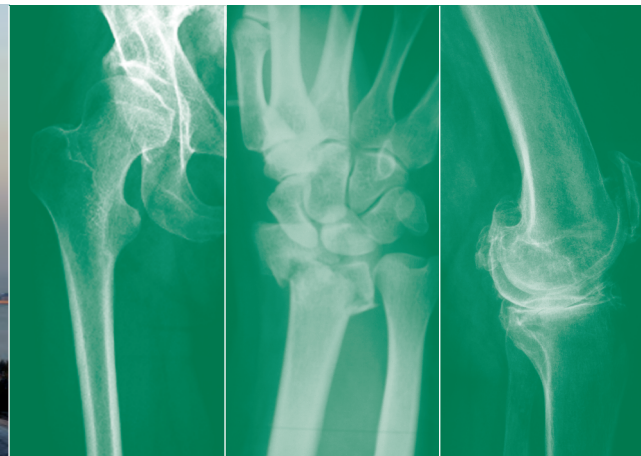


CRC Course

Istanbul, 6 June 2013

www.efort.org/istanbul2013



EFORT SYLLABUS

The Comprehensive Orthopaedic Review Course (CRC)

During the 14th EFORT Congress Istanbul: 6 June 2013

Course highlights

- Basic Science
- Tumour
- Paediatrics
- Spine (incl. Trauma)
- Reconstruction
- Sports knee
- Trauma



EPOS – EFORT Instructional Course – 09 to 11 October, Vienna

Paediatrics: Basic Course II

- Disorders of the upper limb
- Disorders of the knee
- Musculoskeletal infections
- Spine



EFORT Instructional Course – 15 & 16 November, Basel

Osteoarthritis: Joint preserving surgery (JPS) of the lower extremity

- Joint preserving surgery around the hip
- Joint preserving surgery around the knee
- Joint preserving surgery around the ankle

Hands-on Workshops:

- Hip: Osteotomies, arthroscopy, impingement surgery, chondral repair
- Knee: Osteotomies, arthroscopy, chondral repair, ligament repair
- Ankle/Hindfoot: Osteotomies, arthroscopy, chondral repair, ligament repair

Introduction

Welcome



Prof. Dr. Pierre Hoffmeyer

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Welcome to this fifth edition of the EFORT Comprehensive Review Course. This course, held during the 2013 Istanbul Congress, aims to provide a basis for the core theoretical knowledge expected of all orthopaedic trainees at the end of their specialty curriculum. This course must be seen only as an outline that obviously cannot be exhaustive. It is a bare skeleton upon which the participants must add the sinews and the flesh. However, seeing its past success, it certainly addresses the essentials and these will be found in the lectures and in the syllabus. The course is also a convenient way for experienced and senior surgeons to obtain an update of current practices and state of the art information encompassing the whole field of orthopaedics and traumatology. My thanks go to all the course lecturers and authors of the syllabus who put considerable effort into making this CRC course a worthwhile experience. Dr Domizio Suva and his team are to be thanked for their commitment in the organization and running of the CRC.

I also extend my gratitude to the dedicated secretarial, administrative and IT staff, both from the EFORT Head Office and from the Congress Centre in charge of this project. Ms Larissa Welti is to be especially commended for her hard and untiring labors in organizing the Syllabus. This collective effort will certainly be of benefit our participants and ultimately to our patients.

Prof. Dr. Pierre Hoffmeyer, EFORT President



The full attendance of the CRC course entitles to 6 European CME credits (ECMEC's)

The Certificate will be sent to each participants as a PDF file to the registered e-mail address after the course. If you have not yet registered your e-mail account with us, please contact the "Faculty desk" desk in the registration hall.

EFORT does not in any way monitor or endorse the content of any given lecture by any of the speakers during the course. EFORT does not accept any responsibility for the content of any individual session as presented by the speakers nor printed in the syllabus and thus declines all liability of whatever nature arising thereof.

Foreword



Dr. Domizio Suva

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The concept behind the CRC is to provide a complete one-day overview of the essential material for the specialist exam of the European Board of Orthopaedics and Traumatology (EBOT exam). It will enable participants who have mastered this material to be even better prepared to take the exam. Year after year we listen to every idea, and each positive and negative criticism, to make this intensive day even more attractive and useful. This results in a wide and well-balanced scientific programme and an interactive course, while also considering participant energy and receptivity. The themes of the day are drawn from a combination of essential areas of our specialty, combined with the practical knowledge that everyone is expected to have when facing everyday reality. We hope that this fifth CRC course will fulfil your expectations, and eagerly await your feedback!

Dr. Domizio Suva, CRC supervisor



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Infection after prosthetic joint replacement: The best possible complication you can have

In an ever-aging population with higher and higher functional demands the need for prosthetic joint replacement is increasing enormously. After total joint arthroplasty (TJA) considerable improvement of patient mobility, independence in the activities of daily living as well as in the pain scale can be found. Today there are about 4'500'000 artificial joints implanted every year worldwide.

The good results of TJA can of course not be reached in every patient and complications do occur. In almost every scientific article dealing with complications after TJA the most mentioned and most feared is the periprosthetic joint infection (PJI).

A long and painful road is supposed to lie in front of our patients with never healing open wounds, multiple operations, impossibility of eradication of the infection and exploding costs. Cure rates are reported to be as low as 30% and never ending disputes exist to decide whether 1-stage or 2-stage prosthetic exchange is the way to go.

With patient adapted treatment algorithmes based on the patient himself, the type of infection (early vs. delayed vs. late), the appropriate surgical options and systemic and local antibiotic treatment adapted to the type of germ the chances of successful treatment leading to eradication of the infection associated with good functional outcome can be reached in above 93% of the patients.

There is hardly any other complication in orthopaedic surgery with such a high success rate making the treatment of infected total joint arthroplasties an extremely rewarding and interesting job.



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Biomechanics of musculoskeletal tissue – Biomaterials (trauma, prosthesis)

This lecture will consider biomechanics of bones and joints that are applied due to movement by people and then the biomaterial considerations relevant to orthopaedic implants. Biomechanics and biomaterials are obviously both huge subjects but so only the areas of each of importance to orthopaedic surgeons will be considered.

Biomechanics

The mechanics of moving objects, including the human body, are governed by Newton's Laws of Motion. The 1st Law states that "a body will remain in a state of rest, or move at constant velocity, unless acted upon by a force". The 2nd Law states that "a body acted upon by a force will change its velocity in proportion to the applied force". While the 3rd Law says that "when two bodies exert a force upon each other the force acts on the line connecting them and the two force vectors are equal and opposite". What do these laws mean when applied to the human body? Firstly for anything to start moving a force has to act on it, secondly how fast it moves depends on the magnitude of the applied force. The applications of these two laws to the human body are relatively obvious, muscles act by contracting and thus generating a force. What needs to be considered is that shortening a muscle against no resisting force requires no muscle force, what produces the force is the muscle shortening against some form of resistance. The third law is commonly restated as "every action has an equal and opposite reaction" and it is this law combined with the first law that is used in calculating forces generated in the body and how these effect the movement of parts of the body.

The second basic element needing to be considered in biomechanics is the behaviour of levers. Archimedes (287-212BC) is quoted as having said "Give me a fulcrum and I will move the world". We can analyse the behaviour of the human body as a mechanical system by modelling the bones as levers, the weight of components of the body as the loads which need to be moved and the muscles as the applying forces. Levers come in three classes, depending on the relative positions of the fulcrum, the pivot point about which the lever moves, and the load force which the force which needs to be moved and the effort force which is the

force doing the moving, that is the muscle force in the body (Fig. 1). An example of a Class I lever is the child's seesaw, where the fulcrum is in the centre and the two people are the load and effort forces. In the human body there are few Class I levers, one example is at the head where the C1 vertebra acts as the fulcrum, mass of the head is the load force and is anterior to this fulcrum, while the extensor muscles of the neck supply the effort force. In Class II and III levers the fulcrum is at one end of the lever and the load and effort forces are to the same side of the fulcrum. In Class II the load force is between the fulcrum and the effort force while in Class III the effort force is between the load force and the fulcrum and the Class III lever is the most common type of lever found in the body. As the forces multiplied by their distance from the fulcrum have to balance where the effort force is nearer the fulcrum than the load force the effort force has to be higher than the load force.

Force is measured in Newtons (N) in the SI (Système International) unit scheme. 1 Newton is the force exerted by 1 kg (kilogram) when accelerated at 1ms^{-2} , thus force exerted by 1kg on earth is 9.81N as the acceleration due to gravity on earth is 9.81ms^{-2} . One simple way to remember the value of a Newton is that the force exerted on earth by a typical apple weighing about 100g is about 1N.

In analysing the biomechanics of the body we can consider a simple action, holding a weight in the hand with the forearm held horizontal and the upper arm horizontal (Fig. 2). The weight is acting downwards and to be held still the vertical forces in the arm through to the body must be equal and the moments about the elbow joint must be equal. If we assume the weight of the lower arm is 20N and the weight held in the hand is 10N (thus approximately 2kg and 1 kg mass respectively) and that the length from the elbow joint to the hand is 300mm and to the the centre of mass of the forearm is 130mm with the line of action of the biceps muscle being 50mm. We can calculate that the force in the biceps has to be 112N.

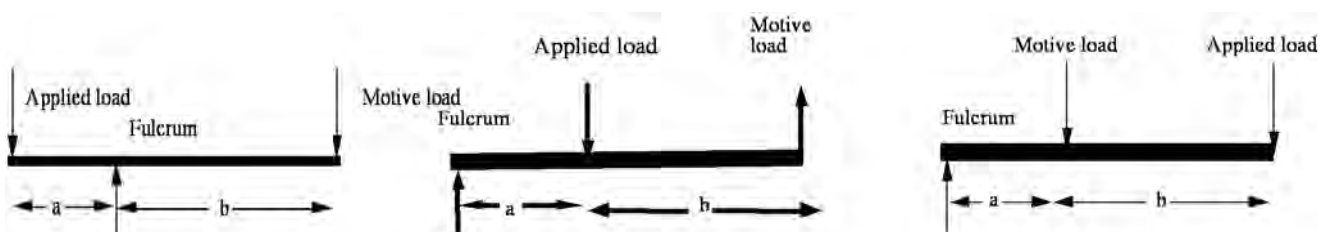


Fig. 1 Lever types

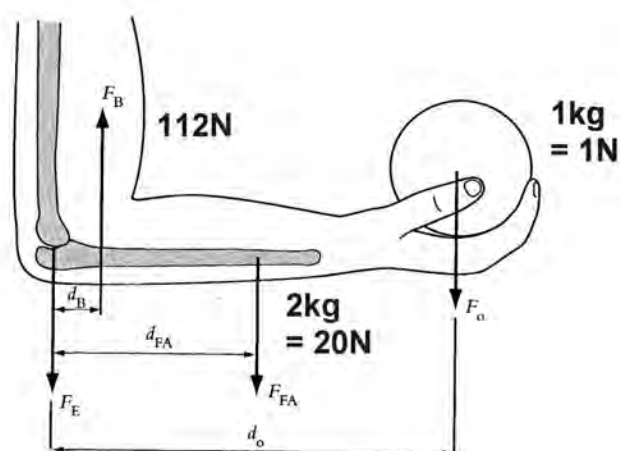


Fig. 2 Forces involved in carrying a 1kg weight in the hand.

If we apply similar calculations to a person standing on one leg and making appropriate assumptions of distances in the body then we can calculate that the load on the femoral head is 2.58 times the subject's body weight and that the forces in the abductor muscles is 1.77 times body weight. The calculations have to include that the distance between the hip joint and the line of action of the hip abductors must be taken as perpendicular to the line of the muscle (Fig. 3). If these simple calculations are compared with the data from an instrumented hip prosthesis [Bergmann, Graichen et al. 1993] then it can be seen that the forces calculated using a simple two dimensional analysis can give a good estimate of the actual forces occurring in vivo. These types of analysis can be applied throughout the body.

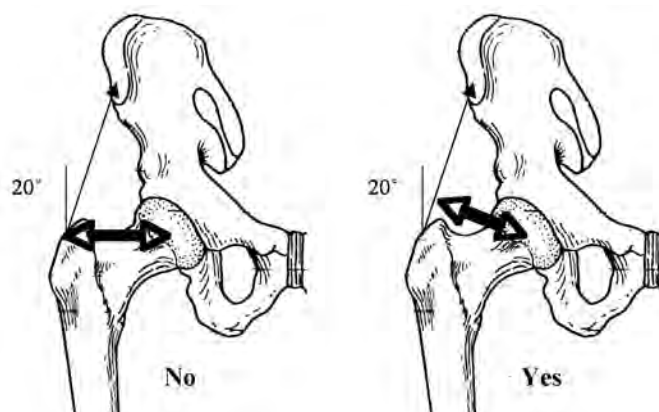


Fig. 3 Incorrect and correct methods of calculating the moment about a hip joint

The final factor to be considered is the number of load cycles applied during walking and other activities. [Wallbridge and Dowson 1982] found that the number of load cycles applied to the legs dropped from an average of 2 million per year when people were in their 20s down to 0.5 million in their 80s. The interesting factor was that they also measured some joint replacement patients and found they these people were applying more load cycles than the average for their age group. In the hand Joyce and Unsworth (2000) estimated similar number of load cycles for the fingers, but estimated that the loading the fingers considered of two groups, high movement with low loads interspersed with limited motion but high loads.

Biomaterials

"A biomaterial is a non viable material used in a medical device, intended to interact with biological systems" according to [Williams 1999] and to function successfully it needs to be biocompatible, that is it "has the ability to perform with an appropriate host response in a specific application" [Williams 1999]. The behaviour of a material in the body depends on two factors: the effect the implant material has on the body and the effect the body has on the implant material. The reaction to an implanted material (and thus implant) can be divided into four types: **Toxic**, that is it kills cells in contact with or away from implant, **Bioinert**, that is produces no response by the body and which never truly occurs as there is always a response to implantation, but when the response is minimal the material is called bioinert. **Bioactive**, which is encourages an advantageous response from the body and this will depend on where the implant is placed in the body and thus the required bioactive response and finally **Biodegradable** where the implant breaks down in the body to non-toxic components which are excreted by the body. The effects the body has on an implant can be defined as the response of the material to the internal environment of the body from the physiological environment, protein absorption, which is a particular problem with polymers, degradation whether required or not and finally corrosion, which particularly applies to metal implants.

When we are considering the mechanical properties of a material these are measured using stress, which is the force per unit area and strain which is a measure of the change in dimension and the ratio of these two is called Young's Modulus or stiffness. Further important mechanical factors are the ultimate strength, that is how much force a material can take before it breaks, the ductility, the amount a material deforms before it breaks and toughness which is a measure of how fast a crack progresses through a material once fracture starts. When choosing a material for use in the body one of the considerations is the mechanical properties of the material compared to those of the body component being replaced.

Cortical bone has Young's modulus of 7-25GPa, strength of 50-150MPa and a fracture toughness of 2-12 MN m^{-3/2}, while cancellous bone has modulus of 0.1-1.0GPa and compressive strength of 1-10MPa [Currey 1998; Currey 2006]. Cortical and cancellous bone are both brittle, but being able to react to their mechanical environment can be considered to be "smart" materials. **Cancellous bone** behaves as a typical foam, that is increasing the density (or decreasing the porosity) increases the stiffness and strength [Gibson and Ashby 1999]. Ligaments and tendons have non-linear mechanical properties with the stiffness increasing as the load increases.

Materials can be defined into four basic groups: **metals**, ceramics, polymers and composites. Metals are normally used as alloys, that is small or larger amounts of other atoms are added to tailor the properties. Metals are reasonably stiff, ductile, that is they deform before they fracture, they generally have good fatigue properties and can be plastically deformed, that is they can be bent into new shape and remain in that shape as is used in the moulding of fracture fixation devices. The major metals used in orthopaedics are the stainless steels, the cobalt chrome alloys, titanium and its alloys. Stainless steel used in medical applications is usually 316 or 316L and consists of 18% chromium, 13% nickel, 2.5% molybdenum, and the rest is iron. The presence of the chromium leads to the alloy being "stainless" as a chromium oxide layer is produced on the surface, which does not easily oxidise further. Stainless steel has a Young's modulus of 210 GPa, is ductile, can be

deformed (cold worked) and the fatigue properties are acceptable. Cobalt Chrome alloy consists of 27–30% chromium, 5–7% molybdenum with the rest cobalt. This formulation means that there is no nickel which is important for those patients who are nickel sensitive. Nickel sensitivity rates are variable within Europe and can reach over 20% in the Scandinavian population. Cobalt chrome has a Young's modulus of 230 GPa, a higher fatigue limit than Stainless Steel and has good wear properties. There are three major groups of titanium: commercially pure which is >99% titanium, Ti-6%Al-4%V which is therefore 90% titanium, 6% aluminium and 4% vanadium and finally the shape memory alloys which are approximately 50:50 titanium:nickel, with the exact composition being used to control the temperature at which the shape memory effect occurs. Most titanium alloys have a lower Young's modulus of 106 GPa, the wear debris is black in body thus looks unsightly to the surgeon, but this wear debris is not known to produced significant extra problems compared to other wear debris which may be as present in the body but is not as obvious to the surgeon. Titanium is notch sensitive, that is any notches or other sharp corners lead to significant reductions in the fatigue life, and also is heat treatment sensitive. [Cook, Thongpreda et al. 1988] showed that with appropriate heat treatment the fatigue limit, which is the fatigue load at which the specimen does not break, was 625MPa, but if a porous coating was applied with an inappropriate heat treatment this fatigue limit was reduced to 200MPa. More recently newer titanium alloys are being developed which have yet lower Young's moduli, at 42GPa, thus bringing their stiffness's closer to those of cortical bone [Hao, Li et al. 2007].

Bioceramics can be divided into 2 major groups, the bioinert which are principally zirconia (ZrO_2) and alumina (Al_2O_3) and the bioactive mainly hydroxyapatite ($Ca_{10}(PO_4)_6(OH)_2$) and tricalcium phosphate ($Ca_3(PO_4)_2$). The bioinert ceramics are principally used for articulating surfaces as either ceramic-on-polymer or ceramic-on-ceramic. Initially Al_2O_3 was preferred as ZrO_2 can be morphologically unstable but now PSZ (Partially Stabilised Zirconia) is available. Al_2O_3 has been used by Sedel in Paris for more than 30 years as ceramic-on-ceramic hip replacements [Nizard, Pourreyron et al. 2008]. In the initial implants the individual grains in the ceramics components were large and failures occurred, now grain size is reduced and failures have reduced to >1:2000. However, very close tolerances on head-cup dimensions are needed so matched pairs are supplied to reduce the fracture risk.

Bioactive ceramics are used in five major applications: bulk implants, that is space filling implants, porous when used as implants for ingrowth or scaffolds for tissue engineering, granules used to bulk out or to replace bone graft, coatings which are either plain HA or HA+TCP (also called biphasic CaP - BCP) and finally as injectable where the calcium phosphate, with or without some calcium sulphate and other additives, is mixed in the operating theatre, injected into the body and sets *in situ*.

Polymers used in orthopaedics are primarily ultrahigh molecular weight polyethylene (UHMWPE), polymethylmethacrylate (PMMA), other methacrylates, polyesters, poly(glycolic acid) and poly(lactic acid) and finally the hydrogels. Polyethylene was introduced by Sir John Charnley in 1960 as the first metal-on-polymer joint replacement. Charnley initially used polytetrafluoroethylene (PTFE) as the bearing surface for his hip replacements and found such drastic wear that after 1 year joint motion was seriously reduced. He originally High Density Polyethylene (HDPE), which was replaced in 1970s with Ultra High Molecular Weight Polyethylene (UHMWPE) and now a range of Enhanced Polyethylene (partially cross linked) or heavily irradiated PE are used to reduce the production of wear particles. PE is used as concave bearing surfaces

against metal or ceramics such as acetabular cups, the tibial plateaux of knee replacements, patella buttons etc. PMMA bone cement is used to fix (grout) joint replacements in place thus is used to space fill. It is supplied as a two phase materials, the powder phase is pre-polymerised polymethylmethacrylate beads plus benzoyl peroxide which initiates the polymerisation of the liquid monomer with a radiopacifier in the form of barium sulphate or zirconia. The liquid phase is methylmethacrylate monomer plus N,N dimethyl-p-toluidene. It is mixed in theatre when polymerisation starts due to the benzoyl peroxide producing free radicals that initiate the polymerisation of the MMA monomer. The rationale for the use of pre-polymerised beads and monomer is that the polymerisation process is exothermic, that is produces heat, and the monomer shrinks by approximately 21% during the polymerisation process. By using about $\frac{2}{3}$ pre-polymerised and $\frac{1}{3}$ monomer the exotherm and shrinkage are both reduced. When in the "dough" state it is inserted into patient, under pressure and then implant pushed into the cement. Initially cement was hand mixed but now mixing is always performed under vacuum as this reduces the porosity [Wang, Franzen et al. 1993] thus improving the mechanical properties and reduces the exposure of theatre staff to the monomer fumes. The mechanical properties of bone cement are very dependent on the formulation and preparation methods, for example in a study using the range of bone cements available in the late 1990s Harper and Bonfield found a factor of over 50 in the fatigue lives of bone cements thought to have good mechanical properties and the really poor cements such as Boneloc® had yet worse properties (Fig. 4). Opacifiers are added to bone cement as being a polymer it is not visible on radiographs, but the opacifiers provide their own problems, acting as brittle fillers and thus reducing the mechanical properties and when the cement breaks up can become embedded in articulating joints increasing the wear in the joint and the presence of opacifier particles can lead to resorption of bone around the implant [Sabokbar, Fijikawa et al. 1997]. Finally, antibiotics are added prophylactically to bone cement to reduce the risk of infection [Jiranek, Hanssen et al. 2006].

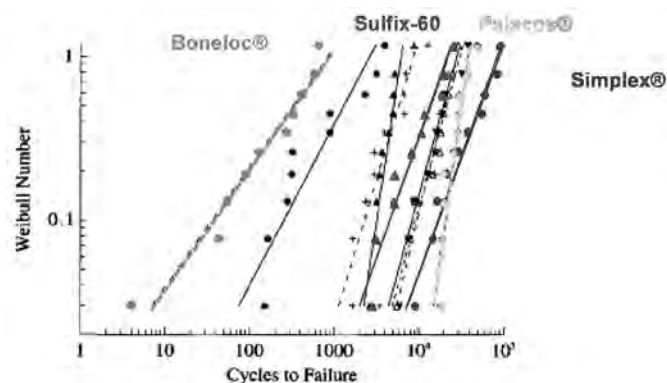


Fig. 4 Comparison of the fatigue lives of a range of bone cements (from Harper and Bonfield, 2000)

The major degradable polymers used are Poly(lactic acid) PLA and Poly(glycolic acid) PGA. Chemically these break down to lactic and glycolic acid, which the body breaks down to CO_2 and H_2O and excretes. Typically PGA is used in degradable sutures as PGA has fast degradation within the body. Due to its lower degradation rate PLA is starting to be used for fracture fixation in low load bearing applications the form of internal fixation plates. The current problems with degradable polymers is the strength and degradation rate. In attempts to improve the strength fibre reinforcement and ceramic reinforcement has been used [Bleach, Nazhat et al. 2002; Huttunen, Törmälä et al. 2008].

Composites are two phase materials where the two phases can be seen as separate either with the naked eye or using a microscope, which is the two phases can be differentiated on the micron scale. Artificial composites are generally used to optimise the properties of the two phases. The individual phases interact be it mechanically or functionally. The major groups of composites are polymer reinforced with ceramics/glasses, polymers reinforced with different polymer or polymer form such as drawn fibres of a polymer in a amorphous matrix of the same polymer, an example is the PLLA in PLDLA used in some degradable fracture fixation plates. Ceramic metal composites, which are also known as metal matrix composites a few of these have been developed for medical applications and finally ceramic-ceramic composites, but neither of these but have as yet reached clinical applications. In a composite there is normally one continuous phase called the matrix and a second phase called the filler distributed in the matrix as particles, fibres or fabric. Generally phases chosen as when specific properties of one phase are "good" in the other they are "bad", but by getting right balance of phases can balance the properties to optimise the material. Applications of biocomposites in medical applications is beginning to increase [Tanner 2010]. The earliest ones were bioinert, but now bioactive implants are beneficially interacting with the human body.

Conclusions

In conclusion when placing implants in the body there are two major interacting factors that need to be considered for the survival of an implant in the body. The first is how heavily it is being loaded and the second is what it is made of. Without appropriate interactions between both of these factors an implant will not be successful.

References

1. Bergmann, G., F. Graichen, et al. (1993). "Hip-joint loading during walking and running, measured in 2 patients." *Journal of Biomechanics* 26(8): 969-990.
2. Bleach, N. C., S. N. Nazhat, et al. (2002). "Effect of Filler Content on Mechanical and Dynamic Mechanical Properties of Particulate Biphasic Calcium Phosphate Polylactide Composites." *Biomaterials* 23(7): 1579-1585.
3. Cook, S. D., N. Thongpreda, et al. (1988). "The effect of post-sintering heat treatments on the fatigue properties of porous coated Ti-6Al-4V alloy." *Journal of Biomedical Materials Research* 22(4): 287-302.
4. Currey, J. D. (1998). "Mechanical properties of vertebrate hard tissues." *Proceedings of the Institution of Mechanical Engineers: Part H Engineering in Medicine* 212-H(6): 399-412.
5. Currey, J. D. (2006). *Bones: Structure and Mechanics*, Princeton University Press.
6. Gibson, L. J. and M. F. Ashby (1999). *Cellular Solids*. Oxford, Pergamon Press.
7. Hao, Y. L., S. J. Li, et al. (2007). "Elastic deformation behaviour of Ti-24Nb-4Zr-7.9Sn for biomedical applications." *Acta Biomaterialia* 3(2): 277-286.
8. Harper, E.J., Bonfield, W. (2000) "Tensile Characteristics of Ten Commercial Acrylic Bone Cements. *Journal of Biomedical Materials Research* 53-A(5): 605-616. Errata 2001;58-A(2):216.
9. Huttunen, M., P. Törmälä, et al. (2008). "Fiber-reinforced bioactive and bioabsorbable hybrid composites." *Biomedical Materials* 3(3).
10. Jiranek, W. A., A. D. Hanssen, et al. (2006). "Antibiotic-loaded bone cement for infection prophylaxis in total joint replacement." *Journal of Bone and Joint Surgery* 88-A(11): 2487-2500.
11. Joyce, T. J. and A. Unsworth (2000). "The design of a finger wear simulator and preliminary results." *Proceedings of the Institution of Mechanical Engineers: Part H Engineering in Medicine* 214-H(5): 519-526.
12. Nizard, R. S., D. Pourreyron, et al. (2008). "Alumina-on-alumina hip arthroplasty in patients younger than 30 years old." *Clinical Orthopaedics and Related Research* 466: 317-323.
13. Sabokbar, A., Y. Fijikawa, et al. (1997). "Radio-opaque agents in bone cement increase bone resorption." *Journal of Bone and Joint Surgery* 79B(1): 129-134.
14. Tanner, K. E. (2010). *Hard tissue applications of biocomposites. Biomedical Composites*. Ed L. Ambrosio. Cambridge, UK, Woodhead Publishers.
15. Wallbridge, N. and D. Dowson (1982). "The walking activity of patients with artificial hip joints." *Engineering in Medicine* 11(3): 95-96.
16. Wang, J. S., H. Franzen, et al. (1993). "Porosity of Bone Cement reduced by mixing and collecting under vacuum." *Acta Orthopaedica Scandinavica* 64(2): 143-146.
17. Williams, D. F. (1999). *The Williams Dictionary of Biomaterials*. Liverpool, Liverpool University Press.



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Metabolic bone disease

Metabolic bone disease is an umbrella term referring to abnormalities of bones caused by a broad spectrum of disorders. Most of these disorders are caused by abnormalities of minerals (e.g. calcium, phosphorus, magnesium, vitamin D) leading to dramatic clinical disorders that are commonly reversible once the underlying defect has been treated. A different group comprises genetic bone disorders where there is a defect in a specific signaling system or cell type that causes the bone disorder.

1. Bone structure and function

The bony skeleton (206 bones in the adult) not only provides structural integrity and strength to the body, it protects vital organs and plays a very critical role in the hematological system in the body. In addition, it is responsible for the mineral homeostasis, mainly storage of essential minerals like calcium (1-2kg), phosphorus (1kg), magnesium, and sodium.

1.1 Cortical and trabecular bone

The hard outer layer of bones is composed of compact bone tissue (porosity 5-30%) and accounts for 80% of the total bone mass. The interior is filled with trabecular (cancellous) bone tissue, an porous network that make the overall organ lighter (porosity 30-90%) and contains blood vessels and marrow. Trabecular bone accounts for only 20% of total bone mass but has nearly ten times the surface area of cortical bone. Because osteoblasts and osteoclasts inhabit the surface of bones, trabecular bone is more active, more subject to bone turnover, to remodeling.

The majority of bone is made of the bone matrix that has inorganic elements (65%) and organic matrix (35%).

1.2 Inorganic component

The bone mineral is formed from calcium hydroxyapatite ($\text{Ca}_{10}(\text{PO}_4)_6\text{OH}_2$) and provides bone strength and hardness. It acts as a storehouse for 99% of the body's calcium, phosphate as well as sodium and magnesium.

1.3 Organic component

The organic part of matrix consists mainly of Type I collagen (90%), synthesised intracellularly as tropocollagen and then exported, forming fibrils. According to the pattern of collagen forming the osteoid two types of bone can differentiate: the mechanically weak woven bone with collagen deposit in random weave or the mechanically strong lamellar bone with a regular parallel alignment of collagen.

When osteoblasts produce osteoid rapidly woven bone occurs. This is the case in the fetal skeleton especially at growth plates, in the fracture healing process and with Paget's Disease. Woven bone is weaker with a smaller number of randomly oriented collagen fibers, but resists forces

equally from all directions. The presence of woven bone in the adult is always pathological.

Lamellar bone gradually replaces woven bone during growth or after a fracture (bone substitution). Lamellar bone formation is much slower (1-2µm per day) but leads to a much stronger consistence. It consists of many collagen fibers parallel to other fibers in the same layer, in alternating layers they run in opposite directions.

1.4 Cellular structure

The bone-forming cells constitute only 2% of bone weight but are responsible for formation and maintenance of bone [5].

1.4.1 Osteoprogenitor cells

Osteoprogenitor cells are pluripotential mesenchymal stem cells differentiating into osteoblast when stimulated.

1.4.2 Osteoblasts

Osteoblasts are mononucleate bone-forming cells located on the surface of bone. They synthesize, transport, and arrange matrix proteins (collagen type I, proteoglycans, glycoproteins) and initiate mineralization by producing osteoid, a protein mixture. They have receptors for parathyroid hormone, vitamin D, estrogen, cytokines, growth factors etc. Bone lining cells are essentially inactive osteoblasts. They cover all of the available bone surface and function as a barrier for certain ions. Osteoblasts are immature bone cells, and eventually become entrapped in the bone matrix to become osteocytes. Estrogen and PTH stimulate the activity of osteoblasts.

1.4.3 Osteocytes

Osteocytes are terminally differentiated bone-forming cells forming a cellular network by connecting with each other and with osteoblasts on the bone surface through canaliculi. They are actively involved in bone turnover including formation of bone, matrix maintenance and calcium and phosphorus homeostasis. Osteocytes play also an important role in sensing extracellular mechanical stress loaded on the bone. These mechanical signals may regulate the overall metabolism of cells in bone tissue. Osteocytes are stimulated by calcitonin and inhibited by PTH.

1.4.4 Osteoclasts

Osteoclasts are responsible for bone resorption. They are large, multi-nucleated cells located on bone surfaces derived from a monocyte stem-cell. Because of their origin they are equipped with phagocytic-like mechanisms similar to circulating macrophages. They migrate to discrete bone surfaces and upon arrival, active enzymes, such as tartrate resistant acid phosphatase, are secreted against the mineral substrate and thus they break down bone to its elemental units.

1.5 Remodeling

Remodeling or bone turnover is a constant process right from the embryonic age to the end of life [5]. Each year 18% of the total skeletal calcium is deposited and removed. This cycle of bone resorption and formation is a process carried out by the basic multicellular unit (BMU), composed of a group of osteoclasts and osteoblasts, and coupled together via paracrine cell signalling. A micro-crack starts the process, the osteocytes sense damage and send signals into the marrow space. Preosteoclasts turn into multi-nucleated osteoclasts and start resorption, meanwhile preosteoblasts turn into osteoblasts and start forming osteoid which then mineralizes. The rate of mineralization varies, but there are normally 12 -to 15- days between formation of matrix and its mineralization.

This delicate balance in bone remodelling results in no net change in skeletal mass. However, osteoblasts can increase bone mass through secretion of osteoid and by inhibiting the ability of osteoclasts to break down osseous tissue. Peak bone mass is achieved in early adulthood, later 5 to 10% of bone mass are remodeled each year. Around the ages of 30-35, cancellous or trabecular bone loss begins. Women may lose as much as 50%, while men lose about 30%.

The purpose of remodeling is to regulate calcium homeostasis, repair micro-damaged bones and to shape and sculpture the skeleton during growth and later. Repeated stress, such as weight-bearing exercise or bone healing, results in the bone thickening at the points of maximum stress (Wolff's law).

1.6 Paracrine cell signaling

A number of chemical factors can either promote or inhibit the activity of the bone remodeling cells. In addition, the cells also use paracrine signalling to control the activity of each other [5].

Bone building through increased secretion of osteoid by the osteoblasts is stimulated by the secretion of growth hormone, thyroid hormone as well as estrogens and androgens. These hormones also promote increased secretion of osteoprotegerin.

Osteoblasts can also secrete a number of cytokines that promote reabsorption of bone by stimulating osteoclast activity and differentiation from progenitor cells. Stimulation from osteocytes as well as vitamin D and parathyroid hormone induce osteoblasts to increase secretion of RANK-ligand and interleukin 6, cytokines then stimulate increased reabsorption of bone by osteoclasts. They also affect osteoblasts to increase secretion of macrophage colony-stimulating factor, which promotes the differentiation of progenitor cells into osteoclasts, and decrease secretion of osteoprotegerin.

The amount of osteoclast induced bone resorption is inhibited by calcitonin and osteoprotegerin. Calcitonin is produced by parafollicular cells in the thyroid gland, and can bind to receptors on osteoclasts to directly inhibit osteoclast activity. Osteoprotegerin is secreted by osteoblasts and is able to bind RANK-L, inhibiting osteoclast stimulation.

2. Metabolic bone disease

- Diseases associated with abnormal matrix = Disorders of osteoblasts
- Diseases associated with abnormal remodelling = Disorders of osteoclasts
- Diseases associated with abnormal mineral homeostasis

2.1 Diseases associated with abnormal matrix

2.1.1 Osteogenesis imperfecta

Osteogenesis imperfecta (brittle bone disease) is an autosomal dominant genetic defect, but it can also be caused by a de novo mutation [13]. People with OI are born with defective connective tissue, or without the ability to synthesize it, usually because of a deficiency of Type-I collagen. Qualitatively normal collagen is built in decreased amounts because abnormal collagen molecules are overproduced. Recent works suggest that OI must be understood as a multi-scale phenomenon, which involves mechanisms at the genetic, nano-, micro- and macro-level of tissues [3].

Clinical expression

Osteogenesis imperfecta affects structures rich in type I collagen (joints, eyes, ears, skin, and teeth). There is a wide spectrum of expression of these disorders but all are marked by extreme skeletal fragility. The most common types I and IV are characterized by:

- Discoloration of the sclera, appearing in blue-gray color
- Slight protrusion of the eyes
- Early loss of hearing in some children
- Multiple fractures especially before puberty
- Slight spinal curvature
- Mild to moderate bone deformity
- Poor muscle tone in arm and legs
- Laxity of the joints

Treatment

At present there is no cure for OI. Therefore the main aim is to increase the overall bone strength to prevent fracture and maintain mobility. Physiotherapy is applied to improve muscle strength and mobility in a gentle manner, while minimizing the risk of fracture. This often involves hydrotherapy and the use of support cushions to improve posture. Bisphosphonates are being increasingly administered to increase bone mass and reduce the incidence of fracture [14,18].

2.1.2 Mucopolysaccharidoses

The mucopolysaccharidoses are part of the lysosomal storage disease group, a group of metabolic disorders caused by the absence or malfunctioning of lysosomal enzymes needed to break down glycosaminoglycans. Over time, these glycosaminoglycans collect in the cells, blood and connective tissues. The result is permanent, progressive cellular damage which affects appearance, physical abilities, organ and system functioning, and in most cases mental development. Skeletal manifestations result from abnormalities in hyaline cartilage caused by a deficiency in the acid hydrolases required to degrade cartilage matrix [1].

Clinical expression

The mucopolysaccharidoses share many clinical features but have varying degrees of severity. These features may not be apparent at birth but progress as storage of glycosaminoglycans affects bone, skeletal structure, connective tissues, and organs.

On the skeletal site short stature, short stature with disproportionately short trunk (dwarfism), malformed bones and chest wall abnormalities are typical. Short hands, progressive joint stiffness, and carpal tunnel syndrome can restrict hand mobility and function.

Treatment

At present there is no cure. Medical care is directed at treating systemic

conditions and improving the person's quality of life. Changes to the diet will not prevent disease progression. Physical therapy and daily exercise may delay joint problems and improve the ability to move.

2.1.3. Osteoporosis

Osteoporosis is a major public health threat which afflicts 1 in 3 women and 1 in 12 men over the age of 50 worldwide. It is responsible for millions of fractures annually, mostly involving the lumbar vertebrae, hip, and wrist.

Osteoporosis is defined by the WHO as "a systemic skeletal disease characterized by low bone mass and micro-architectural deterioration of bone tissue, leading to enhanced bone fragility and a consequent increase in fracture risk" [20].

The form of osteoporosis most common in women after menopause is referred to as postmenopausal osteoporosis. Senile osteoporosis occurs after age 75 and is seen in both females and males at a ratio of 2:1. Secondary osteoporosis may arise at any age and affects men and women equally, resulting from chronic predisposing medical problems or disease, or prolonged use of medications such as glucocorticoids.

Pathophysiology

The underlying mechanism in all cases of osteoporosis is an imbalance between bone resorption and bone formation [11]. The three main mechanisms by which osteoporosis develops are an inadequate peak bone mass (insufficient development of mass and strength during growth), excessive bone resorption and inadequate formation of new bone during remodeling.

The rate of bone resorption is determined by hormonal factors: lack of estrogen (menopause) increases bone resorption as well as decreasing the deposition of new bone that normally takes place in weight-bearing bones. Parathyroid hormone (PTH, parathormone) increases bone resorption to ensure sufficient calcium in the blood, calcitonin, a hormone generated by the thyroid, increases bone deposition.

Calcium metabolism plays also a significant role in bone turnover, and deficiency of calcium and vitamin D leads to impaired bone deposition; in addition, the parathyroid glands react to low calcium levels by secreting PTH.

In osteoporosis not only bone density is decreased, but the micro-architecture of bone is disrupted. The weaker spicules of trabecular bone break ("microcracks"), and are replaced by weaker bone. Common osteoporotic fracture sites, the wrist, the hip and the spine, have a relatively high trabecular bone to cortical bone ratio. These areas rely on trabecular bone for strength, and therefore the intense remodeling causes these areas to degenerate most when the remodeling is imbalanced.

Risk factors

The most important risk factors for osteoporosis are advanced age (in both men and women) and female gender [19]; While these are nonmodifiable risk factors other can potentially be modified:

- Vitamin D deficiency is associated with increased Parathyroid Hormone (PTH) production leading to bone resorption.
- Malnutrition including low dietary calcium and/or phosphorus, magnesium, zinc, boron, iron, fluoride, copper, vitamins A, K, E and C (and D where skin exposure to sunlight provides an inadequate supply).
- Physical inactivity can lead to significant bone loss since bone remodeling occurs in response to physical stress, and weight bearing exercise can increase peak bone mass achieved in

adolescence.

- Tobacco smoking inhibits the activity of osteoblasts, and results also in increased breakdown of exogenous estrogen, lower body weight and earlier menopause.
- Excess alcohol (alcohol intake greater than 3 units/day) increases risk significantly.

Many diseases and disorders as well as certain medications have been associated with an increase in osteoporosis risk:

- Hypogonadal states with estrogen (oophorectomy, premature ovarian failure, anorexia nervosa, Turner syndrome, Klinefelter syndrome) or testosterone deficiency.
- Endocrine disorders including Cushing's syndrome, hyperparathyroidism, thyrotoxicosis, hypothyroidism, diabetes mellitus type 1 and 2, acromegaly and adrenal insufficiency. In pregnancy and lactation, there can be a reversible bone loss.
- Nutritional and gastrointestinal disorders including coeliac disease, Crohn's disease, lactose intolerance, gastric or bowel resection.
- Rheumatologic disorders like rheumatoid arthritis, ankylosing spondylitis, systemic lupus erythematosus, either as part of the disease or because of corticosteroid therapy.
- Renal insufficiency.
- Steroid-induced osteoporosis (SIOP) especially in patients taking the equivalent of more than 30 mg hydrocortisone (7.5 mg of prednisolone) in excess of three months.
- Enzyme-inducing antiepileptics (eg. Barbiturates, phenytoin) probably accelerate the metabolism of vitamin D
- L-Thyroxine over-replacement in a similar fashion as thyrotoxicosis.
- Hypogandism-inducing drugs, eg. aromatase inhibitors (used in breast cancer), methotrexate, depot progesterone and gonadotropin-releasing hormone agonists.
- Proton pump inhibitors lowering the production of stomach acid, so interfering with calcium absorption.
- Anticoagulants.
- Chronic lithium therapy.

Falls risk

The risk of falling is increased by balance disorder, movement disorders (e.g. Parkinson's disease), impaired eyesight (e.g. due to glaucoma, macular degeneration), dementia, and sarcopenia (age-related loss of skeletal muscle). Transient loss of postural tone due to cardiac arrhythmias, vasovagal syncope, orthostatic hypotension and seizures leads to a significant risk of falls. Previous falls and gait or balance disorder are additional risk factors. Removal of obstacles and loose carpets in the living environment may substantially reduce falls.

Clinical expression

Osteoporosis itself has no specific symptoms; its main consequence is the increased risk of so called fragility fractures, since they occur in situations where healthy people would not normally break a bone. Typical osteoporotic fractures occur in the vertebral column, rib, hip and wrist.

Fracture Risk Calculators assess the risk of fracture based upon several criteria, including BMD, age, smoking, alcohol usage, weight, and gender. Recognised calculators are the FRAX and the DVO fracture risk assessment.

Diagnosis

Dual energy X-ray absorptiometry (DXA) is considered the gold standard for the diagnosis of osteoporosis. According to the World Health Organization, osteoporosis is diagnosed when the bone mineral density is less than or equal to 2.5 standard deviations below that of a young adult reference population [20]. This is translated as a T-score

- T-score ≥ -1.0 is normal
- T-score between -1.0 and -2.5 is osteopenia (low bone mass)
- T-score ≤ -2.5 or below is osteoporosis

Conventional radiography is relatively insensitive to detection of early disease and requires a substantial amount of bone loss (about 30%) to be apparent on x-ray images. The relevant radiographic features of osteoporosis are cortical thinning and increased radiolucency.

Prevention

Methods to prevent osteoporosis include changes of lifestyle, medications, orthoses and fall prevention.

Lifestyle prevention addresses primarily modifiable risk factors such as immobility, tobacco smoking and unsafe alcohol intake. Achieving a maximum peak bone mass through exercise and proper nutrition during adolescence is important for the prevention of osteoporosis. Exercise and nutrition throughout the rest of the life delays bone degeneration. Proper nutrition includes a diet sufficient in calcium and vitamin D. Patients at risk for osteoporosis (e.g. elderly, steroid use) are generally treated with vitamin D (1,25-dihydroxycholecalciferol or calcitriol) and calcium supplements (calcium carbonate or citrate). Aerobics, weight bearing, and resistance exercises can all maintain or increase BMD in postmenopausal women.

Treatment

There are several medications used to treat osteoporosis. Antiresorptive agents work primarily by reducing bone resorption, while anabolic agents build rather bone [8,10].

Antiresorptive agents include bisphosphonates, selective estrogen receptor modulators SERMs and calcitonin, anabolic agents comprise of teriparatide (recombinant parathyroid hormone) and sodium fluoride. Other agents include RANKL inhibitors (human monoclonal antibody mimicking the activity of osteoprotegerin) and strontium ranelate (dual action bone agents) stimulating the proliferation of osteoblasts as well as inhibiting the proliferation of osteoclasts.

3. Diseases caused by osteoclast dysfunction

3.1 Osteopetrosis

Osteopetrosis (marble bone disease) is a rare inherited disorder characterized by osteoclast dysfunction, the number may be reduced, normal, or increased [6,17]. Deficient carbonic anhydrase might result in defective hydrogen ion pumping in osteoclasts. This might cause defective bone resorption, since an acidic environment is needed for dissociation of calcium hydroxyapatite from bone matrix and its release into blood circulation. If bone resorption fails while formation persists, excessive bone is formed.

Despite a diffuse symmetric skeletal sclerosis, bones are brittle and fracture frequently. Many bones do not develop a medullary cavity. Mild forms may cause no symptoms. However, serious forms can result in stunted growth, deformity and an increased likelihood of fractures. Bone marrow narrowing leads to extramedullary hematopoiesis, resulting in

hepatosplenomegaly. Patients suffer from anemia and recurrent infections. Due to the increased pressure put on the nerves by the extra bone it can also lead to blindness, facial paralysis, and deafness.

The only durable cure for osteopetrosis is bone marrow transplant [15].

3.2 Paget's disease (Osteodystrophia deformans)

This chronic disorder typically results in enlarged and deformed bones. Sir James Paget first described this condition in 1876. It is common in whites in England, France and Austria with global prevalences between 1,5 and 8%, rarely occurring before the age of 40.

In situ hybridization studies have localized a type of paramyxovirus in osteoclasts, so a slow virus infection is discussed as causal agent. Other evidence suggests an intrinsic hyperresponsive reaction to vitamin D and RANK ligand might be the cause [21].

The pathogenesis of Paget's disease is described in 3 stages. Periods of furious bone resorption are followed by compensatory increase of bone formation in a disorganized fashion. Intense cellular activity produces a mosaic-like picture of trabecular bone instead of the normal linear lamellar pattern, resulting in a gain in bone mass but the newly formed bone is disordered. The marrow spaces are filled by an excess of fibrous connective tissue with a marked increase in blood vessels, causing the bone to become hypervascular. In the final phase (burnt out) the bone hypercellularity may diminish, leaving a dense typical pagetic bone [12].

Clinical expression

Bone pain is the most common symptom, headaches and hearing loss may occur when Paget's disease affects the skull. Increased head size, bowing of the tibia, or curvature of spine may occur in advanced cases. Hip pain may be caused by Paget's disease affecting the pelvic bone or secondary osteoarthritis due to damage of the joint cartilage. Pathological fractures and rarely malignant transformation (osteosarcoma) are serious problems.

Diagnosis

An elevated level of alkaline phosphatase in the blood in combination with normal calcium, phosphate, and aminotransferase levels in an elderly patient are suggestive of Paget's disease. In the late phase pagetic bone has a characteristic appearance on X-rays. Bone scans are useful in determining the extent and activity of the condition.

Treatment

There is no cure. However, prognosis is generally good, particularly if treatment is given before major changes in the affected bones have occurred. Bisphosphonates can relieve bone pain and prevent the progression of the disease; in addition Vitamin D and Calcium should be supplemented [12].

4. Diseases associated with abnormal mineral homeostasis

4.1 Hyperparathyroidism

Normally parathyroid hormone (PTH) stimulates osteoclastic resorption of bone, with the release of calcium from the bone into the plasma [7]. Hyperparathyroidism is an overactivity of the parathyroid glands resulting in excess production of parathyroid hormone (PTH). It is classified into primary and secondary types. Primary hyperparathyroidism results from hyperplasia, adenoma or rarely carcinoma of the parathyroid gland and leads to hypercalcemia.

Secondary hyperparathyroidism is caused by prolonged hypocalcemia, eg., due to Vitamin D deficiency or chronic renal failure.

Failure of the feed back mechanisms leads to excessive. Parathormone secretion with continuing PTH output. Increased parathyroid hormone is detected by osteoblasts, which then initiate the release of mediators that stimulate osteoclast activity resulting in excessive osteoclastic destruction of bone. Uncontrolled absorption of bone is followed by compensatory attempts of osteoblasts to deposit new bone. Subperiosteal resorption are accompanied by fibrous tissue replacement of marrow spaces.

In addition to affecting all bones single or multiple focal osteolytic lesions are also present in bone. These osteolytic lesions appear as soft, semi fluid brown material because of old and recent hemorrhages called as "brown tumors". Multiple brown tumors produce numerous osteolytic lesions in many bones know as "Von Recklinghousin's disease' of bone" or "osteitis fibrosa cystica".

Clinical expression

High blood calcium levels have a direct effect on the nervous system, so common manifestations of hyperparathyroidism include weakness and fatigue, depression, bone pain, myalgias, decreased appetite, feelings of nausea and vomiting, constipation, polyuria, polydipsia, cognitive impairment and kidney stones. Decrease in bone mass predisposes to fractures.

Diagnosis

The gold standard of diagnosis is the Parathyroid immunoassay. Once an elevated Parathyroid hormone has been confirmed, serum calcium level allows differentiating between primary (high) and secondary (low or normal) hyperparathyroidism.

Treatment

The immediate goal is to control the hypercalcemia; in primary cases surgical removal of the parathyroid tumor or parathyroid gland will normalize the situation. Control of hyperparathyroidism allows the bony changes to regress significantly or disappear completely. A calcimimetic drug might be considered as a potential therapy for some people with primary and secondary hyperparathyroidism on dialysis.

4.2 Renal osteodystrophy

Chronic kidney disease-mineral and bone disorder (CKD-MBD) refers to metabolic and structural abnormalities of bone caused by presence of chronic renal failure [16]. There are two main components to renal osteodystrophy:

- a. Osteomalacia of renal origin due to failure of conversion of 25 hydroxy vitamin D3 to the active principle 1,25 dihydroxy vitamin D3 in the kidney because of tubular damage.
- b. Secondary hyperparathyroid effects secondary to hyperphosphatemia and hypocalcemia due to phosphate retention and excess calcium loss in urine of the damaged kidney.

The bone in renal osteodystrophy therefore shows combination of excessive bone erosion by osteoclasts, failure of mineralisation of osteoid collagen (osteomalacia), osteosclerosis and osteoporosis.

Renal osteodystrophy may be asymptomatic; if it does show symptoms, they include bone and joint pain, bone deformation and sometimes fracture.

Blood tests will indicate decreased calcium and calcitrol and increased phosphate and parathyroid hormone. X-rays might show chondrocalcinosis at the knees and pubic symphysis, osteopenia and bone fractures Symptomatic treatment includes calcium and vitamin D supplementation, restriction of dietary phosphate and phosphate binders such as calcium carbonate, calcium acetate, sevelamer hydrochloride, cinacalcet [9]. Renal transplantation might be a curative treatment option for renal osteodystrophy, since full recovery has been observed post transplantation.

4.3 Osteomalacia and rickets

Both disorders are characterized by delayed and / or inadequate bone mineralization leading to an excess of un-mineralized matrix. The name osteomalacia is often restricted to the milder, adult form of the disease, while in children the disease is known as rickets [2,4].

A common cause of the disease is a deficiency in vitamin D, due to insufficient calcium absorption from the intestine because of lack of dietary calcium or a deficiency of or resistance to the action of vitamin D. In addition, phosphate deficiency caused by increased renal losses can also lead to osteomalacia.

Patients may show general signs as diffuse body pains, muscle weakness, and fragility of the bones. Manifestations during infancy and childhood include softened flattened occipital bones, frontal bossing, deformation of the chest with anterior protrusion of the sternum-pigeon-breast, lumbar lordosis and bowing of the legs. Osteomalacia in the adult is most of the time unspecific and characterized by loss of skeletal mass and osteopenia. Skeletal deformities do not appear in osteomalacia, but fractures might occur, most often of the vertebrae, hips, wrists, and ribs. Relevant for the diagnosis is an abnormally low vitamin D concentration in blood serum. In addition serum calcium and urinary calcium is low, serum phosphate is low and serum alkaline phosphatase is high. Furthermore, a technetium bone scan will show increased activity.

Comparison of bone pathology					
Condition	Calcium	Phosphate	Alkaline Phosphat	Parathyroid	Comments
Osteomalacia Rickets	▼	▼	▲ ∅ ▼	▲	soft bones
Osteitis fibrosa cystica	▲	▼	▲	▲	brown tumors
Osteoporosis	(▲)	∅	▲ ∅ ▼	∅	decreased bone mass
Osteopetrosis	∅	∅	∅	∅	thick dense bones
Paget's disease	∅	∅	▲	∅	abnormal bone architecture

Radiologically cortical microfractures (Looser's zone or Milkman's fractures), most common in the bones of the lower limbs, and a protrusion acetabuli can be seen.

Treatment

Nutritional osteomalacia might be appropriately supplemented by administration of 10,000 IU weekly of vitamin D for four to six weeks. Osteomalacia due to malabsorption may require treatment by injection or daily oral dosing of significant amounts of vitamin D.

References

1. Aldenhoven M, Sakkers RJ, Boelens J, de Koning TJ, Wulffraat NM (2009) Musculoskeletal manifestations of lysosomal storage disorders. *Ann Rheum Dis* 68(11):1659-65.
2. Allgrove J (2009) A practical approach to rickets. *Endocr Dev* 16:115-32.
3. Basel D, Steiner RD (2009) Osteogenesis imperfecta: recent findings shed new light on this once well-understood condition. *Genet Med*. 2009 Jun;11(6):375-85.
4. Bhan A, Rao AD, Rao DS (2010) Osteomalacia as a result of vitamin D deficiency. *Endocrinol Metab Clin North Am* 39(2):321-31.
5. Datta HK, Ng WF, Walker JA, Tuck SP, Varanasi SS (2008) The cell biology of bone metabolism. *J Clin Pathol*. 2008 May;61(5):577-87.
6. de Vernejoul MC, Kornak U (2010) Heritable sclerosing bone disorders: presentation and new molecular mechanisms. *Ann N Y Acad Sci*. 1192:269-77.
7. Fraser WD (2009). Hyperparathyroidism. *Lancet* 374 (9684): 145-58.
8. Kanis JA, Burlet N, Cooper C, Delmas PD, Reginster JY, Borgstrom F, Rizzoli R (2008) European guidance for the diagnosis and management of osteoporosis in postmenopausal women. *Osteoporos Int* 19(4):399-428.
9. Pelletier S, Chapurlat R (2010) Optimizing bone health in chronic kidney disease. *Maturitas* 65(4):325-33.
10. Poole KE, Compston JE (2006). Osteoporosis and its management. *BMJ* 333 (7581): 1251-6.
11. Raisz L (2005) Pathogenesis of osteoporosis: concepts, conflicts, and prospects. *J Clin Invest* 115 (12): 3318-25.
12. Ralston SH, Langston AL, Reid IR (2008) Pathogenesis and management of Paget's disease of bone. *Lancet* 372 (9633): 155-63.
13. Rauch F, Glorieux FH (2004) Osteogenesis imperfecta. *Lancet* 363 (9418): 1377-85.
14. Silverman SL. (2010) Bisphosphonate use in conditions other than osteoporosis. *Ann N Y Acad Sci*. 2010 Sep 28.
15. Steward CG (2010) Hematopoietic stem cell transplantation for osteopetrosis. *Pediatr Clin North Am* 57(1):171-80.
16. Tejwani NC, Schachter AK, Immerman I, Achan P (2006) Renal osteodystrophy. *J Am Acad Orthop Surg*. 14(5):303-11.
17. Tolar J, Teitelbaum S, Orchard PJ (2004). Osteopetrosis. *New England Journal of Medicine* 351 (27): 2839-49.
18. Ward LM, Rauch F, Whyte MP, D'Astous J, Gates PE, Grogan D, Lester EL, McCall RE, Pressly TA, Sanders JO, Smith PA, Steiner RD, Sullivan E, Tyerman G, Smith-Wright DL, Verbruggen N, Heyden N, Lombardi A, Glorieux FH (2010) Alendronate for the Treatment of Pediatric Osteogenesis Imperfecta: A Randomized Placebo-Controlled Study. *J Clin Endocrinol Metab*. 2010 Nov 24.
19. Waugh, EJ; Lam, MA, Hawker, GA, McGowan, J, Papaioannou, A, Cheung, AM, Hodsman, AB, Leslie, WD, Siminoski, K, Jamal, SA (2009) Risk factors for low bone mass in healthy 40-60 year old women: a systematic review of the literature. *Osteoporosis international* 20 (1): 1-21-
20. WHO (1994) Assessment of fracture risk and its application to screening for postmenopausal osteoporosis. Report of a WHO Study Group. *World Health Organization technical report series* 843: 1-129.
21. Whyte MP (2006) Paget's Disease of Bone and Genetic Disorders of RANKL/OPG/RANK/NF-B Signaling. *Ann. N. Y. Acad. Sci.* 1068: 143-64.



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Diagnostic work up and recognition of primary bone tumours

Although the frequency of metastases from primary cancers to bone is unknown, it is very much more common than the incidence of sarcoma. Soft tissue sarcoma has an incidence of around 2,500 cases per annum in the population of the United Kingdom (66 Million) and is treated by a number of different surgeons, including orthopaedic surgeons. Bone sarcomata are treated only by orthopaedic surgeons and medical oncologists and the incidence in the United Kingdom is around 6 cases per million, per annum. Therefore, in the United Kingdom we are likely to see around 360 cases per annum. This rarity of occurrence means that the orthopaedic surgeon must always be wary and suspicious of the probability of the occurrence of a primary bone sarcoma, which usually presents as an uncharacteristic, unrelenting and progressive pain, swelling or pathological fracture.

Rarely in the immature skeleton disorders of growth can occur, particularly around the knee joint. Bone sarcoma is rare beneath the age of five years where the common diagnosis is osteomyelitis, metastatic neuroblastoma, leukaemia, eosinophilic granuloma or unicameral bone cyst. Primary bone sarcomata, therefore, tend to affect the adolescent population with an age range between 5 and 20 years and malignant sarcomas must be differentiated from primary benign disease and fibrous dysplasia. The commonest sarcoma is osteosarcoma and this has a peak incidence at 13.5 years in girls and 17 years in boys. Ewing's sarcoma has a similar age distribution to osteosarcoma, but tends to affect slightly older patients in addition. The rare condition of chondrosarcoma is very unusual below the age of 20 and tends to afflict the older age group of 50 years and above.

When a bone tumour is considered the first and primary investigation that is required is a simple x-ray and the diagnostic process which follows the recognition of a lesion on the x-ray should be as follows:

1. What is the lesion doing to the bone?
2. What is the bone doing in response to the presence of the lesion has any response been formed?
3. Are there any characteristic features detectable?

The presence or absence of response from the host bones usually gives an indication of the degree of rate of growth of the tumour. Well corticated areas with a very narrow zone of transition are obviously tumours which have a slower growth rate and allow host-bone to respond, compared to those where there is a wide zone of transition or large soft tissue mass present. When considering an x-ray a number of features need to be assessed:

1. The age of the patient.
2. The site of the lesion within the skeleton.
3. The site of the lesion within the bone – whether it is epiphyseal metaphyseal or diaphyseal.
4. Whether it is intramedullary, cortical or on the surface.

5. The radiological appearances of the lesion itself.

The majority of tumours are in fact metaphyseal and intramedullary in their origin. They affect usually the distal femur, the proximal tibia or the proximal humerus, which are areas of rapid growth.

In conclusion, the plain radiograph is the most important investigation for characterisation of the lesion and assessing the general characteristics of the tumour. The MRI is a very important tool for local staging, accurately mapping out the area of involvement of the tumour, but the MRI although useful for characterisation can often be misinterpreted due to reactive bone and soft tissue oedema. Bone tumours generally should be referred to a specialist centre where a multi-disciplinary approach should be taken.

Having established the possible diagnosis of a primary bone tumour by plain x-ray it is important to stage the patient both locally and distally. The local staging essentially takes place by an MRI. Distal staging takes place by CT scanning and Technetium bone scanning or occasionally by PET scanning. Once the lesion has undergone radiological staging then a tissue diagnosis by bone biopsy is required. The usefulness of the various modalities available and their strengths and weaknesses at answering various questions is outlined in Figure 1.

Imaging Modalities Primary Bone Tumours					
	MRI	CT	Angiography	Scintigraphy	Plain flim
Extraosseous extn.	4.1	3.6	2.9	2.6	1.7
Intraosseous extn.	4.5	4.2	2.9	4.4	3.3
Cortial destruction	3.0	4.0			3.6
Calcification ossification	1.6	3.8			3.0
Periosteal/enosteal react	1.6	2.1			3.4

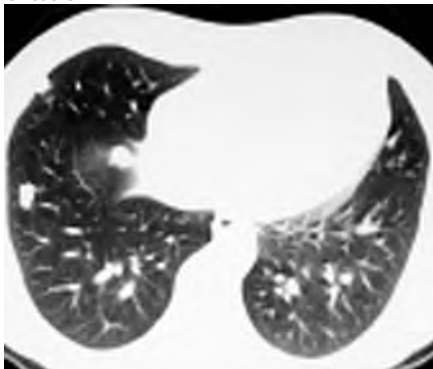
Local staging of the disease essentially wishes to draw out the local extent of the lesion and by use of an MRI of the whole bone define any local skip metastases. The local staging also requires an estimation of the extraosseus extent of the disease, the involvement of soft tissue muscle compartments, the involvement of the joint and the relationship of the tumour to the neurovascular bundle, which is of tantamount importance in considering limb salvage procedures. Magnetic Resonance Imaging has the advantage of being extremely sensitive to bone pathology. It is excellent for local staging due to direct multi-planer

imaging capabilities. It involves no ionising radiation and can occasionally be tissue specific. Its disadvantages are that generally it is not tissue specific, it has poor capability of detecting calcification and it is relatively poor for imaging very small bone lesions, such as osteoid osteoma and is somewhat over sensitive to soft tissue reaction and marrow oedema. Occasionally it can detect lesions which are not visible by direct bone radiography and therefore is the investigation of choice in patients where the plain x-ray has been unhelpful.

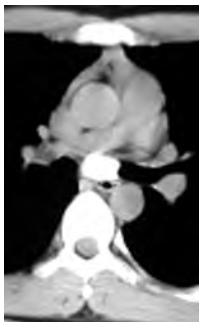
Distal staging is performed by the use of CT scan of the pulmonary lung fields as 95% of metastatic disease involves the pulmonary tissue, but a Technetium bone scan is also useful for picking up both soft tissue and bony metastases. If the bone scan picks up a distant metastasis in bone then further imaging of that bone is required.

Distant Staging

Chest CT



Lung metastases



Mediastinal nodes



Vertebral metastases

Once the lesion is staged it is important to obtain a tissue diagnosis. Most commonly throughout the world this is performed by targeted (CT, plain radiographs or ultrasound) Jamshidi needle biopsy, although some countries maintain the use of open biopsy. The method of needle biopsy is 98% accurate in peripheral malignancies in centres of excellence. If pathological excellence is not available then perhaps open biopsy should still be considered. It has long been understood that open biopsy leads to larger local contamination and clinical morbidity. Mankin's paper of 1982 shows that it can worsen the prognosis in 8% and therefore will increase the risk of amputation. Once staging and biopsy are complete the tumour is placed in Enneking's clinical staging system, which is as pertinent today as when it was first described in 1986.

Clinical Staging

Stage	Grade	Compartment	Mets
IA	Low	Intra	No
IB	Low	Extra	No
IIA	High	Intra	No
IIB	High	Extra	No
IIIA	Any	Intra	Yes
IIIB	Any	Extra	Yes

Since the pioneering work of Rosen, et al in the United States it has become established practice across Europe to give all sarcomas, except chondrosarcoma, pre-operative chemotherapy. There is no doubt that the use of neoadjuvant and adjuvant chemotherapy in both osteosarcoma, Ewing's sarcoma and fibrous malignancies of bone has led to improved survival rates across all centres and probably also influences the ability to perform limb salvage surgery. Unfortunately primary amputation rates remain at around 8% and usually involves late diagnosis with consequential wide spread soft tissue contamination or neurovascular involvement, pathological fracture and a lack of response to neoadjuvant chemotherapy.

References

1. Mankin H J, Lange T A, Spanier S S. The hazards of biopsy in patients with malignant primary bone and soft tissue sarcomas. *J. Bone Joint Surgery*, 1982. Oct 78. 656-663.
2. Stoker D J, Cobb J P, Pringle J A S. Needle biopsy of musculoskeletal lesions: A review of 208 procedures. *J. Bone Joint Surgery. Br.* 1991. 73B. 498-500.
3. Saifuddin A, Mitchell R, Burnett S J, Sandison A, Pringle J A. Ultrasound guided needle biopsy of primary bone tumours. *J. Bone Joint Surgery*, 2000. 82B. 50-54.
4. Enneking W F. *Clinical musculoskeletal pathology*. Storter, 1986.
5. Rosen G, Caparros B, Huvoos A G, et al. Pre-operative chemotherapy of osteosarcoma. *Cancer* 49. 1221-1230, 1982.



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Diagnostic algorithm and treatments options in bone metastasis

Oncology management is becoming an increasingly more serious task in orthopaedic and trauma surgery. A significant number of patients, who have solitary or multiple bone metastases can survive on cytostatic treatment for years.

The significance of the treatment of bone metastases is indicated by the fact that they are 80-100 times more common than primary malignant bone tumors. Various cancers have very different „bone affinities” as concerns their metastases (Table 1).

Table 1

Characteristics of skeletal metastases
- In 65- 70% of the bone metastases the primary site is: lung, breast, kidney and prostate
- Imaging: lytic, mixed or sclerotic lesions
- Periosteal reaction is usually absent
- 10- 20% are solitary at recognition but multiplication occurs in 1-3 years
- Pathological fracture in 20% of the cases
- Risk of pathological fractures: <ul style="list-style-type: none"> • more than 2 cm • lower limb (peritrochanteric region) • lytic type

In 65-85% of bone metastases the primary site of the tumor is in the breast, lung, kidney and prostate. The bones most frequently involved in decreasing sequence are: lumbar, dorsal, cervical spine, ribs, proximal femur and tibia, skull, pelvis, sternum and humerus. Only 1-2% of these secondaries affect the short tubular bones of the hand and foot.

Symptoms: Deep intermittent pain that is independent of the movement, often presents weeks or month before the X-ray changes are detected. The case history (primary cancer!) and laboratory tests must be thoroughly evaluated. In 10-30% of cases the first episode is a pathologic fracture of a lytic metastasis of kidney or lung cancer. Osteoplastic metastases of prostate cancer rarely break and have good propensity to heal.

Imaging: In suspected cases, e.g. when there is local bone pain after history of tumor, an X-ray is taken of the area in question and CT, MR (occasionally PET-CT) scans are added if necessary. In spine, in the opposite of spondylitis the tumor involves single vertebral bodies, invading the intervertebral space only in later stage. In the long tubular bones, the lesion may be central, though it is more often eccentric, involving the cortex. Periosteal reaction is in most cases absent. Bone scan is also extremely important to decide if the process is single or multiple (Fig. 1a and b).

