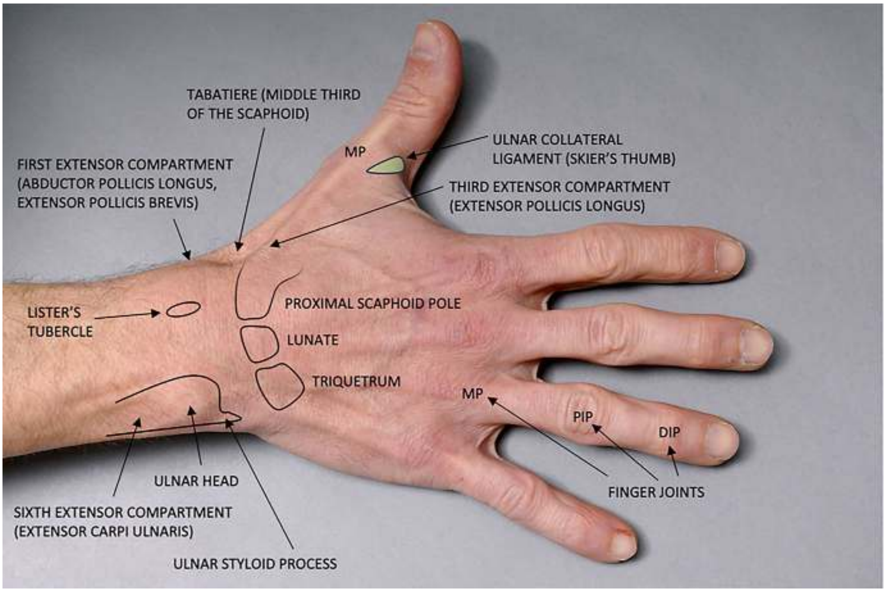


Landmarks of the hand, dorsal

Carpal Tunnel Syndrome (CTS)

Epidemiology

Most common peripheral nerve compression syndrome.

Women > men (3:1), especially postmenopausal, incidence 0.1–1% annually.

Etiology

- Compression of the median nerve in the carpal tunnel, mostly idiopathic
- Anatomical variants such as a persistent median artery (embryologically the main artery to the hand which typically regresses during gestation) increase the likelihood of developing carpal tunnel syndrome
- Carpal tunnel syndrome is very rare in children. However, metabolic disorders (especially mucopolysaccharidosis) are the most common cause of CTS in children
- Acute carpal tunnel syndrome can result from high-energy trauma, haemorrhage, infection, or thrombosis of a median artery and requires emergency surgical decompression
- Increased incidence during pregnancy; this can be temporarily relieved with steroid infiltrations

Anatomy of the Carpal Tunnel

Roof: Transverse carpal ligament (= Retinaculum flexorum)

Floor: Proximal row of carpal bones and deep extrinsic palmar carpal ligaments

Contains the median nerve and nine tendons (flexor pollicis longus tendon, 4 flexor digitorum superficialis tendons, and 4 flexor digitorum profundus tendons)

Clinical Presentation

Pain in the wrist, hand and fingers, occasionally radiating to the shoulder

Intermittent paresthesia and/or numbness (→ thumb to middle finger and radial ring finger)

Diagnosis

- Clinical diagnosis, which can be confirmed and quantified by electrophysiological examinations (electromyography [EMG]/electroneurography [ENG])
- **Clinical examination:**
  - Muscle weakness/atrophy of the thenar muscles
  - Provocation test to trigger paresthesia:
    Hoffmann-Tinel sign (tap the palmar side of the wrist)
    Phalen's test (hold wrist in maximum flexion for 1 min)
    Durkan test (gentle pressure on carpal tunnel for 30 s)
- **Radiographic examination:**
  - Ultrasound provides information about possible pseudoneuroma (swelling of the nerve proximal to the compression site) and nerves ability to freely glide (laterally) during finger movement

Therapy

Conservative Nighttime wrist splint, steroid infiltration

Operative Open or endoscopic carpal tunnel release

→ Surgical interventions are superior to all non-operative methods

Differential Diagnoses

Cervical radiculopathy (specifically C6-C7 syndrome), brachial plexopathy, thoracic outlet syndrome, pronator syndrome, and/or polyneuropathy

Cubital Tunnel Syndrome

Epidemiology

Second most common nerve compression syndrome of the upper extremity
Males > females (females more likely to present at earlier age)

Etiology

Compression and traction of the ulnar nerve at the medial elbow
Sites of compression:

- Medial head of triceps (cross country skier)
- Within the Arcade of Struthers (fascial thickening at hiatus of medial intermuscular septum as the ulnar nerve passes from the anterior to the posterior compartment)
- Medial intermuscular septum
- The distal transverse fibers of the arcade of Struthers
- Osborne ligament (cubital tunnel roof)
- Anconeus epitrochlearis muscle (anomalous muscle—from the medial olecranon to medial epicondyle)
- Between two heads of flexor carpi ulnaris muscle/fascia (aponeurosis)
- Aponeurosis of proximal edge of flexor digitorum superficialis muscle
- Other external sources of compression:
  - Tumors, ganglions
  - Osteophytes, heterotopic ossification, medial epicondyle nonunion
  - Burns
  - Cubitus varus or valgus deformities
  - Medial epicondylitis
  - Repetitive elbow flexion/valgus stress during occupational or athletic activities

Clinical Presentation

Intermittent paresthesia and/or numbness of the small finger and ulnar half of the ring finger/ulnar dorsal hand; exacerbated by prolonged and excessive flexion of the elbow

Diagnosis

- Clinical diagnosis, which can be confirmed and quantified by electrophysiological examinations (electromyography [EMG]/electroneurography [ENG])
- **Clinical examination:**
  - Provocative tests:
    - Direct cubital tunnel compression
    - Hoffmann-Tinel sign
    - Elbow hyperflexion (reproduce or intensify paresthesia)
  - Check for subluxation of ulnar nerve over medial epicondyle during elbow flexion and extension
  - Examination findings secondary to motor weakness
    - Froment sign (compensatory thumb IP joint flexion (FPL) during key pinch due to adductor pollicis weakness)
    - Wartenberg sign (persistent abduction and extension of the small finger during attempted adduction due to weakness of the middle finger palmar interosseous and small finger lumbrical muscles)
    - Masse sign (flattening of the palmar arch and loss of ulnar hand elevation due to weak opponens digiti minimi and reduced small finger MCP flexion)
    - Interosseous and/or first web space atrophy
    - Ring and small finger clawing
- **Imaging**
  - Ultrasound provides information about possible pseudoneuroma (swelling of the nerve proximal to the compression site) and (sub-) luxation of the ulnar nerve

Therapy

Conservative Activity modification, nighttime elbow splint, and NSAIDs

Operative Endoscopic or open release

- In situ decompression
- Anterior transposition
  - Subcutaneous
  - Submuscular
  - Intramuscular
- Medial epicondylectomy
- Higher rate of recurrence than after carpal tunnel release

- Surgery should be performed before motor denervation
- Persistent postoperative medial/posterior elbow pain may be secondary to neuroma formation from iatrogenic injury to branches of the medial antebrachial cutaneous nerve

Differential Diagnoses

- Cervical radiculopathy (C8)
- Ulnar tunnel syndrome (Guyon's canal syndrome): no sensory deficit to dorsum of the hand, no motor deficit to extrinsic muscles, negative elbow flexion test

Wartenberg Syndrome

Synonym: Cheiralgia paresthetica.

Etiology

Compressive neuropathy of the superficial sensory radial nerve at the wrist, between brachioradialis and extensor carpi radialis longus tendons with forearm pronation

Epidemiology

Females > males

Clinical Presentation

Pain and paresthesias over the dorsoradial hand without any motor deficits

Diagnosis

- **Clinical examination**
  - Clinical diagnosis, electrophysiological examinations are of limited value
  - Hoffmann-Tinel
  - Forceful forearm pronation for 60 s (as a provocative test)
- **Imaging**
  - Ultrasound examination provides information regarding possible nerve entrapment

Therapy

Conservative Initially treated by activity modification, splinting, and NSAIDs

Operative Surgical decompression if symptoms persist after 6 months of nonoperative treatment

Differential Diagnoses

- External compression (handcuffs, tight wrist band)
- Tendovaginosis, De Quervain
- Intersection syndrome: irritation and inflammation at the junction of the first and second dorsal compartments
- Supinator tunnel syndrome: Compression of the posterior interosseous nerve at the nerve's entrance into the supinator muscle (Frohse's arcade)

Trigger Finger and Trigger Thumb

Synonym: Stenosing Flexor Tenosynovitis

Etiology

- Inflammatory/degenerative thickening of the first (A1) pulley and swelling of the flexor tendons (chronic tenosynovitis) leads to "catching" when trying to glide through the stenotic area
- In children, the thumb is usually the affected digit whereas in adults the ring finger is most commonly affected. Children also typically present with a nodule of the flexor tendon (Notta's nodule)
- Trigger finger (index to little finger) in childhood is very rare and is usually caused by a metabolic disorder

Symptoms and Findings

Finger snapping, which occur at the PIP or IP joint (sudden recoil of the finger or thumb during extension from a clenched fist). In severe cases, the digit is locked in either flexion or extension.

Diagnosis

- Diagnosis is primarily clinical, but an ultrasound examination is confirmatory. Patients will report tenderness to palpation (tendonitis) over the flexor tendon at the A1 pulley of the affected digit
- Palpation of a thickened tendon or tendon nodule is common. Direct visualization of the trigger phenomenon (thumb in the IP joint, index to little finger in the PIP joints) is confirmatory

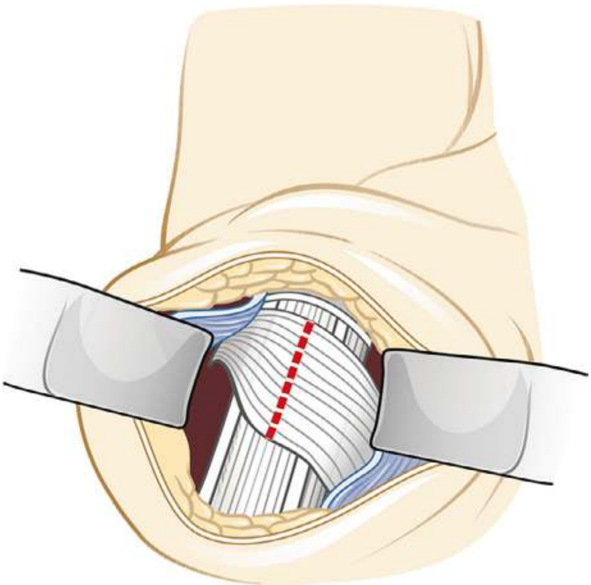
Therapy

Conservative Oral NSAID, flexor tendon sheath injection/infiltration with steroid

Operative Ultrasound guided percutaneous or open A1 pulley ligament release are both effective

Differential Diagnoses

Differential diagnosis for snapping phenomenon in the PIP joint: Swan neck deformity, dislocating lateral band of the extensor tendon



A1 pulley release

De Quervain Tenosynovitis

Epidemiology

Women > Men (6:1), mostly between the ages of 30–50 years

Etiology

Inflammation of the first extensor compartment with the tendons of the extensor pollicis brevis and abductor pollicis longus muscles at the point where they pass the fibro-osseous tunnel at the styloid process of the radius

Clinical Presentation

Pain, swelling, and tenderness to palpation over the first extensor compartment.

Commonly, this presents with pain and loss of strength when lifting/holding objects. This phenomenon is common in the post-partum period (and is often called the "baby wrist").

Rarely, trigger phenomenon occurs with ulnar-radial motion.

Diagnosis

Diagnosis is primarily clinical but an ultrasound examination (to assess the extent of inflammation of the first extensor compartment and the gliding ability of the tendons) is confirmatory

- **Clinical examination:**
  - Finkelstein test: Pain provocation over the radius styloid, when the examiner grasps the patient's thumb and quickly deviates the hand to the ulnar side
  - Eichoff's test: Pain provocation over the radius styloid, when the wrist is ulnar deviated, and while the thumb is grasped in the palm
  - WHAT (Wrist Hyperflexion and Abduction of the Thumb) test: Most specific/sensitive test, pain provocation over the radius styloid, when the thumb is abducted while the wrist is in a hyperflexed position

Therapy

Conservative Immobilization using a wrist splint including the thumb, oral/topical NSAIDs, infiltration of the 1st extensor compartment with steroid

Operative Surgical release of the first extensor (dorsal) compartment

- The tendon of the abductor pollicis longus muscle typically has 2 or more tendinous slips
- The entire extensor pollicis brevis is absent in approximately 6% of the population
- The first extensor compartment is sub-divided in approximately 30% of patients by a longitudinal septum into two tunnels
- → If one is not aware of these anatomical variations, there is the risk of incomplete release of the first extensor compartment

Differential Diagnoses

Basal thumb arthritis, scaphoid fracture, radiocarpal osteoarthritis, intersection syndrome, and/or Wartenberg syndrome

Basal Thumb Arthritis

Synonym: Rhizarthrosis, Osteoarthritis of the CMC joint

Etiology

Degenerative changes of the saddle joint of the thumb

Clinical Presentation

- Pain elicited with key pinch, grasping objects, and spreading of the thumb
- Periarticular soft tissue swelling
- Loss of strength and restriction of movement (adduction contracture)
- Compensatory hyperextension and instability in the IP joint

Diagnosis

- **Clinical examination:**
  - Local pressure tenderness at the palmo-radial corner of the thumb CMC joint
  - Grind test: pain and crepitus in the thumb CMC joint elicited while using combined axial compression and circumduction of the joint

- **Radiographic examination:**
  - Subchondral sclerosis
  - Joint space narrowing
  - Osteophytes
  - Bone cysts

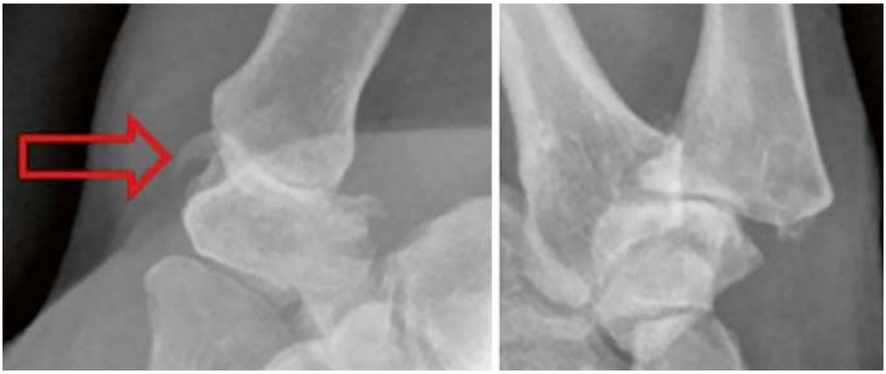
Therapy

Conservative Immobilization in a short thumb splint, oral/topical NSAIDs, infiltration into the CMC joint of the thumb with steroids

Operative Many treatment options exist, most common is the trapeziectomy with suspension/interposition arthroplasty

Surgical therapy options for basal thumb arthritis

- Wilson's osteotomy: A closing wedge osteotomy of the thumb metacarpal bone is performed to relieve the palmar part of the saddle joint and optimize the load zone in the joint. The joint itself is not opened. Indicated for younger patients and those with moderate disease.
- Thumb CMC arthrodesis: Suitable for younger patients, especially those with heavy manual use of the hand (e.g., craftsmen). This procedure is contraindicated in the presence of additional arthritis of the adjacent segments (e.g., realignment osteotomy of the scapho-trapezio-trapezoid joint (STT joint)).
- Resection/Suspension/Interposition arthroplasty: Trapeziectomy (resection) followed by stabilization of the base of the thumb metacarpal bone using tendon strips of the flexor carpi radialis or abductor pollicis longus (suspension) and insertion of the remaining tendon strip into the gap where the trapezium was originally located (interposition). Indicated for older patients with more severe disease.
- Prosthetic joint replacement (arthroplasty): Possible for older patients who do not require a high level of manual use of the hand. Loosening and subluxation are the most common complications.



Saddle joint of the thumb with joint space narrowing and osteophytes (arrow)

Osteoarthritis of the Fingers

Etiology

Degenerative disease of the DIP joints (= Heberden's osteoarthritis) and PIP joints (= Bouchard's osteoarthritis)

Primary osteoarthritis of the MP joints is rare and can occur as part of hemochromatosis

Clinical Presentation

- Pain under load, loss of strength and restriction of movement
- Heberden's nodules dorsal to the joint, mucoid cysts periungual
- Axial deviation and subluxation

Diagnosis

Clinical diagnosis, which is confirmed by imaging (conventional X-ray of the hands in the posterior-anterior view as well as affected fingers individually in posterior-anterior and lateral views)

Therapy

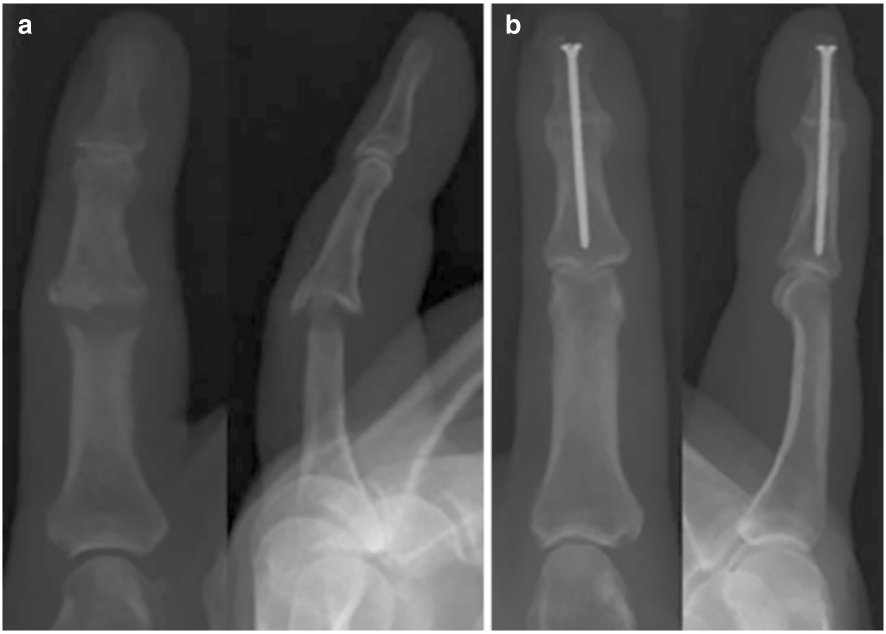
Conservative Immobilization (only short-term), NSAIDs (oral and/or topical), ultrasound-guided steroid infiltration

Operative

DIP joints: Arthrodesis (optimal position for DIP joint arthrodesis: 0–30°)

PIP joints: Arthroplasty (prosthetic joint replacement), rarely arthrodesis (optimal position for PIP joint arthrodesis: Index finger 35°, middle finger 40°, ring finger 45°, little finger 50°)

MP joints: Silicone prosthesis [Swanson prosthesis] or 2-component metal backed PE total joint replacement implants.



(a) Arthroplasty of the PIP joint; (b) Arthrodesis of the DIP joint

Ganglion of the Wrist

Epidemiology

Dorsal wrist ganglions are the most common cause of dorsal central wrist pain without trauma and have a higher incidence than palmar wrist ganglions in adults.

Etiology

Fluid filled swelling overlying a joint synovium or tendon sheath. Typically originates at either the dorsal wrist (from the scapholunate ligament) or the palmar wrist (radially from the radio-scaphoid joint).

Clinical Presentation

- The cyst is typically smooth and rounded with a rubbery consistency upon palpation. Patients typically report load-dependent pain during movement (extension) or end-stage flexion in the wrist
- Small occult ganglions can be difficult to palpate and may have greater pain intensity than larger cysts (due to compression of the cyst by the adjacent bony structures during dorsal extension)

Diagnosis

Clinical diagnosis, which is confirmed by imaging (ultrasound or MRI).

Therapy

Conservative Immobilization in a wrist splint

Aspiration: Ultrasound-guided under local anesthetic with traumatization of the ganglion stem (risk of recurrence greater than with surgery)

Operative Open excision of the ganglion including the stem or arthroscopic resection of the stem

Dorsal wrist ganglion (right side)

Dupuytren's Contracture

Etiology

The exact etiology is not yet fully clarified. Benign fibro-proliferative change, which typically begins as nodules in the palmar aponeurosis with insidious progression to form diseased cords and eventually leads to flexion contractures in the MCP and/or PIP joints.

Clinical Presentation

- Visible and palpable subcutaneous nodules and/or cords, extension deficit in the MCP and PIP joints of the affected ray
- In 50% of patients, bilateral findings are present; men > women; most > 50 years old

Dupuytren's diathesis describes patients with an early onset of disease (< 50 years), a familial predisposition, a rapid progression of joint contractures, often bilateral involvement of the hands with involvement of the radial fingers, and additional extrapalmar manifestations such as:

- Dorsal of the PIP joints (Garrod knuckle pads)
- Penis (Peyronie's disease, Induratio penis plastica)
- Planta pedis (Ledderhose disease)

Diagnosis

Clinical diagnosis (typical finding).

The severity of Dupuytren's contracture is classified according to the Tubiana classification, where the extension deficits in the MCP, PIP and DIP joints of the affected ray are cumulative, known as the total flexion deformity (TFD).

Stage 0	No lesion
Stage N	Palmar nodule without presence of contracture
Stage I	TFD between 0° and 45°
Stage II	TFD between 46° and 90°
Stage III	TFD between 91° and 135°
Stage IV	TFD greater than 136°

Therapy

Conservative

- Collagenase injection into the cords: An enzyme (from Clostridium histolyticum) is directly injected into the cords. After a few days, the cords can be "broken" by extending the fingers. Despite ongoing approval in Europe, in 2019, the manufacturer decided to distribute this drug exclusively in the United States.
- Percutaneous needle fasciotomy
- External beam radiation therapy

Operative Excision of the altered tissue

Kienböck Disease

Synonym: Idiopathic osteonecrosis of the lunate

Epidemiology

- Most common in young males, 20–40 years old
- Rare in children but better prognosis

Etiology

Unclear; multifactorial etiology postulated: Lunate geometry, anatomic variability of lunate blood supply, ulnar minus variance (increased shear stress on marginally perfused lunate)

- Progressive, often debilitating disease, characterized by fragmentation and collapse of lunate

Clinical Presentation

Dorsal wrist pain (tenderness over dorsal lunate), mild swelling, stiffness, and weakness

Diagnosis

➔ Unexplained and persitent dorsal wrist pain in a young adult, unrelated to activity and accompanied by negative ulnar variance, should warrant an MRI evaluation for early disease detection.

Signal change(s) on MRI must be seen throughout the entire lunate to make the diagnosis of Kienböck disease ("black lunate sign")

X-ray may be initially normal

Carpal Height Index (radiographic measurement): Carpal height divided by the 3rd metacarpal bone height (normal range between 0.51 and 0.57)

Stages of Kienböck Disease

Stage I:	Plain X-ray normal, but a linear fracture through the lunate may be noted. MRI demonstrates diffuse T1 signal decrease in lunate.
Stage II:	Sclerosis of the lunate is seen on plain X-ray
	Multiple fracture lines may be seen, though collapse of the lunate has not occurred
Stage III A:	Lunate collapse has occurred, but carpal height normal.
Stage III B:	Lunate collapse has occurred, carpal height decreased.
	Radioscaphoid angle >60°, scaphoid in hyperflexed position ("ring sign")
Stage VI:	Additional carpal osteoarthritis (radiocarpal and/or midcarpal)

Therapy

Depends on disease stage

Conservative Only for Stage I disease, cast/immobilization for 3 months

Operative

Stage II or higher: Joint-levelling procedure. Radial shortening osteotomy (if ulnar-negative variance) or capitate shortening with capito-hamate fusion is used (if ulnar-positive variance).

Stages I to IIIA: Vascularized bone grafting.

Stage IIIB: Addresses the associated carpal instability by STT-fusion, scapho-capitate fusion, or proximal row carpectomy.

Stage IV: Proximal row carpectomy or wrist fusion.

Differential Diagnoses

Ulnar impaction syndrome (MRI findings limited to ulnar side of lunate), lunate fracture, intraosseous ganglion, enchondroma (focal MRI changes within lunate), or intersection syndrome

- Preiser disease is a rare idiopathic osteonecrosis of the scaphoid. In contrast to Kienböck disease, there is no known predisposing vascular patterns in Preiser disease.

Distal Radius Fracture

Epidemiology

Most common fracture of the upper extremities, **1/6 of all treated fractures**

Incidence: two age peaks → (1) Adolescents (high-energy trauma), (2) (mainly) women > 50 years (low-energy trauma in osteoporotic bone)

Etiology

Fall on the extended or flexed wrist

Clinical Presentation

Pain, swelling, and deformity at the wrist

Diagnosis

Clinical presentation and X-ray of the wrist in posteroanterior and lateral views. CT for uncertainties or for assessment of intra-articular fractures.

Several different classifications exist:

- By proper names:
  Colles (dorsal tilt)
  Smith (palmar tilt)
  Barton (intraarticular with dorsal tilt)
  Hutchinson (radius styloid fracture, chauffeur fracture)
- Schematic (> 10 different classification schemes exist)
  AO classification is the most commonly used (3 main types (extra-articular, partially articular and intra-articular) with additional subtypes)

Therapy

Conservative

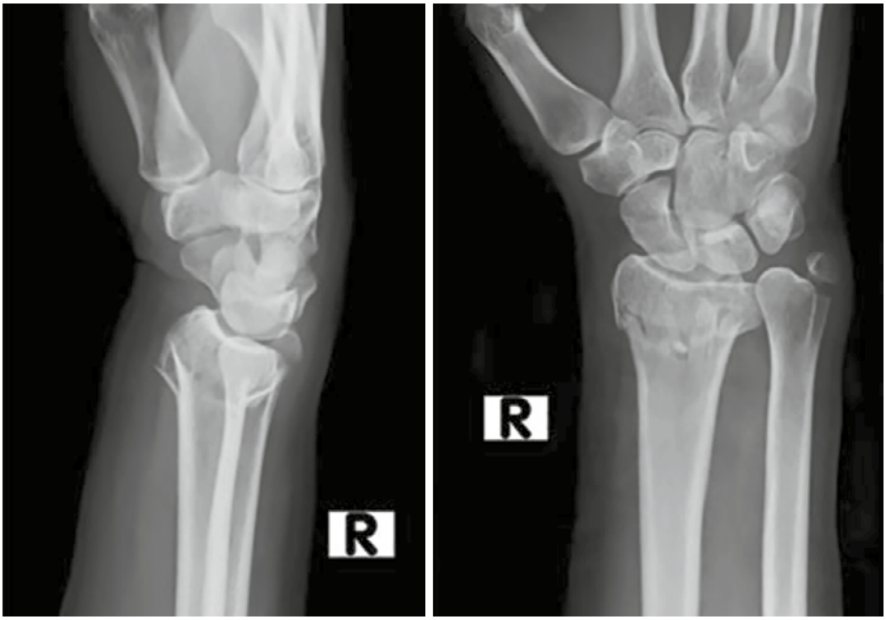
- Non-displaced: Immobilization (cast) for 4–6 weeks
- Displaced and stable: Repositioning under sufficient analgesia followed by immobilization in a forearm cast
- Radiological reevaluation after 10–14 days

Operative

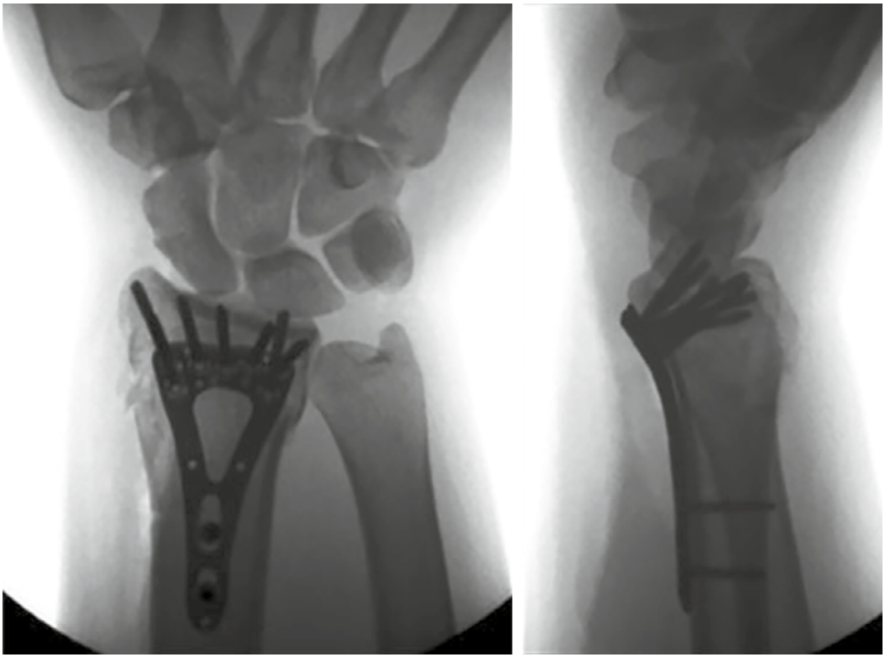
If 3 or more of the following instability criteria are met, there is an indication for surgery:

– Intra-articular radiocarpal fracture
– Dorsal comminution
– Dorsal angulation of > 10–20°
– Age < 65 years
– Additional ulna fracture
– Palmar angulation any

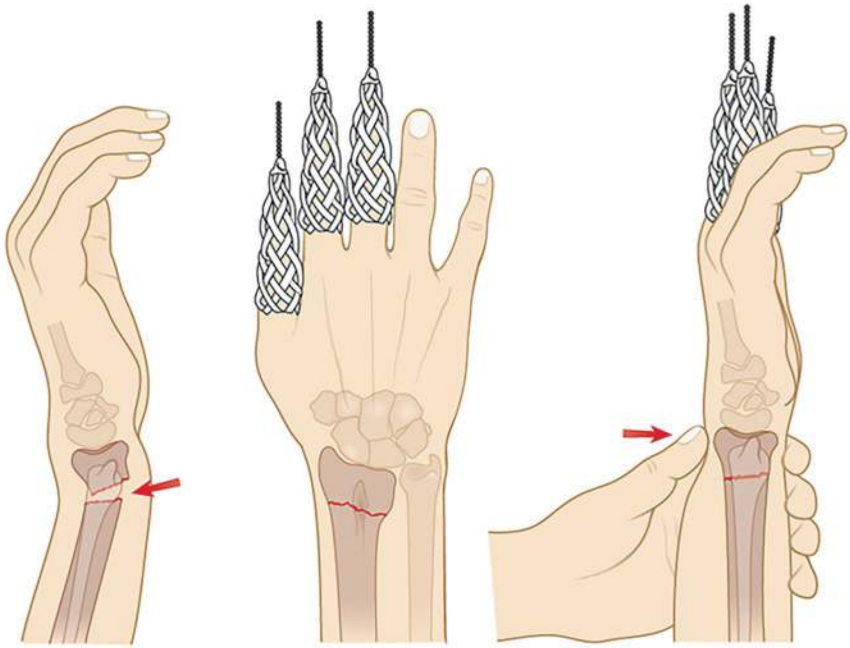
Most common operative treatment: palmar or dorsal plate osteosynthesis

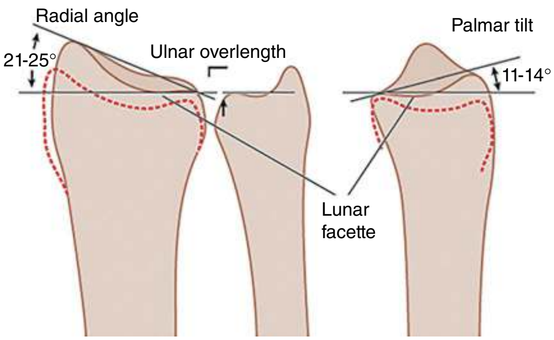


Distal radius fracture with ulnar styloid fracture near the base



Palmar plate osteosynthesis





Goal of therapy: correct reduction with radial angle of 21–25° and palmar tilt of 11–14°, no ulnar overlength

Colles: tilting dorsally; Smith: tilting palmarly

Perilunate Dislocation/Dislocation Fracture

Etiology

Result of high-energy hyperextension trauma; complex, partially combined carpal injury (ligamentous, osseous) with dislocation of the hand (typically dorsally or rarely palmarly)

Can go through up to 4 stages (Mayfield stages)

(1) Stage 1: Scapholunate disruption due to SL ligament rupture (Lesser-arc injury) or scaphoid fracture (Greater-arc injury)
(2) Stage 2: Additional mediocarpal disruption due to additional radio-scapho-capitate ligament rupture
    CAUTION: This is overlooked in up to 25% of cases in the emergency department
(3) Stage 3: additional luno-triquetral disruption due to additional luno-triquetral ligament rupture → dorsal mediocarpal dislocation (capitate is dorsal to lunate on the lateral X-ray view)
(4) Stage 4: additional radio-triquetral ligament rupture → complete palmar dislocation of the lunate

Clinical Presentation

Severely swollen wrist with significant restriction of movement

Dislocations are associated with sensory disturbances in the supply area of the median nerve in up to 25% of patients

Diagnosis

Clinical examination and X-ray of the wrist (posteroanterior/lateral views). If X-ray is unrevealing but suspicion for perilunate dislocation persists, CT should be obtained.

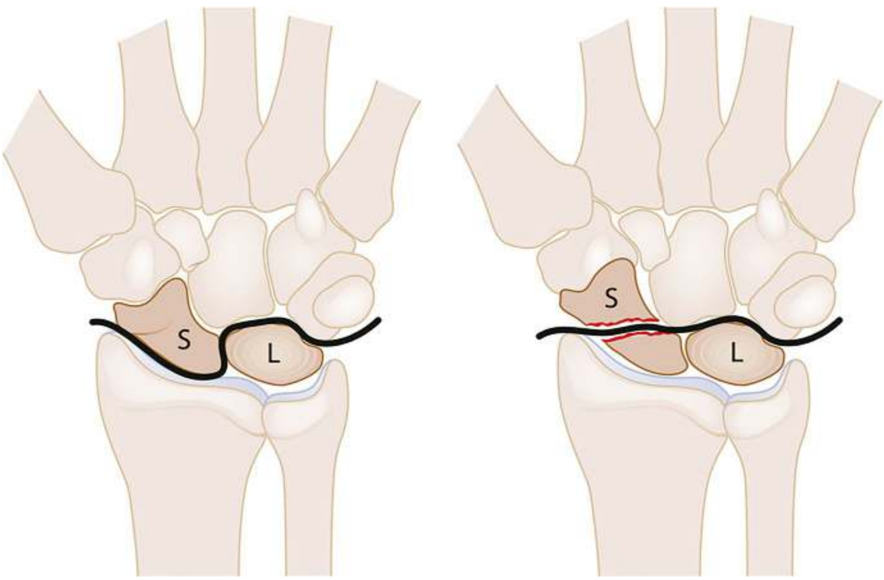
Classification:

- Perilunar dislocation (Lesser-arc injury): purely ligamentous injuries without fracture
- Perilunar dislocation fracture (Greater-arc injury): additional fracture of one or more carpal bones, usually scaphoid

Therapy

Perilunar dislocation injuries are a surgical hand emergency!

- Immediate **reduction** of the carpus and **surgical repair** of the fractures and/or ligaments. Late diagnosis leads to consistently poor outcomes.



Pattern of perilunar dislocation (left); perilunar dislocation fracture (right)

P-A X-ray: Superimposition of the proximal carpal row on the distal carpal row. Lateral X-ray: dorsal dislocation of the distal carpal row (arrow = empty articular surface of the lunate bone)

Scaphoid Fractures and Scaphoid Fracture Nonunion

Epidemiology

Most common fracture of the carpal bones (79% of all carpal bone fractures)

Men > women (4:1), peak age between 20 and 30 years

Etiology

Typical mechanism of injury results from a fall on an outstretched hand with wrist in hyperextension and radial deviation

In 70–80% the middle third of the scaphoid is fractured and in about 20% the proximal third; fractures of the distal pole are the rarest

Diagnosis

- **Clinical examination**
  - Tenderness on palpation over the anatomical snuffbox, over the proximal and/or distal pole of the scaphoid
  - Painful axial compression of the thumb
- **Imaging**
  - X-ray of the wrist (posteroanterior/lateral views)
  - CT (or MRI) if there is the slightest suspicion of a scaphoid fracture
  - The **humpback deformity** can occur in fractures in the middle third of the scaphoid due to a tilting of the distal scaphoid portion in flexion position (towards palmar) and the proximal portion in extension position. This “hump formation” is clearly visible in the lateral X-ray image.
  - The **DISI malalignment** (*Dorsal Intercalated Segment Instability*) refers to a malalignment of the carpal bones with the lunate bone rotated dorsally (in extension position) and occurs due to bony changes (humpback deformity of the scaphoid) or ligamentous injuries (scapho-lunate ligament rupture). In a DISI malalignment, the scaphoid-lunate angle is >60° and the capitate-lunate angle is >15° on a lateral wrist X-ray.

Therapy

Conservative Non-displaced scaphoid fracture: Immobilization of the wrist for 8–12 weeks in a cast, followed by a CT to confirm consolidation (timing of cast immobilization should be longer for more proximal fractures)

Operative Displaced fracture or fracture of the proximal pole: Open reduction and internal fixation with cannulated screw osteosynthesis

Potential Complications

Healing of the scaphoid is most uncertain in fractures involving the proximal pole due to the retrograde (distal) blood supply. Healing is best in the distal third.

Scaphoid nonunion—can occur after non-operative or operative therapy

Risk factors:

- Overlooked fracture or delay of therapy onset >4 weeks
- Fracture of the proximal pole

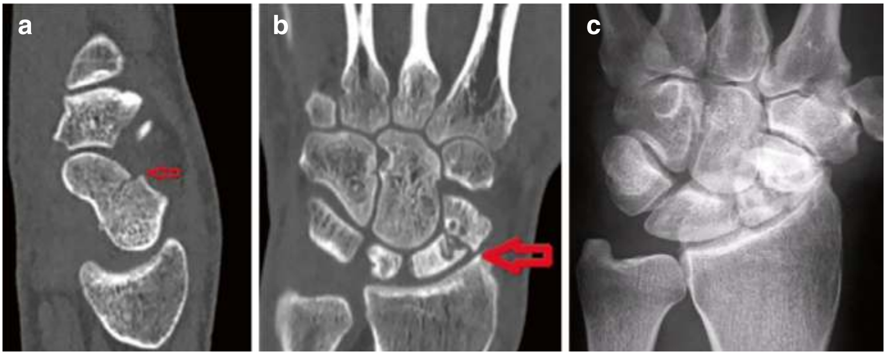
– Displacement of >1 mm
– Smoking

Treatment of scaphoid nonunion

– Nonunion resection and scaphoid reconstruction with bone graft or vascularized bone graft from the distal radius

Scaphoid Nonunion Advanced Collapse (SNAC)—refers to the instability and resulting arthritic changes in the wrist due to a chronic/improperly healed scaphoid fracture. SNAC wrist progresses in 4 distinct stages:

(1) Stage I: Osteoarthritis is localized to the radial side of the scaphoid and radial styloid
(2) Stage II: Additional osteoarthritic changes occur between the scaphoid and scaphoid fossa of the radius
(3) Stage III: Additional osteoarthritic changes occur between the lunate and capitate
(4) Stage IV: Panosteoarthritis of the wrist



(**a**) Acute scaphoid fracture; (**b**) Scaphoid pseudoarthrosis; (**c**) SNAC wrist

CAUTION
- At the slightest suspicion of a scaphoid fracture, a CT should be obtained
- Missed scaphoid fractures can lead to chronic scaphoid pseudarthrosis

Fractures of the Metacarpal Bones

Clinical Presentation

Pain, swelling of the hand, possible misalignment—especially **rotational deviation.**

Diagnosis

Clinical examination and X-ray of the hand (posteroanterior/lateral views)

Therapy

The tolerance limit of axial deviation for conservative treatment varies depending on the ray and location of the fracture (diaphyseal vs. metaphyseal/neck fractures):
- The lowest tolerance of less than 15° palmar tilt is for diaphyseal metacarpal fractures of the index finger
- The highest tolerance limit of up to 50° palmar tilt is for metacarpal neck fractures of the little finger (= Boxer's fracture*)*

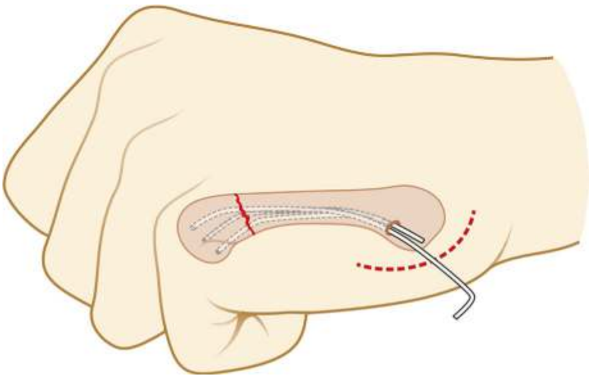
Conservative
- Stable fractures without rotational deviation (often internal rays (middle and ring finger) due to high intrinsic stability) or
- Not or only slightly (within the tolerance limit, see above) displaced fractures

→ Immobilization in a splint for 4–6 weeks

Operative Osteosynthesis using Kirschner wires, screws, screws/plate, or intramedullary nailing is appropriate for:

- Unstable fractures
- Fractures displaced beyond the tolerance limit
- Rotational deviation
- Multiple fractures

(**a**) oblique fracture; (**b**) spiral fracture; (**c**) subcapital fracture



Intramedullary nailing

CAUTION
Rotational deviations must not be missed!

Intraarticular Fracture of the Metacarpal Bone of the Thumb

Clinical Presentation

Pain, limited movement, and swelling

Diagnosis

Clinical examination and X-ray of the thumb or CT

Classification of the base fractures of the metacarpal bone of the thumb:

- Winterstein fracture: extraarticular base fracture
- Bennett fracture: classic intraarticular two-part dislocation fracture
- Rolando fracture: intraarticular three-part fracture/comminuted fracture

Therapy

Operative Reposition and osteosynthesis

All of these fracture types are usually unstable and typically dislocate due to the pull of the abductor pollicis longus muscle while the base fragment remains fixed by the strong carpo-metacarpal ligaments.

Fractures of the Phalanges

Epidemiology

Most common fractures in the hand

Clinical Presentation

Swelling, pain with limited movement, possible axis deviation, and rotational deviation

Diagnosis

Clinical presentation and X-ray of the finger or CT if intraarticular involvement is suspected

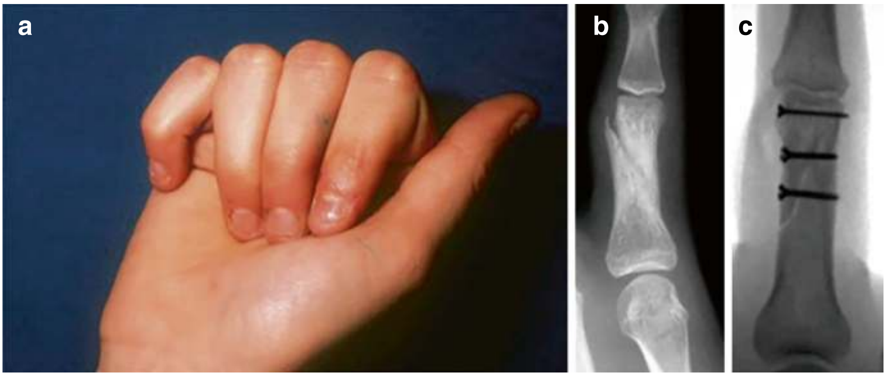
Therapy

Conservative Stable fractures without rotational deviation

→ Immobilization in splint for 4 weeks

Operative Unstable fractures, axis deviation, and/or rotational deviation

→ Osteosynthesis using Kirschner wires, screws, or screws/plate



(**a**) Rotational deviation due to a phalangeal fracture; (**b**) Oblique fracture of the basal phalanx; (**c**) Screw osteosynthesis

Dislocations of the PIP Joint

Etiology

Axial trauma or hyperextension trauma
- Dislocation direction (lateral vs. dorsal vs. palmar) is significant in terms of injured structures, therapy, and outcome.