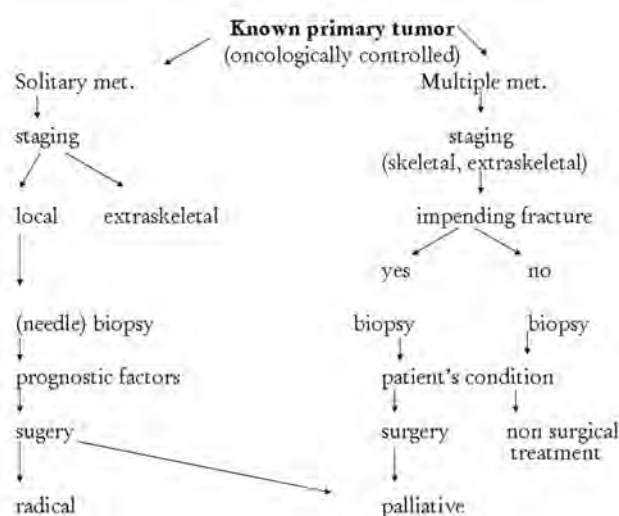


Fig. 1a Diagnostic algorithm at impending fracture (Known primary tumor)

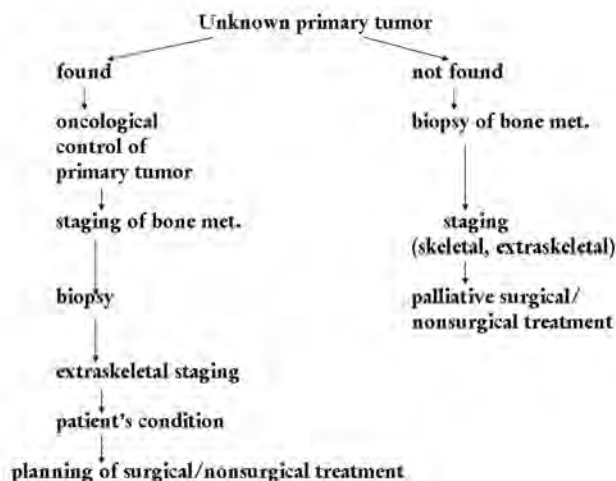


Fig. 1b Diagnostic algorithm at impending fracture (Unknown primary tumor)

Prognostic factors: The most sensitive prognostic factor is the origin of the primary tumor (Table 2). In cases of breast, prostate, thyroid and kidney cancers, the expected survival time is much longer than in cases of lung cancers or bone metastases of melanoma (Fig. 2 and 3). The life expectancy is poor (Table 3) when the primary tumor is unknown or inoperable, or when the primary is discovered at the same time as the metastases, if the metastases are inoperable, multiple or multiorgananic.

Table 2

Favourable prognostic factors
- Primary site: breast, kidney
- Interval between primary tu and met: more 4 years
- Solitary metastases
- Radical excision
- Grade 1, less vascular metastases kidney)
- Chemo-, radio-, hormone sensitive tumors

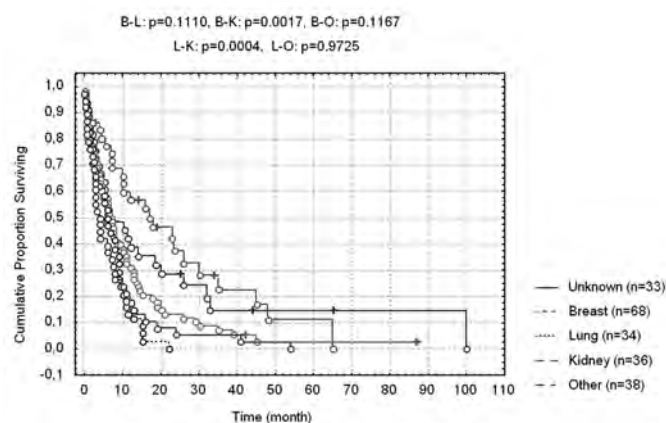


Fig. 2 Survival according to the primary site in 209 metastatic patients

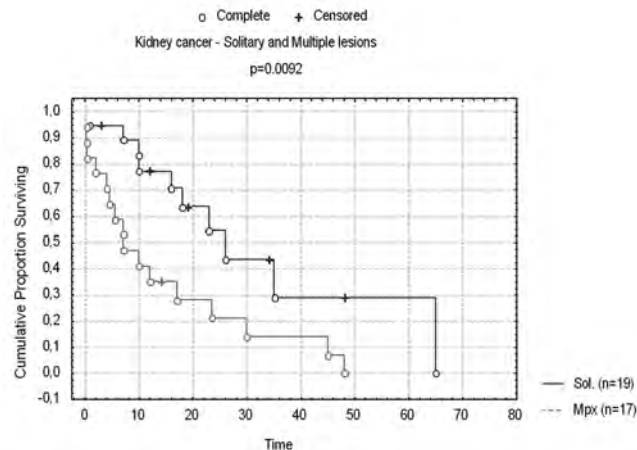


Fig. 3 Survival according to the solitary and multiple manifestations of bone metastases in kidney cancer

Table 3

Unfavourable prognostic factors
- Primary tumor: unknown, or oncologically uncontrolled
- Primary site: lung, liver, pancreas or melanoma
- Axial location
- Multiple/ multiorganic metastases
- Short doubling time of metastases
- Radio-, chemotherapy resistency
- Synchron or metachron appearance of metastases
- Poor general condition of patient

Surgical treatment: The surgical intervention can be palliative or curative. The aims of palliative surgical treatment are: to alleviate the pain, to prevent the imminent fracture, to osteosynthesize and strengthen the bone in case of pathologic fracture using the less invasive technique, to reconstruct the motion and mobility of the patient ensuring a better quality of life. There is a broad range of the possible surgical procedures for reconstruction of the defect, i.g. plating (Fig. 4), intramedullary nailing (Fig. 5), curetting the defect and filling up with bone cement or insertion of a normal (Fig. 6) or tumor endoprosthesis. Intramedullary nailing is advantageous for it is stable weight-bearing, and even if the tumor progresses, loosening of the implant is not likely. In 10-20% of the cases a curative-type radical tumor excision (Table 4) is warranted using limb-saving surgery and reconstruction of the defect by modular tumor endoprosthesis or allograft.



Fig. 4 Plating with cementation



Fig. 5 Intramedullary nailing

Table 4

Indications for radical excision
- Oncologically controlled primary tumor site
- Solitary bone metastasis
- Positive prognostic factors
- Conditions present for radical excision



Fig. 6 Conventional cemented revision endoprosthesis

References

1. Baloch KG, Grimer RJ, Carter SR, et al. Radical surgery for the solitary-bone metastasis from renal cell carcinoma. *J Bone Joint Surg.* 82:62-67, 2000.
2. DeVita VT, Hellman Jr S, Rosenberg SA. *Principles and practice of oncology* 5th ed. Lippincott-Raven Publishers, Philadelphia. Chapter 50. Treatment of metastatic cancer. Pp.2570-2585, 1997.
3. Jemal A, Siegel R, Ward E et al. Cancer statistics, 2008. *CA Cancer J Clin* 58:71-96, 2008.
4. Jung ST, Ghert MA, Harrelson JM et al.: Treatment of osseous metastases in patients with renal cell carcinoma. *Clin Orthop Rel Res.* 409:223-231, 2003.
5. Szendrői M, Sárváry A: Surgery of bone metastases. In: Besznyák I (Ed.) *Diagnosis and surgery of organ metastases*, 1st ed. Akadémiai Kiadó, Budapest, 2001, pp:213-248.



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Missed diagnosis in children's orthopaedics

Monteggia equivalent fracture – Bado type I

Missed Monteggia equivalent fracture leading to persistent anterior radial head dislocation, Bado type I is a well known entity although rare. However, the diagnosis is often missed because of lack of awareness. The dislocated radial head is often diagnosed months after the injury. Patients complain are typically reduced elbow flexion combined with elbow pain. On examination restricted range of elbow movement is usual, especially flexion. Missed Monteggia equivalent fracture occurs typically in age group 4–10 years of old and is more common in boys. Diagnosis is confirmed on conventional X-rays. Obligatory anterior-posterior and lateral views of both forearms with elbow included. The ulnar bow is determined on the lateral view (Fig. 1). Preferred treatment is transverse proximal ulnar osteotomy without annular ligament reconstruction (Fig. 2).



Fig. 1

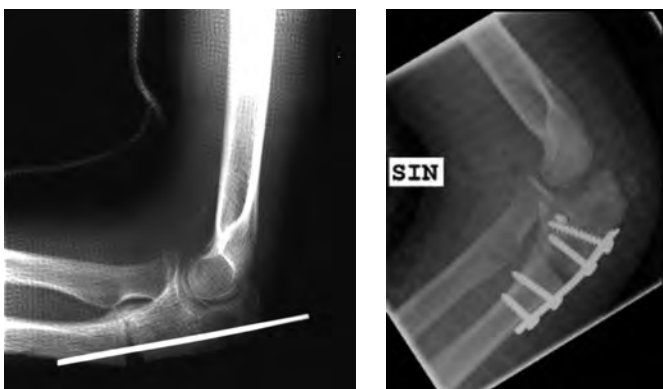


Fig. 2

Lateral humeral fracture

Lateral condylar fractures are avulsion fractures and constitute approximately 20% of all pediatric distal humerus fractures. The fracture

is often missed, and if not managed appropriately the fracture can displace and non-union and deformity may be observed. It typically occurs in children aged approximately 6 years. Patients complain are typically localized swelling and tenderness to palpation on the lateral aspect of the elbow. Deformity is seldom seen at the elbow. X-rays, anterior-posterior, internal oblique (for posterior displaced fragments) and lateral views often confirm the diagnosis. Prior to complete ossification an arthrography may be helpful to determine the fracture pattern and intraoperatively to assess reduction. MRI may be helpful to visualize a cartilage hinge fracture. Lateral humeral condyle fractures are traditionally classified according to Milch 1964 or Jakob, 1975. Milch type I fracture line pass through the lateral ossific nucleus of the capitum into the joint (Fig. 3) and Milch type II fracture is characterized by a fracture line going lateral to the ossific nucleus into the apex of the trochlea (Fig. 3). Jakob's type I fracture is stable, non-displaced as it does not violate the cartilage or goes intraarticular. Type II fracture is comparable to Milch fracture type II as the fracture line goes through the articular surface into the trochlea. Type III fracture is characterized by displacement and often rotation (Fig. 4).

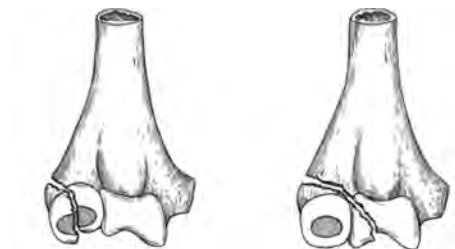


Fig. 3

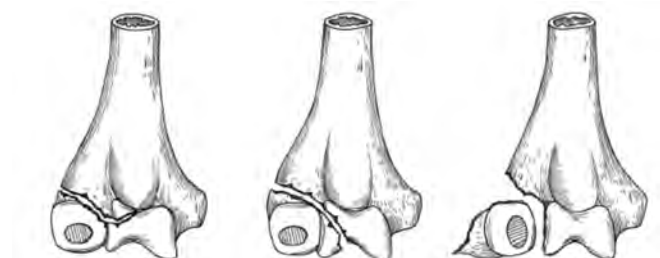


Fig. 4

Jakob's type I and II fractures displaced less than 2 mm may be treated by percutaneous pinning. Type II if displaced more than 2 mm open reduction and internal fixation is required. All type III fractures are unstable and require open reduction and internal fixation as they are prone to a high rate of non-union (Fig. 5). Post operative care involves X-rays after one week and then after three more weeks. If new bone are detected pinns can be removed and immobilization continuous two more weeks making six weeks in total. If non-union is detected after six

weeks six more weeks of immobilization are required. Complications associated with lateral humeral condyle fractures are cubitus varus (the cause of this deformity is not clear), cubitus valgus, fish tail deformity and ulnar nerve palsy.

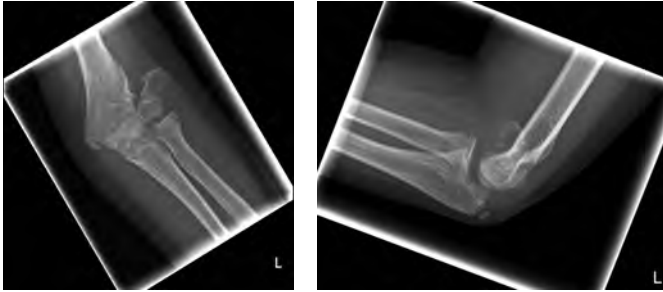


Fig. 5

References

1. *J Am Acad Orthop Surg* 1998;6, 215-224
2. *Clin Orthop Relat Res* 1967; 50:71-86.
3. *Clin Orthop Relat Res* 1997; 337:208-215.
4. *J Trauma* 1964; 4:592-607.
5. *J Bone Joint Surg Br* 1975; 57(4):430-436.



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Neuro-orthopaedics

Neuromuscular disorders in children include a group of conditions that affect the brain, spinal cord, peripheral nerves and muscles. By causing muscle weakness, hypotonia or spasticity, these entities negatively impact the structure, function and development of the musculoskeletal system.

While some of these children can be diagnosed at birth merely by their clinical appearance (such as spina bifida), many others are evaluated for complaints such as delayed ambulation, abnormal gait or parents' concern regarding the motor milestones of their child by orthopedic surgeons first. For this reason, orthopedic surgeons need to be well-versed in the neurodevelopmental steps of a normal child. Minor neurological abnormalities (retention of primitive reflexes, claw toe deformity, severe in-toeing, toe-walking, very brisk or absent reflexes) should be regarded as warning signs to obtain further testing and after a thorough examination, the child should be referred to the appropriate consultations.

The primary responsibility of the orthopaedic surgeon is to determine what is abnormal. Evaluating the meaning of this abnormality and reaching a conclusive diagnosis may oftentimes require the assistance of other disciplines. Naturally, the first step in recognizing the abnormal is to know what is normal.

Neuroorthopaedic Evaluation

Physical examination begins with observation from the instant the child enters the exam room. In ambulatory children, gait abnormalities (Trendelenburg limp: gluteal muscle weakness, drop foot: myopathies, positions and movements of the limbs during walking: cerebral palsy), and in sitting children, trunk asymmetry (muscular dystrophies) must be noted carefully. Involuntary movements during the child's attempt to rest, move or communicate verbally should be considered warning signs.

The family should be questioned as to family history (peripheral neuropathies), the pregnancy itself and obstetric history (cerebral palsy),

success in school and social intelligence, and sphincter functions (occult dysraphisms).

During the examination, the clothes should be removed and all areas of the child's body inspected separately (i.e. spine: signs of occult dysraphism, calf hypertrophy: myopathy, ptosis: congenital muscular dystrophy). Mildly increased lumbar lordosis or a cavus deformity of the foot may be only the tip of the iceberg and disclose a more serious underlying condition. While walking the child should be observed for the initiation of the first step, unsteadiness during gait, speed, synchrony between the upper and lower extremities and the spatial relationship of the joints during walking. Toe- and heel-walking may help disclose subtle problems. Usually, an abnormality in gait is easily detected. However, the determination of the cause of this abnormality may usually require detailed and sophisticated examinations. By recognizing the abnormality and performing correct referrals, the orthopaedic surgeon will have done his or her part.

Determination of joint range of motion and resistance during movement (rapid angle vs. slow angle, spasticity vs. contractures) are some of the essentials of the orthopaedic examination. During evaluation of joint range of motion, the surgeon should keep in mind that some muscles span more than one joint and consider the positions of adjacent joints while evaluating these muscles (a contracture of the psoas may be masked by increasing lumbar lordosis, a contracture of the hamstrings by hip flexion posture; the child may appear to walk on their heels despite a contracture of the tendo Achilles because of valgus deformation of the subtalar joint).

It is important that the patients be evaluated for overall function as well as having their joints and muscles examined individually. One of the most well-known and commonly used methods for this is the Gross Motor Function Classification System that was conceived for the evaluation of cerebral palsy (CP). This system provides an objective measure for the evaluation patient function, treatment plan and success of the chosen method of treatment while creating a common language for health professionals to use (Fig. 1).



Fig. 1 (From Centre for Childhood Disability Research of the McMaster University, 1997)

During patient evaluation, imaging modalities are also sometimes used along with physical examination. Observational and computerized motion analysis should be an essential part of the neuromuscular patient's work-up. EMG aids in the diagnosis of spinal cord and peripheral neuropathies while providing dynamic data for the activation of different muscle groups during walking in patients with CP.

The role and responsibility of orthopaedic surgery in the management of neuromuscular disorders.

The priorities of a child with a neuromuscular disease can be listed as achieving verbal and physical communication with their environment, performing actions for daily living independently or with help, locomotion and ambulation. The orthopaedic surgeon can assist the child in these objectives in a myriad of ways:

- Providing guidance for the selection of appropriate physical therapy,
- Deciding on and prescribing assistive devices that will prevent deformity formation, aid in the maintenance of posture and help employ present potential more efficiently,
- Planning and performing surgical procedures that will prevent the formation of deformity, correct existing deformity and as a result improve function and comfort.

While rebalancing the existing musculature is sufficient in some cases, oftentimes planning of the procedure to correct the deformity, ordering the appropriate device for the maintenance of the correction achieved and planning rehabilitation modalities for the maximum gain in functional status are required. Orthopaedic management does not address only musculoskeletal problems; with increased functional capacity, the child contributes more to activities of daily living and can establish better communication with other persons. The neurodevelopmental rehabilitation of a child that can use their extremities better will be infinitely more effective. For these reasons, orthopaedic management has a deep impact on cognitive, emotional, psychological and neurological functions beyond the musculoskeletal system.

Neuromuscular disorders: similarities and differences from a musculoskeletal perspective.

Although the title 'neuromuscular' contains pathologies very different from each other regarding pathophysiology and prognosis, they create similar problems in the growing skeleton. While sometimes disease-specific measures are needed, there are several common principles that can be applied to most of these problems.

A team approach is the sine qua non of the successful management of neuromuscular disorders. The most important member of this team are those that have understood the disease from every aspect, are aware of current and possible problems, in possession of the determination, diligence and necessary social and economic resources required for a tedious, difficult treatment open to disappointment: the family. If there is no supportive and dedicated family, the struggle has already been lost. It is impossible for a single physician or a group of physicians to manage neuromuscular diseases. The core team consisting of the physical therapist, orthopaedic surgeon and neurologist will require the help of other health professionals such as neurosurgeons, psychologists, occupational therapists and others. These members should never consider themselves competitors but recognize that they are players of

the same team simply manning different positions.

Musculoskeletal problems secondary to neuromuscular disorders change over time. For this reason, establishing a long-term treatment plan is a mistake. The severity of the disease, the age at which symptoms appeared, the efficacy of rehabilitation, compliance to brace wear, the timing of necessary medical and surgical interventions are only some of the factors that directly have an effect on the extent and severity of musculoskeletal problems. Two patients who started out in similar clinical and functional settings may, after a while, end up in very different situations, and therefore, with very different requirements. On the other hand, basing serious treatment decisions, especially of the surgical kind, on a single examination usually results in serious mistakes that cannot be corrected. These patients should be examined at different points in time before a surgical decision is made.

Abnormal tonus or weakness of muscles causing imbalance at a joint will cause a contracture that is dynamic at first and becomes static unless treated in time. Contractures negatively impact muscular growth and skeletal development and result in permanent bony deformities and/or joint instability. True contractures along with permanent bone/joint deformities in turn result in functional disability and painful states, such as degenerative arthritis. Breaking this vicious cycle should be an objective apart from the disease itself (Fig. 2). For this reason, protection or restoration of joint range of motion, preserving muscle strength as much as possible should be a priority in the treatment of all neuromuscular disorders.

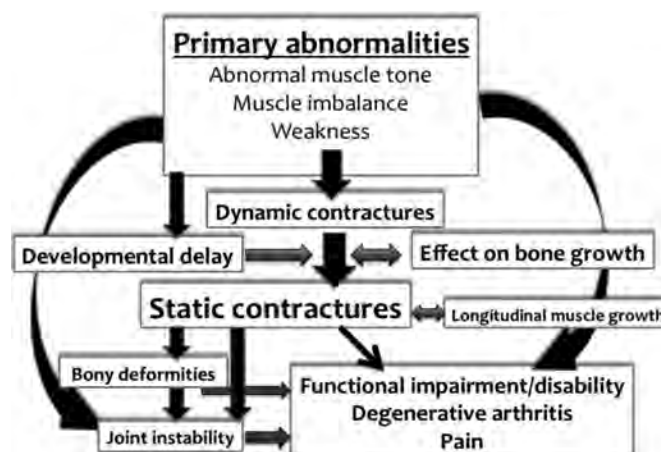


Fig. 2 (From Uri Narayanan, 6th IPOS Syllabus, Page 308, 2009)

While in cerebral palsy, spasticity needs to be decreased and selectivity increased for this purpose, in peripheral neuropathies, muscle strengthening exercises should be primarily employed. In disorders where there is no expected change in the geographical distribution such as cerebral palsy, surgical procedures that will diminish muscle strength and cause irreversible changes are put off as much as possible. This is in contrast to those disorders that decrease life expectancy and functional capacity by steady progress to different levels in the body (progressive muscular dystrophy), where early muscle transfers may be indicated in order to maintain function for as long as possible. Regardless of etiology, immobilization following surgery should be kept short and the patient referred to rehabilitation as soon as possible. Prolonged immobilization will further exacerbate a common problem of these children: disuse osteoporosis and the increased risk of fracture that goes with it. Atrophy and loss of range of motion will negatively affect an already diminished capacity for locomotion and at the very

least inhibit the expected benefits of the procedure, if not cause functional deterioration.

Neuromuscular disorders affect the axial spine as well as the appendicular skeleton. While the treatment of spinal deformities requires more serious intervention than those of the extremities, usually the decision process is somewhat simpler. Curves that have reached a certain degree of severity, interfere with trunk balance and threaten normal respiratory function should be treated surgically. Pelvic instrumentation in these deformities, which often include the pelvis and have a collapsing nature, can be construed as a standard of care. However, differences among specific diseases exist. A scoliosis in SMA which can often maintain good flexibility despite high Cobb angles may benefit from waiting until surgery is absolutely necessary, while a curve in DMD that will always invariably and unrelentingly progress after the child becomes wheel-chair-bound should be treated as soon as it appears to decrease possible complications. Again, although SMA 2 scolioses may not affect the pelvis at presentation, pelvic instrumentation is indicated as this group of curves have been shown to progress to pelvic involvement in time. However, some CP scolioses only concern the thoracolumbar or lumbar spines and may be evaluated like idiopathic scoliosis and segment preservation attempted.

Conclusion

In conclusion, the great majority of the neuromuscular disorders of children affect the musculoskeletal system to variable degrees. Bone and joint deformities caused by muscle spasticity and/or weakness will both impede the child's functional capacity and preclude the progression of normal development. For the improvement or, at the very least, preservation and optimization of present functional capacity, a team approach is needed. The orthopaedic surgeon plays an instrumental role in the management of these children from first diagnosis to orthotic treatment, decreasing of spasticity to the restoration of architecture with tendon-muscle-bone procedures.

Suggested readings

1. Birch JG. *Orthopedic management of neuromuscular disorders in children. Seminars in Ped Neurol.* 5: 78-91, 1998.
2. Cottalorda J, Violas P, Seringe R. *Neuro-orthopaedic evaluation of children and adolescents: A simplified algorithm. Orthop Traumatol Surg Res* 98: S146-53, 2012.
3. Staheli LT. *Neuromuscular disorders. In Practice in pediatric orthopedics.* Ed Lynn T Staheli. Lippincott Williams&Wilkins 2001. pp: 323-46.
4. Warner WC. *Orthopedic surgery in neuromuscular disorders. In Neuromuscular disorders: Treatment and management.* Ed. Tulio E Bertorini. Saunders Elsevier 2011. pp:137-53.



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Abnormalities of newborns feet

Foot deformities are the most common congenital deformities seen at birth. In addition to true congenital deformities there is a variety of molding deformities, which are usually mild and resolve spontaneously. The incidence of foot deformities in a consecutive series of 2401 patients was reported with 4.2% with 87% of those feet being normal at reexamination [32].

Differentiating between deformities that are mild and correct well with massage and benign neglect and those needing treatment and even may recur is not always easy but nonetheless crucial.

In the newborn all foot deformities can be diagnosed clinically by inspection and palpation. The ankle, hindfoot, midfoot and forefoot and the position, range of motion and flexibility of these segments should be evaluated. Documentation should include digital images and clinical scoring systems whenever available. Radiographs are never necessary at presentation in the neonatal period but might be helpful in the course of treatment to add information to the decision making process or as documentation in cases that received prior treatment [26].

In all cases a thorough examination including a basic neuro-orthopedic evaluation has to be performed. The pregnancy and birth history can give valuable hints at the possible etiology of the abnormality. A hip-ultrasound should be performed as an association between foot deformities like metatarsus adductus or clubfoot and hip dysplasia is controversially reported [23, 24].

1. Molding deformities

Molding deformities are deformities which are connected to the position of the child in utero. As the crowding of the infant increases especially in the last weeks of pregnancy those deformities are usually less frequent in preterm infants [17]. However, the etiologies are overlapping and special considerations must be given to feet presenting muscle imbalance on the basis of a still undeveloped or unbalanced neuro-motoric system.

1.1 Metatarsus adducts (MTA)

Metatarsus adducts (MTA) also known as "bean-shaped foot" is the most common molding deformity. The forefoot is adducted in relation to the hindfoot often presenting a prominent fifth metatarsal base. However, the Achilles tendon is not shortened and full passive dorsiflexion is possible. (Fig. 1) Clinical examination can differentiate between a flexible MTA and a rigid MTA. In flexible MTA the forefoot can passively be abducted to more than neutral, while rigid MTAs can not be abducted to a neutral position. Flexible MTAs usually resolve spontaneously. However, the mother can perform a stretching of the medial foot and especially stimulation of the lateral foot and thigh which usually stimulate the peroneal muscle group and results in abduction, dorsiflexion

and eversion of the whole foot and extension and abduction of the toes. In more resistant cases an abduction orthotic which is available in different forms of shoes like the IPOS shoe or reverse-last shoes might be used up to the age of 6–9 months.

Rigid cases should be casted. For casting the foot is abducted with counter pressure on the calcaneo-cuboid area. This is similar to Kites technique of casting for clubfoot which is obsolete for clubfoot correction but works very well for MTA [18]. A well molded above the knee cast should be applied, as this minimizes the risk of cast slipping. With experience in infant casting a below the knee cast can be applied [16]. In rigid cases a follow up treatment with IPOS shoes or reverse-last shoes should be performed.

Most cases will correct well with the described treatment regime. However, there is a small minority of cases that will tend to recur and might need prolonged orthotic treatment. These are usually cases with distinct muscle imbalance and overpowering of the tibialis anterior tendon.

MTA should not require surgery as even residual forefoot adduction does not lead to functional limitations or foot wear problems. In a long term study of patients with MTA no functional problems were found and no foot was graded as a poor [10].

The cosmetic concerns of the parents should not be accepted as indication for surgery; a cosmetic procedure can still be performed after the end of growth.



Fig 1 Mild but persistent metatarsus adducts

1.2 Skew foot

Skew foot is a combination of MTA with increased heel valgus (as seen in talus obliquus or congenital flatfoot) which is very rarely seen after birth but usually is diagnosed later in life from 4 to 6 years onwards. If present at birth it can be very rigid with casting being very difficult. For casting abduction of the forefoot in relation to the hindfoot must be achieved using the prominent fifth metatarsal base and cuboid as a

fulcrum, while supporting the plantar and medially displaced talus head. In older children the correction can be achieved surgically. Usually a combination of surgical procedures used for correction of flat foot like calcaneal lengthening and for correction of forefoot adduction like medial cuneiform opening wedge osteotomy and tibialis anterior split tendon transfer is successful [11].

1.3 Talipes calcaneovalgus

Talipes calcaneovalgus is a quite frequent molding deformity and is characterized by an extremely dorsiflexed foot with the dorsum of the foot often touching the shin bone (Fig. 2). The forefoot might even be mildly abducted as the calcaneus is in a marked valgus position. It can easily be differentiated from congenital vertical talus (CVT) as the calcaneus is not in equinus and the Achilles tendon is not shortened. Posteromedial bowing of the tibia which is a deformity of the most distal tibia sometimes presents with the foot in a quite similar position (Fig. 3). However, in this deformity the tibia is bowed distally into recurvatum and valgus which results in a foot which is parallel to the tibia but itself totally normal and in normal relationship to the distal part of the tibia [22]. If the foot can be plantarflexed beyond neutral massage and stretching should be performed by the parents and will help the deformity to resolve. In more rigid cases one or two casts might be preferable to achieve correction, and might help to prevent subsequent hypermobile pes planovalgus in the older child [9].



Fig. 2 Schematic drawing of a talipes calcaneovalgus



Fig. 3 Posteromedial bowing. Note that the deformity is in the tibia only

1.4 Positional clubfoot (postural talipes equinovarus)

Positional clubfoot is a molding deformity which resembles clubfoot. The foot is in mild equinus, in adduction and is rotated inwards. However, the foot is passively fully flexible with dorsiflexion well above neutral and no sign of the rigid deformities seen in true clubfoot (Fig. 4 a,b). Positional clubfoot usually responds to stretching and massage. However, some cases are somewhere between severe positional clubfoot and mild idiopathic clubfoot and should be followed to at least two years of age. Whenever a cast is needed for correction, abduction bracing as for clubfoot should be performed for a limited amount of time.



Fig. 4. Positional clubfoot looks like a clubfoot at birth (a) but is passively correctable with free dorsiflexion (b).

2. Structural deformities

All congenital deformities of the foot show a structural deformity compared to the molding deformities which present a positional deformity. Especially in clubfoot the muscles, ligaments, bones are not normal.

2.1 Clubfoot

Clubfoot is the most common true congenital deformity with an incidence of 1.25 in 1000 live birth. It can be found bilateral in 40–50% and male patients are affected more often with a sex ratio male to female of 2.5:1.

The etiology is still unknown; however there is a multitude of factors associated with an increased incidence of clubfoot. Genetic studies have favored the hypothesis of a heterogeneous disorder with a polygenic threshold model explaining the inheritance patterns [5].

Clubfoot is a very complex deformity that shows different components which are often described separately but are all related nonetheless: There is equinus of the hindfoot due to shortening and retraction of the Achilles tendon, varus (inversion) of the hindfoot (calcaneus) and adduction of the calcaneus with the calcaneus being near the lateral malleolus. As a result the calcaneus stands parallel under the talus. The forefoot is adducted and in cavus with the navicular being displaced to medial being near the medial malleolus. Although the forefoot is in supination related to the axes of the tibia, the forefoot is pronated in relation to the hindfoot adding to the cavus and adductus and the medial deep skin crease (Fig. 5 a,b).

Additionally to the displacements of the bones there are marked structural deformities like the talus dome which is mildly flattened [13] which might be increased during treatment by over- vigorous casting, the ligaments are less flexible and contracted, the muscles are more fibrotic and cells of the medial ligamentous tissue have myofibroblastic characteristics [14, 29].

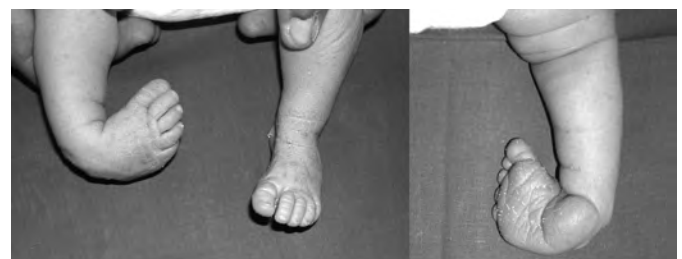


Fig. 5a Unilateral clubfoot in a 5 days old girl with Pirani 5.5.



Fig. 5b Same patient at age 9 years after Ponseti treatment with additional tibialis anterior tendon transfer

Secondary clubfoot must be differentiated from idiopathic clubfoot. Secondary clubfoot might be seen in patients with arthrogryposis, myelomeningocele (MMC) spina bifida and other neurogenic disorders, as part of a syndrome or in association with a fibular hemimelia. Prenatal diagnosis has increased in the last years and most mothers prefer to know about their baby's clubfoot before birth. However, prenatal counseling is necessary and should include a treatment outline and discussing the possibility of non-idiopathic clubfoot and additionally the possibility of a false positive diagnosis [28].

Treatment should be started within the first weeks of life. However, there is no need to rush treatment as it was traditionally done in many countries. I recommend starting treatment after the infant and mother have been released from the hospital and feel well and strong enough for travelling. At presentation a thorough pediatric orthopaedic examination of the newborn must be performed which should include a hip ultrasound. Documentation should include digital pictures and a clinical scoring. The Pirani score is a very well reproducible and validated score that can even guide the treatment process [30].

In the last 10 years the Ponseti method has become the gold standard for the treatment of clubfoot worldwide [15]. The Ponseti method is a mostly non-operative treatment regime with serial casting with above the knee casts and weekly cast changes. The superior results are achieved by following the very well defined treatment regime in great detail [25]. Short manipulation is recommended before casting. In the first cast the first metatarsal must be raised to align the forefoot with the hindfoot and to decrease cavus. The foot should never be pronated. In the following casts a pure abduction with counter pressure on neck of talus, not calcaneus, is performed. Long leg casts weekly for about 4–7 weeks are necessary to achieve full correction with abduction, eversion and dorsiflexion of the calcaneus from underneath the talus. An Achilles tenotomy is necessary in about 90% of cases, when after clinical examination dorsiflexion of at least 15 degrees cannot be obtained. In cases with dorsiflexion over 15 degrees and no need for a tenotomy from clinical evaluation I recommend a lateral stress dorsiflexion view radiograph to evaluate the true correction of the hindfoot and especially the tibiocalcaneal angle [27]. The tenotomy is a full percutaneous tenotomy performed from medial to lateral to avoid bleeding complications. The last cast after the tenotomy stays for three weeks and is molded in full abduction and dorsiflexion. After cast removal a foot abduction orthosis (Denis Browne bar), with the clubfoot in 70 degrees abduction and the normal foot in 45 degrees abduction must be used for 22 hours for 3 months and for 3–4 years for nights and naps. A tibialis anterior tendon transfer is necessary in about 20 % of patients usually between age 3–6 in case of weak peroneal muscles and an overpowering tibialis anterior muscle.

Long- and midterm results are very good and superior to all other treatment regimens or surgical approaches reported in literature [3, 4].

2.2 Congenital vertical talus (CVT)

Congenital vertical talus presents as a rocker-bottom deformity with fixed equinus of the calcaneus and dorsal dislocation of the navicular on the talus. It can be found bilateral in about 50% of patients and can even be associated with a contralateral clubfoot (Fig. 6). Mostly cases are associated with syndromes like arthrogryposis, trisomy 18, sacral agenesis or Larson's syndrome. The incidence is with approximately 1:10.000 about ten times lower than clubfoot.



Fig. 6 Patient with congenital vertical talus on the right and clubfoot on the left side.

The exact etiology is still unknown but muscle imbalance, intrauterine compression particularly when coupled with arthrogryposis, or arrest in fetal development of the foot have been discussed. More recent studies suggest that skeletal muscle biopsy abnormalities are common in patients with vertical talus and are more frequently seen in patients with congenital myopathy and distal arthrogryposis [19]. However, isolated CVT can be transmitted as an autosomal dominant trait with variable expression and incomplete penetrance [7].

Classifications usually are based on the underlying etiology. Ogata differentiated in his series between a primary isolated form (44%) and an associated form with and one without a neurological deficit (50%) [20].

As differential diagnoses flexible flatfoot, oblique talus and talipes calcaneovalgus must be ruled out. Flexible flatfoot presents without fixed equinus and with a navicular which is less prominent and can easily be repositioned in front of the talus by dorsiflexion. Oblique talus is a less severe manifestation of CVT with a rocker-bottom deformity of the foot and equinus contracture but with a reducible navicular.

Congenital vertical talus is a very complex deformity. The navicular articulates with the dorsal aspect of the neck of the talus, the calcaneus is displaced posterolaterally in relation to the talus and is in equinus. Additionally there is a variable degree of subluxation of the calcaneocuboid joint. On clinical examination the plantar prominent talus can be palpated. The forefoot is abducted and dorsiflexed and the hindfoot in equinus. Radiographs in ap.-and lateral view and lateral with maximum plantarflexion and dorsiflexion view can help to differentiate CVT from Talus obliquus.

Traditionally treatment was surgically with the most recent approach being an open release through a Cincinnati approach, tendo Achilles lengthening, capsulotomy and medial release with reposition of navicular, reconstruction of capsule and transfixation with k-wires. A treatment based on serial manipulation and casting combined with a minimal surgical intervention was introduced by Dobbs [6]. After correction of the talonavicular luxation by casting which could be summed up as a reversed Ponseti casting a minimal medial approach is used to reposition the navicular and transfix the navicular to talus with

a k-wire. Afterwards a percutaneous Achilles tendon tenotomy is performed and the result fixed in a cast. An orthotic treatment is necessary to prevent recurrence. Encouraging results of the Dobbs technique have been reported in literature [6,8].

2.3 Toe deformities and duplications

2.3.1 Curly toes

Abnormalities of the toes are almost always visible at birth. Many infants and toddlers are presented with mild deformities of the toes usually referred to as curly toes. Curly toes are typically bilateral and mostly affect the second to fourth toes. The deformity corrects spontaneously in many cases. In resistant cases taping or splinting can only temporarily improve the position but is very bothersome for the small patient and the parents. Surgical correction should only be considered when significant clinical problems arise due to overlapping of the toes and resulting pressure sores. For surgical correction flexor tenotomy has been shown to be effective. [12]

2.3.2 Syndactyly

Syndactyly is a congenital webbing of toes most commonly affecting the second and third toe. The webbing can reach until the tip of the toe or can be incomplete. Syndactyly does not lead to any kind of functional impairment or limitation and does therefore not require surgical correction. Cosmetic concerns should not be considered as an indication for the operation. Separation of the toes usually requires Z-plasties or skin grafts which lead to cosmetically unsatisfying results.

2.3.3 Polydactyly

Polydactyly can affect the lateral fifth ray (fibular or postaxial polydactyly) the middle rays (central polydactyly) or the medial first ray (preaxial polydactyly). Polydactyly can be part of a syndrome but is most often isolated and is bilateral in about half of the cases.

Depending on the extent of involvement a distal phalangeal type, a middle phalangeal type, a proximal phalangeal type and a metatarsal type can be differentiated [2,31]. A tarsal type with duplication or partial duplication of the hindfoot is very rare.

In fibular polydactyly resection of the supernumerary ray is usually performed between age 9 to 12 months. Radiographs should be performed to confirm the most lateral ray as the hypoplastic one.

Tibial polydactyly comes in many different shapes and forms and is more complex regarding surgical correction (Fig. 7). Acute lengthening with interposition of a small fibular segment or gradual lengthening with external fixation is often necessary for shortening of the first ray. The very rare central duplications can be excised through a racquet incision and need reconstruction of the plantar intermetatarsal ligament.

Although good alignment of the toe can usually be achieved widening of the forefoot sometimes persists

2.3.4 Ray deficiencies

Ray deficiencies are usually found in association with other deformities like fibula hemimelia or tibial hemimelia. As the lateral developmental field is affected in fibular hemimelia usually the lateral rays are missing [21]. In a similar way tibial hemimelia is often associated with tibial polydactyly.

Central ray deficiencies can be found in the form of a cleft foot (formerly unfortunately known as lobster claw). This deformity is very rare with an incidence of about 1 on 90000 births and mostly bilateral. Classification is mainly based on the number of existing metatarsals [1]. Surgical correction is difficult and aims at narrowing the wide foot [33].



Fig. 7 Tibial preaxial polydactyly with duplication of the first ray.

References:

1. Blauth W, Borisch NC 1990 Cleft feet. *Proposals for a new classification based on roentgenographic morphology. Clin Orthop Relat Res* (258):41-8.
2. Blauth W, Olason AT 1988 Classification of polydactyly of the hands and feet. *Arch. Orthop. Trauma. Surg* 107, 334-344.
3. Bor N, Coplan JA, Herzenberg JE (2009) Ponseti treatment for idiopathic clubfoot: minimum 5-year followup. *Clin Orthop* 467: 1263-1270.
4. Cooper DM, Dietz FR (1995) Treatment of idiopathic clubfoot: a thirty-year follow-up note. *J Bone Joint Surg Am* 77-A: 1477-1489.
5. Dobbs MB, Gurnett CA (2012) Genetics of clubfoot. *J Pediatr Orthop B* 21(1):7-9.
6. Dobbs MB, Purcell DB, Nunley R, Morcuende JA (2006) Early results of a new method of treatment for idiopathic congenital vertical talus. *J Bone Joint Surg Am* 88(6):1192-200.
7. Dobbs MB, Schoenecker PL, Gordon JE (2002) Autosomal dominant transmission of isolated congenital vertical talus. *Iowa Orthop J* 22:25-7.
8. Eberhardt O, Fernandez FF, Wirth T. [Treatment of Vertical Talus with the Dobbs Method.] *Z Orthop Unfall*. 2011;149(2):219-224.
9. Edwards ER, Menelaus MB 1987 Reverse club foot. *Rigid and recalcitrant talipes calcaneovalgus. J Bone Joint Surg Br* 69:330.
10. Farsetti P, Weinstein SL, Ponseti IV (1994) The long-term functional and radiographic outcomes of untreated and non-operatively treated metatarsus adductus. *J Bone Joint Surg Am* 76(2):257-65.
11. Hagmann S, Dreher T, Wenz W (2009) Skewfoot. *Foot Ankle Clin*. 14(3):409-34.
12. Hamer AJ, Stanley D, Smith TW (1993) Surgery for curly toe deformity: a double-blind, randomised, prospective trial. *J Bone Joint Surg Br* 75(4):662-3.
13. Herzenberg JE, Carroll NC, Christofersen MR, Lee EH, White S, Munroe R (1988) Clubfoot analysis with three-dimensional computer modeling. *J Pediatr Orthop* 8(3):257-62.
14. Ippolito E, Ponseti IV (1980) Congenital clubfoot in the human fetus: A histological study. *J Bone Joint Surg Am* 62: 8-22.
15. Jowett CR, Morcuende JA, Ramachandran M (2011) Management of congenital talipes equinovarus using the Ponseti method: a systematic review. *J Bone Joint Surg Br* 93(9):1160-4.
16. Katz K, David R, Soudry M (1999) Below-knee plaster cast for the treatment of metatarsus adductus. *J Pediatr Orthop* 19(1):49-50.
17. Katz K, Naor N, Merlob P, Wielunsky E (1990) Rotational deformities of the tibia and foot in preterm infants. *J Pediatr Orthop*

- 10(4):483-5.
18. Kite JH (1967) Congenital metatarsus varus. *J Bone Joint Surg Am* 49(2):388-97.
19. Merrill LJ, Gurnett CA, Connolly AM, Pestronk A, Dobbs MB (2011) Skeletal muscle abnormalities and genetic factors related to vertical talus. *Clin Orthop Relat Res* 469(4):1167-74.
20. Ogata K, Schoenecker PL, Sheridan J (1979) Congenital vertical talus and its familial occurrence: an analysis of 36 patients. *Clin Orthop Relat Res* (139):128-32.
21. Opitz JM 1985 The developmental field concept. *Am J Med Genet* 21(1):1-11.
22. Pappas AM (1984) Congenital posteromedial bowing of the tibia and fibula. *J Pediatr Orthop* 4:525.
23. Paton RW, Choudry Q (2009) Neonatal foot deformities and their relationship to developmental dysplasia of the hip: an 11-year prospective, longitudinal observational study. *J Bone Joint Surg Br* 91(5):655-8.
24. Perry DC, Tawfiq SM, Roche A, Shariff R, Garg NK, James LA, Sampath J, Bruce CE (2010) The association between clubfoot and developmental dysplasia of the hip. *J Bone Joint Surg Br* 92(11):1586-8.
25. Ponseti IV (1996) Congenital clubfoot. *Fundamentals of treatment*. New York: Oxford University Press Inc.
26. Radler C, Egermann M, Riedl K, Ganger R, Grill F (2010) Interobserver reliability of radiographic measurements of contralateral feet of pediatric patients with unilateral clubfoot. *J Bone Joint Surg Am* 20;92(14):2427-35.
27. Radler C, Manner HM, Suda R, Burghardt R, Herzenberg JE, Ganger R, Grill F (2007) Radiographic Evaluation of Idiopathic Clubfeet Undergoing Ponseti Treatment. *J Bone Joint Surg Am* 89: 1177-1183.
28. Radler C, Myers AK, Burghardt RD, Arrabal PP, Herzenberg JE, Grill F (2011) Maternal attitudes towards prenatal diagnosis of idiopathic clubfoot. *Ultrasound Obstet Gynecol* 37(6):658-62.
29. Sano H, Uhthoff HK, Jarvis JG, Mansingh A, Wenckebach GF (1998) Pathogenesis of soft-tissue contracture in club foot. *J Bone Joint Surg Br* 80(4):641-4.
30. Shaheen S, Jaiballa H, Pirani S (2012) Interobserver reliability in Pirani clubfoot severity scoring between a paediatric orthopaedic surgeon and a physiotherapy assistant. *J Pediatr Orthop B* 21(4):366-8.
31. Watanabe H, Fujita S, Oka H (1992) Polydactyly of the foot: an analysis of 265 cases and a morphological classification. *Plast Reconstr Surg* 89:856-877.
32. Widhe T, Aaro S, Elmstedt E (1988) Foot deformities in the newborn-incidence and prognosis. *Acta Orthop Scand* 59(2):176-9.
33. Wood VE, Peppers TA, Shook J 1997 Cleft-foot closure: a simplified technique and review of the literature. *J Pediatr Orthop* 17(4):501-4.



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Hip diseases in the childhood

Hip disorders in the childhood are common in the pediatric population. The initial symptoms may be limping or an abnormal gait. In addition, the child may complain of knee rather than hip pain. Many hip problems in the adult have their origin during growth. This chapter provides an overview of the principal orthopedic hip disorders from birth to adolescence.

1. Developmental Dysplasia of the Hip (DDH)

Developmental dysplasia of the hip (DDH) occurs in different forms at different ages and includes all grades of instability and morphological abnormalities detected by ultrasound or radiography. The true incidence of DDH is difficult to determine because of disparities in the definition, the type of examinations used to detect DDH and the population being studied. Estimates range from 0.5 to 4 % [1, 2].

Understanding the natural history of DDH requires knowledge of the growth and development of the hip joint from birth to skeletal maturity.

Due to differences in the diagnosis and the management of the disease, this entity is divided in two age groups:

- 1.1. DDH in newborns (0 to 1 year)
- 1.2. DDH after walking age including
 - Neglected cases of hip dislocation
 - Residual dysplasia of the hip
 - Osteonecrosis of the hip after treatment of DDH

1. DDH in newborns (0 to 1 year)

Since the introduction of hip sonography and its standardization, results of treatment have improved due to early diagnosis of deformities at birth [3]. Every newborn should be screened for signs of DDH or instability. Clinical examinations include the Barlow and Ortolani techniques and require experienced examiners [4]. Ultrasound according to Graf as a screening method is independent to the examiner's experience and skills and provides a tool for early detection and early adequate therapy of DDH. Each sonographic type can be assigned to a specific phase of treatment correlated with a specific procedure effective in the given pathoanatomical situation (Table 1). Splinting therapy should be started up to the beginning of the sixth week of life in order not to miss the best time of treatment [5,6]. In cases, which are detected early, conservative treatment can be completed before walking age. The α -angle according to Graf should be at least 60°. A radiograph should be performed in every treated child to exclude osteonecrosis of the hip. Uncritical splinting which is not adapted to the given sonographic pathoanatomical situation of the hip or noncompliance of the parents during the splinting phase often leads to surgery with the need for open reduction even when early diagnosed [7, 8].

Table 1 Sonographic-based treatment system according to the given hip types.

Treatment phase	Hip type	Therapy	Alternative
Reduction	IV, III, D	Manual reduction	Reduction orthosis (i.e. Pavlik harness,...)
Retention	Reduced IV, III, D IIc unstable	Fettweis plaster cast in human position	Retention orthosis (i.e. Pavlik harness,...)
Maturity	IIa, IIb, IIc stable	Abduction device according to Mittelmeier-Graf	Maturity orthosis (i.e. Pavlik harness, Tübinger orthosis,...)

2. DDH after walking age

In cases of DDH after walking age, surgery to correct acetabular anatomy is often required. Operative interventions should be performed before the age of five years in order to provide complete anatomical recovery [9–11].

2.1. Neglected cases of dislocation after walking age

In countries with general ultrasound screening programs neglected dislocated hips have become rare cases [7, 8].

Clinical findings in children with neglected hip dislocation after walking age are limping because of the shortened leg combined with restricted range of motion (restricted abduction and flexion contracture) and abductor muscle insufficiency (Trendelenburg gait). In bilateral cases, compensatory hyperlordosis of the lumbar spine is evident.

The therapeutic management depends on the age of the child and the pathomorphology of the acetabulum and the femur. In unilateral and bilateral cases of hip dislocation, the upper age limit for an open reduction is 8 years [11]. Due to the morphological changes of the hip joint in this age group, open reduction should be performed with pelvic and femoral (varus, derotation and shortening) osteotomies in a single stage procedure (Fig. 1a, b). Femoral shortening is necessary to reduce the risk of osteonecrosis following open reduction in older children [10, 12].

The type of pelvic osteotomy depends on the age of the patient and the degree of acetabular dysplasia. Pemberton acetabuloplasty is preferred in children less than 6 to 7 years with a shallow and steep acetabulum. This intervention keeps the pelvic ring intact and can be performed in bilateral cases in one session. Pin fixation is not necessary. Salter innominate osteotomy represents a redirection osteotomy with the symphysis as the center of rotation. The upper age limit is 8 years.



Fig. 1a Two-year-old girl with unilateral untreated dislocation of the left hip.



Fig. 1b The reduced left hip of the same girl. Six weeks after open reduction, Pemberton acetabuloplasty and femoral osteotomy (varus, shortening and derotation).

2.2. Residual dysplasia of the hip after walking age

During periods of growth spurts the anatomy of the hip can change rapidly and unexpected deterioration can occur especially in cases which have been treated before. Children with treated DDH in the first year of life should be followed radiographically to detect residual or recurrent acetabular dysplasia or subluxation [13, 14].

In childhood residual dysplasia and subluxation of the hip may not have clinical signs or symptoms. Diagnosis is made according to the radiographs. Radiographic assessment should be performed by measuring the acetabular index or the center edge angle on AP radiographs. Residual dysplasia is evident if the acetabular index is over 25 degrees and the center edge angle under 20 degrees [15]. The subluxated hip shows a lateralization of the femoral head. The Shenton line may be disrupted.



Fig. 2 Three-year-old girl with bilateral treated dislocation of the hip at the age of five months (closed reduction of the right hip, open reduction of the left hip). At follow up both hips show residual acetabular dysplasia with subluxation of the right hip and disrupted Shenton lines on both sides.

Surgical treatment includes different types of pelvic and femoral osteotomies to improve the coverage of the femoral head and to achieve nearly normal anatomy. Pemberton acetabuloplasty and Salter innominate osteotomy are preferred in the younger age group. In children older than 8 years with residual acetabular dysplasia the triple pelvic osteotomy is often the method of choice to correct hip dysplasia when spherical congruity is present. During this procedure the acetabulum becomes a free, rotating fragment, and overcorrection, impingement and retroversion of the acetabulum can occur. The triple pelvic osteotomy is a challenging procedure and requires an experienced surgeon. After closure of the triradiate cartilage, the periacetabular osteotomy (PAO) according to Ganz can be performed in cases with mild osteoarthritis (Toennis < 2). The PAO is even more challenging than the triple pelvic osteotomy.

2.3. Osteonecrosis (ON) of the hip after treatment of DDH

ON of the proximal femoral epiphysis is one of the main complications associated with the treatment of developmental dislocated hips. The overall rates of ON reported in studies have been inconsistent, ranging from 6% to 88% after treatment of the dislocated hip [16, 17]. The complication can lead to acetabular dysplasia and joint incongruity, resulting in early osteoarthritis.

Clinical symptoms vary according to severity from limping, to restricted range of motion and hip pain if osteoarthritis is progressive. Radiographs are characterized by acetabular dysplasia, deformed femoral epiphysis and femoral neck shortening. The Bucholz-Ogden system is most widely used to classify ON into four types (grade I to IV) [18].

The treatment goal is to achieve remodeling of the deformed femoral head by concentric reduction and pelvic osteotomy. Additionally, femoral valgus osteotomy, intertrochanteric double osteotomy with trochanteric advancement or greater trochanter transfer are often necessary.



Fig. 3a 11-year-old girl with an closed reduction of the dislocated left hip at the age of five months. At follow up signs of osteonecrosis with a trochanteric overgrowth were evident.



Fig. 3b Three months after the trochanteric advancement surgery. The greater trochanter is now below the level of the top of the femoral head.

2. Transient (toxic) Synovitis

Transient synovitis of the hip joint is probably one of the most common cause of hip pain and responsible for the majority of cases of limping. The hip joint is irritated because of an effusion underlying inflammatory synovitis. There is often a history of antecedent viral illness.

Transient synovitis is seen in children between 3 and 8 years and manifests with the rapid onset of hip pain, limited range of motion and limping. Clinical presentation may mimic that of septic arthritis. Patients rarely have a temperature above 38° or indication of systemic illness. The blood parameters are usually within normal limits, radiographs are normally unremarkable. Ultrasound of the affected hip shows effusion with an increased distance between the femoral neck and the joint capsule. Treatment include a brief period of bed rest and the use of nonsteroidal anti-inflammatory drugs to expedite spontaneous resolution of the inflammatory synovitis. Clinical symptoms usually resolve completely over several days.

3. Septic Arthritis

Septic arthritis of the hip requires urgent medical management because of the potential for significant joint destruction. Patients present with the acute onset of hip pain similar to children with transient synovitis. They may walk with a limp and have a history of antecedent infection, trauma or illness. Unlike transient synovitis, septic arthritis progresses to a febrile systemic illness with fever, chills and malaise. Local clinical symptoms are obvious: swelling of the joint, erythema, warmth, tenderness and the child holds the affected extremity immobile in an abducted, flexed and external rotated position.

Laboratory values (WBC count, C-reactive protein, and ESR) are usually elevated. Radiographs remain normal following several days after initial symptoms. Lateralization and subluxation of the femoral head because of the massive effusion can occur. Ultrasound demonstrates joint effusion. A sonographic differentiation between septic or transient synovitis is usually not possible. If clinical suspicion remains high, consideration should be given to a supplemental MRI.

Treatment should be urgent and requires immediate surgical drainage of the joint and the metaphysis in cases of additional underlying osteomyelitis of the proximal femur. The metaphysis is located intra-articularly and may produce septic arthritis. Intravenous antibiotic therapy starts immediately with empirical antibiotics coverage and should be changed according to the sensitivity of obtained cultures [19, 20].

According to the recommendation to start urgent treatment in cases of septic arthritis, the hip joint can be saved without morphological changes. In late treated cases, deformities vary from subluxation to complete destruction of the proximal femur (Fig. 4).



Fig. 4 Radiograph of a three-year-old girl after neonatal septic arthritis of the right hip. Complete destruction of the femoral head and neck are visible.

4. Legg-Calve-Perthes Disease (LCPD)

Legg-Calve-Perthes Diseases (LCPD) is a femoral head disorder of unknown aetiology. It involves temporary interruption of the blood supply to the bony nucleus of the proximal femoral epiphysis, impairing the epiphyseal growth and increasing bone density. The necrotic bone is subsequently replaced by new bone, flattening and enlarging the femoral head. Once the new bone of the femoral head is contained within the acetabulum, the femoral head slowly remodels until skeletal maturity. LCPD is four times more common in boys than in girls, and the disease has been reported in patients from 2 to 12 years. At clinical examination, the most common presenting symptom is a painless limping. Pain, if present, may be localized in the groin, thigh or knee region. Hip motion, primarily internal rotation and abduction, is limited. Atrophy of the muscles of the thigh or calf secondary to pain may be evident. Diagnosis is made according to the clinical symptoms and the typical radiographic findings.

LCPD has been divided into four radiographic stages (Table 2):

- In the initial stage, a decreased size of the ossific nucleus, lateralization of the femoral head, a subchondral fracture, and physeal irregularity may be evident.
- In the fragmentation stage, the epiphysis appears fragmented with areas of radiolucency and radiodensity.
- During the reossification stage, the bone density returns to normal by new bone formation.
- In the residual stage, the radiographic findings include a gradual remodeling of the head shape and the acetabulum until skeletal maturity.

Several radiographic classification systems are currently used:

1. Catterall proposed a four-group classification, based on the amount of the involvement of the femoral epiphysis (Catterall group I-IV) [21].
2. Salter and Thompson reported a two-group classification based on the extent of subchondral fracture, which corresponded to the amount of subsequent resorption (Salter-Thompson group A and B) [22].
3. The Herring lateral pillar classification is the most widely used radiographic classification system helping to determine treatment and prognosis during the fragmentation stage of the disease. The degree of involvement of the lateral pillar can subdivided into three groups (Herring group A, B and C) [23]

Table 2 Radiographic stages of Legg-Calve-Perthes disease.

Stage	Findings
Initial stage	Lateralization of the femoral head and smaller ossific nucleus Subchondral fracture
Fragmentation stage	Segments of femoral head are demarcated Lucent areas appear Increased density resolves
Reossification stage	New bone formation occurs in the femoral head
Residual stage	Acetabulum and head shape remodels gradually until skeletal maturity

Catterall identified prognostic factors, known as radiographic "head-at-risk" signs, associated with poor results [24]. These "head-at-risk" sings include the following:

- 1. Lateral subluxation of the femoral head
- 2. Gage sign: a radiolucency in the lateral epiphysis and metaphysic
- 3. Calcification lateral to the femoral epiphysis
- 4. Horizontal physeal line

The main goal of the treatment is to contain the femoral head in the acetabulum during the active period of the disease. This "containment" provides remodeling of the femoral head to the same shape of the acetabular cup, creating a spherical, well-covered femoral head with a normal hip range of motion. Various nonoperative and operative treatment methods for containment were developed over time (bracing, casting, physiotherapy, femoral and pelvic osteotomy ("super-containment")). The choice of treatment is based on the severity of involvement and age of onset and based on the surgeon's experience and institutional treatment protocol. There is still a lack of consensus among the pediatric orthopedic surgeons.

Our institutional treatment philosophy includes the following:

- 1. Nonoperative containment is used in patients younger than 6 years at onset and Herring A and B. Symptomatic treatment includes nonsteroidal anti-inflammatory medication, reduction of activities and physiotherapy to increase range of motion.
- 2. Operative treatment is indicated in patients after the age of 6 years at onset and Herring B, B/C and in all patients regardless of age with Herring C or "head-at-risk" signs.

Till the age of 6 years, Salter innominate osteotomy is used. Salter osteotomy and femoral varus osteotomy ("super-containment") in combination is indicated in patients younger than 8 years (Fig. 5a, b). In patients more than 8 years old triple pelvic osteotomy is indicated. In all groups, hip range of motion should be achieved before operative containment. In cases of aspherical incongruency and hinge abduction secondary containment or salvage procedures are used: valgus femoral osteotomy, shelf acetabuloplasty, cheilectomy and Chiari osteotomy.



Fig. 5a Seven-year-old boy suffering from Perthes disease on the right hip. Radiological signs of "head-at-risk" with lateralization of the femoral epiphysis, horizontal physeal line and the Gage sign are evident.



Fig. 5b Radiographs of the same boy, five months after Salter innominate and femoral varus osteotomy. The femoral head is well covered.

5. Slipped Capital Femoral Epiphysis (SCFE)

Slipped capital femoral epiphysis (SCFE) is a well-known hip disorder that affects adolescents between 11 and 15 years, and involves the displacement of the femoral epiphysis from the metaphysic [25, 26]. In early adolescence, the growth plate is weaker and vulnerable, as it carries about four times its body weight. If obesity or trauma is added to the physeal weakness, the growth plate may fail gradually or acutely. SCFE occurs in about 2 in 100.000 most commonly in obese boys [25, 26]. It is bilateral in about one-fourth of cases, with possibly slight silent slippage in even more.

SCFE may be classified according to onset of symptoms (acute, chronic or acute on chronic) or according to ability of weight bearing (unstable or stable). Clinical symptoms include: 1. Pain in the affected hip or groin or referred to the anteromedial thigh, 2. Limited hip range of motion (decreased internal rotation and abduction) 3. Gait abnormality. The symptoms and clinical findings vary according to whether the symptoms are acute, acute-on-chronic or chronic, whether the slip is stable or unstable, and with the coexistence of osteonecrosis or chondrolysis [27,28]. Plain radiographs in anteroposterior and lateral views are the primary imaging studies needed to evaluate SCFE. Southwick recommended measuring the femoral-head-shaft angle on frog-leg lateral views. The head-shaft angle is less than 30 degrees in mild slips, between 30 and 50 degrees in moderate slips, and more than 50 degrees in severe slips [29].

Treatment can be divided into two categories: treatment to prevent further slippage in mild and moderate cases, and treatment to reduce the degree of slippage in severe slips. Prevention of further slippage in

mild and moderate slips can be accomplished by in situ pinning or screw fixation. In situ fixation allows a minimal invasive, percutaneously performed treatment with the goal to stabilize the femoral epiphysis to the femoral neck. The fixation device must be placed perpendicular to the plane of the proximal femoral epiphysis and must be of appropriate strength to avoid failure before physeal plate closure. Almost all SCFEs should be able to be stabilized with percutaneous placement of a single 6.5- to 7.5-mm cannulated screw [30–32]. Because of the high prevalence of contralateral slip, prophylactic pinning is recommended, especially in patients who have SCFE associated with known metabolic and endocrine disorders [33]. Treatment methods that reduce the degree of slip, and lead to improved motion and function include open reduction and subcapital osteotomy through a Ganz surgical hip dislocation approach, and intertrochanteric osteotomy according to Imhaeuser/Southwick [34, 29, 35]. The choice of treatment to reduce the degree of slip is based on the surgeon's experience. Ganz surgical hip dislocation and open reduction is a technically challenging procedure (Fig. 6a, b). Intertrochanteric osteotomies remain the most frequently used procedures for realignment in SCFE. Two major complications, osteonecrosis and chondrolysis, are specifically associated with SCFE. After the diagnosis has been made, treatment must be directed at maintaining motion and preventing collapse including anti-inflammatory medication and relieved weight-bearing until healing occurs.



Fig 6a 14-year-old boy with a severe, chronic slipped capital femoral epiphysis of 60 degrees.



Fig. 6b Radiographs of the same boy two years after surgery. The right hip was treated with open reduction and subcapital osteotomy through a surgical dislocation approach. The left hip was fixed prophylactically with a cannulated single screw.

References

1. Toennis D (1984) *Die angeborene Hüftdysplasie und Hüftluxation*. Springer, Berlin Heidelberg New York.
2. Rosendahl K, Markestad T, Lie RT (1994) Ultrasound screening for developmental dysplasia of the hip in the neonate: the effect on treatment rate and prevalence of late cases. *Pediatrics* 94 (1):47–52
3. Graf R (2006) *Hip Sonography. Diagnosis and Treatment of Infant Hip Dysplasia*. Springer, Heidelberg.
4. Barlow TG (1963) Early Diagnosis and Treatment of Congenital Dislocation of the Hip. *Proc R Soc Med* 56:804–806.
5. Matthiessen HD (1996) Forensic problems in the treatment of hip dysplasias and dislocations. *Z Orthop Ihre Grenzgeb* 134 (6):0a10–12.
6. Matthiessen HD (1997) Dysplasia and therapy factors in hip developmental disorders. *Z Orthop Ihre Grenzgeb* 135 (1):0a12–13.
7. Grill F, Muller D (1997) Results of hip ultrasonographic screening in Austria. *Orthopade* 26 (1):25–32.
8. von Kries R, Ihme N, Oberle D, Lorani A, Stark R, Altenhofen L, Niethard FU (2003) Effect of ultrasound screening on the rate of first operative procedures for developmental hip dysplasia in Germany. *Lancet* 362 (9399):1883–1887.
9. Salter RB (1966) Role of innominate osteotomy in the treatment of congenital dislocation and subluxation of the hip in the older child. *J Bone Joint Surg Am* 48 (7):1413–1439.
10. Galpin RD, Roach JW, Wenger DR, Herring JA, Birch JG (1989) One-stage treatment of congenital dislocation of the hip in older children, including femoral shortening. *J Bone Joint Surg Am* 71 (5):734–741.
11. Lindstrom JR, Ponseti IV, Wenger DR (1979) Acetabular development after reduction in congenital dislocation of the hip. *J Bone Joint Surg Am* 61 (1):112–118.
12. Schoenecker PL, Strecker WB (1984) Congenital dislocation of the hip in children. Comparison of the effects of femoral shortening and of skeletal traction in treatment. *J Bone Joint Surg Am* 66 (1):21–27.
13. Kim HT, Kim JI, Yoo CI (2000) Acetabular development after closed reduction of developmental dislocation of the hip. *J Pediatr Orthop* 20 (6):701–708.
14. Albinana J, Dolan LA, Spratt KF, Morcuende J, Meyer MD, Weinstein SL (2004) Acetabular dysplasia after treatment for developmental dysplasia of the hip. Implications for secondary procedures. *J Bone Joint Surg Br* 86 (6):876–886.
15. Toennis D (1987) *Congenital dysplasia and dislocation of the hip in children and adults*. Springer, Berlin Heidelberg.
16. Pospischill R, Weninger J, Ganger R, Altenhuber J, Grill F (2012) Does open reduction of the developmental dislocated hip increase the risk of osteonecrosis? *Clin Orthop Relat Res* 470 (1):250–260.
17. Firth GB, Robertson AJ, Schepers A, Fatti L (2010) Developmental dysplasia of the hip: open reduction as a risk factor for substantial osteonecrosis. *Clin Orthop Relat Res* 468 (9):2485–2494.
18. Bucholz R, Ogden J. (1978) Patterns of ischemic necrosis of the proximal femur in nonoperatively treated congenital hip diseases. *The Hip: Proceedings of the Sixth Open Scientific Meeting of the Hip Society*. CV Mosby, St. Louis.
19. Morrey BF, Bianco AJ, Jr., Rhodes KH (1975) Septic arthritis in children. *Orthop Clin North Am* 6 (4):923–934.
20. Morrey BF, Peterson HA (1975) Hematogenous pyogenic osteomyelitis in children. *Orthop Clin North Am* 6 (4):935–951.

21. Catterall A, Pringle J, Byers PD, Fulford GE, Kemp HB, Dolman CL, Bell HM, McKibbin B, Ralis Z, Jensen OM, Lauritzen J, Ponseti IV, Ogden J (1982) A review of the morphology of Perthes' disease. *J Bone Joint Surg Br* 64 (3):269-275.
22. Salter RB, Thompson GH (1984) Legg-Calve-Perthes disease. The prognostic significance of the subchondral fracture and a two-group classification of the femoral head involvement. *J Bone Joint Surg Am* 66 (4):479-489.
23. Herring JA, Neustadt JB, Williams JJ, Early JS, Browne RH (1992) The lateral pillar classification of Legg-Calve-Perthes disease. *J Pediatr Orthop* 12 (2):143-150.
24. Catterall A (1971) The natural history of Perthes' disease. *J Bone Joint Surg Br* 53 (1):37-53.
25. Kelsey JL, Keggi KJ, Southwick WO (1970) The incidence and distribution of slipped capital femoral epiphysis in Connecticut and Southwestern United States. *J Bone Joint Surg Am* 52 (6):1203-1216.
26. Loder RT (1996) The demographics of slipped capital femoral epiphysis. An international multicenter study. *Clin Orthop Relat Res* (322):8-27.
27. Aronson J, Tursky EA (1996) The torsional basis for slipped capital femoral epiphysis. *Clin Orthop Relat Res* (322):37-42.
28. Loder RT, Richards BS, Shapiro PS, Reznick LR, Aronson DD (1993) Acute slipped capital femoral epiphysis: the importance of physeal stability. *J Bone Joint Surg Am* 75 (8):1134-1140.
29. Southwick WO (1967) Osteotomy through the lesser trochanter for slipped capital femoral epiphysis. *J Bone Joint Surg Am* 49 (5):807-835.
30. Goodman WW, Johnson JT, Robertson WW, Jr. (1996) Single screw fixation for acute and acute-on-chronic slipped capital femoral epiphysis. *Clin Orthop Relat Res* (322):86-90.
31. Herman MJ, Dormans JP, Davidson RS, Drummond DS, Gregg JR (1996) Screw fixation of Grade III slipped capital femoral epiphysis. *Clin Orthop Relat Res* (322):77-85.
32. Koval KJ, Lehman WB, Rose D, Koval RP, Grant A, Strongwater A (1989) Treatment of slipped capital femoral epiphysis with a cannulated-screw technique. *J Bone Joint Surg Am* 71 (9):1370-1377.
33. Jerre R, Billing L, Hansson G, Wallin J (1994) The contralateral hip in patients primarily treated for unilateral slipped upper femoral epiphysis. Long-term follow-up of 61 hips. *J Bone Joint Surg Br* 76 (4):563-567.
34. Imhauser G (1954) [Surgical treatment of pathological anteversion of the proximal femur]. *Z Orthop Ihre Grenzgeb* 85 (3):395-405.
35. Leunig M, Slongo T, Kleinschmidt M, Ganz R (2007) Subcapital correction osteotomy in slipped capital femoral epiphysis by means of surgical hip dislocation. *Oper Orthop Traumatol* 19 (4):389-410.