Clinical Presentation

Misalignment, restricted movement, pain, and swelling

Diagnosis

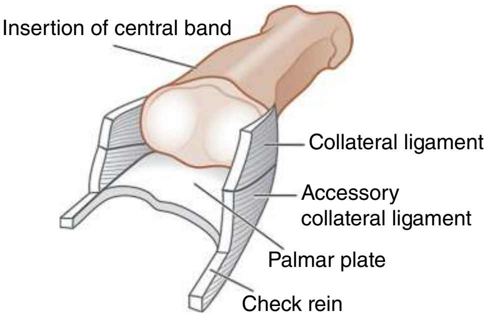
Clinical examination and X-ray of the finger (posteroanterior/lateral views), ultrasound

Pathomechanics

- Lateral PIP dislocation: Unilateral collateral ligament rupture and partial or complete avulsion of the palmar plate
- Dorsal PIP dislocation: Lesion of the palmar plate/fracture of the palmar base of the middle phalanx
- Palmar PIP dislocation: Rupture of the central band (rare)

Therapy

- Lesion of the collateral ligaments: Buddy taping
- Lesion of the palmar plate: Extension stop splint / buddy taping
- Lesion of the central band: Immobilization of the PIP joint in extension for 6 weeks



The stabilizing structures of the PIP joint

Ulnar Collateral Ligament Injury (Skier's Thumb)

Etiology

Acute lesion of the ulnar collateral ligament of the thumb MCP joint *(incl. lesion of the palmar plate)* due to forced radial deviation of the thumb

- An interposition of the adductor aponeurosis between the ligament stumps is called a Stener lesion. This situation makes conservative therapy impossible *(due to lack of proximity)*.
- Lesions of the ulnar collateral ligament of the thumb MCP joint are 10 times more common than lesions of the radial collateral ligament.

Clinical Presentation

Pain, swelling, hematoma

Diagnosis

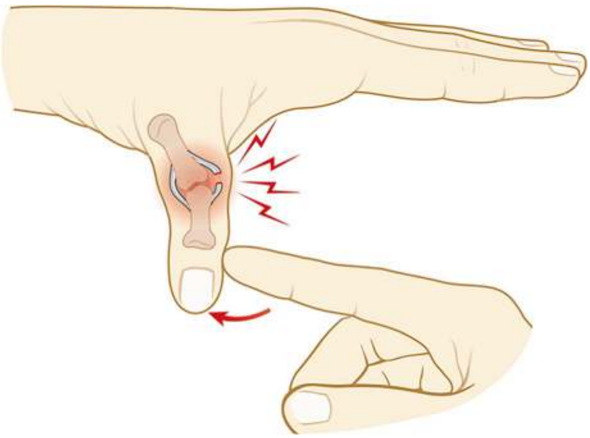
- **Clinical examination**
  - Lateral instability and increased laxity to lateral stress

    The ulnar collateral ligament of the thumb MCP joint is under maximum tension at 40° flexion. This is therefore the best position to clinically test the integrity of the ligament.
- **Imaging**
  - Ultrasound (if suspicion for a Stener lesion)
  - X-ray thumb

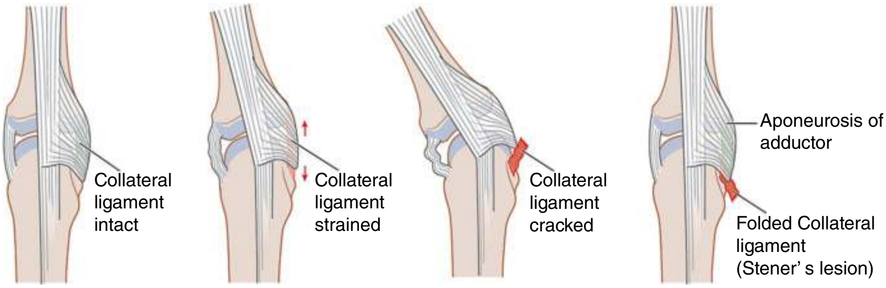
Therapy

Conservative Without Stener lesion: Immobilization in a splint for 6 weeks

Surgical With Stener lesion: Refixation of the ulnar collateral ligament



Increased lateral instability and increased laxity to lateral stress



Pathomechanics of a Stener lesion

Mallet Finger

Classification

A distinction is made between a tendinous mallet finger and an osseous mallet finger.

Etiology

Rupture of the extensor tendon above the DIP joint *(tendinous mallet finger)* or fracture of the dorsal terminal phalanx base *(osseous mallet finger)* with consecutive active extensor deficit

Clinical Presentation

Extension deficit, swelling and pain over the DIP joint

Diagnosis

Clinical examination
- Pain over the DIP joint

Imaging
- Ultrasound
- X-ray of the finger

Therapy

Conservative Consistent immobilization of the DIP joint in an extension position in a splint for 8 weeks

Surgical Reduction of dislocated osseous mallet lesions / large fragments and fixation with Kirschner wires or plates

Giant Cell Tumor of the Tendon Sheath

Epidemiology

Second most common soft tissue mass of the hand (after ganglions). Benign soft tissue tumor. Most common in the third to fifth decades of life.

Clinical Presentation

- Presents as slow-growing, nontender, multi-lobulated mass
- The tumor is most often firm, nodular, and nontender
- Typically located on palmar side of the hand
- Often involves the radial three digits and the DIP joint region
- Contains multinucleated giant cells, xanthoma cells, and hemosiderin deposits (histology similar to pigmented villonodular synovitis)
  - Giant cell tumor of the tendon sheath is not a particularly good term because the lesion does not uniformly contain giant cells and is not necessarily associated with a tendon sheath

Diagnosis

Imaging

- Ultrasound, X-ray of the finger/hand (anteroposterior/lateral views), and/or MRI
  - Soft tissue mass or pressure erosion of underlying bone
  - Bone invasion not typical—suggests a more aggressive neoplasm (seen as well with diffuse pigmented villonodular tenosynovitis)

Therapy

Operative Marginal excision

CAUTION: high risk for recurrence (up to 50%).
➔ satellite lesions or incomplete excision

– Risk factors for local recurrence:
  degenerative joint disease
  location at the DIP joint
  radiographic evidence of pressure erosion

Sarcoma

Epidemiology

15% of all soft tissue sarcomas occur in upper extremity
Risk factors:

- Radiation and toxins (herbicides)
- Genetic factors (Neurofibromatosis and Li-Fraumeni syndrome)

Most common histological subtypes of soft tissue sarcomas in the hand are:

- Epithelioid sarcoma
- Synovial sarcoma
- Malignant fibrous histiocytoma

Clinical Presentation

Painless mass that may have been present for a long time with recent growth

Soft tissue sarcomas are frequently misdiagnosed as an infection, ganglion, or lipoma

Diagnosis

History and clinical examination, ultrasound, X-ray of the finger/hand, and/or CT/MRI

Biopsy

- **Excisional biopsy**: For very small lesions. Excision with a adequate border of normal tissue, before diagnosis is known
- **Incisional biopsy:** For large lesions or lesions in proximity to major nerves, vessels, or tendons
  - Incisional biopsy can result in the contamination of all exposed tissues

Staging

After the diagnosis is made, a systemic staging should be done with CT (chest & axilla)

➔ Soft tissue sarcomas of the upper extremity most commonly metastasize to the lungs or regional lymph nodes (LN)

- However, LN metastasis in <5% of soft tissue sarcomas of extremities
- Exception: Epithelioid sarcoma ➔ risk for LN metastasis up to 42%

Therapy

Operative

Think and plan for **resection** and **reconstruction**

- Resection of the lesion is of primary importance
- Reconstruction must be given secondary importance

The goal of surgery is complete removal of the tumor with a sufficient surrounding cuff of normal tissue

If negative margins cannot be achieved with wide excision, amputation indicated

Extensive reconstruction may be required after wide excision in the hand

Adjuvant Therapy

Adjuvant Therapy such as brachytherapy/radiation or chemotherapy has to be evaluated in an interdisciplinary team of oncologists, radiologists, pathologists, and hand surgeons

Malignant Bone Tumors

Epidemiology

Most common hand malignancy are metastases from a primary lung carcinoma, usually involving distal phalanx

Colon, kidney, and breast malignancies may also metastasize in the hand

Clinical Presentation

Acute, localized pain and/or swelling. Trauma (frequently) can be the event that directs attention to the tumor.

Acral metastasis have a poor prognosis (< 6 months survival expected at time of discovery).

The three most common primary malignant bone tumors of the hand are:

1. Chondrosarcoma
2. Osteosarcoma (most common primary malignancy of bone in pediatric patients)
3. Ewing sarcoma

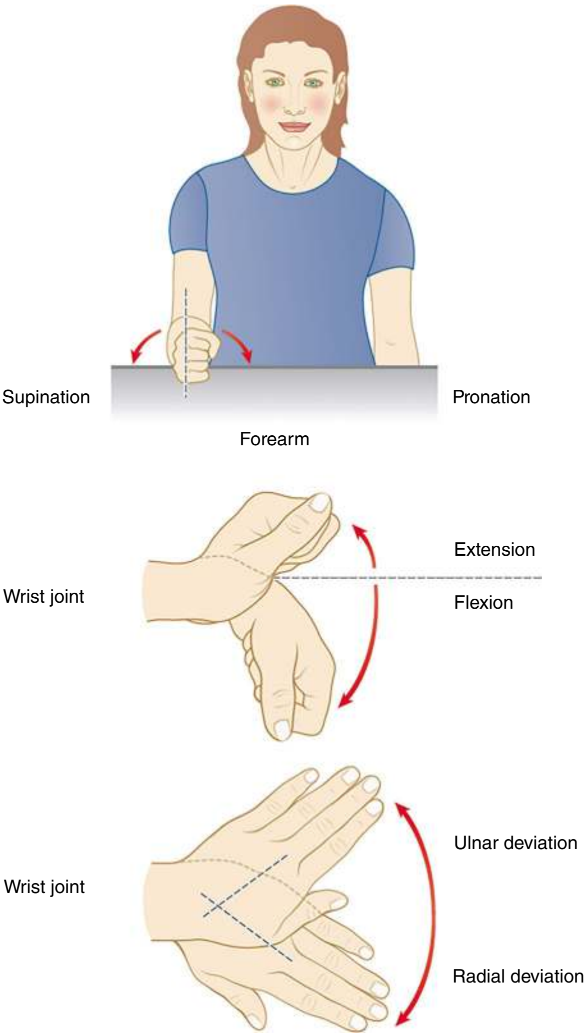
Location is usually metacarpals or phalanges

Therapy

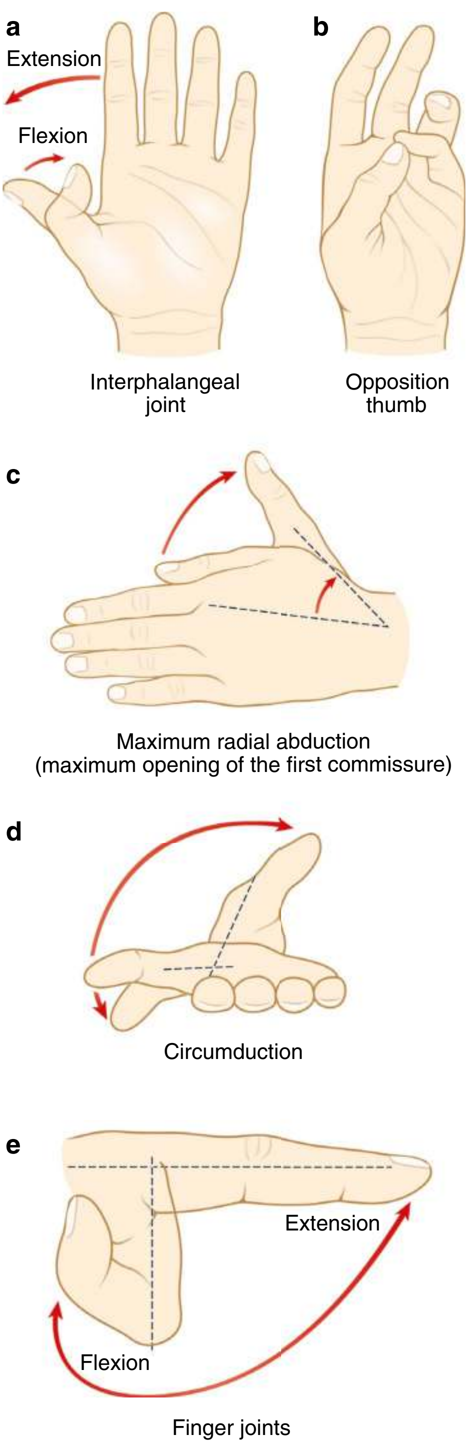
- The treatment for each tumor is the same as elsewhere in the body.

Important Examinations

1. **Inspection**
2. **Palpation → Anatomical landmarks**
3. **Global test/Grasping forms**
4. **Range of motion measurement**



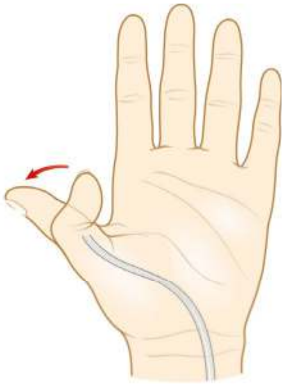
Directions of movement and their nomenclature



Further directions of movement and their nomenclature

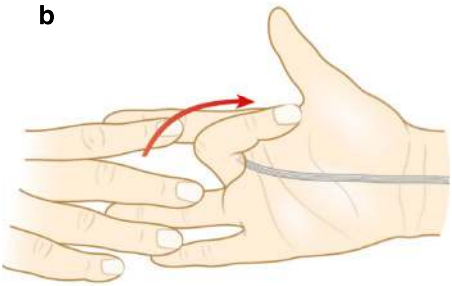
5. **Examination of tendon function**

a



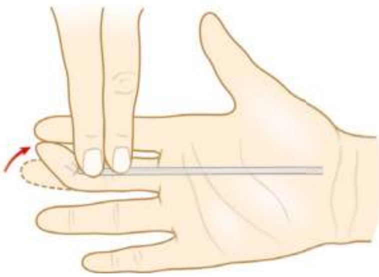
Flexor pollicis longus tendon

b

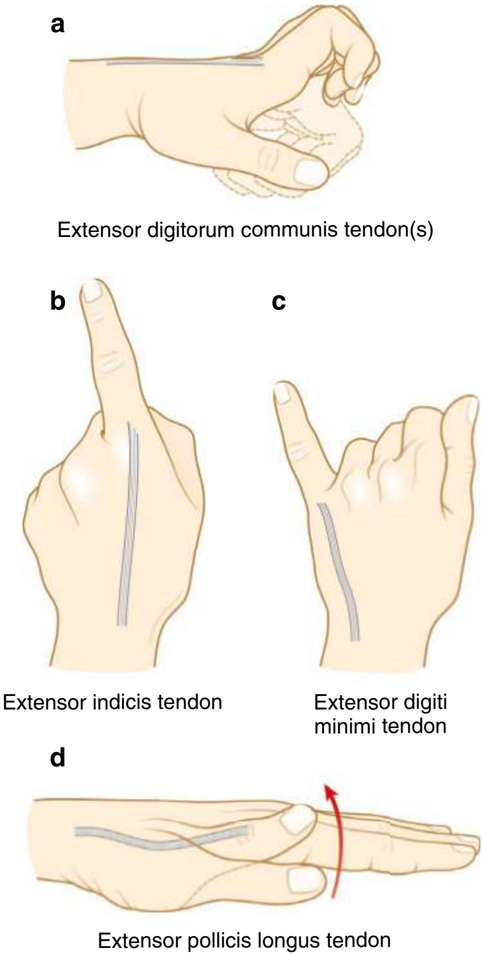


Flexor digitorum superficialis (III) tendon

c



Flexor digitorum profundus (III) tendon

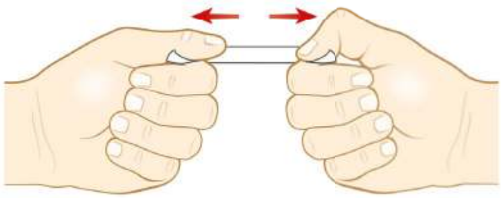


6. **Examination of the intrinsic function**

a

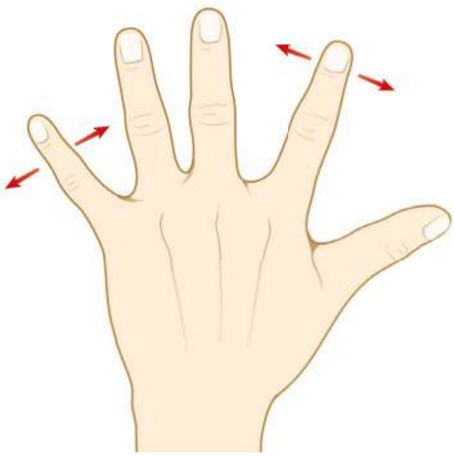
Opposition: M. opponens, M. abductor brevis

b



Froment´s test: M. adductor pollicis

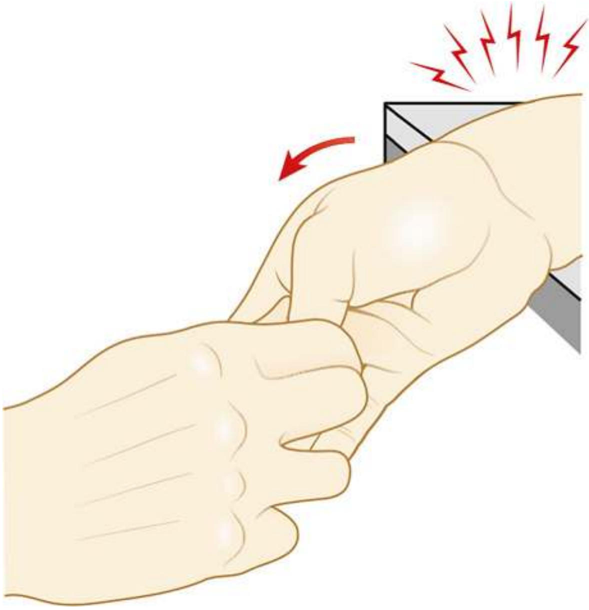
c



Finger abduction/adduction: Mm. interossei

Froment test (middle): Testing the function of the adductor pollicis muscle: Hold a piece of paper between the thumb and the radial side of the index finger, apply traction—the test is positive when the thumb flexes in the IP joint

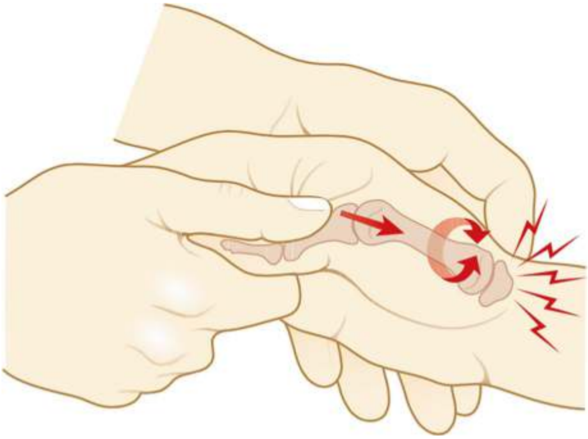
7. **Palpation/pain provocation tests (tendon/tendon sheath)**



Triggering pain in synovitis in the first extensor tendon compartment (Finkelstein's test)

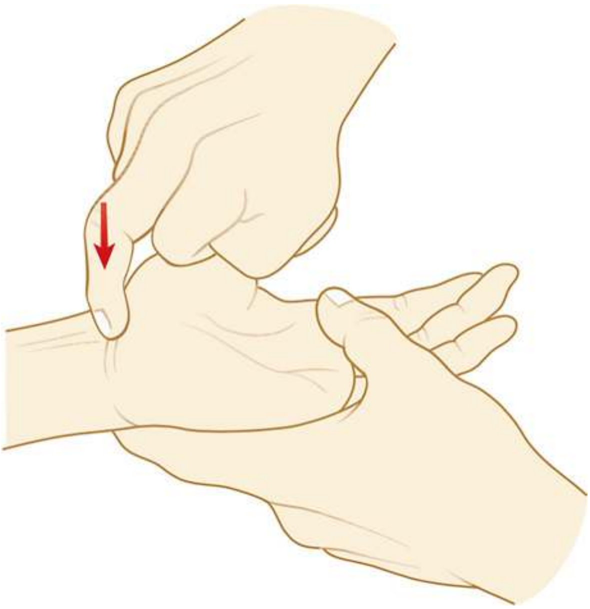
8. **Palpation/Pain Provocation Test (Joints)**

Gaenslen's test: painful compression of the metacarpophalangeal joints indicating arthritis

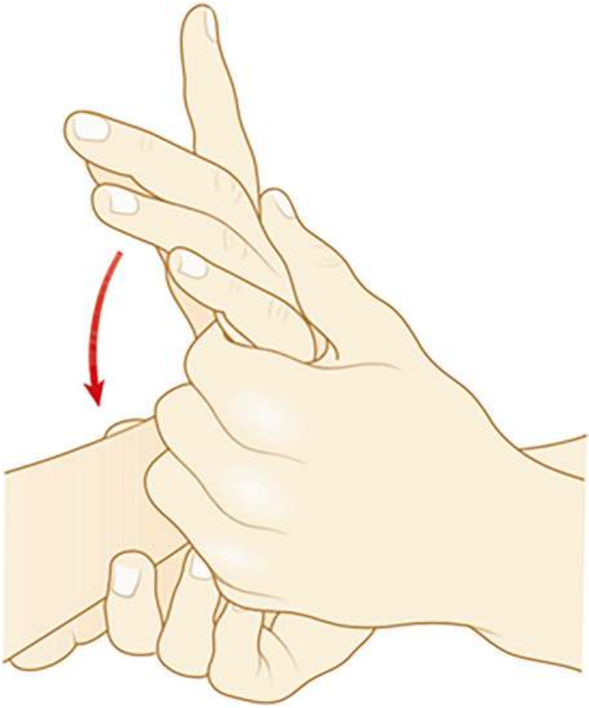


Grind test: Axial sprain pain with crepitus during rotational movement. Typical for Osteoarthrosis of the saddle joint of the thumb

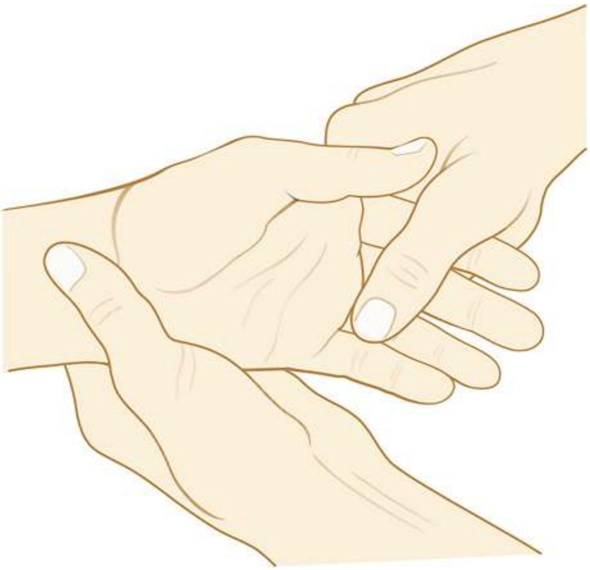
9. Nerve (dys)function tests



Hoffmann-Tinel sign: tapping of the median nerve. Test positive: triggering of paresthesia



Phalen test: 60 s of maximal wrist flexion. Test positive: triggering of paresthesia



Durkan test: Local pressure on carpal tunnel. Test positive: triggering of paresthesia

Hip

6

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Anatomy and Biomechanics

Hip Joint

- Ball-and-socket joint
  - *Socket extends beyond the equator of the sphere*
  - Socket is extended by the labrum (fibrous cartilage)
- Head covered by hyaline cartilage
  - Only the lunate surface is covered by articular cartilage, the acetabular fossa is cartilage-free
    - Located in the acetabular fossa: the pulvinar (a collection of fat and fibrous tissue in the space adjacent to the medial wall of the acetabulum) and the caput femoris ligament (attaches to the fovea)

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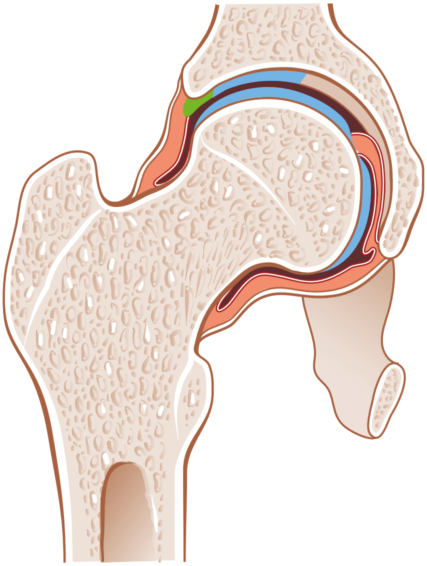
M. Farshad (ed.), *Orthopedics Must Knows*,
https://doi.org/10.1007/978-3-662-70893-4_6


Articular cartilage in blue, labrum in green

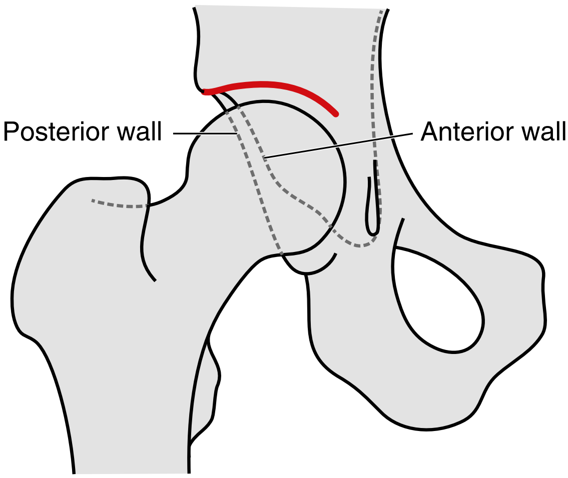
Acetabulum

- Three bones form the acetabulum: ilium, pubis, and ischium
  - The anterior column is composed of the anterior/upper ilium (anterior wall and dome of the acetabulum) and pubis (superior pubic ramus)
  - The posterior column is composed of the ischium (posterior wall, posterior dome of the acetabulum, greater and lesser sciatic notches) and the posterior aspect of the ilium (ischial tuberosity)

    The quadrilateral plate is the flat surface of bone lying directly medial to the acetabulum and connects the anterior and posterior columns of the acetabulum. It is still controversial whether the quadrilateral plate belongs to the anterior or posterior column
- Orientation:
  - Anteversion of approximately 15°
  - Inclination of approximately 45°

    The anterior and posterior walls usually do not cross on a plain pelvis radiograph

    The acetabular sourcil is a radiographic feature referring to the roof or the weight-bearing area of the acetabulum and is characterized by increased sclerosis. Clinical importance: increased upsloping indicates acetabular dyplasia (Tönnis angle)



The anterior and posterior wall usually do not cross on pain pelvis radiographs. The acetabular sourcil is shown in red

Femur

- CCD (caput-collum-diaphyseal) angle:
  - Normal range in adults is between 125 and 135° (<120° coxa vara, >140° coxa valga)
  - The CCD is higher at birth (140°) and decreases with ageing (120°)
  - Used for surgical planning in proximal femur fractures but also several other pathological conditions

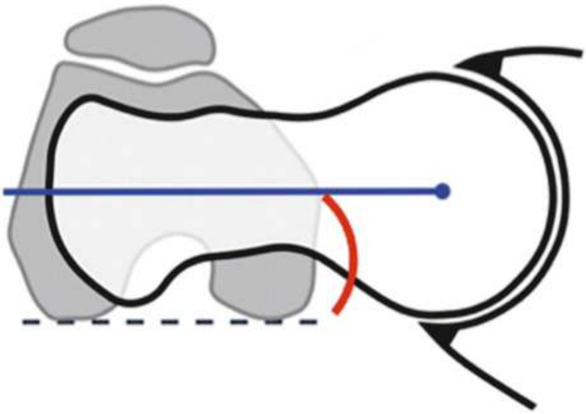
The caput-collum-diaphyseal (CCD) angle

- Femoral torsion:
  - Refers to the orientation of the femoral neck in the coronal plane in relation to the femoral condyles at the level of the knee
  - Femoral antetorsion is higher at birth (30–40°) and decreases to 12 ± 10° in adults
  - Pathological femoral torsion affects the hip range of motion:

    Femoral torsion ↑ = increased internal hip rotation

    1. May lead to ischiofemoral hip impingement (and therefore anterior subluxation due to dorsal impingement)

    Femoral torsion ↓ = decreased internal hip rotation



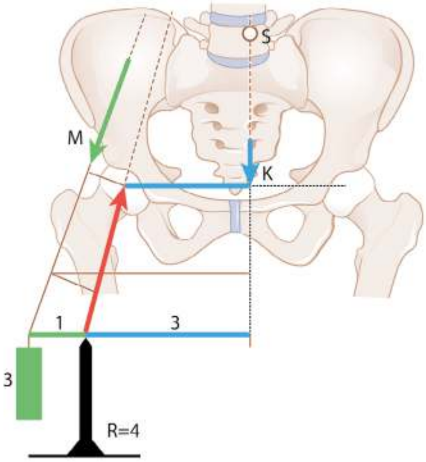
Femoral torsion is the axial angle between the femoral neck and a line connecting the femoral condyles. 0° degrees of femoral torsion is shown here, corresponding to reduced femoral torsion or femoral retrotorsion

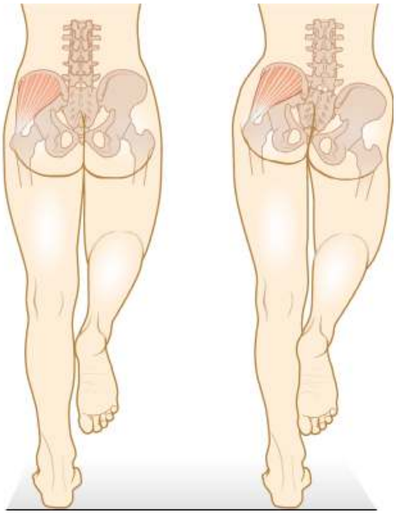
- Main blood supply of the femoral head
  - Medial circumflex femoral artery (MCFA)
  - Topography: The deep branch runs along the proximal border of the quadratus femoris and extends to the lateral aspect of the greater trochanter where it crosses the obturator externus tendon posteriorly. As the deep branch moves superiorly, it crosses anterior to the conjoint tendons of the inferior gemellus, internus obturator and superior gemellus. It then perforates the joint capsule at the level of gemellus superior. In its intracapsular segment it runs along the posterosuperior aspect of the femoral neck, dividing into two to four subsynovial retinacular vessels
  - Clinical relevance: The integrity of the external obturator is critical for femoral blood supply in surgical hip dislocation procedures

    If indicated for ORIF of posterior wall/column fractures, dissect conjoint tendons 2 cm from insertion to protect blood supply. Tenotomy (of the conjoint tendon) does not affect the tension and course of the MCFA branches as long as the external obturator is intact

Traumatic hip dislocation (with rupture of the external obturator tendon) or displaced femoral neck fractures are associated with a high risk of avascular necrosis (up to >40%)

Hip Forces According to Pauwels





Schematic representation of the forces in the hip according to Pauwels on the left. Trendelenburg's sign (hip drop to the opposite side) shown on the right

> Body weight × Load arm = Hip abductor lever arm × Hip abductor muscle force.

The hip abductor lever arm is about three times smaller than the load arm, so the abductors must exert a multiple of the muscle force (M; green) to compensate for the body weight (K; blue). This results in a correspondingly high hip joint reaction force (R; red), which is 4 times the body weight. Depending on the activity, this can increase to between 4 and 6 times body weight

Clinical Relevance

- Trendelenburg's sign
  - Weak or paralyzed abductor muscles
  - Positive when the pelvis drops to the opposite side when standing on one leg
  - To compensate for the impaired abductors, the patient shifts the center of gravity to the affected side, and shortens the lever arm of the bodyweight,

thereby reducing the forces required by the hip abductors to maintain posture (Duchenne limp). A cane held in the contralateral hand reduces joint reactive forces through the affected hip by up to 50% by reducing abductor muscle pull

- Differential diagnosis of Trendelenburg's sign
  - Extended load arm
    - Dysplasia with subluxation
    - Iatrogenic after hip joint replacement
  - Shortened hip abductor lever arm
    - Coxa breva or valga
    - Iatrogenic after hip joint replacement with decreased femoral offset
  - Reduced abductor preload
    - Trochanteric elevation due to varisation osteotomy, trauma (malunion of fracture), deformity (coxa plana)
  - Disturbed neuromuscular unit
    - Muscle/tendon tear of the abductors (*"rotator cuff of the hip"*), superior gluteal nerve injury, L5 radiculopathy
- Coxa valga is associated with a reduced abductor lever arm length with increased joint reaction force and overuse of the hip abductors leading to lateral hip pain
  - The same applies for excessive femoral antetorsion. However, it depends on how the patient holds the leg in space ➔ biomechanically relevant when the patient holds the foot straight but not when it is internally rotated

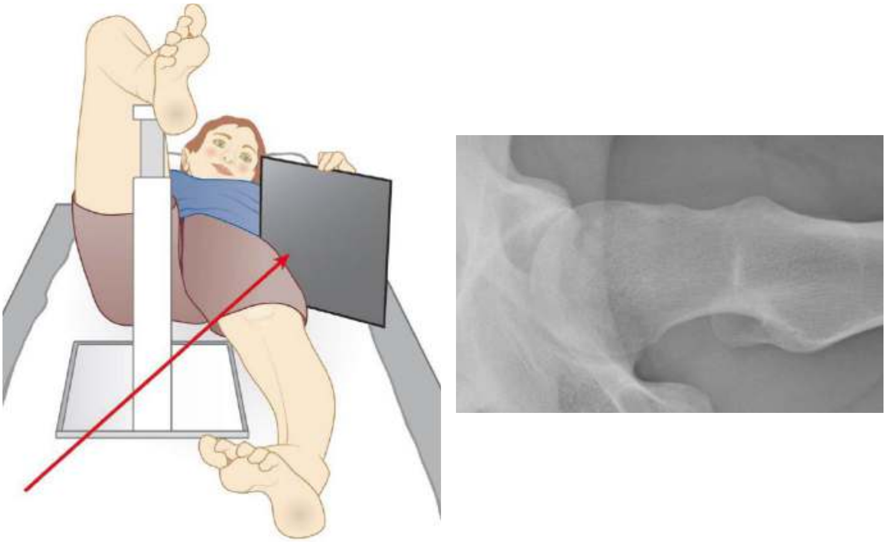
Imaging

Conventional X-Ray

- Plain radiographs in 2 planes: AP pelvis and lateral hip ("cross table" most common)
  - Ensure quality of AP pelvis radiograph in terms of inclination and rotation:
    - Coccyx and symphysis pubis should be in a straight line and positioned in the middle line of the image. The distance between the superior border of the pubic symphysis and the tip of the coccyx should be between 1 and 2 cm (less important)
    - Both sides of the iliac wings and obturator foramina should be symmetric
  - Teardrop:
    - Radiographic feature resulting from projection of a bony ridge running along the floor of the acetabular fossa. Boundaries of the teardrop are formed medially by the quadrilateral plate, laterally by the acetabular fossa, and basally by the acetabular notch
  - Radiographic assessment:
    - Lateral center-edge (*LCE*) angle: <23° indicates dysplasia, >34° signals acetabular overcoverage, and >40° protrusion

Acetabular roof angle or AC index or Tönnis angle (angle between the sourcil and a horizontal): >13° indicates hip dysplasia, values less than 3° indicate acetabular overcoverage

Signs of acetabular retroversion: crossover sign (anterosuperior rim extends laterally to the posterosuperior rim), prominence of ischial spine sign (PRISS) (ischial spine projects medially to the pelvic brim), posterior wall sign (posterior wall deficiency so that the wall is medial to the femoral head center). Highest sensitivity/specificity for acetabular retroversion with all three signs and a crossover (or retroversion) index of >30% (quantifies overlap of anterior and posterior wall in case of a positive crossover sign)



Cross table lateral view of the left hip

Plain AP hip radiograph with anatomic landmarks

- Specialized views:
  - Outlet view:
    The patient is positioned as in the AP view of the pelvis, but with the beam tilted 40° cephalad
    Shows the anterior ring superimposed on the posterior ring, and the sacrum appears in its longest dimension with the neural foramina visible
    For assessment of cephalic/caudal translation and superior migration of the hemipelvis following trauma
  - Inlet view:
    Patient is positioned as in AP view of pelvis but with beam tilted 40° caudally
    S1 overlaps S2
    Assesses for anteroposterior displacement of the pelvic ring or rupture of the pubic symphysis
  - Iliac oblique:
    The beam is rotated 45° away from the injured side
    For assessment of the ilioischial line of the posterior column, the posterior column, the roof of the acetabulum, the anterior wall, and iliac crest
  - Obturator oblique:
    The beam is rotated 45° opposite from the injured side
    Demonstrates the iliopectineal line of the anterior column, the anterior column of the pelvis, the posterior acetabular wall and the obturator foramen

Computed Tomography (CT)

- For assessment of fractures or osseous deformities and torsion

Magnetic Resonance Imaging (MRI)

- Standard MRI is used to assess soft tissue (muscle or tendon inflammation or tears) or bone pathology (oedema, necrosis, fracture)
- Combined with arthrography, it allows to visualize the articular cartilage, labrum, and capsule, as well the bone marrow of the femur and acetabulum. This is the exam of choice for any intra-articular pathology
- Alpha angle:
  - Angle between the axis of the femoral neck and the axis between the center of the femoral head and the head-neck junction
  - Quantification of bony deformity at the femoral head-neck junction
  - Radial images allow assessment of the alpha angle over the entire circumference. Most commonly prominent in the anterosuperior region
  - A threshold of 55° to 60° best discriminates between volunteers and patients with cam deformities and normal head-neck junction

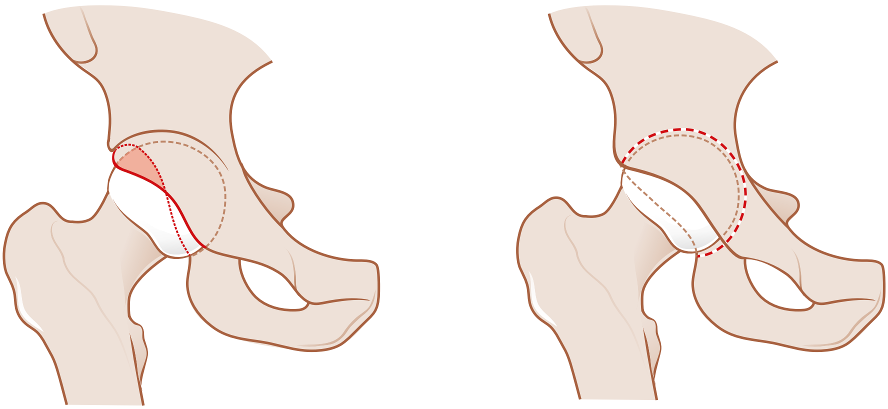
- For assessment of femoral torsion (requires axial images of the knee)
  – The most commonly used technique is that described by Murphy, in which a line is drawn from the center of the femoral head to the centroid of the femoral neck just above the lesser trochanter

Femoroacetabular Impingement

Etiology

Eccentric closure of the growth plate. Competitive sports in childhood: Stop-and-go and jumping sports (e.g. ice hockey, football) have a tenfold increased risk of cam deformity. Secondary impingement occurs after Perthes, SCFE, PAO (iatrogenic) or trauma (femoral neck fracture)

- Cam impingement:
  – Refers to femur-based disorder
  – Males > females
  – Sphericity mismatch causes shearing at the chondro-labral junction
- Pincer impingement:
  – Refers to acetabulum-based disorders and/or reduced femoral torsion
  – Common in active, middle-aged women
  – Femoral neck collides with acetabular bone overhang and crushes the labrum
  – Localized conflict in acetabular retroversion, focal antero-superior overcoverage, or global conflict in deep acetabulum (acetabular protrusion)



left = retroversion, right = acetabular protrusion

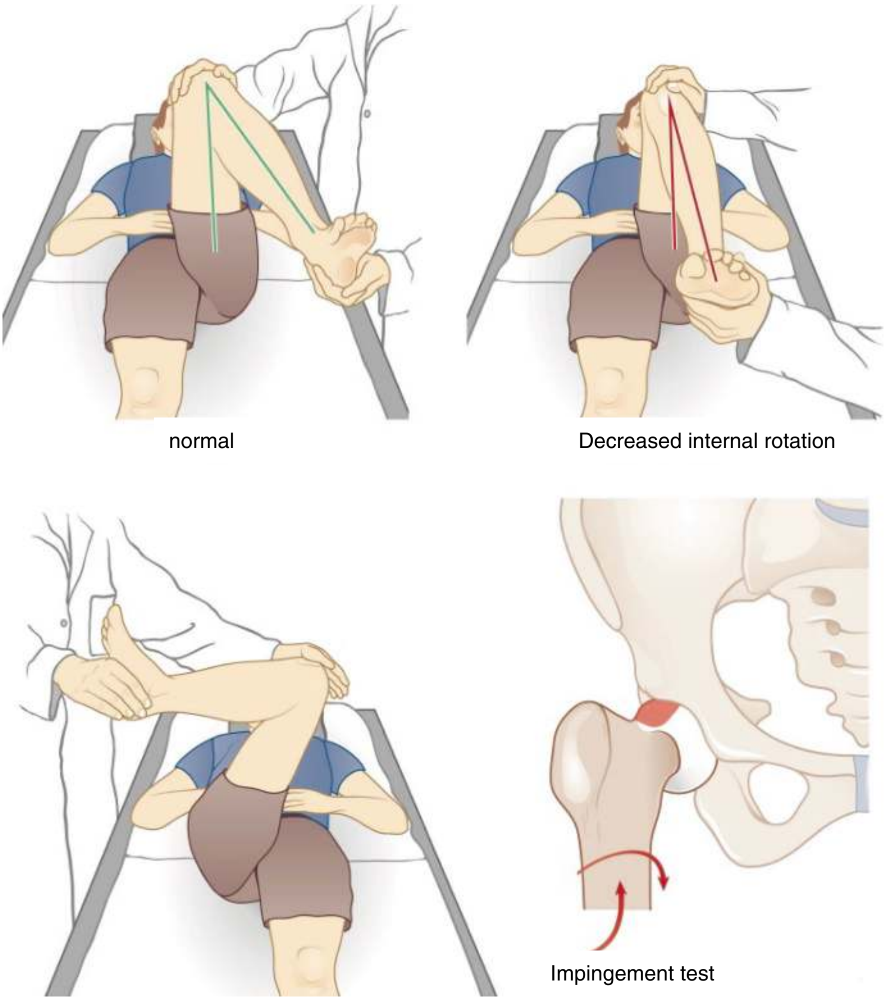
- A combination of the above is present in >85% of cases

Clinical Presentation

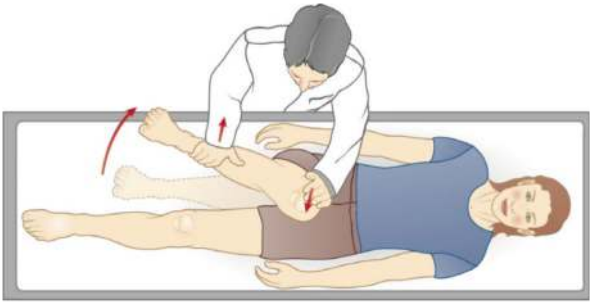
Activity-related groin pain, mainly on hip flexion with difficulty sitting. May be associated with gluteal or trochanteric (lateral) pain due to abnormal gait mechanics

Diagnosis

- Clinical examination
  - Limited internal rotation and flexion
  - Positive anterior impingement test (provocation of the labrum and therefore pain with flexion, adduction, internal rotation; FADIR)



Anterior hip impingement test



- Radiographic examination
  - X-ray
    - Asphericity and contour of femoral head and neck (pistol grip deformity)
    - Acetabular retroversion or acetabular protrusion, focal antero-superior overcoverage
    - Impingement cysts in the area of the acetabular roof or at the head-neck transition (herniation pits)
    - Labrum calcifications, os acetabuli (rim fracture due to impingement)
  - MR arthrography
    - Assesses for articular cartilage and labral damage
    - Can assess anatomy of femoral head/neck junction abnormalities (alpha angle) on radial sequences
    - Allows measurement of femoral torsion when additional sequences are obtained at knee level