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Paediatric idiopathic scoliosis

The goal of this work is to provide a non-exhaustive overview with up-to-date information on idiopathic scoliosis (IS), including relevant aspects of its initial evaluation, diagnosis, classification, orthopedic and surgical treatment. IS is defined on X-rays as a lateral curvature of the spine greater than or equal to 10° Cobb angle with rotation, of unknown etiology [1]. The initial evaluation by an orthopedic surgeon of a child with a spinal deformity should focus on specific items during anamnesis and physical examination. The presence of back pain and/or neurological symptoms (like bowel or bladder dysfunction, or progressive weakness in the legs) are crucial associated findings during patients and parents interview. Similarly, familial history of idiopathic scoliosis is an important element to ask at the first visit. Physical examination should seek waistline and shoulder asymmetry, pelvis level, presence of a rib hump during Adam's forward bending test, physical development and finally neurological evaluation, including superficial abdominal reflexes. In most of the cases, this initial evaluation will be completed by standard radiographic evaluation and the clinician will then be able at this point to classify the scoliosis according to its etiology; idiopathic, congenital, neuromuscular, or syndromic. One must keep in mind that, even if recent evidence suggests that a substantial proportion of patients with adolescent IS (AIS) present with back pain, IS in a child is still considered a painless deformity [15, 17].

Low-dose X-ray diagnostic modalities or surface topography imaging techniques offer the ability to initially diagnose and follow at risk patients with a significantly reduced or even absent irradiation. They also provide the possibility to evaluate quantitatively assess the three-dimensional parameters of the spinal deformity, some of them being highly correlated with planar evaluation and risk of progression [18, 23].

Additional workup with MRI should be done in cases of skin abnormalities along the spine, IS in a male patient, a left main thoracic curve, a rapidly progressing curve, scoliosis in a child under 10 year, scoliosis associated to kyphosis, neurological abnormalities at physical examination, pes cavus and/or unilateral amyotrophic calf.

According to the *Scoliosis Research Society (SRS)* revised glossary of terms, the chronologic definition of IS presentations is:

- Infantile scoliosis - presenting from birth through age 2+11
- Juvenile scoliosis - presenting from age 3 through age 9+11
- Adolescent scoliosis - presenting from age 10 through age 17+11
- Adult scoliosis - presenting from age 18 and beyond

Additionally, the term Early Onset Scoliosis (EOS) is used for any scoliosis deformity diagnosed before age 10. It represents a group of heterogeneous diagnoses only unified by age at presentation and including infantile and juvenile IS, congenital scoliosis, neuromuscular scoliosis and syndromic scoliosis [1, 5].

Location-related classification of a scoliotic curve is based on its apex location. The apex is the vertebra or the disc in a curve most deviated laterally from the vertical axis of the patient that passes through the sacrum (the central sacral line) [1].

- Thoracic: apex at a point between the T2 vertebral body through the T11-T12 disc
- Thoracolumbar: apex at a point between T12 and L1
- Lumbar: apex: apex at a point between the L1-L2 disc space through the L4-L5 disc space

Several additional classifications and specific criteria have been proposed for the 3 different types of IS affecting children. A classification for IS should ideally meet the following requirements:

- Being comprehensive, including all curve types encountered in clinical practice
- Helping to orient the treatment strategy and enabling comparison between different treatments
- Demonstrating acceptable inter- and intraobserver reliability
- Taking into account the sagittal profile of the spine, i.e. evaluating three-dimensionally the deformity
- Being easy to understand and to use in daily practice

King classification is still widely used for AIS, despite the fact it was described at the Harrington rod instrumentation era. It describes 5 thoracic curve types of IS based on the coronal radiographs of the spine [6]. It has three main disadvantages:

- The sagittal alignment is not included in the evaluation
- Some curve types encountered in clinical practice are missing (thoracolumbar, lumbar, double and triple major curves)
- It offers poor-to-fair validity, reliability, and reproducibility.

In 2001, Lenke proposed a new classification system for AIS meeting almost all of the above-mentioned criteria. It is based on four radiographs of the spine (standing long-cassette coronal and lateral as well as right and left supine side-bending) which will define 6 curve types. These 6 curve types are determined by the localization, degree, and flexibility of the manifested curves. Curve types are further classified according to the degree of associated lumbar deformity (lumbar spine modifier) for thoracic curves and according to the sagittal alignment of the thoracic spine (sagittal thoracic modifiers). With these additional parameters, this complex classification system enables the categorization of 42 subtypes of AIS. Its inter- and intraobserver reliability was shown to be superior to the King classification [7]. This useful clinical tool is progressively becoming established as a standard worldwide. Although its complexity makes it difficult to use clinically for the general orthopaedic surgeon, it procures a differentiated instrument to the experienced pediatric spine surgeon, facilitating and standardizing the surgical treatment strategy.

The need for follow-up or treatment, and choice treatment for IS are mainly dictated by the risk of curve progression. In this setting, knowledge of the natural history and long-term prognosis associated with untreated IS is mandatory. There are clearly significant differences of natural history between the different types of pediatric IS. Untreated patient with AIS have been shown to be productive and functional at a high level at 50-year follow-up. Although the prevalence of back pain in this patient population is likely to be higher than in the general population, many studies tend to show that this issue is not a significant problem for these patients. On the other hand, cosmetic concerns associated with the development of significant deformity should not be underestimated in patient with untreated AIS [22]. These long-term favorable outcomes of untreated AIS have clearly to be opposed to those of the other subtypes of pediatric IS (infantile and juvenile), belonging to the early onset scoliosis group. The prognosis of these patients, when left untreated, is associated with significantly increased mortality rate at a younger age when compared to AIS patient, because of respiratory failure in a large number of cases [13, 14]. Unfortunately, spinal fusion in young children with progressing scoliosis deformity, even anterior and posterior, does not offer systematic prevention of progression and is associated with negative effects on thoracic growth and hence pulmonary function [5]. Taken together, these data support aggressive screening of spinal deformity in children under the age of 10 and, when indicated, the choice of treatment modality preserving the growth of the spine, i.e. with brace or growth friendly surgical treatment based on distraction (dual growing rods (Fig. 1), vertical expandable prosthetic expandable rib (VEPTR)) or guided growth (Shilla procedure or modern Luqué-Trolley) [8, 11, 19, 20]. None of these surgical techniques are associated with low complications rates and experienced surgical teams should use them accordingly.



Fig. 1 Infantile scoliosis: dual growing rods technique.

Regarding AIS, the choice of treatment is still based most of the times on curve magnitude and potential for growth. The Risser grading system is often used to estimate skeletal maturity. Observation is usually proposed for growing adolescent patient with a major curve of less than 25° or for skeletally mature patient with a curve measuring less than 50°. Bracing is proposed for growing adolescent with curves from 25° to 40°. Surgery is generally offered for growing AIS patients with curves exceeding 40° or for skeletally mature patient with curve beyond 50°. A large number of research studies have demonstrated potential disorders associated to AIS, including abnormalities affecting the central and the peripheral nervous system maturation, the connective tissues, muscles and bones. Platelet disorders and several molecular biology anomalies (such as melatonin, calmodulin and growth hormones levels) have also been related to AIS [3]. However daily practice will probably

be more positively affected by establishment of definitive prognostic factors for AIS progression derived from genetic testing, with the hope to further decrease repetitive radiographs, unnecessary bracing, psychological burden and costs-of-care related to follow-up in low-risk patients [9, 10].

The whole perioperative strategy for the surgical treatment of IS should be directed at preventing complications. A blood management protocol including preoperative evaluation, intraoperative cell salvage, topical hemostasis, antifibrinolytics, and hypotensive anesthesia is generally used for the surgical treatment of the pediatric patient with IS, as well as in general for pediatric deformity surgery. Preoperative oral iron and erythropoietin or autologous blood predonation can be used in this setting. Implementation of such a protocol is associated with a low perioperative transfusion rate together with preoperative diagnosis of patients with coagulopathy [4]. Intraoperative neuromonitoring combining data of somatosensory-evoked potential (SSEP) and transcranial motor-evoked potential (tcMEP), to increase the sensitivity and specificity for detection of intraoperative neurologic injury, should also probably be considered as a standard of care for the surgical treatment of IS patients [12]. Intraoperative computed tomography-guided navigation is a promising tool to further assist in the accuracy and safe placement of pedicle screws [21], especially with the increasing trend to use posterior-only approach (Fig. 2 and 3) with a high number of pedicle screws for definitive fusion in adolescent patients.

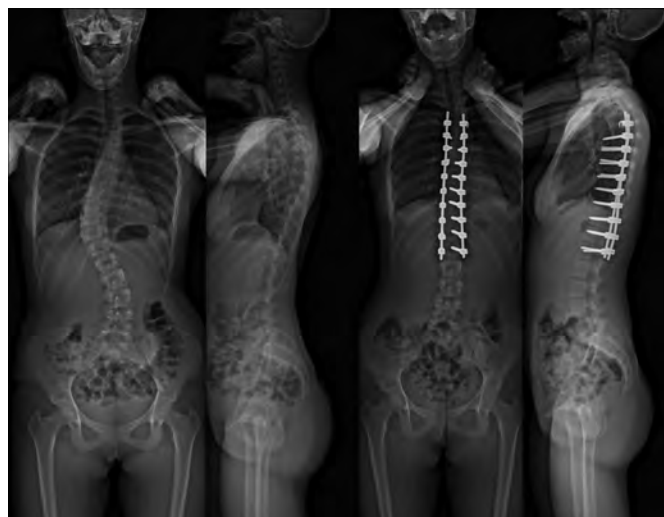


Fig. 2 AIS: posterior-only approach and instrumented fusion of a thoracic curve.

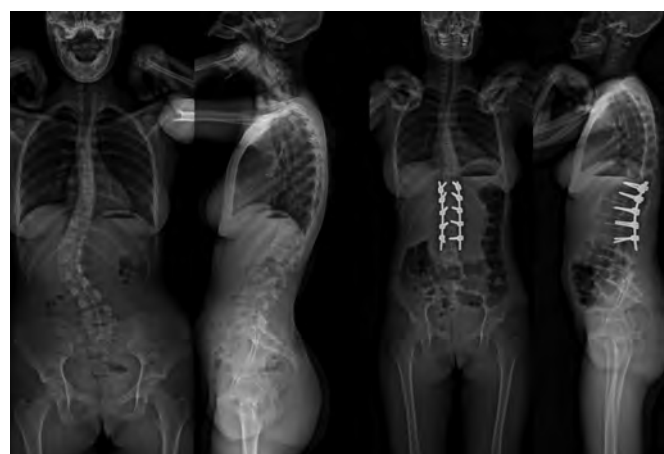


Fig. 3 AIS: posterior-only approach and instrumented fusion of a thoraco-lumbar curve.

This surgical approach is clearly the most frequently favored for the surgical treatment of AIS together with aggressive derotation technique, with the potential benefit to decrease the number of fused levels and increase postoperative patient satisfaction regarding cosmesis [2]. Anterior-only approach (open or endoscopic) is less frequently used but is still a valuable option for the treatment of thoracic or thoracolumbar/lumbar curves (Fig. 4). Finally, some surgeons developed for selected AIS cases a minimally invasive posterior approach. This technique is technically demanding and more time-consuming than the traditional posterior approach, but early results suggest encouraging perspectives with reduced blood losses and length of hospital stay [16].



Fig. 4 AIS: anterior-only approach and instrumented fusion of a thoraco-lumbar curve.

In conclusion, IS current areas of clinical research could probably be summarized as two main challenges. The first one concern patients affected by infantile and juvenile IS, with the priority to better define, standardize and secure the surgical treatment of their growing spine. The second one is devoted to adolescent patients with the aim to more accurately identify patients at risk of significant curve progression and concomitantly to increase the effectiveness of AIS follow-up in low-risk patients.

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References

1. Scoliosis Research Society (SRS) Available from: <http://www.srs.org/>.
2. Cuartas E, Rasouli A, O'Brien M, et al. Use of all-pedicle-screw constructs in the treatment of adolescent idiopathic scoliosis. *J Am Acad Orthop Surg* 2009; 17:550-61.
3. Dayer R, Lascombes P. Idiopathic scoliosis: etiological concepts and hypotheses. *Journal of Children's Orthopaedics* 2013;In Press.
4. Hassan N, Halanski M, Wincek J, et al. Blood management in pediatric spinal deformity surgery: review of a 2-year experience. *Transfusion*; 51:2133-41.
5. Karol LA. Early definitive spinal fusion in young children: what we have learned. *Clin Orthop Relat Res*; 469:1323-9.
6. King HA, Moe JH, Bradford DS, et al. The selection of fusion levels in thoracic idiopathic scoliosis. *J Bone Joint Surg Am* 1983;65:1302-13.
7. Lenke LG, Betz RR, Harms J, et al. Adolescent idiopathic scoliosis: a new classification to determine extent of spinal arthrodesis. *J Bone*

- Joint Surg Am* 2001;83-A: 1169-81.
8. McCarthy RE, Sucato D, Turner JL, et al. Shilla growing rods in a caprine animal model: a pilot study. *Clin Orthop Relat Res*; 468:705-10.
9. Ogilvie JW. Update on prognostic genetic testing in adolescent idiopathic scoliosis (AIS). *J Pediatr Orthop*; 31:S 46-8.
10. Ogilvie JW, Braun J, Argyle V, et al. The search for idiopathic scoliosis genes. *Spine (Phila Pa 1976)* 2006; 31:679-81.
11. Ouellet J. Surgical technique: modern Luque trolley, a self-growing rod technique. *Clin Orthop Relat Res*; 469:1356-67.
12. Pahys JM, Guille JT, D'Andrea LP, et al. Neurologic injury in the surgical treatment of idiopathic scoliosis: guidelines for assessment and management. *J Am Acad Orthop Surg* 2009; 17:426-34.
13. Pehrsson K, Larsson S, Oden A, et al. Long-term follow-up of patients with untreated scoliosis. A study of mortality, causes of death, and symptoms. *Spine (Phila Pa 1976)* 1992; 17:1091-6.
14. Pehrsson K, Nachemson A, Olofson J, et al. Respiratory failure in scoliosis and other thoracic deformities. A survey of patients with home oxygen or ventilator therapy in Sweden. *Spine (Phila Pa 1976)* 1992; 17:714-8.
15. Ramirez N, Johnston CE, Browne RH. The prevalence of back pain in children who have idiopathic scoliosis. *J Bone Joint Surg Am* 1997; 79:364-8.
16. Sarwahi V, Wollowick AL, Sugarman EP, et al. Minimally invasive scoliosis surgery: an innovative technique in patients with adolescent idiopathic scoliosis. *Scoliosis*; 6:16.
17. Sato T, Hirano T, Ito T, et al. Back pain in adolescents with idiopathic scoliosis: epidemiological study for 43,630 pupils in Niigata City, Japan. *Eur Spine J*; 20:274-9.
18. Schulte TL, Hierholzer E, Boerke A, et al. Raster stereography versus radiography in the long-term follow-up of idiopathic scoliosis. *J Spinal Disord Tech* 2008; 21:23-8.
19. Smith JT. Bilateral rib-to-pelvis technique for managing early-onset scoliosis. *Clin Orthop Relat Res*; 469:1349-55.
20. Tis JE, Karlin LI, Akbarnia BA, et al. Early onset scoliosis: modern treatment and results. *J Pediatr Orthop*; 32:647-57.
21. Ughwanogho E, Patel NM, Baldwin KD, et al. Computed tomography-guided navigation of thoracic pedicle screws for adolescent idiopathic scoliosis results in more accurate placement and less screw removal. *Spine (Phila Pa 1976)*; 37:E473-8.
22. Weinstein SL, Dolan LA, Spratt KF, et al. Health and function of patients with untreated idiopathic scoliosis: a 50-year natural history study. *Jama* 2003; 289:559-67.
23. Wybier M, Bossard P. Musculoskeletal imaging in progress: The EOS imaging system. *Joint Bone Spine*.



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Management of spinal trauma

Acute traumatic spinal cord injuries and spinal column fractures are common. SCI has a major impact on function. Although initial trauma to spinal cord cannot be controlled, prevention of secondary damage to spinal cord is utmost importance. Most of the research is either on the prevention of secondary damage or repair of the damaged spinal cord roots or cauda equina. Outcome depends on well-conducted specialized management both in the emergency and treatment phases.

1. Epidemiology

It is shown that although spinal injury occurs more commonly in the older child, it can be seen in younger children also. However, when it does occur in younger children (aged 8 or less), it is associated with a higher prevalence of cord injury. In particular, the SCIWORA syndrome is primarily a disease of the young child. This is consistent with data from the National Pediatric Trauma Registry [1, 2].

Road traffic accident (RTA) is the most frequent mechanism of spinal column injury in children. This accounted for 39% to 52% of all injuries, rising to 66.1% of cord injuries [3-7].

7.4% of children with spine fractures/dislocations have injuries in more than one spinal region [3].

In general population the estimated incidence varied from 3.3 to 130.6 individuals with traumatic SCI per million inhabitants a year in Europe. Most recent estimates from Europe indicate MVCs to be the leading cause of SCIs in regions of Spain, and Turkey, and the second most common in regions of Norway [8], and so on.

SCI primarily affects young adults. Reports from National Spinal Cord Injury Statistical Center in USA the average age at injury was 28.7 years from 1973 to 1979. However, as the median age of the general population of the United States has increased by approximately 9 years since the mid-1970, the average age at injury has also steadily increased over time. Since 2005, the average age at injury is 41.0 years. 80.6% of spinal cord injuries reported have occurred among males. Causes of SCI were Motor vehicle crashes 39.2%, falls 28.3%, violence 14.6%, sports 8.2% and other or unknown 9.7% [9].

In 10% of patients, injury involves more than one level, emphasizing the importance of a careful and complete spinal work-up. The combination of chest trauma, head trauma and severely altered consciousness points to a high risk of spinal and / or spinal cord injury [10].

2. Classification

Classification systems in spinal fractures have evolved parallel with the understanding of spine biomechanics and stability. An ideal classification should address the severity of injury, neurological involvement and the stability of the spine as well as guiding physician in treatment options.

Such classification should also be reliable, reproducible, all-inclusive, mutually exclusive, user friendly and clinically useful. Second role of classification system is to provide a common communication platform between spine specialists. The notion of instability, introduced by Watson-Jones [11] and later developed by White and Panjabi [12] has been used in many classifications. Emerging information on the importance of soft tissue involvement has been reflected in the classification systems and treatment algorithms [13,14].

2.1 Cervical spine

Upper cervical spine and sub axial spine have distinct anatomical differences.

Therefore one unified classification system cannot address specific injuries. Levin and Edwards (C1 fractures), Fielding (C1-C2 dislocations), Anderson Alonzo (Odontoid fractures) and Effendi (C2 ring fractures) classifications are used in upper cervical spine [15-18]. Ferguson and Allen classification system is usually used for sub axial spine fractures [19]. A new classification of the lower cervical spine has been recently proposed associating a morphological description of the injuries with the ligament involvement [20].

2.2 Thoracolumbar spine

Böhler was the first to classify spine fractures with mechanistic approach and introduced flexion-distraction and torsional injuries in 1929 [21]. Later in 1938 Watson-Jones classified fractures upon their morphology [22]. Nicoll in 1942 introduced the stability concept [23].

Holsworth introduced two-column classification in 1963 and emphasized the importance of posterior column. In 1977 R. Louis brought three-column theory where anterior vertebral corpus and two posterior facet joint lines constituted the columns.

Denis further developed the three-column concept and created an imaginary third middle column. Middle column is composed of the posterior longitudinal ligament and the posterior half of the disc and the vertebral body; although this is not an anatomic column biomechanically it has been shown to act like one.

The columns described in Denis's classification are:

- The anterior column includes the anterior halves of the vertebral bodies and the anterior longitudinal ligament.
- The middle column includes the posterior halves of the vertebral bodies and the posterior longitudinal ligament.
- The posterior column includes the facet joints, pedicles, and supraspinous ligaments.

Denis described four types of injuries: (compression, burst, seat belt, dislocation) with involvement of one or more columns and the basic mode of fracture (compression, flexion-distraction, rotation) [24].

- Compression fractures are classified according to acting force vector being anterior or lateral flexion. These injuries cause anterior column compression and sometimes associated posterior column distraction.
- Burst fracture results from an axial load vector. However, flexion, rotation, or lateral flexion forces may accompany and results in anterior and middle column compression, and may sometimes also produce associated posterior column distraction.
- Seat belt fractures: flexion-disruption mechanism. The anterior column may be intact or distracted, but the middle and posterior columns are distracted.
- Fracture-dislocations are classified according to whether the dislocation involves rotation, shear, or flexion-distraction. These injuries can produce any pattern of column involvement.

Magerl in his classification united morphological and mechanical criteria to classify thoracolumbar vertebra fractures [25]. Spinal injuries are described by a 3-3-3 grid for three types of morphological damage of increasing severity.

A. Compression (posterior soft tissue must be intact even if there is some posterior element fracture)

- A1: Wedge
- A2: Split or coronal
- A3: Burst.

B. Distraction

- B1: Distraction of the posterior soft tissues
- B2: Distraction of the posterior arch
- B3: Distraction of the anterior disc

C. Multi-directional with translation

- C1: Anterior-posterior (dislocation)
- C2: Lateral (lateral shear)
- C3: Rotational (rotational burst)

Magerl classification is commonly used, as it provides a comprehensive classification describing the nature of injury, the degree of instability, and prognostic aspects that are important for choosing the most appropriate treatment. However, there have been reports that AO system lacks reliability and reproducibility [26, 27].

McCormack and Gaines evaluated patients who underwent posterior short segment fixation and failed. They came out with an algorithm based on the comminution, apposition of the fractured vertebra and the residual kyphosis grading from 1 to 3 with increasing severity. Authors recommended anterior support to those received 7 or more points with their proposed classification [28].

New classification system for thoracolumbar fractures, also known as the Thoracolumbar Injury Severity Score (TLISS), was devised by the Spine Trauma Study Group (STSG) and has been validated and shown to have good reliability [20, 29, 30].

This classification system is based on 3 injury components:

1. Morphology of injury determined by radiographic appearance,
 - Compression fracture: 1 point
 - Compression fracture plus burst: 2 points
 - Translational/rotational fracture: 3 points
 - Distraction fracture: 4 points

2. Integrity of the posterior ligamentous complex, and

- Intact: 0 points
- Suspected/indeterminate: 2 points
- Injured: 3 points

3. Neurological status of the patient.

- Intact: 0 points
- Nerve root: 2 points
- Cord, conus medullaris
 - Complete: 2 points
 - Incomplete: 3 points
 - Cauda equina: 3 points

3 or less points suggests a non-operative management, while a total score of 5 or more suggests that surgical intervention may be considered.

A score of 4 might be handled conservatively or surgically.

This classification system also provides a guide for determining the optimal approach (anterior, posterior, combined anterior-posterior) for surgically treated patients based on:

1. Disruption of the posterior ligamentous complex, and
2. Neurological status.

3. Management of acute spinal injuries

The management of acute spinal injuries starts at the injury scene. The goal of the prehospital management is to stabilize vital functions and avoid neurological deterioration and secure patient to be transferred safely to hospital.

Initial evaluation at the scene includes airway maintenance with cervical spine immobilization, assessment of breathing and ventilation, and assessment of circulation with hemorrhage control.

Use of trauma board and rigid immobilization of the spinal column should be achieved. Care should be taken when immobilizing small child as their head is bigger compared to their body. Achieving hemodynamic stability and sufficient oxygenation is essential to avoid secondary spinal cord injury due to hypoxia. All trauma victims should be considered and addresses as having spinal injury until otherwise proven by the secondary evaluation at the hospital. Use of ASIA neurologic injury data collection charts is very important to detect progressive neurologic deteriorating patients [31].

Patients may have one of 2 types of hypotension.

a -Hypotension with tachycardia: Usually not due to the spine injury, and other causes should be sought (e.g., hemorrhage, dehydration).

b -Hypotension with bradycardia or normal heart rate: This is neurogenic shock and should be managed with fluid/blood replacement, taking care not to cause fluid overload and pulmonary edema; if it persists, vasopressors should be started. The use of vasopressors however, should be left to the intensive care setting use [32].

4. Imaging modalities

4.1 Plain film radiography

Today multislice CT (MSCT) became the initial imaging modality. In cases where multislice MSCT is not available, plain film radiographs still remains the initial imaging modality. Lateral, anteroposterior, and open-mouth, odontoid views are needed for cervical spine traumatized patients. Often, additional views, such as oblique views or the swimmer's view, are performed in an attempt to clear the cervicothoracic junction.

Every effort should be made to visualize C7 vertebra to clear the cervical spine. The sensitivity of plain X-Ray films in diagnosing spinal injuries varies, ranging from 39% to 94%, with variable specificity [33-39]. If only plain radiographs are used, 23% to 57% of all fractures of the cervical spine might be missed therefore delays in the diagnosis and treatment cervical spine injuries are noted (5% to 23%) [39-41].

4.2 Computerized Tomography

Today most level-1 trauma centers prefer CT (MSCT) as the initial method of choice when evaluating the cervical spine for bone injuries [42-47]. CT has higher sensitivity than plain X-Ray films in detecting fractures and soft tissue abnormalities, such as disc hernia ion and Para vertebral soft tissue and intraspinal hematoma may also be shown. Other advantages of MSCT would be 1-short examination time of whole spine, 2-sagittal and frontal reformat images in desired planes, 3-three dimensional viewing of complex injuries [48].

4.3 MR imaging

It has been shown that MR is the gold standard for assessing soft tissue injuries, spinal cord injury, intervertebral discs and ligaments, and vascular injuries [49, 50]. MR imaging is the only method that can differentiate spinal cord hemorrhage from edema, which might be of prognostic significance and possibly may change the treatment.

5. Treatment

The goals of spine trauma care should include protection against further injury during transport, evaluation and management; identification of spine injury and documentation of the clinical findings; optimization of conditions for neurological recovery; maintenance and restoration of spinal alignment; obtaining a stable and healed spinal column; and facilitation of rehabilitation [51]. The most important factors to consider when deciding on treatment for patients with a thoracolumbar spine fracture are neurological status, spinal stability, degree of deformity, and associated injuries.

Spinal fracture with spinal cord injury.

There are two controversial issues in the management of spinal cord injury: timing and the use of neuroprotective agents.

Intravenous methylprednisolone has been recommended for acute spinal cord injury in the past; [52-54] however, its use is controversial due to severe adverse effects (e.g., decreased wound healing, infections, increased mortality), and problems with the method of studies concerning its use [55]. Riluzole is a sodium channel blocking benzothiazole anticonvulsant drug that is FDA approved for the treatment of Amyotrophic Lateral Sclerosis and has shown efficacy in preclinical models of SCI in reducing the extent of sodium and glutamate-mediated secondary injury. It is currently in the early stages of clinical investigation for SCI and shows promise as an acute neuroprotective therapy in this context [56].

Recently published results of STASCIS study revealed beneficial outcome in early (<24 hours) decompressive surgery in spinal cord injury [57]. Those operated earlier than 24 hours benefited average 2 grades change in ASIA impairment score.

Non-operative management is indicated if all the following general conditions are met: no neurological deficit, resolving deficit, or deficit that does not correlate to demonstrable compression, deformity, or instability; acceptable alignment (initial or after postural reduction), compression fracture <50% of vertebral body height; and angulation

<20°.

We can define 6 clinical scenarios that are usually encountered at clinical setting: 1-Posterior ligamentous complex intact and neurologically intact, 2 -Neurologically Intact and Posterior Ligaments Disrupted, 3. Neurologically Incomplete or Cauda Equina Injury and Posterior Ligaments Intact, 4. Neurologically Incomplete or Cauda Equina Injury and Posterior Ligaments Disrupted, 5. Neurologically Complete and Posterior Ligaments Intact, 6. Neurologically Complete and Posterior Ligaments Disrupted. These fractures occur in the combination of types (compression, burst, fracture dislocation, chance) as previously described [58]. In clinical scenario one, surgical intervention is rarely needed. Most compression fractures are relatively stable injuries that nonoperative treatment in a thoracolumbar orthosis for twelve weeks can be made. It is very important to rule out a potentially more unstable burst fracture and to confirm the integrity of the posterior column [59].

The extent of collapse and kyphosis as well as the integrity of the posterior column are key factors of stability and should play a role in deciding between surgical and nonoperative treatment. Progressive neurological deterioration in the presence of substantial canal compromise is an indication for surgical decompression and stabilization. Moreover, when the type of treatment is being decided, it must be kept in mind that many studies have failed to reveal a substantial difference in functional outcome between operative and nonoperative treatment of thoracolumbar spine fractures, regardless of the presence of neurological injury [59-67]. There may be instances that surgical treatment is warranted. Burst fractures with greater comminution and vertebral height loss greater than 50% may need posterior or anterior surgery in surgeons' preference.

2. Neurologically Intact and Posterior Ligaments Disrupted: Most thoracolumbar injuries involving disruption of the posterior ligaments include severe compression fractures, burst fractures, distraction injuries, or translational injuries. Most of the surgeons participating in Vaccaro's study [58] agreed on operating neurologically intact patient with radiographic evidence of compromised posterior ligaments would be best stabilized by a posterior approach.

3. Neurologically Incomplete or Cauda Equina Injury and Posterior Ligaments Intact: Most commonly seen in severe burst fractures but may also be seen in a distraction extension injury or a flexion distraction injury through bone only. A neurologically incomplete patient (ASIA B-D) without evidence of posterior ligamentous disruption is best served by spinal cord or cauda equina decompression to allow for maximal neurologic recovery. In thoracolumbar spine injuries, this decompression is most directly achieved through an anterior approach

4. Neurologically Incomplete or Cauda Equina Injury and Posterior Ligaments Disrupted: most commonly seen in severe burst fractures, flexion distraction injuries (through posterior ligaments), and translational injuries (Fig. 1). Burst-type injury accompanied by incomplete spinal cord/cauda equina injury with documented neural compression, warrants an anterior decompression. Due to the presence of a ruptured posterior ligamentous complex, posterior stabilization may be needed to augment anterior stabilization. The need for combined anterior and posterior (360°) approaches in a severe burst injury with incomplete neurologic injury and disruption of the posterior ligaments was agreed upon by 82% of study population in Vaccaro's study [58].

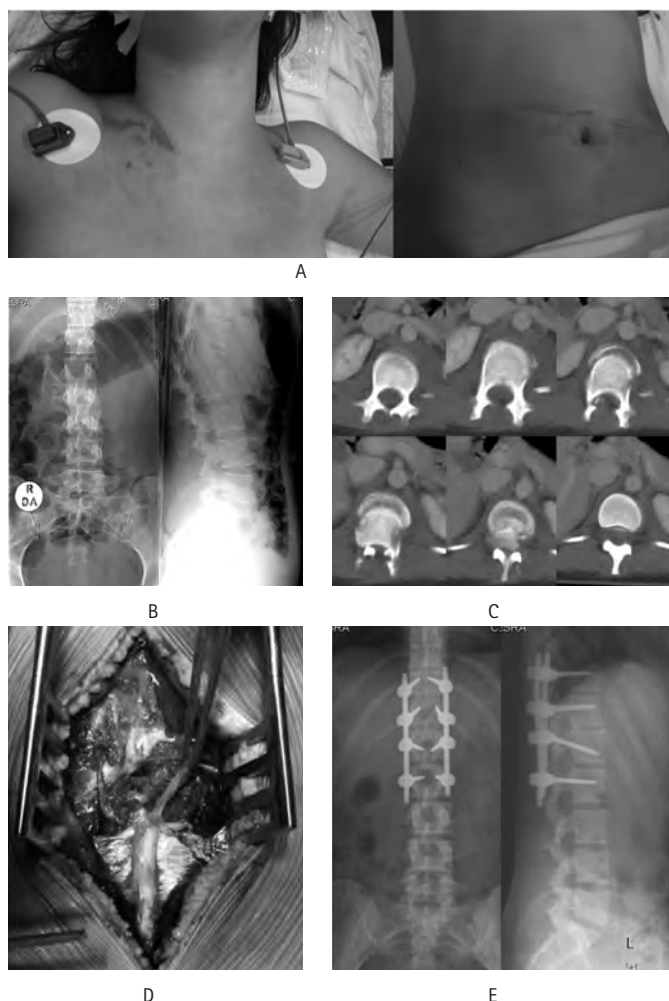


Fig. 1 17 years old Female patient sustained a motor vehicle accident. A. Clinical pictures showing seat belt injury. B. preoperative AP and lateral thoracolumbar X-Rays depicts a flexion extension injury. C. CT images clearly show "empty facet sign" typical to facet dislocation. Superior facet of the caudal vertebral is located within the spinal canal thus compressing spinal cord. D. Intraoperative images show a complete rupture of posterior ligamentous complex. E. Post operative images depicting posterior instrumentation.

5. Neurologically Complete and Posterior Ligaments Intact: Mostly they are severe burst fractures, flexion distraction injuries through bone or a distraction extension injuries through the posterior elements. Spinal cord or neural element injury occurs due to canal compromise due to translation or bone fragments from anterior to the canal. In case of a total spinal cord injury decompression usually is not beneficial. Preferred treatment of choice usually is posterior approach aimed at stabilization and realignment. These types of injuries are generally due to high-energy trauma and the spine is biomechanically destabilized. Anterior and posterior stabilization might be preferred [58].

6. Neurologically Complete and Posterior Ligaments Disrupted

Severe compressive burst injuries, translational injuries, and distraction injuries all contribute to this type of clinical presentation. In the absence of salvageable neurologic function, a posterior exposure and fixation procedure is the surgical pathway of choice [58].

Isolated anterior column injuries in elderly

Generally analgesia and bed rest for 1 to 3 weeks would be sufficient. If pain subsides, mobilization with or without brace may be commenced. Follow-up is recommended to rule out progressive deformity. Vertebro-

plasty or kyphoplasty will be an option in elderly patients with intractable pain, or anterior wedging of vertebral body with an intact posterior cortex of vertebral body [68]. Vertebroplasty is used for painful osteoporotic compression fractures of less than 1 month's duration and those of <30% vertebral body compression. Kyphoplasty is used for painful osteoporotic compression fractures of less than 3 months' duration and those of ≥30% vertebral body compression.

DVT prophylaxis

Appropriate DVT prophylaxis is recommended to prevent DVT and pulmonary embolism. Compression stockings and long-term anticoagulation with either low molecular weight heparin or adjusted-dose unfractionated heparin should be commenced within 72 hours of the initial injury. Compression stockings should be continued for 2 weeks. Anticoagulation should be continued for 8 weeks. However, if additional risk factors for thromboembolism are present (e.g., lower limb fractures, a history of thrombosis, cancer, heart failure, obesity, or age >70 years), anticoagulation should be continued for 12 weeks, or until discharge from rehabilitation.

References

- Patel JC, Tepas III JJ, Mollitt DL, et al. Pediatric cervical spine injuries: defining the disease. *J Pediatr Surg* 2001;36:373–6.
- Kokoska ER, Keller MS, Rallo MC, et al. Characteristics of pediatric cervical spine injuries. *J Pediatr Surg* 2001;36:100–5.
- Martin BW, Dykes E, Lecky F E. Patterns and risks in spinal trauma. *Arch. Dis. Child.* 2004;89:860–865.
- Hadley MN, Zabramski JM, Browner CM, et al. Pediatric spinal trauma. Review of 122 cases of spinal cord and vertebral column injuries. *J Neurosurg* 1998;68:18–24.
- Osenbach RK, Menezes AH. Pediatric spinal cord and vertebral column injury. *Neurosurgery* 1992;30:385–90.
- Hamilton MG, Myles ST. Pediatric spinal injury: review of 174 hospital admissions. *J Neurosurg* 1992;77:700–4.
- Mann DC, Dodds JA. Spinal injuries in 57 patients 17 years or younger. *Orthopedics* 1993;16:159–64.
- <http://www.scireproject.com/book/export/html/10>
- https://www.nscisc.uab.edu/public_content/annual_stat_report.aspx
- Hasler RM, Exadaktylos AK, Bouamra O et al. Epidemiology and predictors of spinal injury in adult major trauma patients: European cohort study. *Eur Spine J* 5 June 2011.
- Watson-Jones R. (1938) *The results of postural reduction of fractures of the spine.* *J Bone Joint Surg Am*;20:567–86.
- White AA, Panjabi MM. (1978) *Clinical Biomechanics of the Spine.* Philadelphia: Lippincott.
- Weidenbaum M, farcy JP *The Textbook of Spinal surgery* 2nd edition Philadelphia, PA: Lippincott Raven; 1997:1839.
- Vaccaro AR, Lehman RA, Hurlbert PA, et al. A new classification of thoracolumbar injuries: the importance of injury morphology, the integrity of the posterior ligamentous complex, and neurologic status. *Spine.* 2005;30:2325–2333.
- Levine AM, Edwards CC *The management of traumatic spondylolisthesis of the axis.* *J Bone Joint Surg* (1985), 67A: 217–26.
- Fielding WJ, Hawkins RJ *Atlanto-axial rotatory fixation.* *J Bone Joint Surg* (1977) 59A: 37–44.
- Anderson LD, d'Alonzo RT (1974) *Fractures of the odontoid process of the axis.* *J Bone J Surg* 56-A:1663–74.

20. Effendi B, Roy D, Cornish B, Dussault RG, Laurin CA (1981) Fractures of the ring of the axis. A classification based on the analysis of 131 cases. *J Bone Joint Surg* 63B: 319–27.
21. Allen BL, Ferguson RI, Lehmann TR, O'Brien RP (1982) A mechanistic classification of closed, indirect fractures and dislocations of the lower cervical spine. *Spine* 7: 1–27.
22. Vaccaro AR, Anderson PA. Classification of Lower Cervical Spine Injuries. *Spine* 2006; 31:11 Suppl, pp S37–S43.
23. Böhler L. Mechanisms of fracture and dislocation of the spine. In: Böhler L, ed. *The Treatment of Fractures*, vol. 1, ed. 5. New York: Grune & Stratton, 1956:300–29.
24. Watson Jones JBJS 1938; 20A:567–586.
25. Nicoll JBJS 1949; 31B:376–394.
26. Denis F. The three-column spine and its significance in the classification of acute thoracolumbar spinal injuries. *Spine* 1983;8:817–31.
27. Magerl F, Aebi M, Gertzbein SD, et al. A comprehensive classification of thoracic and lumbar injuries. *Eur Spine J* 1994;3:184–201.
28. Blauth M, Bastian L, Knop C, et al. Inter-observer reliability in the classification of thoraco-lumbar spinal injuries. *Orthopaedics* 1999;28:662–81.
29. Wood, K.B., et al. Assessment of two thoracolumbar fracture classification systems as used by multiple surgeons. *J Bone Joint Surg Am* 2005;87:1423–9.
30. McCormac T, Karaikovic E, Gaines RW. The load sharing classification of spinal fractures.
31. Lenarz CJ, Place HM. Evaluation of a new spine classification system, does it accurately predict treatment? *J Spinal Disord Tech*. 2010;23(3):192–196. Abstract(external link).
32. Vaccaro AR, Baron EM, Sanfilippo J, et al. Reliability of a novel classification system for thoracolumbar injuries: the Thoracolumbar Injury Severity Score. *Spine*. 2006;31: S62–S69.
33. Fisher CG, Noonan V, Smith DE, Wing PC, Dvorak M, Kwon B (2005) Motor Recovery, Functional Status, and Health-Related Quality of Life in Patients With Complete Spinal Cord Injuries. *Spine* 30: 2200–7.
34. American College of Surgeons. Advanced trauma life support manual, 7th ed. Chicago, IL: American College of Surgeons; 2004.
35. Stiell IG, Wells GA, Vandemheen KL, et al. The Canadian C-spine rule for radiography in alert and stable trauma patients. *JAMA* 2001;286: 1841–8.
36. Frankel HL, Rozycki GS, Ochsner MG, et al. Indications of obtaining surveillance thoracic and lumbar spine radiographs. *J Trauma* 1994;37:673–6.
37. Widder S, Doig C, Burrows P, et al. Prospective evaluation of computed tomographic scanning for spinal clearance of obtunded trauma patients: preliminary results. *J Trauma* 2004;56: 1179–84.
38. Blackmore CC, Ramsey SD, Mann FA, et al. Cervical spine screening with CT in trauma patients: a cost effectiveness analysis. *Radiology* 1999;212:117–25.
39. Brandt MM, Wahl WL, Yeom K, et al. Computed tomographic scanning reduces costs and time of complete spine evaluation. *J Trauma* 2004;56:1022–8.
40. Tins BJ, Cessar-Pullicino VN. Imaging of acute cervical spine injuries: review and outlook. *Clin Radiol* 2004;59:865–80.
41. Blackmore CC, Mann FA, Wilson AJ. Helical CT in the primary trauma evaluation of the cervical spine: an evidence based approach. *Skeletal Radiol* 2000;29:632–9.
42. Nunez DB, Quencer RM. The role of helical CT in the assessment of cervical spine injuries. *AJR Am J Roentgenol* 1998;171:951–7.
43. Nunez DB, Zuluaga A, Fuentes Bernardo DA, et al. Cervical spine trauma; how much do we learn by routinely using helical CT? *Radiographics* 1996;16:1307–18.
44. Diaz JJ Jr, Gillman C, Morris JA Jr, et al. Are five view plain films of the cervical spine unreliable? A prospective evaluation in blunt trauma in patients with altered mental status. *J Trauma* 2003;55:658–63.
45. Griffen MM, Frykberg ER, Kerwin AJ, et al. Radiographic clearance of blunt cervical spine injury: plain radiograph or computed tomography scan? *J Trauma* 2003;55:222–6.
46. Holmes JF, Mirvis SE, Panacek EA, et al. For the NEXUS Group. Variability in computed tomography and magnetic resonance imaging in patients with cervical spine injuries. *J Trauma* 2002;53:524–9.
47. Kligman M, Vasili C, Roffman M. The role of computed tomography in cervical spine injury due to diving. *Arch Orthop Trauma Surg* 2001;121:139–41.
48. Schenarts PJ, Diaz J, Kaiser C, et al. Prospective comparison of admission computed tomographic scan and plain films of the upper cervical spine in trauma patients with altered mental status. *J Trauma* 2001;51:663–8.
49. Berne JD, Velmahos GC, El Tawil Q, et al. Value of complete cervical helical computed tomographic scanning in identifying cervical spine injury in the unevaluable blunt trauma patient with multiple injuries: a prospective study. *J Trauma* 1999;47:896–902.
50. Woodring JH, Lee C. The role and limitations of computed tomography scanning in the evaluation of cervical trauma. *J Trauma* 1992;33(5): 698–708.
51. Flanders AE, Schaefer DM, Doan HT, et al. Acute cervical spine trauma; correlation of MR imaging findings with degree of neurological deficit. *Radiology* 1990;177:25–33.
52. Wilmink JT. MR imaging of the spine: trauma and degenerative disease. *Eur Radiol* 1999;9(7): 1259–66.
53. Bucholz RM, Heckman JD. Rockwood and Green's fractures in adults, 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2002:1303.
54. Bracken MB, Collins WF, Freeman DF, et al. Efficacy of methylprednisolone in acute spinal cord injury. *JAMA*. 1984;251:45–52.
55. Bracken MB, Sheppard MJ, Collins WF, et al. A randomized, controlled trial of methylprednisolone or naloxone in the treatment of acute spinal-cord injury: results of the Second National Acute Spinal Cord Injury Study. *New Engl J Med*. 1990;322:1405–1411.
56. Bracken MB, Shepard MJ, Holford TR, et al. Administration of methylprednisolone for 24 or 48 hours or tirilazad mesylate for 48 hours in the treatment of acute spinal cord injury: results of the Third National Acute Spinal Cord Injury Randomized Controlled Trial. *National Acute Spinal Cord Injury Study*. *JAMA*. 1997;277:1597–1604.
57. Hurlbert RJ, Moulton R. Why do you prescribe methylprednisolone for acute spinal cord injury? A Canadian perspective and a position statement. *Can J Neurol Sci*. 2002; 29:236–239.
58. Wilson JR, Fehlings MG, Riluzole for Acute Traumatic Spinal Cord Injury: A Promising Neuroprotective Treatment Strategy, *World Neurosurgery* (2013), doi: 10.1016/j.wneu.2013.01.001.
59. Fehlings MG, Vaccaro A, Wilson JR, Singh A, Cadotte DW, et al. (2012) Early versus Delayed Decompression for Traumatic Cervical Spinal Cord Injury: Results of the Surgical Timing in Acute Spinal Cord Injury Study (STASCIS). *PLoS ONE* 7(2): e32037.
60. Vaccaro AR, Lim MR, Hurlbert JR, Lehman RA, Harrop J, Fisher C, MD, Dvorak M, Anderson G, Zeiller SC, Lee JY, Fehlings MG, Oner FC, and Spine Trauma Study Group. Surgical Decision Making for Unstable Thoracolumbar Spine Injuries Results of a Consensus Panel Review by the Spine Trauma Study Group. *J Spinal Disord Tech* 2006;19:1–10.

61. Vaccaro AR, Kim DH, Brodke DS, Harris M, Chapman J, Schildhauer T, Chip Routt ML, Sasso RC. *Diagnosis and Management of Thoracolumbar Spine Fractures*. *J Bone Joint Surg Am*. 2003; 85:2456-2470.
62. Burke DC, Murray DD. *The management of thoracic and thoracolumbar injuries of the spine with neurological involvement*. *J Bone Joint Surg Br*. 1976;58:72-8.
63. Cantor JB, Lebowitz NH, Garvey T, Eismont FJ. *Nonoperative management of stable thoracolumbar burst fractures with early ambulation and bracing*. *Spine*. 1993;18:971-6.
64. Fredrickson BE, Yuan HA, Bayley JC. *The nonoperative treatment of thoracolumbar injuries*. *Semin Spine Surg*. 1990;2:70-8.
65. Kraemer WJ, Schemitsch EH, Lever J, McBroom RJ, McKee MD, Waddell JP. *Functional outcome of thoracolumbar burst fractures without neurological deficit*. *J Orthop Trauma*. 1996;10:541-4.
66. Mumford J, Weinstein JN, Spratt KF, Goel VK. *Thoracolumbar burst fractures. The clinical efficacy and outcome of nonoperative management*. *Spine*. 1993;18:955-70.
67. Rehtine GR 2nd, Cahill D, Chrin AM. *Treatment of thoracolumbar trauma: comparison of complications of operative versus nonoperative treatment*. *J Spinal Disord*. 1999;12:406-9.
68. Weinstein JN, Collalto P, Lehmann TR. *Longterm follow-up of nonoperatively treated thoracolumbar spine fractures*. *J Orthop Trauma*. 1987;1:152-9.
69. Weinstein JN, Collalto P, Lehmann TR. *Thoracolumbar "burst" fractures treated conservatively: a long-term follow-up*. *Spine*. 1988;13:33-8.
70. Lovi A, Teli M, Ortalina A, et al. *Vertebroplasty and kyphoplasty: complementary techniques for the treatment of painful osteoporotic vertebral compression fractures: a prospective non-randomised study on 154 patients*. *Eur Spine J*. 2009;18(suppl 1): S. 95-S101.



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Hip: Osteotomy and arthroplasty

1. Osteotomies and Osteochondroplasties

1.1 Classic Femoral Osteotomy

Historically, the most frequent osteotomy around the hip joint was and perhaps still is the intertrochanteric osteotomy. Indications in earlier years included reorientation of the proximal femur in hips with abnormal morphologies like dysplasia, coxa antetorta or residual distorted deformities after childhood diseases (slipped capital femoral epiphyses, Perthes disease, etc.) [1, 2]. In addition intertrochanteric osteotomies have been used in osteoarthritic hips in order to relieve pain and/or to prevent further progression of disease [2]. The indication to perform intertrochanteric osteotomies in arthritic hip joints was based on improvement of load transmission in order to stimulate regeneration of the cartilage (Fig. 1) [2, 3].

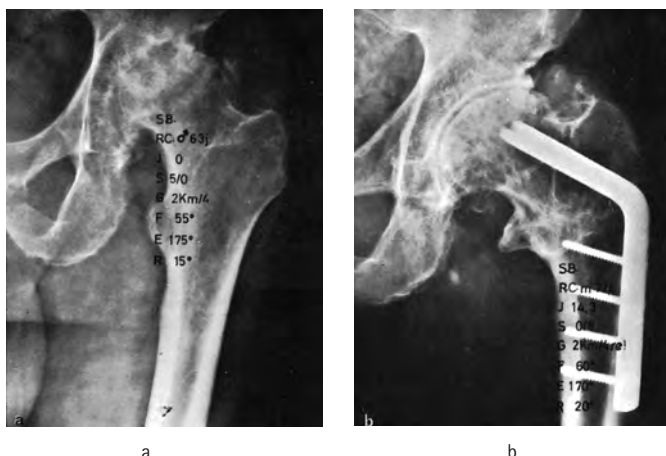


Fig. 1a shows the radiograph of a 63 year-old farmer with an arthritic left hip joint. The patient was treated with an intertrochanteric varus osteotomy.

Fig. 1b shows the radiographic result 14 years postoperatively.

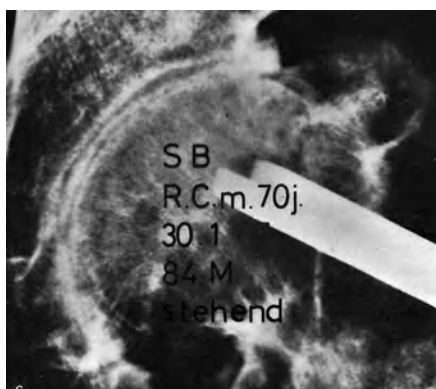


Fig. 1c The arthrogram demonstrates cartilage remodeling on the acetabular, as well as the femoral side at 7 years postoperatively. Image reprinted with permission from [2].

However long-term followup after 12- 15 years showed unsatisfactory results and/or functional deficits in close to 40% of 109 intertrochanteric osteotomies for osteoarthritis of the hip [2]. Thus, indications for intertrochanteric osteotomies in osteoarthritic hips have largely been abandoned today.

1.2 Osteochondroplasties around the hip

Nearly a decade ago the concept of femoroacetabular impingement (FAI) as a cause of early hip osteoarthritis was brought to attention by R. Ganz and his co-workers [4]. This concept implemented new surgical treatment options to delay or prevent early osteoarthritis in hips with abnormal morphologies. Pathomorphologies leading to FAI can be found on the femoral side, the acetabular side or on both sides. Accordingly FAI can be subdivided into

1. Cam-type impingement (femoral abnormality)
2. Pincer-type impingement (acetabular abnormality)
3. Combined impingement (abnormalities on both sides of the hip)

The treatment concept is based predominantly on:

1. Trimming of abnormal bone at the femoral and/or acetabular side and/or
2. Reorientation of malalignment or torsional deformities on both sides of the hip.

The „classical“ impingement is treated by trimming of an abnormal head-neck junction and / or trimming of a prominent acetabular rim. The head-neck trimming is called improvement of offset and is typically required at the antero-superior head-neck junction. On the acetabular side most common a localized prominent antero-superior rim or less frequently a circumferential prominent acetabular rim, e.g. in protrusio acetabuli are trimmed back. Labral refixation after acetabular rim trimming has shown to provide better results than labral resection [5, 6]. The goal of surgery is to prevent further cartilage damage by providing an impingement-free physiological range of motion. This goal can be reached with different surgical techniques, such as:

1. Open surgery via a safe surgical dislocation of the hip [7]
2. Combined minimal anterior approach and hip arthroscopy [8]
3. Hip arthroscopy (HAS) [9]

Treatment results will depend on surgeon's experience in analyzing the impingement conflict and detection of required zones for trimming as well as the surgeon's surgical ability especially to perform labral refixation during hip arthroscopy [10]. An overview of surgical short to midterm outcome with the different types of approaches is summarized in table 1.

Table 1 Selected studies are presented, comparing outcome after operative treatment of femoroacetabular impingement. Different surgical techniques ranging from open surgical dislocation, over mini-open approaches with arthroscopic assistance to pure arthroscopic procedures are summarized [8, 51–55].

Study (Year)	Surgical Technique	Number of Patients [hips]	Mean Patient Age [years]	Mean Followup	Scores Employed	Score Improvement at Last Followup	Revision Surgery (other than THA)	Failure (Conversion to THA)
Naal et al. (2012) [54]	Open	185 [233]	30 ± 9 (14–55)	60.7 ± 11.8 (24–120 month)	WOMAC HOS SF-12 UCLA	N.A.	18 (7.7%)	7 (3%)
Steppacher et al. (unpublished 2012)	Open	75 [97]	32 ± 8 (15 – 52)	6.0 ± 0.5 (5.0 – 7.1 years)	M.d'A. WOMAC SF-12 UCLA	1.9 points N.A. N.A. N.A.	7 (7.2%)	7 (7%)
Laude et al. (2009) [53]	Combined	97 [100]	33 (16– 56)	4.9 (2.4–8.7 years)	NAHS	29.1 points	16 (16%)	11 (11%)
Clohisy et al. (2010) [8]	Combined	35 [35]	34 (16–48)	2.2 (2–3 years)	mod. HHS NAHS UCLA	23.6 points 15.1 points 2.3 points	None	None
Byrd and Jones (2009) [51]	HAS	200 [207]	33 (w/o range)	16 (12–24 month)	mod. HHS	20 points	3 (1.4%)	1 (0.5%)
Horisberger et al. (2010) [52]	HAS	88 (105)	41 (17–66)	2.3 (1.3–4.1 years)	NAHS	27.8 points	None	9 (9%)

HAS = hip arthroscopy; THA = Total Hip Arthroplasty; WOMAC = Western Ontario and McMaster Universities Arthritis Index, HOS = Hip Outcome Score; NAHS = Nonarthritic Hip Score; M.d'A. = Merle d'Aubigne Score; mod. HHS = modified Harris Hip Score; UCLA = UCLA Activity Index

1.3 Osteotomies on both sides of the hip joint

Hip joints with substantial malalignment or torsional deformities may be better treated with realignment or reorientation of either side of the hip joint. Acetabular retroversion has been treated with an anteverting periacetabular osteotomy with good and excellent midterm results in 26 out of 29 hips [11, 12]. Coxa valga and antetortia can lead to posterior impingement which may be best treated with a corrective intertrochanteric osteotomy [13]. The approach for surgical dislocation of the hip can be expanded by an additional extended soft-tissue retinacular flap [14]. While protecting the supero-posterior retinaculum to the femoral head including the blood vessels to the head, the entire neck, trochanteric area and large areas of the femoral head can be exposed. This technique has amplified the potential for surgical corrections of the proximal femur including relative lengthening of the femoral neck, trimming and distalization of the greater trochanter, a true neck osteotomy and even a head reduction-plasty in grossly deformed femoral heads (Fig. 2).

Developmental dysplasia of the hip joint leads to mechanical overload due to a reduced load transmission area and /or to joint instability. Acetabular reorientation techniques have largely replaced augmentation techniques like a Chiari osteotomy [15] or shelf plasties [16] at least in adolescents and adults. Reorientation osteotomies include

1. Spherical or rotational osteotomies [17, 18]
2. Triple osteotomy [19]
3. Bernese Periacetabular Osteotomy [20]

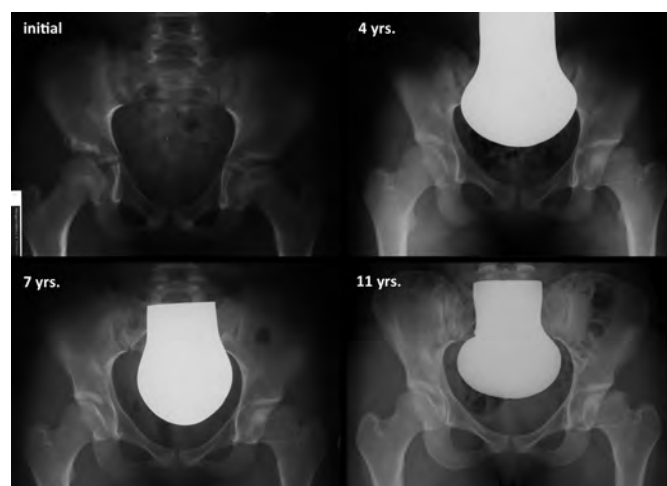


Fig. 2a A consecutive series of anteroposterior pelvis radiographs acquired over an 11-year period are shown. On initial presentation (upper left), this 10-year-old female patient presented with clinical symptoms and radiographic evidence of Perthes disease and concomitant marginal hip dysplasia on the left side. In the light of acceptable hip joint congruity, this patient was treated conservatively with physical therapy. While containment of the femoral head was initially maintained, it became apparent that the central head portion underwent consecutive collapse, leading to the incongruent left hip joint with a too large, subluxated head depicted in the lower right corner.



Fig. 2b Intraoperative images of the same patient introduced in figure 2a) are shown. At 22 years of age, a surgical hip dislocation with an extended retinacular soft-tissue flap was performed, allowing for inspection and remodeling of the deformed femoral head. In this case, the subsequent steps of a head reduction-plasty are illustrated from 1.-3. A wedge-shaped part of the femoral head is marked in a region not associated with the retinacular vessels. This wedge is subsequently excised and the femoral head reduced in shape in order to fit into the acetabulum.



Fig. 2c The postoperative radiographs are shown. Apart from the head reduction-plasty, the patient received a periacetabular osteotomy of the left hip joint. The combined femoral and acetabular interventions significantly increased the containment of the femoral head and the congruity of the articulation of the left hip joint.

The expected joint preservation after reorientation procedures is within the range of 80 – 90% at 10-year follow-up and between 60 – 70% at 20-year follow-up [21–25]. The expected joint preservation rises to 85 – 90% at 20-year follow-up after surgery in an ideal patient less than 30 years and without preoperative osteoarthritic radiographic signs. There is recent evidence that additional correction of a non-spherical head might further improve results in these patients (Fig. 3) [26]. Typically the success rates for joint preservation drop with patient's age between 35 to 40 years. Thereafter only well selected cases seem suitable for joint preservation surgery.

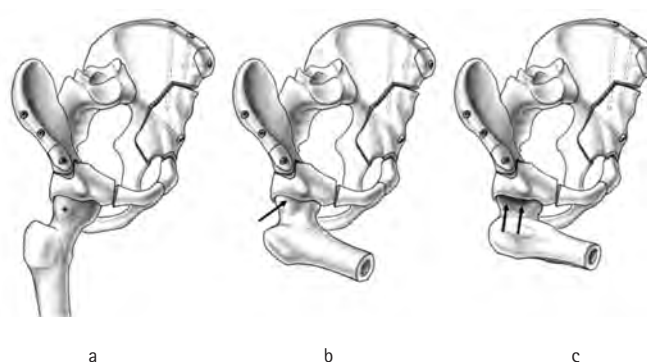


Fig. 3 The drawings depict a pelvis treated with bilateral periacetabular osteotomy for correction of acetabular dysplasia (a). With the acetabulum in a new, more anteriorly covered portion, it becomes apparent that a coexistent femoral cam-type deformity leads to an antero-superior impingement conflict between femoral head-neck junction acetabular rim (b). Hence, after acetabular reorientation, it is mandatory to evaluate for co-existent femoroacetabular cam-type impingement and restricted internal rotation. In the latter case, an additional femoral head-neck osteochondroplasty is required in order to allow for an impingement-free range of motion (c).

2. Hip Joint Replacement

2.1 Classical primary total hip arthroplasty

The total hip arthroplasty could be called the „second half of life period“ surgery for a painful hip. Survival rates generally have been better than expected in earlier years. The classic total hip arthroplasty (THA) is the low friction arthroplasty with cemented components and a metal on polyethylene bearing [27]. Survival rates in regard to aseptic loosening of cemented THAs in the very long-term follow-up over 30 years are very favorable in specialized centers and experienced hands [28, 29]. The corresponding figures for revision for aseptic loosening range from 3.2% to 4.5% for the cemented stem and from 8.1 to 8.5% for cemented cups [28, 29]. Although still in debate, longer term follow-up data from large arthroplasty registers show that revision rates for uncemented components in THA are approaching the results for cemented components specifically on the femoral side or may even perform better in younger patients [30, 31]. In the annual report 2010 of the Swedish arthroplasty register [32], implant survival in regard to cup wear and loosening rates range between 84 – 96% at 15 year follow-up. In general, cup survival was better with cemented implants in older patients and younger patients seemed to benefit from non-cemented cups. For the stems the annual report of the Swedish arthroplasty register showed survival rates between 95 and 97% at 15-year follow-up. Younger men under 50 years of age had lower stem revision rates with uncemented stems whereas older patients over 70 years of age seem to benefit from cemented stems [32].

2.2 Hard-on-hard bearings

Over time different joint bearing surfaces have been developed in order to reduce wear, osteolysis and implant loosening. One example is ceramic-on-ceramic bearings, which typically should help younger patients to reduce implant loosening by decreased wear [33, 34]. In -vitro-studies of reduced wear rates have shown to be reproducible in-vivo and mid- to long-term results and are comparable with metal-on -poly bearings. Implant survival rates range from 96% at 8 years to 85% at 20 years follow-up with ceramic-on-ceramic components with very low wear [33–35]. In opposite, modern metal-on-metal bearings specifically with larger femoral heads have failed to demonstrate an

advantage. Based on joint registries and meta-analyses larger femoral metal heads have a more than three-fold higher revision and failure rate [36, 37]. Reasons for failure are suboptimal positioning with higher edge loading and wear, increased local debris and subsequent adverse soft tissue reactions. In addition there is increasing concern about elevated metal ion serum concentrations [36, 37].

2.3 Resurfacing arthroplasty

Hip resurfacing arthroplasty can be considered a revival of a concept proposed and developed more than 30 years ago by Wagner [38]. The revival was based on the fact that new metal-on-metal bearings might solve the problems of the initial designs [38]. While the idea of bone preservation may be appealing, many advocates for revival of hip resurfacing arthroplasties wrongly advertised potential advantages of this type of joint replacement. The critical side in THAs is not the femoral bone, but the acetabular bone stock, which is not at all better preserved in resurfacing arthroplasties. Hip resurfacing arthroplasty (HRA) does not lead to a increased range of motion compared to standard THA with 32 mm heads and thus HRA as such can not fulfill the promise of increased physical or athletic activities [39, 40]. Adverse tissue reactions on larger metal-on-metal bearings and femoral neck fractures, virtually unknown in conventional THA with standard bearings, have led to a decrease of indications and number of HRAs. Increasing reports on failures rates have led to the recommendation that HRA is not used in women any more and should only be considered in selected young men, then using a construct with larger femoral heads [32, 41–43].

2.4 Surgical approaches

In the recent years a diversification in surgical approaches in regard to implantation of a THA has occurred. While the posterior approach is still largely used, there is an increasing trend to favor an antero-lateral or anterior approach over the standard lateral (Hardinge) approach [32]. The direct anterior [44] and antero-lateral modified Watson-Jones approaches [45] have also been called minimal invasive approaches. The basic advantage of these two latter approaches is that they use muscle intervals in order to reach the hip without the need for muscle detachment. Specifically the gluteus medius tendon can be preserved. Early weight bearing and rehabilitation may be facilitated which might lead to a decreased time to discharge and represent a secondary cost reduction. However, clinical studies did not prove a superior functional outcome 1 year after surgery [46–49]. In addition, repetition of learning curves and a somewhat limited exposure with minimal approaches may raise the complication rates with minimal invasive approaches [46, 49, 50]. The Swedish and Norwegian arthroplasty registers have independently found an increasing infection and dislocation rate after primary total hip replacements implanted in the recent years [32, 41]. Although not proven yet, suspicion arouse whether sub-optimally placed components or technical problems with the use of minimal invasive approaches may compromise longevity of the prosthetic construct in the long-term followup. Thus, it remains mandatory to stay with established principles for implant positioning and implant design for THA in order to keep this surgery one of the most successful interventions in humans. Meticulous and structured training programs for orthopedic surgeons are essential. Improvements on the field of THA have to be measured against the gold standard.

References

1. M.E. Müller, *DIE HÜFTNAHEN FEMUROSTEOTOMIEN. Unter Berücksichtigung der Form, Funktion und Beanspruchung des Hüftgelenkes.* 1957: Georg Thieme Verlag.
2. R. Schneider, *Die intratrochantäre Osteotomie bei Coxarthrose.* 1979, Berlin Heidelberg New York: Springer.
3. F. Pauwels, *Atlas zur Biomechanik der gesunden und kranken Hüfte.* 1973: Springer Verlag, New York.
4. R. Ganz, J. Parvizi, M. Beck, M. Leunig, H. Notzli, and K.A. Siebenrock, *Femoroacetabular impingement: a cause for osteoarthritis of the hip.* *Clin Orthop Relat Res*, 2003(417): p. 112–20.
5. N. Espinosa, D.A. Rothenfluh, M. Beck, R. Ganz, and M. Leunig, *Treatment of femoro-acetabular impingement: preliminary results of labral refixation.* *J Bone Joint Surg Am*, 2006. 88(5): p. 925–35.
6. C.M. Larson, M.R. Givens, and R.M. Stone, *Arthroscopic debridement versus refixation of the acetabular labrum associated with femoroacetabular impingement: mean 3.5-year follow-up.* *Am J Sports Med*, 2012. 40(5): p. 1015–21.
7. M. Lavigne, J. Parvizi, M. Beck, K.A. Siebenrock, R. Ganz, and M. Leunig, *Anterior femoroacetabular impingement: part I. Techniques of joint preserving surgery.* *Clin Orthop Relat Res*, 2004(418): p. 61–6.
8. J.C. Clohisy, L.P. Zebala, J.J. Nepple, and G. Pashos, *Combined hip arthroscopy and limited open osteochondroplasty for anterior femoroacetabular impingement.* *J Bone Joint Surg Am*, 2010. 92(8): p. 1697–706.
9. J.W. Byrd and K.S. Jones, *Arthroscopic management of femoroacetabular impingement.* *Instr Course Lect*, 2009. 58: p. 231–9.
10. J.C. Clohisy, L.C. St John, and A.L. Schutz, *Surgical treatment of femoroacetabular impingement: a systematic review of the literature.* *Clin Orthop Relat Res*, 2010. 468(2): p. 555–64.
11. C.L. Peters, L.A. Anderson, J.A. Erickson, A.E. Anderson, and J.A. Weiss, *An algorithmic approach to surgical decision making in acetabular retroversion.* *Orthopedics*, 2011. 34(1): p. 10.
12. K.A. Siebenrock, R. Schoeniger, and R. Ganz, *Anterior femoroacetabular impingement due to acetabular retroversion. Treatment with periacetabular osteotomy.* *J Bone Joint Surg Am*, 2003. 85-A(2): p. 278–86.
13. *Valgus Hip With High Antetorsion Causes Pain Through Posterior Extraarticular FAI* Siebenrock KA, Steppacher SD, Haefeli PC, Schwab JM, Tannast M *Clin Orthop Relat Res.* 2013 Mar 6. [Epub ahead of print]
14. R. Ganz, T.W. Huff, and M. Leunig, *Extended retinacular soft-tissue flap for intra-articular hip surgery: surgical technique, indications, and results of application.* *Instr Course Lect*, 2009. 58: p. 241–55.
15. K. Chiari, [Pelvic osteotomy in hip arthroplasty]. *Wien Med Wochenschr*, 1953. 103(38): p. 707–9.
16. M. Lance, *Consitution d'une butr e ost oplastique dans les luxations subluxations cong nitales de la hanche [in french].* *Presse Med*, 1925(33): p. 945–958.
17. S. Ninomiya and H. Tagawa, *Rotational acetabular osteotomy for the dysplastic hip.* *J Bone Joint Surg Am*, 1984. 66(3): p. 430–6.
18. H. Wagner, *Osteotomies for congenital hip dislocation.* In: *The hip Proceedings of the fourth open scientific meeting of the hip society.* 1976. St.Louis: CV Mosby.
19. D. Tonniss, K. Behrens, and F. Tscharni, *A modified technique of the triple pelvic osteotomy: early results.* *J Pediatr Orthop*, 1981. 1(3): p. 241–9.

20. R. Ganz, K. Klaue, T.S. Vinh, and J.W. Mast, A new periacetabular osteotomy for the treatment of hip dysplasias. Technique and preliminary results. *Clin Orthop Relat Res*, 1988(232): p. 26-36.
21. S.D. Steppacher, M. Tannast, R. Ganz, and K.A. Siebenrock, Mean 20-year followup of Bernese periacetabular osteotomy. *Clin Orthop Relat Res*, 2008. 466(7): p. 1633-44.
22. M. Kralj, B. Mavcic, V. Antolic, A. Iglic, and V. Kralj-Iglic, The Bernese periacetabular osteotomy: clinical, radiographic and mechanical 7-15-year follow-up of 26 hips. *Acta Orthop*, 2005. 76(6): p. 833-40.
23. X. Flecher, A. Casiraghi, J.M. Aubaniac, and J.N. Argenson, [Periacetabular osteotomy medium term survival in adult acetabular dysplasia]. *Rev Chir Orthop Reparatrice Appar Mot*, 2008. 94(4): p. 336-45.
24. L. Zagra, M. Corbella, and R. Giacometti Ceroni, Wagner's spherical periacetabular osteotomy: long term results. *Hip Int*, 2007. 17 Suppl 5: p. S65-71.
25. Y. Takatori, S. Ninomiya, S. Nakamura, S. Morimoto, T. Moro, I. Nagai, and A. Mabuchi, Long-term results of rotational acetabular osteotomy in patients with slight narrowing of the joint space on preoperative radiographic findings. *J Orthop Sci*, 2001. 6(2): p. 137-40.
26. C.E. Albers, S.D. Steppacher, R. Ganz, M. Tannast, and K.A. Siebenrock, Impingement Adversely Affects 10-year Survivorship After Periacetabular Osteotomy for DDH Albers CE, Steppacher SD, Ganz R, Tannast M, Siebenrock KAClin *Orthop Relat Res*. 2013 Jan 25. [Epub ahead of print]
27. J. Charnley, Total hip replacement by low-friction arthroplasty. *Clin Orthop Relat Res*, 1970. 72: p. 7-21.
28. B.M. Wroblewski, P.D. Siney, and P.A. Fleming, Charnley low-frictional torque arthroplasty: follow-up for 30 to 40 years. *J Bone Joint Surg Br*, 2009. 91(4): p. 447-50.
29. R.S. Ling, J. Charity, A.J. Lee, S.L. Whitehouse, A.J. Timperley, and G.A. Gie, The long-term results of the original Exeter polished cemented femoral component: a follow-up report. *J Arthroplasty*, 2009. 24(4): p. 511-7.
30. S. Morshed, K.J. Bozic, M.D. Ries, H. Malchau, and J.M. Colford, Jr., Comparison of cemented and uncemented fixation in total hip replacement: a meta-analysis. *Acta Orthop*, 2007. 78(3): p. 315-26.
31. N.P. Hailer, G. Garellick, and J. Karrholm, Uncemented and cemented primary total hip arthroplasty in the Swedish Hip Arthroplasty Register. *Acta Orthop*, 2010. 81(1): p. 34-41.
32. G. Garrellick, J. Kärrholm, C. Rogmark, and P. Herberts, Swedish Hip Arthroplasty Register - Annual Report 2010. 2010.
33. M. Hamadouche, P. Boutin, J. Daussange, M.E. Bolander, and L. Sedel, Alumina-on-alumina total hip arthroplasty: a minimum 18.5-year follow-up study. *J Bone Joint Surg Am*, 2002. 84-A(1): p. 69-77.
34. S.B. Murphy, T.M. Ecker, and M. Tannast, Two- to 9-year clinical results of alumina ceramic-on-ceramic THA. *Clin Orthop Relat Res*, 2006. 453: p. 97-102.
35. M.G. Zywiell, S.A. Sayeed, A.J. Johnson, T.P. Schmalzried, and M.A. Mont, Survival of hard-on-hard bearings in total hip arthroplasty: a systematic review. *Clin Orthop Relat Res*, 2011. 469(6): p. 1536-46.
36. P.B. Voleti, K.D. Baldwin, and G.C. Lee, Metal-on-Metal vs Conventional Total Hip Arthroplasty: A Systematic Review and Meta-Analysis of Randomized Controlled Trials. *J Arthroplasty*, 2012. 27(10): p. 1844-9.
37. K.J. Bozic, J. Browne, C.J. Dangles, P.A. Manner, A.J. Yates, Jr., K.L. Weber, K.M. Boyer, P. Zemaitis, A. Woznica, C.M. Turkelson, and J.L. Wies, Modern metal-on-metal hip implants. *J Am Acad Orthop Surg*, 2012. 20(6): p. 402-6.
38. H. Wagner, Surface replacement arthroplasty of the hip. *Clin Orthop Relat Res*, 1978(134): p. 102-30.
39. B.C. Bengs, S.N. Sangiorgio, and E. Ebramzadeh, Less range of motion with resurfacing arthroplasty than with total hip arthroplasty: in vitro examination of 8 designs. *Acta Orthop*, 2008. 79(6): p. 755-62.
40. M. Lavigne, M. Ganapathi, S. Mottard, J. Girard, and P.A. Vendittoli, Range of motion of large head total hip arthroplasty is greater than 28 mm total hip arthroplasty or hip resurfacing. *Clin Biomech (Bristol, Avon)*, 2011. 26(3): p. 267-73.
41. H. Bergen, Centre of Excellence for Joint Replacement - The Norwegian Arthroplasty Register - Report June 2010. 2010.
42. A.D. Carrothers, R.E. Gilbert, A. Jaiswal, and J.B. Richardson, Birmingham hip resurfacing: the prevalence of failure. *J Bone Joint Surg Br*, 2010. 92(10): p. 1344-50.
43. A.J. Smith, P. Dieppe, P.W. Howard, and A.W. Blom, Failure rates of metal-on-metal hip resurfacings: analysis of data from the National Joint Registry for England and Wales. *Lancet*, 2012.
44. M.N. Smith-Petersen, Approach to and exposure of the hip joint for mold arthroplasty. *J Bone Joint Surg Am*, 1949. 31A(1): p. 40-6.
45. K.C. Bertin and H. Rottinger, Anterolateral mini-incision hip replacement surgery: a modified Watson-Jones approach. *Clin Orthop Relat Res*, 2004(429): p. 248-55.
46. J.H. Goosen, B.J. Kollen, R.M. Castelein, B.M. Kuipers, and C.C. Verheyen, Minimally invasive versus classic procedures in total hip arthroplasty: a double-blind randomized controlled trial. *Clin Orthop Relat Res*, 2011. 469(1): p. 200-8.
47. J. Jung, K. Anagnostakos, and D. Kohn, Klinische Ergebnisse nach minimal-invasiver Hüftendoprothetik. *Orthopade*, 2011(41): p. 399-406.
48. S.B. Murphy, T.M. Ecker, and M. Tannast, THA performed using conventional and navigated tissue-preserving techniques. *Clin Orthop Relat Res*, 2006. 453: p. 160-7.
49. T.O. Smith, V. Blake, and C.B. Hing, Minimally invasive versus conventional exposure for total hip arthroplasty: a systematic review and meta-analysis of clinical and radiological outcomes. *Int Orthop*, 2011. 35(2): p. 173-84.
50. T. Cheng, J.G. Feng, T. Liu, and X.L. Zhang, Minimally invasive total hip arthroplasty: a systematic review. *Int Orthop*, 2009. 33(6): p. 1473-81.
51. J.W. Byrd and K.S. Jones, Arthroscopic femoroplasty in the management of cam-type femoroacetabular impingement. *Clin Orthop Relat Res*, 2009. 467(3): p. 739-46.
52. M. Horisberger, A. Brunner, and R.F. Herzog, Arthroscopic treatment of femoroacetabular impingement of the hip: a new technique to access the joint. *Clin Orthop Relat Res*, 2010. 468(1): p. 182-90.
53. F. Laude and E. Soriali, [Treatment of FAI via a minimally invasive ventral approach with arthroscopic assistance. Technique and midterm results]. *Orthopade*, 2009. 38(5): p. 419-28.
54. F.D. Naal, H.H. Miozzari, M. Schar, T. Hesper, and H.P. Notzli, Midterm results of surgical hip dislocation for the treatment of femoroacetabular impingement. *Am J Sports Med*, 2012. 40(7): p. 1501-10.



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Knee: Osteotomy and arthroplasty

Symptomatic knee osteoarthritis (OA) is highly prevalent among people aged 50 years and over with a consistently higher prevalence among women compared with men. The typical symptoms include effusion, joint pain and stiffness leading to loss of joint function. Patient history, physical examination, radiological and laboratory findings are the diagnostic criteria for knee OA. If, after a well managed initial conservative treatment, the symptoms are not relieved, surgery should be considered and consists of many options such as: arthroscopic debridement, cartilage repair, osteotomies around the knee and unicompartmental knee arthroplasty (UKA) or total knee arthroplasty (TKA).

1. Osteotomies around the knee

Osteotomies around the knee are standard well-documented methods for the treatment of unicompartmental knee osteoarthritis associated with malalignment of the lower limb. These procedures belong to conservative surgery. The aim is to unload an altered compartment of the knee and transfer the peak load by slightly overcorrecting into a valgus or varus axis in order to slow the degenerative process, reduce pain and delay joint replacement. Osteotomies may be done either on the distal femur or the proximal tibia, depending on the deformity of the lower limb. They have gained in popularity in the 1960 and consisted classically of a tibial valgisation closing wedge type including an osteotomy of the fibula as described by Coventry. Later on, these procedures lost importance due to the success of knee arthroplasty. Also, they were considered as demanding procedures associated with significant complications. Still, the development during the last 10 years of new fixation devices (plates with angular stability) has brought osteotomies again into light, especially for younger and active patients.

1.1 Patient selection

The outcome of such procedure is among others depending on proper patient selection. The stage of OA shall be precisely addressed and if there already is a 4th degree (Outerbridge) wear, only limited pain relief shall be expected. The range of motion is evaluated and at least 120° of flexion and no more than 20° of extension deficit are mandatory. Instability of the knee joint is not an absolute contraindication because tibial slope correction is used to address ACL or PCL deficient knees. The patellofemoral joint may show signs of degenerative changes but shall be totally asymptomatic. Considering the age, >65 years is a relative contraindication but the activity and biologic age must also be considered. A BMI under 30 gives the best results. Also, the patient shall not suffer from inflammatory diseases such as rheumatoid arthritis. Ideally, before the osteotomy it is interesting to confirm the clinical and radiological findings by an arthroscopy that can be done during the same procedure. This will also ensure that the unaffected compartment is healthy.

1.2 Preoperative planning

The key for a successful osteotomy is a correct pre-operative planning; therefore, it is important to understand the normal lower limb anatomy and its physiological angles and axes. The physiological mechanical axis of the leg, also called "Mikulicz line", runs from the center of the femoral head to the center of the ankle joint and crosses the knee joint about 4 (± 2) mm medial to its center. This point is used to quantify the mechanical axis deviation (MAD) of the lower limb mechanical axis. It may be measured in millimetres from the center of the knee or like Fujisawa described, as a percentage of a medial or lateral compartment (Fig. 1d). The anatomical axes of the femur and tibia correspond to the diaphyseal midline of these bones. The mechanical axis of the femur, running from its head to the center of the knee therefore forms an angle of $6 \pm 1^\circ$ with the anatomical axis (Fig. 1 a-c). The tibia has a mechanical axis nearly identical to the anatomical axis.

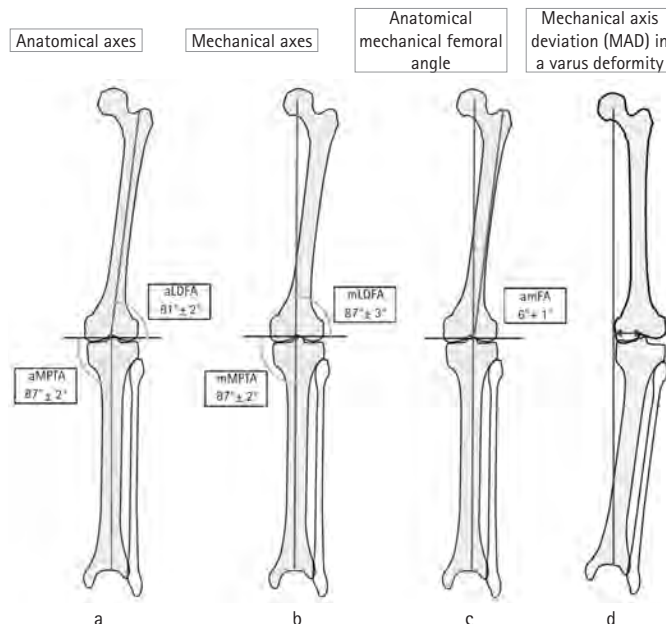


Fig. 1 (a-c) Anatomical and mechanical angle values of the femur and the tibia. d measurement of the MAD (mm) from the knee centre in a varus deviation.

aMPtA: anatomical mechanical proximal tibial angle. aLDFa: anatomical mechanical distal femoral angle. mMPtA: mechanical medial proximal tibial angle. mLDFa: mechanical lateral distal femoral angle. amFA: anatomical – mechanical femoral angle.

1.3 Measurement and localisation of the axial deformity

The lower limb deformities occur most often in the frontal plane and are described as varus or valgus deviations. This malalignment is defined as a significant deviation from the mechanical axis, (MAD: mechanical axis deviation). It is diagnosed as a varus when the weight bearing axis of

the lower runs 15mm medial to the center of the knee and valgus when it runs 10mm lateral to the center. The measures of the anatomical and mechanical angles of the femur and the tibia are then necessary to point out the source of the deviation because axial deviations may exist due to isolated of either femur or tibia deformation, or due to a combination of both (Fig. 2 a-b). These more complex situations often need double osteotomies around the knee.

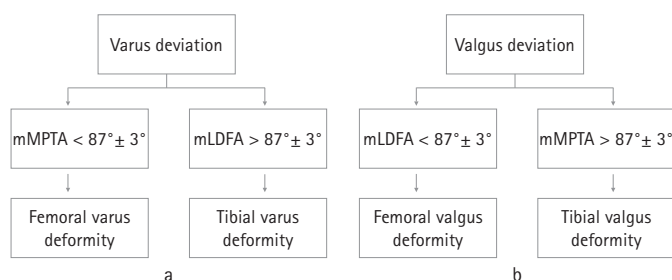


Fig. 2 (a-b) Femoral and tibial analysis of varus and valgus deformity

1.4 Level of the osteotomy

On optimal correction is obtained when the osteotomy is performed at the apex of the deformity and depends of the preoperative planning. It may be done either on the distal femur or proximal tibia, or both. The osteotomy line shall stay in the metaphyseal bone because of better healing properties. The open- wedge osteotomies are generally easier and more precise to achieve than closing-wedge and in most of cases there is no need for bone grafting if an implant with angular stability is used.

1.5 Correction

The first goal of an osteotomy is to achieve a correction in the frontal plane to unload an altered knee compartment as described above. However, it may also influence the sagittal and transverse planes. A correction of the sagittal plane is used in cases of anterior or posterior knee instability by varying the tibial slope. In case of a chronic ACL insufficiency, the tibial slope shall be decreased up to 5° (extension osteotomy) in order to improve the sagittal instability and gain some extension. In posterior or posterolateral knee instabilities, the slope shall be increased up to 12° (flexion osteotomy) to reduce the posterior subluxation of the tibia and to eliminate the hyperextension of the knee. Corrections in the transverse plane are rare and are used to correct rotational deformities. As the patellar tracking may be significantly altered, the patellofemoral alignment shall be analysed and understood preoperatively.

1.6 Preoperative planning

Several methods for osteotomy planning have been described in the literature. Loebenhoffer and al. have developed an accurate technique to define the correction angle based on the study by Fujisawa and the planning method described by Miniaci. In facts, a varus malalignment is brought to a slight overcorrection, between 10 and 35% in the lateral compartment depending on the severity of the medial cartilage loss. On the other hand, a valgus deformity is corrected up to neutral. As an example, for a high tibial valgisation osteotomy, first trace the Mikulicz line and then draw the new weight-bearing line from the centre of the hip and passing through the lateral compartment of the knee at the chosen level. Define the hinge of osteotomy, one centimetre from the lateral cortex of the tibia and connect it distally to the old and new centre of the ankle. These two lines form the correction angle (Fig. 3 a-c).

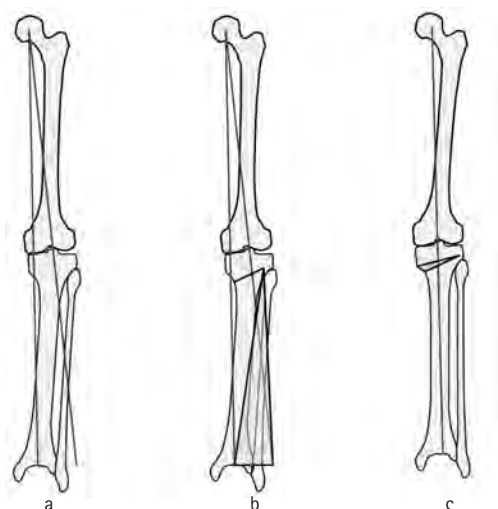


Fig. 3 (a-c) Determination of the correction angle in high tibial osteotomy opening wedge (Miniaci).

1.7 Surgery and fixation

As an example for a high-tibial open-wedge valgisation osteotomy, the surgical procedure will start with a knee arthroscopy to evaluate and document the amount of cartilage in the lateral compartment (Fig. 4 a-b). An adjuvant treatment such as microfractures, osteochondral autograft transfers, matrix-associated chondrocyte implantations or others may be done during the same procedure.

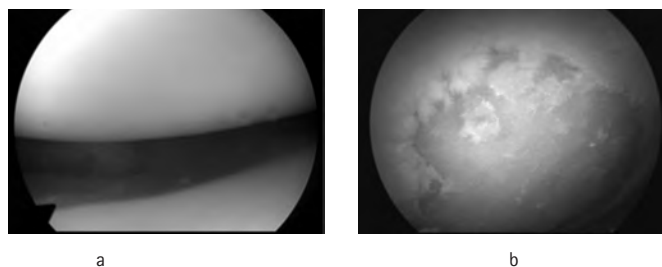


Fig. 4 Arthroscopic views of a right knee.

- a lateral compartment.
- b medial compartment with cartilage wear

A longitudinal skin incision to expose the medial part of the tibial metaphysis is preferred to oblique approaches because it may be used again for future surgeries (total knee arthroplasty).

The pes anserinus is exposed and two guide wires are placed along the virtual osteotomy line. The proper position of the wires is checked with fluoroscopy in the frontal and sagittal planes. The medial and posterior tibial cortices are cut with a saw blade along the wires. The anterior ascending cut is then performed to free the anterior tibial tubercle.

The osteotomy is progressively and carefully completed with chisels. Take care to stop the osteotomy about 1 cm from the lateral cortex to preserve a lateral bony hinge. Once the gap is completed, a spreader can be introduced to progressively open the osteotomy line. This opening takes several minutes to prevent intra-articulaire fractures of the lateral tibial plateau.

Due to the superficial medial collateral ligament, the osteotomy tends to open more in its anterior portion. Therefore it is important to release enough the superficial fibres to obtain a symmetrical medial opening. At this stage, anterior or posterior knee instabilities can be treated by varying the tibial slope.

Once the planned correction is obtained, a definitive fixation is made by

the insertion of a plate that is fixed with angular stability screws (Fig. 5).



Fig. 5 Post-operative views of right knee after high-tibial opening-wedge valgisation osteotomy

2. Unicompartmental knee arthroplasty (UKA)

When describing the anatomy of the knee, three separate anatomic compartments are mentioned; medial, lateral and patellofemoral. Each may be individually considered in terms of replacement arthroplasty. For a successful UKA, the patient selection plays an important role. The cruciate ligaments as well as the remaining two compartments must be well preserved in order to allow proper knee kinematics. A preoperatively correctable varus or valgus deformity to neutral alignment, a flexion contracture less than 10° and a minimum of 90° of flexion are mandatory. A fixed deformity will not be adequately balanced during surgery so that the implant will be overstressed and will likely fail. Also, there shall not be a collapse of the opposite compartment on stress radiographs and the patient should not suffer from an inflammatory disease. UKA is contraindicated in patient with high demand or labourer as well as those in overweight (>90kg). The most commonly used UKA involves the medial compartment and replaces both the femoral and tibial surfaces (Fig. 6).



Fig. 6 Medial UKA

The main advantage of the UKA is that it is a less aggressive surgery where the extensor mechanism is not damaged, thus allowing a quicker recovery. Also, it preserves the bone stock and normal knee kinematics for a more physiological function. The outcome for UKA is variable and ranges from 80.2 to 98% in terms of 10-years survival. Still, UKA has a significantly poorer long-term survival than total knee arthroplasty.

Isolated patellofemoral OA occurs in about 9% of patients over 40 years old in is predominant in females. Underlying causes include prior patellar fractures, patellar instability (patellar maltracking, trochlear dysplasia) and ancient surgery. The number of patellofemoral arthroplasties is rising but remains low because such arthroplasty often leads to failure

and the results are frustrating. Therefore, TKA should be considered as standard also for patellofemoral OA, especially for elderly patients.

3. Total knee arthroplasty

Total joint arthroplasty is a safe, cost-effective procedure for the management of advanced stage knee OA and results in a significant improvement of life quality. Prosthetic survival now approaches 90% at 15 years in the elderly but sinks to about 76% at 10 years for the younger population. The main complications after a TKA are the loosening of components, femoropatellar pain, stiffness and infection. In order to improve the outcome and lower the rate of unsatisfactory results several options have been explored these last years and include the use of computer-assisted surgery (CAS), minimally invasive surgery (MIS), patient specific cutting bone blocs, improvement of the design of implants and fixation of implants. Still, for a successful outcome, restoration of the mechanical alignment, preservation of the joint line, soft tissue balancing and femoral rotation remain essential. Basically, there are three main designs of total knee prostheses; unconstrained, semi-constrained and constrained hinged. In the unconstrained category, two different types are used, the posterior cruciate retaining and the posterior cruciate substituting (also called posterior stabilized) implants. The described advantage of posterior cruciate retaining implants is that as the posterior cruciate ligament gets taught in flexion it prevents an anterior dislocation of the femur on the tibia. Also, the femoral rollback is reproduced during the flexion of the knee and mimics a more physiological function. However, this is more a slide and roll movement which may create high stresses on the polyethylene (PE). Posterior cruciate substituting implants combine a cam situated between the condyles and a tibial post in the centre of the tibial PE. As, the knee flexes, the femoral cam will engage against the tibial post and thus the femur will not be able to translate anteriorly, providing stability of the knee joint. These implants are recommended for patients with previous patellectomy, those suffering from inflammatory diseases, having a severe fixed deformity or presenting a prior trauma with PCL rupture. As already mentioned, a good preoperative planning and clinical evaluation are predictors of the clinical outcome of a TKA. Preoperative radiographs are used to identify the correction needed in alignment and points out the bony defects that will need bone grafting or augmentation (Fig. 7, Table 1).

Table 1

Preoperative X-Rays
- Standing full-length AP view form hip to ankle
- Standard AP and side view of the knee
- Standing AP (Rosenberg's) view in 45° of flexion
- Femoropatellar (Merchant's) view
- Varus / valgus stress views (optional)



Fig. 7 (a-c) a Standing antero posterior view
b Frontal and lateral knee views
c Patellofemoral view

3.1 Mechanical alignment

It is important to clearly identify the mechanical and anatomical axes of the femur. The angle they form, called the valgus cut angle, allows a perpendicular distal femoral cut to the mechanical axis. In that situation, the femoral component will point toward the center of the femoral head and allows an optimal load share through the medial and lateral compartment. In most cases, this angle measures 5°–7° (Fig. 8a). On the tibial side, the mechanical and anatomical axes are also identified and in most of the cases are the same. However, the axes may be divergent like in congenital deformities, post-traumatic conditions or after prior surgery such as closing wedge osteotomies. The aim is to have a proximal tibial cut perpendicular to the mechanical axis so that the lower limb stresses run through the center of the tibial plateau

3.2 Preservation of the joint line

The goal is to remove sufficient amount of bone from the femur and the tibia so that the prosthesis when in place will re-create the original thickness of cartilage and bone. Also, the height of the joint line has to be respected in order to keep the patella in a proper position. Cutting too much from the distal femur may lead to patella baja what is poorly tolerated. In severe deformities, there is frequently a bone defect that has to be identified and restored. Bony defects of less than 1cm may be filled with cement whereas larger defects need metallic augmentation.

3.3 Soft tissue releases

It is probably the most fundamental step in TKA. During the degenerative process, ligaments and soft tissues will become contracted on the concave side of the deformity and stretched to lose on the convex side (Fig. 8 b-c). For proper knee function these structures need to be released and balanced in the frontal and sagittal planes. For example, in case of a varus deformity, the medial side will be concave and require a release. The release shall be progressive until the initial deformity is corrected to the neutral axis in the frontal plane. In the frontal plane, the anatomical structures to be released in a varus (Table 2) or valgus (Table 3) condition are listed below.

Table 2 Varus Deformity

MEDIAL RELEASE
- Osteophytes
- PCL (if not PCL not retaining)
- MCL deep portion
- Posteromedial corner
- Semi- membranous
- Pes anserinus

Table 3 Valgus Deformity

LATERAL RELEASE
- Osteophytes
- PCL
- Lateral capsule
- Posterolateral corner
- Ilio – tibial band from Gerdi
- Lateral condyle osteotomy

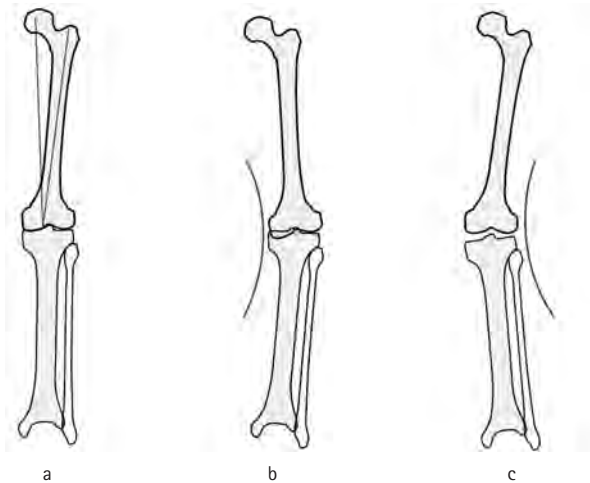


Fig. 8 (a-c) a Measure of the femoral valgus cut angle
b Medial contracted structures in varus deformity
c Lateral contracted structures in valgus deformity

When addressing the sagittal plane, the surgeon shall keep in mind that the physiological knee presents two curvatures; one for the patellofemoral articulation and one for the weight bearing portion of the knee. Therefore, to achieve a correct balancing in flexion and extension, it is necessary to release not only the soft tissue but also sometimes to add some amount of bone resection. The knee will be well balanced in the sagittal plane if the tibial insert remains stable during the full range of motion. As a general rule, if the gap problem is symmetric the tibia needs to be adjusted whereas if the gap problem is asymmetric the femur needs an adjustment (Table 4).

Table 4 Sagittal Plane Balancing

Situation	Problem	Solution
Tight in flexion and extension	Symmetric gap	Cut more tibia
Loose in flexion and extension	Symmetric gap	1. Thicker insert 2. Metallic tibia augmentation
Tight in extension / Good in flexion	Asymmetrical gap	1. Release posterior capsule 2. Cut more distal femur
Good in extension / Tight in flexion	Asymmetrical gap	1. Resect PCL if not done 2. Decrease size of the femoral component 3. Check tibial slope
Good in extension / Loose in flexion	Asymmetrical gap	Increase size of the femoral component (posterior metallic augmentation)
Loose in extension / Good in flexion	Asymmetrical gap	Distal femoral augmentation

3.4 Patellofemoral alignment

To prevent femoropatellar maltracking there are some situations to avoid. For instance, internal rotation of the femoral component should be avoided. It shall be placed in a slight external rotation. This is because the tibia presents anatomically a light varus of about 3° and as the cut is made perpendicular to the tibial axis, the femoral component has to be externally rotated to create a symmetric flexion gap. Two methods are used to get a correct rotation: the flexion gap balancing technique and the measured resection technique. The first uses the tensioning of the collateral ligaments in 90° of flexion to rotate the femur in the proper position. The latter uses bony landmarks (3° to 5° of the posterior condyles line) to get the proper femoral position (Fig. 9 a-b).

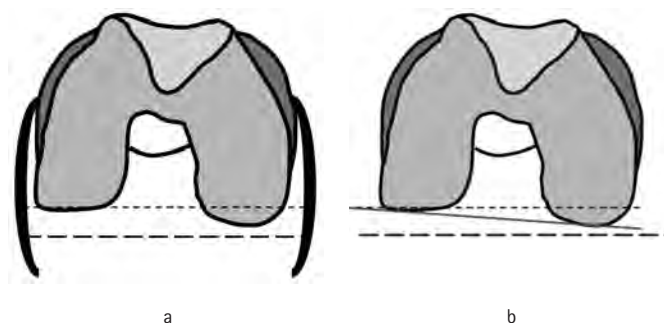


Fig. 9 (a-b) Optimal rotational positioning of femoral implant

----- Proximal tibial cut

----- Posterior femoral cut

a Posterior cut using a tensioning device

b Posterior cut about 3° to the posterior condyle line

On the tibial side, internal rotation of the component must be avoided and its center has to point to the medial third of the anterior tibial tubercle. If resurfacing the patella, the patellar dome shall be centred or even better, slightly medial. If necessary, a release of the femoropatellar lateral retinaculum is done from the articular side.

3.5 Implant fixation

Cemented fixation of TKA is a standard procedure with good long term results. It is also less technically challenging because the bone cuts do not need to fit perfectly to the prosthesis and the cement may fill the defects up to 1cm. None cemented implants have the advantage to lower the operation time but non-cemented tibial components have shown higher loosening rates so that only femoral components should be non cemented.

Due to the development of new implants and techniques, the outcome and function of TKA have improved. Still, for a successful outcome a well-balanced implant with a good patellofemoral tracking are essential (Fig. 10).



Fig. 10 Total knee arthroplasty

4. Conclusion

The choice of a surgical option and the patient selection is the most challenging part in the treatment of osteoarthritis of the knee. It is the surgeon's duty to correctly analyse the stage of osteoarthritis, the ligamentous status, the type of deformity and reducibility, the age, the range of motion and the expectations of the patient before proposing a surgery. There is a place for osteotomies around the knee, not only for monocompartmental osteoarthritis but also to address specific knee instability or to protect an ACL reconstruction in younger patients. Unicompartmental knee arthroplasties give also good results but are to be considered as resurfacing surgery and need an optimal comprehension of the lower limb deformity and clinical status. Total knee arthroplasty remains the gold standard for the definite treatment of knee osteoarthritis. Table 5 lists the ideal patient for each type of surgery.

Table 5

Osteotomies	UKA	TKA
55 to 70 years (biological age)	Older than 55 years	Older than 70 years
Correct extraarticular deformity	No influence on extraarticular deformity	May have fixed axis deviation
Monocompartmental osteoarthritis	Monocompartmental osteoarthritis	Generalised osteoarthritis
Complete range of motion	Complete range of motion	May have flexion or extension deficit
No inflammatory disease	No inflammatory disease	May have inflammatory disease
May have ACL / PCL deficiency	Intact ACL/PCL	May have ACL / PCL deficiency

References

- Lobenhoffer P, van Heerwaarden J, Staubli A, Jakob RP. Osteotomies Around the Knee. Thieme; 2008.
- Miller M. Review of Orthopaedics. 5th edition, Saunders; 2008.
- Jackson JP. Osteotomy for osteoarthritis of the knee. Proceedings of the Sheffield Regional Orthopaedic Club. The Journal of Bone and Joint Surgery. 1958;40(4):p. 826.
- Coventry MB. Osteotomy of the upper portion of the tibia for degenerative arthritis of the knee. A preliminary report. The Journal of Bone and Joint Surgery. 1965;47:984–990.
- Lobenhoffer P, Agneskirchner JD. Improvements in surgical technique of valgus high tibial osteotomy. Knee Surgery, Sports Traumatology, Arthroscopy. 2003;11(3):132–138.
- Staubli AE, De Simoni C, Babst R, Lobenhoffer P. TomoFix: a new LCP-concept for open wedge osteotomy of the medial proximal tibia—early results in 92 cases. Injury. 2003;34(supplement 2):SB55–SB62.
- Maquet P. Valgus osteotomy for osteoarthritis of the knee. Clinical Orthopaedics and Related Research. 1976;120:143–148.
- Fujisawa Y, Masuhara K, Shiomi S. The effect of high tibial osteotomy on osteoarthritis of the knee. An arthroscopic study of 54 knee joints. Orthopedic Clinics of North America. 1979;10(3):585–608.
- Paley D, Pfeil J. Principles of deformity correction around the knee. Orthopade. 2000;29(1):18–38.
- Insall JN, Joseph DM, Msika C. High tibial osteotomy for varus gonarthrosis: a long-term follow-up study. Journal of Bone and Joint Surgery A. 1984;66(7):1040–1048.
- Murray DW. Unicompartamental knee replacement: now or never? Orthopedics. 2000;23(9):979–980.
- Moller JT, Weeth RE, Keller JO, Nielsen S. Unicompartamental arthroplasty of the knee. Cadaver study of the importance of the anterior cruciate ligament. Acta Orthopaedica Scandinavica. 1985;56(2):120–123.
- Borus T, Thornhill T. Unicompartamental knee arthroplasty. Journal of the American Academy of Orthopaedic Surgeons. 2008;16(1):9–18.
- Sah AP, Scott RD. Lateral unicompartamental knee arthroplasty through a medial approach: study with an average five-year follow-up. Journal of Bone and Joint Surgery A. 2007;89(9):1948–1954.
- Ackroyd CE, Newman JH, Evans R, Edridge JDJ, Joslin CC. The avon patellofemoral arthroplasty: five-year survivorship and functional results. Journal of Bone and Joint Surgery B. 2007;89(3):310–315.
- Cartier P, Sanouiller JL, Khefacha A. Long-term results with the first patellofemoral prosthesis. Clinical Orthopaedics and Related Research. 2005;436:47–54.
- Keating EM, Meding JB, Faris PM, Ritter MA. Long-term followup of nonmodular total knee replacements. Clinical Orthopaedics and Related Research. 2002;404:34–39.
- Lundblad H, Kreicbergs A, Jansson KÅ. Prediction of persistent pain after total knee replacement for osteoarthritis. Journal of Bone and Joint Surgery B. 2008;90(2):166–171.
- O. Rönn K, Reischl N, Gautier E, Jacobi M. Current surgical treatment of knee osteoarthritis. Arthritis; 2011:454873.



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Ankle arthrodesis, arthroplasty, hallux valgus & flat foot

Preface: Reconstructive Foot and Ankle Surgery

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Foot and ankle disorders are a common and well recognized problem in the Health Care. However, only limited number of studies is existing addressing epidemiology of foot and ankle disorders [2]. This pathologic entity seems to be more frequent in the industrial countries and may be partially related to shoe wearing, because foot and ankle problems are rare in those populations who do not wear shoes [6,15]. In contrast to these findings, another study has revealed that 80% of 356 healthy women with an age ranged between 20 and 60 years have reported to have pain while wearing shoes [8]. More than 75% of all persons had substantial foot deformities and in 88% the used shoes were on average 1.2 cm smaller in width than their feet [8].

An epidemiologic study has been performed in the Italian town of Dicomano including 459 persons, 73% of whom were 65 years old or older [3]. Females were more often affected by foot and ankle disorders. The foot and ankle pain was indicated in 22% of all cases. The most common pathological findings were calluses and corns (68%), hypertrophic nails (30%), hallux deformities (21%) and absence of arterial pulses in affected foot and ankles (16%) [3]. The American National Estimate showed that the hallux deformity with painful bunions is a common problem in females with 60% of women aged 65 years and older [13]. However, also more than 40% of older men were affected by the same problem [13]. Women's Health and Aging Study [11] has shown that 32% of 990 disabled women had foot and ankle pain classified between moderate and severe. The most common foot and ankle disorders in this patient cohort were painful bunions and hammer toes with 70% and 50%, respectively. The study findings clearly suggest that painful foot and ankle problems may play an important role in general disability in older women [11]. Dunn et al [7] addressed the prevalence of foot and ankle problems in a multiethnic community sample including 784 persons aged 65 or more years. The five most common foot and ankle problems were toenail disorders (75%), lesser toe deformities (60%), corns and calluses (58%), bunions (37%), and signs of fungal infection, cracks/fissures, or maceration between toes (36%). In total, almost 15% had substantial foot and ankle pain on most days in the past 4 weeks [7]. Hill et al¹⁰ performed the North West Adelaide Health Study – a representative longitudinal cohort study of a total of 4,060 persons. Overall, almost one of five persons indicated that they had some foot and ankle problems including pain, aching, or stiffness. Following factors have been identified as independent risk factors for development of foot and ankle problems: female gender, higher age (50

years and older), and obesity [10]. Menz et al [12] used data from the Consultations in Primary Care Archive to identify the characteristics of primary care consultations for musculoskeletal foot and ankle problems in the UK. Overall, 8% of all 55,033 musculoskeletal consultations were related to foot and ankle problems [12].

While tumors of the foot and ankle are rare,[1, 4, 14] the degenerative conditions are the most common problem in patients with foot and ankle disorders. Approximately 1% of the entire world's adult population is affected by ankle osteoarthritis [9]. Ankle osteoarthritis is definitely less common than osteoarthritis of the other major joint of the lower extremity – knee or hip joint. However, the clinical importance of ankle osteoarthritis should not be underestimated. Glazebrook et al [9] have demonstrated that the patients with end-stage ankle osteoarthritis have comparable mental and physical disability as patients with end-stage hip osteoarthritis.

The economic burden of foot and ankle disorders is enormous. Only foot problems caused by ill-fitting shoes are responsible for costs of 1.5 billion \$ including annual direct cost of surgery and postoperative care for foot and ankle deformities [5]. Additional indirect costs were estimated as further 1.5 billion \$ [5].

Foot and ankle disorders may be classified due to their localization: hindfoot, midfoot, and forefoot (Fig. 1). The aim of this book chapter was to highlight the most common foot and ankle disorders and their treatment options, including ankle osteoarthritis, ankle arthrodesis, total ankle arthroplasty, flat foot deformity, and hallux valgus deformity.



Fig. 1 Classification of foot and ankle disorders depending on its localization: hindfoot, midfoot, and forefoot.

References

1. Azevedo CP, Casanova JM, Guerra MG, Santos AL, Portela MI, Tavares PF. Tumors of the Foot and Ankle: A Single-institution Experience.

- J Foot Ankle Surg* 2013.
- Balint GP, Korda J, Hangody L, Balint PV. Regional musculoskeletal conditions: foot and ankle disorders. *Best Pract Res Clin Rheumatol* 2003; 17(1):87-111.
 - Benvenuti F, Ferrucci L, Guralnik JM, Gangemi S, Baroni A. Foot pain and disability in older persons: an epidemiologic survey. *J Am Geriatr Soc* 1995; 43(5):479-484.
 - Chou LB, Ho YY, Malawer MM. Tumors of the foot and ankle: experience with 153 cases. *Foot Ankle Int* 2009; 30(9):836-841.
 - Coughlin MJ, Thompson FM. The high price of high-fashion footwear. *Instr Course Lect* 1995; 44:371-377.
 - Didia BC, Omu ET, Obuoforibo AA. The use of footprint contact index II for classification of flat feet in a Nigerian population. *Foot Ankle* 1987; 7(5):285-289.
 - Dunn JE, Link CL, Felson DT, Crincoli MG, Keysor JJ, McKinlay JB. Prevalence of foot and ankle conditions in a multiethnic community sample of older adults. *Am J Epidemiol* 2004; 159(5):491-498.
 - Frey C, Thompson F, Smith J, Sanders M, Horstman H. American Orthopaedic Foot and Ankle Society women's shoe survey. *Foot Ankle* 1993; 14(2):78-81.
 - Glazebrook M, Daniels T, Younger A, Foote CJ, Penner M, Wing K et al. Comparison of health-related quality of life between patients with end-stage ankle and hip arthrosis. *J Bone Joint Surg Am* 2008; 90(3):499-505.
 - Hill CL, Gill TK, Menz HB, Taylor AW. Prevalence and correlates of foot pain in a population-based study: the North West Adelaide health study. *J Foot Ankle Res* 2008; 1(1):2.
 - Leveille SG, Guralnik JM, Ferrucci L, Hirsch R, Simonsick E, Hochberg MC. Foot pain and disability in older women. *Am J Epidemiol* 1998; 148(7):657-665.
 - Menz HB, Jordan KP, Roddy E, Croft PR. Characteristics of primary care consultations for musculoskeletal foot and ankle problems in the UK. *Rheumatology (Oxford)* 2010; 49(7):1391-1398.
 - NHANES III Examination Data File. Third National Health and Nutrition Examination Survey, 1988-1994. Public Use Data File Documentation No 76200 Hyattsville, MD: Centers for Disease Control and Prevention 1996.
 - Ozdemir HM, Yildiz Y, Yilmaz C, Saglik Y. Tumors of the foot and ankle: analysis of 196 cases. *J Foot Ankle Surg* 1997; 36(6):403-408.
 - Sim-Fook LA, Hodgson AR. A comparison of foot forms among non-shoe and shoe-wearing Chinese populations. *J Bone Joint Surg Am* 1958; 40:1058-1062.

Ankle Osteoarthritis: Ankle Arthrodesis and Total Ankle Arthroplasty

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Ankle Osteoarthritis

Osteoarthritis as a debilitating chronic disease is a growing problem in Health Care [12]. Approximately 1% of the world's adult population is affected by ankle OA with pain, dysfunction, and impaired mobility [12, 50]. It has been shown, that the mental and physical disability associated with end-stage ankle OA is at least as severe as that associated with end-stage hip OA [12]. While the etiology of hip and knee OA is well understood and widely highlighted in numerous clinical studies, research

related to ankle OA is limited in the current literature [50]. The knowledge and exact analysis of the underlying etiology is important for selecting the best treatment strategy and is key to achieving satisfactory long-term results and avoiding postoperative complications.

Unlike the hip and knee, the ankle joint is rarely affected by primary OA. Instead, numerous clinical and epidemiologic studies have identified previous trauma as the most common origin for ankle OA showing that patients with posttraumatic OA are younger than the patients with primary OA [7, 18, 41, 44, 50]. Saltzman et al evaluated 639 patients presenting with painful end-stage ankle OA (Kellgren grade 3 or 4) [41]. In this patient cohort, 445 patients (70%) had post-traumatic, 76 patients (12%) had rheumatoid, and 46 (7%) had primary ankle OA. While rotational ankle fractures was identified as the most common reason for posttraumatic ankle OA (164 patients), previous ligament injuries were also found as a reason for ankle OA in 126 patients [41]. These trends were confirmed by a study performed by Valderrabano et al, who evaluated etiologies of ankle OA in 390 consecutive patients (406 ankles) with painful end-stage ankle OA [50]. Most patients (78%) presented with posttraumatic OA. In that study, the malleolar fractures were the most common reason for degenerative changes in ankle joint (157 patients) followed by ankle ligament lesions (60 patients). Only 31 patients studied by Valderrabano et al were affected by primary OA while secondary OA was seen as the more common etiology of end-stage ankle OA (46 patients) [50]. Secondary OA has also been found to be associated with the a variety of underlying diseases/pathologies, such as rheumatoid disease, hemochromatosis, hemophilia, gout, neuropathic diseases, avascular talus necrosis, osteochondral lesion, or postinfectious arthritis [41, 50].

Diagnostic Assessment of Ankle Osteoarthritis

Patient History

When diagnosing suspected cases of ankle OA a careful patient history should be taken [19]. All previous medical reports (e.g. surgery reports) and imaging studies should be collected or ordered. The underlying OA etiology should be specifically addressed and evaluated. In general, all concomitant diseases should be carefully assessed [42]. If necessary, a consultation with the department of neurology and/or internal medicine should be arranged to assess the concomitant systematic diseases. The following specific aspects should be addressed regarding the ankle pathology: previous trauma, infections, and/or surgeries, actual pain level (e.g. using visual analogue scale (VAS) ranging from 0 (no pain) to 10 (maximal pain) [22, 30]), limitations in daily activities (e.g. using a short-form (SF)-36 questionnaire [30, 54]) and sports (e.g. using the following score: Grade 0, none; Grade 1, moderate; Grade 2, normal; Grade 3, high; Grade 4, elite [52]).

Physical Examination

The routine physical examination includes careful inspection of the whole lower extremity, including a comparison to the contralateral non-affected limb. Skin and soft tissue should be carefully evaluated, with special attention given to previous surgical scars. Hindfoot stability, especially ligament stability of the ankle and subtalar joints (anterior drawer and tilt tests), should be proven manually with the patient sitting. Ankle alignment should be assessed while the patient is standing. In cases with foot/ankle deformities, the extent to which the deformity can be corrected should be assessed. As many patients with end-stage OA present with lower leg muscular atrophy, [51] basic muscle function should be assessed (e.g. function of tibial and peroneal muscles). ROM

should be determined clinically with a goniometer placed along the lateral border of the foot. For improved accuracy, the ROM should be measured radiographically while patient standing [6]. Systematic assessment of ankle function can be calculated with the American Orthopaedic Foot and Ankle Society (AOFAS) hindfoot score [23, 30]. This widely used AOFAS hindfoot score has not been validated.

Imaging and Other Diagnostic Studies

Routine radiographic imaging includes standard foot/ankle radiographs in three planes. All radiographs should be performed in a weight bearing position for correct assessment of statics and biomechanics of foot and ankle. All possible coexisting degenerative changes in the adjacent joints as well as any deformities (e.g. flattening of the longitudinal arch of the foot) should be identified and carefully analyzed. The special hindfoot alignment view should be acquired for standardized assessment of varus and valgus of the hindfoot [39]. The clinical relevance of this special hindfoot alignment view in TAR patients has been previously proven [11]. A computed tomography (CT) scan may be helpful to assess the bony defects and to analyze the joint incongruency. In patients with painful degenerative changes in adjacent joints a single-photon-emission computed tomography combined with computed tomography with a superimposed bone scintigraphy scan (SPECT-CT) can be performed for exact localization of degenerative changes and for assessment of their biological activity [24,33]. We do not recommend routine use of MRI. However, in some cases MRI may be helpful to assess the concomitant ligamentous injuries, [17, 20], pathological changes of tendons, [9] and avascular necrosis of osseous structures [17, 20]. We recommend pedobarography to assess the preoperative and postoperative alteration in gait characteristics and plantar pressure distribution [21].

Ankle Arthrodesis

Ankle arthrodesis has been considered to be the "gold-standard" treatment option in patients with end-stage ankle osteoarthritis for the long time [1,32]. The surgical technique of the ankle arthrodesis has been described for the first time in the year 1882 [2]. Since then more than 40 different surgical techniques have been described in the current literature with good mid-term results including substantial pain relief [5,16]. The surgical techniques used for ankle arthrodesis can be classified based on surgical method, approach, and fixation type as shown in Table 1 [32].

One of the most common approaches used for ankle arthrodesis is the transfibular approach (Fig. 1). The distal fibula may be used as autograft and also may be incorporated for the lateral support [1, 32]. While the lateral approach is mainly used for standard primary arthrodesis of the tibiotalar joint, the anterior approach for plating fixation can be used for complex cases, e.g. in patients with substantial bony defects or in patients with failed previous total ankle arthroplasty or non-union of ankle fusion [34]. In the current literature also arthroscopic surgical techniques for ankle arthrodesis have been described [10]. Both – anterior and posterior ankle/hindfoot arthroscopic approaches can be used for this purpose [31]. Arthroscopic surgical technique has been shown to be more technically demanding with a steep learning curve [35]. However, the arthroscopic ankle arthrodesis has been shown to provide good functional outcomes with a fusion rate up to 100% in the current literature [15, 32, 55]. Recently, a comparison of arthroscopic versus open ankle arthrodesis was performed in a multicenter comparative case series showing that arthroscopic arthrodesis resulted in a shorter hospital stay and better outcomes at one and two years [46].

Table 1 Classification of surgical techniques used for ankle arthrodesis

Open ankle arthrodesis	anterior approach (horizontal incision, longitudinal incision)
	posterior approach
	lateral approach
	medial approach
	combined lateral and medial approach
Arthroscopic ankle arthrodesis	anterior ankle arthroscopy
	posterior ankle/hindfoot arthroscopy
Mini-open ankle arthrodesis	
External fixation	external Charnley fixateur
	Ilizarov fixateur
	Hoffman double fixateur
	hybrid fixateur
Internal fixation	screw fixation
	plating fixation
	intramedullary nail
No fixation	immobilization using cast
	immobilization using stable walker (e.g. Aircast, Vacoped etc.)



Fig. 1 Tibiotalocalcaneal arthrodesis. A: 59-year old female with severe end-stage osteoarthritis of the tibiotalar and subtalar joints. Saltzman view shows substantial valgus deformity of the hindfoot. B: CT-scans confirm severe osteoarthritis with substantial destruction of the tibiotalar and subtalar joints and subchondral cysts. C: Tibiotalocalcaneal arthrodesis has been performed using intramedullary fixation. Post-operative Saltzman view shows physiological alignment of the hindfoot.

Although most ankle arthrodeses may provide substantial pain relief and satisfactory functional outcomes, at least in the short-term, [43] many clinical reports have described mid- and long-term problems following ankle arthrodesis. These problems include some difficulties in daily activities such as climbing stairs, getting out of a chair, walking on uneven surfaces, fast walking, and running [4, 25, 27, 28].

Total Ankle Arthroplasty

Total ankle arthroplasty has a substantially shorter history than the total arthroplasty of knee or hip joint. The first generation total ankle arthroplasties have been introduced in 1960s-1970s as an alternative treatment option in patients with end-stage ankle osteoarthritis [14]. Muir et al [29] reported 40-year outcome in a 71-year old male who underwent talar dome resurfacing with a custom Vitallium implant for posttraumatic ankle osteoarthritis in 1962. Most first generation total ankle arthroplasty designs were two component prostheses with cement fixation used on both sided, talar and tibial [37]. Overall, the clinical results of first generation ankle prostheses were highly discouraging due to unacceptable high failure rate, along with other complications like wound healing problems, low functional outcomes etc. The critical analysis of the main failure reasons led to the development of the second generation ankle prostheses. All three main second generation total ankle arthroplasty designs – Agility, Buechel-Pappas, and STAR – have been clinically used with encouraging mid- and long-term results and acceptable survival rate of prosthesis components [36].

The most modern third generation total ankle arthroplasty designs are three component prostheses with mobile bearing (Table 2). While numerous biomechanical studies revealed some advantages of 3-component prosthesis designs regarding biomechanics and kinematics, [8, 47-49] not obvious superiority of any prosthesis design could be identified in the current literature [13, 53].

Table 2 Classification of different total ankle arthroplasty designs

Fixation	cemented
	uncemented
Number of components	2-components
	3-components
Constraint type	constrained
	semi-constrained
	unconstrained
Congruency type	congruent
	incongruent
Component shape	anatomic
	non-anatomic
Bearing type	fixed bearing
	mobile bearing

Total ankle arthroplasty is constantly gaining more acceptance among the orthopedic surgeons. Recently, Saltzman et al [40] published initial findings from a prospective controlled trial of the STAR prosthesis versus ankle arthrodesis showing that total ankle arthroplasty led to better

function and similar postoperative pain relief compared with patients who underwent ankle arthrodesis. However, total ankle arthroplasty still remains a technically demanding procedure with a steep learning curve [26, 38]. Especially correct positioning of the talar component is one of the most demanding intraoperative steps with sagittal malpositioning resulting in negative biomechanical consequences [8, 45] and worse clinical outcome [3].

The clinical outcome following total ankle arthroplasty and survival rate of prosthesis components are constantly improving. In the current literature, the 5-year survival rate is approximately 90% with a range between 68% and 100% [13]. Further biomechanical and clinical studies addressing the outcome and biomechanical properties of total ankle arthroplasty should be continued with the aim to improve the current total ankle arthroplasty designs.

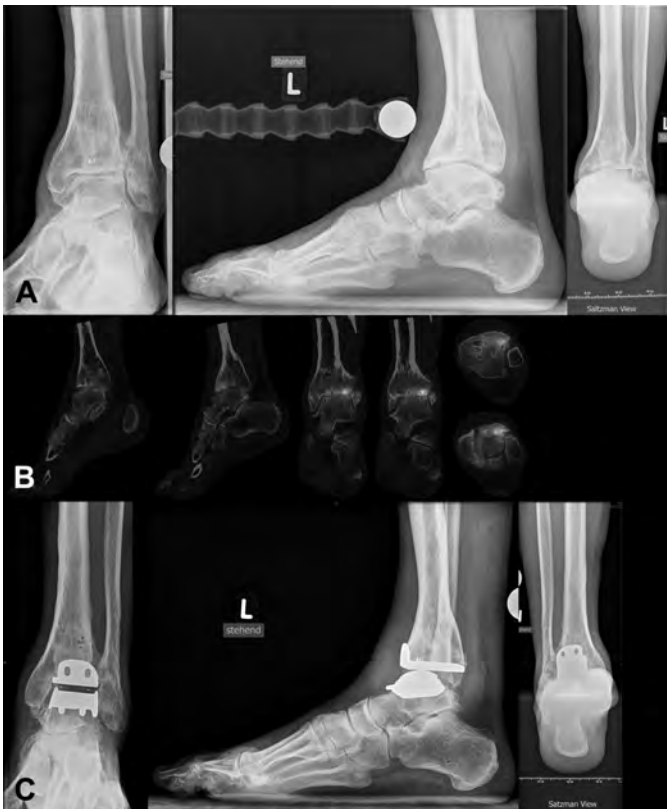


Fig. 2 Total ankle arthroplasty. A: 73-year old male with symmetric osteoarthritis of the tibiotalar joint. Saltzman view shows no concomitant deformity of the hindfoot. B: SPECT-CT shows exact localization and biologic activity of osteoarthritic changes. C: Total ankle arthroplasty has been performed using HINTEGRA prosthesis (a non-constrained, three-component total ankle prosthesis). Postoperative Saltzman view shows physiological hindfoot alignment.

References

1. Ahmad J, Raikin SM. Ankle arthrodesis: the simple and the complex. *Foot Ankle Clin* 2008; 13(3):381-400, viii.
2. Albert E. Einige Falle kunstlicher Ankylosen: Bildung an paralytischen Gliedmassen. *Wien Med Press* 1882; 23:726-728.
3. Barg A, Elsner A, Anderson AE, Hintermann B. The effect of three-component total ankle replacement malalignment on clinical outcome: pain relief and functional outcome in 317 consecutive patients. *J Bone Joint Surg Am* 2011; 93(21):1969-1978.

4. Boobbyer GN. The long-term results of ankle arthrodesis. *Acta Orthop Scand* 1981; 52(1):107-110.
5. Coester LM, Saltzman CL, Leupold J, Pontarelli W. Long-term results following ankle arthrodesis for post-traumatic arthritis. *J Bone Joint Surg Am* 2001; 83-A(2):219-228.
6. Coetzee JC, Castro MD. Accurate measurement of ankle range of motion after total ankle arthroplasty. *Clin Orthop Relat Res* 2004; 424(424):27-31.
7. Daniels T, Thomas R. Etiology and biomechanics of ankle arthritis. *Foot Ankle Clin* 2008; 13(3):341-352.
8. Espinosa N, Walti M, Favre P, Snedeker JG. Misalignment of total ankle components can induce high joint contact pressures. *J Bone Joint Surg Am* 2010; 92(5):1179-1187.
9. Feighan J, Towers J, Conti S. The use of magnetic resonance imaging in posterior tibial tendon dysfunction. *Clin Orthop Relat Res* 1999; 365(365):23-38.
10. Ferkel RD, Scranton PE, Jr. Arthroscopy of the ankle and foot. *J Bone Joint Surg Am* 1993; 75(8):1233-1242.
11. Frigg A, Nigg B, Davis E, Pederson B, Valderrabano V. Does Alignment in the Hindfoot Radiograph Influence Dynamic Foot-floor Pressures in Ankle and Tibiotalocalcaneal Fusion? *Clin Orthop Relat Res* 2010; 468(12):3362-3370.
12. Glazebrook M, Daniels T, Younger A, Foote CJ, Penner M, Wing K et al. Comparison of health-related quality of life between patients with end-stage ankle and hip arthrosis. *J Bone Joint Surg Am* 2008; 90(3):499-505.
13. Gougoulis N, Khanna A, Maffulli N. How successful are current ankle replacements?: a systematic review of the literature. *Clin Orthop Relat Res* 2010; 468(1):199-208.
14. Gougoulis N, Maffulli N. History of total ankle replacement. *Clin Podiatr Med Surg* 2013; 30(1):1-20.
15. Gougoulis NE, Agathangelidis FG, Parsons SW. Arthroscopic ankle arthrodesis. *Foot Ankle Int* 2007; 28(6):695-706.
16. Guo C, Yan Z, Barfield WR, Hartstock LA. Ankle arthrodesis using anatomically contoured anterior plate. *Foot Ankle Surg* 2010; 31:492-498.
17. Haygood TM. Magnetic resonance imaging of the musculoskeletal system: part 7. The ankle. *Clin Orthop Relat Res* 1997; 336(336):318-336.
18. Hintermann B. Characteristics of the diseased ankle. In: Hintermann B, editor. *Total ankle arthroplasty: Historical overview, current concepts and future perspectives*. Wien New York: Springer, 2005: 5-9.
19. Hintermann B. Preoperative considerations for total ankle arthroplasty. In: Hintermann B, editor. *Total ankle arthroplasty: Historical overview, current concepts and future perspectives*. Wien New York: Springer, 2005: 91-104.
20. Hintermann B. What the orthopaedic foot and ankle surgeon wants to know from MR Imaging. *Semin Musculoskelet Radiol* 2005; 9(3):260-271.
21. Horisberger M, Hintermann B, Valderrabano V. Alterations of plantar pressure distribution in posttraumatic end-stage ankle osteoarthritis. *Clin Biomech (Bristol, Avon)* 2009; 24(3):303-307.
22. Huskisson EC. Measurement of pain. *Lancet* 1974; 2(7889):1127-1131.
23. Kitaoka HB, Alexander IJ, Adelaar RS, Nunley JA, Myerson MS, Sanders M. Clinical rating systems for the ankle-hindfoot, midfoot, hallux, and lesser toes. *Foot Ankle Int* 1994; 15(7):349-353.
24. Knupp M, Pagenstert GI, Barg A, Bolliger L, Easley ME, Hintermann B. SPECT-CT compared with conventional imaging modalities for the assessment of the varus and valgus malaligned hindfoot. *J Orthop Res* 2009; 27(11):1461-1466.
25. Lance EM, Paval A, Fries I, Larsen I, Patterson RL, Jr. Arthrodesis of the ankle joint. A follow-up study. *Clin Orthop Relat Res* 1979; (142):146-158.
26. Lee KB, Cho SG, Hur CI, Yoon TR. Perioperative complications of HINTEGRA total ankle replacement: our initial 50 cases. *Foot Ankle Int* 2008; 29(10):978-984.
27. Mazur JM, Schwartz E, Simon SR. Ankle arthrodesis. Long-term follow-up with gait analysis. *J Bone Joint Surg Am* 1979; 61(7):964-975.
28. Morgan CD, Henke JA, Bailey RW, Kaufer H. Long-term results of tibiotalar arthrodesis. *J Bone Joint Surg Am* 1985; 67(4):546-550.
29. Muir DC, Amendola A, Saltzman CL. Forty-year outcome of ankle "cup" arthroplasty for post-traumatic arthritis. *Iowa Orthop J* 2002; 22:99-102.
30. Naal FD, Impellizzeri FM, Rippstein PF. Which are the most frequently used outcome instruments in studies on total ankle arthroplasty? *Clin Orthop Relat Res* 2010; 468(3):815-826.
31. Nickisch F, Barg A, Saltzman CL, Beals TC, Bonasia DE, Phisitkul P et al. Postoperative complications of posterior ankle and hindfoot arthroscopy. *J Bone Joint Surg Am* 2012; 94(5):439-446.
32. Nihal A, Gellman RE, Embil JM, Trepman E. Ankle arthrodesis. *Foot Ankle Surg* 2008; 14(1):1-10.
33. Pagenstert GI, Barg A, Leumann AG, Rasch H, Muller-Brand J, Hintermann B et al. SPECT-CT imaging in degenerative joint disease of the foot and ankle. *J Bone Joint Surg Br* 2009; 91(9):1191-1196.
34. Plassa C, Knupp M, Barg A, Hintermann B. Anterior double plating for rigid fixation of isolated tibiotalar arthrodesis. *Foot Ankle Int* 2009; 30(7):631-639.
35. Raikin SM. Arthrodesis of the ankle: arthroscopic, mini-open, and open techniques. *Foot Ankle Clin* 2003; 8(2):347-359.
36. Rippstein PF. Clinical experiences with three different designs of ankle prostheses. *Foot Ankle Clin* 2002; 7(4):817-831.
37. Saltzman CL. Perspective on total ankle replacement. *Foot Ankle Clin* 2000; 5(4):761-775.
38. Saltzman CL, Amendola A, Anderson R, Coetzee JC, Gall RJ, Haddad SL et al. Surgeon training and complications in total ankle arthroplasty. *Foot Ankle Int* 2003; 24(6):514-518.
39. Saltzman CL, el Khoury GY. The hindfoot alignment view. *Foot Ankle Int* 1995; 16(9):572-576.
40. Saltzman CL, Mann RA, Ahrens JE, Amendola A, Anderson RB, Berlet GC et al. Prospective controlled trial of STAR total ankle replacement versus ankle fusion: initial results. *Foot Ankle Int* 2009; 30(7):579-596.
41. Saltzman CL, Salamon ML, Blanchard GM, Huff T, Hayes A, Buckwalter JA et al. Epidemiology of ankle arthritis: report of a consecutive series of 639 patients from a tertiary orthopaedic center. *Iowa Orthop J* 2005; 25:44-46.
42. Saltzman CL, Zimmerman MB, O'Rourke M, Brown TD, Buckwalter JA, Johnston R. Impact of comorbidities on the measurement of health in patients with ankle osteoarthritis. *J Bone Joint Surg Am* 2006; 88(11):2366-2372.
43. Sealey RJ, Myerson MS, Molloy A, Gamba C, Jeng C, Kalesan B. Sagittal plane motion of the hindfoot following ankle arthrodesis: a prospective analysis. *Foot Ankle Int* 2009; 30(3):187-196.
44. Thomas RH, Daniels TR. Ankle arthritis. *J Bone Joint Surg Am* 2003; 85-A(5):923-936.
45. Tochigi Y, Rudert MJ, Brown TD, McIlff TE, Saltzman CL. The effect of accuracy of implantation on range of movement of the Scandinavian Total Ankle Replacement. *J Bone Joint Surg Br* 2005; 87(5):736-740.
46. Townshend D, Di Silvestro M, Krause F, Penner M, Younger A, Glazebrook M et al. Arthroscopic versus open ankle arthrodesis: a multicenter

- comparative case series. *J Bone Joint Surg Am* 2013; 95(2):98-102.
47. Valderrabano V, Hintermann B, Nigg BM, Stefanyshyn D, Stergiou P. Kinematic changes after fusion and total replacement of the ankle: part 1: Range of motion. *Foot Ankle Int* 2003; 24(12):881-887.
 48. Valderrabano V, Hintermann B, Nigg BM, Stefanyshyn D, Stergiou P. Kinematic changes after fusion and total replacement of the ankle: part 2: Movement transfer. *Foot Ankle Int* 2003; 24(12):888-896.
 49. Valderrabano V, Hintermann B, Nigg BM, Stefanyshyn D, Stergiou P. Kinematic changes after fusion and total replacement of the ankle: part 3: Talar movement. *Foot Ankle Int* 2003; 24(12):897-900.
 50. Valderrabano V, Horisberger M, Russell I, Dougall H, Hintermann B. Etiology of ankle osteoarthritis. *Clin Orthop Relat Res* 2009; 467(7):1800-1806.
 51. Valderrabano V, Nigg BM, von T, V, Frank CB, Hintermann B. J. Leonard Goldner Award 2006. Total ankle replacement in ankle osteoarthritis: an analysis of muscle rehabilitation. *Foot Ankle Int* 2007; 28(2): 281-291.
 52. Valderrabano V, Pagenstert G, Horisberger M, Knupp M, Hintermann B. Sports and recreation activity of ankle arthritis patients before and after total ankle replacement. *Am J Sports Med* 2006; 34(6):993-999.
 53. Valderrabano V, Pagenstert G, Muller AM, Paul J, Henninger HB, Barg A. Mobile- and fixed-bearing total ankle prostheses: is there really a difference? *Foot Ankle Clin* 2012; 17(4):565-585.
 54. Ware JE, Jr., Sherbourne CD. The MOS 36-item short-form health survey (SF-36). I. Conceptual framework and item selection. *Med Care* 1992; 30(6):473-483.
 55. Winson IG, Robinson DE, Allen PE. Arthroscopic ankle arthrodesis. *J Bone Joint Surg Br* 2005; 87(3):343-347.

Adult Flat Foot

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The adult flatfoot is a common pathology in adult people. A careful clinical and radiographic assessment is crucial to provide the appropriate treatment. A conservative therapy may be initiated in patients with mild symptoms, however, severe flatfoot deformity with breakdown of the medial arch and substantial deformity of the hindfoot often need surgical correction. Soft tissue and bony procedures are often combined to address tendon pathologies and osseous malalignment. Postoperative outcomes presented in the current literature are promising with a high patients' satisfaction rate.

Etiology and Biomechanics

The flatfoot deformity (Pes planus deformity) is defined as the loss of the normal medial longitudinal arch of the foot, which can be combined with other morbidities such as valgus malalignment of the hindfoot, subluxation of the subtalar joint, eversion of the subtalar calcaneus, lateral abduction in the midtarsal joint, and adaptive forefoot supination [16]. The hindfoot valgus deformity is often aggravated by a shortened Achilles tendon. In total, 15%-20% of adults have a remaining flexible flatfoot from childhood, meaning the pes planus deformity may be corrected in non-weight bearing situations [6]. Most of the patients are asymptomatic or have only minor problems with those feet [4]. The

deformity is caused by a dysfunction of the static and dynamic structures of the medial longitudinal arch of the adult foot. In many such cases, a dysfunction of the posterior tibial tendon (PTT) seems to be the most common reason for this disorders [4, 10, 11, 34]. Acute trauma, inflammation, chronic stress, coalitio, Charcot neuroarthropathy, neuromuscular imbalances (e.g. polio), or iatrogenic reasons (e.g. repetitive injections of steroid) can lead to PTT dysfunction. [4,6] Especially chronic stress on the PTT and biomechanical misuse can lead to stepwise progression of the pathology.