Therapy

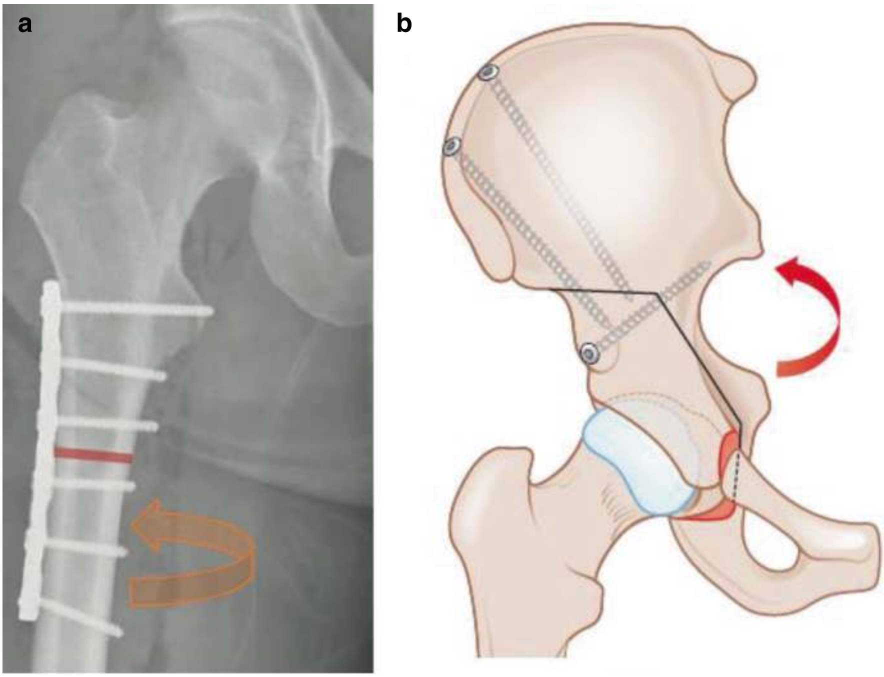
For symptomatic patients only (cam and pincer deformities present in 37% and 67% of the asymptomatic population, respectively)

Conservative symptomatic therapy with the aim of relieving symptoms

- Activity modification (load reduction), NSAIDs, physiotherapy, intra-articular infiltration (should be used restrictively in young patients)
- Even slight residual symptoms with conservative therapy are a sign of persistent joint overuse → Cam morphology increases the risk of osteoarthritis by 5–10% and should therefore be treated early with joint-preserving surgery, preferably before cartilage lesions occur

Operative address underlying bony pathology with the goal of an impingement-free internal rotation of 25–30°

- Hip arthroscopy vs. open surgical hip dislocation
  - Cam impingement → femoral head/neck trimming
  - Pincer impingement → acetabular rim trimming (in case of minor retroversion/focal overcoverage with negative posterior wall sign and retroversion index <30%). Iatrogenic dysplasia has to be avoided
  - Labral repair versus debridement
- Subtrochanteric rotational osteotomy of the femur
  - Indicated for reduced femoral torsion of ≤0°
- Reversed periacetabular osteotomy (PAO)
  - Indicated for truly retroverted acetabulum
- Negative prognostic factors for the surgical outcome: Extent of cartilage damage, patient age > 40 years



Adult Hip Dysplasia

Etiology and Presentation

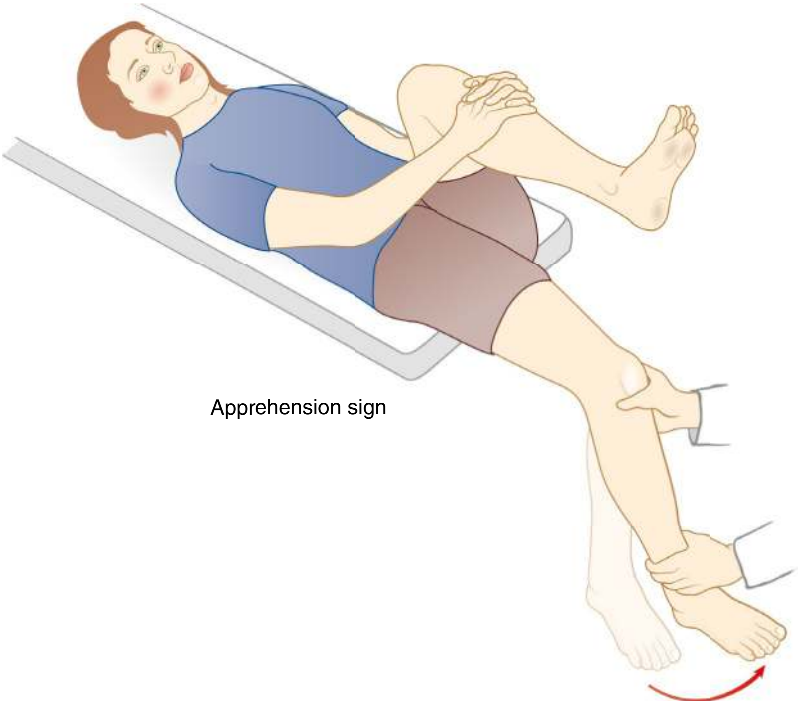
Abnormal movement of the femoral head within the acetabulum due to bony abnormalities. It is estimated that 10% of all total hip arthroplasties (less in Switzerland) are performed as a result of dysplasia. Half of all people under 50 with a hip replacement have hip dysplasia as the cause of OA. Risk factors: fetal breech position, female sex, primiparity and positive family history

Clinical Presentation

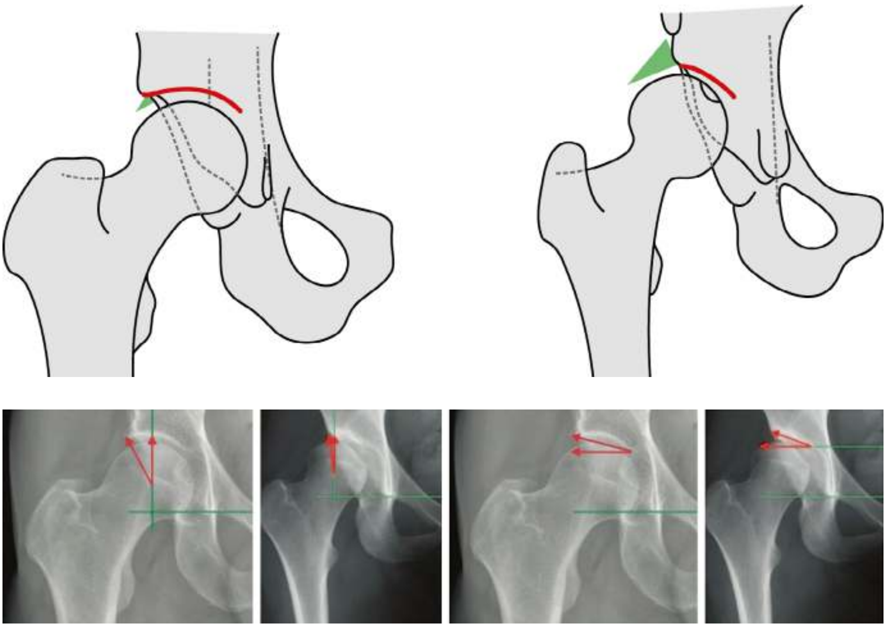
Hip pain with prolonged walking or standing. Lateral hip pain due to abductor overload (s*ubluxation of the head laterally → Load arm becomes longer and power arm shorter, lateral lesion of labrum due to subluxation*), limping (Trendelenburg) due to abductor fatigue

Diagnosis

- Clinical examination
  - Above-average hip range of motion (with excessive internal rotation in case of excessive femoral antetorsion)
  - Abductor irritation or insufficiency
  - Positive provocation test with flexion, internal rotation, adduction (equivalent to the anterior impingement test) as the femoral neck strikes the hypertrophic, degenerated labrum
  - Apprehension sign: External rotation at full hip extension causes anterior subluxation of the femoral head, resulting in anterior hip pain



- Radiographic examination
  - X-Ray:
    Short and oblique sourcil (insufficient head coverage)
    AC index >13°
    LCE <23°
    Coxa valga
    Look for signs of acetabular retroversion or cam deformity, which may coexist and lead to impingement, especially if not corrected during PAO
    Degree of proximal displacement and femoral head subluxation according to the Crowe classification. The Hartofilakidis classification distinguishes between dysplasia, low and high dislocations, where the femoral head creates a false acetabulum superior to the true acetabulum
  - MR arthrography
    Hypertrophic labrum with ganglion formation due to overload
    Maltorsion



LCE (left) and AC index (right) in normal and dysplastic hips

Therapy

Conservative Inadequate femoral coverage and mechanical instability lead to increased stress, resulting in degeneration of the labrum and cartilage. Supportive measures are of limited value given premature progression to secondary OA. Key is prevention through screening and treatment of congenital dysplasia. Ideally, newborn babies should have an ultrasound measurement of the acetabulum (using the Graf method)

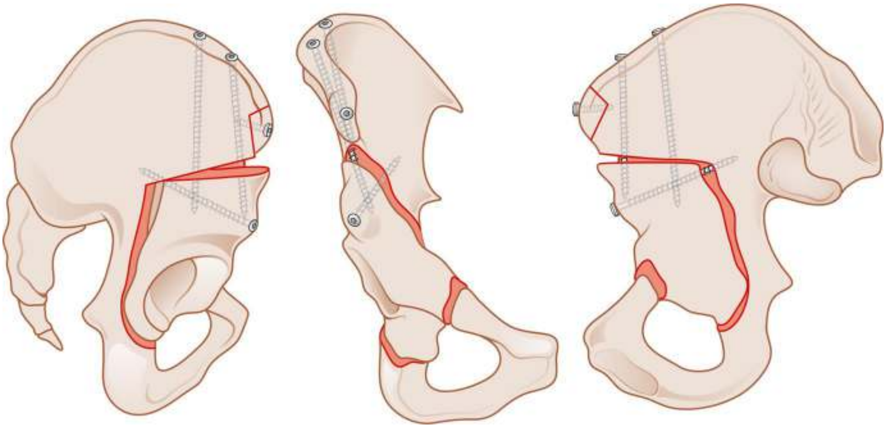
Operative Periacetabular osteotomy (PAO)

- Indicated for symptomatic dysplasia in adolescents or adults
- PAO allows large multidirectional corrections. The acetabular fragment is released from the rest of the pelvis by four osteotomies and a controlled fracture, but the posterior column is preserved

• Hip replacement
  - Treatment of choice for patients with end-stage OA secondary to dysplasia
  - Long-term follow-up shows higher revision rate for THA in dysplastic hips

    Increased dislocation profile due to increased femoral antetorsion and CCD—Requiring specific intraoperative care with component orientation

– Crowe IV hips may need:
  Femoral shortening osteotomy due to risk of sciatic nerve injury (as a traction injury if limb length change >4 cm)
  Repositioning trochanteric osteotomy rarely required, but should be considered if the trochanter is malpositioned, which can lead to poor abductor mechanics



Periacetabular osteotomy

Hip Osteonecrosis

Etiology

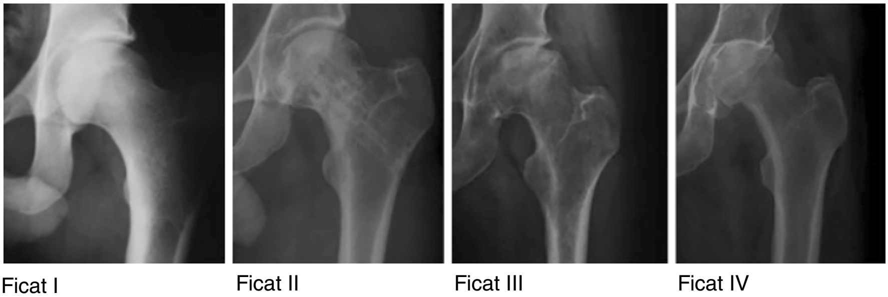
May be a result of trauma with damage to the vascular blood supply, such as hip dislocation or femoral neck fracture. Risk factors for atraumatic avascular necrosis (pathophysiology: intravascular coagulation) include radiation therapy, chemotherapy, alcohol, high dose steroids, sickle cell disease, diving (dysbaric), smoking, and metabolic disorders (Morbus Gaucher, etc.)

Clinical Presentation

The most common presenting symptom is pain in the region of the affected hip, thigh, groin and buttock, which is more likely to occur when walking or standing. However, a small number of patients may remain asymptomatic until the late stages

Diagnosis

- Clinical examination
  - Painful and reduced range of motion (especially rotation)
  - Eventually cracking or locking of the joint when moving the hip
  - Decreased leg length if the head is collapsed
- Radiographic examination
  - X-ray:
    The distinction between pre-collapse and post-collapse stage has prognostic relevance (corresponding to stage 2b according to *Ficat-Arlet classification*)
    Crescent sign: linear subchondral sclerotic zone, usually in the anterolateral portion of the femoral head. If a crescent sign is present, the probability is high that the head will collapse in the further course



Ficat-Arlet classification of hip osteonecrosis

- MRI (gold standard):
  To be ordered when radiographs are negative but osteonecrosis is still suspected (has highest sensitivity and specificity)
  The presence of bone marrow edema is predictive of worsening pain and future disease progression. The modified Kerboul method adds the angles of visible hip necrosis on sagittal and coronal MR images. A combined angle of necrosis > 240° correlates with a high risk of collapse. 50% chance of collapse after 3 years with an angle of 190–240°

Therapy

Conservative low evidence

- Restricted weight bearing: to reduce pain but does not prevent collapse
- Modification of risk factors

Table 6.1 Ficat-Arlet classification

Stage	Symptoms	Radiograph	Scintigraphy		Therapy
0	Asymptomatic	Normal	Uptake ↓	**Pre-collapse stage**	Symptomatic treatment versus core decompression Rotational osteotomies
1	Symptomatic	Normal	Uptake ↓		
2a	Symptomatic	Sclerosis or cystic changes	Uptake ↑		
2b	Symptomatic	Crescent sign (subchondral collapse)	Uptake ↑	**Post-collapse stage**	Rotational osteotomies versus Total hip replacement
3	Symptomatic	Subchondral collapse with flattening of femoral head	Uptake ↑		
4	Symptomatic	Advanced degenerative changes	Uptake ↑		Total hip replacement

- Prostaglandins, LMWH (low molecular weight heparin), statins to improve circulation (vasodilatation) for patients with a hypercoagulable state
- Bisphosphonates: inconsistent und limited evidence. Indicated for precollaps necrosis (Ficat stages 0-II)

Operative According to the Ficat stage (see Table 6.1)

- Pre-collapse stage (Ficat ≤2a): conservative or joint-preserving treatment
  - No clinical superiority of core decompression over conservative management according to recent studies
- Post-collapse stage (Ficat >2b):
  - Rotational osteotomies for small/focal lesions in the weight-bearing area (Kerboul angle <200°) and signs of early subchondral collapse (Ficat IIb)

    Survival rates of intertrochanteric osteotomies vary widely at mid-term follow-up (40–70%)

    Distorts the femoral metaphysis, making secondary THA more difficult. Functional results are inferior to primary total hip arthroplasty
  - Total hip replacement: survival rate of 83% at 15 years in patients <40 years old (old data—results probably better with modern arthroplasty)

Hip Osteoarthritis

Etiology

Mechanical deformity (e.g., dysplasia, impingement, osteonecrosis, post-traumatic after acetabular fracture), inflammatory disease (rheumatoid arthritis, monarthritis), septic (bacterial) arthritis, or idiopathic (which has almost disappeared since the description of FAI)

Clinical Presentation

Function-limiting hip pain, pain at rest/night, stiffness

Diagnosis

- Clinical examination:
  - Restricted range of motion (especially internal rotation)
  - Inguinal pain provoked by extreme movements
  - Distinguish between articular vs. muscular (peritrochanteric, psoas) or spinal/neurologic pain

    Failure to respond to intra-articular injection of local anesthetic is a strong negative predictor of surgical outcome
- Radiographic examination
  - X-ray: Joint space narrowing, joint space incongruence, subchondral sclerosis, subchondral cysts, osteophytes
  - MRI or aMRI is rarely indicated when osteoarthritis is clearly visible on standard radiographs. They may be useful if there is a discrepancy between early signs of osteoarthritis on radiographs and clinical examination, or to diagnose muscle pathology, such as gluteal tendonitis or tears

Therapy

Conservative symptomatic treatment → NSAIDs, activity modification, physical therapy, intra-articular injections (corticosteroids)

Operative

- Total hip arthroplasty:
  - 96–98% survival at 20 years (but depending on patient's age at time of implantation)
- Rare:
  - Femoral head resection (Girdlestone): only in case of repeated infections, and /or if the general condition does not allow a total hip replacement

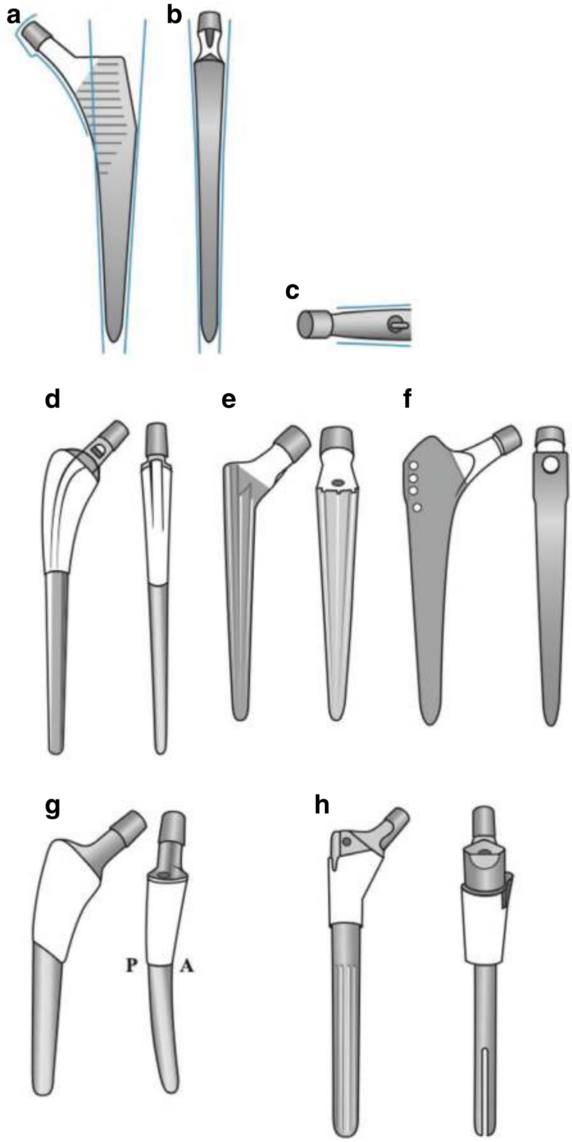
    Walking with crutches is still possible, but the leg is significantly shortened and there is no abduction strength
  - Hip arthrodesis: very rare indication, in cases of recurrent infection and relative contraindication to total hip arthroplasty

Total Hip Arthroplasty

The artificial hip joint functions like a natural ball-and-socket joint, allowing for a wide range of motion and mimicking the natural mechanics of the hip.

Stem:

- Press-fit stems rely on biologic fixation, cemented stems on cement fixation
- Cementless stem designs: single wedge, tapered round/cone/rectangular, anatomic, modular, and with full or proximal coating
  - Made from titanium (highly biocompatible and corrosion resistant with high durability, Young's modulus closer to bone than CoCr)
  - Most stems are proximally coated (for bone ingrowth) that taper distally
    - Stem coating: plasma-sprayed titanium and hydroxyapatite
    - Theoretically, short stems maintain better bone density (less stress shielding) in the trochanteric zone by finding fixation in the metaphysis, but the effect on survival rates is not yet proven
  - Specific complications: increased risk of intraoperative fracture or early loosening in the absence of osseointegration
- Cemented stem designs:
  - Made of stainless steel
  - Preferred for irradiated bone due to the bone's limited ability for ingrowth
  - Preferred for poor proximal femoral bone stock (Dorr C) or osteoporosis
- Revision stems:
  - Uncemented or cemented, proximally porous-coated long-stem prosthesis with or without a modular junction
  - The stem must bypass the most distal defect by 2 cortical diameters
  - Endoprosthetic replacement (tumour prosthesis): in case of massive bone loss with non-supporting diaphysis



Tapered femoral stems can be described by their geometry: single taper, double taper, and triple taper. Single-taper stems have a reduction (taper) in the medial-lateral dimension in the frontal plane, but have a constant anteroposterior dimension (**a**). Double-taper stems have tapers in the frontal and sagittal planes (**b**). Triple-taper stems have a reduction in anteroposterior dimension through the cross-section of the stem from lateral to medial (**c**). Tapered round (**d**), tapered spline/cone (**e**), tapered rectangular (**f**), anatomic (**g**) and modular (**h**)

Cup:

- Cemented or uncemented (hemispherical or elliptical) cups
  - Pressfit occurs at ischium, anterior inferior iliac spine, and os pubis
  - Hemispherical cups are often "oversized" (i.e. 50 cup prepared with a 49 reamer), elliptical cups are usually prepared "line-to-line" (i.e. 50 cup prepared with a 50 reamer)
  - Double mobility cup systems offer greater range of motion, reducing the risk of impingement and dislocation
- Acetabular revision, or difficult primary cases:
  - Implant selection according to the bone defect

    Primary cups or revision cup, eventually secured with screws

    (i) Postero-superior zone as a safe zone for screw placement

    (ii) In the postero-inferior zone, the screws should not exceed 20 mm, otherwise the sciatic nerve as well as the inferior gluteal vessels and nerve are at risk

    (iii) Anterior zone should be avoided (danger zone for iliac vessels and femoral nerve)

    Acetabular augments

    (i) Usually trabecular metal to enhance stability and support in significant bone loss. Its structure promotes bone ingrowth and provides a secure interface with the implant, helping to restore anatomy and distribute loads more naturally. This is an alternative for bone graft, decreasing the potential risk of non-healing/necrosis of the graft.

CAGES:

The Ganz ring:

(i) Support provided cranially and on the medial acetabular wall, eventually on the lateral ilium if used with an additional lateral flange

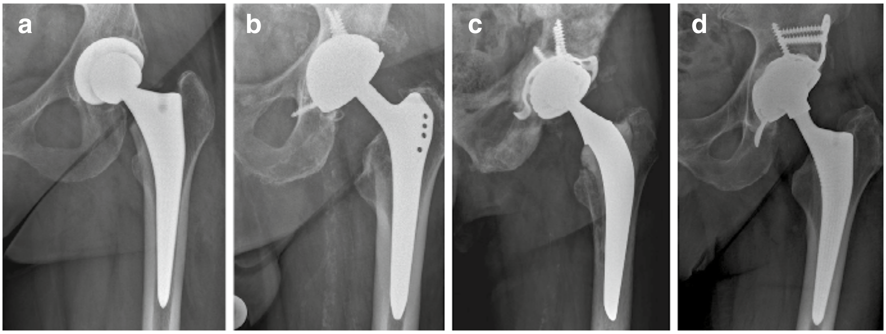
(ii) Inferior hook meant to be placed under the teardrop as an additional feature to the Müller ring (helps identify the anatomical center of rotation)

(iii) With structural bone allograft

The Burch-Schneider ring:

(i) Two flanges: superior (resists ilium) and inferior (slotted into the ischium for stability and to avoid sciatic nerve injury)

(ii) In case of unsupportive columns



Primary cup (**a**), revision cup fixed with screws (**b**), Ganz ring (**c**), Burch-Schneider ring (**d**)

Component Orientation:

- Acetabular safe zone: anteversion 15 ± 10° and inclination 40 ± 10°
  - The dislocation rate is not necessarily higher outside this safe zone. The position of the cup should be adapted to the individual anatomical characteristics of each patient (lumbo-pelvic balance)
  - Specific considerations for soft tissue handling and implant positioning are required depending on the approach
  - Avoid anterior cup overhang as this may cause iliopsoas impingement, but do not over-antevert the cup to avoid potential anterior instability
- Stem: Significant varus or valgus placement (of standard straight stems) should be avoided as this will alter load transmission and may increase the risk of loosening

Preoperative Planning:

- Surgical goal is anatomical reconstruction of global offset, leg length and torsion. The center of rotation is shifted closer to the body's center of mass (medial position of the cup at the ilioischial line), increasing the hip abductor muscle lever arm if the global offset is maintained (less exertion)

Tribology (Bearing Surfaces):

- Metal (CoCr) on polyethylene
  - Advantage: cheap, longest track record of bearing surfaces with good results
  - Disadvantage: Osteolysis (hard on soft, see *Polyethylene wear*)
- Ceramic on cross-linked polyethylene
  - Standard of care
  - Advantage: result in less polyethylene wear than metal-on-polyethylene bearings (ceramic particles are bio-inert and thus better tolerated)
  - Disadvantage: Special attention in case of revision

When changing the head, a metal sleeve must be applied to the roughened stem neck to protect the head and prevent ceramic fracture

After a ceramic head fracture, do not change to a CoCr head (or to a double mobility cup system), as the smallest ceramic particles remaining in the situs will cause severe wear of the softer CoCr head with subsequent pseudotumor formation and systemic exposure to metal ions. In this situation, a ceramic-on-ceramic bearing is ideal

- Ceramic on ceramic
  - Advantage: best wear properties of all bearing surfaces (hard on hard)
  - Disadvantage: squeaking (hard-on-hard combination can cause noise when walking)
- Metal on metal
  - No longer used
  - Advantage: better wear properties than metal-on-polyethylene (hard on hard versus hard on soft, see *Polyethylene wear)*
  - Disadvantages:

    Metal ions are detectable in serum and urine, correlating with wear

    Over time, a pseudotumor may form (also known as an aseptic lymphocyte-dominant vasculitis-associated lesion (ALVAL), a mass-forming tissue caused by type IV delayed-type hypersensitivity to metal debris)

    Serum metal ion concentration correlates poorly with the presence of a pseudotumor (no cut-off) and should not be used as a standard for follow-up in these cases

    40% of patients with pseudotumor are asymptomatic. Damage to the abductor muscles is feared. However, there is no evidence of an increased risk of malignancy
  - Management of patients with metallosis:

    Low risk group (asymptomatic, small diameter femoral head (<36 mm), optimal cup orientation, no osteolysis/loosening), normal MRI (with MARS = Metal Artifact Reduction Sequence) ➔ annual radiographic follow-up. In case of doubt or clinical or radiological suspicion, a MARS/DIXON MRI should be performed to look for pseudotumors

    Moderate risk group (mild local hip symptoms but no systemic symptoms, no abductor weakness or gait change, large diameter head (>36 mm), modular neck device, optimal cup orientation without signs of loosening/osteolysis), presence of abnormal tissue reactions on MRI) ➔ consider revision surgery or close follow-up (in 6 months)

    High-risk group (symptomatic, abductor weakness and gait change, implant osteolysis/loosening, MRI with solid lesions involving surrounding muscles/bone) ➔ revision surgery (change to ceramic to polyethylene)
  - Trunionosis: release of metal ions from corrosion at the femoral head-neck interface (regardless of bearing combination). Risk factor: modular designs, large CoCr heads, taper design

- Polyethylene wear
  - Pathophysiology: debris formation due to interaction of two surfaces of different (harder/softer) material (=abrasive and adhesive wear) ➔ macrophage-activated (RANKL-mediated) bone resorption (acetabular/femoral osteolysis) ➔ prosthesis micromotion ➔ debris dissemination around the implants, which promotes loosening and pain under load
  - Cross-linked polyethylene:
    Linear wear rates >0.1 mm/year have been associated with osteolysis and subsequent component loosening
    Non-crosslinked polyethylene wear rate is 0.1–0.2 mm/year
    Highly cross-linked polyethylene: wear rate 0.02–0.05 mm/year. Produces smaller wear debris and is more wear resistant: In highly cross-linked polyethylene, a 32 mm femoral head shows no increased wear compared to a 28 mm femoral head, allowing the use of larger diameter femoral heads that reduce the risk of joint dislocation

Complications:

- Most common early complication: Dislocation
  - Factors influencing stability:
    Femoral head size: a larger head (>28 mm) allows for greater range of motion, less component impingement and increases jump distance. Current practice: 32–36 mm ➔ Optimal balance between stability and wear; no relevant effect on wear parameters as compared to 28 mm for highly cross-linked polyethylene
    Loss of soft tissue tension: abductor damage, gluteal nerve lesion, decreased offset, decreased leg length
    Component malorientation: too high or too low combined anteversion of the cup and stem. Optimal combined anteversion is approximately 30° to 50°
    The orientation of the cup should be individually adapted to each patient according to their spino-pelvic balance
    Patient factors: Neurological deficits (e.g., Parkinson's disease, cerebral palsy), muscular deficits (e.g. insufficiency of hip abductors)
- Most serious complication: implant-associated infection
- Periprosthetic fractures are becoming more common. Intraoperative fractures occur in patients with brittle bone and press-fit implants. The anterior approach has been described as a higher risk, especially for greater trochanteric fractures. These should be recognized during surgery and treated immediately with fixation if necessary. Later fractures occur as a result of trauma. The stability or instability of the implants must be determined, as this changes the type of revision between internal fixation or internal fixation with revision of the implant (Vancouver classification)

- Reasons for revision of total hip arthroplasty according to SIRIS report 2021: stem loosening (21.3%), infection (19.8%), cup loosening (17.2%), periprosthetic fracture (16.7%), dislocation (11.2%), wear (6.2%), metallosis (4.9%)

"Periarticular" Pain

Causes:

- Overuse
- Reactive (due to intra-articular pathology)
- Pseudoradicular from spine/sacroiliac joint

Common structures involved in periarticular hip pain:

- Trochanteric bursa → trochanteric bursitis
- Abductors → abductor tendinopathy/(partial) tears
- Iliotibial tract → external snapping hip
- Adductors → adductor tendinosis
- Iliopsoas → internal snapping hip

Greater Trochanteric Pain Syndrome (GTPS)

- General term for a number of different conditions, often occurring in combination. It is not a precise diagnosis and should be avoided
- Painful structure not always well identified (see above*: involved structures*)
- Common symptom: Lateral hip pain, possibly radiating down the lateral thigh to the knee
- Much more common in women

Trochanteric Bursitis

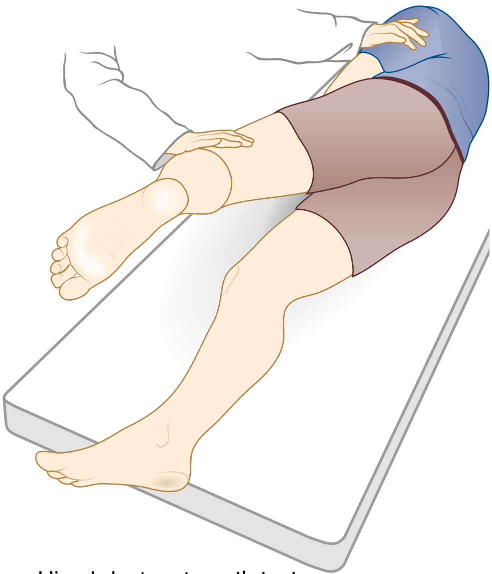
- Mostly a symptom of another pathology, rarely the actual cause of the complaints
  - An abductor lesion should always be suspected until proven otherwise
- Pain with local pressure (lateral position), prolonged standing or walking, adduction
- Therapy (same as GTPS):
  - Local and systemic anti-inflammatory medication, stretching and strengthening exercises for the hip abductors
  - Corticosteroid injection of the trochanteric bursa

Hip Abductor Tendon Lesions

- Degenerative or iatrogenic (post-operative) hip abductor tendon lesion (gluteus minimus and medius)
  - Without hip arthroplasty: 25% of patients undergoing total hip arthroplasty for severe hip osteoarthritis have a partial abductor tear
  - With hip arthroplasty: Excessive wear/osteolysis/metallosis (see *Tribology*) leads to inflammation, tissue fatigue, tendon injury, and atrophy
  - During hip arthroplasty: approach-related (e.g., transgluteal)
- Complaints similar to trochanteric bursitis, loss of strength in hip abduction, positive Trendelenburg sign, frequently associated with coxa saltans externa
  - Straight hip abduction tests several abductor muscles. To specifically test the gluteus medius, the hip should be internally rotated. Abduction against resistance is then assessed
- Natural history of partial tears: risk of clinically relevant progression or fatty infiltration of the muscle
- Therapy:
  - Conservative: same as above
  - Operative:

    Good muscle quality on MRI → Endoscopic vs. open tendon reattachment

    Poor muscle quality → gluteus maximus tendon transfer. Improves pain levels, but effect on hip abductor strength and patient-reported functional outcome scores is limited. Remains the preferred salvage option in the absence of better alternatives



Hip abductor strength test

Coxa Saltans Externa

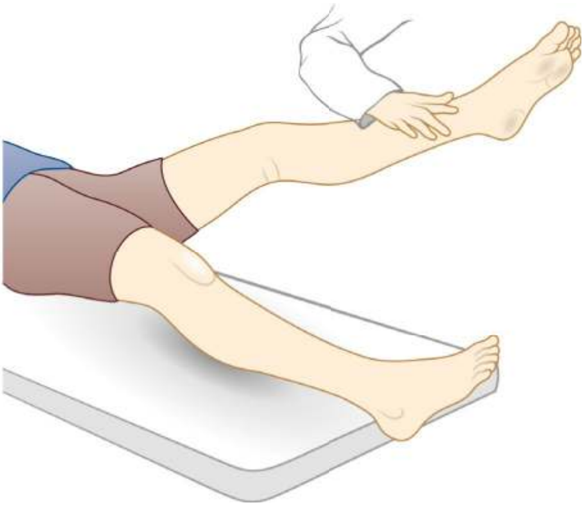
- Snapping hip caused by sliding of the iliotibial tract over the greater trochanter
- More common in athletes (e.g., road cyclists) or young hyperlax patients, may be compensatory in cases of abductor weakness
- Snapping may be painful or painless, aggravated by activity, trochanteric bursitis may develop
- Clinical examination: Tractus stress test (cycling movements in lateral position)

Tractus stress test

- Therapy:
  - Conservative: Activity modification, physical therapy, NSAIDs, physical therapy (stretching and strengthening exercises for the hip abductors, corticosteroid injection of the trochanteric bursa
  - Operative: rarely indicated (tract release)

Coxa Saltans Interna

- Iliopsoas tendon snapping
- Snapping is typically reproduced by extending (and internally rotating) the hip from a flexed and externally rotated position
- Clinical presentation: snapping (painful or painless) in or around the hip, groin pain, painful resisted single leg raise
- Therapy:
  - Conservative: Iliopsoas stretching exercises, diagnostic-therapeutic injection of the iliopectineal bursa
  - Surgical: after failure of conservative treatment
    - Iliopsoas tendon release (arthroscopic tenotomy is the technique of choice, as open techniques have a significantly higher complication rate)
    - With significant anterior cup overhang (more than 5 mm): Acetabular component revision



Provocative test for the iliopsoas

Traumatology

Femoral Neck Fractures

- Intracapsular fractures have a higher risk of nonunion and osteonecrosis than other hip fractures
- Classification:
  - Garden I to IV: I incomplete (valgus-impacted), II complete non-displaced, III varus displacement, or IV fully displaced/dislocated
  - Pauwels: fracture angle (I < 30°, II 30–50°, III >50°)
    The steeper the angle, the more shearing forces are generated, and the less likely the fracture will heal and varus dislocation will occur
- Therapy:
  - Young patients with good bone quality:
    Closed or open reduction and internal fixation with dynamic hip screw, 2 or 3 cannulated screws (reversed triangle construct), Femoral Neck System
    (i) Cannulated screws are biomechanically inferior (in Pauwels III and II, not I), but this has not been confirmed in clinical studies. A medial buttress plate in Pauwels II and III fractures increases the stability
    (ii) Total hip arthroplasty in patients over 50 years of age with a high risk of secondary necrosis (Garden III and IV)

Intraosseous healing only due to lack of periosteal and extraosseous blood supply intraosseous healing only ➔ No callus formation. Achieving and maintaining anatomic reduction is essential

- Elderly patients:

  Valgus impacted/nondisplaced: conservative management versus cannulated screw fixation

  Displaced: total hip arthroplasty (THA) vs. hemiarthroplasty (HA)

  (i) Dislocation rate: THA > HA, but similar within first 2 years after surgery ➔ preference of HA due to dislocation is controversial. Dual mobility or larger heads reduce dislocation risk in THA

  (ii) THA is associated with longer operative times and higher blood loss, but no difference in medical complications (overall, in the first year, or within 30 days). Even lower mortality rates are reported after THA which is mainly attributed to patient selection for THA

  (iii) Function: THA > HA (clinical scores and mobility at 5 years)

  (iv) Acetabular erosion is one main reason for revision (80% of indications for revision in HA). Revision rate significantly higher with HA (especially >4 years), so patient selection is important (HA for demented or immobilized patients)

Pertrochanteric Fractures

- Mortality rate is higher than in femoral neck fractures with 20–30% within the first year (patients are mostly >85 years old and ASA 3/4)
- Therapy:
  - Operative:

    Stable fracture patterns: dynamic hip screw or short cephalomedullary nail (CMN)

    (i) No differences in functional outcome, non-union, revision rate or infection. Quality of reduction is essential to achieve a stable construct

    (ii) CMN biomechanically more stable than extramedullary implants

    (iii) CMN is associated with mild hip abductor muscle strength deficit

    Unstable fracture patterns (multifragmentary, fractured or incompetent (<20.5 mm) lateral wall): CMN

    Reverse oblique fractures: long CMN

    (i) Lag screw in same fragment as distal locking screw for short CMN ➔ less reliable fixation

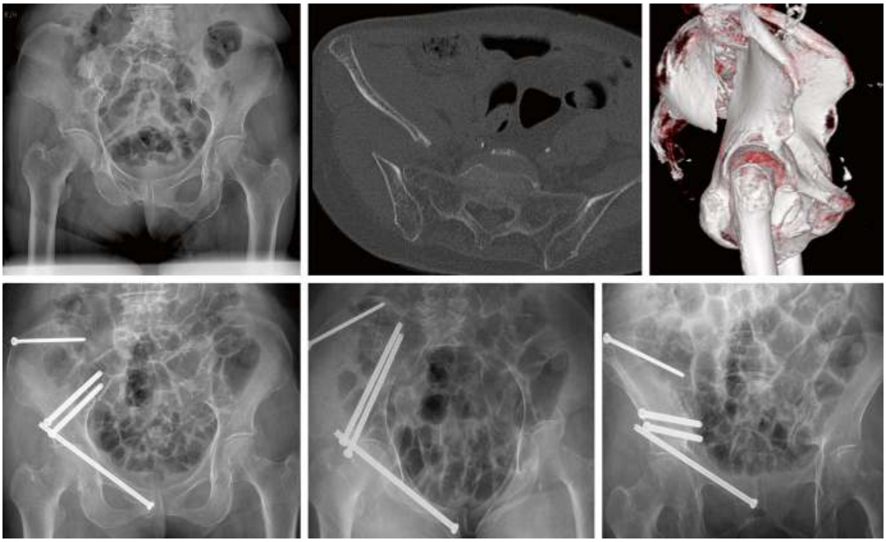
    Subtrochanteric extension: long CMN

    Surgical technique:

    (i) Tip-apex distance and anatomic reduction remain the most important predictors of construct failure

Pelvis Fragility Fractures

- 60–80% of patients with anterior pubic rami fractures have associated posterior injuries found on CT
  - A significant proportion of patients who complain of persistent pain or have difficulty with mobilization are showing injury progression to a more unstable fracture pattern
  - Typically, the posterior involvement corresponds to a vertically oriented sacral fracture through the ala that does not involve the neuroforamina, but may be joined by a horizontal fracture to create an H-type pattern
    - A so-called “alar void” located in the sacral ala has been found as a zone of decreased bone mass compared to the S1 vertebral body ➔ predisposed fracture site
    - Technically, this corresponds to a spinopelvic dissociation, but the clinical presentation and degree of resulting instability are different from those seen in high-energy trauma
- Medical management:
  - Pain control: Even type I fractures, which are commonly managed non-operatively, carry a significant morbidity, with an increase in the requirements for long-term assistance, and a decrease in quality of life measures
  - Haemorrhage: less than 2% in contrast to high-energy fractures
  - Osteoporosis: secondary fracture prevention must be a key concern in this patient population. Over 40% develop a further fragility fracture within 2 years
- Therapy:
  - Conservative:
    - *Nondisplaced* pelvic fragility fractures (FFP type 1/2, AO type A/B)
  - Operative:
    - Failed conservative treatment (mobilization not possible, pain level)
    - Indicated for significantly *displaced* fractures (FFP type 3/4, AO type C)
    - Surgical indication less clear for fractures with *subtle displacement* (minimally/non-displaced FFP type 3, nondisplaced AO type C)
    - Open procedures result in significantly longer hospital stays, increased surgical complications and higher all-cause mortality compared to percutaneous procedures
    - Construct of choice: combined anterior-posterior fixation biomechanically superior. However, some believe that for many injuries, the anterior ring rarely requires fixation after stable fixation of the posterior pelvic ring



The CT scan shows a significantly displaced crescent fracture of the ilium and an oblique superior ramus fracture. The fracture was reduced minimally invasively/indirect and fixed with percutaneous screw fixation (in the LC2 and anterior column corridors)

Acetabular Fractures

- Judet and Letournel classification system:
  - 5 elementary and 5 complex fracture patterns
  - Fracture pattern mainly determined by force vector, position of the femoral head at time of injury, and bone quality

    Younger patients: posterior wall, transverse ±posterior wall, T-type

    Elderly: anterior column ±posterior hemitransverse, both columns
- Poor and fair results in 20% of elementary (simple) fractures in the medium term, 28% in complex fractures
  - Total hip replacement required in 20% after 2–20 years
  - Prognostic factors: fracture pattern (complex fractures have a worse prognosis than simple fractures, except for posterior wall fractures), superomedial acetabular dome impaction, degree of fracture dislocation, femoral head injury, increasing patient age, delayed surgical treatment (>3 weeks), quality of reduction
- Therapy:

- Conservative: suitable for non-displaced or minimally displaced fractures with stable joints

  Examination under anesthesia to assess hip joint stability in case of small (<40%) and non-displaced (<2 mm) posterior wall fractures
- Operative: Indicated for displaced fractures

  Approaches include posterior, ilioinguinal/anterior intrapelvic approach (modified Stoppa plus lateral window)/pararectus, surgical hip dislocation, and sometimes combined approaches for complex fractures
  - Anterior column fractures, both column fractures, anterior column posterior hemitransverse, T-type, and certain transverse fractures (depending on which portion is primarily displaced) are managed from anterior
  - Posterior wall, posterior column, T-type and transverse fractures are managed from posterior or with a surgical hip dislocation
  - Percutaneous fixation of acetabular fractures is a powerful tool in the treatment of minimally displaced fractures. The addition of minimally invasive and indirect reduction can help with more displaced fractures. Benefits: Reduced operative time, blood loss, heterotopic ossification and earlier patient mobilization. However, it requires significant experience with intraoperative pelvic fluoroscopy
  - Total hip replacement with internal fixation (plates, cerclage) may be indicated in older patients with complex fractures and brittle bone

Displaced transverse/posterior wall fracture in a young patient treated with a posterior approach

Both column fracture with comminuted quadrilateral wall. The fracture pattern is similar, but by definition not an anterior column/posterior hemitransverse fracture because there is no articular surface attached to the ilium. Treated with an anterior intrapelvic approach

Knee

7

Andreas Flury, Leo Kronberger, Bernhard Christen,
Hermes Miozzari, and Sandro Fucentese

Anatomy

Osseous Anatomy

- Joints: patellofemoral, femorotibial, proximal tibiofibular
- Femorotibial: not a simple hinge joint, but has a complex motion with a "screw-home" mechanism
  - Six degrees of freedom include 3 rotational (flexion/extension; internal/external rotation; varus/valgus) and 3 translational (anterior and posterior glide, medial and lateral shift, compression and distraction) motions
  - "Screw-home" mechanism: The tibia rotates outward in terminal extension to the femur (or, with the leg upright, the femur rotates inward). The rotation (and anterior translation) is due to anatomic conditions such as the incongruence of the lateral tibiofemoral joint (convex tibial plateau and convex femoral condyle). This final rotation in full extension maximizes tension on the ACL, iliotibial tract, and collateral ligaments, increasing stability. The popliteus muscle lifts this final rotation as it initiates knee flexion. By internally

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rotating the tibia (or externally rotating the femur with the leg upright), the collateral ligaments are relaxed and flexion is allowed
- Patellofemoral:
  - The patella is stabilized in the sulcus from 30–100° of knee flexion
  - ➔ congruence of patella and trochlea most important
  - From 0–30° of knee flexion, active (mainly M. vastus medialis obliquus) and passive (MPFL and medial patellar retinaculum) factors stabilize the patella
    The stabilizing effect of the M. vastus medialis obliquus is almost negligible. The muscle vector only becomes relevant after 60° of knee flexion. It reaches its maximum at > 90°, but then the patella is already inside the sulcus
    The lateral patellar retinaculum (LPFL) is less commonly injured than the medial patellar retinaculum, but it is often disrupted during surgery to correct abnormal lateral patellar tracking or dislocation

Soft-Tissue

Cruciate Ligaments

- Anterior cruciate ligament (ACL):
  - Origin: medial aspect of lateral femoral condyle, with intercondylar ridge as the anterior edge of the ACL
  - Insertion: 2 bundles named for tibial attachment
    Anteromedial bundle: tight in flexion and lax in extension (Lachman test)
    Posterolateral bundle: tight in extension, lax in flexion, responsible for rotational stability (pivot shift test)
  - Blood supply: middle geniculate artery
  - Covered by synovial sheath containing nerve fibers that provide proprioception
- Posterior cruciate ligament (PCL):
  - Origin: anterolateral aspect of medial femoral condyle
  - Insertion: posterior tibial sulcus below the articular surface
    Anterolateral bundle (constitutes 85% of the PCL) and posteromedial (tight in extension) bundle
  - Covered by a synovial sheath and surrounded by well-vascularized (middle geniculate artery) fat tissue, extending from extra- to intra-articular → *intrinsic healing potential*

Collateral Ligaments

- Medial collateral ligament (MCL):
  - Capsular and extracapsular part (dMCL and sMCL)
  - Origin: posterior and proximal to the medial femoral condyle
  - Insertion: deep and posterior to the pes anserinus (8–10 cm)
  - sMCL is the primary passive restraint to valgus rotation, and its rupture leads to valgus laxity at all knee flexion angles, whilst isolated rupture of the dMCL does not cause a clinically discernible increase in valgus laxity

sMCL is also the most important medial restraint to external tibial rotation ➔ anteromedial draw test (see Chapter "MCL injury")

- Other stabilizers of the medial knee:

  The MCL is part of the posteromedial corner along with the posterior oblique ligament (POL), the semimembranosus attachments, capsule, and oblique popliteal ligament (OPL)

  The posterior oblique ligament (POL) is located in the posterior third of the MCL and extends from the adductor tubercle to the posteromedial aspect of the tibia. It resists tibial internal rotation and is a secondary restraint to valgus stress at full knee extension.