The most common classification for the adult flatfoot refers to PTT dysfunction and was introduced by Johnson and Strom [13]. They initially described three stages but their classification was modified by Myerson [22] later on and amended by a fourth stage.

Stage 1

Swelling and pain on the medial aspect of the foot
 Painful tenosynovitis of PTT, tendon length is often normal
 Single-limb heel rise test can be performed

Stage 2

Elongation and degeneration of the PTT
 Obvious deformity of the foot (hindfoot valgus, collapse the talonavicular joint)
 Valgus deformity is flexible (reduction manually possible)

Stage 3

Rigid valgus hindfoot deformity (no reduction of subtalar joint)
 Subfibular impingement and possible pain at rest (Osteoarthritis)
 Single-limb heel rise cannot be performed

Stage 4

Lateral compartment Osteoarthritis
 Possible valgus deformity of the talus (obvious in X-ray)
 Loss of function of the deltoid ligament

Conservative Treatment

Conservative treatment should be initiated first in treatment of adult flatfoot deformity. Therapy options are: immobilization, anti-inflammatory medication, physical therapy, footwear changes or modifications, and the use of braces or orthotics. However, an accurate and well-defined diagnosis prior to initiation of a conservative strategy is essential for the choice of the appropriate treatment [20]. Moreover treatment options are dependent on the extent and stage of the deformity (see above).

Immobilization/Rest

Acute pain along the posterior tibial tendon according to Stage I insufficiency can be relieved and even the valgus deformity of the hindfoot can be corrected with a rigid stirrup brace or lace-up sport brace. However, this does not stabilize the ankle in the sagittal plane allowing motion of the tendon and thus not inducing complete rest. Therefore repetitive stress and inflammation may remain, which is the main problem in stage I dysfunction [23]. To achieve a complete immobilization a below-knee cast should be applied. This also provides stabilization in the sagittal plane. However, a simultaneous physical therapy is not possible, muscle atrophy may occur, and a higher risk for venous thrombosis remains. An interim solution is provided by a below-knee cast walker, which can be taken off for physical therapy but provides a comparable stability of the hindfoot. If asymptomatic the patient is allowed to full weight bearing within the cast or walker. The time of immobilization is usually 4 to 8 weeks [23].

Medication

Usually a course of 2 weeks of anti-inflammatory drugs (NSAID) is recommended in addition with rest of the tendon to calm down the inflammation in the acute phase. NSAIDs can also be applied locally by the patient. Local treatment together with local physical therapy might positively influence the course. Although widely used, corticosteroids should not be used neither systemically nor with local injections due to their possible devastating side effects up to causing tendon rupture.

Physical Therapy

Passive physical therapy including local therapy and iontophoresis, in which dexamethasone is repelled into the deep soft tissue with electric current, has the potential to calm down the inflammatory process, which again may be useful in the early stages. So far, there is no documented risk of tendon rupture in iontophoresis making this a safe therapy option [2]. Ice massage or cryotherapy can be performed by the patient himself and is particular beneficial after activity. As ultrasound can heat the tissue it should rather be used in a pulsed manner not to increase inflammation by its heat.

Active treatment with isolated strengthening of the tendon should be hold up to the patient's freedom of pain, again not to increase inflammation and consequently prolong the healing process. Selective activation of the posterior tibial tendon is achieved by a forced adduction against resistance (e.g. Thera-Band) [18]. Moreover stretching of the muscles should be integrated in the active training program.

Orthotics

Orthotics can be classified according to their rigidity as rigid, semirigid, or soft. In any case, a soft top cover should overlay the harder portion of the orthotics to avoid excessive plantar pressure. The stress on the PTT can be simply reduced by supporting the medial arch from extern using an appropriate device. This eliminates pronation, preconditioned that the flatfoot deformity is flexibly (Stage I and II). In this case the orthotic must keep the heel in a subtalar neutral position, which can be achieved by a full-foot-length semirigid device for Stage I and a rather rigid device for Stage II. Ideally, the customized orthotics can be transferred from one pair of shoes to another. In a rigid deformity the orthotic is fitted in situ and must not correct the hindfoot position to increase the comfort. In these cases a modification of the footwear is important. First to have enough space for the malaligned foot and the orthotics, second the surrounding soft tissue needs to be protected around the deformity to avoid compromising. In higher stages of flatfoot deformity a foot-ankle orthotics gives more stability compared to the foot only orthoses by stabilizing also the ankle joint in the sagittal plane and thus avoiding subfibular impingement. However, patients with rigid deformities possibly profit as well from a describe devise, even though only an operative treatment can definitively correct the deformity.

Operative Treatment

Soft Tissue Procedures

All soft tissue procedures for the adult flatfoot are quite often accompanied by osteotomies or osseous corrections. Most of the procedures strengthen, reinforce or replace the PTT to compensate for the dysfunction therefore we focus in this chapter on these techniques. The term PTT dysfunction has replaced many of the traditional terms such as 'flatfoot deformity in the adult patient' or 'adult-acquired flatfoot deformity' and has been used exchangeable [4, 10, 11]. This indicates that the insufficiency of the PTT plays an important role and is therefore addressed with the soft tissue procedures. Isolated medial

soft tissue procedures are only an option if the deformity is flexible and fully reducible [11]. Especially in patients with mild complaints an promptly after onset of symptoms these procedures seem to be an option. Combinations with osteotomies or other osseous corrections are often indicated when the PTT dysfunction proceeds or symptoms have been prevalent over a longer time [11].

Cobb Procedure

The Cobb procedure is a tendon transfer, where the anterior tibial tendon (ATT) is transferred to the PTT. The ATT is not sacrificed but rather split in length to strengthen the PTT with the transferred part. An advantage of this technique is the long distance of PTT defect, which can be restored. Defects up to the medial malleolus or even more proximal can be addressed with this technique [11]. The pathology of the PTT can be evaluated during the operation and the split of the ATT can be addressed according to it. The distal insertion of the ATT is left in place (first cuneiform and first metatarsal) and the tendon is identified proximally through a mini open approach and then mobilized. The ATT is split in half from proximally and one portion is detached from the distal insertion. The authors prefer to insert it to the original insertion of the PTT plantar (e.g. tenodesis screw). It has been described that an anastomosis of the ATT to the PTT side-to-side or end-to-end is reasonable as well [11].

Kidner Procedure

An accessory navicular bone, as seen in d.p. (dorso-plantar) x-ray, may lead to an irregular insertion of the PTT [14, 15]. As a result, the PTT becomes insufficient and a symptomatic pes planovalgus occurs, as described above. In cases with an hypertrophic navicular bone or an accessory navicular bone the so called "Kidner procedure" provides sufficient surgical relief. In this procedure the accessory bone is removed and the insertion of the PTT is readapted to the anatomic position (the plantar side of the navicular bone) [32]. After mobilization of the PTT it is readapted and fixated transosseous or with interference screws. However it is important to protect al medial structures (e.g. spring ligament) because it has been described that injuries might have worsened the symptoms of medial pain in flatfoot patients after the procedure [11].

Spring Ligament Reconstruction

The spring (plantar talonavicular) ligament is an important static stabilizer of the medial arch of the foot and if it is ruptured or insufficient progression of flatfoot deformity is described [3, 33]. The clinical impression of a spring ligament rupture might even be similar to a PTT ruptur [3]. Operative repair of the insufficient or ruptured spring ligament is important to restore biomechanical properties of the medial arch and provide functional stability. For operative repair the spring ligament might either be sutured directly or it can be reconstructed in severe cases with a tendon graft (e.g. flexor digitorum tendon or the peroneus longus tendon) which is fixated on the medial aspect of the foot to provide stability [3, 11]. In cases of severe Pes planus or a traumatic history the authors advice to inspect the spring ligament compulsively to test for potential lesions and enable a direct operative repair.

Posterior Tibial Tendon Repair and Reconstruction

Soft tissue PTT repair and reconstruction is often accompanied by various types of osteotomies. Isolated soft tissue procedures are rare and in most of the cases in isolated, early stage PTT lesions. Especially MRT provides valuable information about the PTT concerning inflammation, tendonitis or complete/partial ruptures [5]. Debridement and synovectomy

are valuable options for inflammatory changes of the PTT [8]. Direct repair of the tendon should be performed whenever possible to restore the natural anatomy. In cases of a structural deficit a tendon transfer is a good option. As a graft the flexor digitorum longus or the flexor hallucis longus tendons are often used [11, 12]. With both tendons multiple techniques and variations have been described including an end-to-end repair or a tenodesis with an insertion of the graft tendon to the navicular bone. For fixation interference screws, trans osseous drill holes or soft tissue anchors can be used [11, 12].

Calcaneal Osteotomies

Different types of calcaneal osteotomies with different biomechanical impact of the foot have been described in the current literature. Lateral column lengthening (Fig. 1), displacement osteotomy of the posterior calcaneus (e.g. medial sliding osteotomy, Fig. 2) and combinations of those are the most common [7, 9, 17, 31].



Fig. 1 Lateral column lengthening osteotomy of the calcaneus. A: 42-year old female with substantial abductus deformity of the midfoot and valgus malalignment of the hindfoot. B: Lateral column lengthening osteotomy of the calcaneus with use of a spongy allograft block and a FDL on PTT transfer were performed. Fixation of the calcaneus osteotomy can be alternatively performed using a screw.

Calcaneal osteotomies of both types are biomechanical powerful procedures in Stage II PTT disorders and are often combined with the above mentioned soft tissue procedures.

Lateral column lengthening of the calcaneus was first described by Evans [7]. However it has been shown that placing the osteotomy more proximally (between the posterior and the middle facet of the subtalar joint) does not violate the joint surfaces and Hintermann et al. showed good clinical results in their patient group [9]. Lateral lengthening of the calcaneus provides a triplanar correction. The forefoot comes out of abduction, it supinates the subtalar joint and elevates the medial arch through an more powerful plantar pull of (the peroneus longus is tightened) [17].

The medial sliding osteotomy of the calcaneus in contrast redirects the forces of the M. triceps surae from valgus (pronatory force) to a neutral or even supinatory force [10]. This results in a powerful correction of the hindfoot [24, 25, 30]. Preoperative radiographs including the Saltzman view [28] is very important to evaluate the valgus component of the TPP dysfunction in contrast to the flatfoot portion. The combination of a

lateral lengthening osteotomy and a medial sliding osteotomy of the calcaneus is a good treatment option for stage II PTT dysfunction with severe valgus and flatfoot component [9]. Patients treated with this double-osteotomies are reported to sustained a lasting correction of their pes planovalgus foot deformity and a high patient satisfaction is described [21, 26]. The authors use these double osteotomies of the calcaneus as well and often in combination with a flexor digitorum longus transfer in combined pathologies. The extra-articular locations of these procedures might impede osteoarthritis development of the joints with a powerful biomechanical correction.

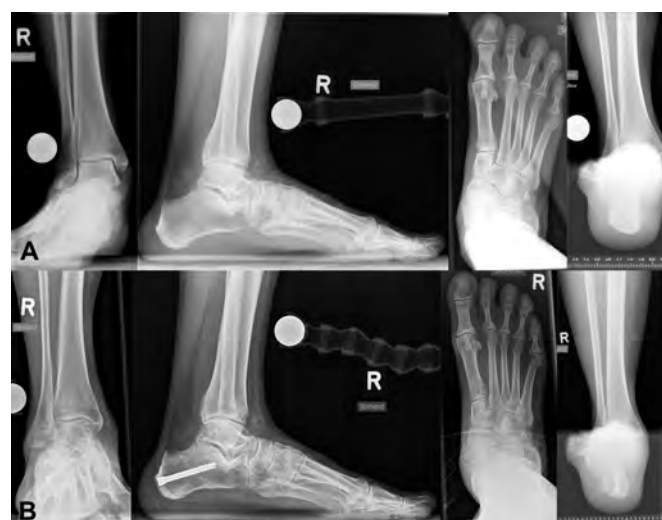


Fig. 2 Medial sliding osteotomy of the calcaneus. A: 59-year old male with severe valgus malalignment of the hindfoot and substantial flattening of the medial arch. B: Medial sliding osteotomy of the calcaneus was performed resulting in physiological alignment of the hindfoot.

Arthrodesis

In cases of end-stage rigid flatfoot deformity with stage III and IV PTT insufficiency a correcting soft tissue procedure or osteotomies are not sufficient enough to correct misalignment [1]. Depending on the severity of the deformity a subtalar fusion or even a modified triple arthrodesis (subtalar and talonavicular joints without calcaneocuboidal joint) need to be performed to ensure a sufficient hind foot realignment and stability [19, 27]. After having removed the remaining cartilage and realigned the hindfoot the arthrodesis is secured with compression screws or possibly with locking plates. To allow motion within the lateral column of the foot fusing the calcaneocuboidal joint is not recommended by the authors unless the patient has rheumatoid arthritis. Malunion, inadequate correction, continued instability, gait disturbances, and adjacent joint degeneration are possible complications as a result of inadequate realignment [27].

In case of stage IV PTT dysfunction the tibiotalar osteoarthritis needs to be addressed as well by either a joint preserving realigning procedure or a joint sacrificing procedure such as ankle fusion or a total ankle replacement [29]. Satisfaction rates greater than 90% are reported after arthrodesis and nonunion rates were reduced significantly by improved osteosynthesis and postoperative non-weight bearing [27].

References

1. Ahmad J, Pedowitz D. Management of the rigid arthritic flatfoot in adults: triple arthrodesis. *Foot Ankle Clin* 2012; 17(2):309-322.

2. Bare AA. Tenosynovitis of the posterior tibial tendon. *Foot Ankle Clin* 2001; 6(1):37-66.
3. Borton DC, Saxby TS. Tear of the plantar calcaneonavicular (spring) ligament causing flatfoot. A case report. *J Bone Joint Surg Br* 1997; 79(4):641-643.
4. Chang TJ, Lee J. Subtalar joint arthroereisis in adult-acquired flatfoot and posterior tibial tendon dysfunction. *Clin Podiatr Med Surg* 2007; 24(4):687-697.
5. Conti S, Michelson J, Jahss M. Clinical significance of magnetic resonance imaging in preoperative planning for reconstruction of posterior tibial tendon ruptures. *Foot Ankle* 1992; 13(4):208-214.
6. Coughlin MJ, Mann RA, Saltzman CL. *Surgery of the foot and ankle. 8th Edition ed. Philadelphia: MOSBY Elsevier, 2007.*
7. Evans D. Calcaneo-valgus deformity. *J Bone Joint Surg Br* 1975; 57(3):270-278.
8. Gould JS. Direct repair of the posterior tibial tendon. *Foot Ankle Clin* 1997; 2(2):275-280.
9. Hintermann B, Valderrabano V, Kundert HP. Lengthening of the lateral column and reconstruction of the medial soft tissue for treatment of acquired flatfoot deformity associated with insufficiency of the posterior tibial tendon. *Foot Ankle Int* 1999; 20(10):622-629.
10. Hix J, Kim C, Mendicino RW, Saltrick K, Catanzariti AR. Calcaneal osteotomies for the treatment of adult-acquired flatfoot. *Clin Podiatr Med Surg* 2007; 24(4):699-719.
11. Jacobs AM. Soft tissue procedures for the stabilization of medial arch pathology in the management of flexible flatfoot deformity. *Clin Podiatr Med Surg* 2007; 24(4):657-665.
12. Johnson KA, Strom DE. Tibialis posterior tendon dysfunction. *Clin Orthop Relat Res* 1989; 239:196-206.
13. Johnson KA, Strom DE. Tibialis posterior tendon dysfunction. *Clin Orthop Relat Res* 1989; 239:196-206.
14. Kidner FC. The pre-hallux (accessory scaphoid) in its relation to flat foot. *J Bone Joint Surg Am* 1929; 11:831-837.
15. Kidner FC. The pre-hallux in relation to flatfoot. *JAMA* 1933; 101:1539-1542.
16. Kitaoka HB, Luo ZP, An KN. Three-dimensional analysis of flatfoot deformity: cadaver study. *Foot Ankle Int* 1998; 19(7):447-451.
17. Koutsogiannis E. Treatment of mobile flat foot by displacement osteotomy of the calcaneus. *J Bone Joint Surg Br* 1971; 53(1): 96-100.
18. Kulig K, Burnfield JM, Requejo SM, Sperry M, Terk M. Selective activation of tibialis posterior: evaluation by magnetic resonance imaging. *Med Sci Sports Exerc* 2004; 36(5):862-867.
19. Mann RA. Posterior tibial tendon dysfunction. Treatment by flexor digitorum longus transfer. *Foot Ankle Clin* 2001; 6(1):77-87.
20. Marzano R. Functional bracing of the adult acquired flatfoot. *Clin Podiatr Med Surg* 2007; 24(4):645-656.
21. Moseir-LaClair S, Pomeroy G, Manoli A. Intermediate follow-up on the double osteotomy and tendon transfer procedure for stage II posterior tibial tendon insufficiency. *Foot Ankle Int* 2001; 22(4):283-291.
22. Myerson M, Solomon G, Shereff M. Posterior tibial tendon dysfunction: its association with seronegative inflammatory disease. *Foot Ankle* 1989; 9(5):219-225.
23. Myerson MS. Adult acquired flatfoot deformity: treatment of dysfunction of the posterior tibial tendon. *Instr Course Lect* 1997; 46:393-405.
24. Myerson MS, Corrigan J, Thompson F, Schon LC. Tendon transfer combined with calcaneal osteotomy for treatment of posterior tibial tendon insufficiency: a radiological investigation. *Foot Ankle Int* 1995; 16(11):712-718.
25. Nyska M, Parks BG, Chu IT, Myerson MS. The contribution of the medial calcaneal osteotomy to the correction of flatfoot deformities. *Foot Ankle Int* 2001; 22(4):278-282.
26. Pomeroy GC, Manoli A. A new operative approach for flatfoot secondary to posterior tibial tendon insufficiency: a preliminary report. *Foot Ankle Int* 1997; 18(4):206-212.
27. Rush SM. Reconstructive options for failed flatfoot surgery. *Clin Podiatr Med Surg* 2007; 24(4):779-788.
28. Saltzman CL, el Khoury GY. The hindfoot alignment view. *Foot Ankle Int* 1995; 16(9):572-576.
29. Smith JT, Bluman EM. Update on stage IV acquired adult flatfoot disorder: when the deltoid ligament becomes dysfunctional. *Foot Ankle Clin* 2012; 17(2):351-360.
30. Steffensmeier SJ, Saltzman CL, Berbaum KS, Brown TD. Effects of medial and lateral displacement calcaneal osteotomies on tibiotalar joint contact stresses. *J Orthop Res* 1996; 14(6):980-985.
31. Stufkens SA, Knupp M, Hintermann B. Medial displacement calcaneal osteotomy. *Tech Foot & Ankle* 2009; 8:85-90.
32. Sullivan JA, Miller WA. The relationship of the accessory navicular to the development of the flat foot. *Clin Orthop Relat Res* 1979; 144:233-237.
33. Weinraub GM, Heilala MA. Isolated talonavicular arthrodesis for adult onset flatfoot deformity/posterior tibial tendon dysfunction. *Clin Podiatr Med Surg* 2007; 24(4):745-752.
34. Wiewiorski M, Valderrabano V. Painful flatfoot deformity. *Acta Chir Orthop Traumatol Cech* 2011; 78(1):20-26.

Hallux Valgus

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Hallux valgus describes a pathological adduction of the great toe towards the second toe, which is a valgisation towards the body axis. The major deformity is located in the first metatarsophalangeal joint but the more proximal and the more distal joint influence the exact morphology of the hallux valgus deformity. Hallux valgus is the most common and the most relevant deformity of the forefoot. It is estimated that 2-4% of the population suffer of symptomatic hallux valgus deformity. Asymptomatic cases are probably even more common.

Etiology and Classification

Development of hallux valgus is a multifactorial development and details are still unknown and a matter of debate [3]. Several etiologies for hallux valgus deformity have been suggested. These can basically be divided into primary and secondary causes. Primary causes include hallux valgus deformity due to genetically predefined anatomical predispositions. This type of hallux valgus deformity often appears as juvenile hallux valgus. Predisposing pathologies such as increased valgus angle of the distal metatarsal articular angle (DMAA), varus alignment of the distal articular surface of the medial os cuneiforme or hallux valgus interphalangeus let hallux valgus appear in young life and often in patients with a positive family history.

Secondary causes include intrinsic and extrinsic factors. Structural abnormalities such as flatfoot with subsequent supination of the forefoot, TMT-I instability and loss of transverse arch of the forefoot with subsequent varisation of the first metatarsal can lead to the development

of a hallux valgus deformity. Extrinsic factors mostly include fashionable shoes with high heels and a much too narrow forefoot box which is forcing the first toe into a valgus position. However, many of these etiological factors are still a matter of debate with contradictory studies having been published in the last years [3]. All these factors lead into a common pathogenetical path where relative medialisation of the metatarsal head leads to failure of the medial restraints and subluxation of the head from the sesamoids which remain in their original position. Medialisation also leads to plantarisation and therefore loss of function of the abductor hallucis tendon with subsequent pronation of the first toe. The now unopposed adductor hallucis tendon further increases the hallux valgus morphology together with the lateralized flexor hallucis longus and brevis as well as extensor hallucis longus tendon which gain more and more adduction force with increasing hallux valgus angle due to their relative lateralization in relation to the metatarsal head. The medial MTP I capsule gets more and more stretched and pronation of the toe brings weaker more dorsal areas of the capsule towards medial, thus further weakening the medial restraints to valgus deviation. After some time the sesamoid crista gets more and more flattened and allow the metatarsal head to fully luxate from the sesamoids and increase the deformity [3, 8, 10].

For description and classification of hallux valgus several radiologic measurements are commonly used (Fig. 1). These are:

- 1. Hallux valgus angle: angle between the long axis of the first metatarsal and the proximal phalanx (<15°)
- 2. Intermetatarsal angle: angle between the long axis of the first and second metatarsal (<9°)
- 3. Distal metatarsal articular angle (DMAA): angle between the long axis of the first metatarsal and the articular surface at the metatarsal head.
- 4. Proximal phalangeal articular angle: angle between the long axis of the proximal phalanx and the articular surface



Fig.1 Radiographic assessment of hallux valgus deformity (angles according to the text)

Further, MTP I joint can be described as congruent and incongruent. Congruent hallux valgus is usually associated with a pathological DMAA. Localization of the sesamoids can be judged according to the position of the medial sesamoid in relation to the long axis of the first metatarsal. The severity of hallux valgus can be classified e.g. according to Coughlin (Table 1) [2]. Severity of the deformity dictates the treatment method that has to be chosen. If the severity of the deformity and the etiological factors are not sufficiently taken into account, recurrence after surgical treatment is very likely.

Table 1 Stages of hallux valgus deformity, modified according to Coughlin. [2]

	Hallux valgus angle	Intermetatarsal angle	Sesamoid subluxation
Mild	<20°	<12°	<50%
Moderate	20-40°	12-15°	50-75%
Severe	>40°	>15	>75%

Diagnosis

Diagnosis is made by clinical appearance and radiological work-up. Clinically, the patient presents with obvious valgus alignment of the MTP-I joint. Pain, calluses or even ulceration due to pressure on the prominent bones in inadequate shoe wear is possible. These are mostly located medially due to pronation of the great toe. Range of motion can be limited and painful. Hallux valgus leads to instability of the transverse arch of the forefoot with subsequent transfer metatarsalgia and associated hammer and claw toe formation. Examination is completed by a full set of standing x-rays (dorsoplantar and lateral foot and a.p. of the ankle). Clinical examination as well as x-rays needs not only to focus on the obvious deformity but rather identify predisposing and associated factors that influence treatment decision and risk for recurrence after surgery.

Treatment Options

Conservative treatment is an option in oligosymptomatic patients with mild to moderate hallux valgus. Some patients improve with accommodative shoe wear with large enough toe box and insoles with retrocapital and medial support of the foot arch [3]. The benefit of night splints is controversial and in our point of view should not be a treatment option. If conservative treatment fails or if there is a severe hallux valgus deformity, surgical treatment should be initiated. In literature, more than 150 surgical procedures have been described [12]. Despite specific technical features, hallux valgus surgery can be divided in 6 groups: medial and lateral soft tissue correction, phalangeal osteotomies, distal, shaft, and proximal metatarsal osteotomies and TMT I arthrodesis. In general, the more proximal the osteotomy is done the more powerful correction of hallux valgus angle can be achieved [4]. Distal metatarsal osteotomies have the additional advantage that multiplanar correction, i.e. for pathological DMAA is possible. Phalangeal osteotomies allow correction of not just the hallux valgus at MTP I joint but also hallux valgus interphalangeus. In about 5% of cases there is concomitant or causative TMT-I instability. In these cases correction of hallux valgus should be done by TMT-I arthrodesis since otherwise there's an increased risk of recurrence of the deformity. Soft tissue balancing by means of medial tightening and lateral release is part of every hallux valgus surgery. Re-centration of the sesamoids is only possible if sufficient lateral release has been achieved. However, pure soft tissue surgery is usually followed by recurrence of the deformity [6].

Postoperative treatment consists of immobilization and partial weight bearing for proximal shaft osteotomies or arthrodesis and full weight bearing in a stiff sole for all other patients.

Complications

Complication rate in literature is as high as 10-55% [9]. Beside general complications such as infection, thrombosis etc., specific complications of hallux valgus surgery include [4, 7, 12]:

- Recurrent hallux valgus: particularly if predisposing factors such as TMT-I instability, pathological DMAA have not been taken into consideration. Insufficient correction and recurrence also results

from choosing a technique that does not have enough corrective potential (i.e. Chevron osteotomy for severe hallux valgus).

- Over-correction , hallux varus : particularly if excessive lateral release is done.
- Non-/Malunion and shortening of the first ray, failure of osteosynthesis material (insufficient fixation, too much weight bearing for proximal osteotomies and TMT I arthrodesis)
- Arthrofibrosis
- Avascular necrosis, especially after Chevron type osteotomies (main vessels enter the bone through the capsule at the level of the base of the head)

Table 2 Surgical treatment methods

Method	Definition, Example	Indication
Medial exostosectomy	Resection of medial pseudo-exostosis and tightening of joint capsule	A part of every hallux valgus surgery, no isolated independent treatment option
Lateral release	Incision of the lateral capsule, intermetatarsal ligament, adductor hallucis tendon	Depending on severity of deformity: Capsule release > adductor release (cave over-release)
Osteotomy of the proximal phalanx	e.g. Akin osteotomy	Hallux valgus interphalangeus / pronatus, pathological proximal phalangeal articular angle ¹
Distal metatarsal osteotomies	e.g. Chevron osteotomy, biplanar distal osteotomies	Mild to moderate hallux valgus (intermetatarsal angle <15°) ⁴
Shaft osteotomies	e.g. Scarf, Ludloff osteotomies	Severe hallux valgus (intermetatarsal angle >15°)
TMT I arthrodesis	Lapidus arthrodesis	Moderate to severe hallux valgus with concomitant TMT I instability, ^{5,11}



Fig. 3 Chevron and Akin osteotomy. 31-year old female with moderate hallux valgus deformity.

References

1. Barouk LS, Barouk P, Baudet B, Toullec E. The great toe proximal phalanx osteotomy: the final step of the bunionectomy. *Foot Ankle Clin* 2005; 10(1):141-155.
2. Coughlin MJ. Hallux valgus. *Instr Course Lect* 1997; 46:357-391.
3. Easley ME, Trnka HJ. Current concepts review: hallux valgus part 1: pathomechanics, clinical assessment, and nonoperative management. *Foot Ankle Int* 2007; 28(5):654-659.
4. Easley ME, Trnka HJ. Current concepts review: hallux valgus part 2: operative treatment. *Foot Ankle Int* 2007; 28(6):748-758.
5. Espinosa N, Wirth SH. Tarsometatarsal arthrodesis for management of unstable first ray and failed bunion surgery. *Foot Ankle Clin* 2011; 16(1):21-34.
6. Kitaoka HB, Franco MG, Weaver AL, Ilstrup DM. Simple bunionectomy with medial capsulorrhaphy. *Foot Ankle* 1991; 12(2):86-91.
7. Lehman DE. Salvage of complications of hallux valgus surgery. *Foot Ankle Clin* 2003; 8(1):15-35.
8. Perera AM, Mason L, Stephens MM. The pathogenesis of hallux valgus. *J Bone Joint Surg Am* 2011; 93(17):1650-1661.
9. Scioli MW. Complications of hallux valgus surgery and subsequent treatment options. *Foot Ankle Clin* 1997; 2:719-739.
10. Stephens MM. Pathogenesis of hallux valgus. *Eur J Foot Ankle Surg* 1994; 1:7-10.
11. Trnka HJ, Hofstatter S. [The modified Lapidus arthrodesis]. *Orthopade* 2005; 34(8):735-741.
12. Wulker N. [Failures of hallux valgus surgery]. *Orthopade* 2011; 40(5):384-391.

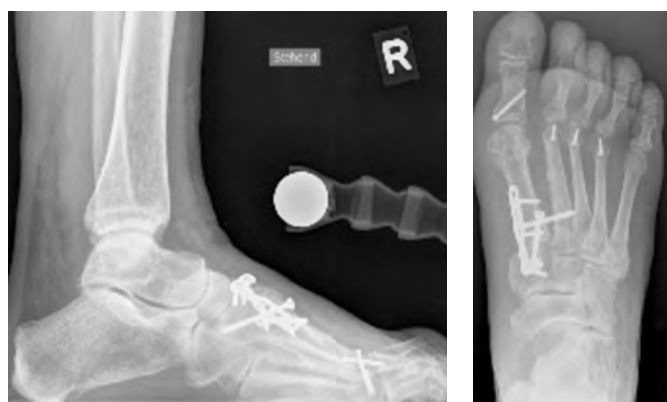


Fig. 2 Lapidus arthrodesis. 56-year female with incongruent hallux valgus deformity, TMT-I instability and metatarsalgia MTP-II/IV



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Shoulder degenerative disorders

Degenerative conditions of the shoulder are common and often result in pain and limitation of function. The tendons of the rotatorcuff, particularly the supraspinatus, are affected by extrinsic and intrinsic factors and tears of the rotatorcuff are a common finding among patients with shoulderpain. In elderly patients conservative treatment may lead to satisfactory outcome, but in younger patients symptomatic tears of the rotatorcuff often need repair. The optimal surgical technique is not defined, but the trend is to use a double row suture configuration. Massive cuff tears may lead to osteoarthritis in the joint and loss of function. Reverse arthroplasty can restore function and give pain relief in these severe cases. Shoulder arthroplasty can give excellent function in patient with osteoarthritis and the number of procedures is increasing. Total shoulder arthroplasty gives better outcome than hemiarthroplasty. Symptomatic osteoarthritis of the AC- or SC joint can often be managed with antiinflammatory medications or steroid injections. Lateral clavicle resection may be necessary in patients with AC-joint osteoarthritis, but such procedure is rarely performed in the SC-joint.

Introduction

Shoulder problems, particularly pain, are very common and it is said that up to 50% of the population experience episodes of shoulder pain every year. The shoulder is the most mobile joint in the body. There are 4 joints that is considered to be part of the shoulder i.e. gleno-humeral, acromio-clavicular, sterno-clavicular and scapula-thoracic joints. Most of the movement is taking place in the glenohumeral and scapulo-thoracic joints. The shoulder is surrounded by two layer of musculature. The deeper layer is called the rotatorcuff and consists of 4 muscles (subscapularis, supraspinatus, infraspinatus and teres minor) and the tendon of the long head of the biceps, which runs in the bicipital groove entering the joint in the interval between the subscapularis and supraspinatus tendons. The superficial layer is the deltoid muscle. Between these layers there is the subacromial bursa. Degenerative conditions resulting in clinical symptoms occurs primarily in the joints and rotatorcuff. Correct diagnosis and treatment depend on careful patient history and clinical examination followed by further exams like radiographs, ultrasound and MRI.

Clinical evaluation

Patient history is very important and can usually give the examining doctor a preliminary diagnosis. Visual inspection is done looking from the front and back to look for asymmetries and atrophy. Four areas are discussed i.e. pain, range of motion, stability and strength, followed by specific testing of for example the stability and function of the rotatorcuff. Pain should be graded on a visual analogue scale from 0 to

10. Pain at rest and pain during activity should be graded separately. Patient history is followed by a careful clinical examination. Scapulo-thoracic rhythm is evaluated by looking from the back during abduction. Stability of the scapula is checked and palpation is done to look for tenderness. Active and passive range of motion should be recorded for forward elevation, abduction, external and internal rotation. Strength of the rotatorcuff muscles can easily be tested by having the patients pushing against resistance in external, internal rotation and abduction. Internal rotation test several muscles (subscapularis, pectoralis major, latissimus dorsi) and in order to specifically test the subscapularis muscle the "lift-off" test is performed. This testing is best done standing posterior of the patient. Subacromial pain syndrome can be evaluated by performing Hawkins test or empty can test. Stability is tested with various test. AC-joint pain can be tested by cross-body motion compressing the AC-joint.

Plain radiographs should always be performed to look for bony abnormalities and osteoarthritic changes. MRI is the best tool to evaluate the soft tissue, but ultrasound and CT scan also give valuable information of the soft tissue.

It can be difficult to determine the source of pain around the shoulder. A helpful tool is to inject local anesthesia into for example the subacromial space or AC-joint and evaluate whether the pain is eliminated or reduced. It is also important to examine the neck, since pathologic processes in the cervical spine can result in shoulder pain.

Degenerative conditions of the rotator cuff

Pathology of the rotatorcuff tendons and subacromial bursa is a common source of pain and functional disability. The etiology of tendinopathy and rotator tendon failure is multifactorial and extrinsic, intrinsic and environmental factors have been studied. The subacromial bursa is innervated by the suprascapular and pectoral nerve (C5-C6) and mechanoreceptors have been identified [17].

A rotatorcuff tear can be degenerative, traumatic or a combination of these. Traumatic rotator cuff tears will not be discussed here. Neer believed that the majority of degenerative rotator cuff tears were caused by mechanical irritation from the anterior-inferior part of the acromion onto the superior aspect of the rotatorcuff [11]. This would explain why the majority of rotatorcuff tears occur in the supraspinatus. An acromioplasty was recommended in patients with subacromial pain to avoid having progression to rotatorcuff tears. The results of acromioplasty have been very good with up to 80-85% patients reporting pain relief [11]. These patients with pain during elevation have been shown to have high concentration of inflammatory, pain-mediating proteins in the bursa [17]. Thus the bursa is an important factor in shoulder pain in patients with impingement. This is further supported by the finding of Budoff et al that outcome for patients having surgery for impingement

syndrome were the same after bursectomy with or without acromioplasty [17]. The concept of mechanical impingement between acromion and the rotatorcuff has therefore been challenged and the removal of the bursa may be the most important part of the procedure. Weakness of the rotatorcuff leads to superior migration of the humeral head increasing the pressure in the subacromial space. To counteract this migration a specific rehabilitation program for patients with impingement has been recommended [11]. Strengthening excercises of the anterior and posterior rotatorcuff can counteract the tendency for superior migration and relieve pain.

Intrinsic changes in the rotatorcuff tendons due to overload affecting the collagen, proteoglycans, vascularity and cells have been described. Hashimoto et al postulated that intrinsic degeneration is the primary cause of rotator cuff tears [17]. Animal studies have shown that rats exposed to an overuse running program developed the same tendon changes seen in human tendinopathy resulting in weakening of the tendon.

The concensus today is that degenerative rotator cuff disease is the result of intrinsic and extrinsic factors and may result in structural failure of the tendons. Such a tear is defined as a partial (bursal side, intratendinous, intraarticular) or a full thickness. This degenerative process is not always symptomatic. These degenerative tears are usually located in the supraspinatus and initiate in a region about a centimeter posterior of the biceps tendon [13]. An asymptomatic rotator cuff tear may become symptomatic [18]. Mall et al followed 195 patients with an asymptomatic rotatorcuff tear [18]. Within 2 years 44 patients developed pain and 18% of the full thickness tears had progressed in size and 40% of partial-thickness tears had progressed to a full thickness tear. Fucentese et al studied symptomatic full-thickness supraspinatus tears in 24 patients who declined surgery [7]. At a mean follow up of 3,5 years and reported no tear size progression. 17 patients were satisfied or very satisfied with their shoulder function.

Rotator tendon tears lead to muscular degeneration with fatty infiltration and atrophy (Fig. 1). The degree of atrophy and fatty infiltration has been classified by Goutallier and can be done using CT-scan or MRI (Table 1) [8,10]. The exact mechanism for the muscular atrophy and fatty infiltration is not known [12,15]. Rotatorcuff tears changes the extra-cellular matrix which surrounds the myofibers and these changes may lead to fibrosis and atrophy [15]. When the tendon is torn and retracted the pennation angle changes thereby increasing the space between the muscle fibers where fat could infiltrate [26]. Other authors have postulated that stem cell populations within the muscle differentiate into adipogenic cells, maybe as a result of nerve injury to the suprascapular nerve [12,15]. In a group of 26 patients with massive rotator cuff tears Costouros et al found that 14 had evidence of suprascapular nerve pathology [4]. Six of seven patients that underwent repair of the cufftear showed partial or full recovery of the nervepalsy. Thus, there are several factors that may contribute to muscular atrophy and fatty infiltration in patients with rotator cuff tears. Furthermore, such changes have a negative effect on clinical outcome after surgical repair. The fatty infiltration and atrophy have been regarded as irreversible, but Yamaguchi reported improvement in both degree of atrophy and fatty infiltration after successful repair of massive rotator cuff tears [25].

Table 1 Classification of fatty degeneration of the rotator cuff muscles

Goutailier Stage (CT)	Degree of fat	Fuchs stage (MRI)
0	Normal muscle	I
I	Some fat seen	I
II	More muscle than fat	II
III	Equal muscle and fat	III
IV	More fat than muscle	III

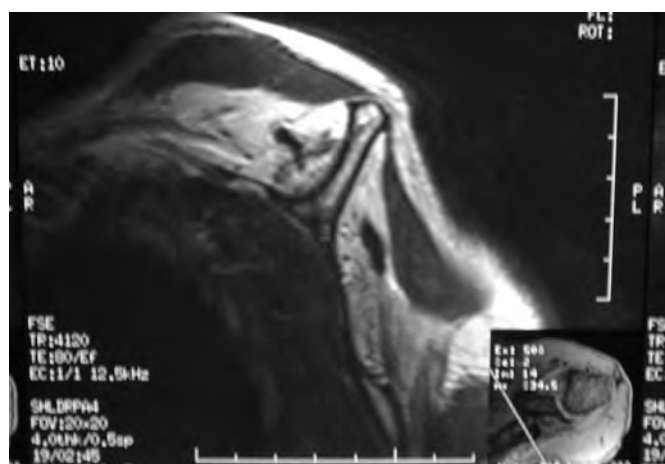


Fig. 1 Severe atrophy of supraspinatus, infraspinatus and teres minor muscles in a patient with a chronic rotator cuff tear

Treatment

In patients with subacromial pain without significant rotatorcuff tears a specific rehabilitation program is initiated, sometimes in combination with a steroid injection. If that is not successful, an arthroscopic bursectomy with or without an acromioplasty is performed (depending on the degree of spurformation on the acromion). The treatment of a partial rotatorcuff tear is more controversial and debridement and/or repair have been advocated.

Repair is usually recommended in a degenerative symptomatic full thickness rotator cuff, particularly in younger patients, where strength is important. However, non operative treatment in patients with a small supraspinatus tear may lead to satisfactory outcome [7]. Today there is an intense debate regarding optimal repair technique. Various suture configurations with single or double row techniques have been advocated. In a review 2010 Saridakis concluded that the present literature suggest a better structural healing after an arthroscopic double-row repair compared to single-row [23]. However, no difference in functional outcomes has been reported, except for maybe in massive rotator cuff tears [5,23]. During recent years there have been attempts to improve the structural results after cuff-repair by augmenting the repair with different biological scaffolds [3, 22]. There are several different types on the market and they have different characteristics. There is little evidence supporting the use of these devices and further research is needed before the use of these scaffolds can be recommended. Patients with significant muscular atrophy and fatty infiltration have a poor prognosis and a high retear-rate. Further research is needed to understand the degenerative process in the rotatorcuff and the etiology of atrophy and fatty infiltration. This may lead to an optimal structural repair technique combined with biological treatment to restore muscular

function.

Chronic massive rotatorcuff tears results in superior migration of the humeral head and osteoarthritis in the glenohumeral joint. This is often called cuff-tear arthropathy, even though that term was first used by Neer and Fukuda in patients with a severe destruction of the rotatorcuff and glenohumeral joint [20]. The combination of massive rotatorcuff tear and degenerative changes in the joint is a challenge to treat. It may lead to pain and poor active range of motion (pseudoparalysis). A reverse arthroplasty (Delta arthroplasty) can restore function and relieve pain in these patients (Fig. 2)[6]. The modern design of reverse arthroplasty is based on the idea of Grammont [9]. It has a large ball on the glenoid side and a socket on the humeral side with a non anatomical inclination angle. The center of rotation is medialized, which increases the lever arm for the deltoid muscle. Furthermore the center of rotation is fixed in a more inferior position thereby restoring the length of the deltoid muscle. The biomechanical balance is restored. In the first reported series a high complication rate was reported, but with modern technique and new implants the rate of complication has significantly been reduced [6]. The reverse arthroplasty cannot restore external rotation and therefore it has been recommended to combine the reverse arthroplasty with a latissimus dorsi transfer if there is lack of functional external rotation as well as pseudoparalysis.



Fig. 2 Pre- and postop X-rays of a patient with cufftear arthropathy treated with a Delta Xtend reverse arthroplasty (preop 2A, postop 2B)

Glenohumeral osteoarthritis (OA)

In autopsy studies the incidence of osteoarthritis in the shoulder is 10–20%. It can be primary osteoarthritis or secondary to infection, fracture, traumatic instability, avascular necrosis of the humeral head or other significant trauma [19]. Levy et al found a high incidence of propionibacterium acnes in nonoperated shoulders undergoing shoulder arthroplasty suggesting this gram-positive anaerobic bacillus may play a role in the development of osteoarthritis [16]. In primary cases inferior osteophytes are often seen and can be quite large. In many cases a posterior subluxation of the head is present with posterior wear and bone loss of the glenoid. Walch has classified these changes into glenoid type A, B and C. Type A is characterized by central erosion with no posterior subluxation of the humeral head, Type B has posterior subluxation of the head without (B1) or with bone loss (B2), and type C is a severe retroversion of the glenoid surface (dysplasia) (Fig. 3) [24]. Walch et al found that 59% was type A, 32 % was B and 9% had a type C glenoid in a series of 113 patients.

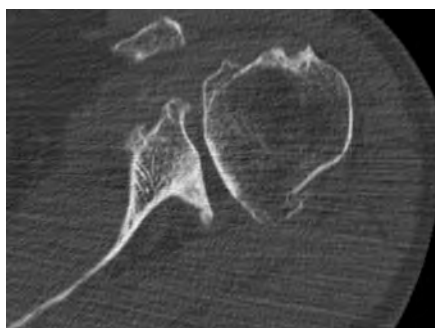


Fig. 3 CT scan of a B2 glenoid with posterior erosion

Osteoarthritis leads to restriction in range of motion, particularly external rotation, and various degrees of pain. If non operative treatment is unsatisfactory surgery is recommended. Shoulder arthroplasty with modern implants and surgical techniques have very good outcome with excellent pain relief and range of motion (Fig 4). There has been an intense debate whether to perform a hemiarthroplasty or total shoulder arthroplasty. There is increasing evidence that total shoulder arthroplasty results in better pain relief and function compared to hemiarthroplasty [21]. The traditional humeral component has a stem, but now the trend is to use more resurfacing or metaphyseal fixed implants. On the glenoid side a cemented all polyethylene component is still the golden standard. The trend is to use more pegged implants compared to the traditional keel design. However, metal backed non cemented components are being tested and promising midterm results have been reported [1]. Soft tissue balancing is important and particularly subscapularis management is crucial. To minimize the risk for postoperative subscapularis failure an osteotomy of the lesser tuberosity has been advocated [2]. However, traditional tenotomy with side to side repair is still being widely used. Further studies are needed to define the optimal technique. Lafosse has developed a technique where the total shoulder arthroplasty is done through the rotator interval without taking down any part of the rotatorcuff allowing for immediate mobilization [14].

To optimize function anatomical reconstruction is important. Therefore a preoperative CT scan is recommended in order to classify the glenoid. If there is posterior bone loss reaming down the anterior high side can be done or a posterior bonegraft used. Recently a posterior augmented glenoid component was designed and now being tested clinically. The CT scan can also give important information regarding the rotator cuff muscles. If there is significant cuff disease, particularly atrophy and fatty infiltration, a reverse arthroplasty may be indicated. The author also uses a reverse arthroplasty in severe B2 and C glenoid in elderly patients. In the Swedish registry for shoulder arthroplasty an annual increase of the number of arthroplasty performed has been seen the last 10 years. In 2011 one arthroplasty/ 9000 inhabitants was recorded. The trend is that more total shoulder and reverse arthroplasty are being done and less resurfacing and hemiarthroplasty.



Fig. 4 Preoperative radiograph (4A) showing osteoarthritis with narrowing of the joint space and typical inferior osteophytes of the humeral head and postoperative image of a total shoulder arthroplasty (4B)

Acromioclavicular joint (AC joint)

It is very common to find osteoarthritic changes in this joint on radiographs, but often it is asymptomatic due to limited motion. Inside the joint there is a fibrocartilaginous disc that can degenerate and cause pain and crepitus. Typical symptoms are pain during overhead activities and cross-body maneuvers and local tenderness over the joint can be seen. If clinical examination indicates pain coming from the AC joint a corticosteroid injection can often relieve symptoms. If symptoms recur a lateral clavicle resection (5–10 mm) open or arthroscopic can be performed.

Sternoclavicular joint (SC joint)

Osteoarthritic changes can cause pain and crepitus. Nonoperative treatment with anti-inflammatory agents or corticosteroid injections is usually enough to achieve pain relief. However, in rare cases a medial clavicle resection is necessary. When such a procedure is performed it is important to keep the ligaments stabilizing the joint to avoid postoperative instability. Patients with rheumatoid disorders often have degenerative changes in the SC-joint that may be symptomatic.

References

1. Castagna A, Randelli M, Garofalo R, et al (2010) Mid-term results of a metal-backed glenoid component in total shoulder replacement. *J Bone Joint Surg* 92:1410–1415.
2. Gerber C, Yian EH, Pfirrmann AW, et al (2005) Subscapularis muscle function and structure after total shoulder replacement with lesser tuberosity osteotomy and repair. *J Bone Joint Surg* 87-A:1739–174
3. Cheung EV, Silverio L, Sperling JW (2010) Strategies in biologic augmentation of rotator cuff repair. *Clin Orthop Rel Res* 468:1476–1484.
4. Costouros JG, Porramatikul M, Lie DT et al. (2007) Reversal of suprascapular neuropathy following arthroscopic repair of massive supraspinatus and infraspinatus rotator cuff tears. *Arthroscopy* 23:1152–1161.
5. DeHaan AM, Axelrad TW, Kaye E et al. (2012) Does double-row rotator cuff repair improve functional outcome of patients compared with single-row technique? *Am J Sports Med* 40:1176–1185.
6. Ekellund A (2009) Reverse shoulder arthroplasty. *Shoulder Elbow* 1:68–75.
7. Fucentese SF, von Roll AL, Pfirrmann CWA et al. (2012) Evolution of nonoperatively treated symptomatic isolated full-thickness supraspinatus tears. *J Bone Joint Surg* 94:801–808.
8. Fuchs B, Weishaupt D, Zanetti M et al. (1999) Fatty degeneration of the muscles of the rotator cuff: assessment by computed tomography versus magnetic resonance imaging. *S Shoulder Elbow Surg* 8:599–605.
9. Grammont PM, Baulot E (1993) Delta shoulder prosthesis for rotator cuff rupture. *Orthopaedics* 16:65–8.
10. Goutallier D, Postel JM, Bernageau J et al. (1994) Fatty muscle degeneration in cuff ruptures. Pre- and postoperative evaluation by CT scan. *Clin Orthop Rel Res* 304:78–83.
11. Harrison AK, Flatow EL (2011) Subacromial impingement syndrome. *JAAOS* 19:701–708.
12. Kang JR, Gupta R (2012) Mechanisms of fatty degeneration in massive rotatorcuff tears. *J Shoulder Elbow Surg* 21:175–180.
13. Kim HM, Dahiya N, Teefey SA et al (2010) Location and initiation of degenerative rotator cuff tears. *J Bone Joint Surg* 92:1088–1096.
14. Lafosse L, Schnaser E, Haag M, et al (2009) Primary total shoulder arthroplasty performed entirely thru the rotator interval: technique and minimum two-year outcomes. *J Shoulder Elbow Surg* 18:864–73.
15. Laron D, Samagh SP, Liu X et al. (2012) Muscle degeneration in rotator cuff tears. *J Shoulder Elbow Surg* 21:164–174.
16. Levy O, Iyer S, Atoun E et al. (2012) *Propionibacterium acnes*: an underestimated etiology in the pathogenesis of osteoarthritis. *J Shoulder Elbow Surg* In press.
17. Lewis JS (2012) Rotator cuff tendinopathy. *Br J Sports Med* 43:236–241.
18. Mall NA, Kim HM, Keener JD et al. (2010) Symptomatic progression of asymptomatic rotator cuff tears. *J Bone Joint Surg* 92:2623–2633
19. Matsen FA, Clinton J, Rockwood CA et al. Glenohumeral arthritis and its management. In Rockwood CA, Matsen FA, Wirth MA, Lippitt SB (2009) *The Shoulder*. Saunders Elsevier Philadelphia.
20. Neer CS, Craig EV, Fukuda H (1983) Cuff-tear arthropathy. *J Bone Joint Surg* 65:1232–1244.
21. Radnay CS, Setter KJ, Levine WN, et al (2007) Total shoulder replacement compared with humeral head replacement for the treatment of primary glenohumeral osteoarthritis: A systematic review. *J Shoulder Elbow Surg* 16:396–402.
22. Richetti ET, Aurora A, Eng D et al. (2012) Scaffold devices for rotator cuff repair. *J Shoulder Elbow Surg* 21:251–265.
23. Saridakis P, Jones G (2010) Outcomes of single-row and double-row arthroscopic cuff repair: A systematic review. *J Bone Joint Surg* 92:732–742.
24. Walch G, Badet R, Boulahia A, et al (1999) Morphologic study of the glenoid in primary glenohumeral osteoarthritis. *J Arthroplasty* 14:756–60.
25. Yamaguchi H, Suenaga N, Oizumi N et al. (2012) Will preoperative atrophy and fatty degeneration of the shoulder muscles improve after rotator cuff repair in patients with massive rotator cuff tears? *Adv Orthop Epub* Jan 12.
26. Zuo J, Sano J, Itoi E (2012) Changes in pennation angle in rotator cuff muscles with torn tendons. *J Orthop Sci* 17:58–63.



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Denenerative disorders of the elbow

Osteoarthritis of the elbow

Primary osteoarthritis (OA) of the elbow is relatively rare, affecting approximately 2% of the population. The average age of patients at initial presentation is 50 years old, and the male-to-female ratio is 4:1. The etiology is unclear, but primary elbow OA is usually associated with a history of heavy use of the arm, such as is the case with manual workers. Although the elbow is a non-weight-bearing joint, studies have reported forces up to 2 times body weight during motions commonly seen in occupational duties such as lifting, moving, and placing 2kg weights. Therefore, individuals who perform strenuous activities, or require the use of a wheelchair, may be expected to produce large loads across the elbow. Secondary causes of elbow OA include trauma, osteochondritis dissecans, synovial chondromatosis, and valgus extension overload. Patients under the age of forty often have a history of a traumatic event.

Symptoms in the involved elbow include loss of terminal extension, painful catching or clicking, or locking of the joint. Pain is typically noted at the terminal range of both flexion and extension. Patients report that it is painful to carry heavy objects at the side of the body with the elbow in extension. The arc of motion is restricted by the presence of osteophytes, as well as secondary to capsular contracture. Night pain is not typical, and forearm rotation is relatively well-preserved. Ulnar neuropathy is present in up to 50% of patients. The degree of pain and disability varies among patients and is affected by functional demand.

Radiographs show osteophytes involving the coronoid process, coronoid fossa, olecranon, and olecranon fossa. Preservation of the joint space at the ulnohumeral and radiocapitellar joints is common. Loose bodies may be seen, but up to 30% of them are not detected on plain radiographs.



Conservative treatment includes rest, activity modification, and non-steroidal anti-inflammatory medication (NSAID). Surgery is indicated for those patients who fail to respond to nonoperative treatment, particularly when loss of motion interferes with activities of daily living (loss of

extension >30°), when there is painful locking or clicking, or ulnar nerve symptoms are present. Current treatment options include (1) classic open procedure, (2) ulnohumeral arthroplasty, (3) arthroscopic osteocapsular arthroplasty, and rarely (4) total elbow replacement.

The classic Outerbridge-Kashiwagi (OK) procedure includes a posterior approach to the elbow with a triceps split, removal of the tip of the olecranon, and osteophyte removal through olecranon fossa trephination. Limitations of this procedure are an incomplete anterior release and inability for osteophyte removal anteriorly. Any flexion contracture can be more reliably addressed via an ulnohumeral arthroplasty, which is a modification of the OK procedure including triceps elevation rather than splitting, and a lateral column procedure to perform an anterior capsule release. Ulnar nerve decompression is advocated if preoperative symptoms are present, when preoperative flexion is <100°, or when a gain of 30°–40° of flexion is expected. Postoperative complications include ulnar neuropathy, and recurrence of a flexion contracture or osteophytes. Arthroscopic débridement appears to give good results, although currently there is a lack of long term follow-up.

Total elbow arthroplasty is only indicated for patients older than 65 years for whom other interventions have failed, and who are willing to accept low activity levels with regards to the elbow.

The rheumatoid elbow

Rheumatoid arthritis affects 1–2% of the population and involves the elbow joint in 20–50% of patients. The great majority of patients also have wrist and shoulder involvement. Initially, patients present with a painful stiff elbow. Secondary changes may develop over time, leading to a fixed flexion contracture, pain throughout the range of motion, instability due to soft tissue deterioration, and ulnar and radial nerve neuropathies.

Anteroposterior and lateral radiographs of the elbow are needed to stage the disease according to the classification of Larsen or Mayo Clinic, which are based upon the radiographic degree of joint involvement as well as clinical symptoms. Radiographic signs include periarticular erosions, symmetric joint space narrowing, osteopenia, subchondral plate erosions, and finally gross destruction of most or all articular architecture.

Nonsurgical management is appropriate for patients with early disease, and includes physical therapy, resting splints, NSAID, and occasional corticosteroid injections. Surgical options include synovectomy (Larsen stages 1 to 2), radial head excision, and total elbow arthroplasty (Larsen stages 3 to 4). Open synovectomy is usually performed via a lateral

approach. The most common complication is recurrence of pain over time. Arthroscopic synovectomy is less invasive but technically demanding and carries the risk of neurovascular injury. Radial head excision is controversial. Rheumatoid arthritis is the primary indication for total elbow arthroplasty. Due to bone loss and soft tissue involvement, semi-constrained implants are the prostheses of choice. Complication rates may be as high as 50% and patient selection is very critical. Age less than 65 years old is only a relative contraindication and total elbow arthroplasty can be performed in low-demand patients with severe disease.



Synovial chondromatosis

Synovial chondromatosis is a rare benign pathology of the synovium in which cartilaginous material is formed within synovial tissue. The cartilaginous nodules may become intraarticular loose bodies or undergo ossification, described as osteochondromatosis. The symptoms are non-specific, and include pain on exertion, swelling, locking episodes, and flexion/extension deficit. It is a monoarticular process most often occurring in middle-aged men, with the knee being the most frequently affected joint. With standard radiographs it may be difficult to diagnose intraarticular chondromatosis when there is no calcification of the cartilage nodules, and magnetic resonance imaging (MRI) or computed tomography (CT) may be helpful. Treatment consists of open or arthroscopic removal of loose bodies and partial synovectomy. Additional procedures may be necessary according to the local status of the elbow, such as removal of osteophytes, anterior capsulotomy, etc. Recurrence rates between 3-22% have been reported after surgery. Chondromatosis of the elbow frequently leads to secondary osteoarthritis.



Distal biceps tendon rupture

This usually affects men between 40 and 60 years of age, and generally occurs in the dominant extremity. The mechanism is a single traumatic event in heavy workers or weight lifters. Patients report pain and weakness in flexion and supination. Clinical examination reveals an absent distal biceps tendon, with proximal retraction of the biceps muscle. Most often the diagnosis is clear, but in cases of partial tendon rupture these signs may be absent and MRI is useful. Conservative treatment leads to poor results in terms of flexion and supination strength, and is recommended only for elderly patients with low functional demand. Surgery involves anatomic repair of the tendon to the radial tuberosity. Controversies exist regarding single versus two-incision technique, and method of fixation (anchors, endobutton, transosseous tunnel). There is a higher risk of nerve lesions with a single incision, and a higher risk of heterotopic ossification with two incisions. We use the Mayo modified Boyd and Anderson two incision technique with transosseous tunnels.



A transverse incision is made at the anterior aspect of the elbow. The tendon is identified, prepared at its end to fit into the tuberosity, and two #6 Ethibond sutures are placed through its substance. With the forearm at 90° of flexion and full pronation, a curved clamp is introduced into the empty bicipital canal and directed to the ulnar side of the radius, curving it away from the ulna, until it emerges through the extensor muscles. A second dorsolateral incision is made slightly anterior to the tip of the clamp. A muscle splitting approach is used to expose the biceps tuberosity which lessens the risk of heterotopic ossification. The ulna is never exposed to minimize the risk of a radioulnar synostosis. A 15x5 mm excavation with two 2.5 mm holes is performed, and the tendon is repaired to the tuberosity.

After surgery, the arm is placed in a splint for 3 weeks and then range of motion (ROM) exercises are started aiming at full motion by 6 weeks. Nonstrenuous activity is permitted at 3 months and heavy activities after 4 months.



Complications include (1) lateral antebrachial cutaneous nerve injury (usually self-resolving), (2) radial nerve lesion (decreased by using two-incisions), (3) heterotopic ossification and radio-ulnar synostosis (higher risk in two-incisions), and (4) re-rupture which is uncommon. Overall, clinical results are good to excellent, with 80-90% recovery in strength and an almost complete ROM.

Septic olecranon bursitis

This is a common condition that requires prompt recognition and treatment in order to avoid potentially life threatening complications. Septic bursitis generally arises following blunt trauma or a superficial wound. Clinically there is local tenderness over the bursa, but the range of motion of the elbow is usually full and pain-free. The diagnosis is based upon clinical evaluation, with standard laboratory findings including elevated white cell count and high C-reactive protein levels. The organisms that are found are generally staphylococci or streptococci. Treatment consists of incision and drainage with removal of the bursa. The wound is left open, and after several days secondary closure can be performed. During the treatment period appropriate antibiotics are administered intravenously.



References

1. Keener JD. Controversies in the surgical treatment of distal biceps tendon ruptures: single versus double-incision repairs. *J Shoulder Elbow Surg.* 2011;20:113-125.
2. Miyamoto RG, Elser F, Millett PJ. Distal biceps tendon injuries. *JBJS Am.* 2010;92:2128-2138.
3. Sutton KM, Dodds SD, Ahmad CS, et al. Surgical treatment of distal biceps rupture. *J Am Acad Orthop Surg.* 2010;18:139-148.
4. Peeters T, Ching-Soon NG, Jansen N, et al. Functional outcome after repair of distal biceps tendon ruptures using the endobutton technique. *J Shoulder Elbow Surg.* 2009;18:283-287.
5. Lieberman JR. AAOS Comprehensive orthopedic review, 2009.
6. Gregory DG, Leesa MG. Current concept review. Management of elbow osteoarthritis. *J Bone Joint Surg Am.* 2006;88:421-430.
7. Cheung EV, Adams R, Morrey BF. Primary osteoarthritis of the elbow: current treatment options. *J Am Acad Orthop Surg.* 2008;16:77-88.
8. Vingerhoeds B, Degreef I, De Smet L. Débridement arthroplasty for osteoarthritis of the elbow (Outerbridge-Kashiwagi procedure). *Acta orthop Belg.* 2004;70:306-310.
9. Kozak TK, Adams RA, Morrey BF. Total elbow arthroplasty in primary osteoarthritis of the elbow. *J Arthroplasty.* 1998;13:837-842.
10. Aldridge JM, Lightdale NR, Mallon WJ et al. Total elbow arthroplasty with the Coonrad/Coonrad-Morrey prosthesis. A 10- to 31-year survival analysis. *J Bone Joint Surg Br.* 2006;88:509-514.
11. Kauffman JJ, Chen AL, Stuchin S, et al. Surgical management of the rheumatoid elbow. *J Am Acad Orthop Surg.* 2003;11:100-108.
12. Mansat P. Surgical treatment of the rheumatoid elbow. *J Bone Spine.* 2001;68:198-210.
13. Jazrawi LM, Ong B, Jazrawi BS, et al. Synovial chondromatosis of the elbow. A case report and literature review. *Am J of Orthopedics.* 2001;223-224.
14. Mueller Th, Barthel Th, Cramer A, et al. Primary synovial chondromatosis of the elbow. *J Shoulder Elbow Surg.* 2000;9:319-322.
15. Kamineni S, O'Driscoll SW, Morrey BF et al. Synovial osteochondromatosis of the elbow. *J Bone Joint Surg Br.* 2002;84:961-966.
16. Christensen JH, Poulsen JO. Synovial chondromatosis. *Acta Orthop Scand.* 1975;46:919-925.
17. Hoffmeyer P, Chalmers A, Price GE. Septic olecranon bursitis in a general hospital population. *CMA Journal.* 1980;122:874-876.



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ACL, PCL, Collaterals

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Anterior cruciate ligament (ACL)

ACL is the primary restraint to anterior knee laxity. ACL tear is the most frequent lesion in sports traumatology. Indeed 85% of ACL tears occur during sports as soccer, ski, rugby or other sports implying knee pivoting as tennis, handball, basketball.

Anamnesis

Patient often feel and sometimes hear a painful «crack» during trauma, followed by knee swelling which is actually an hemarthrosis. ACL tear may be isolated: it is often due to a non-contact trauma (active knee hyperextension during a shoot for example); or ACL tear may be combined to other knee lesions, which occurs during contact trauma (for example combined knee valgus-flexion-external rotation leading to the «unhappy triad» of O'Donoghue = ACL tear + medial collateral ligament tear + external meniscus tear). Combined lesions should always be searched in case of ACL tear.

Clinical exam

Clinical diagnosis can be done with three tests:

- the **Lachmann-Trillat test**: tibial anterior drawer at 30° of knee flexion. The end point is evaluated and may be a «soft end point» meaning a complete ACL tear, a «hard end point» meaning an intact ACL, or a «delayed hard end point» meaning a partial ACL tear (only one of the two bundles of ACL is torn). The differential laxity with the controlateral knee can be evaluated with an arthrometer, for example KT-1000®. More than 3mm difference is considered abnormal.
- the **anterior drawer test**: tibial anterior drawer at 90° of knee flexion.
- the **pivot-shift test**: the patient must relax to prevent muscle guarding and the physician must be familiar with this test which reproduces event that occurs when knee gives way because of loss of ACL. The physician applies a combined valgus - tibial anterior drawer and internal rotation, and progressively flex the knee. At 30-40°, the iliotibial band reduces the tibial anterior drawer and a sudden jerk is felt, which confirms ACL tear.

Imagery

MRI is the gold standard radiographic exam (Fig. 1). However, standard radiographs should always be prescribed because indirect signs of ACL rupture as the Segond fracture can be seen (Fig. 2). They also can help and inform on the ACL biomechanical properties. Anterior tibial translation can be measured and compared to the controlateral knee on standard profile weightbearing radiographs, or on stress radiographs (Telos®) with a 50N anterior tibial drawer.



Fig. 1 ACL rupture.



Fig. 2 "Segond" fracture.

Treatment

Due to its poor vascularisation, ACL has a poor ability to heal and has to be reconstructed surgically most of the time. Conservative treatment is an option for sedentary patients and for patients who don't complain of knee instability: a program of rehabilitation is necessary to recover knee range of motion, to strengthen thigh muscles and improve proprioception. However, for patients who want to get back to sports and/or who complain of knee instability with giving way episodes, ACL reconstruction is mandatory to prevent meniscus tear and early osteoarthritis. The goal of this surgery is to reconstruct the ACL anatomically by positioning the graft inside the knee joint as a native

ACL. Since few years, some surgeons reconstruct the two bundles of ACL (anteromedial and posterolateral bundles) in order to get the most anatomical reconstructed ACL. Long term clinical results did not show a clear benefit of this technique, except for a lower rate of persisting pivo-shift. For single bundle ACL reconstruction, various types of ACL grafts can be used:

- central third of the patellar tendon also called «bone-tendon-bone» which has a bone plug of anterior tibial tuberosity on one end and a bone plug of patella on the other end
- central third of the quadriceps tendon with a bone plug of patella
- hamstring tendons (gracilis and semi-tendinous tendons)
- allografts (occasionally used in Europe for multiligamentar reconstruction, more frequently in USA for isolated ACL reconstruction)

Grafts with bone plugs incorporate in bony tunnels by a bone-to-bone healing; hamstring grafts incorporate by tendon-to-bone healing which sometimes is longer and leads to tunnel widening.

Nowadays ACL is reconstructed arthroscopically. A tibial and a femoral tunnel are done to pass the graft inside the joint and to fix it. The tibial tunnel is always drilled outside-in with a special guide. The femoral tunnel is drilled either inside-out through the tibial tunnel or through another arthroscopic portal creating a blunt tunnel, or outside-in with a specific guide. Whatever the technique, the goal is to place the tunnels anatomically in the joint. Indeed non-anatomical positioning of the graft may lead to persistent anterior laxity or pivot-shift, and is the first risk factor of graft failure.

The graft is fixed in the tunnels by interference screws, or outside the tunnel by Endobutton®, or may also be impacted in the tunnel (press-fit). Double tibial fixation is preferred with both an interference screw and a trans-osseous suture. Graft fixation should be done at 30° of knee flexion.

In case of partial ACL rupture where either the anteromedial bundle or the posterolateral bundle is torn, the torn bundle can be reconstructed solely (for example with the semi-tendinous tendon) and the other bundle is preserved: it is also called «ACL augmentation». In all ACL reconstructions, preserving the stumps of the torn ACL is favorable as it helps graft incorporation, revascularisation and ligamentisation.

Posterior cruciate ligament (PCL)

Posterior cruciate ligament tears are rare and may be undiagnosed. Part of the diagnosis of a PCL tear is made by knowing how the injury happened.

Anamnesis

Any trauma doing a sudden tibial posterior drawer can result in PCL tear, as in traffic road accident with dash-board injury, which is the most frequent trauma mechanism. Accidental kneeling with ankle plantarflexion is another trauma mechanism frequent in soccer. As for ACL tears, trauma is followed by knee pain, swelling and decreased motion.

Clinical exam

The injured knee sags backwards when flexed. The tibial posterior drawer test at 90° of flexion is positive and the tibial step-off has to be quantified by the physician to grade the severity of injury. In general, grading of the injury corresponds to the following:

Grade I: partial tear of the PCL

Grade II: isolated, complete tear of the PCL

Grade III: complete tear of the PCL with other associated ligament injury as postero-lateral corner tear which can be diagnosed clinically with the Hugston recurvatum test, or by finding a postero-lateral rotatory laxity with the dial test or Whipple test.

Imagery

MRI is the gold standard radiological exam to diagnose PCL tear and to find associated lesions (Fig. 3). Stress radiographs as the kneeling view (Bartlett view) are helpful to measure posterior tibial drawer and to compare it to contralateral side.



Fig. 3 PCL rupture.

Treatment

As opposed to ACL, PCL has a good healing potential. Conservative treatment of isolated PCL tear often leads to PCL healing, both clinically and on MRI, and most patients go back to normal daily and sports activities within few months. If not diagnosed, the natural evolution of a PCL tear will be in three phases as described by Prof. Tillat: a 1st phase of functional adaptation (3-18 months), a 2nd phase of functional tolerance (until 15 years), a 3rd phase of arthritis.

Knee immobilisation by a cast or a brace in full extension is known to favor healing of the PCL. Alternatively, a custom-made knee brace applying anterior tibia drawer allows early mobilisation and walking without compromising PCL healing. Knee rehabilitation is done prone by a physiotherapist to recover knee range of motion, and quadriceps strengthening is mandatory.

In case of failure of conservative treatment or in case of combined PCL-PLC lesion with posterior laxity greater than 12 mm, a PCL surgical reconstruction is required. It is also done arthroscopically with a long autograft (for example central third of quadriceps tendon) or allograft. Articular tunnel positioning must also be anatomic. The tibial tunnel is drilled from anterior to posterior with a special guide, and the femoral tunnel is drilled either inside-out through the antero-lateral arthroscopic portal (blunt tunnel), or outside-in with a guide. The graft is fixed inside tunnels by interference screws, or outside with an Endobutton® for example. If arthroscopic reconstruction is not technically possible, tibial PCL graft fixation can be done by a posterior knee approach with a staple.

Medial collateral ligament (MCL)

Trauma in valgus may damage the MCL. If isolated, this injury is often considered as a «benign injury» by the surgeon! However, it can significantly impair the patient function. The anatomical structures

involved are: the medial collateral ligament (MCL) with its superficial and deep bundles (Fig. 4) and the posteromedial capsule with its main ligamentar reinforcement: the postero-medial capsule the posterior oblique ligament (POL) described by Hughston with its three arms. The lesional mechanism is usually a combined knee valgus and tibial external rotation. Initially an elastic deformation occurs, and then a plastic deformation when the elongation is more than 5%. The MCL can be avulsed from its tibial or femoral insertion, or can be torn within its substance. Healing occurs in 4 steps:

- bleeding
- inflammation
- proliferation
- remodeling

Elasticity of the healing ligament quickly returns back to normal. However, it is thickened during several weeks after trauma, and it progressively but incompletely gets thinner. Eventually, its stress rupture point will remain lower, even more if it healed with a fibrotic gap.

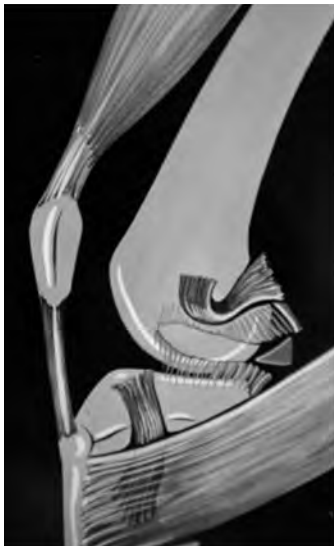


Fig. 4 MCL rupture

Clinical exam

Diagnosis is mainly done clinically (traumatic mechanism, pain, anamnestic instability, knee range of motion, swelling, clinical laxity), and completed by imagery (X-Rays, arthro-CT scan, MRI).

Imagery

MRI is the gold standard exam to evaluate partial rupture (edema) or complete rupture of MCL and to precise the location of the tear (femoral versus tibial side).

Combining clinical and radiological datas, medial knee sprains can be classified in three grades:

- Grade I:** pain, localized edema, no laxity; peri-ligamentous edema with intact ligament on MRI
- Grade II:** localized pain, slight joint effusion, medial laxity less than 5 mm; partial rupture with hyper signal on MRI
- Grade III:** severe pain, significant effusion, medial laxity more than 5 mm; complete rupture on MRI

Treatment

Most isolated medial compartment sprains can be treated conservatively. Patient must be aware that medial knee pain may last longer than expected.

Grade I: no brace, elastic contention, pain killers, ice, full weight bearing

Grade II and III: early knee mobilization in flexion/extension with hinged brace to protect the knee from valgus stress for 3 to 6 weeks; crutches if needed

Surgical treatment, primary repair or sometimes delayed reconstruction, is sometimes necessary in these cases:

- tibial or femoral MCL avulsion with a bony fragment
- Grade III combined with ACL rupture

Potential complications of medial knee structures sprain are chronic medial laxity, stiffness, and medial meniscus hypermobility.

Lateral collateral ligament (LCL)

Anamnesis

A trauma in pure varus can damage the LCL. The postero-lateral corner (PLC) may be damage if the varus is combined with a tibial externa, rotation during trauma. The major structures of the posterolateral corner of the knee include the iliotibial tract, the popliteus complex consisting of both dynamic components (the popliteus muscle-tendon unit) and static components (the popliteofibular ligament, popliteotibial fascicle, and popliteomeniscal fascicles), the middle third of the lateral capsular ligament, the fabellofibular ligament, the arcuate ligament, the posterior horn of the lateral meniscus, the lateral coronary ligament, and the posterolateral part of the joint capsule.

Clinical exam

Palpation of LCL is painful and a gap can be felt when the patient lies in «figure of four» position. An asymmetric lateral laxity in varus is found. To diagnose combined PLC injury, physical examination should include the recurvatum test (Hughston), the external tibial hypermobility test (Bousquet), and the dial test or Whipple test (external tibial rotation at 20° and 90° knee flexion). When the patient is able to walk with full weightbearing, a knee «varus-thrust» appears during the stance phase of gait. Neurologic leg examination is required as the common fibular nerve or the superficial fibular nerve may be stretched or even torn during trauma in varus.

Imagery

Standard X-Rays may show a fibular head avulsion (distal insertion of LCL) or a femoral epicondylar avulsion (proximal insertion of LCL). Stress radiographs in varus show an asymmetric lateral compartment opening and can be compared to the controlateral side. MRI has a high sensibility of LCL and PLC lesions : coronal slice show obviously LCL lesions, but PLC lesions need slices in three planes to be diagnosed. LCL tear may be isolated or combined to PLC, ACL or PCL tears. MRI is very useful to diagnose every intraarticular lesions and to plan the treatment.

Treatment

Conservative treatment gives good results for patients with mild and moderate ligamentous instability.

Early surgical treatment (within three weeks) is required for patients with severe lateral/postero-lateral instability, isolated or combined. Early repair consists of reinsertion with screws or anchors in case of avulsion, or direct sutures in case of interstitial ruptures. Chronic injury, whether

isolated or combined with other tissue injury, is probably best treated by reconstruction of the posterolateral corner along with reconstruction of any coexisting cruciate ligament injury. Many techniques have been described for reconstruction of LCL and/or PLC, with autografts (for example, the «8» hamstring augmentation described by Bousquet, or the biceps augmentation described by Dejour) or allografts. Anatomical reconstruction of each ligament torn seems to give better results.

Conclusions

Each ligament of the knee may be injured during a knee sprain. Understanding the trauma mechanism helps in diagnosis. MRI is nowadays largely prescribed after knee sprains as it confirms the diagnosis and then helps in treatment planning. Treatment depend on which ligament(s) is(are) torn and depend also on patients characteristics (age, level of activity and sports, job, etc). Perfect knowledge of knee anatomy remains the key of success in clinical examination and in surgery.

References

1. Siegel L, Vandenakker-Albanese C, Siegel D. Anterior cruciate ligament injuries: anatomy, physiology, biomechanics, and management. *Clin J Sport Med* 2012;349-55.
2. Tiamklang T, Sumanont S, Foocharoen T, Laopaiboon M. Double-bundle versus single-bundle reconstruction for anterior cruciate ligament rupture in adults. *Cochrane Database Syst Rev* 2012.
3. Voos JE, Mauro CS, Wente T, Warren RF, Wickiewicz TL. Posterior cruciate ligament: anatomy, biomechanics, and outcomes. *Am J Sports Med* 2012;222-231.
4. Fanelli GC, Beck JD, Edson CJ. Current concepts review: the posterior cruciate ligament. *J Knee Surg* 2010;61-72.
5. Robinson JR, Sanchez-Ballester J, Bull AMJ, Thomas R, Amis A. The posteromedial corner revisited. An anatomical description of the passive restraining structures of the medial aspect of the human knee. *J Bone Joint Surg* 2004 ; 674-681.
6. Covey D.C. Injuries of the posterolateral corner of the knee. Current concepts review. *J Bone Joint Surg* 2001 ; 106-118.



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Meniscus and cartilage repair

The etymology of the term meniscus comes from the Greek, "meniskos" which means a crescent-moon shape.

The morphology of the meniscus strongly resembles this shape.

The kinematics of the menisci in humans is quite asymmetric, with the lateral meniscus being much more mobile than that of the medial meniscus.

Because the medial compartment of the knee is substantially more constrained than that of the lateral compartment, chronic ligamentous injuries – such as that of the anterior cruciate ligament which results in pathological increases in AP translation – often is associated with increased frequency of damage of the medial meniscus compared to that of the lateral meniscus.

All of the various components of the knee are important to normal functioning of this living, self-maintaining transmission system. (Fig. 1)

A knee that has sustained a tear of the meniscus can be thought of as a transmission that has a damaged bearing.



Fig. 1 All of the various components of the knee are important to normal functioning of this living self-maintaining transmission system (Sott Dye, courtesy Werner Müller)

2. Meniscus repair

2.1. Biology

Since Kohn and Siebert's study in 1989, the biomechanical basis of meniscus repair – and meniscus repair itself – has significantly evolved. Evaluation of the first-generation repair techniques with sutures showed that the biomechanical conditions of meniscal repair were dependent on the anatomy of the meniscus, the quality of this tissue and the type

of suture and suture material.

Studies published in the 90s and in the current decade evaluated the second and third-generation repair devices.

While the second-generation devices represented a significant step forward with respect to the invasiveness of surgery, their biomechanical properties were generally inferior to those of the "gold standard" sutures. However, as biomechanical testing became more complex with the introduction of cyclic loading, the evaluation of meniscus repair could be extended to include criteria such as the resistance of the repair and gapping of the tear site under more physiologic loading conditions. The third-generation flexible suture anchors meet both the criteria of minimal invasiveness and biomechanical properties, which are comparable to those obtained with suture techniques.

These anchors as well as improved all-inside suture techniques will probably represent the first choice of meniscal repair techniques in the coming years.

From a scientific point of view, further studies should be performed to achieve a better understanding of the forces acting on meniscus repair under certain pathologic conditions and of the biomechanical properties of regenerated or "healed" meniscus tissue after repair.

2.2. Techniques

Saving the meniscus, especially in young patients, to decrease the risk of secondary osteoarthritis is challenging. Meniscal repair techniques are well established and allow surgeons to address tears of different complexity and location. There exists no universal technique, but rather several techniques which are adapted to different indications. Even if all-inside fourth-generation devices are now the gold standard in the majority of cases, inside-out, outside-in, and even open techniques are still indicated in selected cases. The ultimate goal is to achieve a strong repair.

In the future, the next step will be biological meniscus repair by introducing factors such as stem cells, growth factors, or cytokines at the site of the repair to enhance healing. These can be regarded as biological mediators, which regulate key processes in tissue repair (cell proliferation, directed cell migration, cell differentiation, and extracellular matrix synthesis).

2.3. Results

Menisci are no vestigial structures, but form an integral part of the 'self-maintaining transmission system' which the knee joint is.

Minimal tissue resection, which very often can be described as 'adequate', e.g. leaving the meniscal rim, should be the rule. Care should be taken to resect what has been torn and remove meniscal tissue only to avoid any further impingement that may remain sensitive to rotational painful stress and may thus produce clinical symptoms.

Arthroscopic techniques allow for repeat surgery, which may be required in case of persistent mechanical derangement. However, the fulcrum to

proceed to repeat arthroscopy surgery needs to remain clinical. All too often, repeat surgery does not alter the clinical findings if it is based on – needless – imaging alone.

Therefore, potential meniscal repair is warranted in all cases where meniscal resection has been considered. Full options remain when, in addition to partial resection, suture of the meniscal remnant to the meniscal wall appears to be required.

Biomechanical investigation and testing of meniscal repair devices has received ample consideration. While tensile forces, which are of lesser importance in clinical practice, have been extensively investigated, shearing forces acting on the meniscus are of paramount clinical importance but cannot be reliably reproduced in in-vitro studies.

Experience has taught us that a red-on-red tear heals spontaneously within four to six weeks, provided that the necessary immobilization is applied. (Fig. 2) The purpose of meniscal stabilization is to safely bridge this period in order for the scar tissue to heal and stabilize the lesion. Because in vivo testing is not possible as yet, clinicians investigate implant material by essentially focusing on material properties, safety guidelines and ease of insertion, with convincing evidence based on physiological meniscal healing.

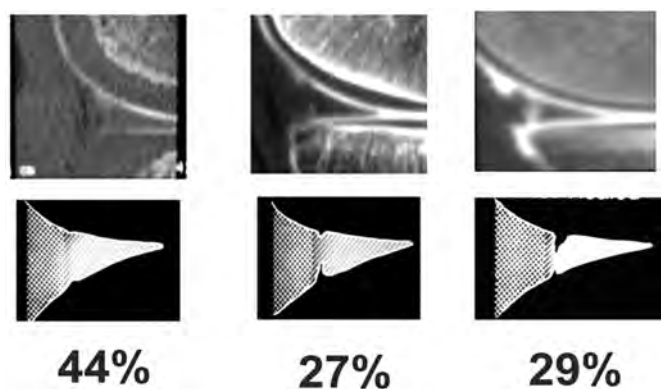


Fig. 2 The healing rate in meniscal repair is highly satisfactory when considering CT scan arthrography (courtesy Société Française d'Arthroscopie)

The implants developed in recent years allow for arthroscopic meniscal suturing all around the meniscal rim. Good stabilization is obtained in the majority of cases. Average results are defined as up to 80% of clinical healing at long-term follow-up. Failures are mainly due to improper indications or knee joint instability. Less well-documented reasons could be poor meniscal tissue, low cellularity and thus poor healing response. These findings are obviously difficult to document, but are recognized when at surgery yellowish degeneration of the meniscal core is found, which is often related to age and overload and compromises the healing response.

One of these 'degenerative' findings is the meniscal cyst. Prone to increased shearing forces at its fixation around the popliteus muscle tendon, the lateral meniscus may sustain a horizontal tear associated with cyst formation. Depending on its intra-articular 'opening', the symptomatic cyst needs to be resected and the torn meniscus repaired. Repair is mandatory at all costs in order to avoid underlying cartilage degeneration.

2.4. Rehabilitation

Although evolving continuously, concepts of postoperative rehabilitation after meniscus repair still remain controversial. Two rehabilitation protocols, applied in clinical practice, are described in the current literature: the conventional and the accelerated rehabilitation protocol.

The optimum rehabilitation is yet to be identified, and the lack of scientific data in the literature does not allow us to endorse a specific rehabilitation program.

However, it is the authors' opinion that all intrinsic factors should be taken into account when designing a rehabilitation program. Individualizing the rehabilitation according to the size and type of meniscal tear, vascular supply, localization, concomitant procedure, and presence of other intra-articular disorders (ACL, cartilage lesions,...) seems to be an interesting concept. If less than two intrinsic risk factors are present, healing will occur fairly rapidly and the risk of failure is low, so that an accelerated rehabilitation protocol is recommended. However, the presence of more than two risk factors (e.g., a large tear in a red-white zone) increases the risk of meniscus failure and slow healing, and in this case a more conservative approach is probably the best guarantee for success.

However, well-designed longitudinal studies are mandatory to determine the actual efficacy of this rehabilitative approach with regard to patient function and satisfaction.

3. Meniscus replacement

Carl Wirth and Gabriela von Lewinski investigated the basic science in meniscal transplantation. The interest taken by their German group in meniscal transplants was fueled by clinical needs.

The concept of the meniscus also being a stabilizing structure in the knee joint is not new, but they were the first to consider the meniscus as a primary stabilizer after knee ligament injury and repair. Simply removing the meniscus had proven deleterious to the long-term results after ligament repair.

In animal experiments, the authors were able to show healing after meniscal allograft implantation.

Also in human clinical studies, satisfactory healing occurred at the meniscosynovial junction, but whether this was also true for the meniscal horns remained a controversial issue.

Horn fixation is indeed mandatory for true hoop stress protection.

In addition, animal experiments showed increased cartilage degradation when the normal anatomy had not been restored. Nowadays, no clear consensus is available on whether bone fixation of meniscal allografts is mandatory for normal homeostasis.

Choosing allograft tissue such as meniscal tissue, although of limited availability, is a logical option.

Deep-freezing appears to be the most accepted method of preservation and standards of procurement have been well established.

If procured in a sterile fashion, the allografts can be used when the tissue bank has found the donor to be free of transmissible diseases. When harvesting has been done in an unsterile fashion, the issue of sterility requires appropriate attention and management. Avoiding irradiation as such is essential in order not to be detrimental to meniscal structure and thus good postoperative function.

However, national laws and regulations can interfere with good clinical practice on grounds of legal constraints based on earlier infringements and exposures.

Meniscal surgery, as it started in the 1990s, required an open approach because at that time arthroscopic meniscal fixation devices were limited and not really appropriate. In the early beginning, meniscal transplantation was very often associated with other repair surgery (mostly ligamentous).

Open surgery is also required for bone plug fixation and to obtain

elementary stability.

It is only because meniscal surgery and repair indications have increased that arthroscopic transplantation has been initiated.

Without bone plug fixation the technique becomes an arthroscopic soft-tissue procedure, also with use of improved fixation and stabilization devices as applied constantly in routine meniscal repair procedures.

With growing surgical expertise and better visualization and anatomic positioning of the anterior and posterior meniscal horns, bone plug fixation has become technically less challenging.

The literature does not indicate whether one or the other technique is superior in terms of results, nor has any clinical difference in results been reported between deep-frozen, cryopreserved or viable (fresh) transplants at 15 to 20 years of follow-up.

Obviously, clinicians are more confronted with issues dealing with partial meniscectomy and functional derangement.

In animal experiments, collagen meniscus implantation (CMI) was found to yield good results and function. The regenerated tissue appeared to be similar to the native meniscus. The implants did not induce degenerative changes, abrasion or synovitis, and were devoid of allergic or immune responses.

Human clinical trials, which were conducted at various centres over longer periods of time, showed a lesser need for revision surgery after CMI implantation in chronic meniscectomized knees, compared to controls. (Fig. 3)



Polyurethane scaffold



Collagen scaffold

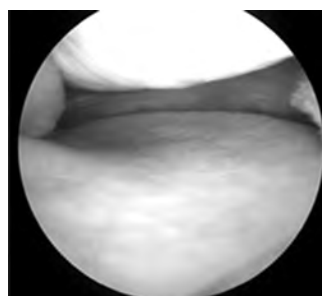


Fig. 3 In case of partial meniscectomy and keeping up with a stable meniscal wall, new scaffolds may generate meniscal tissue to protect the weight bearing cartilage

Good alignment and stability are preoperative requirements.

Alternatives were searched for that would allow working with stronger as well as resorbable materials.

In animal studies, long-term assessment of a polyurethane scaffold showed that transformation into meniscus-like tissue took place as the implant slowly degraded.

Another requirement is the possibility to insert and manipulate the implant into position with use of arthroscopic techniques. A first human safety and efficacy study of 52 patients demonstrated a statistically significant improvement in quality of life and clinical scores at two years (Verdonk P, Beaufils Ph), suggesting that the implant was safe and effective.

Finally, meniscal allografts seem to sustain the hypothesis that meniscal replacement after total meniscectomy is a valid alternative, more specifically in the lateral compartment. For the medial compartment, other useful options are available.

The more common knee dysfunction after partial meniscectomy does not warrant total meniscal allograft replacement.

While we are still constantly searching for useful modes of treatment, partial meniscal replacement is already a first step in the right direction.

4. Ligament instability

4.1. Introduction

True ligament instability needs appropriate diagnosis and treatment.

In this presentation we will not focus on indication and techniques.

We will look into factors associated with ligament instability leading to early arthritis is not appropriately taken care of.

4.2. Sports induced OA predictors

It was formerly accepted that both higher age at the time of meniscectomy as well as longer surgical delay after meniscal rupture would be major factors in inducing osteoarthritis.

It has been shown (Neyret – Verdonk) these factors to be weak predictors for sports induced osteoarthritis.

However, early medial meniscectomy (3x) and even more dramatically medial cartilage lesion at early age (5x) will induce dramatic increase in osteoarthritis.

All these individual facts have even a worse impact in the sports induced osteoarthritis if the patient is confronted with an ACL deficient knee joint. Stabilization of the knee joint after ACL deficiency presents with a satisfactory outcome in a large patient population.

When normal, at 11 years the intact knee remains pristine at further long-term follow-up (24 years) (Neyret – Verdonk).

In malalignment, the index compartment will present overload.

As such, literature is rather scarce in relation to osteotomies and anterior ligament instability around the knee joint. (Fig. 4) With respect to posterior instability only 1 paper is retained (Giffen).

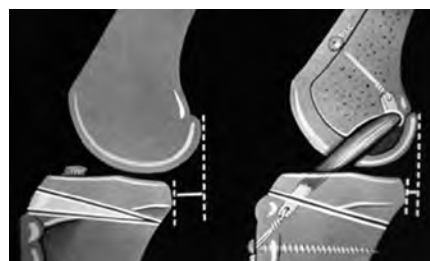
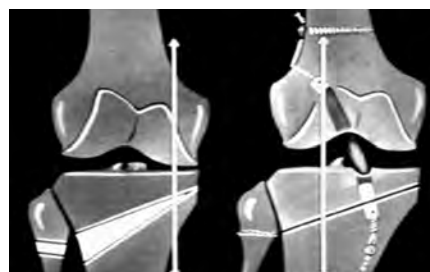


Fig. 4 Valgus HTO in anterior instabilities using a closing wedge osteotomy technique has a tendency to decrease the tibial slope and as such to reduce the anterior tibial translation (courtesy Prof. Dr. Philippe Neyret).

Medline then presents no literature on osteotomies around the antero-posterior instabilities in the knee joint.

In the anterior instabilities with frontal imbalance will allow for closing wedge osteotomy when presenting medial narrowing of the jointline.

In case of sagittal imbalance an AP closing wedge osteotomy will allow for improving biomechanics.

In case of posterior instability after chronic PCL rupture, the sagittal instability will be reduced by an opening wedge osteotomy tilting the tibial plateau.

5. Patella

5.1. Introduction

Many factors, apart from sports overuse, can lead to patellar symptoms.

They are: dislocation, instability, hyperpression, cartilage lesions – more often than not associated with tendon pathology, both below in the patellar tendon as above in the quadriceps tendon –, patellar height.

5.2. Instability factors

Henri Dejour (1987) described 4 main factors inducing instability of the patella.

They are the trochlear dysplasia, the high-riding patella (>1.2), the relationship between the tuberositas tibiae and the trochlear groove ($>20\text{mm}$) and the patellar tilt ($>20^\circ$).

These factors being increased will lead to patellar dislocation.

The instability factors need to be addressed individually and can possibly be combined with other "focal" treatments to eradicate the inducing factors.

The high-riding patella needs to be levelled to index 1 when pathological.

Potential patellar tilt, whether inborn or post-traumatic may need muscle plasty of the vastus medialis internus. If the medial patellar femoral ligament is ruptured because of an earlier dislocation, it needs to be reconstituted anatomically.

In case of frank trochlear dysplasia (grade B and D), a trochlear plasty may be necessitated.

In case of abnormal TT- TG it needs to be reduced to former and normal measurements.

Secondary factors such as malalignment do not need to be addressed in casu.

6. Cartilage

6.1. Introduction

Cartilage repair is physiologically non existent. Surgical approaches have been described using local regeneration using the micro-fracture technique.

Cell transplantation, both autologous as well as allotransplantation of both individual cells, cultured cells or as allografts has been used.

It appears that both in symptoms and in treatment, sizing is essential for inducing valuable results.

In addition, correct alignment is a prerequisite to support good clinical results.

6.2. Cartilage lesions

Arøen et al. (2004) and Curl (1997) have investigated the number of cartilage lesions in routine arthroscopy.

Cartilage lesions in excess of 2cm^2 are present in more than 66% of all cases.

However, only 20% present with a localized defect.

11% are rated grade 3 or 4 according to ICRS.

Only a very limited number of 6% present the same grade 3 – 4 ICRS in a defect that is in excess of 2cm^2 . Figure 5

It is generally accepted that only lesions in the weight bearing zone in excess of 2cm^2 can be symptomatic in need and are in potential need of treatment

It is also common knowledge that degenerative lesions need an appropriate treatment that does not relate to cartilage treatment in itself.



Fig. 5 Limited and focal fresh cartilage lesions can be considered for surgery when limited in extent.

6.3. Treatments

Nowadays small lesions tend to present with good results when treated with microfracture approach as described by Steadman.

The edges of the lesions need to be debrided until sharp.

The micro-fracture technique necessitates perforation until the subchondral lamina every 2 to 3 mm in order to expect bleeding to occur. This suggests the potential of mesenchymal stem cells to approach the lesions surface in order to generate repair cartilage tissue.

Gobbi (2005) has investigated a level 4 comparative study looking into long-term results in young athletes (38y.) retaining pain relief at 6 years, however with lower and sports activities in 80% of cases.

Gudas (2005) looked into younger sportsmen and investigated in a randomized prospective level 1 study microfracture versus mosaicplasty in a 3 year minimal follow-up study.

Lesions were grade 3 to 4. Size, $2,8\text{cm}^2$ and comparing microfracture versus mosaicplasty.

There was a distinct advantage in the use of the mosaicplasty treatment versus microfracture.

7. Conclusion

In painful cartilage lesions in the young athlete, the ideal candidate presents with age below 40. The lesions should be fresh (below 3 months). It is essential to have a lower end BMI ($<30\text{kg/m}^2$) in normal axial alignment without any associated lesions. Small sizing of maximum 3cm^2 is prerequisite.

References

1. R. Verdonk (2010) Meniscal repair: Biomechanics. In: *The Meniscus*, editors R. Verdonk and PH. Beaufils. In press.
2. Dye SF (1996) The knee as a Biologic Transmission with an Envelope of Function. *Clin Orthop Rel Res* 325: 10–18.
3. Müller W (1982) *Le Genou*. Springer Verlag, Berlin
4. Dye SF (1987) An evolutionary perspective of the Knee. *J. Bone Joint Surg* 7:976–983.
5. Seil R, Pape D. (2010) Meniscal repair: biomechanics. In: *The Meniscus*, editors R. Verdonk and PH. Beaufils. In press.
6. Charrois O (2008) Enquête de pratique SFA/ESSKA/SOFCOT. In: *Symposium on Le ménisque latéral. Congrès de la Société Française d'Arthroscopie*, Paris.
7. Seil R, Rupp S, Krauss PW, Benz A, Kohn D (1998) Comparison of initial fixation strength between biodegradable and metallic interference screws and a press-fit fixation technique in a porcine model. *Am J Sports Med* 26(6):815–819.
8. Seil R, Rupp S, Dienst M, Müller B, Bonkhoff H, Kohn D (2000) Chondral lesions after arthroscopic meniscus repair using meniscus arrows. *Arthroscopy* 16(7): E17–37. Seil R, Rupp S, Kohn D (2000) Cyclic testing of meniscal sutures. *Arthroscopy* 16:505–510.
9. Seil R, Rupp S, Jurecka C, Rein R, Kohn D (2001) Der Einfluß verschiedener Nahtstärken auf das Verhalten von Meniskusnähten unter zyklischer Zugbelastung. *Unfallchirurg* 104(5):392–398.
10. Seil R, Rupp S, Mai C, Pape D, Kohn D (2001) The footprint of meniscus fixation devices on the femoral surface of the medial meniscus: a biomechanical cadaver study. *ISAKOS congress*, Montreux 40. Seil R, Rupp S, Jurecka C, Georg T, Kohn D (2003) Réparation méniscale par fixations biodégradables: étude biomécanique comparative. *Rev Chir Orthop* 89:35–43.
11. Jouve F, Ovadia H. (2010) Meniscal repair: Technique. In: *The Meniscus*, editors R. Verdonk and PH. Beaufils. In press.
12. Albrecht-Olsen P, Kristensen G, Tormala P (1993) Meniscus bucket-handle fixation with an absorbable Biofix tack: development of a new technique. *Knee Surg Sports Traumatol Arthrosc* 1:104–106.
13. DeHaven KE (1990) Decision-making features in the treatment of meniscal lesions. *Clin Orthop* 252:49–54.
14. DeHaven KE, Black K, Griffiths HJ (1989) Open meniscus repair. Technique and two to nine year results. *Am J Sports Med* 17:788–795.
15. McDermott ID, Richards SW, Hallam P, Tavares S, Lavelle JR, Amis AA (2003) A biomechanical study of four different meniscal repair systems, comparing pull-out strengths and gapping under cyclic loading. *Knee Surg Sports Traumatol Arthrosc* 11:23–29.
16. R. Verdonk (2010) Synthesis. In: *The Meniscus*, editors R. Verdonk and PH. Beaufils.
17. E. Witvrouw (2010) Rehabilitation. In: *The Meniscus*, editors R. Verdonk and PH. Beaufils.
18. Jokl P, Stull PA, Lynch JK et al. (1989) Independent home versus supervised rehabilitation following arthroscopic knee surgery: a prospective randomized trial. *Arthroscopy*, vol 5, pp 298–305.
19. McLaughlin J, DeMaio M, Noyes FR et al (1994) Rehabilitation after meniscus repair. *Orthopedics*, vol 17, pp 465–471.
20. Shelbourne KD, Patel DV, Adsit WS et al. (1996) Rehabilitation after meniscal repair. *Clin Sports Med*, vol 15, pp 595–612.
21. Giffin JR, Vogrin TM, Zantop T, Woo SL, Harner CD, Effects of increasing tibial slope on the biomechanics of the knee. *Am J Sports Med*. 2004 Mar;32(2):376–82.
22. Arøen A, Løken S, Heir S, Alvik E, Ekeland A, Granlund OG, Engebretsen L. Articular cartilage lesions in 993 consecutive knee arthroscopies. *Am J Sports Med*. 2004 Jan–Feb;32(1):211–5.
23. Curl WW, Smith BP, Marr A, Rosencrance E, Holden M, Smith TL., The effect of contusion and cryotherapy on skeletal muscle microcirculation. *J Sports Med Phys Fitness*. 1997 Dec;37(4):279–86.
24. Gobbi A, Nunag P., Malinowski K., Treatment of full thickness chondral lesions of the knee with microfracture in a group of athletes. Volume 13, Number 3 / April, 2005.
25. Gudas R, Kalesinskas R., Kimtys V, Stankevicius E, Toliusis V, Bernotavicius G, Smailys A. A Prospective Randomized Clinical Study of Mosaic Osteochondral Autologous Transplantation Versus Microfracture for the Treatment of Osteochondral Defects in the Knee Joint in Young Athletes. *Arthroscopy: The Journal of Arthroscopic and Related Surgery* September 2005 (Vol. 21, Issue 9, Pages 1066–1075).
26. Verdonk P, Beaufils Ph, Bellemans J, Djian P, Heinrichs EL, Huysse W, Laprell H, Siebold R, Verdonk R and Actifit Study Group. Successful Treatment of Painful Irreparable Partial Meniscal Defects With a Polyurethane Scaffold: Two-Year Safety and Clinical Outcomes *Am J Sports Med* February 9, 2012.



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Fractures: Pelvic ring und acetabular fractures

1. Pelvic ring injuries

Just 2% of all injuries are pelvic ring injuries with about 70% being stable anterior pelvic ring fractures in the elderly caused by a simple fall. Unstable pelvic ring injuries are caused by a high energy trauma and are part of a polytrauma in one third of the cases. These unstable pelvic ring injuries with disruption of the posterior pelvic ring can be life-threatening because of massive retroperitoneal bleeding. Therefore primary stabilisation of the pelvic ring needs to be included into the polytrauma algorithm. Definitive stabilisation with internal fixation is to be performed as secondary procedure after stabilisation of the general condition of the patient.

1.1. Anatomy

The osseous pelvic ring consists of two ossa innominata and the sacral body. The stability of the pelvic ring is achieved by the ligamentous structures in the symphyseal area and the SI joints. Herby the posterior sacro-iliac ligaments are the strongest ligaments in the whole human body. Forces of more than 1000N are needed to produce a disruption of the posterior pelvic ring. Therefore unstable pelvic ring injuries often are associated with high energy trauma and polytrauma.

Iliac artery and vein as well as the lumbar nerve plexus are running in front of the sacrum. Major bleeding can be caused by disruptions of the posterior pelvic ring, caused mainly by bleeding out of the sacral venous plexus and fracture sites. In only 20% arterial bleeding is the reason for hemodynamic instability.

1.2. Diagnostics

Besides the clinical exam and stability test, the plain X-ray as pelvic view is the base to assess a pelvic ring injury. Additional 45° tilted projections like Inlet and Outlet view can show horizontal and vertical displacements respectively. The computer-tomography is strongly recommended for further evaluation of a pelvic ring injury and for preoperative planning. In up to 70 percent a posterior pelvic ring lesion can only be detected in the CT. Besides a minimum of 2mm cuts additional 2D reconstructions in sagittal and coronal direction are helpful. MRI is rarely needed in the acute setting. An angiography can be applied in cases with suspect for additional vascular injury and existing setup for embolisation.

A precise neurologic exam is mandatory in pelvic ring injuries because of the close relation to the lumbar nerve plexus.

1.3. Classification

The most common classification used is the AO Classification, based on the Tile classification. The main criterion in this classification is the remaining stability of the pelvic ring. Stable A-type injuries are differentiated from partially unstable B-type injuries and completely unstable

C-type injuries

A-type injuries, stable lesions:

A1: iliac wing fractures, apophyseal fractures

A2: anterior pelvic ring fractures

A3: transverse sacral fractures

B-type injuries, partially unstable lesions, horizontal instability:

B1: open book injuries

B2: lateral compression type injuries

B3: bilateral B-type injuries

C-type injuries, lesions with complete disruption of the posterior pelvic ring, horizontal and vertical instability:

C1: unilateral vertical instability

C2: one side vertical instability, other side B-type injury with horizontal instability

C3: bilateral vertically unstable lesion

In C-type injuries the disruption of the posterior pelvic ring can be transiliacal, through the SI joint or transsacral.

1.4. Treatment

The assessment of stability of the pelvic ring and appropriate classification is a mandatory prerequisite for the adequate therapy.

A-type injuries can be treated conservatively most of the times. This means mobilisation with symptom-adapted weight bearing. In case of persisting pain a CT is recommended to exclude a lesion of the posterior pelvic ring. Surgical therapy is only recommended for apophyseal fractures in adolescents, iliac wing fractures with major displacement and transverse sacral fractures with neurology.

B-type injuries need a differentiated therapy concept. Open book injuries (B1 injuries) are usually treated with open reduction and plate fixation (4 hole LCDCP). Lateral compression injuries (B2) with stable impaction can be treated conservatively with partial weight bearing on the injured side. Lateral compression injuries with major displacement and/ or overlap in the anterior pelvic ring need fixation of the anterior pelvic ring, preferably with an external fixator.

As the posterior pelvic ring is not disrupted completely, B-type injuries are treated appropriately with stabilisation of the anterior pelvic ring.

C-type injuries are characterized by complete disruption of the posterior pelvic ring through the ilium, the SI joint or sacrum. Depending on the concomitant bleeding and general condition of the patient, a primary external stabilisation of the pelvis is necessary to stabilize the patient. Besides a simple sheet around the pelvis as "in field maneuver", the external fixator and pelvic C-clamp are used for this primary stabilisation. The C-clamp is an emergency device to stabilize the posterior pelvic ring, thus reducing the intrapelvine volume and the bleeding. The best indications are pure SI disruptions or injuries with major fragments. Contraindication is a transiliac fracture, because of the risk of penetrating pins.

The alternative method for external stabilisation of the pelvic ring is

the external fixator. Preferably the Schanz' screw are placed supra-acetabular because of better biomechanical stability compared to pins into the iliac crest.

Definitive stabilisation is usually performed as a secondary procedure after 5-7 days depending on the general condition of the often polytraumatized patient. If closed reduction is possible, sacral fractures and SI disruptions can be stabilized with SI screw fixation in a minimal invasive technique. If open reduction is necessary, direct plating is preferred for sacral fractures using a posterior approach and anterior plating with 2 plates for SI disruptions. Transiliac dislocation fractures are treated with open reduction and plate fixation from anterior.

For stable fixation and good results, C-type injuries need fixation of the posterior and anterior pelvic ring. Stabilisation of the anterior pelvic ring is achieved at least with an external fixator.

1.5. Results

Stable A-type injuries have good results with conservative treatment. In case of persisting pain in A-type injuries a CT is strongly recommended to exclude additional lesions in the posterior pelvic ring, thus resulting in a B-type injury. For B-type injuries, functional results usually are good. Overall outcome is depending on the extent of urogenital and neurological impairments, especially in B1 and B3 injuries. For C-type injuries the stable reconstruction of the pelvic ring with less than 1cm displacement is a mandatory prerequisite for good results. This can be achieved in more than 70% of the cases. Nevertheless the overall result is depending mainly on the neurological impairments and in case of a polytrauma on the result of the other injuries.

2. Acetabular fractures

2.1. Anatomy

The acetabulum consists of an anterior and a posterior column. In radiographs the iliopectineal line corresponds to the anterior column, the ilioischial line to the posterior column. Additionally the acetabular roof, the anterior wall and the posterior wall can be evaluated.

2.2. Diagnostics

The pelvic X-ray is the base for diagnostics. All lines need to be evaluated in comparison to the noninjured side. Interruptions of the iliopectineal line show a fracture of the anterior column, interruptions of the ilioischial line a fracture of the posterior column. Additional evaluation is possible with the 45° degree tilted iliac view and obturator view. In the iliac view the anterior wall and posterior column are to be seen the best, in the obturator view the anterior column and posterior wall. The additional computertomography is recommended for all acetabular fractures. Intraarticular fragments, marginal impaction of the joint and the full extent of the fracture can be visualized within the axial cuts and the 2D reconstructions. The 3D reconstruction can be helpful to plan the appropriate approach for reconstruction.

2.3. Classification

The most common classification is the Letournel classification. Within this classification 5 simple fractures and 5 combined fractures are differentiated. Simple fracture does not mean easy to treat, but a single fracture line. Anterior wall, posterior wall, anterior column, posterior column and transverse fracture are these basic fractures. Posterior wall fractures are often associated with a posterior dislocation of the femoral head. Combined fractures are posterior column posterior wall, transverse posterior wall,

anterior column posterior hemitransverse, T-type and both column fractures. Both column fractures are characterized by complete separation of the fractured joint from that part of the iliac bone still being attached to the SI joint.

2.4. Treatment

Nondisplaced fractures, fractures below the weight bearing area and fractures with displacement less than 2mm can be treated conservatively. Unstable fractures and fractures with displacement more than 2mm within the weightbearing area are recommended to be treated operatively. The choice of the appropriate approach is essential for anatomic reduction and stable fixation. Fractures with more anterior pathology (anterior wall, anterior column, some transverse, anterior column posterior hemitransverse and most both column fractures) are treated with an anterior, ilioinguinal approach. The posterior Kocher-Langenbeck approach is used for fractures with more posterior pathology (posterior wall, posterior column, some transverse, transverse posterior wall, posterior column posterior wall, T-type). More than 90% of all acetabular fractures can be treated by these two classic approaches. Just rarely extended approaches are needed for fractures in which manipulation and fixation from anterior and posterior is necessary. For specific fracture patterns minimal invasive approaches can be applied, especially with intraoperative 3D C-arm imaging and / or navigation. After reconstruction and fixation of acetabular fractures, partial weight bearing with 15kg is necessary for 12 weeks.

2.5. Results

For good functional long term results near anatomic reduction is essential. With appropriate diagnostics, exact classification, the right approach and extensive expertise good reduction of acetabular fractures with less than 1mm step or gap is possible in up to 80% of the fractures. In case of near anatomic reduction about 75% will have good long term results.

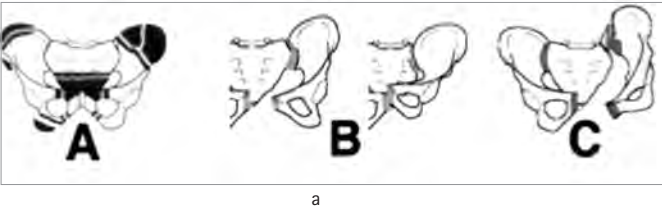
References

1. Letournel E, *The treatment of acetabular fractures through the ilio-inguinal approach. Clin Orthop Relat Res.* 1993 Jul;(292):62-76.
2. Helfet DL, Schmeling GJ., *Management of complex acetabular fractures through single nonextensile exposures. Clin Orthop Relat Res.* 1994 Aug;(305):58-68.
3. Matta JM., *Fractures of the acetabulum: accuracy of reduction and clinical results in patients managed operatively within three weeks after the injury. J Bone Joint Surg Am.* 1996 Nov;78(11):1632-45.
4. Kreitner KF, Mildenerger P, Rommens PM, Thelen M. Rofo.; 2000 Jan;172(1):5-11. [Rational diagnostic imaging of pelvic and acetabulum injuries].
5. Gettys FK, Russell GV, Karunakar MA., *Open treatment of pelvic and acetabular fractures. Orthop Clin North Am.* 2011 Jan;42(1):69-83, vi.
6. Bates P, Gary J, Singh G, Reinert C, Starr A. *Percutaneous treatment of pelvic and acetabular fractures in obese patients. Orthop Clin North Am.* 2011 Jan;42(1):55-67, vi.
7. Stöckle U, Schaser K, König B. *Image guidance in pelvic and acetabular surgery expectations, success and limitations. Injury.* 2007 Apr;38(4):450-62.
8. Giannoudis PV, Pohlemann T, Bircher M. *Pelvic and acetabular surgery within Europe: the need for the co-ordination of treatment concepts. Injury.* 2007 Apr;38(4):410-5. Epub 2007 Mar 29.

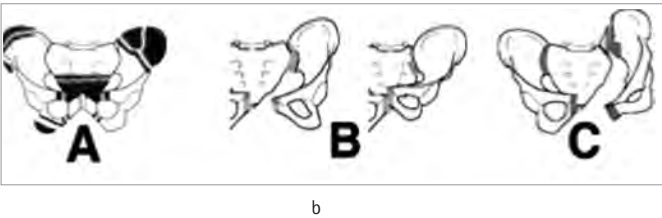
9. H Tscherne, T Pohlemann, Unfallchirurgie 1998. Buch. XVI, 498 S.: 800 s/w-Abbildungen, 30 s/w-Tabellen. Hardcover Springer Berlin ISBN 978-3-540-62481-3

Figures

AO- Classification



- A Anterior and Posterior Arch Intact
Stable Lesions
- B Anterior Complete,
Posterior Incomplete Disruption
Rotational Instability
- C Anterior and Posterior Complete Disruption
Rotational and Vertical Instability



- A Conservative treatment
- B Predominantly operative treatment
- C Operative treatment

Fig. 1a,b AO Classification for pelvic ring injuries and treatment options.

Letournel Classification

Simple fractures

- Posterior wall
- Posterior column
- Anterior wall
- Anterior column
- transverse

Combined fractures

- T-shaped
- Posterior column / posterior wall
- Transverse / posterior wall
- Anterior column / posterior hemitransverse
- both column fracture

Operative treatment acetabular fractures

Anatomic reconstruction (step < 1mm)			
	n	%	good function
Letournel (1993)	492	74	82%
Helfet (1994)	119	74	84%
Matta (1996)	127	90	
Rommens (2000)	225	86	74%
→ anatomic reconstruction prerequisite for good functional results			

Fig. 2a-c Classification of acetabular fractures, results after operative treatment.



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Fractures: Hip, femur, tibia & open fractures

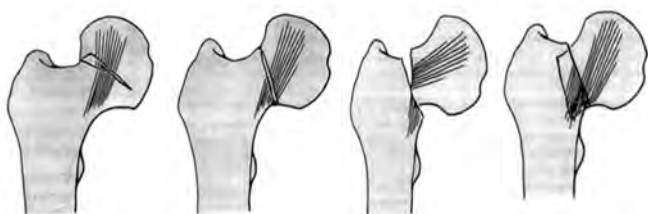
1. Intracapsular hip fractures

Introduction: The absolute number of intracapsular hip fractures has doubled since the mid 1960s leading to an immense increase of the cost. These fractures occur more often in women than in men and more frequently and in younger individuals than intertrochanteric fractures [Parker]. There is a direct correlation between low BMD, low BMI, decreased activity, poor balance, impaired vision and fractures in the elderly. Femoral neck fractures in patients < 50 years make up only 3%-5% and are usually due to high-energy trauma. In this age group 20% of femoral neck fractures are associated with femoral shaft fractures and are missed in up to 30% !

Anatomy: The vascular supply to the proximal femur, including the femoral head, originates from the medial and lateral femoral circumflex arteries, branches of which form an extracapsular arterial ring at the base of the femoral neck. The LFCA being the main artery for blood supply to the head.

Diagnosis: Clinical signs and symptoms can vary according to the type of fracture. In valgus impacted, incomplete or nondisplaced fractures weight bearing can still be possible while patients with displaced fractures have shortening of the affected limb with external rotation, mild abduction and are unable to weight bear. Standard antero-posterior and cross-table lateral RX are required for the diagnosis of a neck fracture. In doubt T1-weighted MRI study will confirm or deny the presence of a fracture in 100%.

Classification: The Garden classification based on the amount of displacement on the antero-posterior radiograph is the most commonly accepted system. There are nondisplaced (GI and II) and the displaced (GIII and IV) fractures. The AO system classification ranges from 31.B1.1. to 31.B3.3.



Undisplaced fracture (Garden 1 & 2), Displaced fracture (Garden 3 & 4)

Treatment: A femoral neck fracture in a young patient should be considered an orthopaedic emergency, elderly patients should be operated as soon as possible once they are medically cleared and ideally within 48h. Young patients with a displaced femoral neck fracture

should benefit from anatomic reduction of the fracture. If this is not possible by closed means surgical dislocation of the hip by trochanter flip or a Watson-Jones approach should be discussed.

Patients with non- or minimally displaced fracture have a low risk for osteonecrosis and nonunion if the fracture is fixed. A displaced fracture places the patient at a high risk of about 25% (range, 10%-45%) of osteonecrosis and nonunion. In elderly patients displaced femoral neck fractures should thus be considered for prosthetic replacement. If fracture fixation is chosen, best results will be achieved with screws. In noncomminuted fractures placing more than 3 screws (or 2 pins) offers no mechanical advantage. In fractures with posterior comminution a forth screw can provide better stability [Kauffmann].

If arthroplasty is chosen bipolar hemiarthroplasty as compared to mono-polar hemi arthroplasty demonstrates a trend to better scores for pain, social function and mobility.

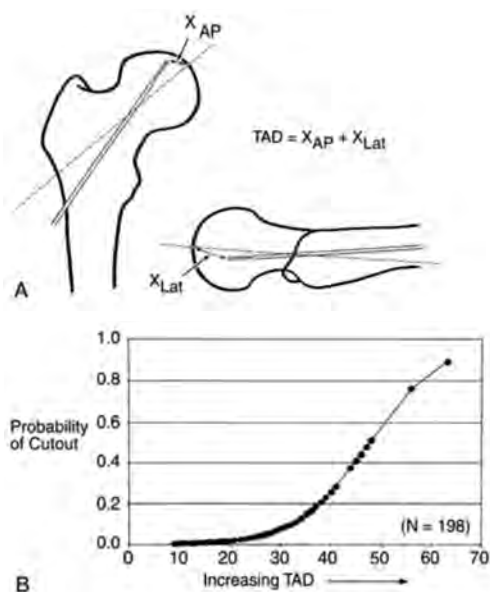
Total hip replacement, initially used mainly for fracture patients with severe preexisting hip disease, is considered now more and more for patients with a life expectancy of more than 5 to 10 years.

Complications: The incidence of osteonecrosis ranges from 10% to 45% adequate and timely reduction being the most important preventing factors. The reported incidence of non-union ranges from 10% to 30% and MRI is indicated to assess the viability of the femoral head. For young patients osteotomy can be indicated whereas prosthetic replacement will be most adequate in elderly patients.

2. Intertrochanteric femur fractures

Diagnosis: Intertrochanteric fractures are rare in young patients but possible in high-energy trauma. In this context they usually come with more soft tissue injury, more fracture displacement and bony fragmentation than in elderly patients. If standard X-ray permits not to make the diagnosis MRI is nowadays the examination of choice for confirming or denying the fracture. A multitude of classifications exists with the Kyle and AO classification being the most widely used.

Treatment: Surgical stabilization is the treatment of choice. Early surgery (within 24-48h) is associated with a reduction in 1-year mortality in all but the most medically unstable patients. Sliding hip screws (SHS) and intramedullary nails are the implants most widely used but the 95° blade plate for reverse oblique fractures and modern angular stable (LCP) plates can be a valuable option. If a SHS or an intramedullary nail are used the correct placement of the screw in the femoral head are of greatest importance. Optimal position is determined by the tip-apex distance [Baumgaertner].



3. Fractures of the femoral diaphysis

Currently antegrade reamed intramedullary nailing is the treatment of choice for femoral shaft fractures with union rates of at least 95%, infection rates of < 1% and low clinically significant malunion rates.

Diagnosis: Patients with femoral shaft fractures have limb shortening, swelling and pain. Except in elderly patients the fracture is usually the result of high-energy trauma and associated injuries are common and should actively be looked for. At initial evaluation the pelvis, ipsilateral knee and foot, neurovascular status should be checked. Initial radiological workup should include hip and knee in order to rule out ipsilateral femoral neck fracture. In doubt CT scan of the pelvis.

Classification: Femoral shaft fractures are classified in proximal third, middle third or distal third fractures, most often the AO classification is used (32.A.1 – 32.C.3).

Treatment: Nonsurgical treatment with only traction can be used but comes with shortening, rotational malunion and knee stiffness and is thus not the treatment of choice. EX FIX is used in severe open fractures as well as for the initial stabilization in hemodynamically unstable poly- trauma patients and in patients with vascular injuries. Plating with MIPO technique has lately become more and more popular but in most hands results in a higher incidence of infection, nonunion and implant failure than does IM-nailing [Bostman]. Reamed antegrade nailing is considered to be the gold standard for the treatment of femoral shaft fractures. Retrograde femoral nails are mainly being used in obese patients, floating knee situations, ipsilateral shaft and neck fractures, pelvic ring fractures and in pregnancy. The results between antegrade and retrograde nailing are almost similar.

4. Intraarticular fractures of the distal femur

In younger patients usually due to high-energy trauma with associated local injuries (cartilage, ligaments, skin, muscle) and systemic complications. In elderly patients osteoporotic bone increases the difficulty of

successful treatment. In more complex intraarticular fractures preoperative planning with the help of a CT scan usually helps a great deal.

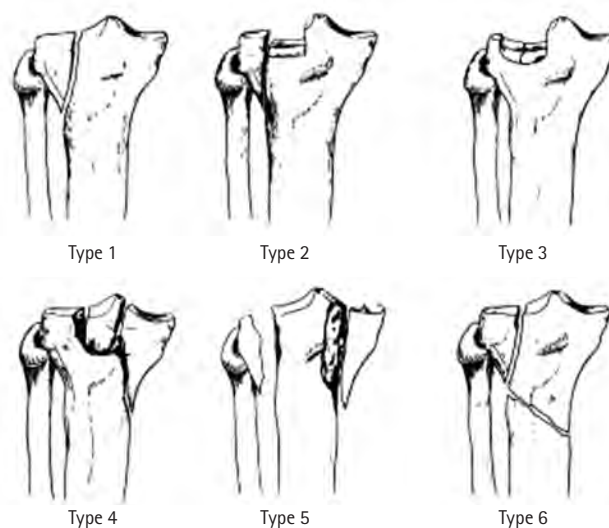
Classification: Usually the AO classification is being used.

Treatment: For successful treatment 4 conditions must be fulfilled: 1) anatomic reduction of fragments particularly joint fragments; 2) preservation of blood supply; 3) stable internal fixation; 4) early, active mobilization. Nowadays retrograde IM- nails and angular stable plates introduced in a MIPO technique are typically used. The 95°fixed-angle plate remains an option.



5. Fractures of the tibial plateau

These fractures occur when the femoral condyle impacts the tibia with a varus or valgus force. This can occur alone or combined with an axial compression force. Lateral plateau fractures are most common followed by bicondylar and finally medial fractures. Schatzker described in his simple and widely used classification system 6 different fracture types: Pure split fractures (Type I) occur mainly in young patients whereas split-depression fractures (Type II) are more common in the elderly. Type III describe pure depression fractures of the lateral plateau; Type IV stand for medial plateau, Type V fractures are bicondylar and finally type VI come with a metaphyseal-diaphyseal separation.



Treatment: Articular congruity, limb alignment and stability are paramount for a good functional result. If minor displacement is present (lateral fracture with <5° of valgus, articular incongruity

<3mm) non-surgical treatment with delayed weight bearing for 8 weeks followed by partial weight bearing for another 4 weeks can be chosen. Medial fractures with any displacement need to be fixed. Surgical treatment can be pure percutaneous screw fixation, with or without arthroscopic visualization or open/closed reduction with internal fixation. External fixation temporary or definitive using non-spanning hybrid fixators can be an option. The fracture fixation technique needs to be adapted to the type of fracture and the soft tissue situation. Patient with complex tibial plateau fractures should be informed that regardless of the fracture fixation mode chosen the potential for a poor outcome with early degenerative arthritis is high. These patients need a long-term follow up as posttraumatic arthritis can take up to 5-7 years to develop.

6. Fractures of the tibial diaphysis

Fractures of the tibial shaft are the most frequent long bone fracture and present with a wide amount of different problems from the minimally displaced closed fracture to the severe open fracture with bone loss and vascular lesion.

Classification: The most common classification scheme is the AO classification dividing bony injuries into simple (Type A), wedge (Type B) and complex (Type C) fractures. As there is a high amount of soft tissue injuries the Oestern/Tscherne classification for closed fractures and the Gustillio/Anderson classification for open fractures are being used.

Treatment: All tibia shaft fracture needs treatment. This can go from simple splinting to amputation. Acceptable displacement for conservative treatment with a cast or brace are: angular malalignment of $\leq 5^\circ$ in all planes, rotational malalignment within 10° , and < 10 - 12 mm of shortening [Brumback]. Plate osteosynthesis, after having fallen out of favor because of frequent soft tissue healing problems, presents with a revival since the introduction of MIPO techniques with LCP plates. Plates are usually used if there is metaphyseal extension of the fracture. External fixation is used for bony and soft tissue stabilization and can be used for definitive treatment but comes with a higher rate of malalignment than does IM nailing. Reamed locked nailing is actually the gold standard for the treatment of tibial midshaft fractures.

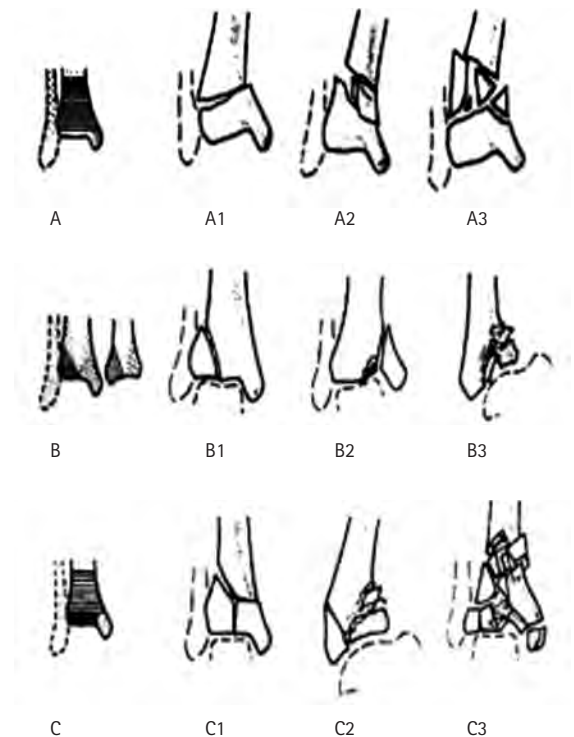
7. Fractures of the tibial plafond

These fractures, also referred to as pilon fractures, are defined by the disruption of the weight bearing articular surface of the tibia most commonly due to an axial loading mechanism. The ipsilateral fibula is fractured in 75% of the time [Rüedi].

Evaluation/classification: In pilon fractures look for associated lesion. Radiographic evaluation consists of standard X-ray exam but is often completed by CT-scan to provide the surgeon with a better understanding of the fracture for preoperative planning. AO classification is next to the Rüedi/Allgöwer classification the most widely used classification system.

Treatment: Nonsurgical treatment with cast and splint is generally chosen for non-displaced fractures or non-ambulators. If surgical treatment is opted for timing and soft tissue management are important.

The safest approach is to delay the operation until the soft-tissue envelope can tolerate surgical aggression. In the time between the accident and the definitive stabilization complex fractures are generally immobilized with joint spanning external fixation. This leaves the time for CT-evaluation and planning of the surgery (Span-Scan-Plan). Definitive fixation can be done with standard ORIF, MIPO, Hybrid Ex Fix or Ilizarov. The technique has to be adapted to the personality of the fracture and the soft-tissue condition.



Complications: Wound breakdown is a major problem after surgical treatment of pilon fractures (10%-15%). Infection occurs in 4% -35%. Often plastic surgeons need to be consulted in order to resolve soft-tissue problems. Malunion and nonunion can lead to the need for arthrodesis.

8. Open fractures

About 3% of the fractures are open [Court-Brown]. They are usually resulting from high-energy trauma, but can also result from simple low-energy trauma such as a fall [Court-Brown]. A fracture is considered to be open when disruption of skin occurs and soft tissue allows a communication between the bone and the outside environment [Okike]. As the body's protective barrier is broken, the potential for contamination in open fractures is high. Subsequently, the high prevalence of microbial contamination predisposes to the development of infection [Gustilo]. The incidences of infection of the bone are influenced by the type of fracture. The most commonly used classification of open fractures is the Gustilo classification. The classification is based on mechanism of injury, the degree of soft tissue damage, the configuration of the fracture, and the level of contamination. It has been modified several times to allow a more accurate prognosis for more severe fractures, i.e. type III injuries [Gustilo]. Management of open fractures is based on the following principles: assessment of the patient, classification of the injury, antibiotic therapy, debridement and wound management, fracture stabilization and supplemental procedures to achieve bony and soft tissue healing

preventing acute or chronic osteomyelitis [Giannoudis]. The management of open fractures always needs a team approach with an orthopaedic trauma surgeon, ID- specialist, microbiologist, plastic surgeon and in 3C open fractures vascular surgeons.

Classification of open fractures

Type 1:

- Skin opening of 1 cm or less, quite clean
- Most likely from inside to outside
- Minimal muscle contusion
- Simple transverse or short oblique fractures

Type 2 :

- Laceration more than 1 cm long, with extensive soft tissue damage, flaps or avulsions
- Minimal to moderate crushing component
- Simple transverse or short oblique fractures with minimal comminution

Type 3:

- Extensive soft tissue damage including muscles, skin, and neurovascular structures.
- Often a high-velocity injury with severe crushing component

3 Subgroups

- IIIA
 - Extensive soft tissue laceration
 - Adequate bone coverage
 - Segmental fractures
 - Gunshot injury
- IIIB
 - Extensive soft tissue injury with periosteal stripping and bone exposure
 - Usually associated with massive contamination
- IIIC
 - Vascular injury requiring repair

intramedullary nailing for definitive management of closed fractures of the femoral and tibial shaft. J Am Acad Orthop Surg 2006; 14(10) 124-7.

9. Borens O, Kloen P, Richmond J, Roederer G, Levine DS, Helfet DL. Minimally invasive treatment of pilon fractures with a low profile plate: Preliminary results in 17 cases. *Arch Orthop Trauma Surg* 2009; 129(5) 649-59.
10. Court-Brown CM, Bugler KE, Clement ND, Duckworth AD, McQueen MM. The epidemiology of open fractures in adults. A 15-year review. *Injury* 2012; 43(6):891-7.
11. Court-Brown CM, Rimmer S, Prakash U, McQueen MM. The epidemiology of open long bone fractures. *Injury* 1998; 29(7):529-34.
12. Okike K, Bhattacharyya T. Trends in the management of open fractures. A critical analysis. *J Bone Joint Surg Am* 2006; 88(12):2739-48.
13. Gustilo RB, Merkow RL, Templeman D. The management of open fractures. *J Bone Joint Surg Am* 1990; 72(2):299-304.
14. Gustilo RB, Mendoza RM, Williams DN. Problems in the management of type III (severe) open fractures: a new classification of type III open fractures. *J Trauma* 1984; 24(8):742-6.
15. Giannoudis PV, Papakostidis C, Roberts C. A review of the management of open fractures of the tibia and femur. *J Bone Joint Surg Br* 2006; 88(3):281-9.

References

1. Caviglia HA, Osorio PQ, Comando D. Classification and diagnosis of intracapsular fractures of the proximal femur. *Clin Orthop Relat Res* 2002; (399): 17-27.
2. Cho MR, Lee SW, Shin DK, et al. A predictive method for subsequent avascular necrosis of the femoral head (AVNHF) by observation of bleeding from the cannulated screw used for fixation of intracapsular femoral neck fractures. 2007.
3. Baumgaertner MR, Curtin SL, Lindskog DM, et al. The value of the tip-apex distance in predicting failure of fixation of pertrochanteric fractures of the hip. *J Bone Joint Surg (Am)* 1995; 77(7): 1058-1064.
4. Collinge C, Liporace F, Koval K, Gilbert GT. Cephalomedullary screws as the standard proximal locking screws for nailing femoral shaft fractures. *J Orthop Trauma* 2010; 24 (12): 717-22.
5. Weinlein J, Schmidt A. Acute compartment syndrome after tibial plateau fracture – beware. *J Knee Surg* 2010; 23 (1) 9-16.
6. Thomas Ch, Athaniso A, Wulschlegler M, Schuetz M. Current concepts in tibial plateau fractures. *Acta Chir Orthop Traumatol Cech* 2009; 76 (5): 363-73.
7. Musahl V, Tarkin I, Kobbe P, Tzioupis C, Siska PA, Pape HC. New trends and techniques in open reduction and internal fixation of fractures of the tibial plateau. *J Bone joint Surg (Br)* 2009; 91 (4): 426-33.
8. Della Rocca GJ, Crist BD. External fixation versus conversion to



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Fractures: Pilon, ankle, talus and calcaneus

1. Fractures: Ankle

Malleolar injuries generally result from rotational forces.

Injuries comprise both bony and soft tissue structures.

Bony anatomy: Medial malleolus, fibula/lateral malleolus, posterior malleolus.

Soft tissue anatomy: Collateral ligaments (deltoid; talofibular and calcaneofibular ligaments; syndesmotic complex consisting of anterior and posterior inferior tibiofibular ligaments, interosseous ligament, interosseous membrane.

Radiographs: Anteroposterior, lateral and mortise (internal rotation 15°) views. Must have full leg films. Key factors to look at: Medial clear space, tibiofibular overlap, posterior articular surface of tibia (posterior malleolus), fibular length.

Classification: AO: 44-A (Weber A), Infrasyndesmotic: The fibular fracture is below the syndesmotic ligament; 44-B (Weber B), Transsyndesmotic fibular fracture: The fibular fracture begins at the level of the joint line and extends proximally. The integrity of the syndesmotic ligaments cannot be determined from radiographs; 44-C (Weber C), Supra-syndesmotic fibular fracture. The fibular fracture is above the syndesmotic ligaments. A very high Type C fracture is also called a Maisonneuve fracture. The fibula fracture is at the neck of the fibula and the medial lesion is either a rupture of the deltoid ligament or a fracture of the medial malleolus.

Goal of treatment is to restore normal articular anatomy of the ankle joint. Fibula must be restored to normal length and rotation. Integrity of the tibiofibular bond (syndesmotic ligaments and interosseous membrane) must be assured. Medial malleolus demands an anatomic reduction. Posterior malleolus requires anatomic reduction if fragment size is >20% of articular surface.

Patient is placed in the supine position with a flank bump. Tourniquet is used. Meticulous care for the soft tissue envelope is mandatory (little or no retraction).

Weber A: The fibular fracture is transverse. Fixation is with tension band wire, lag screw, or plate, or no fixation if nondisplaced. An associated medial malleolar fracture will be an axial shear with possible small impaction of articular surface at medial corner of joint. This requires open reduction internal fixation (ORIF) with lag screws and usually an antilag 1/3rd tubular plate. Any impacted articular surface must be reduced prior to rigidly fixing the medial malleolus.

Weber B: The fibular fracture is oblique from proximal posterior to distal anterior. It requires anatomic reduction restoring length and rotation. Fixation is in one of three ways: (1) Two or more lag screws without plate; (2) A single anterior to posterior lag screw and 1/3 tubular plate laterally as neutralization; or (3) Posterior antilag 1/3rd tubular plate

with posterior to anterior lag screw. It is then mandatory to check for syndesmosis stability by means of a hook test (see below).

Weber C: The fibular fracture requires an anatomic reduction restoring length and alignment. Fixation depends upon the fracture pattern. Will almost always require a plate (1/3rd tubular or 3.5mm standard plate), and lag screw if possible. By definition, the syndesmosis is unstable, but can be confirmed by a hook test.

Hook test is performed after reduction and fixation of the fibula. A small hook is used to pull the fibula away from the tibia while controlling movement with the image intensifier. If the tibiofibular connection is disrupted the talus will follow the fibula laterally.

Stabilization of the syndesmosis with position screws: Generally use a single 3.5mm cortical position screw through three cortices (lateral fibula, medial fibula, lateral tibia). In cases where the fibula has not been internally fixed, such as with a Maisonneuve fracture, one should use a 3.5mm cortex screw through four cortices (the screw passes through the medial tibial cortex), and consideration given for two 3.5mm screws.

After stabilization of the lateral complex in a Weber B or C fracture any associated medial malleolar fracture should be treated by ORIF. An anatomic reduction is required and fixation is usually by means of two 4.0mm partially threaded cancellous screws. With smaller fragments a tension band wire can be used. If the medial injury is a deltoid ligament rupture it is treated nonoperatively.

If the posterior malleolar fracture involves >20% of the articular surface of the distal tibia it should be fixed with one or two screws (depending upon the size of the fragment) from either anterior to posterior or posterior to anterior. The screws should be inserted so as to provide interfragmentary compression (lag screws). In some cases, particularly in more osteoporotic bone, a posterior antilag plate could be used.

Beware of an "isolated" lateral malleolar fracture and normal medial clear space. There may be an associated deltoid ligament rupture ("bimalleolar equivalent") and the patient should have a stress test (eversion/external rotation) to see if there is a lateral talar shift. If there is no talar shift the fracture can be treated nonoperatively.

Postoperative management consists of a posterior splint for six weeks (to prevent ankle equinus and also to allow for good soft tissue healing), during which ankle range of motion exercises are permitted. Very limited weight-bearing (10kg) is permitted. At six weeks a follow-up radiograph is obtained, and if all is satisfactory the splint is discarded and progressive weight-bearing is begun.



Illustrative case (pictures 1, 2, 3 and 4): 54 yo male who sustained an ankle injury. Preoperative radiographs (pictures 1 and 2) show a Weber B bimalleolar fracture. ORIF was performed and the postoperative radiographs show anatomic reduction and stable fixation (pictures 3 and 4).

2. Fractures: Pilon (Plafond)

These are intraarticular fractures of the weight-bearing surface of the distal tibia.

Two types: (1) Rotational type due to a low energy injury with little comminution and few articular fragments. There is usually a fracture of the fibula. Less soft tissue disruption; (2) Axial compression type due to a higher mechanism of injury. There is extensive bony and joint comminution and impaction. There is greater soft tissue damage. There may or may not be a fibular fracture. Frequently the fracture is a combination of these two types.

Fracture analysis requires plain radiographs and CT scan in every case.