  Note: *The hallmark of a posteromedial corner injury is anteromedial rotational instability, defined as "external rotation with anterior subluxation of the medial tibial plateau relative to the femur" as compared to isolated valgus laxity*

- Lateral collateral ligament (LCL):
  - Origin: proximal and posterior to lateral condyle and origin of popliteus
  - Insertion: anterolateral fibular head (most anterior structure of the fibula: LCL ➔ popliteofibular ligament ➔ biceps femoris)
  - Primary passive restraint to varus stress in 5° and 30° of knee flexion

    Resists varus in full extension along with ACL and PCL

    Located behind the axis of rotation of the knee: tight in extension, lax in flexion

    Secondary restraint to posterolateral rotation and part of the posterolateral corner (PLC) along with the popliteofibular ligament and the popliteus tendon. Additional (static and dynamic) structures of the PLC consist of the meniscofemoral and meniscotibial ligaments, the fabellofibular ligament (variable), the arcuate ligament (variable), the biceps femoris, the iliotibial band, and the lateral head of the gastrocnemius muscle

Menisci

- The menisci are composed of fibrocartilage (type I collagen) and are free of synovial membrane
- Function:
  - Increase the contact area between the femur and tibia to optimize force transmission (shock absorption): transmits 50% weight bearing load in extension and 85% in flexion
  - During knee flexion, the menisci are displaced dorsally (*medial: M. semimembranosus; lateral: M. popliteus*). However, there is little movement before 60° of flexion, which is important for postoperative care
- Medial meniscus:
  - C-shaped, covering 50% of the plateau
  - Less mobile due to connection to collateral ligament (➔*"unhappy triad"*)
  - Blood supply: medial inferior genicular artery (20–30%); the posterior root through the middle genicular artery

- Lateral meniscus:
  - More circular in shape and covers more of the plateau (60%)
  - More mobile due to less rigid peripheral fixation (only with thin capsule)
  - Blood supply: lateral inferior genicular artery (20%) and approximately 75% by diffusion

Ligament Injuries

ACL Rupture

Etiology

Four common accident mechanisms, with 40% occurring without external force (non-contact: jumping or landing):

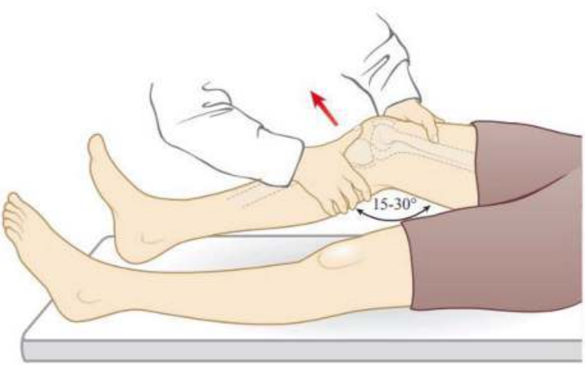
- Valgus and internal rotation (*pivot shift*)
- Anterior translation of the tibia in relation to the femur
  - Axial compression (landing) with heel contact on the ground, slight knee flexion and a large distance between the center of mass (body) and the ground contact point
- Hyperflexion with external rotation of the tibia
- Hyperextension

Clinical Presentation

Acute knee pain, swelling, instability (giving way), "pop" sensation during injury

Diagnosis

- Clinical examination
  - Hemarthrosis / effusion
  - Restricted ROM
  - Lachman test (longer travel with soft or missing stop; highest sensitivity)



Lachman´s test

– Anterior drawer test
– Pivot shift test

In an ACL tear, the tibia will usually sublux anterolaterally on the femur. A flexion and valgus force is applied. As the knee is flexed, the iliotibial band becomes a flexor of the knee (falls behind the axis of rotation of the knee) and will reduce any subluxation

- Assess for meniscal, collateral ligament injuries
- Assess neurovascular status: popliteal artery injury is mainly associated with high-energy injuries, including knee dislocation and complex tibial plateau fractures
  – Due to intimal rupture, the artery may become thrombosed, requiring urgent thrombectomy
- Radiographic examination
  – X-ray in three planes to rule out fractures

    Tibial spine avulsion = bony equivalent of ACL rupture. Fracture is usually confined to the intercondylar eminence (non-articular portion), but may extend to the tibial plateau. Most common, but not exceptional, in children. However, the incidence of tibial spine fractures in alpine skiers is similar among all age groups

Segond fragment = tibial avulsion fracture of the anterolateral ligament/complex; visible in up to 9% of ACL ruptures and pathognomonic for ACL rupture

- MRI: assess ACL, menisci, cartilage

  Acute concomitant meniscal lesions are more likely to involve the lateral > medial meniscus, whereas in chronic ACL unstable knees, medial meniscal injuries are more common than lateral injuries

  Bone marrow edema in the lateral femoral condyle and in the posterolateral part of the tibia is found in >80% and is due to the accident mechanism (pivot shift). The "deep femoral notch sign" (=impression in the femoral condyle) is the maximum variant of this

  The "empty notch sign" describes a free lateral condyle wall, as the ACL is "missing" in the coronal view
- CT to assess tibia/femur fractures (e.g., tibial spine fractures) and for preoperative planning purposes

  CT with contrast to rule out arterial (intimal) lesions in case of knee dislocation

Therapy

Table: Key factors influencing decision between non-operative and operative treatment for ACL injuries

Decision factor	Operative treatment	Non-operative treatment
Activity level	High-demand sports participation (high-risk pivoting sports)	Low-demand lifestyle
Age	Typically younger patients	Age may not be a decisive factor
Functional stability	Significant instability during activities after conservative treatment	Minimal instability or able to compensate
Associated injuries	Concurrent ligament or meniscal injuries	Isolated ACL tear or minimal associated injury, advanced osteoarthritis
Expected outcome	Improved knee stability and function post-surgery	Satisfactory functional outcome anticipated

Conservative Immediate cooling, elevation, NSAIDs. Early rehabilitation program for stability, range of motion and strength. Weight bearing as tolerated. Bracing for support, but no relevant healing potential with conservative treatment

Operative Arthroscopically-assisted ACL reconstruction

- Timing of ACL reconstruction surgery may not influence functional outcomes and postoperative complications, according to recent data

- Technique:
  - Femoral tunnel placement:

    Can be drilled trans-tibially or independently of the tibia, although trans-tibial creates a more vertical graft (associated with rotational laxity—no difference in clinical outcomes has been shown)

    Start near the intercondylar line and the cartilaginous margin of the lateral femoral condyle. Leave a 1-2 mm rim of bone between the tunnel and posterior cortex to prevent posterior wall blowout (and flex the knee >70°).

    To avoid anterior displacement of the femoral bone tunnel, use the posterior horn of the lateral meniscus as an anatomical reference (drill directly above)

    The tunnel should be placed at 10 o'clock for the right knee and at 2 o'clock for the left knee

    Optimal bone tunnel positions in ACL reconstruction are critical for effective restoration of knee stability and function. The placement of the bone tunnels determines the orientation and tension of the graft, which influences postoperative outcomes
  - Tibial tunnel placement:

    Anatomical reference: Posterior border of the anterior horn of the lateral meniscus, approximately 10 mm anterior to the anterior border of the femoral PCL insertion
  - Double bundle reconstruction: mimics natural ACL anatomy with separate bundles for better rotational stability than single bundle reconstruction. Studies have shown no difference in clinical outcomes (subjective stability). Double-bundle reconstructions are more expensive, time-consuming and technically demanding
- ACL ruptures in children:
  - Skeletal maturity on preoperative carpal radiographs guides surgical technique in terms of placement of the tunnel: extraphyseal, transphyseal, or partially transphyseal
- Graft choice (target: 7 cm length, 8 mm thickness):
  - **Hamstring autograft:**

    Advantages: Thick and strong (maximum load up to 4000 N = 2–3 times stronger than the native ACL); reduced donor site morbidity

    Disadvantages: Harvesting may damage the infrapatellar ramus of the saphenous nerve; possible persistent hamstring weakness, which can become symptomatic in sprinters and footballers; slower incorporation of the tendon due to a tendon-bone interface (→ drill channel widening) ➔ higher risk of revision surgery according to registry studies
  - **Quadriceps tendon autograft:**

    Advantages: similar results to other grafts; maximum load up to 2200 N; less donor site morbidity compared to the patellar tendon autograft

    Disadvantages: risk of extensor mechanism dysfunction; graft size may vary, potentially limiting its suitability for patients requiring larger grafts;

incorporation similar to hamstring autograft due to its purely tendinous nature (tendon-bone interface)

- **Patellar tendon autograft:**
  Advantages: Strong initial fixation and faster incorporation due to its bone-to-bone attachment (approximately 6 weeks versus 3 months for pure tendon grafts); significantly less tunnel expansion) → not an option for children with open growth plates; maximum load up to 2600 N
  Disadvantages: donor site morbidity (high risk of anterior knee pain, crepitus and patella infera)
- **Allografts:**
  Advantages: No donor site morbidity; can provide larger grafts
  Disadvantages: cost, slower incorporation, higher risk of re-rupture (4 times higher in patients <20 years old)
- **Synthetic grafts:**
  Advantages: No donor site morbidity; consistent biomechanical properties (reducing variability compared to biological grafts)
  Disadvantages: The long-term clinical outcomes of synthetic grafts are still being evaluated, with limited evidence compared to biological grafts. Synthetic grafts may have higher rates of failure particularly in high performance athletes

- Re-rupture risk:
  - Increased risk in patients aged <20 years, graft thickness < 8 mm, female sex (neuromuscular and hormonal reasons)
  - Associated bony conditions that may be worthy of correction in a revision case: Tibial slope (if >12°) and excessive leg axis malalignment (varus or valgus malalignment increases stress on the ACL)
  - Consider simultaneous ALL reconstruction in professional or competitive athletes with pivoting sports, (preoperative) high-grade (2–3) pivot shift test, general hyperlaxity, and in revision cases

PCL Rupture

Etiology

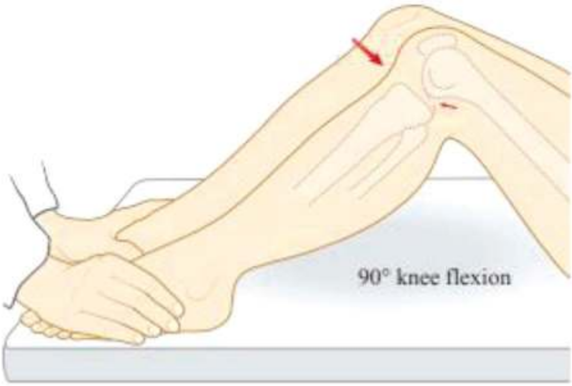
Direct blow to proximal tibia with a bent knee (dashboard injury) or hyperextension injury.

Clinical Presentation

Instability on stairs (due to maximum instability at 90° of knee flexion), but in isolated PCL injuries, instability is often subtle or asymptomatic. Chronic PCL deficiency leads to increased contact pressure (and thus cartilage deterioration) in the patellofemoral and medial compartments.

Diagnosis

- Clinical examination
  - Tenderness and hematoma in the popliteal fossa (in acute cases)
  - Posterior drawer test
  - Posterior sag sign



Gravity sign

- Quadriceps active test
  Positive if there is anterior reduction of the tibia relative to the femur when a 90° flexed knee is extended (quadriceps contraction)
- Dial test (see section "LCL and posterolateral corner injury")
- Assess neurovascular status (CAVE knee joint dislocation)

- Radiographic examination
  - X-ray in three planes to rule out fractures
  - Stress views: assesses the difference between posterior and anterior drawer tests. A translation of >8 mm indicates insufficiency, >12 mm a combined injury. A side-to-side difference (to the intact contralateral side) of 5 mm indicates rupture
  - MRI: assess PCL, PLC, menisci, cartilage

Therapy

Conservative Limited weight bearing for 6 weeks using a lower leg supporting brace for day and night. No active flexion

Operative

- Indication:
- Combined ligamentous injuries
  - Conservative treatment of the PCL with PTS or dynamic PCL brace may be the first-line treatment in selected cases (aiming for intrinsic healing)
- Dislocated bony PCL avulsion
- Symptomatic chronic isolated injuries
  - Not in advanced osteoarthritis
- Technique:
  - ORIF for bony avulsions
  - Arthroscopically assisted reconstruction
    - Tibial inlay or transtibial methods, single vs. double bundle, autograft vs. allograft: no outcome studies clearly support one reconstruction technique over the other. In general, a 10–11 cm long and 7 mm thick graft is required
    - If the tibia is fixed in a posterior subluxed position due to a chronic PCL lesion, it should be released with 6–8 weeks of orthotic treatment prior to surgical PCL reconstruction

MCL and Posteromedial Corner Injury

Etiology

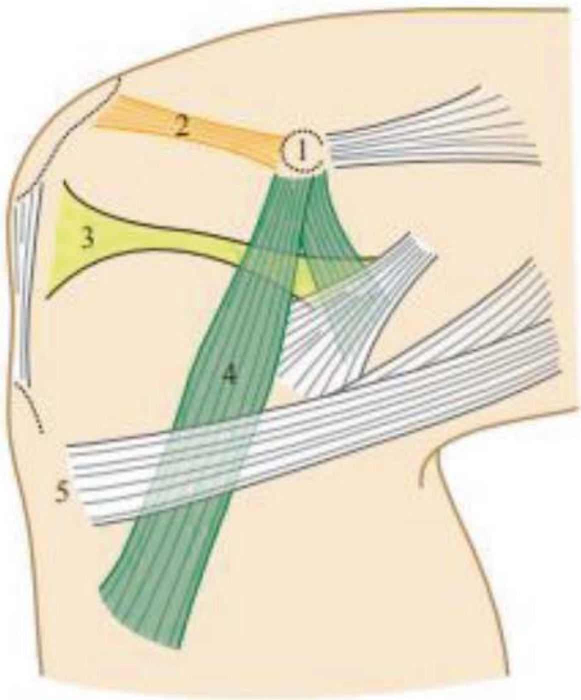
Valgus stress, usually with the knee slightly flexed and externally rotated (*contact* > *non-contact*).

Clinical Presentation

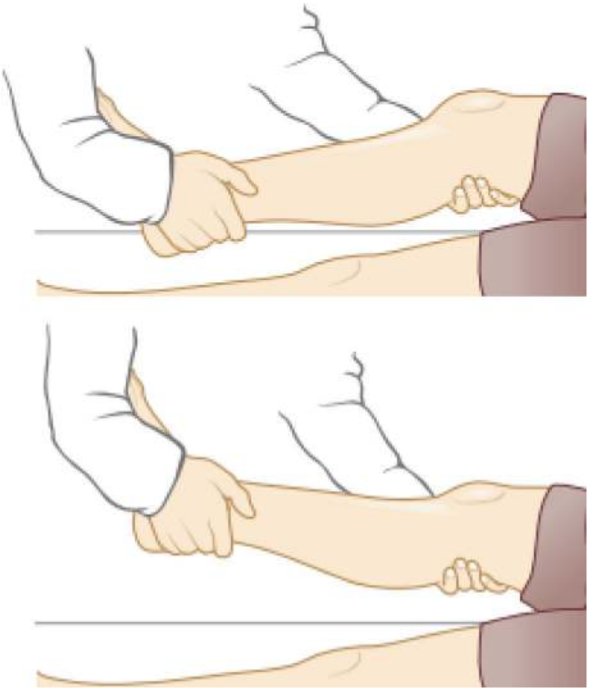
"Pop" at time of injury, swelling, medial knee pain.

Diagnosis

- Clinical examination
  - Tenderness along the medial aspect of the knee
    - Femoral attachment (1)
    - Medial patellofemoral ligament (2)
    - Retinaculum (3)
    - Distal MCL insertion (4)
    - Pes anserinus (5)



- Valgus stress test at 0° and 30° degrees of knee flexion
  Grade I: Tenderness at femoral attachment
  Grade II: Slight increase in joint laxity in flexion, stable in extension
  Grade III: Complete tear with increased joint laxity in 30° flexion and extension
  Pronounced valgus laxity, with the knee in extension, indicates a combined sMCL and POL injury (equivalent to a posteromedial corner injury), and possibly an ACL injury
- Anteromedial rotatory instability:
  Tested by applying a valgus load at 30 degrees of knee flexion *with simultaneous external rotation of the foot*
  Positive test: Anterior subluxation of the anteromedial tibial plateau on the femoral condyle
  Correlates with a combined MCL and POL injury (equivalent to a posteromedial corner)
- Anteromedial drawer test:
  Tested with the knee at 90° of flexion with external rotation of the foot (similar to the posterolateral drawer test but with an anteromedial rotatory force): Look for anteromedial subluxation of the tibial plateau
  May indicate combined injury to the MCL and ACL



Valgus stress test at 0° and 30° degrees of knee flexion

- Radiographic examination
  - X-ray to rule out fractures
    - Usually normal
    - Calcification of the insertion site in chronic MCL tears (Stieda-Pellegrini lesion)
  - MRI: determines extent of injury and evaluates other injuries

Therapy

Conservative

Immediate → Cooling, rest, anti-inflammatory medication.

Grade I	No specific therapy: return to play in 5–7 days if pain free
Grade II	Hinged knee brace for walking with full ROM
Grade III	Immobilization at 20° flexion for 2 weeks; then 60°–20°–0° for 2 weeks. Then varus brace until pain free

- The treatment of choice for MCL and/or complete PMC injuries combined with ACL rupture is a period in a range of motion brace before delayed, isolated ACL reconstruction if medial stability is reasonably restored

Operative Indicated for tibial avulsion fractures of the MCL, Stener-like lesions (distal MCL fibres displaced superficial to the pes anserinus), chronic pain and/or instability

– Displaced femoral sMCL avulsion are also best addressed surgically (acute refixation)
- Ligament repair vs. reconstruction
  – Acute repair of grade III injuries in the setting of multi-ligament knee injuries and Stener-like lesions
  – Anatomical reconstructions with elements to reconstruct the sMCL and POL are the reconstruction of choice for chronic PMC laxity (symptomatic antero-medial rotational instability)

LCL and Posterolateral Corner Injury

Etiology

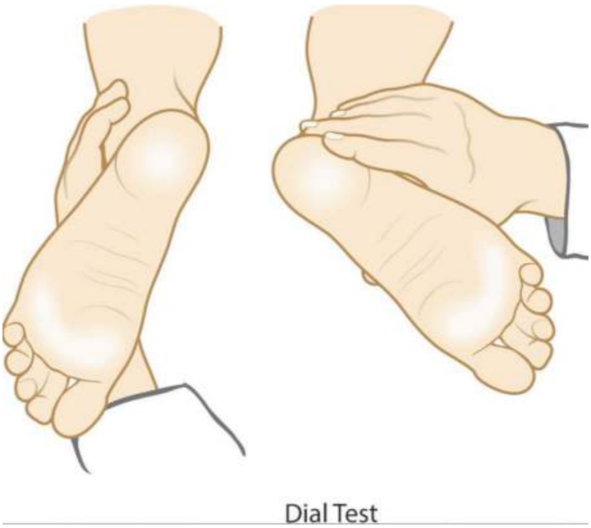
Direct blow or force to the medial side of the knee, excessive varus stress, external tibial rotation and/or hyperextension

Clinical Presentation

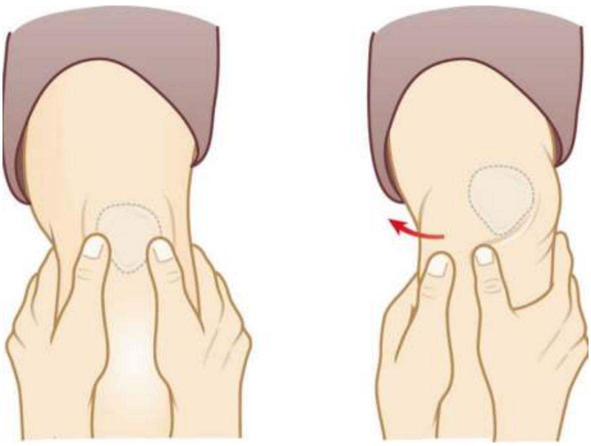
"Pop" at time of injury, swelling, lateral knee pain, instability near full knee extension

Diagnosis

- Clinical examination
  – Tenderness over LCL insertion
  – Hyperextension or lateral thrust gait
  – Varus stress test
    Varus instability at 30° flexion only—isolated LCL injury
    Varus instability at 0 and 30° flexion—combined LCL ± ACL/PCL injury
  – Dial test
    In prone position, the examiner fixes the foot and rotates it outwards, comparing the maximum external rotation of the lower leg in 30 and 90° flexion with the opposite side
    >15° of external rotation to the opposite side at 30° of knee flexion ➔ isolated injury to the posterolateral corner (PLC)
    >15° of external rotation at 30 and 90° of knee flexion ➔ combined injury of the PLC and the PCL



– Posterolateral drawer test
  In the supine position, the knee is flexed to 90° (similar to posterior drawer test) but the foot is externally rotated. In this position, a posteriorizing (and external rotation) force is applied to the proximal tibia
  The test is positive if the lateral tibial head moves posteriorly in relation to the femoral condyle



Posterolateral drawer test

- Radiographic examination:
  - X-ray: Arcuate sign (avulsion fracture of the fibular head)
  - MRI: Gold standard for assessing severity and location of LCL injury
    Findings: Most tears are fibular-sided
    Look for injury to the popliteus and biceps tendon

Therapy

Conservative

Immediate → Cooling, rest, anti-inflammatory medication

- Grades I and II LCL injuries: Valgus brace for 12 weeks (6 weeks with partial weight bearing)

Operative

- Indicated for chronic and complete mid-substance acute grade III isolated LCL injuries with persistent varus instability
- LCL/PLC reconstruction for rotational (posterolateral) instability. Three common ones are listed below:
  - *Larson* (simple): fibula-based, extra-anatomical isometric reconstruction → reconstruction of the LCL/PFL
  - *Arciero* (complex): anatomical reconstruction → additional reconstruction of the popliteus tendon (as opposed to Larson's technique)
  - *LaPrade* (complex): anatomical reconstruction with two soft tissue grafts to reconstruct the LCL/PFL and the popliteus tendon

Meniscal Tears

Etiology

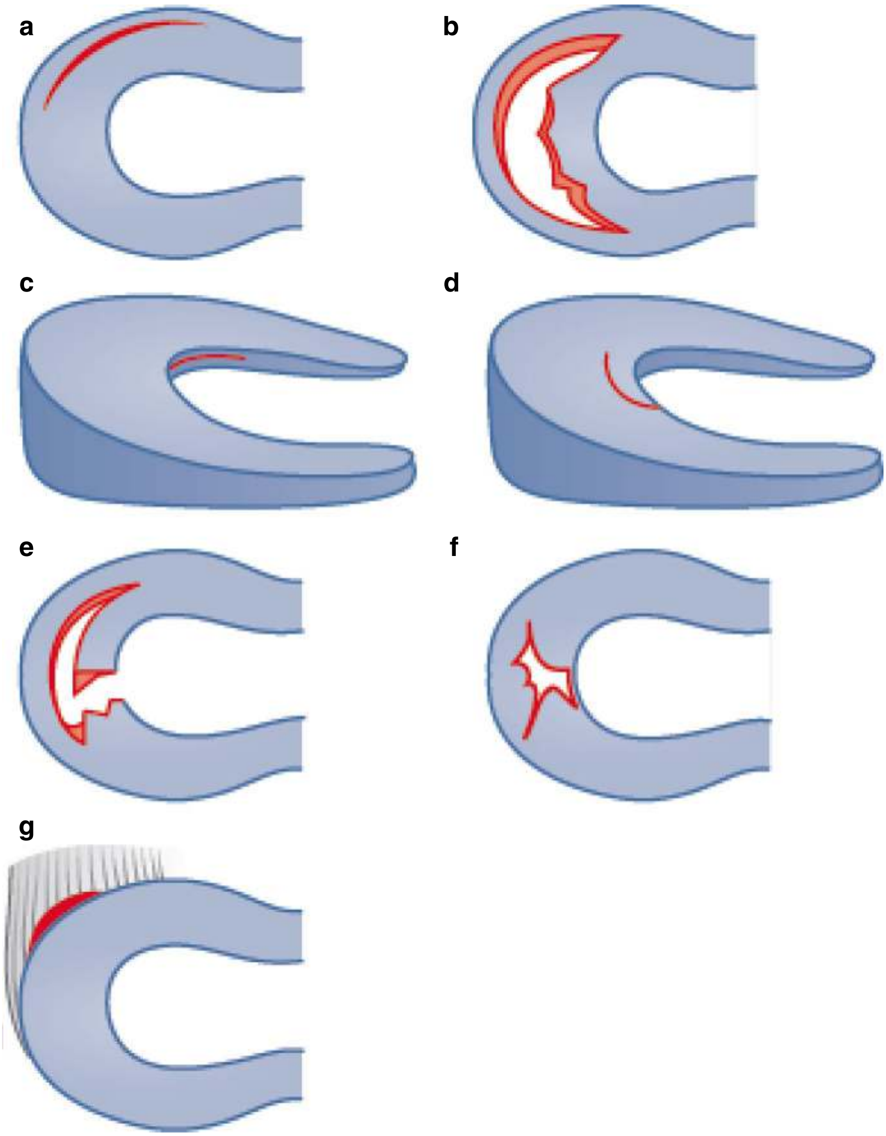
Rotational trauma under axial load, where shear forces occur in the meniscus.

Classification

- **By location:**

Peripheral (outer) third	Red zone	Vascularized (**greatest healing potential)**
Middle third	Red-white zone	Transition zone
Inner third	White zone	Avascular

- **By pattern:**
  - Longitudinal/vertical
    in younger people (traumatic)
  - Bucket handle
    vertical tear which may displace into the notch
  - Horizontal tear
    in older people (degenerative)
  - Radial
  - Oblique/flap
  - Complex degenerative
    A combination of horizontal and radial tears
  - Root
    radial tear within 1 cm of the root attachment or complete avulsion of the root
    posterior medial root in older patients (deep flexion) versus posterior lateral root in younger patients (flexion and rotation in multiligament injuries)
  - Ramp lesion
    meniscocapsular attachment of posterior horn, associated with ACL ruptures
    may compromise knee stability



Meniscal tear patterns

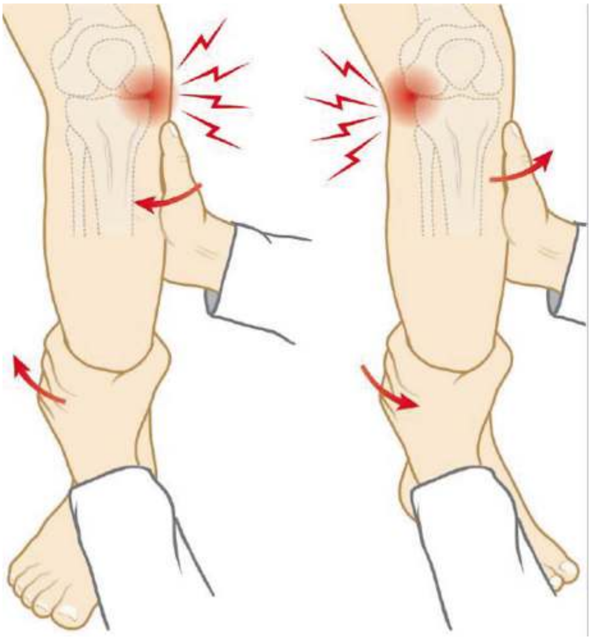
Clinical Presentation

Symptom triad: (rotational) pain, possible blockage, swelling

Diagnosis

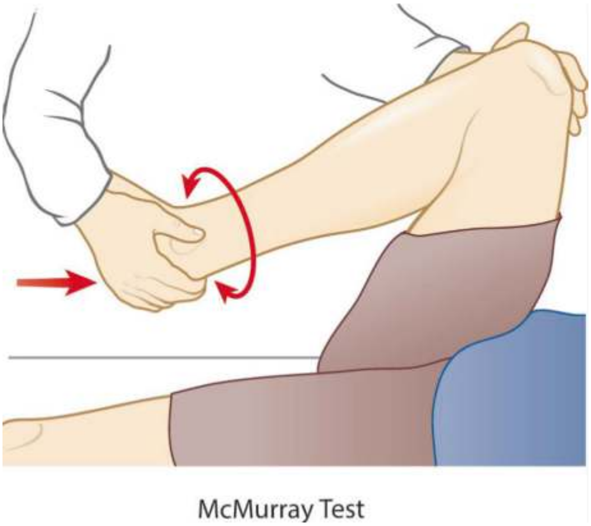
- Clinical examination
  - Joint line tenderness (most sensitive)
  - Provocative tests:

    Steinmann I:
    - Pain with forced rotation of the 90° flexed knee in supine position
    - Pain with external rotation: medial meniscus
    - Pain with internal rotation: lateral meniscus



Meniscus provocative test: Steinmann I

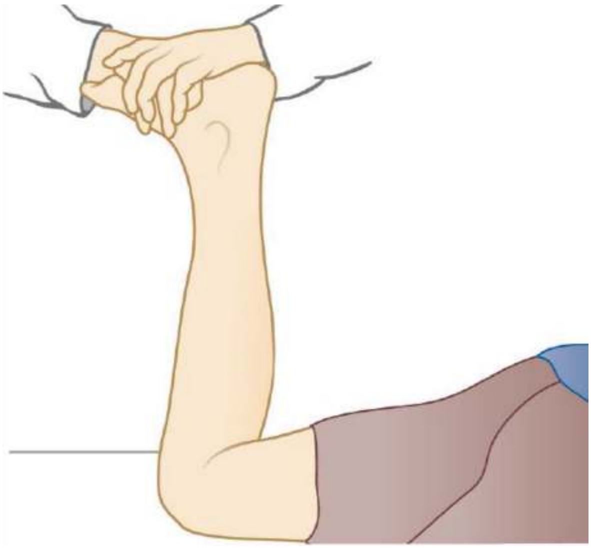
McMurray's test:
- Pain with grinding rotation of the fully flexed knee in supine position
- Pain with external rotation: posterior horn of the medial meniscus
- Pain with internal rotation: posterior horn of the lateral meniscus



Meniscus provocative test: McMurray

Apley compression

- Pain with forced rotation of the 90° flexed knee with axial pressure in prone position
- Pain with external rotation: medial meniscus
- Pain with external rotation: lateral meniscus



Meniscus provocative test: Apley Grinding

- Radiographic examination
  - MRI
    The prevalence of a meniscal tear or of meniscal destruction increases with age and is associated with knee osteoarthritis
    - Incidental (asymptomatic) findings in 61% of the population

    Definition of a degenerative tear:
    - Linear intrameniscal MRI signal (including a horizontal tear component) often extending to the inferior surface of the meniscus (on at least two image slices). A more complex tear pattern (in multiple configurations) may also be present
    - The most common location is the corpus and/or posterior horn of the medial meniscus

    "Double anterior horn" sign: displaced bucket handle tear of the lateral meniscus
    "Double PCL" sign: displaced bucket handle tear of the medial meniscus
    Discoid meniscus (3–5% of the population, mostly lateral, 25% bilateral): continuity of the meniscus on three consecutive MRI slices (with 5 mm thickness)

Therapy

Conservative Rest, immobilization, rehabilitation, possibly intra-articular infiltration

- Indication:
  - Traumatic tears: for stable, small (<10 mm) and mildly symptomatic meniscal tears
    There is no data on self-healing potential, but untreated tears at the time of ACL reconstruction have a low reoperation rate
    Lower threshold for lateral meniscus treatment due to better prognosis with surgical fixation
  - Degenerative tears: always indicated as first-line treatment
    "non-inferior" when compared to arthroscopic partial meniscectomy
    except for mechanical symptoms (locking and clicking)

Operative

- Partial meniscectomy
- A subluxed medial meniscal flap in the tibial gutter with impingement on the bone and MCL is a specific degenerative meniscal lesion that can be associated with significant mechanical symptoms and pain

- Not indicated in advanced knee osteoarthritis
- Minimize resection
  The severity of future arthritis is proportional to the amount of meniscus removed: Total meniscectomy → 70% have degenerative changes after 3 years; 100% have arthritis after 20 years

- Meniscal repair
  - The best candidate for repair is a tear with the following characteristics
    Red-red zone
    Vertical or longitudinal tear
    - Bucket handle tear

    Root tear
    - A root tear is biomechanically equivalent to a total meniscectomy and non-surgical treatment is therefore reserved for knees with severe OA
    - Knee OA 10 years after root repair: 50%, after total meniscectomy: 99%, after non-operative treatment: 95%

    As opposed to radial, horizontal and degenerative tears
    - In younger patients without osteoarthritis, meniscal repair may be considered for horizontal (degenerative) meniscus tears. Meniscal repair has biomechanical advantages over partial meniscectomy. However, outcomes are similar to partial resection, but with a higher complication rate. It is still unclear whether it stops or slows the development of osteoarthritis
  - Technique
    Anterior horn: outside-in
    Pars intermedia: inside-out or all-inside
    Posterior horn: all-inside
    - Lateral meniscus is more mobile: optional fixation in the popliteus tendon

    Root: transtibial (transosseous)
- Meniscal transplantation
  - Grafts
    Allograft: Correct sizing is essential (5–10% discrepancy accepted)
    Artificial (collagen- or polyurethane-based): good bioavailability, low toxicity, favorable production
  - Indications
    Controversial
    In young patients with near-total meniscectomy (especially lateral due to incongruent compartment morphology)
  - Contraindications
    Instability
    Leg axis deformity (malalignment) → to be addressed first
    Inflammatory arthritis
    Obesity
    Grade III–IV chondral changes

  - Outcomes
    - Slows the progression of osteoarthritis
    - Radiographic progression of osteoarthritis after 10 years, but sustained improvement in pain and function
    - Most common complication: re-tears (due to acellularity)

Patellofemoral Instability

Etiology

Acute traumatic patellar dislocation versus habitual instability.

Risk Factors

General factors

- Ligamentous laxity
- Pre-damaged structures (previous event)

Anatomical factors

- Osseous
  - Patella alta
  - Trochlear dysplasia
  - Increased tibial tuberosity trochlear groove (TTTG) distance
  - Genu valgum
  - Increased tibial and femoral torsion
    - And increased femorotibial torsion (relative rotation of the femur to the tibia), although the relevance has not yet been fully established

Clinical Presentation

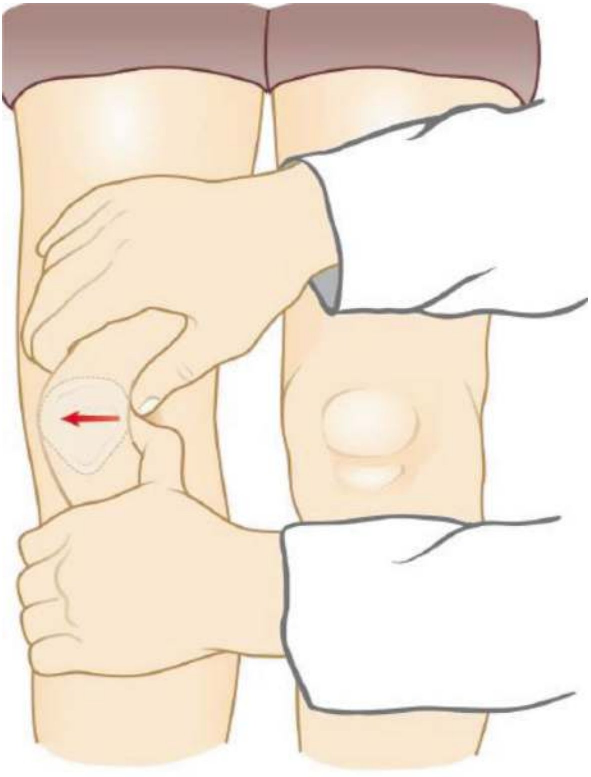
Complaints of instability and anterior knee pain, giving way symptoms.

Diagnosis

- Clinical examination
  - Hemarthrosis (not in case of habitual instability)
  - Medial-sided tenderness (over MPFL)
  - Patellar apprehension

    The patella is pushed laterally at 30° of knee flexion

    Positive if the patient feels the familiar sensation that the patella will soon dislocate (*should not be painful*)



Patella apprehension test

- J-sign

  Excessive lateral translation of the patella in extension, causing the patella to "pop" into the groove as it engages the trochlea early in flexion
- Lower leg alignment and internal/external rotation of the hip (best tested in prone position)

- Radiographic examination
  - X-ray: to exclude fracture (medial patellar facet or lateral femoral condyle) and assess patellar height and trochlear dysplasia according to Déjour
    - Caton-Deschamps index (CDI) for patella alta
      - >1.3 = Patella alta, <0.6 = Patella baja
      - CDI is useful regardless of tuberosity osteotomy and somewhat less dependent on degree of knee flexion and distal patellar pole abnormalities
  - Standing long leg radiographs: Determination of frontal leg alignment (hip-knee-angle).
  - CT: To assess TTTG, tibial tuberosity torsion, femoral and tibial torsion
  - MRI: To assess chondral lesion, trochlear dysplasia and axial deformity

Therapy

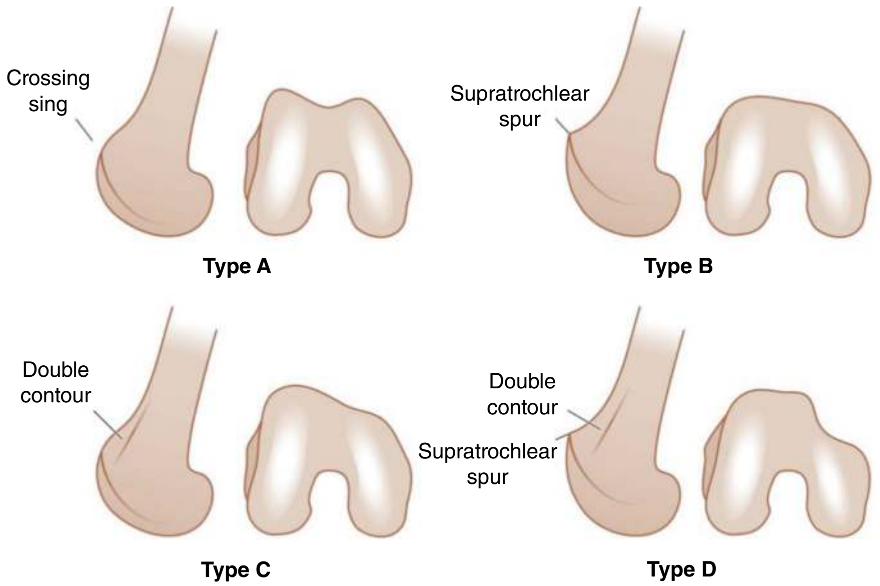
Conservative First-line treatment for first-time patellar dislocation

- Anti-inflammatory medication, bracing, rehabilitation (closed chain quadriceps strengthening, core and hip strengthening to improve limb positioning and balance)

Operative

- Indicated for chronic instability (recurrent dislocation and habitual instability) and for displaced osteochondral fractures or loose bodies following initial dislocation
  - Removal of loose bodies or chondral repair surgery
- Prognosis: 25 years after the initial dislocation, 50% of patients will have patellofemoral arthritis
  - Associated with osteochondral lesions, recurrent dislocations and trochlear dysplasia
- Technique (the therapeutic approach depends on the main cause of the underlying morphological risk factor):
  - MPFL reconstruction and lateral retinaculum lengthening
    - The origin of the MPFL is anterior and distal to the adductor tubercle, but proximal to the MCL and is defined radiographically by the Schöttle point
  - Sulcus deepening trochleaplasty for severe trochlear dysplasia
    - In case of prominent bump (type B or D)
    - Successfully restores patellofemoral stability (re-dislocation rate of 2.4%)
  - Bone realignment osteotomies in cases of increased femoral antetorsion (→ external rotation distal femoral osteotomy) or valgus leg axis (→ varus osteotomy)
    - Increased femoral antetorsion of >25°
    - Valgus deformity above of 3–5° associated with patellofemoral maltracking

– Medializing (and possibly distalisation) of the tibial tuberosity if TTTG >20 mm and still unsatisfactory after correction of other bony risk factors



Dejour classification of trochlear dysplasia

Knee Osteoarthritis

Definition

Degenerative joint disease that can lead to joint deformity through progressive changes in cartilage and bone structures.