Classification: AO 43-B or C (cannot be a Type A because those are extraarticular fractures). 43-B are partial articular, and 43-C are complete articular fractures (no part of the articular surface remains in contact with the shaft).

Treatment largely depends upon the soft tissue envelope (soft tissue injuries with closed fractures are classified according to Tscherne). Generally there is little to no indication for immediate ORIF of pilon fractures. Lesser energy injuries can be managed by splint and elevation, while higher energy injuries should be treated with an ankle-spanning

external fixator. Two anteromedial half-pins should be placed very proximal in the tibia, and a single through and through Steinmann pin placed in the calcaneus (from medial to lateral as far posterior as possible). The pins are connected by a delta or rectangular frame.

Timing of surgery depends upon the resolution of the soft tissue injury. Signs to look for include resorption of edema as seen by the "wrinkle" test, and early healing of any associated blisters. With the higher energy injuries this may require between 10-20 days.

The goal of surgery is first to restore articular congruity by reduction of impacted articular fragments. This is performed by working superior to the impacted articular surface so as to bring it down to the level of the joint and use bone graft or bone graft substitute to support the reduced articular surface. Temporary K-wires are used to maintain the reduction before plate fixation.

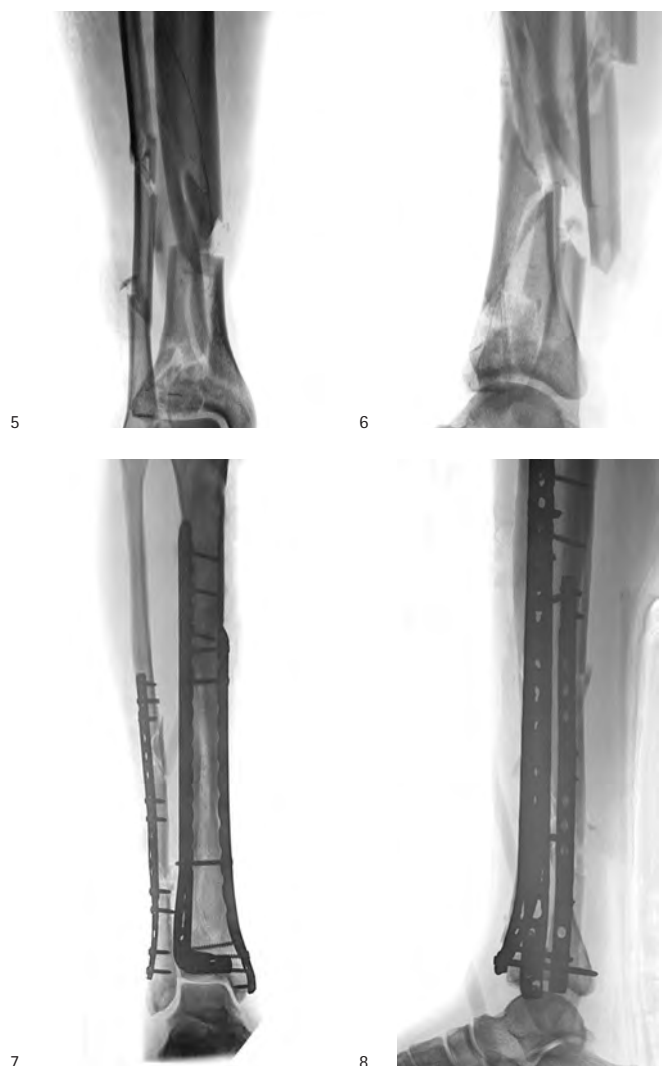
Surgical approaches: Isolated fractures of the lateral column can be addressed by a straight antero-lateral incision. From the same approach ORIF of the fibula can be performed. Isolated fractures of the medial column can be approached by the AO anteromedial approach. Fractures involving both medial and lateral columns require a more extensive surgical incision or combination of incisions. The possibilities include (1) a mini anteromedial incision associated with an anterolateral incision; (2) the extensile approach as described by Assal and Stern; and (3) less commonly a posterolateral incision.

Definitive fixation requires anatomically contoured locking plates. Plate position is determined by its function as a buttress. For fractures with varus (tension deformity of fibula) a medial plate is necessary. For those fractures in valgus (compression deformity of fibula) an anterolateral plate is necessary as a lateral buttress. For the more complex injuries two plates are necessary. Soft tissue management during surgery requires very careful handling of the soft tissues without the use of retractors. K-wires can be positioned in the bone and bent to hold open the soft tissue envelope without pulling on the tissues.

There is debate about the need to fix any associated fibular fracture, and if so, when? The original idea behind fixing the fibula at the time of initial external fixation was to reduce the lateral joint fragment (Chaput) so as to use it to key in the reduction of the rest of the pilon. However, malreduction of the fibula may well make later reduction of the pilon more difficult. Therefore many believe that the fibula should not be fixed at the time of provisional external fixation, but should be fixed after ORIF of the pilon to provide further stability to the construct.

Postoperative care includes application of a splint with the ankle in at least 5° dorsiflexion to prevent late equinus deformity. The limb should be kept elevated until there are no signs of postoperative edema. Ambulation can then be started with 10kg limited weight-bearing.

As far as prognosis is concerned, the outcome following pilon fractures will depend upon whether or not there is primary articular cartilage damage. If so, poor results frequently follow even good surgery.



Illustrative case (pictures 5, 6, 7 and 8): 34 yo female who sustained an axial load injury from falling off a tree. Preoperative radiographs (pictures 5 and 6) show an AO 43-C complete articular fracture of her distal tibia. ORIF was performed and the postoperative radiographs show anatomic reduction and stable fixation (pictures 7 and 8).

3. Fractures: Talus

Injuries are typically high energy and one should search for other musculoskeletal injuries.

The talus articulates with seven joints: both malleoli, the pilon, the three subtalar joints, and the navicular.

The talus is mostly articular (70% covered by cartilage). Any fracture is an articular fracture.

The talus is composed of three parts: (1) Body, which includes the dome; (2) Neck; and (3) Head.

Fracture analysis includes anteroposterior, lateral and mortise views of the ankle, as well as anteroposterior, lateral and Canale (specific view of the talar neck) views of the foot. A CT scan is essential to clearly understand fracture morphology.

A particular problem with fractures of the talus relates to its blood supply, which while is both extra- and intraosseous it is prone to disruption by the injury. The blood supply is coming from the three main arteries of the leg: the tibialis anterior, tibialis posterior, and the fibular arteries. One of the complications of fractures of the talus is avascular necrosis of the talar body, as well as osteoarthritis.

Talar body fractures: Generally caused by axial loading secondary to a fall from a height or a motor vehicle accident. It is frequently associated with either tibiotalar or subtalar dislocation. Closed reduction of any dislocation should be performed immediately after plain radiographs are available, and before CT imaging. If there is no stability after closed reduction, or there is soft tissue compromise, a provisional external fixator should be applied as provisional fixation. Definitive reduction and fixation can wait until soft tissues are stable.

For definitive surgery anatomic reduction with stable internal fixation is the goal. The surgical approach will be dictated by the nature of the fracture. There are two basic approaches: (1) Medial approach through the tibialis anterior and tibialis posterior, which can be extended proximally to incorporate a medial malleolar osteotomy for better visualization of the talar dome; (2) Anterolateral approach just lateral to the extensor digitorum longus and extends from anterior to the lateral malleolus towards the fourth toe; and (3) Posteromedial approach with the patient supine in the interval between the posterior tibial tendon and flexor digitorum longus tendon. It addresses those fractures involving the very back of the talus (Posteromedial talar body fracture).

The fractures are first preliminarily stabilized with K-wires. Definitive fixation has changed from large fragment screws and plates to small and mini implants. Any screws that must pass through articular cartilage have to be countersunk to avoid prominence of the screw head. Bone grafting is occasionally required to support reduced articular segments that have been disimpacted. If a medial malleolar osteotomy is performed local cancellous bone can be used as graft.

Postoperative management includes a posterior splint. The patient is advised to ambulate with no more than 5kg of weight-bearing and remove the splint daily for range of motion exercises.

Talar neck fractures: They account for 50% of all talar fractures. While neck fractures are not intraarticular, they disrupt the normal relationship between the articular facets of the subtalar joint. They are usually cause by forceful dorsiflexion, such as with higher energy motor vehicle accidents when the foot is pushing the brake pedal (historically with early airplane crashes and thus given the name, "aviator's astragalus"). Anatomic reconstruction is critical to restore proper joint mechanics.

Typically patients present with swelling and gross deformity. All obvious dislocations should be reduced as an emergency after obtaining plain radiographs, and before obtaining CT scans. The standard radiographs are mentioned above.

The most widely used classification of talar neck fractures is that of Hawkins. Three groups were originally described. Group 1 fractures are nondisplaced (absolutely nondisplaced; not common). Group 2 are talar neck fractures where the talus is subluxated or dislocated from the subtalar joint. Typically there is a varus and rotational deformity. Group 3 are talar neck fractures where the body of the talus is dislocated from both the subtalar and ankle joints. Immediate closed reduction should be attempted as an emergency, and if not possible emergent open reduction is required. The talar body may have dislocated Posteromedial compressing the neurovascular bundle. Later, a "Type" 4 was added by Canale and Kelly, thus confusing "groups" with "types." Group 4 fractures have all the characteristics of Group 3, with the addition of a dislocation of the head of the talus at the talonavicular joint.

Definitive surgical treatment is almost always performed with two incisions, medial and lateral (dual approach). The medial incision is made between the tibialis anterior and posterior tendons, thus providing exposure to the medial neck. Fractures are typically more

comminuted medially as the neck displaces into varus. This incision can be carried proximally in order to perform a medial malleolar osteotomy. A lateral incision begins just anterior to the fibula, lateral to the peroneus tertius and common extensor tendons and extends distally to the fourth toe. The lateral incision allows for evaluation of length and alignment as there is less comminution laterally. One must be careful not to injure the superficial peroneal nerve.

Stripping of soft tissue should be avoided to preserve the remaining talar blood supply.

Fixation can be from distal to proximal or vice-versa. With distal to proximal fixation screws will pass through the articular surface of the talar head and thus must be countersunk below the cartilage. Lag screws can be used for simple fractures, but with comminution one should not use lag screws but instead fully threaded position screws to avoid shortening of the neck. Alternatively, screws can be placed from the posterior aspect of the talus across the neck fracture into the head. These screws can be placed just inferior to the posterior articular cartilage. In fractures with greater comminution minifragment plates are used to preserve length and alignment and no compression is used. Postoperative management is similar to that described above for talar body fractures.



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Illustrative case (pictures 9, 10, 11 and 12): 22 yo male involved in a car accident. Preoperative radiographs (picture 9) show a talar neck fracture type Hawkins III, both subtalar and tibio-talar joints being dislocated. ORIF was performed through combined medial and lateral incisions (dual approaches). The postoperative radiographs show anatomic reduction and stable fixation (pictures 10, 11 and 12).

4. Fractures: Calcaneus

The calcaneus is the most frequently fractured tarsal bone (60% of all tarsal fractures). Most intraarticular calcaneus fractures are secondary

to an axial load applied directly to the heel, and usually from a fall from a height or motor vehicle accident. Extraarticular fractures are caused more frequently by a twisting or avulsion.

Intraarticular fractures result in two primary fracture lines. The first starts at the crucial angle of Gissane and results from the lateral process of the talus acting as wedge dividing the calcaneus into an anterior and a posterior half. The anteromedial fragment remains in its relationship with the talus, and the posterolateral fragment displaces laterally into varus. Secondary fracture lines form in the sagittal plane along the length of the calcaneus in an anterior-posterior direction. They may extend into the calcaneocuboid joint, split the anterior facet, or exit medially or laterally through the body. Higher energy fractures result in greater comminution.

One must be aware of additional musculoskeletal injuries as axial loading can frequently result in additional injuries to the lower extremities or lumbar spine (careful examination!).

Initial treatment for all calcaneus fractures should be application of a bulky dressing and splint and the limb elevated. Most patients require hospital admission. One must carefully check the neurovascular status and also be alert for possible development of a compartment syndrome (reported to occur in up to 10% of patients with a calcaneus fracture). One must periodically assess the soft tissue envelope as these high energy injuries result in substantial amounts of edema and hematoma, and frequently result in the development of fracture blisters. If the soft tissues are threatened from within by a displaced fracture fragment urgent reduction is required to avoid full-thickness skin necrosis. Fortunately, open fractures are not common. If present, the traumatic wound tends to be medial caused by a sharp spike of the medial wall created by the primary fracture line as the tuberosity dislocates laterally.

Radiographic examination consists of plain radiographs which include a lateral of the foot and ankle, a mortise view of the ankle, and dorsoplantar and oblique X-rays of the foot. In addition, a Harris or axial view is obtained. Contralateral foot and ankle views may be obtained for comparison. A CT scan is mandatory to understand the fracture morphology.

There are primarily three classification systems for calcaneus fractures. The first is from Essex-Lopresti where intraarticular fractures are classified into two broad types, tongue-type and joint depression. It helps to point out those fractures that might be amenable to minimally invasive techniques. A second classification is that of Sanders, and is based upon the number and location of posterior facet fragments as seen on the CT scan. Third is the AO classification where the calcaneus is coded as 81.2, and fractures are divided into types where A are extraarticular fractures, B are intraarticular fractures, and C represent fracture-dislocations. Further subdivision into groups and subgroups follows from these broad types.

Definitive treatment of calcaneus fractures is either nonoperative or operative. The factors to consider in the treatment decision relate to the patient, the fracture, and surgeon experience. Patient factors include the ability to understand the injury and cooperate with treatment, and situations that might affect wound healing such as smoking, diabetes, and peripheral vascular disease. The type of fracture will affect decision-making as well. While extraarticular fractures are frequently treated nonoperatively, the same might be applied to fractures with marked displacement and comminution as these frequently do poorly regardless of treatment. And, as with all fracture treatment, surgeon experience plays an important role.

Extraarticular fractures: The most common is the anterior process fracture. Depending upon the size of the fracture fragment and possible comminution, treatment varies from nonoperative, to ORIF, to excision of small fragments. They often are initially missed and patients present after some time because of persistent pain. Another extraarticular fracture is the calcaneal tuberosity. These are often only minimally displaced and may be treated nonoperatively. Displaced fractures represent an emergency because pressure from the tuberosity fragment on the thin posterior soft tissue envelope may rapidly lead to a full-thickness necrosis.

Intraarticular fractures: For those fractures deemed best treated nonoperatively, the foot and ankle are wrapped in a bulky compression dressing, splinted, and elevated. As the edema subsides and patients are more comfortable, early range of motion of the ankle and subtalar joints is begun, as well as movement of the muscles of the foot. Splinting is continued to avoid an equinus deformity. Weight-bearing is delayed until radiographic signs of healing, between two and three months. The operative treatment of calcaneus fractures is divided into open and percutaneous techniques. Additional operative techniques include external fixation and primary subtalar arthrodesis.

Open reduction internal fixation is usually performed through an extensile lateral approach. There can be a medial approach for certain fractures where it allows for direct visualization of the medial wall and direct reduction of the tuberosity, but this approach does not allow for visualization of the subtalar joint or for decompression of the displaced lateral wall. Additionally, the risk with a medial approach is damage to the neurovascular structures. The extensile lateral approach utilizes a lateral flap based on the calcaneal artery. An L-shaped incision is made over the lateral calcaneus and a full-thickness flap (skin, peroneal tendons, sural nerve, periosteum) is sharply elevated off the lateral wall. This approach allows for direct visualization of the entire subtalar joint from anterior process to calcaneal tuberosity. The major risk of this incision is related to healing of the lateral flap. No retraction of the flap should be performed but K-wires fixed into the bone and bent to 90° can be used to hold back the flap. Fracture fixation is with lag screws and bridging plates, and locking plates may be advantageous in more comminuted fractures or bone of less than good quality. There is no general consensus about the use of bone graft (or bone graft substitute) to fill in the defect following elevation of depressed joint fragments. There is some interest in using calcium phosphate cements in this regard to allow for earlier weight-bearing, although there are no definitive studies showing this to be advantageous.

Minimally invasive or percutaneous techniques may be advantageous in patients deemed poor candidates (see above) for ORIF. They are best used for the Essex-Lopresti tongue-type fracture. This method is best used early before organization of the fracture hematoma. One does not achieve an anatomic reduction of the fracture, but aims to provide satisfactory alignment without the risk of soft tissue complications. Similar percutaneous approaches have been described.

External fixation is not common for definitive treatment, but may be used for some open fractures of the calcaneus or in patients with a poor soft tissue envelope. Pins are placed in the tibia, through the tuberosity, and midfoot. If external fixation is used for provisional fixation of, for example, an open fracture, then one should not place pins in the calcaneus itself as it will compromise definitive exposure. In such cases K-wires can be used (but not in the calcaneus) along with bridging external fixation from tibia to midfoot (no pins in the calcaneus).

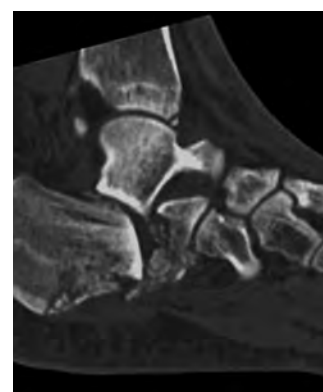
Primary subtalar arthrodesis is a possible option for the more severe

comminuted fractures such as Sanders type 4 fractures). However, others suggest that the results are improved in an ORIF is first carried with the attempt to restore the overall normal shape of the calcaneus, and then performing a subtalar fusion if the need arises after the fracture has healed.

Complications of the fracture itself, regardless of the type of treatment, include malunion (widened heel, hindfoot varus, loss of heel height), and post-traumatic subtalar arthritis. Complications of operative treatment include malunion (as noted above), subtalar arthritis, nerve injury (sural nerve laterally, posterior tibial nerve medially), and wound healing problems with resultant soft tissue and bone infection. Unfortunately, poor results may be the outcome even after careful surgery and an anatomic reduction.



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Illustrative case (pictures 13, 14, 15 and 16): 35 yo male involved in a motobike accident. Preoperative radiographs (pictures 13 and 14) show a calcaneum fracture (Essex-Lopresti tongue-type fracture). ORIF was performed 12 days after the injury. The postoperative radiographs show anatomic reduction and stable fixation (pictures 15 and 16).

References

1. *AO Principles of Fracture Management*, T. Ruedi, R. Buckley and C. Moran (Editors), AOPublishing, Thieme, Stuttgart, 2007
2. *Master Techniques in Orthopaedic Surgery, Fractures*, DA. Wiss (Editor), Second Edition, Lippincott Williams Et Wilkins, Philadelphia, 2006



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Wrist fractures: Distal radius and scaphoid

Distal radius fractures

The recent developments of many osteosynthesis and fixation devices are the reason or the consequences of the rapid changes in the treatment of distal radial fractures (DRF). From a relative conservative policy of treatment we are facing now a very aggressive treatment with open reduction and internal fixation. Confusion is very often done between the different fractures types, the character of the injury and not least the patient groups, their age and activities level or expected activity levels. We have to be careful and not treat all patients in the same way using the latest implant presented on the market. We cannot deny the economical factor and accept that this abundance of implants is driven by the manufacturers who envisage a huge new market. However we are still missing evidence based research which argues definitively for the new approach.

1. Epidemiology

Distal radial fractures are common fractures accounting for about one-sixth of the fractures treated at emergency rooms or one-tenth of the total number of fractures in adults over 35 years. The incidence of DRF is approximately 19–43 per 10000 inhabitants annually with females outnumbering males in overall distribution 4:1. In Sweden, the incidence in the city of Malmö had almost doubled from the 1950s to the 1980s. This change over time could not be explained by an increase in diagnosed DRF as the incidence of shaft fractures of the forearm remained the same. The overall ageing of the population and an increased incidence of osteoporosis may offer an explanation. This trend can be reversed with community interventions which promote health-education programs that address dietary intake, physical activity, smoking habits and environmental risk factors for osteoporosis and falls. Over the last decades there has been an increase in incidence especially in the age group greater than 60 years. The higher incidence among older women could be explained by the increasing incidence of osteoporosis. A screening of patients with wrist fractures between the ages of 50 –75 years revealed that only 19% had normal BMD in the hip and vertebrae. The occurrence of a DRF can be used as a predictor for a later hip fracture. In a Swedish study an overall relative risk to sustain a hip fracture after a previous DRF was 1.54 for women and 2.27 for men and in an American study the relative risk for a hip fracture was 1.4 for women and 2.7 for men. For DRF in younger patients the proportions of men and women are equal. These fractures are often the result of a high energy trauma and should therefore be treated differently than the osteoporotic fractures. The fractures in younger patients are more often intra-articular and associated with a high incidence of ligamentous injuries with the scapho-lunate ligament being the most commonly injured. As the fracture is so common, it imposes large costs to society. In Sweden, the costs in the year following the fracture were 2147€,

including both direct and indirect costs resulting in an annual cost to the country of about 50 million Euro for the adult (7,26 million persons) population (November 2007). However, costs for fractures after the first year, such as costs for surgery of malunions, are not taken to account. With an increasing proportion of elderly people, not only in the western communities but also in the developing countries, the DRF remains an important and increasing economical problem that has to be assessed. However, not only the costs of the fracture are of importance, but also the outcome and disability from the patients' perspective and therefore reliable objective measurements are of importance.

2. Results and how to measure it

The final result of a fracture can be difficult to define and measure. The type of the injury, the expectation of the patient and/or the medical team may have an impact on the real appreciation of the quality of the result. Various modalities have to be considered, such as the subjective, objective and economical outcome; a broad view which incorporates pain, range of motion and cosmetic appearance was suggested by Colles as "One consolation only remains, that the limb will at some remote period again enjoy perfect freedom in all of its motions and be completely exempt from pain: the deformity, however, will remain undiminished through life". This description of the outcome following a DRF is still valid today as found and described by Kopylov et al. in a 30 year follow up of 76 patients with most patients experiencing a good long-term outcome. In a shorter perspective it is somewhat different. In our practice and for research purposes we use the following tool in assessment of the results.

2.1. Objective parameters: The range of motion is measured in the three axes of rotation around the wrist joint. Extension and flexion as well as radial and ulnar deviation take place in the radio-carpal joint and were measured and expressed as one parameter as these could be regarded as one motion around the radio-ulnar and dorso-volar axis. Forearm rotation takes place in the distal and proximal radio-ulnar joints around the longitudinal axis. Grip strength, the next objective clinical parameter of interest, is measured with the Jamar dynamometer, expressed in kg and related to the strength of the contra-lateral hand. Grip strength in an older population has been shown to correlate well to the health related quality of life measured by the SF- 36.

2.2. Radiographs: Radiographs were first used for examination of DRF at the end of the nineteenth century. Since then, radiographic examination has improved technically and forms a basis of classification and outcome. However, it has in some studies been shown to correlate poorly with final clinical outcome and the inter-observer reliability and intra-observer reproducibility of different radiographic classifications is low. In a recent study, the radiographic appearance in the initial radiograph, radial shortening >2mm, dorsal angulation >15 degrees, and

radial angulation >10 degrees were each significantly associated with a poorer DASH score.

2.3. Subjective parameters: In recent years there has been interest in the development of patient related outcome scores – generic, region specific and organ or joint specific. The DASH is one of the most commonly used region specific scoring systems for the upper extremity. DASH is an abbreviation for Disabilities of the Arm Shoulder and Hand, initially published, and later corrected, as the Disabilities of the Arm, Shoulder and Hand. DASH is a self-administered questionnaire developed by the AAOS and the Institute for Work & Health in Canada (<http://www.dash.iwh.on.ca/>). DASH has been translated and validated in many languages for general use in upper extremity disorders but not specifically for DRF. A change in mean DASH score of 10 points after an intervention such as surgery is considered as minimally important change. As the original DASH with 30 questions and items, is sometime perceived as difficult to work with and time consuming, a shorter form, QuickDASH has been developed. It consists of eleven questions from the original DASH and correlate excellently with the standard DASH.

3. Treatment alternatives

3.1. Non Invasive techniques

3.1.1. Conservative: Closed reduction and splinting is still today the most commonly used method of treatment in the DRF. The type of splinting is of importance as is the position to immobilize. In supination there is less likelihood of radiolocation. In the Cochrane data base report on closed reduction methods, only three randomized or quasi-randomized studies were found including 404 patients. Many methods of closed reduction have been developed during the years but there is no evidence based on randomized studies to support the choice of a closed reduction method. Handoll and Madok found more studies [33], when also systematically evaluating non-randomized reports of methods of closed reduction. Even in this study, there is no robust evidence to support any treatment in favour to another and the authors simply recommend the use of a method with which the practitioner is familiar. In many cases conservative treatment, however, is not enough and especially for primarily or secondarily unstable fractures, surgical options are needed.

3.1.2. External fixation: External fixation of DRF has been in use for more than three decades. In Sweden, it is considered to be the standard method for operative treatment of the fracture- and for this reason it can be chosen as the method of reference to which newer methods can be compared. External fixation uses ligamentotaxis to both reduce as well as to keep the fracture in position during healing. The recommended time for immobilization varies, ranging from 4 weeks to 6 weeks. In general, long immobilization time increases the risk for reflex sympathetic dystrophy (RSD). The traction of the wrist ligaments may cause stiffness and therefore dynamic fixation with an articulated device or non-bridging fixation has been proposed with better results reported than for traditional bridging technique. A recent randomized study was unable to find any difference between the bridging and the non-bridging external fixator in regard to clinical results in elderly patients.

3.1.3. Pinning: Other closed reduction techniques includes fixation of the fracture by pinning. Various techniques have been described such as intrafocal pinning, intrafocal intramedullary pinning or pinning in combination with external fixation. In the Cochrane report on percutaneous pinning of DRF it is stated that the high rate of complications casts some doubt on their general.

3.2 Open Surgery

3.2.1. Plates: For volarly dislocated fractures especially of the Barton or Smith type, a volar plate is preferably used. For other types of DRF, other techniques have been considered. Standard AO-plates and screws can be used with good results, however, to get a good stability, usually two or more columns of the radial cortex has to be fixated to achieve good results.

3.2.2. Fragment specific: A fragment specific system addresses the radial and ulnar columns separately as well as single fracture fragments both dorsally and at the volar rim by a combination of plates, pins and screws. It is primarily based on pinning of the fracture but since additional stability is needed to prevent the pins from bending or the fragments from sliding on the pins, a stabilizing plate to secure the pins has been added. In addition, wire forms to support the subchondral bone or small fragments can be used. The system is low profile and offers good stability. The surgical approach is determined by the type of fracture and the type of fixation needed to address the fragments.

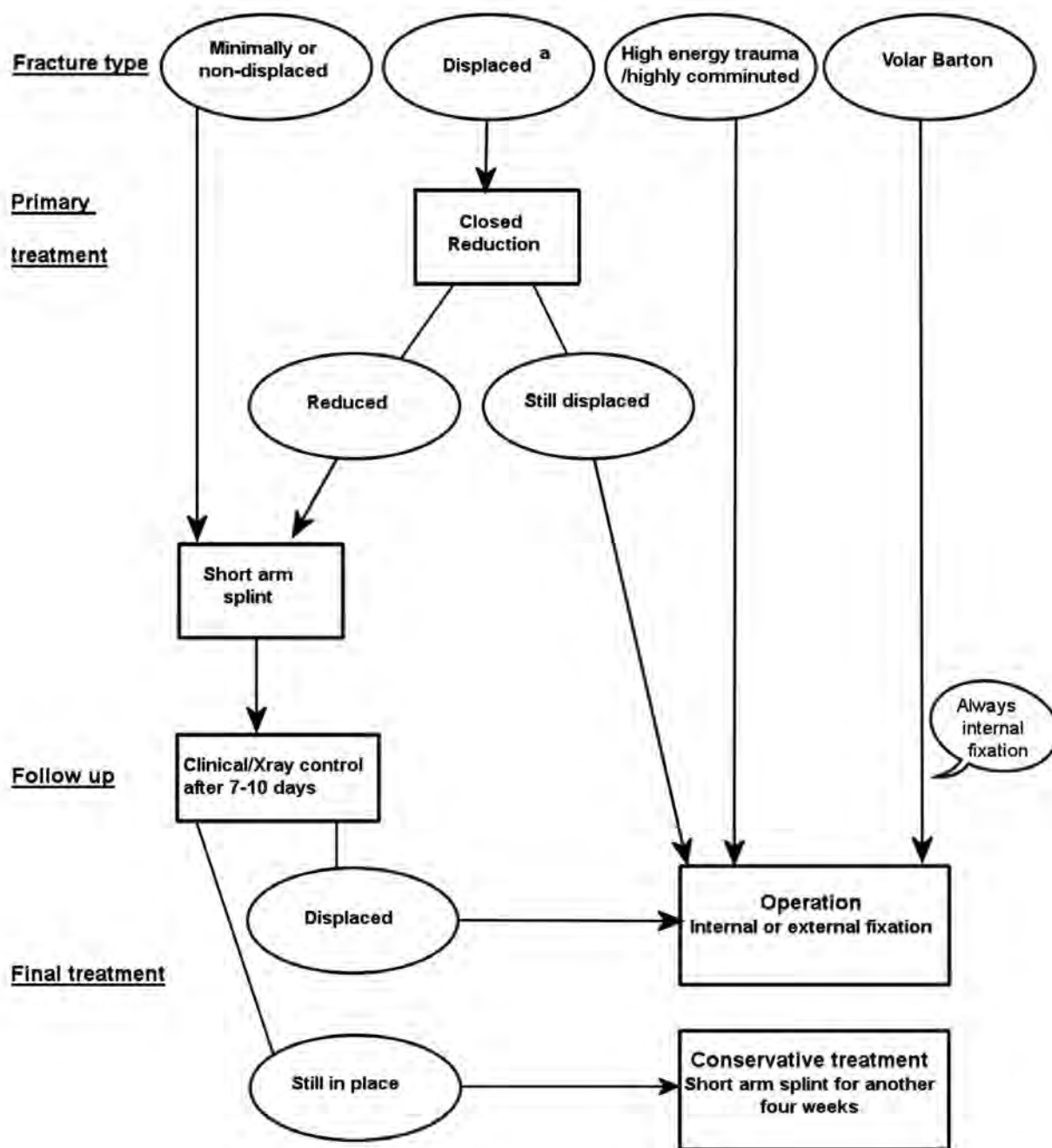
3.2.3. Volar locking plates: The newest concept, the volar locking plates with angle stable screws or pegs is becoming widely used as it offers stability and a safe approach to the fracture. The fracture is approached from the volar side using the Henry approach just radially to the flexor carpi radialis, ulnarly to the radial artery. This offers an easy access to the volar part of the radius. The volar locking plate has, in biomechanical testing, been shown to be sufficiently stable for fixation of the dorsally comminuted fracture and has been shown to offer equivalent stability when compared to the fragment specific fixation.

4. Future

For many it seems that the volar locking plates as given the final solution to the treatment of DRF. From the existing literature it is clear that volar locking plates can be used successfully in both intra and extraarticular DRF. However this treatment is not without complications. The evolution of treating DRF according to the reports in conferences and the publishing results is to treat all fractures undepending types, injury mechanism or age by the same method: open reposition and internal fixation with in the most cases volar locking plates. Using this approach we sure overtreat an undefined number of patients with an increased morbidity and potential complication rates and without control of the potential benefits in term of increase quality of the results for each patient. Therefore, studies of sufficient quality are lacking particularly in important broad diagnoses such as the DRF. The randomized studies most often are limited in size and large differences are necessary to show statistically significant differences. We no longer look upon the DRF as a homogenous entity but instead as a rather heterogenic group. In our department in Lund (Sweden) we have an ongoing registration with a prospective follow up of the DRF with the DASH. This will allow us to pick out smaller groups, analyze the result and perhaps change the treatment for that specific group. Ideally, the registry works as a hypothesis generating tool for selection of randomized studies as the next step.

5. A treatment Protocol

A standardized treatment program, based on the radiographic appearance but taking in account the age and the demands of the patients when selecting the proper treatment was developed by "The consensus group for distal radius fracture in southern Sweden" in 2004. This group consisting of dedicated surgeons from the orthopaedic and hand surgery departments in south of Sweden and with special interests in the treatment of DRF analysed the literature at that time and defined according to it the following protocol (Fig. 1).



^a Displaced= dorsal angulation >10° and/or Ulna + > 2mm and/or articular step >1mm or volar angulation >25°

Fig. 1 The southern Sweden treatment protocol for DRF. When selecting different treatments the patient's age and demands also has to be accounted for.

The treatment protocol is meant to be used as a guideline for treatment but a strict compliance to it is not expected. In a prospective follow up of a large number of patients collected from the previously mentioned DASH – registry have shown that a treatment protocol is of value and might help us to select the optimal treatment for each patient.

6. Conclusion

The use of a standardized treatment protocol may make it possible to select the patients with DRF for appropriate treatment. The chosen treatment will guarantee in each case the expected results with an almost, but not fully, normalized function at one year. All fracture types independently their severity will reach the same good results. There is no evidence based reason, with the actual knowledge in 2009 to apply a standardized treatment with volar locking plate to all patients and/or type of DRF. Further studies on this subject are needed and might change the actual standard of care in the future. We always have to be aware of the morbidity of the applied treatment.

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Scaphoid fractures

The scaphoid a mobile link between the proximal and distal row of the carpus is the most commonly fractured carpal bone. The scaphoid fracture is relatively frequent in the younger adults and decrease with age. The fracture of the scaphoid is difficult to diagnosis and difficult to treat. The very often missed diagnosis because of the difficulty to see the fracture on plain Xrays associated to the long healing time in relation with the poor vascularisation of this bone leads to a great number of scaphoid non unions with carpus collapse and secondary osteoarthritis of the wrist (SNAC). In all wrist trauma the scaphoid fracture can be evoked and has to be treated as a scaphoid fracture until it can be denied.

1. Diagnosis

1.1. Clinical: The scaphoid fracture is caused by fall on an extended hand and needs a relative high energy trauma. Stress fractures are uncommon. Classically the pain localisation after such fracture makes the clinical diagnostic easy. A swelling and pain at palpation on the radial side of the wrist distal to the styloid and between the long and short thumb extensor tendons are typical. But sometimes the complaints are vague, the pain located round or dorsal to the wrist. The traction on the thumb can help in diagnosis but the most important remains that scaphoid fractures has to be evoked in all wrist trauma.

1.2. Radiographic: Many scaphoid fractures are undisplaced and difficult to see on plain Xrays. Only the bone resorption which appears in the fracture line after some days or the displacement with a gap between the fractured scaphoid parts make the diagnosis possible. The initial X rays of the scaphoid needs to be careful, to the AP view and lateral projections so called scaphoid projections has to be added. In ulnar deviation of the wrist the scaphoid will be extended and examined in all its length.

1.3 MRI: This exam is very useful in the diagnosis of scaphoid fracture and gives also information on eventual fracture of other carpal bone or ligaments injury. The marrow oedema describes as bone bruise can be

sources of over diagnostic and for this reason the images of the MRI has to be correlated to a careful and critical clinical exam before diagnosis of one or the other injury is accepted. The high price of this investigation and the difficulties in many hospitals of doing MRI in emergency are important drawbacks.

1.4. CT bone scan: This exam recognizes the fracture very early, is sensitive and specific but unfortunately gives no information about eventual associated ligament injuries.

1.5. Scintigram: The low specificity of this exam make the us of the scintigrafy more rare nowadays

2. Classification

According to J C Botelho the diagnosed scaphoid fracture has to be classified essentially to define the correct treatment. He proposed to consider these following important points

- The fracture location: proximal, middle or distal third. Tuberosity
- Undisplaced or displaced
- The presence of comminution
- The age of the fracture
- Association of the scaphoid fracture as a part of a more severe midcarpal injury/luxation

3. Treatment

The classical conservative treatment, with cast, remains the rule for the majority of scaphoid fractures. New is that the displacement, the comminution or the localisation of the fracture with their consequences as malposition or non union risk make that we have to consider in an increasing number of cases other treatment alternatives. The cast to be used has been discussed in many publications without giving a consensus. It seems that the elbow do not need to be included in the immobilization. In our facility we use a "scaphoid cast" that includes the base of the thumb placed in abduction, immobilize the MCP I leaves the IP free. The wrist is immobilized with the second and third metacarpal in the axis of the radius.

3.1. Tuberosity fractures: This scaphoid fracture localisation has a good prognosis. The healing is relatively easy with almost no problem except a long disability and pain at the base of the thumb. A scaphoid cast can be applied for a period of 4-5 weeks in order to reduce the pain during healing.

3.2. Undisplaced middle third fracture: This fracture normally healed with out problems if the treatment is applied early. A scaphoid cast for a preliminary period of at least 6-8 weeks is recommended. After this time X-rays (without cast) will give information about the healing process. If the healing on X-rays can be confirmed by the absence of pain at palpation, the patient is allowed to start mobilization but a new X-Rays has to confirm the healing at 6 months in order to recognize an eventual painless scaphoid non union. When bone healing on X-Rays at 6-8 weeks is associated with remaining pain at palpation the immobilization time with cast has to be prolonged. Immobilization for more than 3 months is usually worthless.

3.3. Displaced middle third fracture: The deformity, consequence of the fracture displacement with the hump back deformity and the shortening of the scaphoid, the comminution making the fractures unstable are strong argues for the necessity of reposition and internal fixation. The

higher risk of non union of this fracture type is also in favour of the surgical treatment. In selected cases the conservative treatment is still acceptable.

3.4. Proximal third fracture: The high risk of non union and/or necrosis of the proximal pole following this fracture type is in strong favour of surgical treatment. Not only stabilization is here necessary but bone transplantation, vascularised or not, seems to be of importance.

4. Technical considerations

Almost all surgeons have nowadays recognized the advantages of cannulated compression screws in the treatment of scaphoid fractures or scaphoid non unions. With help of these devices or instruments the treatment has been facilitated and can very often be done without opening. Arthroscopy has here an important place and can help visualizing the reduction of the fracture. However it's still questionable if bone transplantation can be done without opening the fracture. The surgeon who has to treat scaphoid fracture or their complications needs to have a good knowledge of carpus anatomy, fixation techniques and wrist arthroscopy. He also needs knowledge about vascularised bone transplantation techniques described for carpus indications.

5. Scaphoid non union

Unfortunately the treatment described above does not guarantee healing of all scaphoid fractures. On the top of that, scaphoid non union can develops without symptoms with a patient who never was in contact with the physician or has forgotten or neglected the initial trauma. The non union can be the source of the instability of the carpus followed by a development of a SNAC wrist (Scaphoid Non union Advanced Collaps) and osteoarthritis. The Non union does not necessary leads to a SNAC and is not always symptomatic. The treatment of a scaphoid non union is only indicated in the absence of osteoarthritis. The non union has to be treated by reposition of the scaphoid, cleaning of the non union site, bone transplantation (vascularised or not) and fixation.

6. Treatment algorithm

Combination of a positive clinical examination associated with a positive X-rays may possible to start the treatment according to fracture type, localisation and displacement. With a negative X-rays the suspicion of a scaphoid fracture remains and the treatment is started with a scaphoid cast for a period of 2 weeks. After this delay a new clinical examination and a conventional radiographic examination of the scaphoid are performed. If both are negative the scaphoid fracture can be denied. With a negative X-Rays and the remaining presence of symptoms the fracture of the scaphoid has to be proved or denied by other examination as MRI or CT bone scan. Only if one or the other of these 2 exams are negative can, in these cases, the scaphoid fracture be denied (Fig. 2).

7. Conclusion

The proper diagnosis of a scaphoid fracture has to be done early in order to immobilize and stabilize the fracture as soon as possible. This can influence positively the outcome of these fractures with a high potential of non union or bone necrosis. A scaphoid fracture has to be suspected in all wrist traumas and treated until the fracture can be denied. The use of a standardized treatment algorithm may make it possible to select the patients for appropriate treatment. The chosen treatment will be chosen in accordance to the fracture type, localization and displacement. The arthroscopic procedures are very important in the treatment of this injury but cannot solve all problems. Bone graft including vascularised bone grafts are often needed for the treatment of the difficult cases.

References and additional readings

1. Abramo A, Kopylov P, Tagil M. Evaluation of a treatment protocol in distal radius fractures: a prospective study in 581 patients using DASH as outcome. *Acta Orthop* 2008;79-3:376-85.
2. Alnot JY. Les fractures et pseudarthroses polaires proximales du scaphoide carpien. *Rev. Chir Orthop* 1988;74: 740-43
3. Amadio PC, Berqvist TH, Smith DK et al. Scaphoid malunion, *J Hand Surg (am)* 1989; 14:679-87
4. Andersen DJ, Blair WF, Steyers CM, Jr., Adams BD, el-Khoury GY, Brandser EA. Classification of distal radius fractures: an analysis of interobserver reliability and intraobserver reproducibility. *J Hand Surg [Am]* 1996;21-4:574-82.
5. Arora R, Lutz M, Hennerbichler A, Krappinger D, Espen D, Gabl M. Complications following internal fixation of unstable distal radius fracture with a palmar locking-plate. *J Orthop Trauma* 2007;21-5:316-22.
6. Atroshi I, Brogren E, Larsson GU, Kloov J, Hofer M, Berggren AM. Wrist-bridging versus non-bridging external fixation for displaced distal radius fractures: a randomized assessor-blind clinical trial of 38 patients followed for 1 year. *Acta Orthop* 2006;77-3:445-53.
7. Beaton DE, Wright JG, Katz JN. Development of the QuickDASH: comparison of three item-reduction approaches. *J Bone Joint Surg Am* 2005;87-5:1038-46.
8. Botelho JC. Fractures of the scaphoid: Diagnosis and management. *EFFORT European Instructional lectures Madrid Springer Ed, Bentley G* 2010;79-84.
9. Dias JJ, Thompson J, Barton NJ et al. Suspected scaphoid fractures. The value of radiographs. *J Bone Joint surg Br* 1990;72:98-101
10. Downing ND, Karantana A. A revolution in the management of fractures of the distal radius? *J Bone Joint Surg Br* 2008;90-10:1271-5.
11. Gelberman RH, Menon J. The vascularity of the scaphoid bone *J Hand Surg Am* 1980; 5:508-513
12. Gummesson C, Atroshi I, Ekdahl C. The disabilities of the arm, shoulder and hand (DASH) outcome questionnaire: longitudinal construct validity and measuring self-rated health change after surgery. *BMC Musculoskelet Disord* 2003;4-1:11.
13. Handoll HH, Madhok R. Closed reduction methods for treating distal radial fractures in adults. *Cochrane Database Syst Rev* 2003-1:CD003763.
14. Handoll HH, Vaghela MV, Madhok R. Percutaneous pinning for treating distal radial fractures in adults. *Cochrane Database Syst Rev* 2007-3:CD006080.
15. Herbert TJ, Fisher WE. Management of the fractured scaphoid bone using a new bone screw. *J Bone Joint surg* 1984; 66B: 114-23.
16. Hudak PL, Amadio PC, Bombardier C. Development of an upper extremity outcome measure: the DASH (disabilities of the arm, shoulder and hand) [corrected]. *The Upper Extremity Collaborative Group (UECG). Am J Ind Med* 1996;29-6:602-8.
17. Kopylov P, Johnell O, Redlund-Johnell I, Bengner U. Fractures of the distal end of the radius in young adults: a 30-year follow-up. *J Hand Surg [Br]* 1993;18-1:45-9.
18. Mathoulin C, Haerle M, Vandeputte G. Vascularized bone graft in carpal bone reconstruction, *Ann Chir Plast Esthet.* 2005 Feb;50(1):43-8.
19. McQueen MM. Redisplaced unstable fractures of the distal radius. A randomised, prospective study of bridging versus non-bridging external fixation. *J Bone Joint Surg Br* 1998;80-4:665-9.
20. Rikli DA, Regazzoni P. The double plating technique for distal radius fractures. *Tech Hand Up Extrem Surg* 2000;4-2:107-14.

Treatment algorithm of diagnosis of a scaphoid fracture

Wrist Trauma.

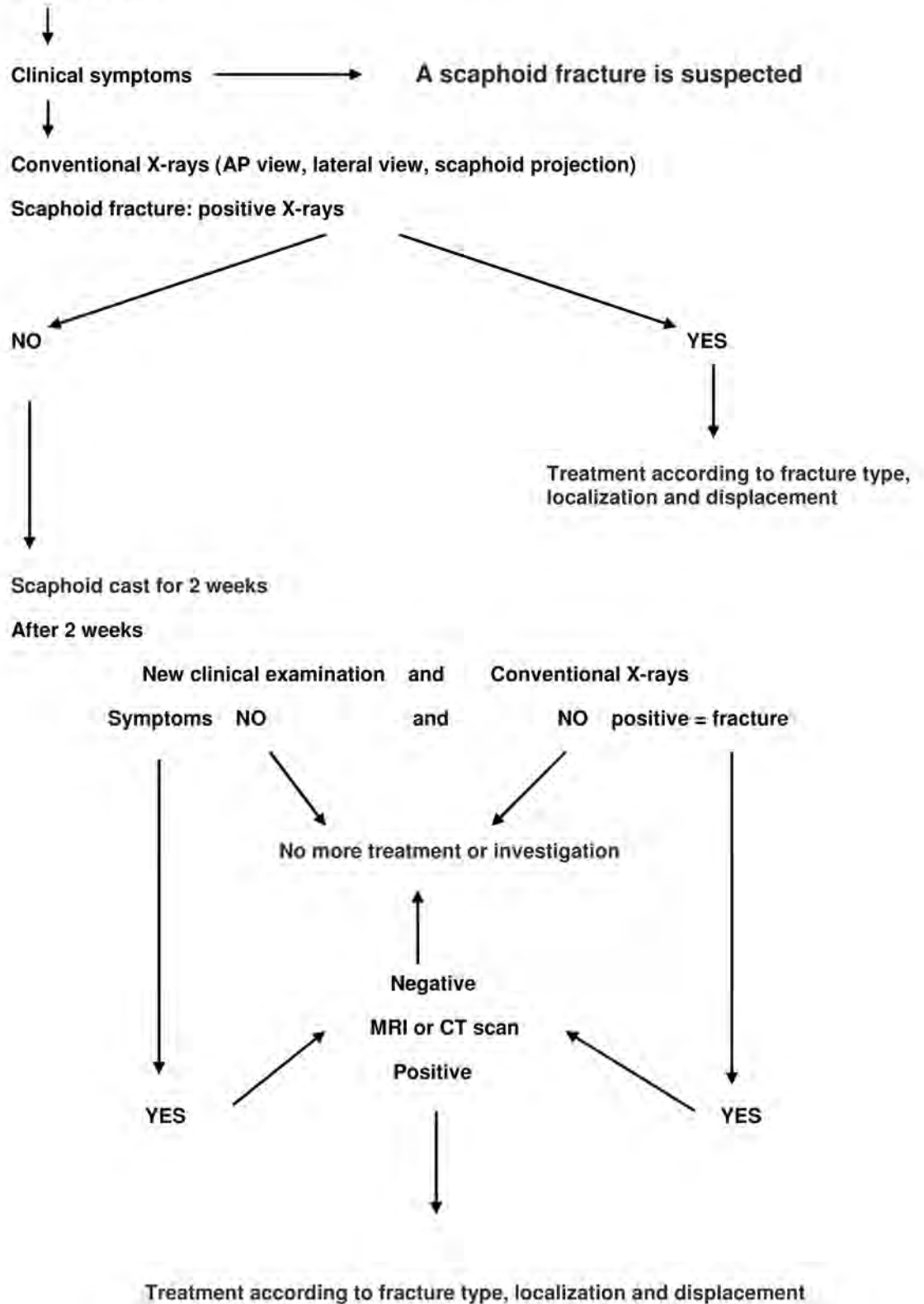


Fig. 2 Treatment algorithm of diagnosis of a scaphoid fracture



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Upper limb trauma: Shoulder girdle, proximal humerus, humeral shaft

For shoulder pathology the major reference is: *The Shoulder, Fourth Edition*. Editors CA Rockwood, FA Matsen, MA Wirth, SB Lippitt. 2009, Philadelphia, Saunders Elsevier. In the AO manual that covers all important fixation techniques the reader will find pertinent facts related to the topic at hand: *AO Principles of Fracture Management. Second expanded edition*, TP Rüedi, RE Buckley, CG Moran. 2007, Georg Thieme Verlag. For the latest in fracture fixation techniques the reader is also invited to visit the AO surgery reference site: *AO surgery reference*: <http://www.aofoundation.org>. Another most useful publication containing pertinent facts related to orthopaedics and musculoskeletal trauma is the *AAOS Comprehensive Orthopaedic Review*. Jay R. Lieberman, MD, Editor 2009 Rosemont, IL, American Academy of Orthopaedic Surgeons. For surgical approaches the most useful reference is without doubt: *Surgical Exposures in Orthopaedics: The Anatomic Approach*. Hoppenfeld S, deBoer P, Buckley R. Lippincott Williams & Wilkins; 2009, Fourth Edition.

Fixation may be accomplished with a 3.5 mm reconstruction or dynamic compression with or without locked screws. Nails of different types have been advocated and reported to be successful by many authors.

1. Robertson C, Celestre P, Mahar A, Schwartz A. Reconstruction plates for stabilization of mid-shaft clavicle fractures: differences between nonlocked and locked plates in two different positions. *J Shoulder Elbow Surg*. 2009;18(2):204-9.
2. Lee YS, Lin CC, Huang CR, Chen CN, Liao WY. Operative treatment of midclavicular fractures in 62 elderly patients: Knowles pin versus plate. *Orthopedics*. 2007;30(11):959-64.
3. Canadian Orthopaedic Trauma Society Trial. Midshaft Clavicular Fractures. A Multicenter, Randomized Clinical Nonoperative Treatment Compared with Plate Fixation of Displaced.
4. *J Bone Joint Surg Am*. 2007;89:1-10.

Clavicle fractures

Clavicular fractures are some of the most common fractures accounting for 5% to 10% of all fractures and 35% to 45% of shoulder girdle injuries. The clavicle struts the shoulder girdle.

Clavicular fractures are the result of falls, rarely direct trauma and rarely secondary to metastatic disease.

Clinical examination shows deformity of the shoulder girdle; a careful neurovascular examination must be performed due to the vicinity of fragile structures deep to this unprotected and subcutaneous bone.

Diagnosis necessitates an AP X-ray of the clavicle and often an AP view of the whole shoulder girdle will be of help to comparatively determine the amount of displacement. In some rare cases a CT will define the fracture.

Fractures of the clavicle are divided into proximal third, mid-third and distal third.

- Proximal third fractures

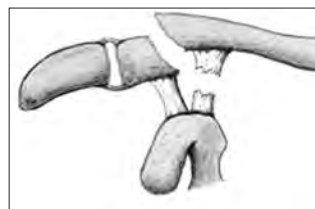
Usually conservative treatment will be sufficient, if displaced will benefit from fracture fixation, preferably with a plate. Beware of free pins that tend to migrate.

- Mid-third fractures

If little displacement is present conservative treatment with a sling will be sufficient. In cases of displacement >100% or > 2 cm of shortening, fixation is indicated. Flail chest, scapulothoracic dissociation, fractures menacing the integrity of the skin or open fractures are also indications for operative fixation. Activities such as professional cycling cannot tolerate unequal clavicular lengths and in these cases reconstruction is indicated.



Neer Type 1



Neer Type 2



Neer Type 3

- Distal third fractures

In case of displacement > 100%, skin menace or open fracture fixation is indicated. Depending on the size of the distal fragment the surgical intervention can vary from simple excision, to figure of 8 wiring with pins, to heavy sutures to specific plates or hook plates. If the coracoclavicular ligaments are compromised (Neer type II fractures) coracoclavicular fixation (sutures or screws) may be indicated.

1. Khan LA, Bradnock TJ, Scott C, Robinson CM. Fractures of the clavicle. *J Bone Joint Surg Am.* 2009;91(2):447-60.
2. Neer CS II. Fractures of the distal third of the clavicle. *Clin Orthop.* 1968;58:43-50.
3. Fann CY, Chiu FY, Chuang TY, Chen CM, Chen TH. Transacromial Knowles pin in the treatment of Neer type 2 distal clavicle fractures. A prospective evaluation of 32 cases. *J Trauma.* 2004;56(5):1102-5; discussion 1105-6.
4. Goldberg JA, Bruce WJ, Sonnabend DH, Walsh WR. *J Shoulder Elbow Surg.* Type 2 fractures of the distal clavicle: a new surgical technique. 1997;6(4):380-2.
5. *The Shoulder, Fourth Edition.* Editors CA Rockwood, FA Matsen, MA Wirth, SB Lippitt. 2009, Philadelphia, Saunders Elsevier.
6. Muramatsu K, Shigetomi M, Matsunaga T, Murata Y, Taguchi T. Use of the AO hook-plate for treatment of unstable fractures of the distal clavicle. *Arch Orthop Trauma Surg.* 2007;127(3):191-4.

Complications

Infections, nonunions or neurovascular compromise dominate the scene.

1. *The Shoulder, Fourth Edition.* Editors CA Rockwood, FA Matsen, MA Wirth, SB Lippitt. 2009, Philadelphia, Saunders Elsevier.
2. Endrizzi DP, White RR, Babikian GM, Old AB. Nonunion of the clavicle treated with plate fixation: a review of forty-seven consecutive cases. *J Shoulder Elbow Surg.* 2008;17(6):951-3.

Sternoclavicular dislocations

- Anatomy

With relatively no osseous constraints, stability is provided by the anterior capsular ligament, the posterior capsular ligament, and a joint meniscus. The costoclavicular and interclavicular ligaments provide adjunct stability.

- Antero-superior dislocation

Unstable and needs surgical intervention for stability. Usually reassurance and conservative treatment will suffice however.

- Postero-inferior dislocation

This is potentially a life threatening situation. Symptoms are related to the posterior structures under compression (dyspnea,

dysphagia, vascular compromise or thrombosis). CT is helpful to make the diagnosis. Reduction under anaesthesia with a bolster under the dorsal spine and simultaneously pulling the arm in extension while grabbing the clavicle end with a towel clip will usually reduce the clavicle that will stay stable.

Beware of fractures passing through the proximal growth plate, which is the last to ossify at age 25.

1. Jaggard MK, Gupte CM, Gulati V, Reilly P. A comprehensive review of trauma and disruption to the sternoclavicular joint with the proposal of a new classification system. *J Trauma.* 2009;66(2):576-84.

Acromioclavicular dislocations

Usually a consequence of a fall on the tip of the shoulder in a young to middle-aged male athlete, the acromion is pushed downwards and the coraco- and acromio-clavicular ligaments are damaged to varying degrees along with a displacement of the clavicle with respect to the shoulder girdle.

The patient presents with a deformity due to the antero-inferior position of the shoulder girdle. Check for instability in the frontal and transverse planes. Inspect the skin to rule out abrasions.

AP X-rays of the shoulder, Zanca views (10°-15° cephalic tilt) and axillary views are necessary and sufficient. An AP X-ray view of the shoulder girdle is a useful adjunct. Stress views are not necessary.

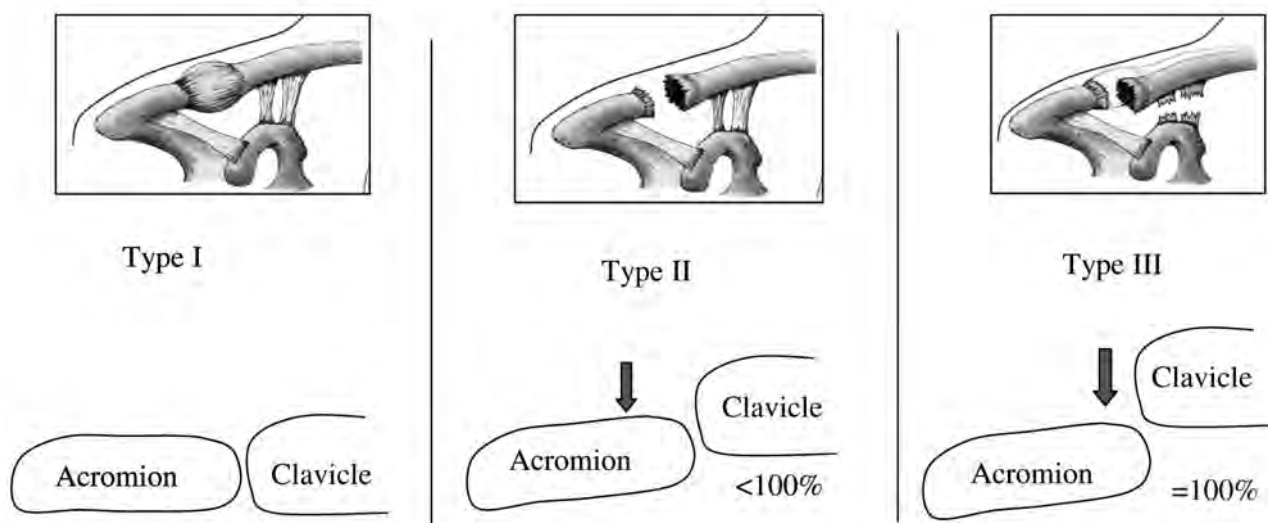
AC dislocations are classified according to Rockwood:

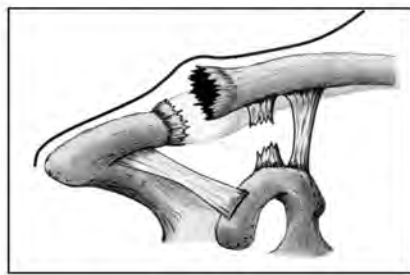
Type I: Strain without tear, Type II tearing of AC ligaments, Type III: Tearing of AC and CC ligaments (Trapezoid and conoid), Type IV: posterior displacement of the clavicle in relation to the acromion. Type V: More than 100% displacement with tearing of AC and CC ligaments and overlying trapezius muscle.

Types I and II need conservative treatment.

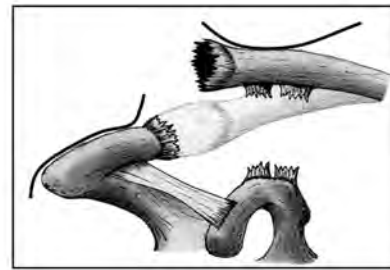
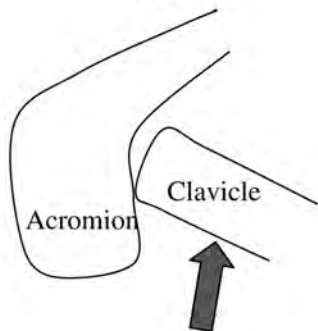
Surgery is usually recommended in types IV and V. Type III is controversial in frail patients in may be recommended. The techniques may involve coraco-clavicular screws, CC and AC heavy sutures or tapes or transarticular pinning.

In long standing cases the Weaver-Dunn procedure is recommended, with removal of 1 cm of the distal clavicle and using the coraco-acromial ligament as a substitute inserted into the hollowed out distal clavicle. Hook plates are used by some authors but will require reoperation for their removal.

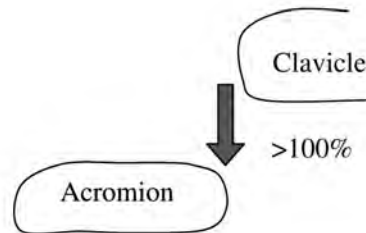




Type IV



Type V



1. *The Shoulder, Fourth Edition.* Editors CA Rockwood, FA Matsen, MA Wirth, SB Lippitt. 2009, Philadelphia, Saunders Elsevier.
2. *AAOS Comprehensive Orthopaedic Review.* Jay R. Lieberman, MD, Editor 2009 Rosemont, IL, American Academy of Orthopaedic Surgeons.

Scapular fractures

Fractures of the scapula result from high energy trauma with 80 to 95% incidence of associated trauma 50% of which are thoracic trauma. Mortality is 10% to 15% principally due to associated thoracic and cranial injuries. Thorough clinical examination is mandatory and CT with 3D reconstruction is of great help in determining the exact extent of the fracture.

- Scapulothoracic dissociation

This is the equivalent of an internal amputation entailing serious neurovascular damages. This injury is associated with a traumatic break or dislocation of the shoulder girdle (AC, clavicle, SC) and a lateral displacement of the scapula as seen on AP chest X-Ray. Consequences are dire and in many cases lead to loss of the upper extremity or death from major thoracic injury or massive haemorrhage.

1. Ebraheim NA, An HS, Jackson WT, Pearlstein SR, Burgess A, Tschern H, Hass N, Kellam J, Wipparman BU. *Scapulothoracic dissociation.* *J Bone Joint Surgery Am.* 1988;70:428-432.

- Body Fractures

Most of these fractures may be treated conservatively, the scapula being well protected and surrounded by muscles. The most popular classification is the Ideberg classification.

1. Ideberg R, Grevsten S, Larsson S. *Epidemiology of scapular fractures. Incidence and classification of 338 fractures.* *Acta Orthop Scand.* 1995;66(5):395-7.
2. Schofer MD, Sehr AC, Timmesfeld N, Störmer S, Kortmann HR. *Fractures of the scapula: long-term results after conservative treatment.* *Arch Orthop Trauma Surg.* 2009.
3. Lapner PC, Uthoff HK, Papp S. *Scapula fractures.* *Orthop Clin North Am.* 2008 ;39(4):459-74, vi.

4. Lantry JM, Roberts CS, Giannoudis PV. *Operative treatment of scapular fractures: a systematic review.* *Injury.* 2008;39(3):271-83.

- Glenoid Fractures

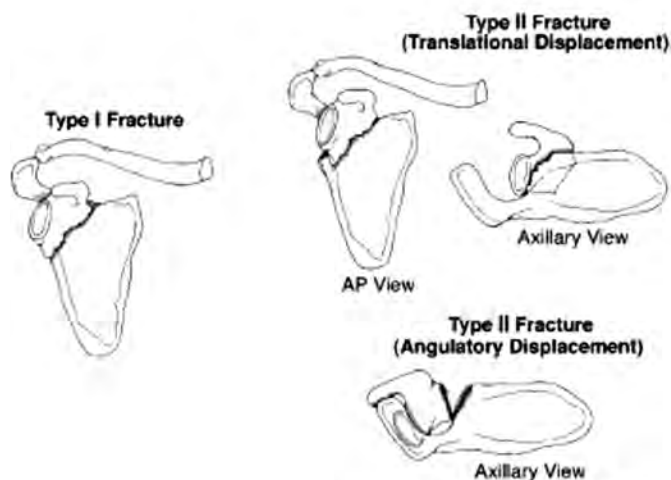
Fractures of the glenoid surface and rim must be reduced and fixed if they are accompanied by instability or subluxation of the glenohumeral joint. If the humeral head does not appear centered in AP and axillary views and in CT cuts then the indication is absolute. When the joint remains centered, the indication for fixation becomes relative.

1. Goss TP. *Fractures of the glenoid cavity.* *J Bone Joint Surg Am.* 1992;74-A:299-305.
2. Schandelmaier P, Blauth M, Schneider C, Krettek C. *Fractures of the glenoid treated by operation. A 5- to 23-year follow-up of 22 cases.* *J Bone Joint Surg Br.* 2002;84(2):173-7.
3. Maquieira GJ, Espinosa N, Gerber C, Eid K. *Non-operative treatment of large anterior glenoid rim fractures after traumatic anterior dislocation of the shoulder.* *J Bone Joint Surg Br.* 2007;89(10):1347-51.

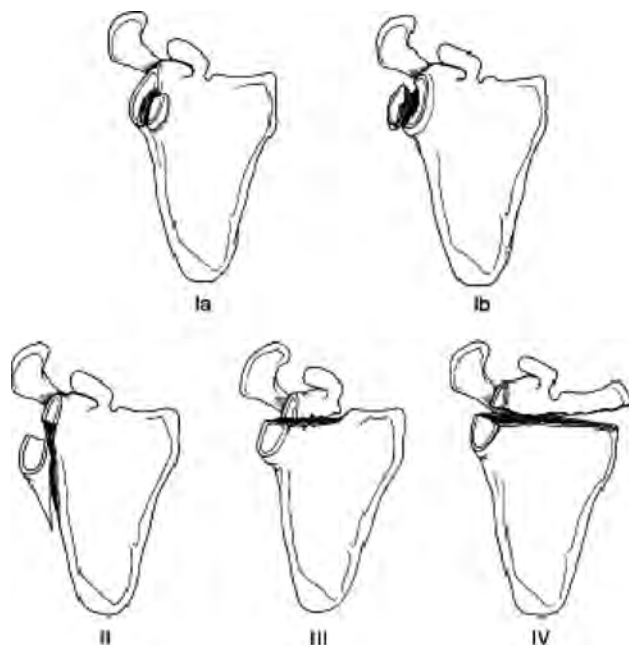
- Acromion and Spine Fractures

Displaced fractures of the acromion or the spine of the scapula need plate fixation or tension band fixation. Constant pull of the deltoid will displace the fragments and lead to a secondary impingement that may be difficult to treat.

1. Ogawa K, Naniwa T. *Fractures of the acromion and the lateral scapular spine.* *J Shoulder Elbow Surg.* 1997;6(6):544-8.



Classification of fractures of the scapular neck TB Goss. J AM Acad Orthop Surg 1995; 3: 22-23



Classification of the scapular body Ideberg R, Grevsten S, Larsson S. Epidemiology of scapular fractures. Incidence and classification of 338 fractures. Acta Orthop Scand. 1995;66(5):395-7.

Glenohumeral dislocation

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Surgical Exposures in Orthopaedics: The Anatomic Approach. Hoppenfeld S, deBoer P, Buckley R. Lippincott Williams & Wilkins; 2009, Fourth Edition.

Introduction

Dislocation applies to a complete loss of contact between two joint surfaces. Subluxation implies partial loss of contact. Laxity is the result of a clinical examination showing more than "normal" passive motion or translation. Instability is a subjective sensation described by the patient that includes subluxation up to dislocation.

Glenohumeral instability is a spectrum that includes hyperlaxity and traumatic dislocation whether anterior or posterior or multidirectional. Classification of the different types of instability include:

- Traumatic anterior dislocation: Accidental fall
- Traumatic posterior dislocation: Accidental fall
- Atraumatic instability due to capsular stretching because repeated "micro-trauma"
- Multidirectional instability due to capsular laxity

Pathoanatomy

- Traumatic anterior instability

Generally accompanied by a tear of the capsulo-labral complex that sometimes includes osseous fragments off from the glenoid rim:

The *Bankart* lesion.

Anterior capsular stretching.

In many cases a bony trough in the posterior-superior region of the head will be caused by impaction against the glenoid rim sometimes leading up to a fracture of the greater tuberosity: The *Hill-Sachs* lesion.

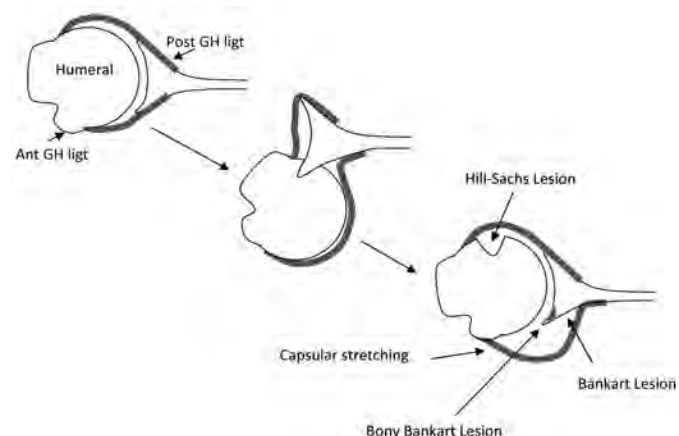
- Traumatic posterior instability

The inferior glenohumeral ligament is the main restraint in abduction/external rotation and found to be torn or detached in all cases of traumatic dislocation.

Generally accompanied by detachment and stretching of the posterior capsulo-ligamentous complex, rarely with osseous lesions involving the glenoid rim: The *reverse Bankart* lesion.

Posterior capsular stretching. Impaction of the anterior region of the head just medial to the lesser tuberosity leading up to a head-split fracture: The *reverse Hill-Sachs* lesion.

Anterior dislocation injuries



Hill-Sachs impression; Bankart bony and labral; capsular stretching.