Etiology

- Primary: "idiopathic"
- Secondary: post-traumatic, high BMI and metabolic syndrome, rheumatoid arthritis, post-infectious, hemophilia, hemochromatosis, gout

Clinical Presentation

Function-limiting knee pain, pain at night or rest (late stage), swelling, stiffness, mechanical symptoms (instability, locking sensation)

Diagnosis

- Clinical examination
  - Gait
  - Effusion
  - Limb alignment
  - Limited range of motion
  - Ligament integrity
- Radiographic examination
  - X-rays in 3 planes (Kellgren & Lawrence grading): Joint space narrowing, subchondral sclerosis, osteophytes, and bone-on-bone deformity, cysts
    - Weight-bearing AP, lateral and tangential (Sunrise view) radiographs
    - Schuss (Rosenberg) view allows assessment of posterior condylar wear that may not be apparent in the AP view
    - Long leg axis view for alignment

Therapy

Conservative

- Goal: reduce pain, improve mobility (and extrinsic stability) and delay/avoid surgery
- Weight reduction
- Physical therapy (e.g., GLA:D, cryotherapy or heat therapy)
- Pharmacological therapy (local, oral, intra-articular)
  - No significant difference in pain relief between steroid, hyaluronic acid, or PRP injections
- Bracing (moderate evidence)

Operative

- Arthroscopic procedures
  - Strong evidence against joint debridement or lavage
  - Inconclusive evidence for meniscal debridement (but certainly not as a first-line treatment)
- Limb realignment osteotomies
  - Indication: Unicompartmental chondropathy grade 3–4, but grade < 3 in the contralateral compartment in younger patients (age up to 60 years, but no absolute value)
    - Correlation between frontal limb malalignment and later unicompartmental knee osteoarthritis: 3.5-fold risk for malalignment without cartilage damage and 4 to ten-fold risk for malalignment and osteoarthritis (K&L 2 and 3)

- Location of the correction: Femoral, tibial, or double level, based on the mechanical lateral distal femoral (mLDF) and mechanical medial proximal tibial (mMPT) angles (normal values: 87 ± 3°)
  - Less than one third of patients with mechanical varus ≥3° have a tibial deformity. If anatomical correction (mMPTA ≤90°) is desired, only about 10% of patients can be corrected with an isolated HTO, while 60% of patients require a double-level osteotomy. If a slight overcorrection is accepted (mMPTA ≤95°), 60% of patients can be corrected with an isolated HTO, while one third of patients would still require a double-level osteotomy
  - In general, JCLA should not exceed >3–4° (due to shear forces not parallel to the ground) and mMPTA should not exceed 93° (certainly not 95°)
- Amount of correction: up to 0.5–3.5° of valgus (50–65% of the transverse diameter of the medial tibial plateau; Fujisawa point at 62.5% or 2.5° valgus)
- Gap filler: iliac crest with accelerated healing but no functional benefit
  - No gap filler needed for correction under 10 mm

- Unicompartmental arthroplasty
  - Contraindications: multicompartmental OA, ligament instability (ACL deficiency), limited ROM, fixed varus >10°, inflammatory arthritis (if untreated or unsuccessfully controlled)
  - Lower and delayed conversion rate of HTO to TKA than UKA, otherwise HTO and UKA are both equivalent solutions for medial OA of the knee
- Total knee arthroplasty
  - Types:
    - Cruciate retaining (CR; medial pivot)
    - Cruciate substituting (PS; UC; medial pivot)
    - Constrained nonhinged (semi-constrained) and constrained hinged (with rotating platform) prosthesis
  - Patella resurfacing: no difference in pain or function with or without patella resurfacing (no clear data pro or con). In selected cases, resurfacing should be strongly considered (inflammatory arthritis, severe and symptomatic patellofemoral osteoarthritis)
    - There is evidence that revision rates for anterior knee pain are higher without patellar resurfacing and that outcomes after revision with patellar resurfacing are moderate
  - Fixed versus mobile bearing inlay: No advantage with mobile bearing inlays, but risk of dislocation. Theoretical advantages: lower contact stress (stress dissipation) and better kinematic properties
    - Higher costs and significantly worse results in arthroplasty registries with some exceptions (manufacturer-specific)
  - Cemented versus uncemented: greater primary stability with cementation, but no long-term benefit
  - Reasons for revision (according to SIRIS report): patella problems (27%), infection (20.5%), tibial loosening (18%), femorotibial instability (18%), pain of unknown origin (10%), femoral loosening (11%), joint stiffness/

arthrofibrosis (6%), inlay wear (5%), femoral (4.5%) and tibial (4%) component malalignment

– **TKA infection**:

Incidence 1–1.5%, caused by skin bacteria in 95% of cases

Frequent risk associations with primary surgical site infections: BMI > 40, immunosuppression (medication); wound dehiscence; prolonged operation time; previous operation (ipsilateral); diabetes mellitus; alcohol abuse; rheumatoid arthritis; liver cirrhosis; dialysis; high ASA score; high perioperative blood loss; intraoperative surgical complications; inadequate antibiotic prophylaxis (omitted, given too early or too late); lack of aseptic principles during and/or immediately after surgery

Usually *Staphylococcus aureus* (often highly virulent) or coagulase-negative *staphyolocci* such as *S. epidermidis* (often less virulent)

Difficult to treat infections: pathogens resistant to biofilm-active antimicrobials (Rifampicin-resistant staphylococci, Ciprofloxacin-resistant gram-negative bacteria, fungi)

Major diagnostic criteria:

1. (Non-traumatic) sinus tract communicating with the prosthesis
2. Pathogen isolated by culture (or PCR) from at least 2 separate tissue/fluid/pus samples from the affected joint and/or blood cultures
3. Visible pus around the prosthesis

Minor diagnostic criteria as a selection because they are not sacrosanct: elevated CRP, elevated synovial leukocytes (>2000–3000 cells μL) and > 75% polynuclear cells in synovial fluid analysis, corresponding clinical presentation (febrile knee swelling), pus, radiological loosening of the prosthesis, and several others

Periprosthetic joint infection can be perioperative or secondary (haematogenous). Classification is based on time since symptom onset with implications for management: <4 weeks is considered an acute infection given the mature biofilm formation within 4 weeks

Therapy:

1. Early (and first-time) infection: Irrigation and debridement with extensive wound drainage, replacement of replaceable components (inlay) = DAIR (debridement and implant retention) procedure. Success rate: 60–85% but may require a second look
2. Late or recurrent infection with the same pathogen, with implant loosening or soft tissue deficits not amenable to primary closure: Revision surgery (one or two stage, depending on the context and surgical experience). Sometimes an early second look is necessary (high infection inoculum, spacer dislocation, haematoma) without representing a failure
3. Systemic antibiotic therapy, but no antibiotics before diagnosis! Probably no antibiotic regimen is superior to another, except for staphylococcal biofilms. Rifampicin as combination therapy for

staphylococcal infections where definitive implants (not temporary spacers) are in place. No rifampicin monotherapy (rapid development of resistance). Additional local antibiotic treatment (often using prefabricated gentamicin and/or vancomycin in spacers) is recommended. The duration of postsurgical treatment is the subject of research. Current expert opinion ranges from 6 to 12 weeks

Arthritis

Etiology

- Bacterial arthritis:
  - Hematogenous/direct route; results in rapid chondrolysis
  - Typical pathogens: Staphylococcus aureus, gonococci, streptococci
- Systemic diseases:
  - Chronic synovitis (e.g., rheumatoid arthritis) leading to cartilage/bone destruction
- Crystal arthropathy:
  - Both acute synovitis and chronic damage due to crystal deposition (gout, pseudogout)

Clinical Presentation

Signs of inflammation (pain, heat, redness, swelling, and loss of function)

Diagnosis

- Medical history (e.g., gout, rheumatoid arthritis?)
- Physical examination: Fever, malaise, signs of inflammation
- Serum labs: CRP, ESR, leukocytosis, blood cultures
- Knee aspiration (no local anesthetic prior to puncture, as bactericidal)
  - Consistency and colour
  - A cell count > 50,000 cells/mL is indicative of septic arthritis, although such a high number can also be found in acute inflammatory/aseptic arthritis
  - Gram stain identifies only 1/3 of the microorganisms
  - Culture
  - Crystals
- Imaging: X-ray, MRI, bone scan

Therapy

Treatment depends on the underlying cause:

- Septic versus aseptic arthritis
  - Even in non-bacterial arthritis, CRP and leukocytes may be elevated. The indication for emergency knee joint lavage is based on the clinical symptoms (patient condition? sepsis?) and the aspirate (purulent?)
  - Bacterial arthritis is an orthopaedic emergency
    - Rapid arthroscopic lavage, synovectomy and debridement, possibly a second look, intravenous antibiotics

Traumatology

Distal Femur Fractures

Etiology

Bimodal distribution: low energy (older patients with osteoporosis) vs. high energy (younger patients) trauma

Diagnosis

Radiographic work-up

- XR: Double density sign on AP view: Hoffa fragment
- CT:
  - low threshold for preoperative planning and evaluation of intra-articular involvement
  - Detects coronally oriented Hoffa fracture (incidence 38%, missed in 31%)
- CT angiogram:
  - Indication:
    - Diminished / absent pulse
    - Expanding hematoma
    - ABI <0.9
    - Persistent arterial bleeding
    - Damage to associated nervous structures

Therapy

Conservative rarely indicated for stable, nondisplaced fractures or non-ambulatory patients

Operative with the goal of anatomical reduction of the articular surface (absolute stability), functional reduction (relative stability) of the metaphysis restoring length, alignment and rotation

- Fracture deforming forces:
  - Gastrosoleus ➔ use bump or triangle under knee to counteract
  - Adductors ➔ varus
  - Quadriceps/hamstrings ➔ shortening
- Open reduction internal fixation (ORIF):
  - Fixed-angle plates are required for metaphyseal comminution. Non-fixed-angle plates (e.g., condylar plates) are prone to varus collapse
  - Surgical approaches: Lateral approach or modified Swashbuckler (allows more articular surface exposure)
- Retrograde nailing
  - Transtendon or medial parapatellar approach, combined with intra-articular exposure for intra-articular fracture extension
  - Fracture morphology: metaphyseal zone with shaft extension

    For younger patients: prefer plate fixation to avoid violating the knee joint
- Combined fixation constructs
  - Dual plate or plate-nail combinations

    biomechanically stronger but no clinical trials have shown superiority
  - To resist varus collapse in extended comminution in the medial metaphyseal zone (as a substitute for a deficient medial cortex)

    Potentially allows earlier weight bearing and reduces non-union rates
- Non-union
  - Rate between 10–20%
  - Patient factors: smoking, diabetes, obesity, open injuries
  - Technical factors: malreduction (varus, medial translation) and longer working length
- Distal femoral replacement
  - For highly comminuted fractures, especially in older patients
  - Resects the fracture and allows for immediate full weight bearing
  - Complications associated with arthroplasty

Tibial Plateau Fractures

Etiology

Bimodal distribution: low energy falls (older patients with osteoporosis) vs. high energy trauma (younger patients).

Diagnosis

Clinical work-up:

- Neurovascular injuries (ABI, Doppler, angiography)
- Compartment syndrome
- Injury to the knee joint: cruciate/collateral ligaments; meniscus, cartilage injury

Radiographic work-up with XR and possibly CT

- Schatzker classification
  - I-III most common
  - I: younger patients, better bone quality
  - II: higher energy, with associated depression of lateral plateau
  - III: poor bone quality in elderly patients
  - IV

    A: low energy, simple depression due to varus loading, fracture line exits medial to the spines

    B: high energy in young patients, medial shearing with the fracture line exiting laterally lateral to the spines; fracture dislocation variant; with a high risk of vascular injury
  - V + VI: bicondylar fractures, high energy; assessment for compartment syndrome and vascular examination a must
- Hohl and Moore classification
  - Useful for true fracture dislocations that do not fit the Schatzker classification (10% of all tibial plateau fractures)
  - Fractures associated with knee instability

Therapy

Conservative rarely indicated for minimally displaced split or depressed fractures stable to varus/valgus and for non-ambulatory patients.

Operative with the goal of anatomical reduction of the articular surface (congruence), leg axis (alignment) and joint stability (strongest predictor of long-term outcome)

- Indication: articular depression >5-10 mm, condylar widening of >5 mm, varus/valgus instability of >10°, medial plateau fractures, bicondylar fractures
- ORIF:
  - Three column principle ➔ approach according to fracture pattern
  - Anterolateral approach: Open-book or keyhole technique for articular reconstruction
  - Medial or posteromedial approaches (supine)

    Indicated if medially open, comminuted, significantly displaced and if the posteromedial fragment will not be captured by lateral screws
  - Lobenhoffer approach (prone): for more posteromedial access, indicated for posteromedial coronal shear fractures (when the fixation obtained from the medial aspect of the tibia is suboptimal)

  - Bone grafting may not always be necessary. Options:
    - Iliac bone autograft or (freeze-dried) structural bone allograft
    - Calcium sulfate: very bioresorbable and non-inferior to iliac bone graft
    - Calcium phosphate: takes years to resolve (less bioresorbable), non-inferior to iliac bone graft
- External fixator:
  - Indicated for tibial plateau fractures associated with the presence of circumferential blistering, extensive skin bruising or necrosis and compartment syndrome (definitive ORIF after skin closure reduces the risk of infection). Otherwise, early definitive ORIF for bicondylar tibial plateau fractures is safe
- Primary revision arthroplasty for elderly patients with comminuted fractures with the goal of immediate full weight bearing

Patella Fractures

Etiology

Direct impact.

Diagnosis

Clinical examination:

- Extensor mechanism intact (single leg raise)?

Radiographic work-up with XR and possibly CT

Therapy

Conservative

- Basically, the treatment algorithm assesses intra-articular step-off, displacement/distraction/gap and the extensor mechanism
- Indication:
  - Vertical fractures are rarely treated surgically as the displacement is almost never significant
  - Non-displaced transverse and comminuted fractures can be treated non-operatively
    - Non-displacement indicates that the retinaculum, and therefore the extensor mechanism, is intact

Operative

- Indication:
  - 2-3 mm of intra-articular step-off
  - Transverse/comminuted fractures with an intact extensor mechanism but a distraction/gap of >1-2 mm can cause an extensor lag and affect strength and stability, especially when descending stairs. In young patients <50 years, this is rarely tolerated. In older patients, however, some displacement is accepted if the extensor mechanism is intact
  - Technique: Tension band construct, screws or (less commonly) plate fixation

Foot and Ankle

8

Andreas Flury, Ines Unterfrauner, Norman Espinosa, Markus Knupp, and Stephan Wirth

Anatomy and Biomechanics

Bones of the foot

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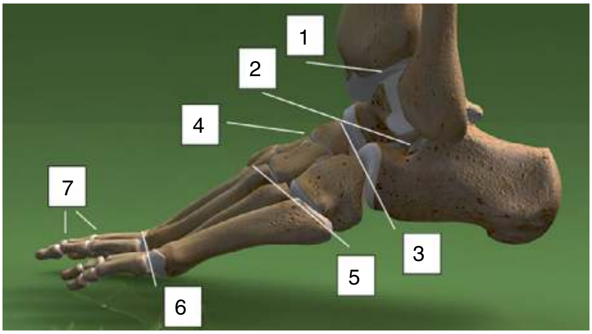
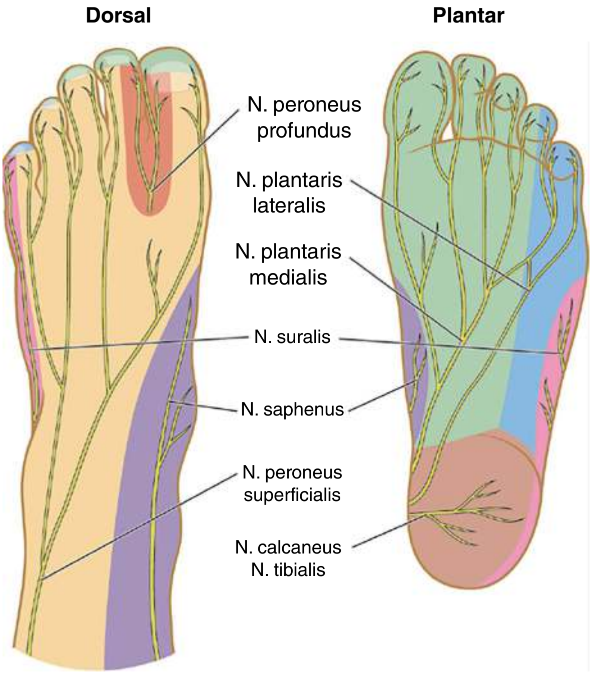


Joints of the foot

1. Ankle joint	Art. talocruralis
2. Subtalar joint	Art. subtalaris
3. Chopart joint	Art. tarsi transversa = Art. calcaneocuboidea + Art. talonavicularis
4. Art. cuneonavicularis	
5. Lisfranc joint	Artt. tarsometatarsales
6. MTP joints	Artt. metatarsophalangeales
7. Artt. interphalangeales	PIP + DIP joints

Innervation

N.tibialis	→ N. plantaris medialis + N. plantaris lateralis + calcaneal branch
N.peroneus communis	→ N. peroneus profundus (*"flip-flop" nerv*) + N. peroneus superficialis (*sensory function at foot level only!*)
N.suralis	→ Sensory function only! – (*Union N. tibialis* + *N. peroneus communis*)
N.saphenus	→ Sensory function only!



Nerves of the foot

Arterial Blood Supply

- A. tibialis anterior
- A. tibialis posterior
- A. peronea

CAVE

Pronation + supination at the foot are **movements made of combinations of**:

- **Pronation** = forefoot abduction + eversion + dorsiflexion
  AND
- **Supination** = forefoot adduction + inversion + plantar flexion

- Key element of foot movement: The Chopart joint
  - Talonavicular joint: is flexible and allows mobility

    Articulates posteriorly with the anterior and middle facets of the calcaneus, and antero-inferiorly with the articular surface and the spring ligament (the structure form the so-called acetabulum pedis)
  - Calcaneocuboid joint: is rigid for stability and follows the talonavicular joint
  - Biomechanical principle:

    At heel strike, the heel is everted and the midfoot is pronated. The talar head glides inferiorly and medially (within the acetabulum pedis) ➔ The TN and CC joints are parallel, making the hindfoot/midfoot transition flexible. This allows shock absorption. The longitudinal arch is flattened

    At toe-off, the heel is inverted, resulting in a divergent alignment of the transverse tarsal joint. The longitudinal arch is elevated and stabilized (rear foot is stable), allowing the foot to act as an extension of the leg (rigid lever arm) and therefore a stable push-off

Imaging

Conventional X-Ray

- Easy and quick to obtain
- Bilateral views allow comparison between both feet and ankles
  - Alignment Deformities
  - Fracture assessment
  - Indirect signs of ligamentous injury
- Weight-bearing images are mandatory
- Four essential views are necessary:
  - Ankle anteroposterior (ap)
  - Foot dorsoplantar (dp)
  - Foot lateral
  - Foot oblique
- Comparison with the opposite side in case of suspected ligament lesion (*e.g., syndesmosis rupture*)

Upper row: dorsoplantar, oblique and lateral views of the foot. Bottom row: lateral and anteroposterior view of the ankle

Computed Tomography (CT)

- Depicts bones at best
- 3D analysis possible
- For assessment of complex fractures (*pilon, calcaneus, midfoot, Lisfranc*), osseous bridging of fractures or fusions, and imaging of osteochondral lesions (*CT* arthrography)

Magnetic Resonance (MRI)

- Depicts soft-tissue at best
- 3D analysis possible
- Accurately depicts osteochondral lesions, bone marrow oedema, osteonecrosis, osteomyelitis, neoplasms (tumours), tendon and soft tissue injuries

Hallux Valgus

Etiology

Multifactorial: intrinsic (genetics, *ligamentous laxity*), extrinsic (*tight footwear*)

Clinical Presentation

Difficulty wearing shoes due to medial eminence (bunion). Consequence: deviation of the remaining toes with possible mechanical conflict and transfer metatarsalgia (weakened Windlass effect, as the transmission of force via the big toe is reduced, provoking load redistribution)

Diagnosis

- Clinical examination
  - Tender bunion
  - 1st MTP joint ROM
  - (Negative) grind test
  - Hypermobility of the 1st TMT joint
- Radiographic examination
  - "Hallux valgus angle" → (*HVA*) = Metatarsophalangeal angle I; *normal value* 8–15°
  - "Intermetatarsal angle" → (*IMA*) I–II; *normal value* < 9°
  - Decentering of the sesamoids
  - Round sign → Lateral rounding of the metatarsal head corresponds to the profile of the plantar condyle and is therefore a sign of metatarsal rotation

Hallux valgus radiographic assessment: HVA 23°, IMA 11.6°

Therapy

Conservative

– Shoe modifications: Wider toe boxes, soft leather shell
– Splints: night splints, functional splints, special socks (little effect, palliative only)
– Insoles: *inserts or orthotics (little lasting effect)*

Operative

Indication: Unbearable pain. In total, over 100 different operations are described

- Lapidus procedure: Fusion of the 1st TMT joint;
  - Indications:
    Hypermobile first ray
    (i) Higher recurrence rates with distal corrections
    (ii) The clinical significance of a hypermobile first ray in the surgical treatment of hallux valgus is uncertain, especially considering that

realignment with distal osteotomies also stabilizes the first ray. Clinical assessment of first ray instability is highly dependent on the examiner. Radiographically, there is no clear indication unless there is an obvious gap between the 1st and 2nd ray in the lateral view. A Lapidus procedure should be considered in cases of flat foot due to the associated (and relevant/obvious) instability

Severe deformity and/or previously failed hallux valgus correction

(i) HVA >40°, IMA >20°

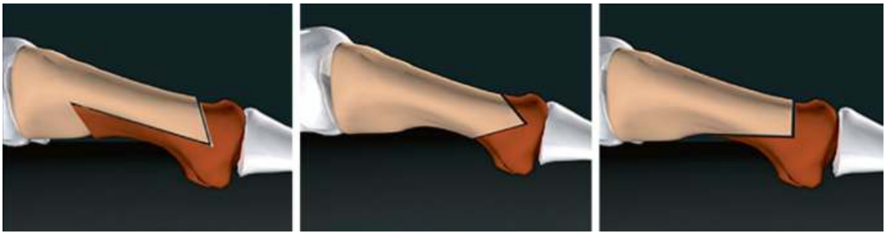
(ii) Metatarsal pronation can be assessed with MRI or CT and may help guide surgical decision making. To correct the underlying deformity, the metatarsal can either be de-rotated with a Lapidus procedure or the distal portion can be medialized with a distal osteotomy

- Various distal options: Osteotomy of the first metatarsal and capsular release (*at the level of the 1st MTP joint*):
  - Scarf
  - Chevron
  - Reversed-L
  - Percutaneous minimal-invasive technique

    Allows rotation of the distal fragment
- Concomitant Akin osteotomy to correct interphalangeal component

Table: Comparison of different surgical treatments for hallux valgus

	Lapidus procedure	Distal MT osteotomy	Percutaneous minimal-invasive distal correction
MTP I osteoarthritis	NO	Ok	Ok
IMA >20°	Ok	NO	Ok
Rotational deformity	Ok	Depending on surgeon	Ok
Clinical outcome	Comparable	Comparable	Comparable
Post-operative aftercare	Partial weight bearing in cast for 6 weeks	Full heel weight bearing	Full heel weight bearing
Complications	Comparable	Comparable	Comparable
Radiographic outcome	Similar	Similar	Similar
Learning curve	Ok	Ok	High



Surgical techniques from Vienne, P. et al. Comparative mechanical testing of different geometric designs of distal first metatarsal osteotomies. Foot Ankle Int **28**, 232–236 (2007): Scarf, Chevron, Reverse-L (from left to right)

Hallux Rigidus

Etiology

Overload e.g. in flatfoot with hindfoot valgus (= *increased load on the 1st ray*), post-traumatic, micro-traumatic

Clinical Presentation

1st MTP joint pain, worse with push off, restricted ROM of movement, dorsomedial paresthesias due to compression of the Nervus cutaneus medialis dorsalis by osteophytes. Clinical examination includes 1st MTP joint ROM (limited and painful dorsiflexion) and grind test

Diagnosis

- Clinical examination
  - Limited and painful 1st MTP joint ROM (especially dorsiflexion)
  - Positive grind test
- Radiographic examination

Therapy

Conservative

Goal:	To reduce mechanical stress within the 1st MTP-joint and thus to reduce pain
Options:	First line treatment is mechanical by applying a stiff carbon support underneath the first ray. In addition to this NSAIDs, local cortison injections and further shoe adjustments with toe box widening over the exostosis can be considered

Operative
Goal: To relieve 1st MTP-joint from mechanical stress and to diminish pain

- Dorsal cheilectomy → Removal of osteophytes
  - No more than 30% of the metatarsal head may be removed, otherwise there is a risk of joint subluxation
  - Increased postoperative ROM may lead to increased pain
- 1st MTP joint fusion → In neutral rotation, 15° valgus and 25° dorsal extension
  - Satisfactory clinical results in all age groups
- MTP-I-Prosthesis
  - High complication rates

Turf Toe

Etiology

Hyperextension injury of the 1st MTP joint resulting in tear to capsular-ligamentous-sesamoid complex

Clinical Presentation

Inability to push off, acute pain and reduced ROM

Diagnosis

- Clinical examination
  - Limited and painful ROM
  - Plantar 1st MTP joint tenderness
  - Positive vertical Lachman test (positive if greater laxity compared to contralateral side)
- Radiographic examination
  - X-Ray: sesamoids may be displaced proximally or may show fracture
  - MRI: rupture of the plantar plate

Therapy

Conservative hard insole for grades I–III (sprain, partial tear, complete tear)

Operative

- Surgical repair, usually for grade III injuries (complete tear) where conservative treatment has failed, sesamoids are retracted, there are loose bodies in the joint or progressive traumatic hallux valgus deformity

Differential Diagnosis

- Fracture (usually of the tibial sesamoid, which carries more weight and is larger)
- Sesamoiditis: due to repetitive microtrauma; clinical diagnosis, no radiological abnormalities
- Chondromalacia: e.g. as a sequela of sesamoiditis: fragmentation, sclerosis, collapse, flattening after 9–12 months
- Osteoarthritis: post-traumatic or due to hallux valgus deformity

Small Toe Deformities

Etiology

Hammer toe: badly fitting shoes (bent toes), associated with bunion or flattened arch
Claw toe: mechanical or neuromuscular
Mallet toe: traumatic or isolated FDL tightness

Diagnosis

- Clinical examination
  - Hyperkeratosis and tenderness on the dorsal side of PIP joint
  - Hyperkeratosis and tenderness underneath the MT head
  - Painful and unstable MTP joint

	Hammer toe	Claw toe	Mallet toe
MTP	Normal	Hyperextension	Normal
PIP	Flexion	Flexion	Normal
DIP	Extension	Flexion	Flexion

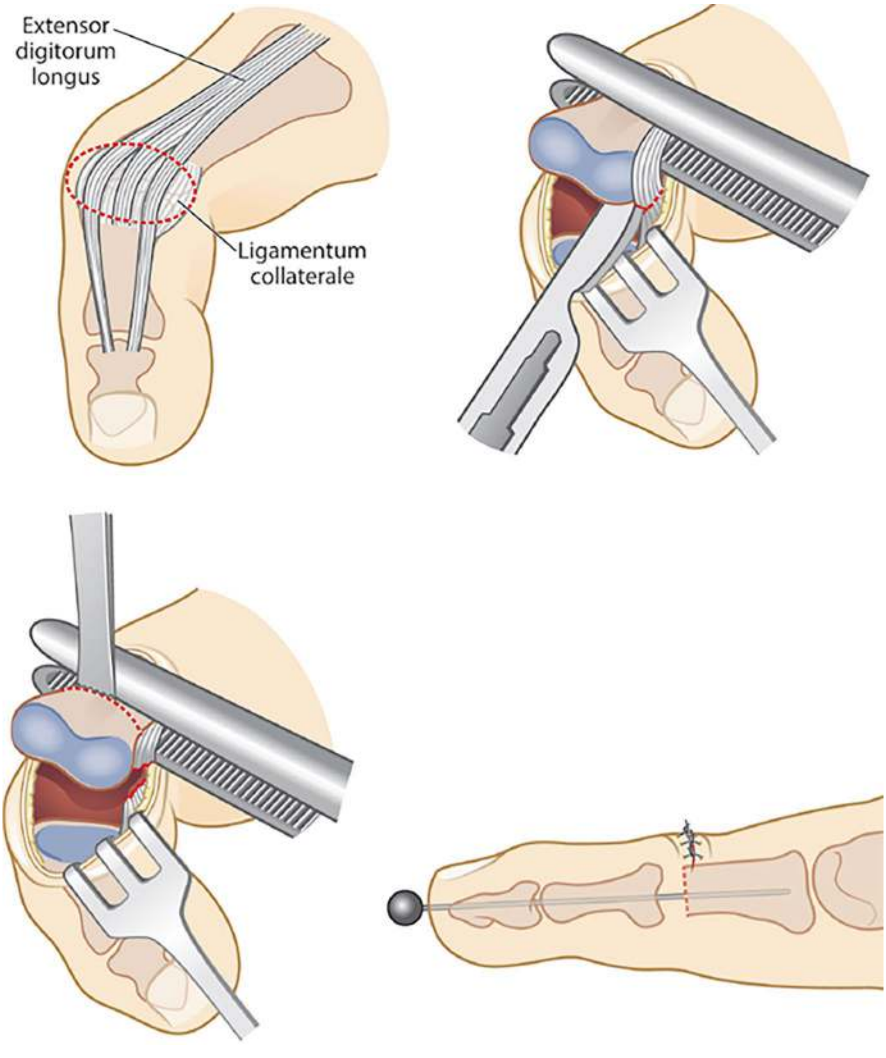
Lesser toe deformities

Therapy

Conservative Shoe modification (wide toe box) and soft bedded/padded insoles with a metatarsal bar

Operative

- Girdlestone procedure (FDL to EDL transfer) for flexible deformities
- Hohmann osteotomy: Resection of the PIP/DIP joint and fixation with K-wire
  - PIP for hammer toe, DIP for mallet toe
- Weil or Maceira osteotomy: shortens and elevates the metatarsal head
  - For claw toes or in case of metatarsalgia



Hohmann procedure

Metatarsalgia

Etiology

Primary: Morphology of the metatarsal bone (increased plantar flexion or length discrepancy) or its relationship to the rest of the foot (first ray insufficiency, forefoot equinus)

Secondary: Malalignment or deformity of the MT head (post-traumatic or due to avascular necrosis = *Köhler-Freiberg's disease*)

Clinical Presentation

Plantar forefoot pain due to increased pressure under the metatarsal heads (*forefoot overload*); patients prefer to mobilize in shoes rather than barefoot (as opposed to Morton's neuroma where tight shoes cause pain)

Diagnosis

- Clinical examination
  - Hyperkeratosis und tenderness sub MT heads II-IV
  - Gait cycle
  - Hindfoot axis (cavovarus deformity)
  - Insufficient first row (hallux valgus, hypermobility, hallux rigidus)
  - Ankle ROM
  - Contracture of the calf muscles (Silfverskjöld test)
  - Plantar fatpad atrophy
  - Interdigital tenderness, pain with forefoot pressure, and Mulder's click are indicators of concomitant Morton's neuroma (not a "real" neuroma, but perineural fibrosis due to compression of the interdigital nerve)
- Radiographic examination
  - X-ray: Length of each MT in relation to the others
  - MRI: Demonstrates bursitis under the MT heads and rules out Morton's neuroma or Köhler-Freiberg's disease

Therapy

Conservative Physical therapy (calf stretching), shoe inserts (retrocapital support, soft padding under the MT heads), steroid injections (for bursitis or Morton's neuroma), podiatry

Operative Various surgical procedures tailored to the underlying problem

- Weil osteotomy (MT shortening) or Maceira osteotomy (MT shortening and head elevation)
- Strayer release
- Excision of Morton Neuroma
  - 10–15% recurrence rate due to inadequate resection
  - Postoperative interdigital numbness is likely
  - Painful scarring more common with plantar > dorsal surgical approach

Lisfranc Injury

The Lisfranc joint consists of the three cuneiform bones and their articulations with the bases of the five metatarsal bones. The bones are held together by strong ligaments called Lisfranc joint complex. When injured, the integrity of the midfoot joint complex (Roman arch), which stabilizes the arch of the foot and is important for pushing off, is lost

Etiology

Axial load and rotational forces with the foot plantarflexed or direct force on the dorsum of the foot

Clinical Presentation

Pain, swelling, deformity; pathognomonic: plantar hematoma

Diagnosis

- Radiographic examination
  - Fractures missed in 50% of cases with unloaded X-rays
  - Weight-bearing with comparison view may be necessary
  - Widening (diastasis) of the interval between the 1st and 2nd ray, disturbed alignment of the medial base of the 2nd metatarsal to the medial cuneiform, a positive fleck sign, dorsal displacement of the base of the 1st or 2nd metatarsal or → perform CT
  - MRI for questionable instability without dislocation

Therapy

Conservative in the absence of signs of instability (undisplaced and stable) → Lower leg cast for 6 weeks, no weight-bearing allowed

Operative

- Swelling is unlikely to subside until reduction is achieved. However, the status of the soft tissue envelope should be considered before surgery
  - Closed reduction and K-wire fixation with delayed definitive fixation in case of doubt
- Lisfranc injuries are usually complex injuries and require a detailed preoperative plan, as the treatment plan varies from case to case depending on which joints are unstable
- General approach: start proximal and medial ➔ cuneiform/navicular relationship; then 2nd MT/intermedium cuneiform; 2nd MT medial cuneiform; 1st, 3rd, 4th and 5th
  - The aim is to achieve anatomic reduction and stable fixation. There are no guidelines as to whether the joints should be fused or temporarily stabilized. Studies have shown no superiority of dorsal plates over trans-articular screws for temporary fixation
  - The 1st–3rd TMTs are reconstructed, whereas the 4th and 5th TMTs should be left functional, which means temporary K-wire stabilization

Lisfranc fracture dislocation before and after internal fixation

Calcaneus Fractures

Subtypes

- Intra-articular fractures (75% of cases)
  - *Joint depression* or *tongue-type*
- Extra-articular fractures (25% of cases)
  - Posterior calcaneal tuberosity avulsion fractures
    Violent pull from gastrosoleus coupled with forced dorsiflexion in osteoporotic bone (common in women in their seventh decade of life)
    Posterior skin at risk and therefore an emergency
  - Anterior process fractures
    Avulsion of the bifurcate ligament due to inversion and plantar flexion
    Non-operative treatment unless >25% (or > 2 mm) of CC joint displaced

Etiology

The *primary* fracture line typically produces a superomedial "constant" fragment. As it is stabilized and thus held in position by strong ligaments (deltoid, interosseus and talocalcaneal) and capsular insertions, it serves as an intraoperative reference for fracture reduction. The *secondary* fracture line determines the classification of the fracture: *joint depression* or *tongue-type*

- *Joint depression*: The secondary fracture line runs superficially directly behind the posterior facet, leaving the posterior articular facet as a free fragment (not attached to the tuberosity). The tuberosity translates laterally, displaces superiorly (Achilles pull), rotates in varus and shortens into the fracture. As a result, the calcaneus loses height, falls into varus position and the heel becomes wider
- *Tongue type*: Secondary fracture line passes through the tuberosity below the posterior facet

Clinical Presentation

Pain, swelling, deformity, inability to bear weight, open fracture

Diagnosis

- Radiographic examination
  - Decreased Böhler's angle (normal 20–40°) and increased Gissane's angle *(normal 120–145°)* in *joint depression*-fractures
  - CT: gold standard

- Sanders: Classifies the fracture based on the number of fragments of the posterior articular facet on the coronal CT slice. Allows statement about prognosis: Number of fragments and fracture reduction correlate with outcome

Therapy

Conservative

Treatment, particularly the decision to use conservative or surgical treatment, is controversial and the data are conflicting, especially with the increasing use of minimally invasive approaches. In general, non-displaced fractures should be treated non-operatively. Non-operative treatment should also be considered for displaced fractures with risk factors such as smoking, diabetes, multimorbidity, etc.

Cast and 12 weeks immobilization, non-weightbearing

Operative

- **Goals:**
  - Anatomical reduction
  - Restoration of height
  - Restoration of width
  - Correction of axis and alignment
  - Correction of Böhler' and Gissane angles
  - Strong fixation for better postoperative mobilization
- ORIF for Sanders type II-III *joint depression*-fractures: Restore articular integrity, restore calcaneal shape (varus malalignment, fibular impingement with the displaced lateral wall fragment)
- Sinus tarsi approach –> Lateral utility approach> exensile lateral approach
- The literature recommends primary arthrodesis for Sanders type IV *joint depression*-fractures. However, there is evidence that ORIF with (best possible) anatomical reduction reduces the number of post-traumatic osteoarthritis cases requiring surgery (subtalar fusion). There is also evidence that the outcome of secondary subtalar fusion is better if the shape of the calcaneus is first restored with ORIF
- Tongue type fractures display an emergency and needs immediate fixation due to increased risk for skin necrosis
- Soft tissue conditioning is critical (positive wrinkle sign prior to surgery), otherwise there is a risk of skin necrosis. Exception: tongue type fractures!

Talus Fractures

Subtypes

- Talus neck (> 50% of cases)
- Talus body (15–25%) and talus head (rarest)
- Lateral process of talus (10%)
  - Forced dorsiflexion with axial force and a rotational component
  - Surgical treatment in case of >2 mm displacement

Etiology

Talus neck fractures: Forced dorsal extension with applied axial force

Clinical Presentation

Deformity, pain, swelling, inability to bear weight, open fracture

Diagnosis

- Radiographic examination
  - AP, lateral and Canale views (maximum equinus, 15° pronated, oblique view to assess talar neck fracutres)
    Classificaiton according to Hawkins:
    Type I: undisplaced (risk of avascular necrosis: 0–13%).
    Type II: subtalar dislocation (20–50%).
    Type III: subtalar and tibiotalar dislocation (20–100%).
    Type IV: subtalar, tibiotalar, talonavicular dislocation (70–100%)
  - CT: gold standard

Therapy

Conservative short leg cast for 8–12 weeks for non-displaced fractures (Hawkins I), no weightbearing allowed

Operative Open reduction and internal fixation

- Injury-related factors are most predictive of osteonecrosis of the talar body, with displaced fractures (according to Hawkins), talar neck fractures and open injuries having higher rates

- The peroneal artery contributes approximately 15% of the blood supply to the talus; the anterior tibial artery 35% (mainly to the anteromedial head of the talus); and the posterior tibial artery approximately 50%. Intra-operative iatrogenic damage to the remaining blood supply appears to be prudent for surgical outcome
- Surgical technique: two approaches are recommended to properly assess reduction. Preserve the remaining blood supply at all costs: deltoid artery in the deltoid ligament (perform a medial malleolar osteotomy if access is required) and the tibialis anterior artery dorsally (no circumferential dissection)
- The *Hawkins sign* in the mortise view shows the resorption of the subchondral bone, indicating a functioning blood supply to the talus body
- Post-traumatic subtalar osteoarthritis is a more common complication (up to 100%) than avascular necrosis (30–40%)

Ankle Osteoarthritis

Etiology

Posttraumatic (70–80%) > secondary OA (rheumatoid arthritis, osteonecrosis, hemophilia, neuropathic or septic) > primary OA (< 10%)

Clinical Presentation

Joint effusion, pain with weight bearing, loss of motion

Diagnosis

- Clinical examination
  - Pain with ROM testing
  - Loss of ROM compared to the contralateral side
  - Angular deformity may be present
- Radiographic examination
  - X-ray: loss of joint space, possible angular deformity

Therapy

Conservative NSAIDs, injections, shoe inserts orthosis customized footwear

Operative
Joint preserving surgery

- Advantages
  - Pain relief
  - Unloading affected part of joint
  - Postponing sacrificing procedures
- Osseous correction of axis deformity
  - For mild/moderate cases with asymmetric joint involvement
  - Supramalleolar osteotomy versus calcaneal sliding osteotomy

Joint sacrificing procedures

- Fusion:
  - Advantages
    Pain relief
    Acceptable function
    High correction potential
  - Disadvantages
    Overloading of adjacent joints ➔ secondary OA
    Prolonged post-operative immobilization
    Psychological factor
  - Techniques
    Arthroscopically assisted with screw fixation: higher fusion rates
    Open with plate (anterior or lateral): biomechanically more stable
- Joint replacement:
  - Advantages
    Pain relief
    Preservation of function
    Protection of adjacent joints
    Immediate weight-bearing
  - Disadvantages
    Limited durability
    Strict indication: stable joint, no hindfoot malalignment (>15° valgus/varus), sufficient tibial bone stock, no infection, preserved ROM and not too young patients
    1. For flatfoot deformity with ankle OA (stage IV according to Johnson and Strom): consider two-stage approach with (1) double arthrodesis and (2) ankle replacement

Flatfoot Deformity

Etiology

Unclear; the dysfunction of the tibialis posterior tendon is accepted as a common cause. Other identified causes include traumatic or degenerative damage to the passive ligamentous structures of the midfoot and hindfoot (e.g., spring ligament), which are key factors in maintaining foot physiology. Recent studies have also shown differences in talus morphology in patients with flat feet. This is why the name has been changed from "posterior tibial tendon insufficiency" to "adult-acquired flat foot". Nowadays, the term progressive collapsing foot deformity is used (PCFD)

Clinical Presentation

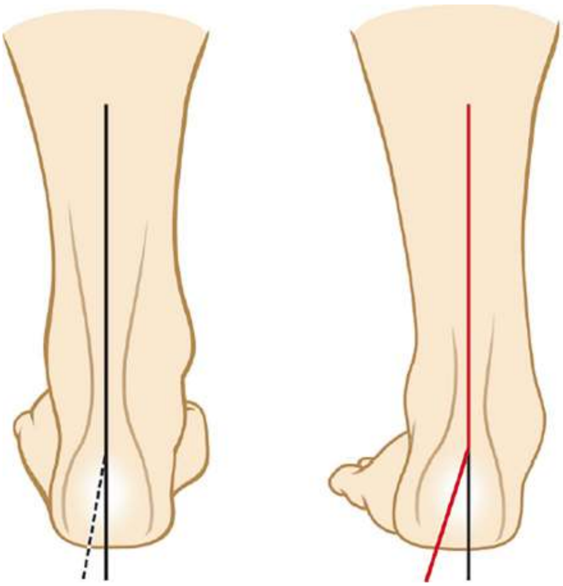
Medial ankle pain and weakness, lateral ankle pain (subfibular impingement), deformity (progressive loss of arch)

Diagnosis

- Clinical examination (◉ Table 8.1)
  - Pes planus
  - Hindfoot deformity (valgus >10°)
  - Forefoot abduction ("*too many toes sign*")
  - Forefoot varus
  - Tenderness along the posterior tibial tendon
  - Tenderness laterally due to impingement
  - Equinus contracture (Silfverskiöld test)
  - Single-limb heel rise: evaluates posterior tibialis tendon
  - Range of motion (flexible or rigid deformity)

Table 8.1 Classification according to Johnson and Strom

Noticeable	Stage 1	Stage 2	Stage 3
Tibialis posterior tendon	**Inflamed (tenosynovitis)**	**Elongated, degenerated**	**Elongated, degenerated**
Deformity	None	**Flexible**	**Rigid**
Pain	Medial	Medial (lateral)	Medial lateral
Single toe stand	Hindfoot in varus	**Weaker**, no varus of the hindfoot	**Not possible**
"*Too many toes sign*"	Negative	**Positive**	**Positive**



Left: Normal (5° valgus). Right: Pes planovalgus (>5° valgus)

Single heel-Rise

- Radiographic examination
  - X-ay DP foot:
    Increased talo-1st metatarsal angle (Simmon angle)
    Increased talonavicular uncoverage (> 40 %)
    Increased talocalcaneal Kite-angle (normal 15–30°)

- Weight bearing lateral foot X-ray:
  - Increased talo-1st metatarsal angle (Meary angle) > 4°
  - Decreased calcaneal pitch (normal 15–30°)
  - Subtalar and talonavicular osteoarthritis (in stages III and IV)
- MRI to assess tendon degeneration and arthritic changes of the hindfoot

Therapy

Conservative
NSAIDs, medial arch supports, brief immobilization, physical therapy.

Operative

- Joint-preserving reconstruction (stage II) versus corrective arthrodesis (stages III/IV)
- Stage II:
  - Flexor digitorum longus transfer
  - Spring ligament suture/reconstruction
  - Osseous correction of hindfoot deformity
    - Fusion of the 1st TMT -joint +/− NC-joint, so-called medial column fusion in order to reduce forefoot-driven hindfoot drive in cases of excessive hypermobility of the first ray
    - Lateral column lengthening: indicated in flatfeet with abducted forefoot. According to Evans: osteotomy parallel to the CC joint, between the anterior and medial joint facets. According to Hintermann: directly in front of the posterior facet, less lateral column overload (CC joint). Does not correct the Achilles pull. Contraindication: supinated forefoot
    - Central calcaneal lengthening: a powerful lengthening of the calcaneus by using medial and lateral approaches. The technique follows the "tiramisu" principle as stated by Klaue
    - Medial sliding calcaneal osteotomy: corrects the Achilles pull
    - Cotton osteotomy (versus 1st TMT fusion): to correct residual rigid forefoot supination
  - Equinus release
    - Strayer release (in Silfverskiöld-positive cases): Incise gastrocnemius aponeurosis from medial to lateral, but protect the suralis nerve laterally
    - Percutaneous Achilles tendon lengthening in Silfverskiöld-negative cases with gastrosoleal contracture
- Stage III:
  - Corrective fusion of the subtalar joint ± in combination with above-mentioned procedures
  - Double Arthrodesis: talonavicular and talocalcaneal
    - Talonavicular fusion corrects forefoot supination and abduction, while subtalar fusion provides hindfoot correction and stability

Collapse of the medial column structures of the midfoot may require restoration of the longitudinal arch by medial column fusion
  - Triple Arthrodesis: the above plus calcaneocuboidal
    Rarely necessary as the CC joint is usually decompressed (opened up) by correcting forefoot abduction with the double arthrodesis

Differential Diagnoses

Müller-Weiss Syndrome Brailsford Disease

- Avascular osteonecrosis of the navicular bone in adults
- *Pes planovarus* or p*es planovalgus* in advanced stage due to lateral navicular collapse. The forefoot falls into abduction and is shortened because the 1st ray is too long. The talus becomes lateralized and the hindfoot falls into varus

Cavovarus Deformity

Etiology

Congenital (residual clubfoot deformity), neuromuscular (up to 70% due to a neurologic condition such as Charcot-Marie-Tooth or Cerebral palsy), post-traumatic (compartment syndrome, crush injury)

Clinical Presentation

Recurrent ankle sprains (instability), lateral ankle pain (peroneal tendons), lateral forefoot overload (painful plantar calluses under 1st metatarsal head and 5th metatarsal head or base)

Diagnosis

- Clinical examination
  - Peek-a-boo heel
  - Coleman block test
    Answers the question of whether the hindfoot varus is forefoot induced and flexible. If so, then a block under the lateral edge of the foot eliminates the plantar flexion of the 1st ray and thus the hindfoot-varizing effect
  - Hindfoot stability (anterior drawer test and talar tilt test)
  - Positive peroneal tendon stress tests (peroneal tendon tear)
  - Interosseous muscle atrophy, lesser toe deformities ➔ Neuro examination
- Radiographic examination

  - X-ay DP foot:
    Metatarsal overlap indicates forefoot pronation
    Talonavicular overcoverage
    Talocalcaneal (Kite) angle <20°
  - Weight bearing lateral foot X-ray:
    Increased talo-1st metatarsal angle (Meary angle) > 4° angle dorsal (due to plantarflexion of the 1st ray)
    Calcaneal pitch >30°
    Sinus tarsi see-through sign and double talar dome sign

Therapy

Conservative
Insoles with lateral arch support with 1st ray deepening

Operative
Surgical strategy depends on deformity (mild/moderate or severe) and rigidity with concomitant osteoarthritis

- Soft-tissue procedures: Lengthening of Achilles tendon, Strayer release, lateral ligament reconstruction (Bröstrom-Gould versus anatomic reconstruction), Steindler procedure (release of the plantar fascia), peroneus longus to brevis transfer
- Realigning osteotomies
  - First ray elevation osteotomy:
    in case of flexible forefoot-driven hindfoot varus
  - Lateral sliding calcaneal osteotomy
    For flexible hindfoot varus
  - Chopart derotational, triple or Lambrinudi osteotomy
    For severe cavovarus foot deformities
    ± with simultaneous supramalleolar varus derotation osteotomy, tendon transfers (of the posterior tibial tendon), Jones procedure (clawing of the 1st toe) and Hohmann osteotomies (for clawing of lesser toes)

Achilles Tendinopathy

Subtypes and Etiology

Insertional tendinopathy, retrocalcaneal bursitis (2–3 cm proximal of the attachment; mostly mechanical, e.g. due to Haglund's exostosis), mid-portion Achilles tendinopathy (in 60% of cases; 2–6 cm proximal of the attachment)

Clinical Presentation

Posterior heel pain, swelling, shoe wear pain due to direct pressure

Diagnosis

- Clinical examination
  - Midline tenderness at insertion site of Achilles tendon
  - Bony enlargement of calcaneus at insertion site (Haglund)
  - More proximal and retrocalcaneal tenderness (retrocalcaneal bursitis)
- Radiographic examination

  Lateral foot X-ray shows bone spur (enthesiophytes), intratendinous calcification, Haglund exostosis

  MRI shows amount of tendon degeneration

Therapy

Conservative

In the absence of mechanical factors → eccentric calf strengthening, stretching, shock wave therapy and night splinting (in dorsiflexion); in the presence of mechanical factors (prominent Haglund's): Heel cap extension

Operative

- Achilles tendon detachment, splitting, debridement (removal of spurs, resection of Haglund's and degenerated tendon tissue), tendon refixation (with suture anchors)
  - Allows to address all three compartments: retrocalcaneal, intratendinous, pretendinous (spur)
  - No scientific support for the Zadek osteotomy (dorsal based closing wedge osteotomy of the calcaneus)
- Endoscopic surgery
  - Allows treatment of retrocalcaneal compartment only: Endoscopic bursal excision and calcaneoplasty
  - Technically more demanding
  - Shorter rehabilitation period

Achilles Tendon Rupture

Etiology

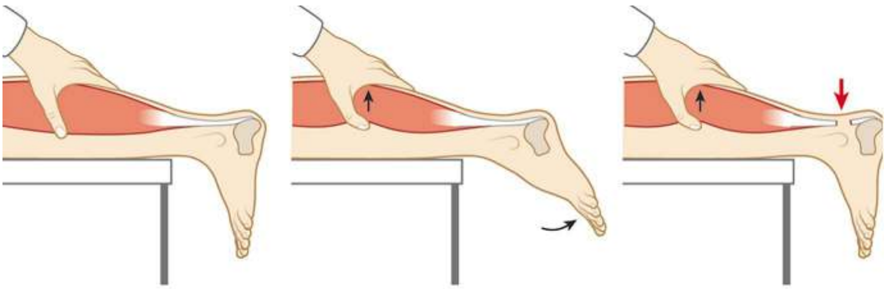
Untrained individuals (*weekend warriors*), unusual strain, after steroid injections or fluoroquinolone use (*Ciprofloxacin*), gout

Clinical Presentation

Audible pop, weakness and difficulty walking, pain in heel

Diagnosis

- Clinical examination
  - Palpable gap
  - Weakness in ankle plantar flexion
  - Positive Thompson test



Thompson test: negative (center), positive (right)

– Increased resting ankle dorsiflexion in prone position (Matles test: decreased tendon pre-tension)

- Radiographic examination
  - X-ray: to rule out other pathology or bony avulsion
  - Ultrasound: may be helpful, but Achilles tendon rupture is a clinical diagnosis
  - MRI: in chronic ruptures to assess fatty infiltration of the gastrosoleus muscle

Therapy

Conservative

Low athletic demands or occupational level, surgical risk factors (e.g., diabetes, smoker, vasculopathy, immunocompromised). Strategy:

- Initial: cast with foot in full equinus, non-weightbearing
- At 2 weeks: transition to cast (or boot) with foot in 20° of plantarflexion, non-weightbearing (or weightbearing as tolerated)
- At 4 weeks: transition to cast (or boot) in neutral, weightbearing as tolerated
- At 6 weeks: document continuity of tendon, transition of boot to daytime wear only (Künzli shoe), physical therapy out of the boot

Operative

- Acute mid-portion ruptures (<6 weeks):
  - Restores proper muscle unit length (pre-tension), greater strength, lower re-rupture rates and higher risk of surgical complications (e.g., wound healing disorders) than non-operative treatment
  - Percutaneous with higher risk of sural nerve damage but lesser risk of wound complications compared with open end-to-end repair
- Insertional ruptures (avulsions)
- Chronic rupture or delayed presentation with tendon gapping:
  - With the gastrosoleus in good condition:
    Reconstruction with V-Y advancement
    (i) A defect of >5 cm cannot be sufficiently closed by this technique
    Repair with allograft (e.g., hamstrings)
  - Fatty infiltration of the gastrosoleus (Goutallier grade 2–3):
    FHL transfer
    (i) The FHL tendon is twice as strong as the FDL tendon and possesses the same force vector as the Achilles tendon. Also contracts in the same gait phase
    (ii) The tendon appears to heal (close the gap) even though the defect is not filled or bridged by the FHL, but due to the restored pre-tension (plantarflexion) after the transfer

Ankle Fractures

Subtypes

- Weber type A: Fibular fracture distal to the joint space/syndesmosis
- Weber type B: Fibular fracture at the level of the joint space/syndesmosis
  - Syndesmotic rupture in 1/3 of cases

- Weber type C: Fibular fracture proximal to the joint space/syndesmosis
  - Maisonneuve fracture: high fibular fracture or dislocation of the proximal tibiofibular joint + rupture of the interosseous membrane + rupture of the syndesmosis, possible rupture of the deltoid ligament, possible fracture of the medial malleolus
- Volkmann fragment: posterior malleolus fracture (wedge-shaped fracture of the posterior distal tibial edge) = osseous avulsion of the posterior syndesmosis
- Tillaux-Chaput tubercle fracture: concomitant avulsion injury of the tibial origin of the anterior syndesmosis
- Wagstaffe-Le Fort fracture: concomitant vertical avulsion fracture of the medial aspect of the distal fibula due to avulsion of the anterior syndesmosis

Fracture of the medial malleolus (red circle). A Tillaux-Chaput fracture is also seen

Diagnosis

- Clinical examination
  – Condition of soft tissue and integument
  – Clinical examination does not reliably detect loss of deltoid ligament integrity. Local tenderness and hematoma may indicate a tear of superficial, but not deep, portions of the deltoid ligament
- Radiographic examination
  – Knee X-ray: consider Weber C fractures and look for proximal fibula fracture
  – AP view in 10–15° internal rotation (= mortise view) to assess distance between tibia and fibula
    Radiographic signs of instability (syndesmotic and/or deltoid integrity): disrupted dime sign, disrupted Shenton's line, increased medial clear space (normal >4 mm), decreased tibiofibular overlap (normal >6 mm on AP view, >1 mm on mortise), increased tibiofibular clear space (normal <6 mm)
  – Stress views (e.g., gravity stress radiographs to assess deltoid integrity): painful and time-consuming. Lead to false-positive results and are therefore inappropriate for surgical decision-making
    Plain weight-bearing radiographs (at presentation or early follow-up) are a reliable assessment of ankle instability
    External rotation stress radiographs under image intensification assess the integrity of the syndesmosis. This can be done intraoperatively after fixation of the ankle fracture or in the case of isolated ligamentous injury with suspicion of syndesmotic instability
  – CT scan for assessment of posterior malleolus and preoperative planning

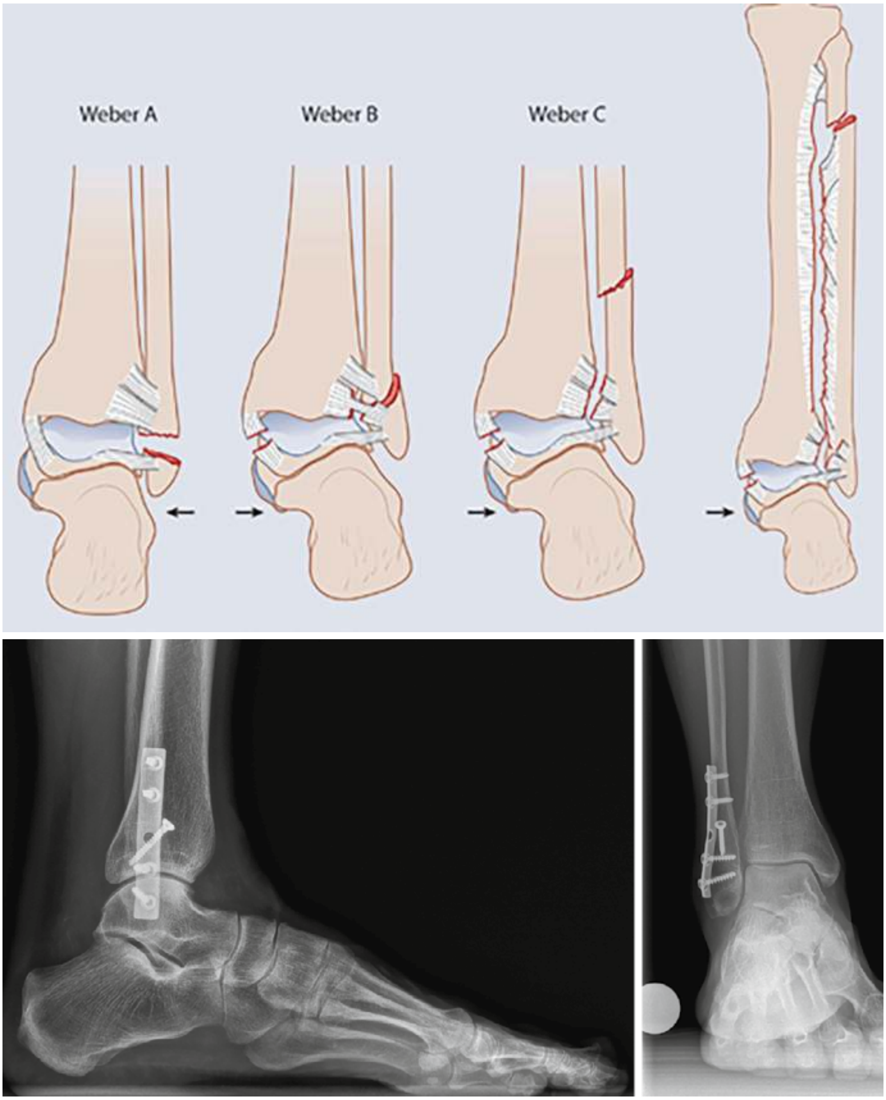
Therapy

Conservative

- Weber Type A without major displacement
- Weber Type B fracture without signs of instability (Lauge Hansen SER stage 2: no Volkmann fragment, intact medial malleolus and stable deltoid ligament)
- Isolated medial malleolus fracture: rule out Maisonneuve fracture and assess integrity of syndesmosis (may require MRI)

Operative

- Weber type B and C fractures with signs of instability (any talar displacement) or fracture of the medial or posterior malleolus (bimalleolar fractures)
- Trimalleolar fractures
- Maisonneuve fractures



Internal fixation of a distal fibula fracture Weber type B

Ankle Sprain

Etiology

- High ankle sprain (1–10% of cases): syndesmosis injury

- Low ankle sprain (90% of cases): ATFL (=*anterior talofibular ligament*) and CFL (=*calcaneofibular ligament*), rarely of PTFL (=*posterior talofibular ligament*) injury

Lateral ligaments of the ankle

Clinical Presentation

Pain with weight bearing, swelling and ecchymosis, recurrent instability

Diagnosis

- Clinical examination
  - Focal tenderness (lateral and medial ligaments, anterior syndesmosis)
    Tenderness at the proximal fibula is highly suspicious for a Maisonneuve fracture
  - Peroneal tendons: swelling, tenderness and stress tests
    Tenderness at the base of the 5th metatarsal
  - Anterior drawer test (for ATFL): push the hindfoot (*slightly plantarflexed*) forward and look for anterior displacement of the talus relative to the tibia—compare to the contralateral side
  - Talar tilt test (for CFL): ankle inversion with the ankle dorsiflexed—compare to the contralateral side (no firm stop, extended path)
  - Squeeze test (for syndesmosis): compression of the tibia and fibula in the middle of the lower leg → pain at the level of the anterior syndesmosis
  - Frick-Test: sensitive test. Hold affected tibia with one hand while applying external rotation onto the foot with the contralateral hand. Pain in the syndesmotic area (anterolateral ankle) is indicative for syndesmotic sprain
- Radiographic examination
  - X-ray: Exclude fracture and syndesmotic instability
  - MRI: acutely for suspected syndesmotic injury or delayed for persistent pain and clinical signs of peroneal tendon injury or osteochondral lesion

Therapy

Conservative

Early functional rehabilitation for grade I and II sprains. Grade III sprains (severe pain with weight bearing) may benefit from a short period (7–10 days) of immobilization (with AFO brace or soft cast). Weight bearing as tolerated, physical therapy for proprioceptive training; follow up with primary care physician

- In case of clinical or radiographic signs of a syndesmotic injury: immobilization in Vacoped or lower leg cast, timely MRI
- Treatment of syndesmotic injuries is controversial, but may be managed nonoperatively (immobilization in a lower leg cast with limited weight bearing for 6 weeks) if there is no evidence of radiographic instability or associated fracture

Operative

- For recurrent sprains due to persistent instability and failed neuromuscular physical therapy
  - Broström procedure: anatomical shortening of the ATFL and CFL
  - Gould modification: Broström reinforced with inferior extensor retinaculum and distal peroneal periosteum
  - Anatomic ligament reconstruction with tendon allograft (for revision cases)
- In case of radiographic instability of the syndesmosis or in association with a fracture
  - Dynamic (e.g., Tight Rope) or static (position screw) syndesmosis stabilization ± fracture fixation

Associated Injuries

Osteochondral Lesion of the Talus

- Etiology: mostly post-traumatic versus repetitive stress causing vascular compromise in osteochondrosis dissecans (OCD) cases
- Incidence: in up to 70% of ankle sprains
- Anatomical location:
  - Medial talar dome: usually no history of trauma, and usually larger and deeper than lateral lesions
  - Lateral talar dome: more superficial and smaller, lower incidence of spontaneous healing
- Diagnosis: MRI or CT arthrography (for more precise evaluation of the cartilage cap)

  - For OCD lesions: assess if fragment is unstable (high signal line separating the fragment from the bone)
- Therapy:
  - *Conservative*

    Initial treatment attempt should always be non-operative (especially for incidental findings and asymptomatic lesions)

    (i) In case of OCD: nonoperative in open physis and stable lesions

    Osteochondral lesions that successfully undergo an initial non-operative treatment period have minimal long-term symptoms, a low failure rate, and no relevant ankle osteoarthritis

    The nonoperative approach is supported by the fact that no clear recommendation for a specific surgical procedure can be made based on the current evidence
  - *Operative*

    Depending on symptoms and size of the lesion

    There are several techniques with similar results: microfracture, autologous chondrocyte implantation, mosaicplasty, AMIC plastic

    (i) In case of OCD: depending on whether the lesion is stable (subchondral drilling), unstable and salvageable (ORIF) or unstable and not unsalvageable (AMIC, OAT, …)

    (ii) Maximum exposure of the medial talar dome can be achieved by performing an additional malleolar osteotomy. Laterally, ATFL dissection and distraction is required for proper access

Peroneal Tendon Tears

- Etiology: forced dorsiflexion of the inverted foot puts stress on the contracted peroneal muscles, leading to a tear of the superior peroneal retinaculum (SPR) (➔ tendon dislocation may occur). Associated with pes cavovarus deformity
- Incidence: Peroneal tenosynovitis in 77%, peroneus brevis tear in 25% in chronic ankle instability
- Diagnosis: X-ray ("fleck sign" = cortical avulsion of the SPR), MRI
- Therapy:
  - *Conservative*

    Temporary immobilization for tenosynovitis and small tears
  - *Operative*

    < 50% rupture: Debridement and tubularizing suture (restores biomechanical properties whereas 50% resection results in elongation and loss of tendon stiffness)

    > 50% rupture: Peroneus longus to brevis transfer

    Tendon dislocation: SPR refixation <u>without</u> groove deepening

Pediatric Orthopedics

9

Thomas Dreher, Arend Nieuwland, Kerstin Schneider, and Sandro Canonica

Anatomy of the Pediatric Hip

- Acetabulum with triradiate cartilage (*Y-shaped junctional epiphyseal plate of ilium, ischium and pubis)*. Mid-adolescent closure (15–16 years in males, 13–14 years in females)
- Arterial blood supply: A. femoralis → A. profunda femoris → A. circumflexa femoris medialis (MFCA) and lateralis (LFCA)
  - A. ligamentum capitis femoris (**intrauterine importance**, subsides at 4y)
  - **medial circumflex artery** (**main supply** after subsidence of a. ligamentum capitis femoris through **posteroinferior and posterosuperior branches**)

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Developmental Dysplasia of the Hip (DDH)

Epidemiology and Etiology

Most common congenital orthopedic anomaly. Female to male ratio (4:1). Ethnic differences: African Americans with low incidence (0%) compared to American Indians/Sami with high incidence (5%). Common risk factors: Breech position, oligohydramnios, firstborn, in the context of syndromic diseases, general joint hypermobility, positive family history.

Diagnosis

- Clinical examination

Barlow's sign	Provocation of a hip (sub-)luxation. Positive if dislocatable, suggestive if subluxable, negative if stable
Ortolani's sign	"Click" during reduction maneuver. Positive if hip can be reduced (typically combined flexion and abduction)
Galeazzi's sign	Appearance of unilateral shortened femur due to hip dislocation. Positioning: hips in 90° flexion, knees in 90° flexion
Classification	Spectrum from subluxable (Barlow suggestive) to dislocatable (Barlow positiv) to dislocated (Ortolani positive/negative)
Abduction	Inhibited on the affected side
Inspection	Asymmetric thigh skin folds

- Radiographic examination

0–3 months: ultrasound examination according to Graf method gold standard. Screening examination at 4–6 weeks, high risk patients sooner. Types: I, IIa/b/c, III, IV.

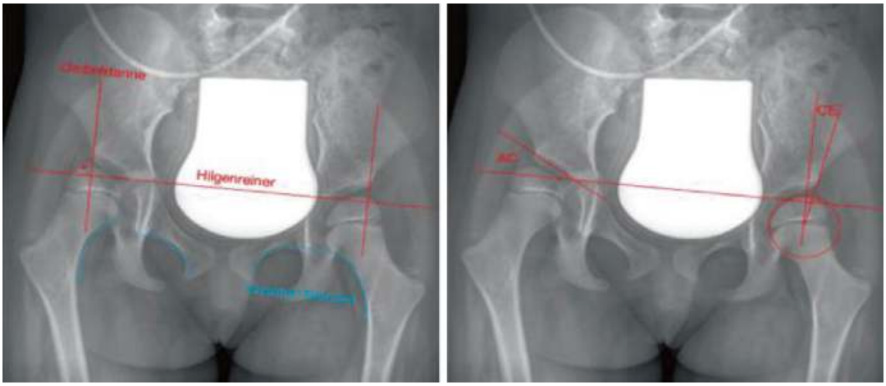
- Assessment of the acetabulum, osseous and cartilaginous acetabular rim, labrum and position of the femoral head in the coronal plane
- Defined as normal/mature, delayed/immature (subdivision: age appropriate vs. inappropriate (age 6–12 weeks)), subluxated, dislocated hip

>4 months: X-ray (AP pelvis)

- Hilgenreiner's line, Perkins-Ombrédanne's line, Shenton-Ménard's line
- Acetabular roof angle according to Hilgenreiner (AC-Index)
- Lateral center-edge angle according to Wiberg (LCE angle)
- Migration Index (Reimers)

MRI

- Gold standard for evaluation after closed reduction and spica casting



Hip development assessment on anteroposterior pelvis X-ray images

Treatment

Goals: Bring immature hip to maturity. Reduce and retain dislocated hips

- **Maturation:** Depending on the ultrasound classification. Pavlik harness (type II-III), Tübinger hip abduction brace (type II-III), spica casting (+/− open reduction), (type III-IV)
- **Dislocated hip (type IV):** Closed/open reduction and retention in spica cast. Overhead extension as a possible addition
- **If closed reduction fails:** Open reduction, because the longer a dislocation exists, the more secondary changes occur that prevent a reduction (e.g. *capsule/labrum interposition, fatty hypertrophied lig. Capitis femoris, contracted lig. Transversum etc.*)

Residual and/or Unrecognized DDH

- **>2nd year of life:** Hip joint reconstruction
- Bony corrections of the pelvis and proximal femur with the goal of (a) reducing the femoral head and (b) increasing congruence between the femoral head and the acetabulum.

Pelvis and femoral osteotomies for DDH

Age	Configuration	Pelvis	Femur[a]
2–8 years	Congruent joint. Inclination too high. Insufficient coverage	Salter osteotomy	+/− shortening osteotomy
2–8 years	Congruent joint. Flattened acetabulum	Pemberton or Dega osteotomy	+/− femoral varus derotational shortening osteotomy
> 8 years	Congruent joint. Spherical femoral head	Triple innominate osteotomy (Tönnis), (*PAO* (*Ganz*))	+/− femoral varus derotational shortening osteotomy
> 8 years	Congruent joint. Flattened acetabulum, triradiate cartilage open	Pemberton or Dega osteotomy	+/− femoral varus derotational shortening osteotomy
> 14 years	Congruent joint. No degeneration	*Triple innominate osteotomy (Tönnis),* (PAO (Ganz))	+/− femoral varus derotational shortening osteotomy
> 14 years	Congruent joint. Degenerative changes	Salvage osteotomies (Shelf, Chiari). THA	

[a]often coxa valga et antetorta is present simultaneously with hip dysplasia

Complications

Conservative/early operative treatment

- inferior hip dislocation
- transient femoral nerve palsy
- AVN

Reconstructive operative procedures

- Growth disturbance at the acetabulum with triradiate cartilage (*treatment: acetabuloplasty*)
- Residual deformities
- Non-unions (*treatment: corrective osteotomies*)
- Neurovascular lesions

Morbus Perthes/Legg-Calve-Perthes Disease

Definition

Idiopathic avascular necrosis of the femoral head *and proximal femoral epiphysis* in an otherwise healthy child. The prognosis is generally good if the course of the disease is favorable.

Epidemiology

Male to female ratio 5:1, peak 4–8 years.

Etiology

Circulatory disorder (*either arterial (under perfusion) or venous (disruption of outflow*). Possibly dietary; genetic (35-fold *increased risk in first-degree relatives*).

Clinical Presentation

- Hip, groin, thigh pain and/or (CAVE) knee pain
- Painless/antalgic limp
- Loss of ROM (internal rotation, abduction)
- Leg length discrepancy (late presentation)

Diagnosis

- Radiographic examination
- Ultrasound: evidence of joint effusion
- X-ray: AP pelvis and Lauenstein: early signs of joint space widening
  - Modified Waldenström classification describes the various stages
  - Lateral pillar (Herring) classification is most predictive of prognosis
  - Caterall classification describes localization and extent of necrosis
  - Stulberg classification describes definitive head configuration and prognosis
- MRI: enables early diagnosis with inconspicuous X-ray image, not indicated as no further consequences

Modified Elizabethtown—Waldenström Classification

IA	Initial stage	Avascular phase, joint space widening
IB	Initial stage	Sclerosis of epiphysis
IIA	Fragmentation stage	Start fragmentation of epiphysis. Crescent sign
IIB	Fragmentation stage	Advanced fragmentation, bone resorption, patchy density
IIIA	Reossification stage	New bone appearing lateral to fragmented epiphysis
IIIB	Reossification stage	New coverage more than one third of width epiphysis
IV	Healing/remodeling stage	Complete healing, no radiographically identifiable avascular bone. Remodelling occurs until skeletal maturity

[a]The final stage can also include the development of a fixed head deformity

Catterall Classification of Localization & Extent of Femoral Head Necrosis

Stage 1	1/4 of the head, anterior epiphysis only
Stage 2	1/3 of the head, anterior and central epiphysis
Stage 3	3/4 of the head, only most medial and lateral epiphysis not involved
Stage 4	Involvement of entire head

Modified Lateral Pillar (Herring) Classification

Stage A	Lateral column without height reduction and without density change	Consistently good prognosis
Stage B	Height maintained > 50%	Good prognosis
Stage B/C	Height maintained > 50%, narrow column (2-3 cm)/poor ossification/depressed relative to central pillar	Intermediate prognosis
Stage C	Height maintained ≤50%	Poor prognosis

Modified Stulberg Classification (Higher Inter-rater Reliability Than Original Classification)

Group A	Spherical femoral head (Stulberg I + II)	Good life time prognosis
Group B	Ovoid femoral head (Stulberg III)	Average prognosis
Group C	Flat femoral head (Stulberg IV + V)	Poor prognosis

"Head-at-Risk" Signs According to Catterall

- Lateralization/subluxation of femoral head
- Metaphyseal involvement
- Lateral calcification
- Horizontal proximal femoral physis

Risk Factors for Unfavorable Course

- Onset after 6 years of age
- Female sex
- Severely restricted hip joint mobility: especially abduction
- Catterall stage 3 + 4
- Lateral Pillar (Herring) classification types B/C + C
- Presence of Catterall "head at risk" signs

Intraoperative hip orthography to assess femoral head containment and congruency, as well as the 'hinge abduction' phenomenon

Hinge abduction: lateral extrusion of proximal femoral physis creates a hinge effect. Clinically: abduction of the hip causes the femoral head to be decentralized over the lateral acetabular rim

- The lever effect, with the acetabular rim as the fulcrum for the femoral head, can be demonstrated by intraoperative arthrography
- If demonstrated, containment surgery by femoral varus osteotomy is no longer appropriate. Acetabular rotations (triple osteotomy) and possibly valgus femoral osteotomies are used

Treatment

Radiological follow up with every 3–6 months.

Conservative treatment	Maintain range of motion through physiotherapy, possibly even certain sports (swimming, *cycling*) Full weight bearing allowed
Contraindicated	Bed rest and partial weight bearing Jumping, contact sports
Exception: Symptomatic	Severely symptomatic weight bearing as tolerated, analgesia
Surgical treatment	Pelvic and/or femoral osteotomies, shelf osteotomy, hip arthroscopy for secondary FAI

1.	no hinge abduction, good abduction = proximal femur varus osteotomy, +/− triple osteotomy
2.	Hinge abduction, but centered in adduction = triple innominate osteotomy
3.	End stage: Stulberg group B or C = offset correction, +/− triple innominate osteotomy/PAO, osteochondroplasty in case of coxa magna (through surgical hip dislocation)
4.	End stage: Stulberg group C = trochanter distalization, +/− femoral neck lengthening, +/− osteochondroplasty in case of coxa magna (through surgical hip dislocation)

Prognosis

Generally good. In case of unfavorable development: femoral head deformity (especially coxa magna/breva), increased risk of osteoarthritis in relatively young adults.

Slipped Capital Femoral Epiphysis (SCFE)

Definition

Slippage of the metaphysis against the epiphysis in the proximal femoral physis.

Epidemiology

Male to female ratio 1.5–2:1; peak age 12–16 years or 10–14 years (*during growth spurt*), incidence 10:100.000.

Risk Factors

Obesity, acetabular retroversion, femoral retroversion.

Classifications

- Temporal classification
  - Acute: Symptoms <3 weeks
  - Chronic: Symptoms >3 weeks
  - Acute on chronic: most common
- Loder classification
  - Stable: Full / partial weight bearing possible
  - Unstable: No weight bearing possible (acute/acute on chronic)

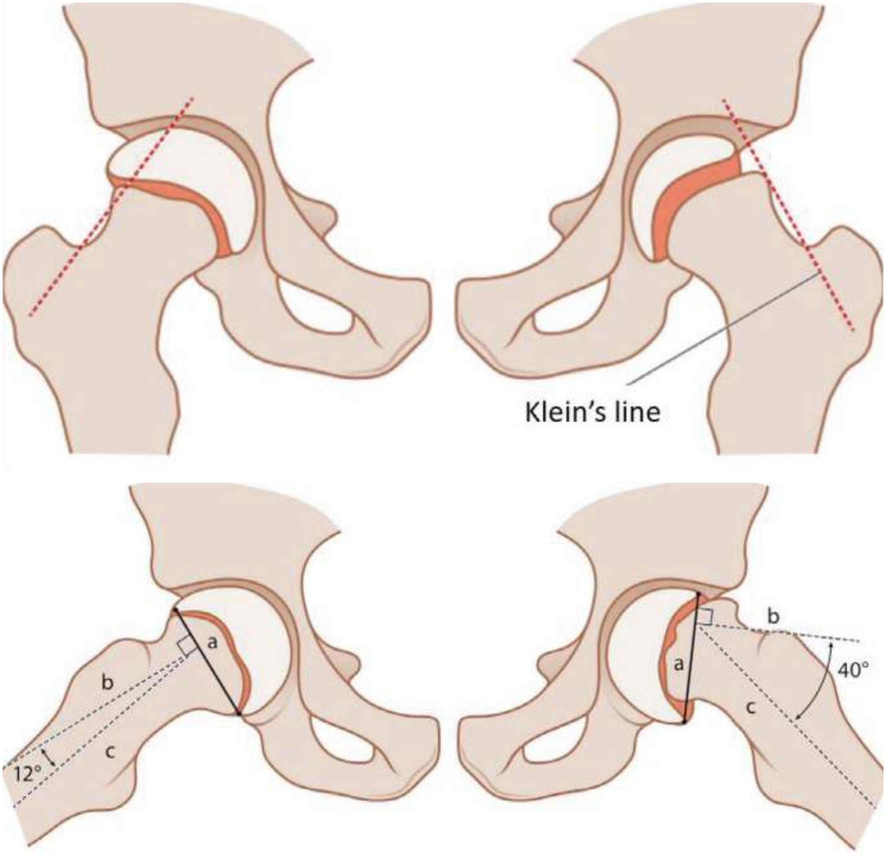
Clinical Presentation

Hip/groin pain, knee pain in up to half of all cases (R. cutaneus n. obturatorii!)

Diagnosis

- Clinical examination
  - Drehmann's sign: Hip flexion limited in neutral position, further hip flexion possible in external rotation position
  - Limping

- Radiographic examination
  - AP pelvis and bilateral (!) Lauenstein (frog leg) views
  - Southwick Slip Angle, Klein's line



Klein's line (AP pelvis X-ray) and the Southwick Slip Angle (bilateral frog leg X-rays)

- Lab studies
  - Not routinely indicated. If patient <10y, weight < 50th percentile, or known endocrine disorder, further analysis required (TSH, fT4, serum creatinine, blood urea nitrogen (BUN))

Treatment

Southwick slip angle <30°	Percutaneous in situ screw fixation (1 vs. 2 screws) Prophylactic ipsilateral screw fixation for open triradiate cartilage, age < 14 years, endocrine disorder
Southwick slip angle >30°	Open reduction of the epiphysis onto the metaphysis & fixation by *modified Dunn procedure*

Complications

Avascular necrosis	Especially in unstable slips. Follow-up for at least 2 years
Contralateral SCFE	Depending on risk factors, simultaneous prophylactic screw fixation
Residual deformity	Is considered a pre-arthrotic deformity → increased risk of osteoarthritis in relatively young adulthood. Discuss hip arthroscopy vs. femoral osteotomy

Transient Synovitis of the Hip (Coxitis Fugax)

Definition

Inflammation of the synovial membrane of the hip joint

- typically as an accompanying symptom during/after viral infection (*upper respiratory tract or gastrointestinal tract*)
- spontaneously resolving, benign condition
- DD septic arthritis of the hip (potentially life and joint threatening condition)

Epidemiology

Transient synovitis of the hip: most common hip disease during growth; peak at 6 (4–8) years.

Clinical Presentation

Hip/groin pain, knee pain (!)

Limping, painful restriction of movement (reduction of internal rotation).

- Typically, "healthy", happy child with transient synovitis
- DD septic arthritis of the hip (potentially life and joint threatening condition)

Diagnosis

Clinical examination and subsequent:

- Blood sample: CRP (<15–20 = septic arthritis less likely), WBC
- Ultrasound: no joint effusion = septic arthritis less likely
- X-ray: pelvis AP, frog leg (primarily to rule out other possible diagnoses such as Perthes disease or SCFE)
- Hip joint aspiration: macroscopic aspect (*clear, purulent, bloody?*), cell count, microbiological assessment

Treatment

Transient Synovitis

- Weight bearing as tolerated, NSAID, observation
- Reduce activities for 24 hours
- Usually symptoms last <1 week, if not: reassess!

Septic Arthritis

- Irrigation and debridement of the joint (arthroscopic or open, possibly repeat surgery depending on the pathogen), antibiotic therapy

Complications and Prognosis

Transient Synovitis

No long-term effects or damage, recurrence rate up to 20%.

Septic Arthritis

- if diagnosed and treated in time, good prognosis with complete recovery
- if diagnosis/treatment is delayed and/or difficult to treat: sepsis, femoral head deformity/destruction, leg length discrepancy

Congenital Clubfoot

Syn	Pes equinovarus (*however, not all components of the malposition are mentioned*)

Epidemiology

Incidence 1:1000, some population groups 1:200 (Polynesians).

Boys > Girls (2:1); 50% bilateral clubfoot.

Etiology

The etiology remains unclear. Most cases are idiopathic.

The following conditions are associated with the development of a congenital clubfoot:

- intrauterine position anomalies
- Amniotic band syndrome
- Oligohydramnios
- Spina bifida
- Neural tube defects

- Aminopterin syndrome
- Arthrogryposis multiplex congenita

Clinical Presentation

- 5 components of deformity: **CAVES** (*Mnemo*)
  - **c** avus = high arch
  - **a** dductus = forefoot adduction
  - **v** arus: hindfoot varus
  - **e** quinus
  - s supinatus: supination of the entire foot, <u>but</u> pronation of the forefoot compared to the hindfoot
- Assessment of rigidity: Can the clubfoot be redressed?
  - redressable: positional clubfoot
  - non-redressable: clubfoot
- Overall status
  - Hip → hip dysplasia associated and therefore more common than in children without clubfoot → ultrasound (!)
  - Spine → signs of spina bifida?

Diagnosis

Clinical diagnosis (X-ray [only for recurrent clubfoot]):

- DP projection
  - Talocalcaneal angle < 20° (n*ormal 20–40*°) = Rearfoot varus
  - Talus-Metatarsal-I angle > 20° (*normal 0–20*°) = Forefoot adduction
- Lateral projection:
  - Talocalcaneal angle < 35° (*normal 35–50*°) = Equinus and rearfoot varus
- Classification according to Catterall/Pirani or Dimeglio

Dorsoplantar view: decreased talocalcaneal angle (rearfoot varus) and increased talus-Metatarsal-I angle (forefoot adduction)

Lateral view: 4 signs of rearfoot varus and external rotation of the ankle and hindfoot: sinus tarsi see-through, double talar dome, shortened appearing calcaneus and posterior position of the fibula compared to the tibia

Differential Diagnosis

Most important differential diagnosis: positional clubfoot (may have all components of clubfoot but is flexible) and metatarsus adductus (has only the adductus component, may be fixed). Clinical examination of ankle range of motion, especially dorsiflexion, and the ability to reduce the hindfoot in valgus is essential to differentiate from true clubfoot.

Therapy

The Ponseti Method has led to a significant improvement in long-term results and a reduction in the number of corrective operations required.

Chronological order:

- Serial manipulation and casting with gradual correction of the deformity for approximately 6 weeks
  - Start as early as possible: 7–10 days after birth
  - Do not touch the heel during manipulation (the calcaneal malposition is corrected passively with abduction)
    1. Correction of cavus foot by elevating the first ray
    2. Correction of adductus and varus
    3. Correction of equinus (complete correction usually only possible with percutaneous tenotomy of the Achilles tendon)
    4. Plaster immobilization for 3 weeks in approx. 10–15° dorsiflexion and 60° abduction
    5. Relapse prevention: Night-time abduction brace until 4 years of age

Alternative Treatment Options

Dynamic therapy, for example according to Bonnet-Dimeglio, is a real therapeutic option to the Ponseti method. They are overall more complex, but offer more flexibility, especially in the case of atypical clubfeet or associated deformities.

CAVEAT
- Positional clubfoot posture is not a true deformity but a temporary deformity caused by intrauterine position
- Differentiation from “true” clubfoot: Examination shows a descending and palpable calcaneus in the heel pad and free dorsiflexion of the ankle

Flexible Flatfoot

Definition

Hindfoot valgus, flattening/loss of medial longitudinal arch of the foot, and forefoot abduction.

By 6–7 years of age, the 'normal' foot shape develops and the fat pad under the medial longitudinal arch disappears. By then, flat feet are most often physiological.

Clinical Presentation

Usually asymptomatic.

Differential Diagnoses

- **Rigid** flatfoot due to coalition of the tarsal bones (*fibrous or bony fusion/connection of tarsal joint*s)
- Recognizable by the following clinical tests:
  - Restricted subtalar joint range of motion (*pro−/supination*)
  - No varus correction of the hindfoot, no accentuation/development of the longitudinal arch when standing on the thoes
  - Passive extension of the big toe (windlass Mechanism) does not result in the formation of the longitudinal arch
  - In severe cases, progression and discomfort, surgical treatment: resection (*and interposition*)
- Shortening of calf muscles (*positive Silfverskjöld test*): Treatment according to severity—stretching, plaster compression, lengthening surgery
- Talus verticalis
- Os tibiale externum
- collagen-related diseases: Marfan, Ehlers-Danlos
- Neuromuscular disease (*cerebral palsy*)
- Muscular dystrophy (*Gowers sign*)
- Osteonecrosis

Diagnosis

- Clinical diagnosis
- X-rays in case of pain and planning of surgery

Prognosis

- Often normalizes in the first 7 years of life
- If persistent, usually good prognosis, lifelong asymptomatic course possible. **Not** a pre-arthrotic deformity per se

Therapy

Conservative

- Barefoot walking to strengthen the foot muscles, toe walking, calf muscle stretching
- Insoles rarely necessary, in case of complaints, increasing talonavicular instability (full foot length, medial arch support, hindfoot varus force)

Operative only in cases of persistent complaints, in particular coalition or shortened calf muscles and/or high tibial torsion.

Toe Walking

Definition

Child walks on toes instead of heel-toe walking

- Up to 2 years: **physiological**
  - To gain stability
  - Coordination of lifting/pushing-off the foot in the swing /stance phase needs to be learnt
- Over 2 years: **"habitual"** toe Walking
  - Muscle tone normal, no muscle hypo−/hypertrophy and reflexes normal
  - Patient is consciously able to place the entire sole of the foot on the ground
  - Secondary shortening of M. triceps surae possible
  - Temporarily observed in approximately 15% of children
  - Cause unclear—idiopathic
- Neurogenic/neuromuscular toe walking;
  - To be considered in case of abnormalities in the examination
  - Associated conditions:
    - (i) Peripheral paresis
    - (ii) Infantile cerebral palsy
    - (iii) Tethered cord syndrome (*DD spinal cord tumor*)
    - (iv) Spina bifida
    - (v) Diastematomyelia
    - (vi) Polyneuropathy (e.g. *Guillain-Barré syndrome*)
    - (vii) Charcot-Marie-Tooth (hereditary motor sensory neuropathy [HMSN] Type I)
    - (viii) Congenital muscular dystrophy
    - (ix) Duchenne muscular dystrophy

- Neuropsychiatric
  - High prevalence in developmental delay or neuropsychiatric disease—up to 40%
  - Examples: global developmental delay, severe speech disorder (40%), intellectual disability (36%), learning disability (20%), low educational level of parents, autism (63%), schizophrenia

Diagnosis

Clinical diagnosis. Further investigations are indicated for: persistence, late onset, unilateral, abnormal reflex status, muscle weakness, abnormal muscle tone, muscle hypotrophy, therapy-resistant gait pattern

- Orthopedic: X-ray, gait analysis
- Neurological: MRI cerebral/spinal, creatine kinase, muscle biopsy (*dystrophies and myopathies*), EMG

Therapy

For idiopathic/habitual toe walking:

- up to 80% with spontaneous heel-to-toe walking as growth continues

Conservative
- wait and see, follow-up every 6 months
- Physiotherapy (stretching, strengthening, gait training)
- Carbon soles, possibly pyramid inserts (*deconditioning*)
- Lower leg cast (deconditioning)
- Lower leg cast with forced dorsiflexion, changed weekly, for 4–6 weeks (stretching)
- possibly Botox

Operative only with insufficient correction/rigidity.

> **CAUTION**
> Toe walking is physiological up to 2 years!

Leg Axis Misalignment and Development

CAUTION
only frontal plane discussed.

Physiological Development

- Varus at birth, more crus varum than genu varum (on average 15°)
- Onset of walking → neutral position; then a valgus axis develops (of about 10°), corresponding to an intermalleolar distance of 2–4 cm
- Corrects itself by the age of 10 to a physiological valgus of 5–7° in girls and straight legs in boys

Definition

1. **Genu varum** = Varus deviation of the lower leg compared to the upper leg
2. **Genu valgum** = Knock-knee: Valgus deviation of the lower leg compared to the upper leg
3. **Crus varum** = Outward curvature of the lower leg

Diagnosis

- Clinical examination
  - Measure intermalleolar distance
- Radiographic examination
  - Requirements for long leg standing view:
    - Extended knee joints
    - Kneecaps must face forward
- Position of the feet is irrelevant
  - Mechanical axis = Mikulicz line
    - Line connecting the center of the femoral head and center of the ankle joint should run 4 ± 2 mm medial to the intercondylar eminence
  - Mechanical lateral distal femur angle:
    Angle between a line connecting the center of the femoral head and the apex of the intercondylar fossa of the femur and a line along distal border of the femoral condyles; norm value: 87° (85–90°)
    (anatomical—Angle between a line centered on the diaphysis of the femur and a line along the distal border of the femoral condyles; norm value: 81° (79–83°))
  - Proximal medial tibia angle (mechanical = anatomical)
    Angle between a line through center of the tibial plateau and the center of the ankle joint and a line on tibial plateau; norm 87° (85–90°)

Therapy

- Permanent epiphysiodesis:
  - Growth guidance by final closure of the growth plate (drilling). Timing is critical (too early: overcorrection, too late: failure to achieve complete correction) ➔ Determine skeletal maturity bone age with hand radiograph, calculate optimal timing through ‘Multiplier method’. Advantage: one-time surgery with small skin incision, no hardware removal necessary
- Temporary epiphysiodesis:
  - Growth guidance with temporary blocking of the growth plate with an implant (e.g. 8-plate). Once the desired correction has been achieved, growth potential remains and risk factors for recurrence are present, partial implant removal of the metaphyseal screw (➔ “sleeper plate”) is performed with the option of simple later reactivation. Once the growth plate is closed, the implant can be removed completely
- Osteotomy:
  - In case of closed growth plates (*completed growth*) and for complex, multiplanar deformities affecting more than just the frontal plane, growth guidance is no longer an option and corrective osteotomies are the therapy of choice

Technical Orthopedics

10

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Basics

Terms and Definitions

TO = Technical Orthopedics (*the term "Prosthetics & Orthotics" is more commonly used in English speaking countries*) is a specialty of orthopedics, which includes both surgical and conservative measures:

Surgical TO	Amputation surgery, amputation stump corrections, surgery of the diabetic foot
Conservative TO	Indication, prescription and control of orthopedic aids for compensation, relief or prevention of functional restrictions of the musculoskeletal system or to avoid the progression of a deformity during growth (see scoliosis)

Orthopedic Technology & Rehab Technology

- Prostheses, orthoses (splints), orthoprotheses, bandages, compression treatment

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- Wheelchair provision, seat shells, lying shells, walking aids, standing aids

Orthopedic Shoe Technology

- Insoles, orthopedic shoe modifications, orthopedic special, serial and custom made shoes, internal foot orthosis (e.g. “Arizona brace”)

Amputation, Exarticulation

- Amputation = Circular removal of harmful or useless body parts; Synonym: *ablatio*. More precisely, the removal of a limb through the bone, as opposed to removal through a joint (= *exarticulation*)

Prosthesis

- Replacement of a limb = exoprosthesis
- Joint replacement = endoprosthesis
- Prostheses, which are anchored in the bone and perforate the skin via an intermediate piece, are referred to as endo-exoprostheses (= osseointegration)

Orthosis

- Brace for improving the function of an existing, but damaged limb (*e.g. due to joint instability*)

Orthoprosthesis

- Combination (hybrid) of orthosis and prosthesis, e.g. in limb malformation with massive shortening

Phantom

- **Phantom sensation:** non-painful sensation in the amputated limb. Normal and not requiring therapy
- **Phantom pain:** Pain in the amputated part of the limb (= *outside the body*)
- **Phantom limb:** “real” sensory replica of the amputated limb (Beware of fall risk: Patient has, for example, the feeling that the lower leg and foot are still present and wants to load the amputated limb when getting up from bed)

- **Telescoping:** Retraction of the phantom limb towards the stump, i.e., for example, after a lower leg amputation, the foot is felt in the middle of the lower leg (*in 30–59% of phantom sensations*)

Note

- Orthopedic aids are only useful if they provide the patient with an increase in ability and a better quality of life or protect him from subsequent damage (**e.g.** ***diabetic foot ulcer***). Especially in children, careful indications are necessary, because sometimes, e.g. after amputations of the upper limb, the ***"stump is the best prosthesis"***
- In Switzerland, orthopedic aids are usually **only** reimbursed by the federal disability insurance, if they are needed permanently (***at least for 1 year***)

Amputations

Amputations of the Lower Limb

Etiology (based on data from the Federal Statistical Office from 2005 to 2015).

Cause	Percentage
Peripheral arterial occlusive disease (PAOD), Diabetes foot disease and infections	85–90%
Trauma[a]	3–4%
Deformities, tumors[b], Varia	5–10%

[a]In young patients, trauma predominates
[b]Tumors are primarily responsible for very high (*proximal*) amputations, e.g. hip/pelvis

Prognostic life expectancy after amputation in PAD (as arteriosclerosis is a systemic disease and also affects the heart, brain and kidneys):

- 1 year after **upper** thigh amputation → 50% survivors
- 5 years after **lower** leg amputation → 50% survivors

Mobility Classes

The mobility class is a prerequisite for the choice of prosthetic components: the higher the mobility class, the more powerful and usually also more expensive are the chosen prosthetic components.

Class 0	**Non-ambulatory:** *Prosthesis for transfer, cosmetic replacement*
Class 1	**Indoor walker:** *Can walk slowly on flat ground for a short time with the prosthesis*
Class 2	**Limited outdoor walker:** *Can walk for a limited time with prosthesis and overcome curbs as well as individual steps*

Class 3	**Unrestricted outdoor walker:** *Can walk with prosthesis even on uneven ground and in open terrain, and can perform a job and therapy*
Class 4	**Unrestricted outdoor walker with particularly high demands:** *Can walk unrestrictedly everywhere, where high shock, rotational and tensile forces can occur, e.g. in sports or demanding physical activity*

Prognosis Regarding Walking Ability with Prosthesis

For achieving walking ability, in addition to comorbidities and walking ability before amputation, primarily the **Level of Amputation** plays a significant role:

Below-knee amputation	70–80% become ambulatory
Knee disarticulation	60–70% become ambulatory
Above-knee amputation	**20–30%** become ambulatory

After bilateral above-knee amputation, usually only therapeutic and short-term walking is possible, and the patients use a wheelchair in everyday life.

Note
- Because of the unfavorable prognosis regarding the expected mobility, the knee joint should be preserved whenever possible!

Load Capacity of the Stump and Energy Consumption

End-bearing:

- An amputation stump is called end-bearing when the stump end can be loaded with full body weight while neither support nor weight reduction further proximal is necessary.
- This is usually the case with forefoot and hindfoot amputations as well as knee and hip disarticulations
- Below-knee and above-knee amputations are **never** end-bearing (exception: in case of transplantation of the calcaneum to the end of the tibia or the femur)

Increased energy expenditure when walking with the prosthesis. Vascular amputations have higher energy expenditure than traumatic amputations:

Hindfoot amputations	+10–25%
Below-knee amputations	+25–50%
Knee and hip disarticulations	+75–100%
Above-knee amputations	+75–100%
Bilateral below-knee amputation	+40–50%
Below-knee plus above-knee amputation	118%
Bilateral above-knee amputation	> 200%

Choice of Amputation Level

- Difficult and requires experience, because the amputation stump should be pain-free, prosthetically manageable, and as durable as possible. Maximum stump length with poor stump quality is of little use to the patient, he wants to be unrestricted and as much as possible able to walk without walking aids
- Prerequisites for good stump quality: sufficient circulation, even under stress, good soft tissue coverage with sensitive and resilient skin, no or only minor misalignments of the adjacent joints (*hip or knee flexion contractures, equinus and varus deformities in the rear foot*), no paralysis

Individual Considerations of Different Amputation Heights

- **Thigh amputation**

With an optimal amputation height 10–15 cm above the knee joint gap (reason: space for the nowadays mostly electronic knee joint) the amputation causes a stump that deviates in flexion and abduction by removing the hip adductors and the ischial muscles. Using an ***Adductor magnus myodesis*** according to Gottschalk (lateral transosseous refixation of the adductor magnus on the femur under optimal pretension of the muscle) the femur can be balanced and the misalignment prevented.

- **Lower leg amputation**

In advanced PAD, the M. soleus should be completely resected due to the risk of necrosis. The long posterior flap in the classic Burgess technique is not supplied by any vessel that passes the M. soleus. The blood supply derives from the Aa. suralis and saphena.

- **Ertl-Dederich amputation**

Special type of lower leg amputation with the creation of a synostosis between the tibia and fibula stump for better load capacity of the amputation stump. Originally described was a corticoperiosteal flap, now fibula strut grafts are also used. Used mainly in younger healthy patients who were amputated due to trauma (especially U.S. military personnel). Should not be used in infection or in PAD. The postulated effect of the Ertl-Dederich amputation on functional outcome has yet to be proven in larger study populations; the RCT "Transtibial amputation outcomes study (TAOS) study", published in 2024, demonstrated more complications in the synostosis group compared to traditional below the knee amputations and found insufficient evidence that the synostosis group had a better functional outcome.

- **Hindfoot amputations (Syme/Spitzy-Pirogoff)**

Allow short, prosthesis-free walking distances (e.g. nocturnal toilet visits). Should only be considered with proven patency of the A. tibialis posterior. The Syme technique also requires an intact and stable calcaneal fat pad.

- **Chopart amputation**

Exarticulation in the Chopart joint with rounding of the osseous contour. Typical misalignment due to unopposed pull of the Achilles tendon is the equinus deformity. Prevented by Achilles tendon lengthening and transfer of the tibialis anterior tendon into the talus neck or arthrodesis of the tibiotalar joint. A tendon refixation should be protected for 4–6 weeks in a lower leg cast.

- **Lisfranc amputation**

Exarticulation in the Lisfranc joint with rounding of the osseous contour. Typical misalignment is the equinovarus deformity due to unopposed pull of the Achilles tendon and the tibialis posterior. The latter only applies if the peroneus brevis tendon insertion is resected during the amputation. By preserving the base of metatarsal V or by refixation of the peroneus brevis tendon into the cuboid, this part of the misalignment can be prevented. In addition, the Achilles tendon lengthening is needed. Any tendon refixation should be protected for 4–6 weeks in a lower leg cast.

Amputation Technique in PAD

Tourniquet	only in standby for PAD/Diabetes mellitus; endangers the wound healing
Exposure	As little tension or pressure on tissue as possible *sharp hooks, forceps*), fingers allowed
Stitches	Single button, little absorbable suture material in depth (*only ligatures, fascia*), sparing subcutaneous stitches to relieve the skin, skin suture not absorbable (*optimal adaptation*), no clamps
Nerves	Shorten, cut smoothly (*for thicker nerves possibly ligature because of vasa vasorum*)
Artery	Removal of vascular prostheses in the prosthesis contact area; crushing or endarterectomy in case of vascular sclerosis
Bone	Low-speed osteotomy under irrigation, rounding of osteotomies, but do not push off periosteum! (no sharp edges in loaded areas)
Muscles	Resection of poorly perfused musculature, which tends to develop thrombosis (*e.g. M. Soleus*)
Drainage	Easy-flow crosswise or thick Redon, deep in the vascular lodge; for 48 h or VAC (v*acuum assisted closure*)
Bandage	Diagonal (***not** circular!*), from dorsal (*posterior flap*) to ventral, without relevant tension, possibly cast as stump protection against early falls

Amputations of the Upper Extremities

Etiology

In contrast to the lower extremity, **trauma** is the most common cause of amputation (50–70%). PAD and diabetes account for only 5–10%.

Phantom and Phantom Limb Pain

Epidemiology

Incidence approx. 50% (*above knee* > *below knee* > *foot*); first manifestation 85–97% < 1 month//10% > 1 year.

Duration: 60% > 1 year, with 5–10% being therapy-resistant.

Risk Factors

Duration and intensity of pre-amputation pain.

Pathogenesis

Cortical reorganization after deafferentation.

Clinic

75% → minutes to days-long attacks (*weather, emotional stress, micturition, defecation, sexual activity*), flashing to continuous pain.

50% Phantom Limb Formation.

Therapy

Conservative

- Medication: Pregabalin
- Anesthesiological: Singular/repetitive/continuous blockades of the N. femoralis/ischiadicus; as well as Epi-/Peridural blockades
- Physical: Mirror therapy, graded motor imagery, stump activation, bandaging, massage, TENS (*transcutaneous electrical nerve stimulation*), prosthetic care. A newer therapeutic approach is augmented reality using a virtual limb (advanced mirror therapy)
- Acupuncture, alternative medicine

Surgical

- Targeted muscle re-innervation (TMR) transferring a transected nerve to a nearby vascularized motor branch. If a TMR is not possible, a regenerative peripheral nerve interface (RPNI) = implanting the divided end of a peripheral nerve into a free muscle graft can be used. If a TMR is used during the primary amputation procedure literature suggests less phantom limb (PLP) and residual limb pain (RLP); secondary TMR in stump revision surgeries seems to improve both PLP and RLP but non-responding is possible

Prosthetic Care

Principles

- Mostly modular design: i.e., only the shaft is made individually for the patient, which has contact with the stump, all other parts (*joints, prosthetic feet, tubes, adapters*) are serially manufactured finished products (= prosthetic components)
- The shaft must have contact everywhere with the stump (***no** cavities or total contact principle*), otherwise edema formation with development of a congestion dermatosis and thus lesions of the skin may occur
- Shaft techniques:
  - Soft socket technique: with supracondylar chamber
  - Liner technique, nowadays standard of care for below the knee amputations
  - Pull-in shaft technique: standard for the thigh, the stump is pulled into the shaft with a bandage or dressing aid and the hole is closed with a one-way valve

Soft socket technique with supracondylar chamber

Liner technique

Prosthetics for the Upper Extremity

General

- **Too high expectations** of function & cosmetics
- About 50% of forearm amputees regularly use a prosthesis but only 25% of upper arm amputees
- In the case of double amputation above the elbow, the patients are usually dependent on outside help

Orthotic Care

Classification of Orthoses

In the international classification of orthoses, the **included joints** or spinal sections are listed with their short designation.

Upper Extremity

- SEWHO/SEO/SO/EO/EWHO/WHO/HO;
- **S** houlder, **E** lbow, **W** rist, **H** and, **O** rthosis

Lower Extremity

- HKAFO/HKO/HO/KAFO/AFO/FO
- **H** ip, **K** nee, **A** nkle, **F** oot, **O** rthosis

Trunk and Neck Orthoses

- CTLSO/CO/TLSO/LSO/LO/SIO
- **C** ervical, **T** horacic, **L** umbar, **S** acral, **I** liac, **O** rthosis

Types of Orthoses

Orthosis	Splint for improving the function of a damaged limb
Static orthosis	For stabilization, support, immobilization, correction, bedding
Dynamic orthosis	For guided movement (*limited, unlimited*), for compensation of paralysis

Example: Quadriceps paresis (e.g., after poliomyelitis):

- Knee joint not stable in sagittal plane: Patient prophylactically shifts body's center of gravity in front of the knee joint axis → genu recurvatum (leads over time to an overstretching of the capsule ligament apparatus and thus to the genu recurvatum)
- Solution: Supply by stabilizing knee or thigh orthosis, depending on the severity of the paresis freely movable but retracted orthosis joint, rigid joint locking in extension (= swiss lock), joint stabilizing under load in any position

Rehabilitation Technology

Serves to maintain or regain autonomy. It mainly includes supportive devices and measures = aids: listed in the Middle and Object List (MiGeL) if they are reimbursed by health insurance companies

- Walkers
- Walking and standing aids
- Mobility aids (wheelchairs, patient transport chairs)
- Grabbing aids
- ADL aids (= **a**ctivities of **d**aily **l**iving)

Orthopedic Shoe Technology

Non -orthopedic: ready-made, serial or factory shoes, custom shoes.

Orthopedic: shoe insoles, shoe adjustments, special shoes, inner shoes, orthopedic serial shoes*, Orthopedic custom-made shoes*.

*: Prescription by orthopedic surgeon.

Principle of pressure redistribution through an insole/footbed: the pressure can be shifted from one place to another.

Shoe adjustment	Definition: Adjustments that improve the biomechanical properties, harmonize the gait, correct the statics and alleviate pain through pressure distribution or relief
Heel	Buffer, roll-off, wing, wedge, rocker sole, drag heel, leg length compensation
Shaft	Adjustment, reinforcement, padding rear, front cap, shaft widths, expansion
Sole	Midfoot, rocker sole, toe, direction, butterfly roll, soft bedding, inner/outer edge elevation, stiffening, widening/narrowing
Special shoes	For insoles, for stabilization, for orthoses, for bandages, forefoot or rearfoot relief shoes, therapy shoe, sickle foot shoe (anti-varus shoe)
Inner shoe/ orthosis	"Shoe in shoe", contains all elements of an orthopedic custom made shoe, which is worn in a ready-made/serial shoe. Indications: Congenital malformation, leg-foot length compensation, paralysis, cerebral palsy, OSG arthrosis/pseudarthrosis.
Orthopedic serial shoe	Semi-finished product, which is suitable for supplying feet that deviate from the usual foot shape and are impaired in function. In some cases, this can avoid the costly custom made shoe
Orthopedic custom made shoe	Custom-made shoe for the treatment of severe foot deformities (dysproportion, unequal foot sizes > 3 cm), foot deformities (malformations, partial amputations), functional disorders (movement restrictions, paralysis), leg shortening (> 25 mm) or as a custom shoe for orthoses

Tumours of the Musculoskeletal System

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Terms

Tumour	lat. *tumour, -oris* – growth, tumour, swelling. Increase in volume, regardless of the cause
Classification	Division into benign (local disease)/intermediate grade/malignant (systemic with risk of metastasis); further classification according to "imitation" of tissue by the tumour
Sarcomas	Malignant tumours arising from mesenchymal tissue with centrifugal growth pattern
Reactive zone	The area between the tumour and the compressed healthy tissue. Contains tumour cells (satellite cells)
Skip lesion	A histologically proven secondary focus of a neoplasm that is distinct from the primary tumour and which is separated by an interval of normal tissue
Pathological fracture	A broken bone that occurs due to an underlying disease (e.g. osteoporosis, infection, altered bone metabolism) or condition (bone tumour) that weakens the bone, rather than from an injury or trauma

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Examination

Clinical Presentation

Often night pain, growing swelling/lump, B-symptoms (fever, night sweats, unintentional weight loss), limping

Diagnosis

- Clinical examination
  - Location, size, shape, consistency of the mass
  - Tenderness
  - Mobility of the lesion under the skin and in relation to the underlying tissue
  - Functional impairment
  - Localization to neurovascular structures
  - General status (further tumour involvement? Symptoms of neurovascular compression distally to the lesion?)
- Radiographic assessment
  - Conventional X-ray in two planes
    - Localization (Epiphysis, Metaphysis, Diaphysis)
    - Description of bone osteolysis (Lodwick classification)
      - (i) Geographic (sclerotic, sharp, blurred)
      - (ii) Moth-eaten
      - (iii) Destructive/permeative
    - Cortical/periosteal reaction
      - (i) Solid (benign)
      - (ii) Lamellar/spiculated (aggressive)
      - (iii) Interrupted (highly aggressive)
  - Staging
    - Process used to determine the extent of a malignant tumour in the body. It is crucial for treatment planning and prognosis estimation
    - Local
      - (i) CT for bone imaging
      - (ii) MRI with contrast agent: extension, soft tissue invasion, involvement of neurovascular structures, skip lesions, soft tissue/bone edema?
    - Systemic
      - (i) Sarcomas metastasize hematogenous → primarily to the lungs (as the fine blood vessels act as a sieve for freely circulating tumour cells)
      - (ii) Conventional CT thorax/abdomen is considered the standard of care
      - (iii) Bone scintigraphy with technetium (is absorbed by osteoblasts) to look for other bone foci; alternatively whole body MRI
      - (iv) PET scan with fluorodeoxyglucose (FDG) tracer to show metabolic activity in bones and soft tissues (marking of tissues with high glucose uptake).

- Biopsy
  – To define the definitive diagnosis
  – The biopsy tract should be as short as possible and lie in the course of the later surgical incision (*no contamination of other tissues/anatomic compartments*)
  – Several biopsy techniques are possible:
    Excisional biopsy (tumour < 3–5 cm, epifascial, superficial)
    Incisional biopsy
    Punch biopsy (CT/MRI/US)
    Fine needle biopsy

CAVE
- In case of clinical and radiological suspicion of a malignant tumour, confirm the diagnosis by means of biopsy before any therapy
- Always determine the biopsy tract in consultation with the treating tumour surgeon

Benign Bone and Soft Tissue Tumours

Benign Bone Tumours

Giant Cell Tumour

Definition
Benign, but locally aggressively growing bone tumour (*atypical for benign tumours, but the giant cell tumour is an exception*)

Epidemiology
Most common between the ages of 30 and 50 years

Localization
Typically manifests in the meta-epiphysis of long tubular bones: Distal femur > proximal tibia > distal radius

Therapy
Operative Surgical extensive curettage and filling of the bone defect with bone cement or allograft bone. For advanced disease and joint destruction: Arthroplasty

Osteochondroma and Multiple Hereditary Exostosis

Definition

In clinical practice, the terms osteochondroma and cartilaginous exostosis are synonym.

Benign tumour that originates from growth cartilage in the form of sessile (broad base) or pedunculated (narrow stalk) lesions

Low lifetime risk (< 1%) of dedifferentiation to secondary malignant chondrosarcoma (especially proximal lesions)

Epidemiology

Mainly affects adolescents and young adults

Multiple hereditary exostosis: condition characterized by multiple osteochondromas due to a mutation affecting the prehypertrophic chondrocytes of the physis. As a result of the number of tumours, the cumulative risk of developing a secondary chondrosarcoma increases up to 10%

Localization

On bone surface at tendon insertion sites, most affected: distal femur + proximal tibia > proximal femur + proximal humerus

Therapy

Conservative Observation. In multiple hereditary exostosis a whole-body MRI every 2–5 years can be considered due to the increased risk of malignant transformation.

Operative Resection when the osteochondroma causes deformity during the growth (distal forearm/distal lower leg) or when the patient becomes symptomatic most frequently because of the tethering effect

Juvenile and Aneurysmal Bone Cyst

Definition

Juvenile bone cyst:	Also called "simple bone cyst": a cavity in the bone filled with serous fluid
Aneurysmal bone cyst:	Multiple cavities that are filled with blood and show a typical mirror formation (liquid-liquid levels, *layered like a "Latte Macchiato"*) on MRI imaging

Epidemiology

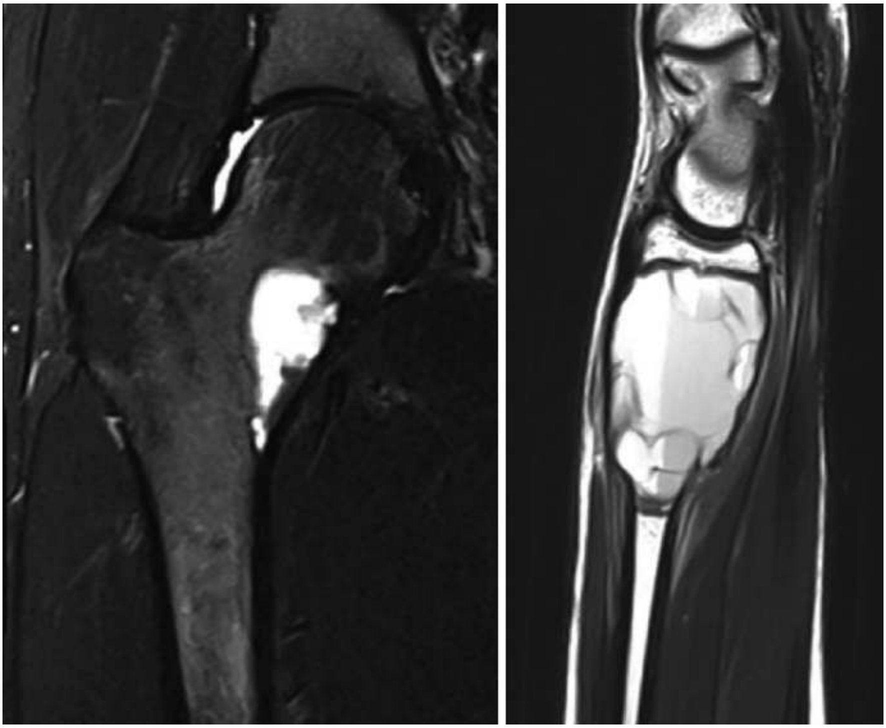
Both conditions typically occur before the age of 20

Localization

- Juvenile bone cyst

  - Central, in the area of the metaphysis during growth, then progressing to the diaphysis
  - Proximal humerus > proximal femur, distal tibia, ilium and calcaneus
- Aneurysmal bone cyst
  - Also in the area of the metaphysis
  - Common location: spine & long bones (*especially distal femur and proximal tibia*)

Therapy
- Juvenile bone cyst:
  - Observation if there is no risk of fracture
  - Curettage with bone grafting is an option if there is a risk of fracture; possibly with additional osteosynthesis
  - Alternatively, aspiration of the cyst fluid, combined with cortisone infiltration or bioresorbable bone cement for preventing fractures
  - In principle, the cyst may regress spontaneously after maturation in about 80%
- Aneurysmal bone cyst:
  - Surgery is usually recommended as progression is expected
  - Curettage with bone defect filling is sufficient
  - Sclerotherapy with polidocanol, ethibloc, or EtOH 96%
  - In huge or difficult anatomical regions embolization or cryotherapy could be an option



Non-ossifying Fibroma

Definition
Benign fibrogenic lesion

Epidemiology
Most common benign bone tumour in childhood: typical between the ages of 5 and 15; overall, approximately 30% of children with open epiphyseal plates have a non-ossifying fibroma

Localization
Eccentrical (cortical), mainly in the area of the metaphysis of the lower extremities
Distal femur > proximal tibia > proximal fibula > distal tibia

Therapy
Conservative "Don't touch", because most of these tumours will regress with growth

Operative Only necessary for large and symptomatic lesions, or when there is an increased risk of pathologic fracture (curettage with defect filling)

Enchondroma

Definition

Benign tumour of cartilaginous tissue resulting from a chondroblast dysfunction in the epiphyseal plate

Risk of malignant transformation approximately 1%

Epidemiology

Usually ages 20–50

Localization

Diaphysis and metaphysis, most commonly in the hand (60%) and the foot

Other common sites of manifestation: distal femur and proximal humerus

Therapy

Conservative Observation

Operative (for size increase or risk of fracture) Curettage and bone defect filling

Osteoid Osteoma and Osteoblastoma

Definition

Both are benign bone tumours

Size: > 2 cm = Osteoblastoma/< 2 cm = Osteoid Osteoma

Epidemiology

Usually ages 10–30

Localization

Osteoid Osteoma: Cortical, lower extremity, proximal femur > tibial diaphysis

Osteoblastoma: most commonly in the area of the posterior elements of the spine

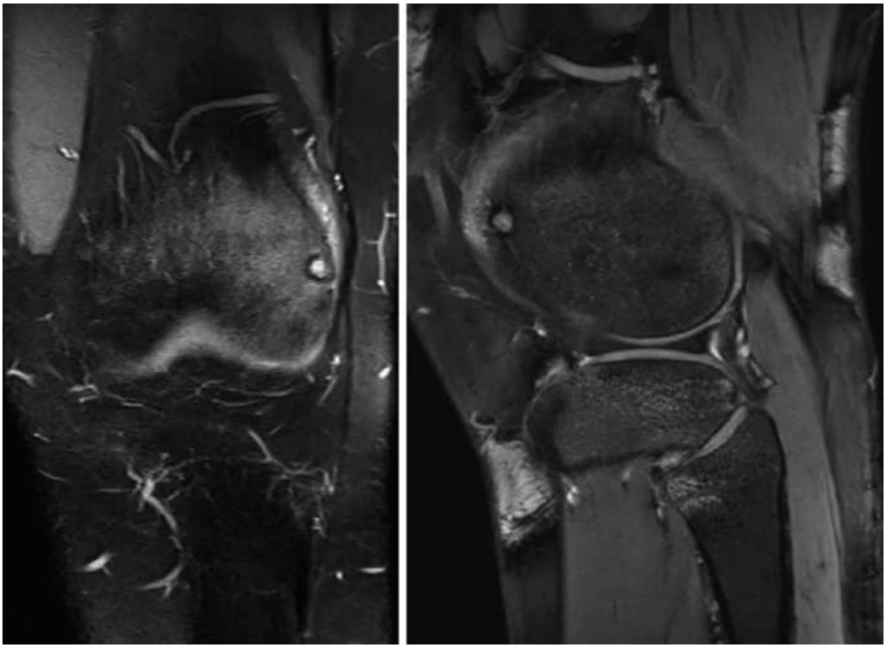
Clinical Presentation

Persistent pain in the affected area, especially at night. Responds very well to NSAIDs (above all aspirin)

Therapy

Osteoid Osteoma: Symptomatic therapy with NSAIDs is usually sufficient. If symptoms persist, percutaneous CT guided biopsy and radiofrequency ablation or curettage may be performed

Osteoblastoma: Usually surgical treatment → Curettage with defect filling



Benign Soft Tissue Tumours

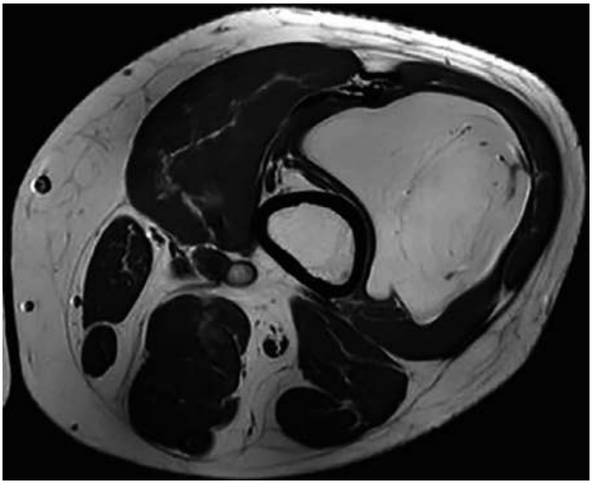
Lipoma	The most common entity in this group. Particularly in the trunk area On MRI: Homogeneous fat mass, clearly circumscribed by a capsule As a rule of thumb: A soft tissue mass that is subcutaneous, mobile on palpation, and smaller than 5 cm is usually benign and can be observed. CAVEAT: A nodule that is deep (below the fascia) and larger than 5 cm should be assessed by imaging (US/MRI)

Therapy

Conservative Most benign soft tissue tumours can be observed and monitored clinically and radiologically over time

Operative Surgical removal is only necessary in cases of discomfort or for aesthetic reasons

Tissue	Benign Tumour
Fat	Lipoma
Smooth muscle	Leiomyoma
Nerve	Neurinoma, Schwannoma
Vessel	Hemangioma
Connective tissue	Fibromatosis



Malignant Bone and Soft Tissue Tumours

Malignant Bone Tumours

Osteosarcoma

Definition

Malignant tumour in which cells form new, unorganized bone

Epidemiology

Osteosarcoma is the most common **primary** sarcoma of the bone

Affects mainly young adults (second decade)

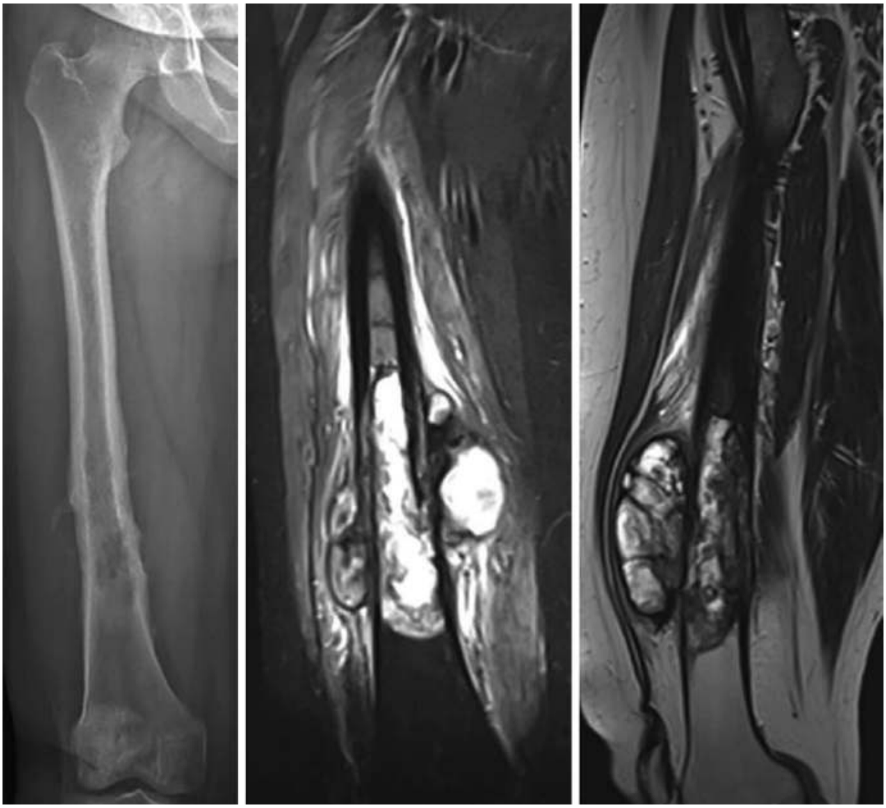
Localization
Distal femur and proximal tibia > proximal femur, proximal humerus + pelvis

Metastases
Hematogenous spread → most commonly to the lungs, secondarily to the bones

Therapy
Multidisciplinary approach:

Preoperative	Chemotherapy (*neoadjuvant chemotherapy*) to contain systemic spread
Operative	Surgical resection with a safety margin around the tumour
Postoperative	Continued chemotherapy (*adjuvant chemotherapy*)

→ Therapy lasts almost a year in total and follows an internationally established protocol



Ewing's Sarcoma

Definition

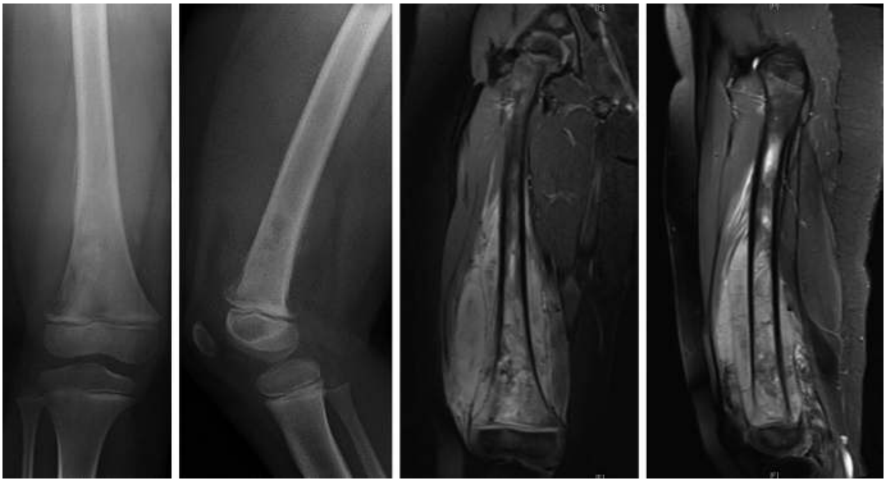
Malignant bone tumour that can also originate from the soft tissues. Histology typically shows small, round, blue cells. The tissue of origin is still unknown. In over 90% of patients, a typical genetic translocation t(11:22) is found in the tumour cells, which serves to confirm the diagnosis

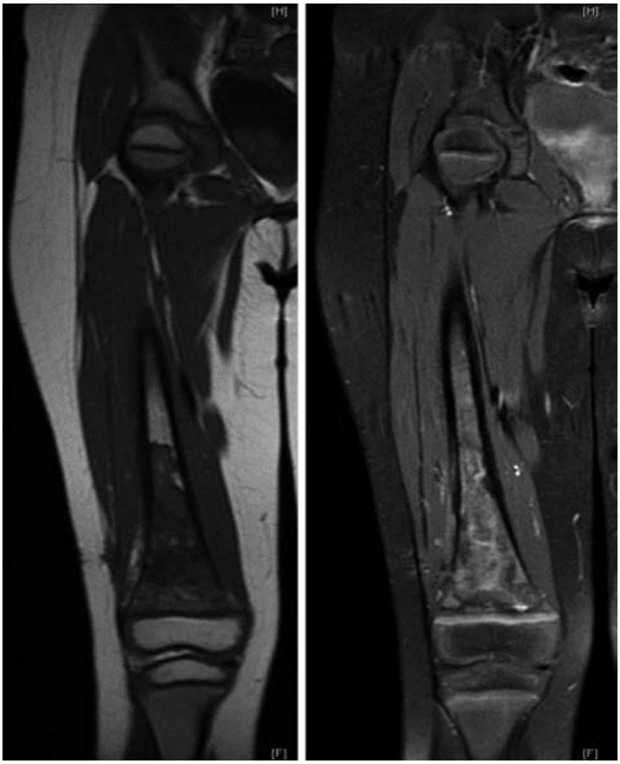
Epidemiology

Usually ages 5–25

Localization

Most commonly affects long tubular bone diaphysis and pelvis





Metastases
High rate of metastasis, most commonly to the lungs and bones

Therapy
Multidisciplinary approach

Preoperative	(Neoadjuvant) chemotherapy
Operative	Surgical resection with a safety margin around the tumour

Ewing's sarcoma also responds very well **to radiation therapy**. In selected cases, definitive radiation therapy may be preferred over surgery

Postoperative	Continued (adjuvant) chemotherapy

Chondrosarcoma

Definition
- Malignant bone tumour whose cells mimic cartilage matrix and, unlike osteosarcoma, do not form bone substance
  - Primary and secondary chondrosarcomas are distinguished:
  - Secondary chondrosarcomas arise from a benign cartilage lesion (*e.g., ex osteochondroma or enchondroma*)

Epidemiology
Mainly "older" patients → 40–70 years of age

Localization
Most commonly in the pelvis, proximal and distal femur, and the scapula

Therapy
Mostly resistant to radiation and chemotherapy

Operative: Grade I, low risk of metastases: Curettage

Higher grade chondrosarcomas (*Grade II + III*): Surgical resection with safety margin

Malignant Soft Tissue Tumours

Definition
Classification analogous to benign soft tissue tumours—according to the tissue of origin. If the tissue of origin is no longer recognizable histologically, it is referred to as an unclassified, undifferentiated sarcoma

Epidemiology
Very rare overall; about 1% of all cancer and proportion of 1 soft tissue sarcoma for every 100 benign soft tissue tumours

Prognosis
Size (*diameter in cm*), histologic grade and the location (*superficial/deep*) are of great importance and determine the prognosis

Metastatic Behavior
Hematogenous spread typically → lung

Individual types may also cause lymph node metastasis

Therapy
Combination of radiotherapy and surgical resection

Chemotherapy usually only for proven metastases or some selected high-grade tumours in younger patients

Special Features
Unfortunately, because they are so rare, they are often overlooked (up to 25%) and not properly treated

Tissue	Malignant Tumour
Fat	Liposarcoma
Musculature	Leiomyosarcoma, Rhabdomyosarcoma
Nerve	Malignant peripheral nerve sheath tumour
Vessel	Angiosarcoma
Connective tissue	Fibrosarcoma

CAVEAT
There is no clinical or radiologic examination that can differentiate reliably between benign and malignant soft tissue tumours. Accurate differentiation/diagnosis is only possible with biopsy. All indeterminate soft tissue nodules larger than 5 cm and deep in location must be considered as soft tissue sarcoma!

Bone Metastases

Epidemiology

Most common tumour disease of the skeleton (much more common than primary bone tumours!)

Etiology

In particular, cancers of the breast, prostate, lung, kidney and thyroid

Localization

Typically: spine, pelvis/hip, ribs, and proximal humerus

Radiographic Examination

X-ray: Osteolytic changes in the bone; however, sclerotic (*reactive bone formation*) or mixed (*lytic + sclerotic*) bone lesions may also occur

Therapy

- The goal is pain control and impending fracture prevention
- Individualized and multidisciplinary approach
- Treatment of the primary tumour and other metastases
- Prophylactic stabilization when the bone is severely weakened and a pathologic fracture is imminent

CAVEAT
If osteolysis is detected in a bone of a patient >40 years of age, an active search for a possible primary tumour with bone metastases must be performed

Surgical Principles

Benign Soft Tissue and Bone Tumours

- Can be gently detached and resected from surrounding tissue
- Safety margin/Removal of healthy tissue is usually not necessary
- For benign bone changes, curettage may be performed to "scrape" the tumour tissue from the bone

Malignant Soft Tissue and Bone Tumours

- The highest priority is the safe and complete removal of the tumour mass
- Because of the reactive zone where the satellite cells are located, a layer of healthy tissue must be removed with the tumour (safety margin)
- The quality of the surgical resection is graded as follows
  - **Radical:** Resection includes the removal of all affected compartments and usually means amputation
  - **Wide:** Tumour including the satellite cells are removed, tumour cells do not reach the resection margin (R0)
  - **Marginal:** Resection through the reactive zone, tumour cells reach the resection margin (R1)
  - **Intralesional:** Resection through the tumour mass, leaving a macroscopic tumour residue (R2)
- R1 and R2 resections lead to a poor prognosis for the patient and should therefore be avoided at all costs!

Infections in Orthopedics

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İlker Uçkay and Carol Strahm

General

- Infections in the osteoarticular/orthopedic area are usually **treated surgically, together with systemic antibiotic treatment**
- The (postsurgical) duration of systemic antibiotic therapy for bone, joint, and material-associated infections is **weeks-long** (typically for 6–12 weeks). The clinical failure rate, e.g. the risk for surgical revision for any reason, can be relatively high (5–20%). In contrast, the incidence of microbiologically-identical recurrences after therapy (with identical pathogen(s) as in the index infection), is low (< 5% to 10%)
- An established bacterial contamination, or a clinical infection, of bones or implants never eradicates without therapy
- Biofilm:
  - The biofilm is formed by the bacteria and the host. It could be described as a syntrophic community of microorganisms in which cells stick to each other and often also to a surface. These cells become embedded within a slimy extracellular matrix consisting of an amalgam of proteins, sugars, and other substances

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 - Antibiotics, as chemical agents, penetrate much worse into this biofilm. In addition, the nested bacteria reduce their metabolism and become less sensitive to most antimicrobial agents, which usually act on the bacterial wall when the bacteria are dividing

Pathogens

- Usually monobacterial infections
  - With the exception of the diabetic foot = classically polymicrobial
  - Gram-positive and Gram-negative; the latter especially when the infection is complicated by local ischemia/necrosis or maceration of soft tissues
- Osteoarticular and implant-associated infections are mainly Gram-positive
  - (*Staphylococcus aureus*, coagulase-negative staphylococci, streptococci)
- Implant-associated infections (*e.g. prostheses*) can be caused by skin commensals, which are typically less virulent, compared to their pyogenic counterparts, in terms of local and systemic inflammation
  - Skin commensals: coagulase-negative staphylococci, corynebacteria, or cutibacteria
- *S. aureus*-associated infections
  - Implant-infections, soft tissue abscesses, iatrogenic and injection abscesses, septic bursitis
- Most infections occur *per continuitatem* from previous surgery, trauma, or skin lesions
- Hematogenously-acquired infections are possible, especially in case of septic arthritis, prosthetic joint infections, and vertebral osteomyelitis (*spondylodiscitis*)

Diagnosis

Clinic

Local and/or systemic inflammation, pus, abscess, rapid deterioration, fever, lymphangitis.

Radiology

In case of material and bone infections: bone lesions and loosening of material.

Microbiology

Microbiology of intraoperative cultures, PCR. The first also provides the pathogen and its sensitivity to various antibiotics. Sonication and histology confirm the clinical suspicion.

CAVE
- The microbiological culture results can be **false-negative** after previous antibiotic therapy (***no growth despite clinical infection***)

A single positive microbiological result, without a corresponding history and clinical presentation of infection, is likely a contamination. For an optimal microbiologically exam, we always take several **deep** tissue microbiological samples (*up to five*), and preferably **without** previous antibiotic administration during several days.

Special Features

- A chronic fistula (sinus tract) with purulent discharge is very suggestive for chronic osteomyelitis
- In case of systemic sepsis (hemodynamic deterioration, chills, high fever) blood cultures should be taken
- The postoperative/posttraumatic serum-CRP is almost always elevated (*maximum on the second-third postoperative day*) and should not been sampled routinely. Its elevation does not prove infection *per se*.
- The same applies to postoperative fever, which can occur occasionally in up to 20% of all patients postoperatively, during several days

Surgical Treatment

- Osteoarticular infections, with or without infected osteosynthesis material, are ideally treated with a combination of surgery and antibiotics
- Healing only through antibiotic therapy is possible in:
  - Superficial soft tissue infections, small abscesses within soft tissues, spondylodiscitis, and diabetic foot infections
  - Implant-associated infections, deep fungal infections, a chronic osteomyelitis, and septic arthritis practically always require a surgical intervention

Therapy

Debridement, lavage, removal of abscesses and sequestra (= *bone abscesses*) and/or the removal of infected implants.

Antibiotic Treatment

Empirical Therapy

- Targets the suspected pathogens: Since most orthopedic infections are Gram-positive, second generation cephalosporins or amoxicillin/clavulanic acid are often used as empirical therapy
- In severe penicillin allergy, clindamycin or vancomycin can be used as a substitute

Choice of Targeted Antibiotic

- As soon as the pathogen + antibiogram are known: → **targeted** antibiotic therapy
- Implant-associated infections caused by staphylococci (*S. aureus or coagulase-negative staphylococci*) are treated with a combination of rifampicin/rifampin in cases where the implant is not removed, as these agents are particularly biofilm-penetrating

CAUTION
Due to the rapid development of resistance, rifampicin should **not** be administered as monotherapy.

Intravenous Antibiotic Therapy

Traditionally, the empirical treatment of orthopedic infections begins intravenously (IV). The treatment is soon switched to oral therapy, after a good evolution, or upon the identification of the pathogen.

Duration of Antibiotic Therapy

- This topic is subject of current research (several prospective-randomized trials)
- The duration of antibiotic therapy for soft tissue infections is determined by the clinic (*i.e. the infection that persists on visual evaluation*) and is thus individualized. Often only a few days are sufficient.

- Native septic arthritis without adjacent osteomyelitis: 3–4 weeks
- Bone and material infections: 6 weeks to 12 weeks in a few exceptions
- *All these duration specifications apply to "classical" bacteria. "Special" infections, such as osseous tuberculosis, are treated longer; or even shorter (→ gonococcal arthritis).*

Follow-Up Checks

No evidence-based laboratory parameters or radiological methods exist, to adjust the duration of antibiotic therapy (**not even the serum CRP levels**).

→ Clinical decision

Prophylaxis of Orthopedic Infections

Epidemiology

Infections of the surgical site (*eng. Surgical Site Infection—SSI*) are rare in (elective) orthopedic surgery.

Hip and knee prostheses	Incidence 1%
Other implants—Incidence	3%
Arthroscopies	Lowest incidence
External fixators	Highest incidence

Risk Factors

- Over 60 different SSI risk factors, half of which are modifiable
- e.g. age, diabetes mellitus, previous infection, rheumatoid arthritis, smoking, high comorbidity scores and a long duration of index surgery, etc.

Evidence-Based Preventive Measures

- Before surgery: Sanitation of active symptomatic infection foci
- Surgical hand disinfection for at least 1.5 min with alcohol-based disinfectant
- Disinfection of the surgical area
- Adequate antibiotic prophylaxis (First- or Second-generation cephalosporins—IV [*immediately before the cut*])
- Use of "established" surgical techniques (together with a short operation time)
- Medical hand hygiene during dressing changes and rounds

- Decolonization of the body (*or the nasal cavities*) from (proven) *S. aureus* carriage is advocated, but is complex to implement and still subject of ongoing research

Chronic (Implant-Free) Osteomyelitis

Diagnosis

Clinical presentation, sinus tract, *CT, MRI; Standard X-ray.*

Therapy

- Removal of all sequestra (*bone abscesses*) and refreshment and resection (*or amputation*) of the affected bone segment
- The antibiotic therapy is targeted at the germ(s). The failure rates are depending on the extent and location **10–15%**, and recurrences are possible even after decades

Orthopedic Material Infections

- Basically: Any infected foreign material should be removed since the success rate of infection eradication is highest this way
- However, if the material is indispensable for stability reasons:
  - Debridement followed by long-term antibiotic therapy
  - Or: temporary external stabilization (*external fixation*)

Prosthetic Infections

Therapy

- For early or acute hematogenous infections (*less than 3–4 weeks*): Debridement; exchange of mobile parts, while retaining the infected prosthesis (DAIR procedure; or one-stage exchange):
- For late infections, substantial soft tissue damage/defect and/or implant loosening: Single-stage (One stage) or two-stage exchange
  - Single-stage: Infected prosthesis is completely replaced
  - Two-stage: Patient has a 6-week antibiotic therapy between the removal and the reinsertion of a new prosthesis, without a prosthesis or functioning joint

Septic Arthritis

Bacterial septic arthritis without foreign material (= *native bacterial arthritis*).

Therapy

Surgical lavage (+/–) synovectomy.

Initial IV antibiotic therapy, then orally according to resistance, 3–4 weeks.

Diabetic Foot Infections

- Practically always secondary The patients, in addition to the actual diabetes, have another underlying chronic problem such as
  - Ischemia and necrosis
  - Polyneuropathy (microtrauma not noticeable)
  - Reduced patient's compliance
- Continuous foot care for prevention, as well as adequate footwear, are essential
- Revascularization, if possible, in case of PAD (peripheral arterial disease)

Therapy

- surgical + antibiotic: for necrosis, malposition, abscesses, and clear destructive osteomyelitis. Off-loading and, if possible, the improvement of patients' compliance

Understanding Statistics and Methodology of Orthopedic Studies

13

Tobias Götschi

Introduction

Foundation of Modern Medicine

- Contemporary medicine places a strong emphasis on evidence-based practices
- Medical practitioners aim to anchor their medical decisions in well-established research evidence
- A critical aspect of correct interpretation of research data lies in a solid understanding of statistical principles
- The integration of statistical knowledge enhances the ability to adapt and apply research findings to the dynamic nature of patient care in orthopedics

Data Types

- Understanding data types is a crucial concept in statistics, essential for accurately applying statistical measurements to orthopaedic data and drawing correct conclusions about specific assumptions related to it

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Different types of data

Type	Description	Examples	Typical visualization tools
Continuous	Numerical data with an infinite number of possible values	Bone mineral density Joint range of motion	Histograms, line charts, scatter plots
Ordinal	Categories with a meaningful order or rank	Fracture severity (mild, moderate, severe)	Ordered bar charts
Categorical	Non-numeric data representing categories or labels	Implant types (plates, screws, prosthetic joints)	Bar charts, pie charts
Binary	A special case of categorical data with only two categories	Treatment (conservative, surgical)	Bar charts, pie charts

Descriptive Statistics

- Descriptive statistics serve to summarize the values within a dataset
- Usually, the values in a dataset are summarized by reporting the most typical value (central tendency measure) and the extent to which the values spread around this (dispersion measures)

Central Tendency Measures

- Central tendency measures are statistical parameters that provide a single representative value of a dataset

Types of measures of central tendency

Name	Description	Computation	Notes	Useful for
Mean	The arithmetic average of a dataset	Sum all values and divide by the number of observations	Sensitive to extreme values and therefore only to be used with caution	Continuous data
Median	The middle value in a sorted dataset	Sort the dataset of interest in order of value. If the number of respective observations is odd, the median is defined as the middle value. Else, average the two middle values to determine the median	Resistant to extreme values, making it a robust measure for skewed distributions	Continuous data Ordinal data
Mode	The most frequently occurring value in a dataset	Count the occurrence of each value and determine the one(s) occurring most often	A dataset may have multiple modes (multimodal)	Ordinal data Categorical data

Dispersion Measures

- Dispersion measures quantify the spread or variability within a dataset
- They are only applicable to continuous or ordinal data

Types of dispersion measures

Name	Description	Computation	Notes	Useful for
Standard deviation	Measures the typical deviation of each data point from the mean	Subtract the mean from each data point, square the differences, find the average of the squared differences, and then take the square root of that average	The standard deviation is sensitive to outliers	Continuous data
Range	The range of a dataset is quantified by the minimum and the maximum value occurring	Determine the lowest and the highest value	While simple to calculate, the range is sensitive to extreme values, making it less robust in the presence of outliers	Continuous data Ordinal data
Interquartile range (IQR)	The IQR is the range of the middle 50% of the data (i.e. the range between the 25th and the 75th percentile	Arrange the dataset in ascending order, find the median and then calculate the median of the lower and upper halves of the values around the primary median	Is less influenced by outliers and therefore usually preferred over the range	Continuous data Ordinal data

Other Descriptive Parameters

- Categorical data cannot be described by a central tendency or a dispersion measure. Such data can be summarized by the absolute or relative occurrence of each category
- Ordinal data can also be summarized in such a way, if preferred

Other descriptive measures

Name	Description	Computation	Notes	Useful for
Absolute counts	Represents the total number of occurrences within a specific category	Count the occurrences of each value or category in the dataset	Provides a straightforward numerical representation of frequency	Ordinal data Categorical data

Name	Description	Computation	Notes	Useful for
Relative counts	Indicates the proportion of occurrences relative to the total sample size, often expressed as percentages	Divide the absolute count of a specific category by the total number of observations and multiply by 100	Facilitates comparison of the prevalence of outcomes across different groups	Ordinal data Categorical data
Ratios	Quantifies the relationship between two variables, offering insights into proportions, rates, or relative magnitudes	Calculate the ratio by dividing one quantity by another, providing a measure of their relationship	Versatile measure applicable to comparing treatment effects, assessing risk, or evaluating interventions	Ordinal data Categorical data

Statistical Inference

Hypothesis Testing

- **Motivation:** In clinical studies, natural (random) variability in patients and measurements induces variations in the outcome of interest. Observed differences between treatment groups in a randomised controlled clinical trial could hence either be a result of the treatment under investigation or it could simply be produced by chance
- **Goal:** Decide, whether the observed data sufficiently supports a particular hypothesis ("alternative hypothesis"). If not, the null hypothesis is assumed to be true (until more evidence is available)
- **Hypotheses:**
  - Null Hypothesis (H_0): No effect (e.g.: There is no difference between the groups).
  - Alternative Hypothesis (H_1): Effect exists (e.g. The outcome in the treatment group differs from the outcome in the control group)
- **P-value:**
  - Provides the basis to accept or reject the null hypothesis
  - Represents the probability of obtaining the observed result (or a more extreme one) under the null hypothesis. In other words, the p-value answers the question:
  - "Is it sufficiently unlikely to obtain the observed result if the null hypothesis is true?"
- **Significance level:**
  - Threshold probability (α) below which the p-value is deemed sufficiently low and the null hypothesis can be rejected (i.e. the alternative hypothesis can be accepted: "There is indeed a difference in outcome between the treatment groups"
  - If $p \leq \alpha$ ➔ The result is deemed to be statistically significant
  - The significance level is to be set before collecting any data for the study
  - Usually set to $\alpha = 5\%$

Type I and Type II Errors

- Any observed effect in a study could potentially be a result of chance. Conversely, a true treatment effect could be masked by natural variations of the outcome parameter
- Type I error:
  - Arises when there is no true effect or difference in the subject of study, but the statistical analysis erroneously detects one
  - The outcome suggests a significant result purely by chance, leading to the incorrect rejection of the null hypothesis
  - Type I errors are associated with mistakenly concluding that there is a real effect when, in fact, there is none
- Type II error:
  - Occurs when there is a true effect or difference in the subject of study, but the statistical analysis fails to detect it
  - In other words, the null hypothesis is not rejected when it should be, leading to a missed opportunity to identify a genuine relationship or effect in the population
  - Type II errors are associated with failing to accept an alternative hypothesis when it is true
- Example:
  - A randomized controlled clinical trial investigates whether a specific exercise regimen reduces the risk of postoperative complications following hip replacement surgery
  - Each included patient is assigned into either of two groups:
    Control group: Is administered the conventional rehabilitation protocol
    Intervention group: Is administered the experimental rehabilitation protocol
  - H_0: There is no difference in the probability of postoperative complications in the two groups
  - H_1: The two groups differ in the probability of postoperative complications

	H_0 is true There truly is no difference in the probability of postoperative complications, irrespective of rehabilitation protocol.	**H1 is true** The probability of postoperative complications truly differs between the two rehabilitation protocols.
H_0 is retained The observed group difference is not large enough to yield a statistically significant result.	Right decision	Type II error
H_0 is rejected The observed group difference is sufficiently large such that $p \leq \alpha$ and the result is hence statistically significant.	Type I error	Right decision

Test Structure

- **Statistical inference tests:** Yield the p-value for the relationship between one or more independent variables and the dependent variable
- **Independent variables (IV):** Parameters or conditions that are manipulated or observed to assess their relationship with the dependent variable.
- (In the study example above, the IV is the group assignment (control / intervention) of each included patient)
- **Dependent variable (DV):** The dependent variable in clinical research is the outcome or response that researchers aim to understand, explain, or predict based on the manipulation or observation of independent variables.
- (In the study example above the DV is the occurrence of postoperative complications)

Choosing the Right Test

- The most suitable statistical test for a specific research question primarily depends on the data type of the independent and the dependent variable

			Independent variable			
			Continuous	**Ordinal**	**Categorical**	
					Binary	**>2 levels**
Dependent variable	**Continuous**		Pearson correlation/ linear regression	Spearman rank correlation	T-test (paired/ unpaired)	ANOVA
	Ordinal		Spearman rank correlation		Mann-Whitney U test/Wilcoxon signed rank test	Kruskal-Wallis test
	Categorical	**Binary**	Logistic regression	n/a	Fisher's exact test/ chi-square test	
		>2 levels	Multinomial logistic regression			

- In many instances, proper inference testing requires taking into account multiple independent variables that may potentially influence the dependent variable of interest (e.g. The probability of postoperative complications may not only be influenced by the type of rehabilitation protocol, but also by patient's adherence to it)
- In such scenarios, "multivariate" models can be used to draw valid conclusions

			>1 IV	
			Continuous	**Categorical/binary**
Dependent variable	**Continuous**		Linear regression	
	Ordinal		Ordinal logistic regression	
	Categorical	**Binary**	Logistic regression	
		>2 levels	Multinomial logistic regression	

Parametric vs. Non-parametric Tests

- Statistical inference tests can be broadly categorized into parametric and non-parametric test. Both groups have distinct advantages and disadvantages

Parametric and non-parametric tests

Test type	Pros	Cons	Examples
Parametric	If they are applied correctly, they tend to carry more statistical power (i.e. they are more efficient in correctly rejecting the null hypothesis)	Rely on various assumptions regarding the distribution of the dependent variable or regarding the relationship between the independent and the dependent variable. Violations of these assumptions might lead to a type I or type II error	Pearson correlation Linear regression T-test (paired/unpaired) ANOVA Logistic regression
Non-parametric	Rely on less assumptions regarding the distribution of the dependent variable or the relationship between the independent and the dependent variable	Yield less information about the nature of the investigated relationship	Spearman rank correlation Mann-Whitney U test Wilcoxon signed rank test Kruskal-Wallis test Fisher's exact test

Key Assumptions of parametric inference tests:

- **Normality**, also referred to as a Gaussian distribution or a "bell-shaped" curve, indicates the extent to which data display a central tendency and symmetrical distribution in relation to the mean. Since parametric tests involve comparing means between data groups, it is essential for the means to faithfully represent the data. The validity of p-values in parametric tests relies on the data conforming to a normal distribution

- Depending on whether parametric or non-parametric tests are applied, different descriptive statistics are usually reported:
  - Parametric tests: Mean and standard deviation
  - Non-parametric tests: Median and interquartile range / relative counts

Test class	Key assumptions
Comparison tests (T-tests, ANOVA)	Normal distribution of the dependent variable within each group Similar variance in all comparison groups
Association-quantifying tests (linear regression, logistic regression)	Linear relationship Normally distributed residuals Homoscedasticity

Study Designs

Observational Studies

- Observational studies are research designs in which investigators observe and analyse individuals or groups without intervening or manipulating any variables (for study purposes)
- These studies aim to understand relationships, patterns, or associations within a population by systematically collecting data through observation, measurement, or other non-interventional methods
- Observational studies are valuable in exploring natural occurrences, identifying potential risk factors, and generating hypotheses for further investigation

Cross-Sectional Studies

- Definition: Examines a population at a single point in time
- Purpose: Descriptive analysis of the prevalence of orthopedic conditions or risk factors
- Application: Snapshot assessments in orthopedic epidemiology
- Advantages:
  - Quick insights
  - Cost-effective
- Limitations:
  - No temporal relationship
  - Potential for bias

Cohort Studies

- Definition: Follows a group over time to assess the development of outcomes
- Types:
  - Prospective (forward in time)
  - Retrospective (backward in time)

- Application: Tracking the incidence of orthopedic events or evaluating interventions
- Challenges:
  - Loss to follow-up
  - Confounding factors

Case-Control Studies

- Definition: Compares individuals with a specific outcome (cases) to those without (controls)
- Application: Investigating associations between potential risk factors and orthopedic conditions
- Challenges:
  - Careful matching required to avoid selection bias
- Limitations:
  - It is difficult to establish causation (as opposed to simple association)

Experimental Studies

- Experimental studies involve the deliberate manipulation of independent variables to observe their effects on dependent variables, allowing researchers to establish causal relationships

Randomized Controlled Trials (RCTs)

- Definition: Experimental studies where participants are randomly assigned to intervention and control groups
- Randomization:
  - Patients are randomly assigned to either intervention or control group
  - Minimizes selection bias, ensuring groups are comparable at baseline
- Blinding:
  - Single blind design: The patients do not know which group they are assigned to
  - Double blind design: Both the patients and the treating physicians do not know group assignment
  - Serves to eliminate bias in outcome assessment
- Application: Assessing the efficacy of new treatments, surgical interventions, or rehabilitation protocols
- Advantages:
  - Causation establishment: RCTs are considered the gold standard for establishing causation due to randomization, reducing confounding factors
  - High internal validity: Random assignment minimizes selection bias, ensuring that observed effects are more likely attributable to the intervention
  - Generalizability: Findings from well-conducted RCTs can be generalized to broader populations, enhancing external validity

- Limitations:
  - Resource intensive: RCTs can be time-consuming and expensive to conduct, especially in large-scale studies with long-term follow-up
  - Ethical concerns: Ethical considerations may limit randomization, particularly when a standard treatment is known to be effective
  - Limited applicability: Strict inclusion and exclusion criteria may limit the applicability of RCT findings to specific patient populations
  - Practical constraints: In certain situations, randomization may be impractical or unacceptable, particularly in emergency or urgent care settings

Quasi-Experimental Designs

- Definition: Research designs with features of both experimental and observational studies
- Lack of Randomization: Participants are not randomly assigned to groups
- Application:
  - Evaluating interventions where randomization is impractical or ethically challenging
- Example: Assessing the long-term success rates of a modified surgical technique compared to conventional methods
- Advantages:
  - Practical when randomization is not possible
  - Provides insights into the impact of an intervention over time
- Limitations:
  - Greater risk of confounding, making causal inferences more challenging

Sample Size Estimation

- Sample size estimation is employed to calculate the optimal number of patients needed for a clinical study, ensuring a sufficient likelihood of detecting a meaningful effect
- Accurate sample size estimation is fundamental for conducting rigorous and meaningful orthopedic studies

Factors Influencing the Required Sample Size

- Effect size:
  - The magnitude of the expected difference or association in the orthopedic outcome
  - Larger effect sizes require smaller sample sizes to achieve statistical significance
- Significance level (α):
  - The probability of committing a Type I error (rejecting a true null hypothesis)
  - Is set by the investigators before conducting the clinical study
  - Smaller significance levels require larger sample sizes

- Statistical power (1-β):
  - The probability of correctly rejecting a false null hypothesis
  - Is set by the investigators before conducting the clinical study
  - A higher power (typically 0.80 or 80%) reduces the risk of Type II errors
  - Higher statistical power requires larger sample sizes
- Variability (Standard Deviation):
  - The degree of dispersion or variability in the outcome of interest
  - Greater variability necessitates larger sample sizes
- Study design:
  - The choice of study design, particularly the selection of outcome parameters and frequency of measurement, can significantly influence the necessary sample size
- Statistical test:
  - Different statistical tests carry more or less statistical power and therefore influence the required sample size

Practical Considerations

- The required sample size associated with a specific study design is a key feature to be considered in the study design phase
- Participant dropout (patients not showing up to follow-up assessments, withdrawing consent, …) and other data loss (logistical difficulties, difficulties during the measurement, …) are to be expected in most study designs and accounted for during sample size estimation. A good rule of thumb is to anticipate a 10% dropout rate
- The sample size has a strong influence on the budget and the required resources to conduct the clinical study

14 Ethics in Orthopedics

Jürg Knessl

Basics/Terms

Ethics is the theory and reflection about the right way to act. Morality, in contrast, is the currently accepted and recognized way of behaving in a particular society.

The "normative ethics" defines, what is right. On the one hand, this can be done "top down", through fixed specifications from the top downwards, or on the other hand "bottom up", according to a consensus reached in mutual exchange.

Relativism Norms are not absolute, but relative to: referring groups, society, culture, time era. They are right depending on the context. Situational relativism: An action is right or wrong depending on the current situation. Problem of relativism: Weakening of a principal position. Binding ethical norms, which apply to everyone, do not exist anymore.

Schools of Thought on Ethics

Consequentialism This theory does not begin with the rules of action, that means its conditions, but with the aims.

Utilitarism An essential school of thought in medical ethics. Mainly prominent in situations, in which need and scarcity prevail, for example in transplant surgery, in use of very expensive medicaments, epidemics, catastrophes and in the context of war medicine. One of the founders of utilitarism is the English lawyer Jeremy

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Bentham. His thesis is as follows: "The greatest amount of happiness for the greatest amount of people".

Immanuel Kant German philosopher and an important ethicist of the eighteenth century. Two of his principles are of great importance:

> Man should act in such a way that his action may become an universal law.
>
> A person may be an end, but never a means to an end.

Jürgen Habermas A representative of the so-called "Discourse Ethics" and the "bottom up" approach. His method is a purely procedural ethics. "Everybody can participate in the decision-making process and to make a demand, no one can be prevented from taking part in the discourse." Criticism: Consensus does not automatically mean "right". Everyone can never be considered to the same extend.

John Rawls An US-American philosopher of the twentieth century. His main topic is "The Theory of Justice", where he analyses the question of justice in the distribution of medical goods. The relevant criteria are: Efficiency, urgency, the random principle and the consideration of own fault.

William David Ross: Prima Facie Duties An ethical system based on several fundamental rules, corresponding to duties:

Fidelity/Reparation (make amends when we have wronged someone)/Gratitude/Non-harm/Beneficence/Self-improvement/Justice.

Four Principles of Beauchamp/Childress

The "Principles of Biomedical Ethics" (1979) by Tom Beauchamp and James Childress is considered as the most important standard work about biomedical ethics. These four principles should be known to all persons working in a medical profession. The first two correspond to proven medical tradition, the following two are quite new.

1. Beneficence: Physicians should act in the best interests of their patient.
2. Non-maleficence: First, do no harm (primum non nocere).
3. Autonomy: The patient is autonomous in his or her decision and has the right to refuse or choose a treatment (quite a new principle).

4. Justice and equality: This principle concerns the distribution of scarce resources in healthcare and the decision as to who receives treatment (Justitia).

Glossary/Meaning of Terms

Autonomy I am «free», when I am doing what I want at the moment. But: Persons acts "autonomously", if they had this wish too, if, after common conceptions, they were acting "sensibly". An autonomous action, respectively decision, is the case, when its is not only free, but also considered, responsible and sensible.

Human Dignity Originally exclusively religiously connotated. The term derives from "Likeness of God", who created man "in his image". Constitutions often contain the phrase: "Human dignity is untouchable". Actually, it would have to say: "should be untouchable", because it is not a descriptive state which is expressed here, but rather a moral commandment, a call.

Logic/Arguing

A man with a thinking mind, when faced with a clear contradiction, feels uncomfortable. In logic applies beside other "the sentence of the excluded third" (Tertium non datur) and "the sentence of contradiction": A statement and its opposite cannot be true at the same time. A representative example:

Correlation/Causation "Post hoc" (after that) is not "propter hoc" (because of that). The following has to be considered: It is not A that causes B and not B that causes A, but another reason causes A and B. A very common mistake of thought, of patients, as well of doctors.

In **orthopedic surgery**, as well as in other fields of surgery, there are further multi-layered ethical requirements. The medical work is, from a judicial point of view, is "an occupation prone to danger". One could damage a person, who was trusted to him for his expertise, irreversibly and for life. Concerning the risk and the number of liability cases, orthopedic surgery is leading. Those occupied in surgery, in contrast to craftsmen, do not have to fulfil a success warranty. Not for the result, but for the diligence in planning, for the correct information, for care and the after treatment. Already for the so called "craftmanship ethics", meaning the obligation to exert the work correctly, is the physician liable. Furthermore, he has to act in the interest of his patients and decide what is right and good for this particular patient. Self-interest of the doctor is submitted to a much stronger limitation than in other occupations.

Clinical Neurophysiology for Orthopedic Surgeons—A Primer

15

Jan Rosner and Armin Curt

Anatomy of the Peripheral and Central Nervous System

Peripheral Nervous System

- Dorsal root: Carries sensory information to the spinal cord (CNS)
- Ventral root: Peripheral motor output from the spinal cord
  - Sympathetic fibers: In the thoracolumbar spinal cord (T1-L2), the ventral root also contains preganglionic sympathetic fibers. These fibers synapse onto postganglionic neurons in the sympathetic chain, which then supply structures such as sweat glands and the smooth muscles of blood vessels.
- Spinal nerve: A nerve formed by the union of the dorsal (afferent) and ventral (efferent) roots of a specific spinal segment, carrying both sensory and motor fibers.
- Plexuses: Networks of nerves formed from the ventral rami, giving rise to peripheral nerves.

Central Nervous System Pathways

- Major ascending pathways:
  - Dorsal columns: Light touch and proprioception
  - Spinothalamic pathways: Pain (including sharp/pinprick sensation) and temperature

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- Major descending pathway:
  – Corticospinal tract: Motor impulses from the cortex to the anterior horn in the spinal cord, where the peripheral axon originates.
- Spinal ventral (anterior) horn: The anterior part of the gray matter in the spinal cord containing lower motoneurons (2nd motoneuron) that send axons into the peripheral nervous system to ultimately innervate skeletal muscles.
- Spinal dorsal (posterior) horn: The posterior part of the gray matter in the spinal cord that receives sensory information from peripheral nerves, processing signals related to touch, pain, and temperature before relaying them to the brain.
- Motor unit: Consists of a group of muscle fibers innervated by a single moto neuron, which is located in the ventral horn of the spinal cord.

Peripheral Neuron

- Consists of a nerve cell body and an axon in the peripheral nervous system.
- The nerve cell body for a peripheral motor axon is located in the central nervous system, i.e., spinal cord (anterior horn) and constitutes the lower motoneuron (alpha-motoneurons).
- The nerve cell body for a peripheral sensory axon is located in the peripheral nervous system, i.e., the dorsal root ganglion (DRG). (Fig. 15.1)
  – Axonal process proximal to the DRG = preganglionic (supraganglionic) axon
  – Axonal process distal to the DRG = postganglionic (infraganglionic) axon
- Function of the cell body: metabolic and trophic support for the axon
- Function of the axon: conduction of electrical impulses
  – Saltatory electrical conduction (fast) in myelinated axons
  – Continuous electrical conduction (slow) in unmyelinated axons

Connective Tissue Sheaths of a Peripheral Nerve

– Endoneurium: covers groups of axons = fascicles
– Perineurium: covers fascicles
– Epineurium: covers groups of fascicles

- Myelin sheath:
  – Composed of Schwann cells in the peripheral nervous system or oligodendrocytes in the central nervous system.
  – Enables saltatory electrical impulse conduction, where action potentials “jump” between the nodes of Ranvier for faster conduction.

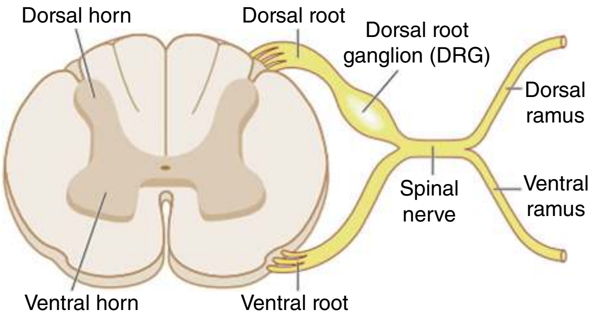

Fig. 15.1 Cross-section of a spinal segment. A spinal segment is defined by the entry of dorsal roots and the exit of ventral roots from the spinal cord. Each segment is the section of the spinal cord that gives rise to a single spinal nerve. A dermatome refers to an area of skin innervated by peripheral nerve fibers stemming from one dorsal root ganglion

Classification of Nerve Fibers

- Large Fibers:
  - A-alpha fibers: efferent motor axons
  - A-beta fibers: afferent, carrying touch and proprioceptive information
- Small Fibers:
  - A-delta fibers: Thin myelin sheath, responsible for pain and temperature
  - C-fibers: No myelin sheath, involved in pain, temperature, and the autonomic nervous system (postganglionic axons)

Classification of Peripheral Nerve Damage (Seddon Classification)

Neurapraxia

- Focal nerve compression with damage to myelin sheath, but intact axons.

Axonotmesis

- Complete or partial axonal and myelin sheath disruption with Wallerian degeneration.
  - Wallerian degeneration: A process that occurs after axonal injury, where the portion of the axon distal to the injury degenerates. For motor nerves this leads to muscle denervation with typical EMG findings (see below).

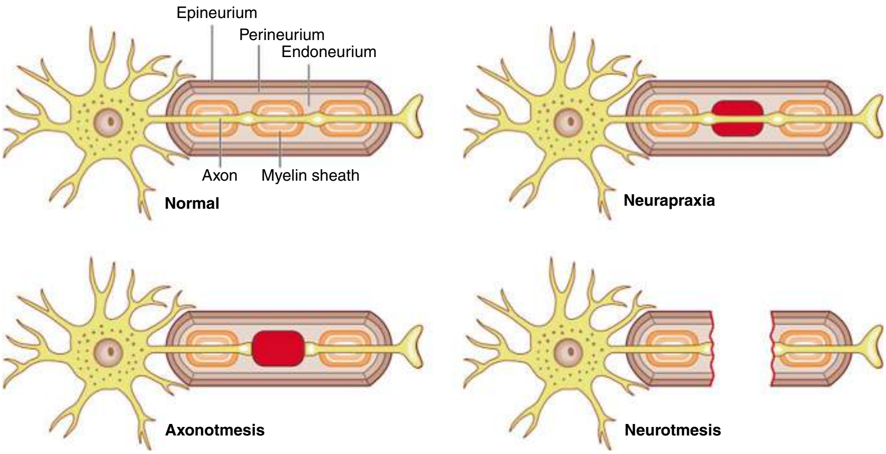

Fig. 15.2 The neuroanatomy of a normal peripheral nerve and the various patterns of nerve damage are described. Each axon is wrapped in a myelin sheath and surrounded by different connective tissue layers. The endoneurium surrounds individual axons within fascicles, while the perineurium encases each fascicle, and the epineurium encases groups of fascicles. Degrees of nerve damage vary, with neurapraxia affecting only the myelin sheath, axonotmesis involving disruption of the axon and myelin with preserved connective tissue continuity, and neurotmesis leading to complete disruption with loss of continuity

Neurotmesis

- Complete loss of nerve continuity, disruption of all connective tissue layers and axons (Fig. 15.2)

Neurophysiological Methods

Nerve Conduction Studies (NCS)

- Measure amplitude and conduction velocity of motor and sensory nerves.
- Motor nerve conduction studies
  - Electrical stimulation of a peripheral nerve (e.g., median nerve at the wrist) -> recording of a compound motor action potential (CMAP) with surface electrodes over innervated muscle (e.g., M. abductor pollicis brevis)
    CMAP amplitude: Provides an estimate of the number of functioning axons or muscle fibers. It is generally assessed by comparing side-to-side differences, with a pathological finding indicated by an amplitude difference of >50%.
  - Nerve Conduction Velocity (NCV): Normal values are >45 m/s for the upper limb and > 40 m/s for the lower limb. A slowing of NCV below these values suggests myelin damage or significant axonal loss, as larger, fast-conducting

axons—often affected early in many pathologies—contribute to the observed reduction in NCV.

- F-waves, a type of late response, occur when an action potential travels up a motor neuron to the spinal cord, where it is "reflected" in the alpha moto neurons in the ventral horn or root, and then travels back down the peripheral nerve, leading to a muscle response (M-wave). This signal travels through proximal nerve segments, making it sensitive to proximal pathologies, such as polyradicular syndromes like Guillain-Barré syndrome. Key parameters include F-wave latency, F-wave persistence (the frequency of waves returning to produce a measurable muscle response), and F-wave chronodispersion, i.e., in cases of demyelination, axons with different conduction velocities can cause F-waves to produce dispersed muscle responses.
- Sensory nerve conduction studies
  - Electrical stimulation of a peripheral sensory nerve (e.g., ulnar nerve at Dig. V)—> proximal recording of a sensory nerve action potential (SNAP) over ulnar nerve at the wrist (orthodromic technique, i.e., corresponding to the physiological direction of travel of the action potential)

Electromyography (EMG)

- Under physiological conditions, a healthy and relaxed muscle shows no electromyographic (EMG) activity, whether assessed by surface or needle EMG recording.
- Needle EMG allows for the detailed investigation of motor unit morphology and the detection of pathological spontaneous muscle activity.
- Pathological spontaneous muscle activity refers to the electrical activity in muscle fibers or motor units in the absence of voluntary contraction:
  - Fibrillations and positive sharp waves: Electrical discharges from single muscle fibers that are not visible to the naked eye.
  - Fasciculations: Involuntary contractions of entire motor units, which may be visible as muscle twitches.

Note: Spontaneous activity often occurs after axonal damage. Neurapraxia does not lead to spontaneous activity. There is typically a delay between the injury and the onset of spontaneous activity, as Wallerian degeneration and changes in neuromuscular transmission (denervation hypersensitivity) take time to manifest. For instance, spontaneous activity in distal muscles may appear approximately 14–21 days after axonal damage.

- The morphology of motor units provides insight into the underlying pathology:
  - Neurogenic conditions, e.g., peripheral nerve damage: Motor units become larger over time as surviving motor neurons take over more muscle fibers after nerve fiber loss.

- Myopathic conditions: Motor units become smaller as the muscle tissue, the effector organ, is directly affected, leading to a reduced electrical signal.
- Voluntary muscle activity can be assessed by examining two key elements:
  - Activation: The increase in the firing frequency of individual motor units.
  - Recruitment: The appearance of additional motor units to increase muscle force.

Note: Normally, these processes result in a dense pattern of activity (interference pattern), where the activity of different motor units is indistinguishable. In cases of neurogenic damage, this pattern becomes less dense due to the loss of motor units, leading to a phenomenon called "rarefaction". An extreme example is when only a few motor units contribute to muscle strength, as seen in poliomyelitis. In such cases, there is no additional recruitment beyond these single units, which exhibit large amplitudes. Force development is therefore dependent on increasing the firing frequency (activation) of these units.

- EMG may reveal signs of nerve regeneration.

Note: Sprouting from injured peripheral nerve terminals leads to characteristic changes in motor unit morphology. Motor units become smaller, and the regenerating fibers exhibit non-uniform conduction velocities, activating muscle fibers at different times. This results in small, polyphasic units, which are indicative of regenerative sprouting (nascent motor units).

Evoked Potentials

- Record the brain's response to repetitive peripheral stimuli.
- Can be used to assess the integrity of sensory and motor pathways.
- Motor Evoked Potentials (MEP): Magnetic stimulation over the motor cortex—> responses recorded in arm or leg muscles. Assessment of corticospinal tract and peripheral A-alpha fibers.
- Somatosensory Evoked Potentials (SSEP): Electrical stimulation of sensory nerve trunk (e.g., tibial nerve at medial malleolus) or dermatome (e.g., C8 dermatome)—> recording of cortical responses. Assessment of peripheral A-beta fibers and dorsal column pathways.
- SSEPs and MEPs yield robust and reliable responses and can therefore also be used for intraoperative neuromonitoring in spinal surgeries.
- Contact heat evoked potential (CHEP): dermatomal stimulation with a heat probe that elicits a rapid heating ramp—> Assessment of peripheral "small fibers" (mainly A-delta) and the central spinothalamic tract (Fig. 15.3).

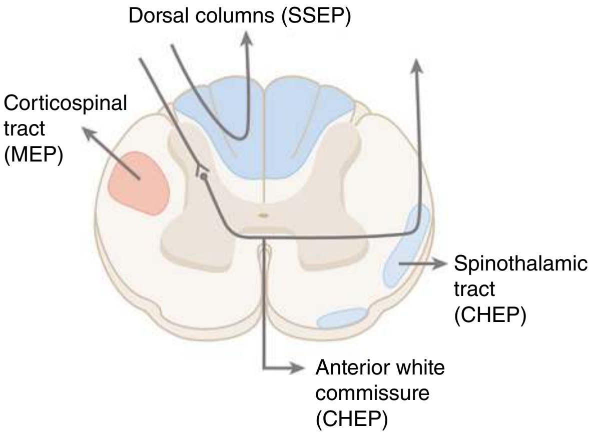

Fig. 15.3 Major spinal ascending and descending pathways, along with assessment techniques, are outlined. Dorsal columns can be assessed using somatosensory evoked potentials (SSEPs), obtained by stimulating a nerve trunk (e.g., the tibial or ulnar nerve) or a specific dermatome (dermatomal SSEPs). Contact heat evoked potentials (CHEPs), generated by cutaneous stimulation with a heat thermode, activate Aδ and C fibers, which synapse in the dorsal horn, cross the midline, and ascend in the anterolateral system via the spinothalamic tract. Both SSEPs and CHEPs can be recorded from the scalp using electroencephalography (EEG). The corticospinal tract is assessed by eliciting a cortical potential through magnetic stimulation, which then travels down the corticospinal tract and synapses in the anterior horn. The final common motor pathway (from alpha motoneuron to muscle) activates muscles, which can be recorded using electromyography. Details on expected electrophysiological findings can be found in Table 15.1

Table 15.1 Typical electrophysiological findings corresponding to the grades of nerve damage

	Neurapraxia	Axonotmesis	Neurotmesis
Mechanism	Compromised myelin sheath Axon intact	Partial or complete axonal damage Anatomical continuity preserved	Complete nerve transection Loss of anatomical continuity
Clinical finding	Paresis Inactivation atrophy Sensory deficits	Paresis Neurogenic atrophy Sensory deficits	Paresis Neurogenic atrophy Sensory deficits
Nerve conduction studies	Conduction block = no propagation of electrical signal across injury site Reduced or absent CMAP/SNAP (when stimulated proximally and recorded distally from myelin pathology)	Partial: Reduced CMAP/SNAP Complete: Absent CMAP/SNAP (after Wallerian degeneration)	Absent CMAP/SNAP (after Wallerian degeneration)

(continued)

Table 15.1 (continued)

	Neurapraxia	Axonotmesis	Neurotmesis
Electromyography	Reduced or absent voluntary activity (interference pattern) No spontaneous activity	Partial: Reduced voluntary activity (interference pattern) Pathological spontaneous activity Complete: Initially no voluntary activity, possible re-occurrence after reinnervation Pathological spontaneous activity	Only pathological spontaneous activity is present, with no EMG activity in response to attempted voluntary contraction No evidence for reinnervation over time

What is the Role of Clinical Neurophysiology?

- Providing objective evidence of nerve damage.
- Assisting in localization of nerve damage, identifying the lesion site (e.g., root, plexus, or peripheral nerve).
- Grading the severity of nerve damage, distinguishing between myelin damage and (in)complete axonal damage.
- Offering insights into prognosis, such as evidence of axonal continuity/conductivity and signs of regeneration.
- Distinguishing focal neuropathies, like entrapment neuropathies, from systemic or generalized conditions, such as polyneuropathies.
- Differentiating between acute and chronic pathologies, for example, identifying new-onset, acute neurogenic changes in lumbar radiculopathy alongside pre-existing chronic neurogenic changes in polyneuropathy (Fig. 15.4).

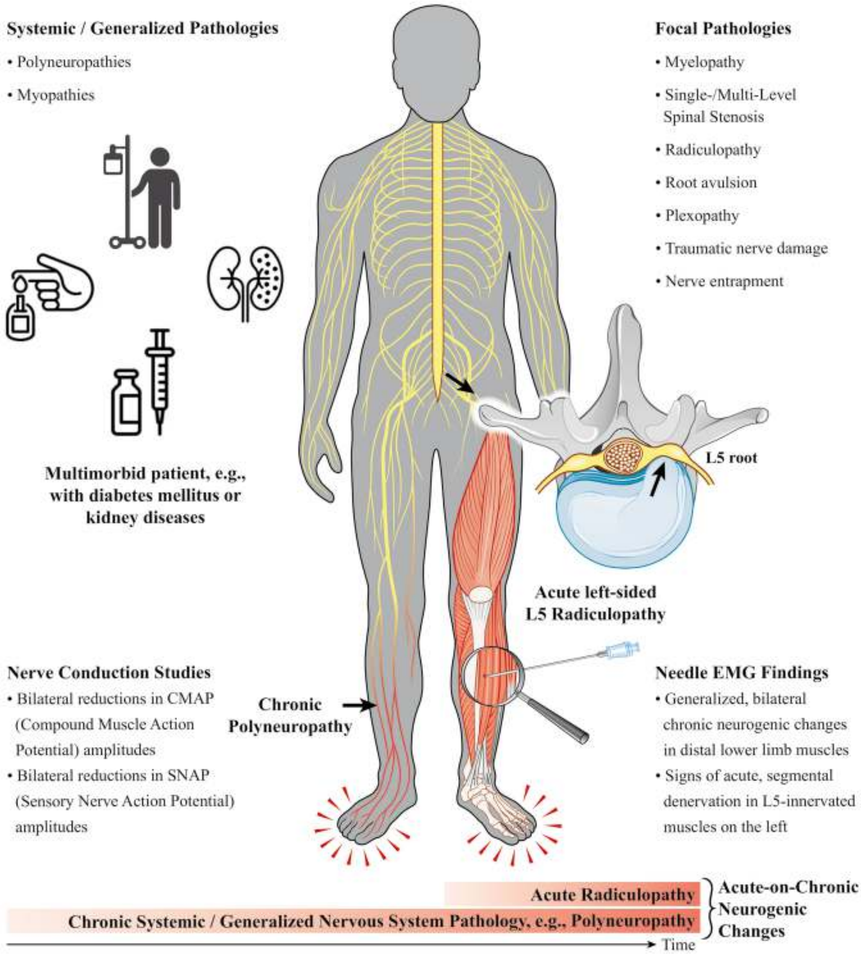

Fig. 15.4 Insights from clinical neurophysiology. Neurophysiological assessments assist in localizing nervous system pathology and distinguishing between focal conditions (e.g., radiculopathy, plexopathy, or focal nerve compression) and generalized nervous system damage, such as that caused by systemic diseases like polyneuropathy due to diabetes. Additionally, clinical neurophysiology enables temporal classification of damage. Electromyography (EMG) is particularly valuable in this context, as it can classify acute axonal damage with signs of denervation, chronic damage with evidence of motor unit reorganization, and signs of regeneration, such as nascent motor units indicative of axonal sprouting

Typical Findings and Assessment Approach in Neuro-Orthopedic Conditions

Radiculopathy

- Pathology of the nerve root, often involving concurrent damage to both sensory and motor roots.
- Preganglionic damage to the peripheral sensory root:
  - Presents as sensory loss in a dermatomal pattern, with preserved sensory nerve action potential (SNAP).
  - Results in impaired dermatomal somatosensory evoked potentials (SSEPs).
- Electromyography (EMG) (diagnostic gold standard): shows reduced recruitment early on and pathological spontaneous activity (after approximately 2–3 weeks) in cases of axonal damage.

Plexopathy

- Sensory nerve conduction studies indicate postganglionic pathology, characterized by sensory loss and the absence of sensory nerve action potential (SNAP).
- Electromyography (EMG) may reveal signs of axonal damage (Fig. 15.5).

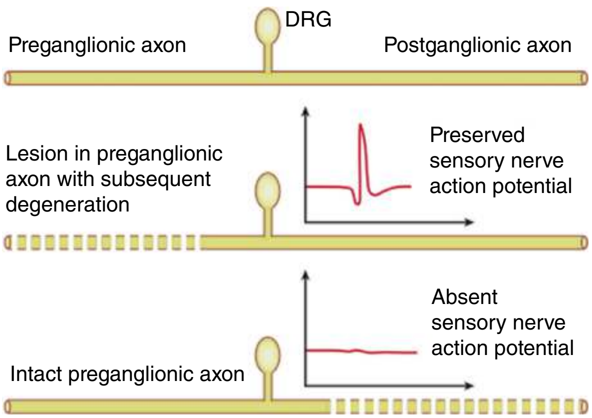

Fig. 15.5 Preganglionic versus postganglionic damage
The dorsal root ganglion (DRG) serves as the trophic support center for both preganglionic and postganglionic axons. In cases of axonal damage, the portion of the axon severed from DRG support undergoes Wallerian degeneration. If this occurs in the preganglionic axon, proximal to the DRG, the distal postganglionic axon still receives trophic support, allowing for a sensory nerve action potential (SNAP) to be recorded despite sensory loss (i.e., the afferent signal is interrupted at the lesion site proximal to the DRG). When the lesion is distal to the DRG, the axon degenerates, resulting in no recordable SNAP. In both situations, clinical findings show sensory loss, but sensory nerve conduction studies help localize the lesion (topodiagnosis), distinguishing preganglionic lesions (e.g., root lesions) from postganglionic ones (e.g., plexus lesions)

Peripheral Nerve Damage (Table 15.1)

Myelopathy

- Depending on the pattern of damage in spinal cord syndromes, different ascending or descending spinal pathways are affected.
- Anterior cord syndrome: Involving roughly the anterior 2/3 of the cord, it affects the anterior horn, anterolateral quadrant (spinothalamic tract), and corticospinal tract, with corresponding loss of pain and/or temperature sensation and motor function. This pattern is seen in cases of anterior spinal cord ischemia or ventral cord compression.
  - MEPs & CHEPs: pathological / abolished
  - SSEPs: preserved
- Posterior cord syndrome: Characterized by loss of light touch and proprioception due to dorsal column involvement, while motor function and pain and/or temperature sensation are preserved. This pattern can be observed in posterior cord compression.
  - MEPs & CHEPs: preserved
  - SSEPs: pathological / abolished
- Hemicord syndrome (Brown-Séquard syndrome): In its classic form, this syndrome affects one side of the cord, commonly in traumatic injuries, such as gunshot or knife wounds. Below the injury, light touch and proprioception are lost ipsilaterally (as dorsal column afferents do not cross at the spinal level), while pain and temperature sensation are lost contralaterally (due to the midline crossing of the spinothalamic tract at the spinal level), resulting in dissociated sensory loss. Incomplete/variable forms of the syndrome are more common in clinical practice.
  - MEPs & SSEPs: pathological/abolished ipsilateral to the lesion
  - CHEPs: pathological/abolished after contralateral stimulation below the lesion
- Central cord syndrome: Here, the commissural fibers of the spinothalamic tract and motor neurons in the anterior horns are affected, leading to segmental deficits in pain and temperature sensation and motor function, confined to the affected segments. This syndrome often presents with a discrepancy between upper limb motor symptoms, e.g., impaired motor function of arms/hand and preserved walking ability. It is typically observed in focal syringomyelia, early stages of cervical myelopathy, or following hyperextension injuries with transient centromedullary cord compression.
  - SSEPs: often unaffected
  - MEPs: affected when recorded from the upper limbs, less so in the lower limbs (as the descending corticospinal tract may bypass centromedullary pathology).

  - CHEPs: pathological / abolished in the affected cervical segments (due to commissural damage to the spinothalamic tract). Stimulation below the lesion may yield preserved cortical responses as the ascending spinothalamic pathway in the anterolateral quadrant may bypass the centromedullary pathology.
- **Note:** Centromedullary cord pathology, as seen in syringomyelia or early degenerative cervical myelopathy, poses a diagnostic challenge since conventional neurophysiological methods, such as MEPs and SSEPs, may bypass the pathology. Contact heat evoked potentials (CHEPs), however, demonstrate high sensitivity to central cord damage where the spinothalamic tract crosses (at the anterior white commissure) and can address this diagnostic gap.