Dislocation and instability types

Anterior dislocation

Usually related to sports activities (soccer, skiing etc.) or falls. Recurrence rates are high in patients below 20 yrs (up to 90%), between 20 and 40 yrs 60% recurrence rates, above 40 yrs 10%. These numbers vary depending on authors but trends remain.

Clinical examination is dominated by apprehension in abduction and external rotation.

Signs of generalized laxity are often present: Antero-posterior drawer, inferior sulcus sign, joint hyperlaxity (fingers, thumb, elbow).

In acute cases axillary nerve injury occurs in 5% of patients.

Imaging involves AP and axillary views. Arthro-CT scans delineate precisely bony morphology of fractures; Hill-Sachs lesions, glenoid rim fractures or rounding are well visualized. MRI may be helpful but bony lesions are poorly demonstrated.

- Treatment for acute dislocations

AFTER diagnostic X-Rays: Reduction techniques include, after neurovascular testing, *Stimson* (Patient prone, arm hanging with 1 to 3 kg weights attached to the wrist), *Saha* (slow elevation in the plane of the scapula), *Kocher* (Adduction in internal rot followed by abduction in ext rotation), Traction after intra-articular injection of lidocaine or equivalent, *Davos* (Patient to cross his fingers around his flexed knee and with elbows extended is instructed to slowly bend backwards), *Hippocrates technique* (anesthetized, traction on the arm and with foot in the axilla which should be replaced by a towel) should only be performed when the non traumatic techniques have failed.

Postreduction treatment includes, after neurovascular testing, immobilisation in internal rotation or in an *external* rotation splint. (The rationale for the external rotation immobilisation is to force the Bankart lesion to stay fixed to the anterior glenoid rim pressured by the subscapularis). Immobilisation should be 2 to 4 weeks followed by strengthening exercises.

- Treatment for recurrent dislocations

Surgical indications for stabilisation include one episode of dislocation too many, or severe apprehension.

Techniques include capsulorrhaphy, Bankart lesion refixation, bony augmentation if severe rounding or fracture of the rim.

Open or arthroscopic techniques are both suitable. Closed arthroscopic techniques are advocated in traumatic Bankart lesions, open techniques are recommended in cases of capsular stretching or large Hill-Sachs lesions. Recurrence rates range between 5% and 30% depending on technique used, strength of reconstruction and patient compliance.

Patients are immobilized from 3 to 6 weeks in internal rotation; rehabilitation emphasizes muscular strengthening in the first weeks followed by range of motion exercises. Patients are advised to avoid contact sports for a year following stabilisation.

Posterior dislocation

Fall on outstretched hand, seizures or electrical shocks are the main causes. AP and axillary X-rays for diagnosis. Relatively rare; less than 5% of all instabilities. Beware of the diagnosis: The cardinal sign is active and passive limitation of external rotation. On the AP X-ray, the joint space is not visible and the axillary is always diagnostic. In doubt a CT will solve the issue.

- Treatment for acute dislocations

If a small i.e. less than 10% reverse Hill-Sachs is present, gentle traction will generally reduce the shoulder which should then be immobilized in an external rotation splint for three to 6 weeks with a rehabilitation programme to follow.

If a large Hill-Sachs lesion is present, reduction under anaesthesia may be necessary followed by the McLaughlin procedure where through an anterior deltopectoral incision the head is gently levered out and the subscapularis or the osteotomized lesser tuberosity is sutured or screwed into the bony defect. External rotation immobilisation 4 to 6 weeks followed by a rehabilitation programme.

- Treatment for recurrent dislocations

If no major Hill-Sachs lesion is present a posterior approach with a cruciate capsulorrhaphy and fixation of the reverse Bankart lesion is performed. A bone graft from the spine of the scapula or the iliac crest may be necessary if a bony defect is present.

If a major Hill-Sachs lesion is present a McLaughlin procedure will be necessary and if insufficient an adjunct posterior procedure may be necessary.

Multidirectional dislocation

This applies to young patients with laxity and instability in more than in one direction, i.e. anterior and posterior or posterior and inferior or all three. Cardinal signs are hyperlaxity, sulcus sign and anterior and posterior drawer signs all causing discomfort or apprehension.

Standard X-rays, arthro-CT or MRI will delineate the existing lesions. Surgery is indicated only after one year of serious muscle strengthening physiotherapy and exercises.

The most commonly accepted operation is Neer's capsular shift which may be performed through an anterior deltopectoral approach but in certain cases may need an adjunct posterior approach. The axillary nerve must be protected during this demanding and complex intervention. 6 weeks of immobilisation in neutral (handshake) position is necessary followed by a muscle strengthening programme.

Chronic dislocation

Usually seen in debilitated patients. The best option may be no treatment. In cases of chronic pain and discomfort shoulder fusion may be another option. Some authors advocate the reverse prosthesis but the danger of dislocation is great.

Recurrent dislocation in the elderly patient

Often these dislocations are associated with minor trauma. A massive rotator cuff tear is the usual cause. If repairable the supra and infraspinatus lesions should be repaired. If not repairable the reverse prosthesis may be an option and if not fusion may have to be performed.

1. *The Shoulder, Fourth Edition. Editors CA Rockwood, FA Matsen, MA Wirth, SB Lippitt. 2009, Philadelphia, Saunders Elsevier.*
2. *AAOS Comprehensive Orthopaedic Review. Jay R. Lieberman, MD, Editor 2009 Rosemont, IL, American Academy of Orthopaedic Surgeons.*
3. *Shoulder Reconstruction. CS Neer. W.B. Saunders Company (January 1990).*

Proximal humerus fractures

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Introduction

Proximal humerus fractures constitute 5% of all fractures. High energy fractures occur in young males and low energy fractures in elderly females. They are intra-articular fractures and treatment modalities should attempt to reconstruct the anatomy so that function may be best restored. 80% of all these fractures need conservative treatment. Avascular necrosis, mal or non unions, stiffness and postoperative sepsis plague the treatment results.

Biomechanics

The quasi sphericity of the humeral head allows smooth articulation on the glenoid. The subacromial arch must be preserved; any bony fragments or overgrowth will lead to impingement inhibiting motion. The rotator cuff plays the roles of transmission belt, spacer and shock absorber. Translation of the humeral head is limited by the glenoid geometry, the labrum, the glenohumeral ligaments and the coaptation force of the cuff muscles. The deltoid muscle provides power in elevation and abduction, the rotator cuff centers the humeral head and provides power in external (infraspinatus) and internal rotation (subscapularis). The supraspinatus fine tunes practically all glenohumeral movements. The pectoralis plays a role in adduction and internal rotation.

1. *The Shoulder, Fourth Edition*. Editors CA Rockwood, FA Matsen, MA Wirth, SB Lippitt. 2009, Philadelphia, Saunders Elsevier.

Anatomy

The humeral head is a half sphere with a diameter between 37 to 57 mm, inclined at 130°, retroverted at 30°. The axillary artery is divided into three segments by the pectoralis minor muscle. The first part is medial to the pectoralis minor muscle, the second part is deep to the pectoralis minor muscle and the third part lateral to the pectoralis minor has three branches: the subscapular artery (the circumflex scapular branch runs through the triangular space), the anterior humeral circumflex artery and the posterior humeral circumflex artery accompanies the axillary nerve and exits posteriorly through the quadrilateral space (medial: long head of triceps, lateral: humeral shaft, superior: teres minor, inferior: teres major). The blood supply of the humeral head is provided by the anterolateral ascending branch of the anterior circumflex artery terminating into the arcuate artery in the

humeral head, the rotator cuff arterial supply, the central metaphyseal artery and the posterior circumflex artery. Innervation of the deltoid and teres minor muscles arises from the axillary nerve along with a sensory component in the lateral shoulder. Innervation of supra and infraspinatus depends on the suprascapular nerve passing through the scapular notch giving off branches to the supraspinatus and then passing around the spinoglenoid notch to innervate the infraspinatus. The subscapularis is innervated by the subscapularis nerve, a direct branch off of the posterior trunk of the brachial plexus. The pectoralis muscle nerve stems off the medial trunk.

1. *AAOS Comprehensive Orthopaedic Review*. Jay R. Lieberman, MD, Editor 2009 Rosemont, IL, American Academy of Orthopaedic Surgeons.
2. Hertel R, Hempfing A, Stiehler M, et al. Predictors of humeral head ischemia after intracapsular fracture of the proximal humerus. *J Shoulder Elbow Surg* 2004; 13(4):427-433.
3. *The Shoulder, Fourth Edition*. Editors CA Rockwood, FA Matsen, MA Wirth, SB Lippitt. 2009, Philadelphia, Saunders Elsevier

Clinical presentation

Deformity and functional impairment are the presenting signs and symptoms. The neurovascular status must be explored namely the status of the axillary nerve. In undisplaced fractures a tell-tale ecchymosis appearing two to three days after a fall will sign an underlying fracture. Diagnosis will be made with well-centered x-rays AP and axillary views. If operative treatment is entertained a CT with 3D reconstruction will give invaluable information. MRI may be occasionally useful for assessment of the rotator cuff or to ascertain the existence of a fracture. Excellent imaging is the only way to accurately classify the fracture and establish a prognosis as to the occurrence of avascular necrosis.

1. *The Shoulder, Fourth Edition*. Editors CA Rockwood, FA Matsen, MA Wirth, SB Lippitt. 2009, Philadelphia, Saunders Elsevier

Classification of proximal humeral fractures

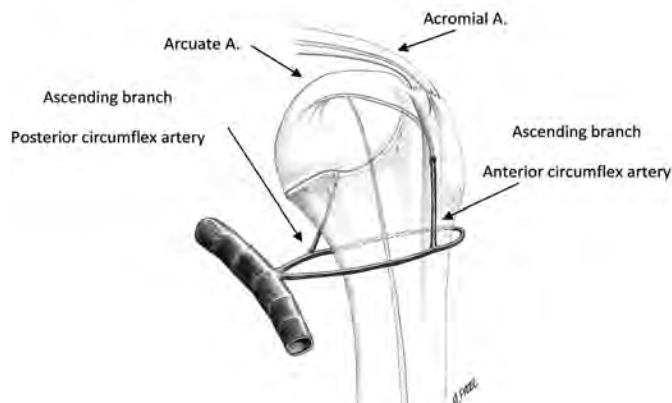
Many classification schemes exist: Neer classification into two, three and four part fractures, a fracture is deemed displaced if there is more than 1 cm of displacement or 45° of angulation. The AO -OTA classification is based on the scheme of the overall AO classification. The "Lego" classification of Hertel is interesting because it allows to combine the different fracture patterns and the Duparc classification which has an anatomic and functional determinant. However although helpful, none of these classifications has perfect inter or intra-observer reliability.

1. Neer CS II: Displaced proximal humeral fractures: Part I. Classification and evaluation. *J Bone Joint Surg Am* 1970;52:1077-1089.
2. *AO Principles of Fracture Management. Second expanded edition*, TP Rüedi, RE Buckley, CG Moran. Georg Thieme Verlag; 2 Har/Dvdr edition (2007).
3. Hertel R. Fractures of the proximal humerus in osteoporotic bone. *Osteoporos Int*. 2005;16 Suppl 2:S65-72.
4. Duparc J. Classification of articular fractures of the upper extremity of the humerus. *Acta Orthop Belg*. 1995;61 Suppl 1:65-70.

Conservative treatment

Most fractures will not be greatly displaced; immobilisation for three to six weeks in a shoulder immobilizer or a Velpeau type bandage will be

indicated. Rarely an abduction splint will be needed to hold the fracture pattern in an acceptable position. Appropriate analgesic medications should be prescribed and personal hygiene measures with removal of the Velpeau every five days should be organized in the first weeks. After 3 to 6 weeks depending on the fracture type gentle physiotherapeutic exercises, emphasising on isometric exercises should be instituted. The fracture will heal in 12 weeks.



Vascular anatomy of the humeral head

Surgical approaches

- Delto-pectoral approach

The cephalic vein should if possible be preserved. The axillary nerve must be palpated in front of the subscapularis. If the long biceps tendon is not anatomically replaced a tenodesis is in order.

- Trans-deltoid approach

The deltoid should not be split further than 5 cm distal to the acromion to protect the axillary nerve.

- Posterior approach

A deltoid split will lead to the unfrazspinatus which may have to be detached to access the capsule for arthroscopy. Rarely used approach in the trauma setting.

1. *The Shoulder, Fourth Edition. Editors CA Rockwood, FA Matsen, MA Wirth, SB Lippitt. 2009, Philadelphia, Saunders Elsevier*
2. *AAOS Comprehensive Orthopaedic Review. Jay R. Lieberman, MD, Editor 2009 Rosemont, IL, American Academy of Orthopaedic Surgeons.*

Operative techniques

- Isolated greater tuberosity fractures

Displacement of more than 0,5 to 1cm warrants operative treatment. Usually a trans-deltoid approach with suture fixation, sometimes augmented by isolated screws or perhaps a plate in case of a large fragment.



Transdeltoid approach and osteosuture of displaced fracture of the greater tuberosity



Two part fracture fixed with a locking plate

- Displaced lesser tuberosity fractures

Anatomic reduction and fixation with screws is warranted to preserve subscapularis function.

- Two part displaced surgical neck fracture

Plating or IM nailing can be used successfully in this indication.

- Three part fractures

In strong bone percutaneous pinning may be used although accurate reduction is best achieved with an open technique. Some authors favour locked nailing for these fractures. In weak bone a deltopectoral approach with plate fixation with or without fixed angle screws or an osteosuture technique will be indicated. The biceps if well aligned in the bicipital groove is a precious indicator as to reduction accuracy. It is wise to check the reduction before closure with an X-ray or an image intensifier.



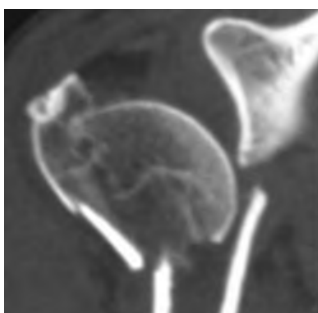
Three part fracture fixation with a locking screw plate



Three part fracture fixation with a third tubular 3.5 locking screw plate

- Four part fractures

Prosthetic replacement respecting height, version and tuberosity fixation will be used in the elderly patient. In high demand young patients it is probably best to attempt plate osteosynthesis with angle stable screws. This is an acceptable solution only if an adequate anatomical reconstruction has been achieved. If not, a hemiarthroplasty with careful reconstruction of the tuberosities is an acceptable option.



Four part fracture: X-Rays, CT evaluation and plate fixation (Deltpectoral approach)



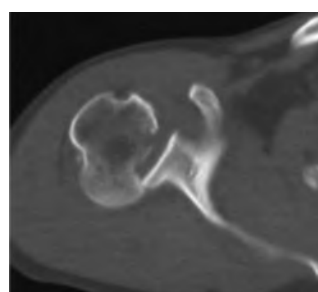
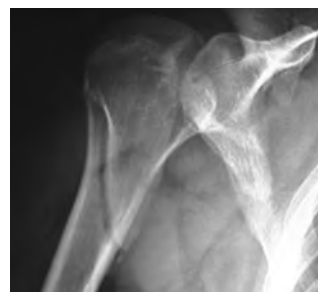
Four part fracture (major displacement) treated with a hemi-prosthesis

- Fracture-dislocations

Reduction must be obtained under anaesthesia so as not to displace a pre-existing humeral neck fracture. If there is doubt an open reduction should be done. Fixation will then depend on the fracture pattern. In very difficult situations it may be necessary to do a deltoid take-down to increase exposure. Careful neurovascular assessment must precede any surgical act and if necessary appropriate vascular imaging should be obtained.

- Posterior dislocations

This may be a difficult to diagnosis often associated with seizures, although a fall on the outstretched hand can cause posterior dislocation. The hallmark is lack of external rotation passive or active. Plain x-rays must be scrutinized and if there is a doubt a CT scan is the best option. Active investigations should include neurological assessment to rule out intracranial tumours or other causes of seizures. If a large reverse Hill Sachs lesion is present or if a head splitting fracture is present the treatment may have to be surgical. The McLaughlin procedure is the insertion of the subscapularis tendon into the reverse Hill-Sachs lesion while the Neer modified approach osteotomizes the lesser tuberosity which is fixed with screws into the bed of the Hill-Sachs lesion. In all cases, whether the treatment is operative or conservative, post-reduction immobilisation is in external rotation often with the help of a splint.



Posterior fracture dislocation : X-rays, CT and screw fixation after reduction

1. *The Shoulder, Fourth Edition. Editors CA Rockwood, FA Matsen, MA Wirth, SB Lippitt. 2009, Philadelphia, Saunders Elsevier.*
2. Hoffmeyer P. The operative management of displaced fractures of the proximal humerus. *J Bone Joint Surg Br*; 2002;84(4):469–480.
3. Gerber C, Werner CM, Vienne P (2004) Internal fixation of complex fractures of the proximal humerus. *J Bone Joint Surg Br*; 86(6): 848–855.
4. Brems JJ (2002) Shoulder arthroplasty in the face of acute fracture: puzzle pieces. *J Arthroplasty*; 17(4 Suppl 1):32–35.

Humeral shaft fractures

In the AO manual that covers all important fixation techniques the reader will find pertinent facts related to the topic at hand: *AO Principles of Fracture Management. Second expanded edition, TP Rüedi, RE Buckley, CG Moran. Georg Thieme Verlag; 2 Har/Dvdr edition (2007).* For the latest in fracture fixation techniques the reader is also invited to visit the AO surgery reference site: *AO surgery reference: <http://www.aofoundation.org>.* Another most useful publication containing pertinent facts related to orthopaedics and musculoskeletal in general trauma is the *AAOS Comprehensive Orthopaedic Review. Jay R. Lieberman, MD, Editor 2009 Rosemont, IL, American Academy of Orthopaedic Surgeons.* For surgical approaches the most useful reference is without doubt: *Surgical Exposures in Orthopaedics: The Anatomic Approach. Hoppenfeld S, deBoer P, Buckley R. Lippincott Williams & Wilkins; Fourth Edition edition 2009.*

Introduction

Fractures of the humeral shaft represent 2–3% of fractures and are distributed in a bimodal mode. High energy fractures are typical of younger patients while low energy fractures are more commonly seen in the elderly osteoporotic individual. These fractures are the result of blunt trauma resulting from a fall and more rarely from gunshot wounds or war injuries. Sports are associated with these fractures such as hangglider injuries or result from arm wrestling. Pathologic fractures are also commonly seen arising from bony fragilisation resulting from metastases (8% of humeral fractures). All neoplastic diseases solid or haematological may cause metastatic disease. The neoplasms most frequently involved are those arising from: Breast, kidney, thyroid, lung, prostate or multiple myeloma. Chronic osteomyelitis either primary or associated with haemoglobinopathies may also cause associated fractures.

1. Ekholm R, Adami J, Tidermark J, Hansson K, Törnkvist H, Ponzer S. Fractures of the shaft of the humerus. An epidemiological study of 401 fractures. *J Bone Joint Surg Br*. 2006;88(11):1469–73.
2. Sarahrudi K, Wolf H, Funovics P, Pajenda G, Hausmann JT, Vécsei V. Surgical treatment of pathological fractures of the shaft of the humerus. *J Trauma*. 2009;66(3):789–94.
3. Frassica FJ, Frassica DA. Metastatic bone disease of the humerus. *J Am Acad Orthop Surg*. 2003;11(4):282–8.

Biomechanics

The main forces acting on the humerus are torsional.

Anatomy

The main anatomical feature is the medial to lateral posteriorly running spiral groove housing the radial nerve beginning at 20 cm medially from

the distal articular surface and ending 14 cm proximal to the distal joint surface. The radial nerve is reported to be injured on average in 11.8% in fractures of the humeral shaft.

1. Shao YC, Harwood P, Grotz MR, Limb D, Giannoudis PV. Radial nerve palsy associated with fractures of the shaft of the humerus: a systematic review. *J Bone Joint Surg Br*. 2005;87:1647–1652.
2. AAOS Comprehensive Orthopaedic Review. Jay R. Lieberman, MD, Editor 2009 Rosemont, IL, American Academy of Orthopaedic Surgeons.

Classification of humeral shaft fractures

The AO classification is popular. It classifies the fracture patterns of the humeral shaft as type A (simple, transverse or spiral) type B (wedge with a butterfly fragment) and type C (segmental or comminuted fragments). Open fractures are classified according to Gustilo and Anderson: Type I inside-out (< 1 cm), Type II outside-in (> 1 cm), Type III A (open, osseous coverage possible), type III B (open, necessitating a local or free flap), Type III C (Open fracture with vascular injury).

1. *AO Principles of Fracture Management. Second expanded edition, Thomas P Rüedi, Richard E Buckley, Christopher G Moran. Georg Thieme Verlag; Har/Dvdr edition (2007).*
2. Gustilo RB, Mendoza RM, Williams DN. Problems in the management of type III (severe) open fractures. A new classification of type III fractures. *J Trauma* 1984; 24:742–746.
3. Gustilo RB, Anderson J. Prevention of infection in the treatment of one thousand and twenty-five open fractures of long bones: Retrospective and prospective analyses. *J Bone Joint Surg Am* 1976;58:453–458.

Clinical presentation

A deformed extremity is present. Look for neurovascular injuries (radial nerve) carefully before any manipulation of the injured extremity. Plain X-rays including the shoulder and elbow are generally sufficient in acute traumatic cases. MRI, CT, Bone scintigraphy are useful in special situations such as chronic infection, metastatic or primary tumors.

Conservative treatment

Usually if conservative treatment is chosen the patient is first immobilised in a Velpeau type bandage and after two to three weeks when swelling has diminished a functional brace is applied. There exist no guidelines but many agree that planar angulations of 20° sagittally and 15° frontally, malrotations up to 15°, and shortening up to 3 cm are acceptable. According to Sarmiento the most common complication of conservative functional bracing is varus angulation: 16% > 10°–20°.

1. Sarmiento A, Latta LL. Humeral diaphyseal fractures: functional bracing. *Unfallchirurg*. 2007;110(10):824–32.
2. Sarmiento A, Zagorski JB, Zych GA: Functional bracing for the treatment of fractures of the humeral diaphysis. *J Bone Joint Surg Am* 2000; 82:478–486.

Surgical approaches

- Antero-lateral approach

The radial nerve may be identified in the intermuscular groove between the brachialis and the brachioradialis. It is followed up into its entry into the groove. The brachialis is then split to reveal the entire length of the shaft if necessary.

1. Mekhail AO, Checroun AJ, Ebraheim NA, Jackson WT, Yeasting RA. Extensile approach to the anterolateral surface of the humerus and the radial nerve. *J Shoulder Elbow Surg.* 1999;8(2):112-8.

- Posterior approach

The radial nerve is identified running obliquely from medial to lateral under the heads of the triceps. The ulnar nerve runs along the medial border of the medial head of the triceps. This is not a suitable approach for proximal fractures because of the deltoid insertion.

1. Zlotolow DA, Catalano LW 3rd, Barron OA, Glickel SZ. Surgical exposures of the humerus. *J Am Acad Orthop Surg.* 2006;14(13):754-65.

Operative treatment indications

The list is not exhaustive and includes the following: Open fractures, bilateral fractures, vascular injury, immediate radial nerve palsy, floating elbow, failure of closed treatment, pathologic fractures (bone metastases), brachial plexus injury, and obesity.

- IM Nailing

Nailing is an advantageous minimally invasive technique that is suitable for unstable fractures. Control of rotation is achieved with locking bolts. Shoulder pain is common after antegrade nailing. Non-unions are more common with nailing than with plating.

- Anterograde
Care must be taken with an adequate point of entry; most nails enter through the cartilaginous surface of the head thus minimizing injury to the rotator cuff.
- Retrograde
Entry point must be well above the olecranon fossa to avoid fragilizing the distal humerus. This is not suited for distal fractures.

- Plating

Using a lateral or a posterior approach, plates suitable to the anatomy (broad plates in a large bone, narrow plates in a small bone) should be used. Attempts at minimally invasive approaches with incisions proximally and distally (radial nerve) allowing closed plate insertions are being developed. Locked screws may be useful in osteoporotic bone. Union rates of more than 94% are achieved with plating.

- External fixation

Indicated in polytrauma (Staged in damage control orthopaedics), open fractures or situations where formal osteosynthesis with nailing or plating is not possible. Open approaches are recommended to avoid injuring nerves. If the elbow must be spanned it is preferable to insert the pins in the ulna.

1. Bhandari M, Devereaux PJ, McKee MD: Compression plating versus intramedullary nailing of humeral shaft fractures—A meta-analysis. *Acta Orthop* 2006;77:279-284.
2. Popescu D, Fernandez-Valencia JA, Rios M, Cuñé J, Domingo A, Prat S. Internal fixation of proximal humerus fractures using the T2-proximal humeral nail. *Arch Orthop Trauma Surg.* 2008
3. Park JY, Pandher DS, Chun JY, et al. Antegrade humeral nailing through the rotator cuff interval: a new entry portal. *J Orthop Trauma.* 2008;22(6):419-25.



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Upper limb trauma: Elbow

The most authoritative and comprehensive textbook about the elbow is certainly *The Elbow and Its Disorders, 4th Edition, Elsevier 2008* by Bernard F. Morrey, MD. In it, the student will find a compilation of the most recent knowledge of all aspects of elbow pathology. For the latest in fracture fixation techniques the reader is invited to visit the *AO surgery reference site: AO surgery reference: <http://www.aofoundation.org>*. Another most useful publication containing pertinent facts related to orthopaedics and musculoskeletal in general trauma is the *AAOS Comprehensive Orthopaedic Review*. Jay R. Lieberman, MD, Editor 2009 Rosemont, IL, American Academy of Orthopaedic Surgeons.

Facts about the elbow

The distal humerus is an arch subtended by two columns of equal importance. The trochlea is a pulley like structure covered by cartilage in a 300° arc. The articular portion of the distal humerus in the lateral plane is inclined 30° anterior with respect to the axis of the humerus, the frontal plane is tilted 6° into valgus, and in the transverse plane is rotated medially about 5°. The capitellum is a half sphere covered anteriorly with cartilage. The radial head is asymmetric and has two articular interactions: The proximal ulno-radial joint and the radio-humeral joint. It has approximately a 240° of articular cartilage coverage which leaves 120° of non cartilage covered area amenable to hardware fixation. The head and neck have an angle of 15° in valgus. The proximal ulna has a coronoid process that has an area equivalent to the radial head. There is no cartilage in the middle of the sigmoid notch. The joint is angled 30° posteriorly in the lateral plane; 1° to 6° in the frontal plane. The carrying angle is the angle between the humerus and the ulna with the elbow extended fully and it varies between 11°-14° in men and 13°-16° in women. The capsule attaches anteriorly above the coronoid and radial fossae and just distal to the coronoid. Posteriorly it attaches above the olecranon fossa, follows the columns and distally attaches along the articular margins of the sigmoid notch. The normal elbow has a range from 0° or slightly hyperextended to 150° of flexion, pronation is 75° and supination is 85°. A 3° to 4° varus-valgus laxity has been measured during F/E. The rotation of the forearm is around an oblique axis passing through the proximal and distal radio-ulnar joints. The primary static stabilizers of the elbow are the ulno-humeral articulation and the collateral ligaments. The secondary static stabilizers are the capsule, the radiohumeral articulation and the common flexor and extensor tendon origins. The dynamic stabilizers are all the muscles that cross the elbow (Anconeus, triceps, brachialis). Finally all forces that cross the elbow joint are directed posterior and this has implications in surgical procedures around the elbow, in the design of elbow prosthesis, and in rehabilitation programmes.

1. *The Elbow and Its Disorders, Editor BF Morrey, 4th Edition, Elsevier 2008.*

Approaches to the elbow

Lateral approach

Kocher (radial head fracture, lat collat reconstruction)

Interval between the anconeus and extensor carpi ulnaris

Column (Stiff elbow)

Extensor carpi radialis longus and distal fibers of the brachial radialis elevated from the lateral column and epicondyle. Brachialis muscle separated from the anterior capsule; safe if the joint penetrated at the radiocapitellar articulation. Triceps may be elevated posterior giving access to the olecranon fossa.

Anterior approach

Henry (PIN, proximal radius, tumors)

After an appropriately curving incision to avoid the flexor crease, brachioradialis and brachialis are gently separated to find the radial nerve. Follow the nerve to the arcade of Frohse where the motor branch plunges into the supinator to course dorsally in the forearm then elevate supinator from its radial insertion laterally thus protecting motor branch in the supinator mass.

Medial approach

Over the top Hotchkiss approach

(Coronoid fracture type 1: transolecranon suture)

50:50 split in the flexor-pronator mass anterior to the ulnar nerve.

Natural split: Taylor and Scham

(Coronoid fracture type 2-3 with plate fixation).

Elevation of the entire flexor-pronator mass, from the dorsal aspect to the volar aspect.

Boyd Posterolateral Exposure (Radial head, proximal radius)

The ulnar insertion of the anconeus and the origin of the supinator muscles are elevated subperiosteally. More distally, the subperiosteal reflection includes the abductor pollicis longus, the extensor carpi ulnaris, and the extensor pollicis longus muscles. The origin of the supinator at the crista supinatoris of the ulna is released, and the entire muscle flap is retracted radially, exposing the radiohumeral joint. The posterior interosseous nerve is protected in the substance of the supinator, which must be gently retracted.

Posterior approach (Fractures distal humerus, arthroplasty, stiff elbow)

- Posterior approach with extensile exposure of the distal humerus:
 - Bilateral tricipital approach (Alonso - Llamas) with lateral and medial retraction of the triceps.
 - Triceps Splitting (Campbell)

- Olecranon osteotomy: Extra-articular, chevron or straight.
- Triceps sparing elevation of triceps according to Gschwend (osseous) or Morrey-Bryan (subperiosteal).
- Triceps reflecting anconeus pedicle approach (TRAP) O'Driscoll.

1. *The Elbow and Its Disorders, 4th Ed, Elsevier 2008 Ed. Morrey BF.*
2. *Fracture of the Anteromedial Facet of the Coronoid Process. Surgical Technique. Ring D, Doornberg JN. J Bone Joint Surg Am. 2007; 89:267-283.*
3. *A posteromedial approach to the proximal end of the ulna for the internal fixation of olecranon fractures. Taylor TK, Scham SM. J Trauma. 1969;9:594-602.*
4. *AO surgery reference: <http://www.aofoundation.org>.*
5. *Surgical Exposures in Orthopaedics: The Anatomic Approach. Hoppenfeld S, deBoer P, Buckley R. Lippincott Williams & Wilkins; Fourth Edition edition 2009.*

Fractures and dislocations

Fractures of the distal humerus

These fractures are relatively rare and constitute about 2% of all fractures, but represent a 1/3 of all elbow fractures. They most commonly occur in patients in the 6th decade and above and are frequently associated with osteoporosis. These fractures are frequently comminuted and operative fixation is therefore technically difficult. Fractures of the distal humerus are articular fractures characteristically *unstable* and prone to displacement. Only in exceptional circumstances is non-operative treatment warranted. For the best results *operative intervention* providing accurate reduction and stable fixation is therefore indicated in these complex fractures. Before intervening careful physical examination is necessary and specifically ascertaining the neurovascular status of the involved extremity. *Compartment syndromes*, a menacing complication with a devastating outcome must be diagnosed early and aggressively treated with fasciotomy. The goal of the treatment is to obtain a stable construct restoring the anatomy and allowing for early motion so as to restore function and strength to the elbow joint.

The anatomy of the distal humerus is complex and for practical purposes the *two column concept* is the best suited. It may be described as two columns, the lateral and the medial, providing the stable structure upon which the articular epiphysis, trochlea and capitellum, is anchored.

Many *classifications* exist; the one best suited being the AO classification which includes type A or extra-articular fracture patterns, type B or partial intra-articular fractures and type C, the most complex, with intra-articular separations and comminution involving the whole joint. To aid in classification it may be useful to obtain x-rays of the contralateral elbow, to perform CT scanning and perhaps most helpful to obtain traction X-rays. Certain fractures such as capitellum fractures are difficult to diagnose and therefore all imaging modalities must be obtained in case of doubt. All these modalities will aid in the diagnosis of the fracture pattern and influence the approach and fixation modalities.

1. *AO surgery reference: <http://www.aofoundation.org>.*

Once the decision to operate is taken, it must be decided on how the patient should be *positioned* during the intervention. This will depend on the fracture pattern and on the patient's condition. The decision should be made in accordance with the anaesthesiologists. For fractures involving the lateral column only a supine approach will be chosen while for fractures involving the medial column or both columns a decubitus lateral position or a ventral position may be necessary to perform a posterior approach, it must be noted that in these positions it will be nigh impossible to access to the front of the elbow, however having to do so is very rare. Also, the surgeon must be very careful of the positioning of the contralateral limb, head and neck, to avoid injury due to compression.

The use of a *tourniquet* is debatable, if the fracture is uncomplicated and the operation is anticipated to be short, a tourniquet will provide a bloodless field and will be useful. In case of a complex fracture, paradoxically, I tend not to use a tourniquet which might have to be inflated for too long a time and I prefer instead to perform careful haemostasis during the approach and operate in the driest field possible.

The *lateral approach* will be directly on the lateral column dissecting off sharply the insertions of the brachioradialis and the extensor carpi radialis longus and brevis from the lateral supracondylar ridge in front and the triceps in the back. The common extensor origin is then sharply lifted off of the epicondyle anteriorly and if necessary the anconeus posteriorly. Proximal extension must be done with caution because of the radial nerve. The joint capsule is incised and elevated to view the capitellum and the radial head.

The *medial approach* is useful for fractures of the epitrochlea and the ulnar nerve must be carefully identified before inserting screws.

The *posterior approach* will be useful for fractures involving the medial or both columns and with intra-articular fractures of the trochlea. The incision will be midline, swerving laterally around the olecranon and in line with ulnar shaft. In all cases the *ulnar nerve* must be visualized and protected. The nerve is easy to find, lying almost subcutaneously at the medial edge of the triceps three finger breadths above the olecranon, once identified it is followed over the epitrochlea into the common flexor mass avoiding injury to the motor branch of the flexor carpi ulnaris that it penetrates between its ulnar (posterior) and humeral (anterior) heads. At the end of the operation it must be decided whether to transpose the nerve anteriorly into a subcutaneous pocket or not. Personally, I avoid this if possible because it renders redo surgery extremely difficult if the whereabouts of the transposed nerve are not exactly described. The next difficulty is exposing the fracture. If there is widespread comminution of the trochlea it is wise to proceed to an *osteotomy of the olecranon* which may be chevron shaped or transverse. The near cortex is cut with an oscillating saw for precision but the articular cortex should be broken off with an osteotome allowing for perfect reposition. At the end of the intervention the olecranon must be repositioned and fixed using a tension band with K-wires or a single 6.5mm spongiosa screw. In the case the hole may be drilled before osteotomy ensuring a good reposition. In cases where the fracture of the trochlea is sagittal with no comminution a *bilateral tricipital reflecting approach* as described by Alonso-LLames may be used or alternatively a *triceps reflecting anconeus pedicle approach (TRAP)* as described by O'Driscoll may be used. It is best to avoid triceps cutting

(V-Y) approaches for they tend to weaken the extensor mechanism without really being efficacious for exposure.

Isolated fractures of the *capitellum* are approached laterally and must be repositioned and fixed with two posterior to anterior small fragment 3.5 mm lag screws or with Herbert type screws.

Isolated fractures of the *epitrochlea* are approached medially and fixed in place with a lag screw after careful reposition. The ulnar nerve must be protected.

Fractures of the *lateral column* are approached through a direct lateral approach. Plate fixation will be needed to augment the screw fixation, usually small fragment implants placed on the posterior aspect of the lateral column will provide adequate fixation.

Both column fractures without articular involvement need a posterior approach and can be addressed through a bilaterotricipital Alonso-Llames or TRAP approach. Both columns are identified and fixed to the articular epiphysis using a lateral $\frac{1}{3}$ tubular plate for the medial column and a posterior 3.5 reconstruction plate for the lateral column. Newer anatomically contoured plates have now reached the marketplace and may also be used.

Both column fractures with articular involvement are the most difficult fracture patterns and need an extensive posterior approach with an accompanying olecranon osteotomy for visualisation. It is necessary to reconstruct the distal epiphysis first. Most of the time the fracture is sagittal line and adequate reduction is easily obtained and held with a lag screw placed in such a way as not to interfere with the ulnar nerve. Sometimes in case of comminution it is necessary to place an intercalary bone graft so as not to squeeze and narrow the epiphysis which renders the joint incongruent. Once the joint surface has been reconstructed it is then possible using various types of implants to fix both columns. In general a $\frac{1}{3}$ tubular plate placed medially on the trochlear column and a posterior 3.5 mm reconstruction plate on the lateral side will provide sufficient fixation. Both plates should be at right angles to each other, the medial plate lying in the sagittal plane and the lateral plate in the frontal plane. Other options include multiple small plates (2.7 mm) or more recently the use of contoured anatomic plates some equipped with locking holes which provide angularly fixed screws. As a general rule it is wise to avoid provisional reduction with too many K-wires as these will interfere with the placement of the definitive implants and reduction will be lost when these are put in place while having to remove the provisional fixation. These fractures tax the anatomical and biomechanical knowledge of the surgeon, as well as his imagination and skill and are amongst the most challenging of articular fractures to undertake.

1. Pollock JW, Faber KJ, Athwal GS. Distal humerus fractures. *Orthop Clin North Am.* 2008;39(2):187-200, vi
2. Bryan RS, Morrey BF: Extensive posterior exposure of the elbow. *Clin Orthop Relat Res* 1982;188-192.
3. *The Elbow and Its Disorders*, 4th Ed, Elsevier 2008 Ed. Morrey BF.
4. Fracture of the Anteromedial Facet of the Coronoid Process. *Surgical Technique.* Ring D, Doornberg JN. *J Bone Joint Surg Am.* 2007; 89:267-283.
5. A posteromedial approach to the proximal end of the ulna for the internal fixation of olecranon fractures. Taylor TK, Scham SM. *J Trauma.* 1969;9:594-602.
6. AO surgery reference: <http://www.aofoundation.org>.
7. *Surgical Exposures in Orthopaedics: The Anatomic Approach.*

Hoppenfeld S, deBoer P, Buckley R. *Lippincott Williams & Wilkins; Fourth Edition edition 2009.*

Comminuted intra-articular fractures in osteoporotic bone. In cases of comminuted fractures of the distal humerus occurring in elderly, osteoporotic, low demand patients it is now a recommended option to place a cemented *Total Elbow Arthroplasty*. Because the epicondyles and their ligamentous attachments are cannot be reconstructed, the chosen prosthesis must provide intrinsic stability. Excision of the radial head must be performed if it impinges upon the prosthesis. Contra-indications include open fractures or a high infectious risk because of extensive soft tissue damage. The technique is demanding and the surgeon must be experienced in TEA for elective procedures before embarking on this intervention. The results are reported to be satisfactory in the literature; however the complication rate is high for this type of operation.

1. Gambirasio R, Riand N, Stern R, Hoffmeyer P. Total elbow replacement for complex fractures of the distal humerus. An option for the elderly patient. *J Bone Joint Surg Br.* 2001 Sep;83(7):974-8.

Comminuted open fractures of the distal humerus. In rare instances one is confronted with a major soft tissue injury with an underlying fracture. In case of Gustilo I and II open fractures the treatment is to debride and wash out the wound and proceed with internal fixation as if it were a closed injury. Whenever possible the opening should be incorporated in the approach and the wound closed over suction drainage at the end of the procedure. Appropriate antibiotic prophylaxis should be started after swabs are obtained for microbiological investigations including culture and sensitivity. In the face of Gustilo III open fractures a *humero-ulnar external fixateur* bridging the fracture zone and immobilising temporarily the joint is a reasonable and useful option. Beware of the radial nerve crossing the humeral diaphysis laterally approximately 7cm above the elbow joint. It is recommended to insert the pins of the external fixateur through a small open incision after having visualized and protected the radial nerve. The fixateur pins, usually a half frame, should be placed as far from the fracture zone as possible so that the pin tracts will not interfere with future osteosynthesis. Once the elbow is bridged, the priority is restoring the integrity of the soft tissue envelope with the help of a plastic surgeon if deemed necessary. Once the soft tissue envelope is restored it may be advisable to remove the fixateur and to proceed with a stable reconstruction of the joint surfaces so as to begin motion and avoid a stiff and painful elbow.

Rehabilitation consists in splinting to protect the soft tissues but with immediate assisted active motion. After 6 to 8 weeks the soft tissues are less swollen the splint may be removed and careful use with non weight carrying may be tolerated.

Fractures of the radial head

Fractures of the radial head represent around 2 % of all fractures and 33% of all elbow fractures. They usually occur after a fall on the slightly flexed outstretched elbow with the hand in supination. The patient complains of immediate pain in the lateral region of the elbow after a fall. There is often a palpable fluctuation outwardly bulging over the radio-humeral joint due to haemorrhagic effusion and active pronosupination is painful or impossible. To assess the amount of displacement the humero-radial joint is aspirated and lidocaine is injected into the joint. If smooth, non-grating, active or passive pronosupination

is possible this is a reliable sign that the fracture is minimally displaced and that non-operative will lead to a satisfactory outcome, otherwise surgical treatment is mandatory if painless motion is to be restored.

Anatomically and biomechanically, the radial head is part of the *forearm articular complex* including the proximal radio-ulnar joint, the interosseous membrane and the distal radio-ulnar joint that allows prono-supination of the forearm. The *radiohumeral joint* also participates in the flexion/extension mobility of the elbow joint. Furthermore the radial head is involved in the stability of the elbow joint and plays the role of a *secondary stabilizer*. If the ulnar collateral ligaments and the distal radioulnar joint are intact, the radial head plays no role in the stability of the elbow and may therefore safely be removed if necessary. However, in the absence of the radial head and disruption of the distal radioulnar ligaments (Essex-Lopresti lesion), the radius will migrate proximally and more so when there is an associated tear of the interosseous membrane. In these circumstances a relative over-lengthening of the ulna will occur at the wrist entailing painful dysfunction. Also, in the absence of the radial head, valgus instability will occur at the elbow if the ulnar collateral ligament is torn or elongated.

Fracture classification of radial head fractures:

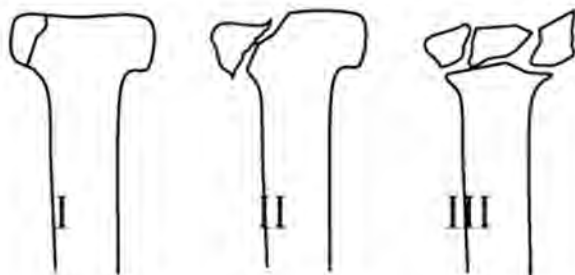
Various classification schemes have been proposed:

Mason Classification:

- Type I: Non-displaced
- Type II: Displaced marginal fractures
- Type III: Comminuted fractures
- Type IV: Associated with elbow dislocation

Hotchkiss modification:

- Type I: No surgery
- Type II: Displaced but fixable
- Type III: Displaced and unfixable



As a general rule displaced fractures need surgical intervention, minimal displacement may benefit from Open Reduction and Internal Fixation (ORIF) and highly displaced or comminuted fractures may necessitate excision in case of a stable Distal Radio-Ulnar Joint and intact interosseous membrane and prosthetic replacement if these conditions are not met.

Approach

The approach is basically lateral starting obliquely from the supracondylar ridge over the radio-humeral joint and trough the Kocher interval between the anconeus and the extensor carpi ulnaris. This protects the motor branch of the radial nerve (Avoid placing a Hohmann type retractor over the anterior neck of the radius) and the approach is sufficiently anterior to spare the ulnar collateral ligament which will not be inadvertently severed. The capsule is revealed and an arthrotomy is performed exposing the radial head. The annular ligament is spared.

Technique

Reconstructible fractures: After assessment they are fixed using small fragment 2.0 or 2.7 AO or Herbert type screws. Sometimes the use of a mini blade plate type of implant may be necessary.

Radial head excision: If excision is necessary be sure that all fragments are excised by reconstructing the head on the instrument table. The head should be removed at the level of the annular ligament. The elbow and wrist should be closely assessed for stability and the lateral collateral reconstructed if necessary.

Prosthetic replacement: It is necessary to provide stability by inserting a radial head prosthesis in cases of fracture dislocations with either frontal plane instability such as with extensive tearing of the lateral collateral ligaments or with longitudinal instability with tearing of the interosseous membrane or DRUJ. Today, the accepted prosthesis is metallic with or without a moving or floating (bipolar) head and a stem that may be cemented or non-cemented. In some cases the prosthesis may be left permanently in place while in other instances such as in very young patients it may be useful to remove the prosthetic head used as a temporary spacer once healing of the ligamentous complex has occurred. Silastic implants once in vogue are now generally abandoned because of the risk of a destructive synovial inflammatory response due to fragmentation of the prosthesis leading to the accumulation of irritative particulate matter. Furthermore biomechanical studies have shown that these prostheses are not stiff enough to allow anatomic healing of torn ligaments.

Rehabilitation

As general rule rehabilitation must be begun early and motion should be started within days of the intervention in case of operative treatment or diagnosis in case of conservative treatment. After a few days of rest start by gentle *active* flexion exercises going from 90° to 110° then progress with *active* extension exercises ranging from 120° to 30° as tolerated. After two to three weeks gentle *active* prono-supination exercises are begun. An articulated brace is useful for protection in cases of instability

The Essex Lopresti injury

This injury was described in 1951 by Essex-Lopresti and associates as a severely comminuted fracture of the radial head with tearing of the interosseous membrane and disrupting the DRUJ. The diagnosis is clinical and radiological. X-rays of the whole forearm are necessary. The radial head must imperatively be fixed or replaced with a prosthesis and the DRUJ has to be stabilized with a cross pin left in situ for 4 to 6 weeks.

1. Frankle MA, Koval KJ, Sanders RW, Zuckerman JD: Radial head fractures with dislocations treated by immediate stabilization and early motion *J Shoulder Elbow Surg* 1999;8:355-356.
2. Cooney WP. Radial head fractures and the role of radial head prosthetic replacement: current update. *Am J Orthop.* 2008;37(8 Suppl 1):21-5.

Fractures of the olecranon

Fractures of the olecranon usually occur after falls directly on the elbow point. They are frequently seen in the osteoporotic patient.

There are various classifications; the most popular are the Mayo classification:

Type I: Undisplaced

Type II: Displaced but stable elbow (Noncomminuted: A / Comminuted: B)

Type III: Displaced and unstable elbow (Noncomminuted: A / Comminuted: B)

The AO classification (Complex: includes the proximal forearm segment: radius and ulna):

A: Extra-articular fractures

B: Intra-articular fractures

C: Fractures of both olecranon and radius

A treatment plan must be elaborated. The great majority of these fractures are displaced and the question arises as to what is the best suited technique.

Approach

The surgical approach is straightforward. The ulna is subcutaneous; the patient may be in a supine or lateral decubitus position with the arm resting on a support. A tourniquet may be used. The incision follows the shaft of the ulna and some recommend arcing the incision radially to avoid the tip of the olecranon and also to avoid a scar over the ulnar nerve.

Technique

Clearly transverse fractures are best treated by a technique associating K-wires and tension band cerclage such as described by the AO Group. The technique must be meticulously followed and especially the placement of the K-wires must be parallel, 5 to 6 cm long, and the tip should be into the opposite cortex distally to the coronoid and the ends must be bent at 180° and deeply buried into the triceps and olecranon. Comminuted fractures will require a plating technique (3.5 LCP or DCP plates) augmented by longitudinal screws (so-called homerun screws). If the fracture is oblique and is near the coronoid a compression screw will be most useful. The main drawbacks of these techniques lie in the high reoperation rate that all authors mention. K-wires tend to back out and must be carefully followed and plates usually present some degree of discomfort and are best removed after adequate consolidation. A more recently described complication is the interference of K-wires or screws placed in the proximal ulna with the proximal radius. Some implants are too long and either impinge or are screwed into the radial head or bicipital tuberosity. This is not an easy diagnosis post-operatively and freedom of pronation-supination must be carefully ascertained at the end of the surgical reconstruction.

Rehabilitation

The patient is placed into a backslab at 80° of flexion and gentle active flexion and extension exercises are started as tolerated. The olecranon is protected for 6 to 8 weeks before any weight bearing exercises are started.

Fractures of the coronoid

This is usually associated with dislocations of the elbow. Regan and Morrey have classified these injuries into:

Type I: Fracture of the tip

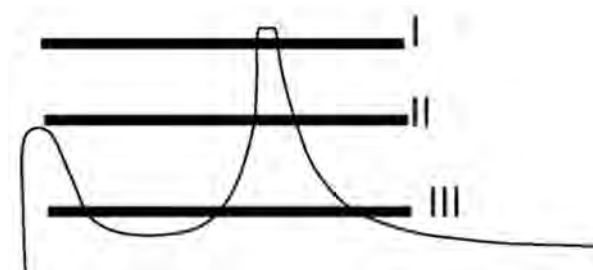
Type II: Less than 50% of the height of the coronoid

Type III: More than 50%

A and B types signify no or associated dislocation

Some have added a:

Type IV: Fracture of the sublime tubercle

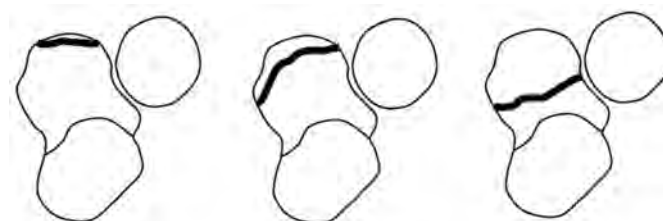


Type I fractures are generally stable and do not need fixation if the elbow is stable. Types III and IV need surgery to insure stability of the elbow because the medial collateral ligament attaches to the medial coronoid and instability will occur if the bony fragments are not fixed. More recently O'Driscoll has modified this classification.

In this classification all types 2 and 3 need fixation and especially if associated with a dislocation or a radial head fracture. Plain x-rays and preferably a CT scan should be used for making the diagnosis and classifying the lesions. Small lesions can be fixed by transolecranon sutures. The fragment is approached from a medial incision in an "Over the top" as approach described by Hotchkiss. Large fragments are approached by a posteromedial route.

In very unstable elbows a hinged external fixateur device will provide stability while allowing early motion.

1. Regan W, Morrey B. Fractures of the coronoid process of the ulna. *J Bone Joint Surg Am* 1989;71:1348-1354.
2. O'Driscoll SW, Jupiter JB, Cohen MS, Ring D, McKee MD. Difficult elbow fractures: pearls and pitfalls. *Instr Course Lect* 2003;52:113-134.
3. Hotchkiss RN. Fractures and dislocations of the elbow. In: Rockwood CA, Green DP, eds. *Rockwood and Green's fractures in adults*. Vol 1. 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 1996:929-1024.



Type 1 Tip fractures

Type 2 Anteromedial fractures

Type 3 Base of coronoid fractures

Dislocation of the elbow

The mechanism is usually a fall on the outstretched hand with the elbow in a varus position. The primary lesion is a tear of the lateral collateral ligament from the lateral humeral insertion and as the mechanism of dislocation the capsule is then torn anteriorly, the coronoid may be damaged by the ram effect of the trochlea and finally the medially collateral may be torn also, leading to a very unstable position. In 5 to 10% of cases a fracture of the radial head may be associated as well as more rarely a fracture of the capitellum. Neurovascular injuries occur infrequently but must be looked for. The median nerve may be stretched by the front riding humerus, and this is the most frequent neurological injury, however the radial nerve and the ulnar nerve may also be damaged. The brachial artery may suffer an intimal tear or a rupture while it is stretched out over the protruding distal humerus and very

rarely the skin may split leading to an open injury. A compartment syndrome is always a possibility and the patient must be monitored.

Dislocations of the elbow are classified as anterior (rare), posterior (most common) and divergent (very rare the radial head will be separated from the ulna and the annular ligament is torn).

The elbow, after proper radiographic and clinical assessment should be reduced, general anaesthesia may be necessary, and tested for stability: varus, valgus and postero-lateral. Postero-lateral rotatory instability occurs when the ulno-radial bloc dislocates off of the humerus laterally in supination and upon reduction in pronation a clunk is heard and felt. The elbow is then flexed to past 90° and held in a splint in pronation. An X-ray is then taken to determine that the reduction is adequate. After 5 to 7 days the elbow is moved, first in flexion then extended as tolerated in an active-assisted mode. A hinged splint may be worn and after 3 to 6 weeks all immobilisation if motion has returned and the patient feels stable all splints are removed.

Indications for surgery range from incarceration of a bony fragment in the joint space, to vascular impairment or gross instability usually associated with a coronoid fracture (see above). Late contracture or heterotopic bone may also lead to surgery at a later stage if mobility is severely limited (>30° of flexion deformity). Late instability may also necessitate surgery and the use of a hinged fixator allowing stable distraction of the joint surfaces and concomitant mobilisation.

1. O'Driscoll SW, Morrey BF, Korinek S, An KN. Elbow subluxation and dislocation: A spectrum of instability. *Clin. Orthop. Relat. Res.* 280:186, 1992.

Medial instability of the elbow

Throwing athletes may develop medial instability due to medial ulnar collateral ligament (MUCL) stretching out or tearing. The patient may experience a pop or a tearing sensation during a throw. Physical examination includes looking for ulnar neuritis and Tinel's sign. The elbow is stressed in valgus at 25° of flexion and the MUCL is palpated for tautness. Further diagnostic imaging using plain stress-test x-rays, dynamic ultrasound or Arthro-MRI will fine tune the diagnosis. MUCL reconstruction using a figure of eight tendon graft as described originally by F Jobe and refined and modified more recently may then be performed.

1. Safran M, Ahmad CS, El Attrache NS. Ulnar collateral ligament of the elbow. *Arthroscopy* 21:1381, 2005.

Postero-lateral rotatory instability of the elbow

After injury or dislocation of the elbow the patient may develop a condition where recurrently he has the impression of the elbow popping or giving way or even dislocating. The symptoms are on the lateral side where the patient often has pain and discomfort. Clinical testing will reproduce the sensation of pain and instability when the elbow is stressed in valgus and supination. An audible pop can occur during this manoeuvre. It signifies that the radius and the ulna although firmly attached by the annular ligament, slip out laterally as a unit from the capitellum because of a tear of the ulnar lateral collateral ligament that laterally unites the humerus to the supinator crista of the lateral ulna. Repair may be accomplished by a tendon graft uniting the humerus to the supinator crista of the ulna and passing under the radial head.

1. Nestor BJ, O'Driscoll SW, Morrey BF. Ligamentous reconstruction for posterolateral rotatory instability of the elbow: *J Bone Joint Surg Am.* 1992;74(8):1235-41.

The stiff elbow

The normal elbow has a range from 0° or slightly hyperextended to 150° of flexion, pronation is 75° and supination is 85°. The stiff elbow becomes a clinical problem when the functional arc accepted in flexion/extension diminishes beyond 130°-30°-0. Very severe stiffness occurs when the total arc is less than 30°, severe stiffness is when the arc is between 31° and 61°, moderate between 61° and 90° and minimal when the arc is greater than 90°. A 100° range of pron/supination (50° pronation and 50° of supination) is necessary for normal function although as a rule lack of pronation is in general less tolerated than lack of supination.

If no bony abnormalities are present the lateral column procedure, where the anterior and posterior contracted capsule is excised from a lateral approach after detaching the distal fibers of the brachioradialis and the extensor carpi radialis longus is recommended. Medial release detaching the flexor-pronator mass is performed in case of arthritic osteophytes, caring for the ulnar nerve. It may be combined with the lateral column procedure. For the rehabilitation it is important to immobilize during night-time the elbow in the position of greatest motion loss. If extension is to be gained the elbow should be immobilized in extension during night-time and flexion during the day.

For more complex conditions with bony deformity, ectopic bone, major osteophytes overgrowth or posttraumatic conditions a posterior approach with of sculpturing of deformed bony surfaces, excision of new bone formation and sectioning of restraining tissues will have to be performed. In some of these cases a hinged uni or bilateral humero-ulnar external fixator allowing controlled motion will need to be used. The ulnar nerve will need special care and transposition may be indicated in some cases. The radial nerve may be at risk when external fixation is used.

Some authors in cases of minimal or moderate stiffness have used arthroscopic release techniques.

1. Ball CM, Meunier M, Galatz LM, Calfee R, Yamaguchi K. Arthroscopic treatment of post-traumatic elbow contracture. *J Shoulder Elbow Surg* 2002;11:624-629.
2. Mansat P, Morrey BF. The column procedure: A limited lateral approach for extrinsic contracture of the elbow. *J Bone Joint Surg Am* 1998;80:1603-1615.
3. Morrey BF. The posttraumatic stiff elbow. *Clin Orthop Relat Res* 2005;26-35.

Tendon ruptures and athletic injuries

Distal biceps tendon ruptures

The distal biceps is the most commonly ruptured tendon around the elbow. This usually occurs with heavy lifting. The patient reports hearing a pop or a crack in the anterior region of his elbow. In the hours that follow the injury an ecchymosis may discolour the antecubital fold. The biceps muscle belly does not retract immediately because it is held down by the lacertosis fibrosus. The patient will have near normal flexion extension strength but will complain of weakness in supination. In an active population the treatment is usually surgical and a two incision reattachment technique as described by Morrey yields satisfactory results. When using this technique care must be taken not to come into contact with the proximal ulna when bringing the distal biceps through the ulno-radial space so as to avoid an osseous synostosis. Gentle flexion-extension exercises follow the surgery and at 6 weeks a full

return to activity is permitted.

Rupture of the brachialis and of the triceps tendons have been reported. These are rare injuries and the best surgical treatment consists in suturing the ruptured tendons.

1. Papandrea RF: Two-incision distal biceps tendon repair, in Yamaguchi K, King GJW, McKee O'Driscoll SW : *Advanced Reconstruction Elbow*. Rosemont, IL, American Academy of Orthopaedic Surgeons, 2006, pp 121-128.

Lateral epicondylitis (Tennis elbow)

The most comprehensive description of the pathoanatomy of epicondylitis is Nirschl's. The essentially this is an overuse lesion causing tearing of the extensor carpi radialis brevis tendon at its distal humerus insertion. Diagnosis is made by eliciting pain on palpation of the lateral epicondyle, wrist extension against resistance as long finger extension against resistance will also produce pain at the elbow in case of epicondylitis. All other conditions leading to elbow pain such as carpal tunnel, radial nerve entrapment under the arcade of Frohse or radiohumeral arthritis should be eliminated. Adjunct imaging such as plain x-rays will not be specific and MRI may be used to image a tear or an edematous area in the region of insertion. Treatment consists of modifying activity, steroidal infiltration, adapted physiotherapy and in case of a long duration of symptoms surgical excision of the ECRB tendon, situated under the Extensor Carpi Radialis Longus tendon. Most authors recommend open procedures although success has been reported using arthroscopic techniques. A characteristic angiofibroblastic hyperplasia-tendinosis has been described by Nirschl which characteristically demonstrates little inflammatory cells. Post-operative treatment consists of a protective splint followed by gentle motion as tolerated with full function possible 6 to 8 weeks postoperatively.

1. Kraushaar BS, Nirschl RP. Tendinosis of the elbow (tennis elbow). *Clinical features and findings of histological, immunohistochemical, and electron microscopy studies*. J Bone Joint Surg Am 1999;81:259-278.
2. Nirschl RP, Pettrone F. Tennis elbow: The surgical treatment of lateral epicondylitis. J. Bone Joint Surg Am. 1979; 61:832.

Medial epicondylitis

Rarely, in the competitive athlete pain will develop following overuse of the flexor-pronator complex. Again the treatment should first be conservative. If symptoms persist surgical excision of the diseased part of the medial conjoint tendon of the flexor-pronator complex may be considered. In some cases a transposition of the ulnar nerve completes the procedure.

1. Vangsness CT, Jobe FW. Surgical treatment of medial epicondylitis. Results in 35 elbows. J Bone Joint Surg Br. 1991;73(3):409-11.

Osteochondritis dissecans

Rare condition affecting mostly skeletally immature patients involved in receptive throwing sports. Symptoms include pain, flexum deformity or catching and locking. This should be differentiated from Panner disease which is an osteochondrosis not requiring treatment. A classification has been evolved that spans from the simple cartilage fissures (I) to the detachment of a large fragment (IV) of cartilage. Treatment is at first conservative with activity modification and if not successful can go,

depending on severity, from simple drilling of the lesion to complex mosaicplasty.

1. Baumgarten TE, Andrews JR, Satterwhite YE: The arthroscopic classification and treatment of osteochondritis dissecans of the capitellum. Am J Sports Med 1998;26:520-523.

Septic olecranon bursitis

This is a potentially life threatening condition caused by a septicaemia originating from an infected bursa under tension. Generally seen in debilitated patients but can arise without a clear cause or after minor trauma in an otherwise healthy individual. Diagnosis is clinical with standard laboratory findings such as high white cell count, left shift of white blood cells, high sedimentation rate and elevated C-reactive protein levels. Organisms found are generally staphylococcus or streptococcus. In mild cases treatment may be antibiotics and splinting. In more severe cases it is mandatory to incise the bursa and leave open, to immobilise in a splint and after a few days to perform a closure secondarily. During the treatment period appropriate IV and oral antibiotics are administered.



1. Pien FD, Ching D, Kim E. Septic bursitis: Experience in a community practice. Orthopaedics 1991; 14:981.
2. Infectious olecranon and patellar bursitis: short-course adjuvant antibiotic therapy is not a risk factor for recurrence in adult hospitalized patients. Perez C, Huttner A, Assal M, Bernard L, Lew D, Hoffmeyer P, Uçkay I. J Antimicrob Chemother. 2010 Mar 1. [Epub ahead of print]



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Upper limb trauma: Forearm

In the AO manual that covers all important fixation techniques the reader will find pertinent facts related to the topic at hand: *AO Principles of Fracture Management. Second expanded edition, TP Rüedi, RE Buckley, CG Moran. Georg Thieme Verlag; 2 Har/Dvdr edition (2007).* For the latest in fracture fixation techniques the reader is also invited to visit the AO surgery reference site: *AO surgery reference: <http://www.aofoundation.org>.* Another most useful publication containing pertinent facts related to orthopaedics and musculoskeletal in general trauma is the *AAOS Comprehensive Orthopaedic Review. Jay R. Lieberman, MD, Editor 2009 Rosemont, IL, American Academy of Orthopaedic Surgeons.* For surgical approaches the most useful reference is without doubt: *Surgical Exposures in Orthopaedics: The Anatomic Approach. Hoppenfeld S, deBoer P, Buckley R. Lippincott Williams & Wilkins; Fourth Edition 2009.*

Introduction forearm

The forearm must be considered as a whole functioning joint allowing pronation of 75° and supination of 85°. The interosseous membrane plays a major stabilising role.

1. *AAOS Comprehensive Orthopaedic Review. Jay R. Lieberman, MD, Editor 2009 Rosemont, IL, American Academy of Orthopaedic Surgeons.*

Classification of forearm fractures

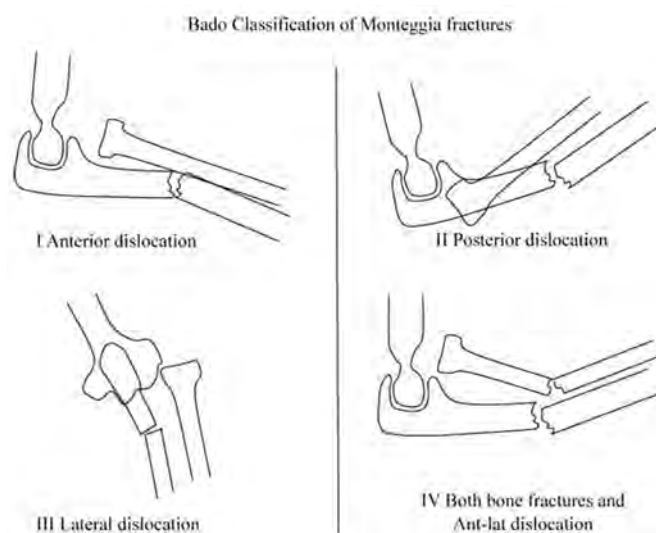
The AO classification is popular. It classifies the fracture patterns of radial and ulnar shafts as type A (simple, transverse or spiral) type B (wedge with a butterfly fragment) and type C (segmental or comminuted fragments). Open fractures are classified according to Gustilo and Anderson: Type I inside-out (< 1 cm), Type II outside-in (> 1cm), Type III A (open but osseous coverage possible), type III B (open necessitating a local or free flap), Type III C any open fracture with vascular injury. Specific to the forearm are the *Monteggia* fracture pattern (Fracture of the ulna with dislocation of the radial head).

Bado classification:

- I Anterior radial head dislocation and proximal ulnar shaft fracture (apex anterior)
- II Posterior or postero-lateral radial head dislocation and proximal ulnar shaft fracture (apex posterior)
- III Lateral radial head dislocation and proximal ulnar shaft fracture (apex posterior)
- IV Anterior radial head dislocation and proximal ulnar and radial shaft fracture (apex posterior)

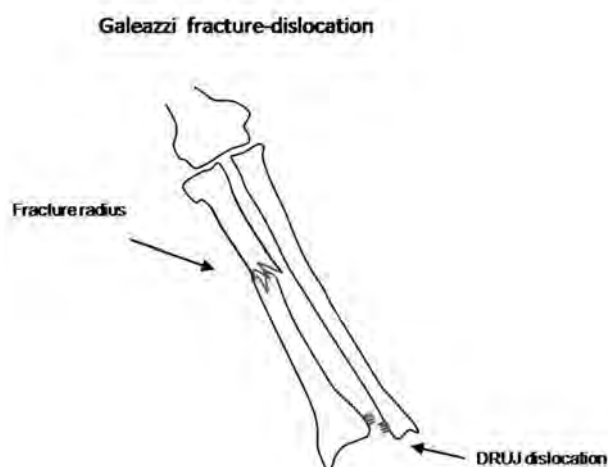
1. *Konrad GG, Kundel K, Kreuz PC, Oberst M, Sudkamp NP. Monteggia*

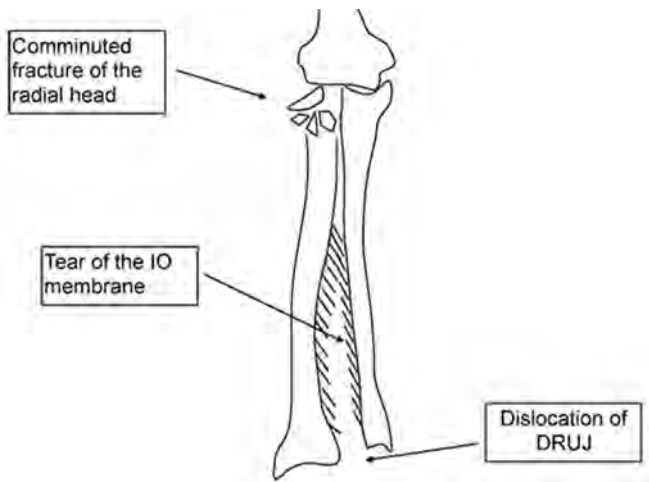
fractures in adults: long-term results and prognostic factors. J Bone Joint Surg Br. 2007;89(3):354-60.



Also specific to the forearm is the *Galeazzi* fracture pattern where the radius shaft is fractured along with a dislocation of the distal radio-ulnar joint (DRUJ). Suture of the triangular ligament or pin fixation of the DRUJ are indicated if after fixation of the radial shaft gross instability is still present.

1. *Rettig ME, Raskin KB. Galeazzi fracture-dislocation: a new treatment-oriented classification. J Hand Surg Am. 2001;26(2):228-35.*





Essex-Lopresti fracture-dislocation

The Essex-Lopresti lesion combines a comminuted fracture of the radial head along with disruption of the interosseous membrane causing a relative overlengthening of the ulna at the wrist. (see Elbow section).

1. Perron AD, Hersh RE, Brady WJ, Keats TE. Orthopedic pitfalls in the ED: Galeazzi and Monteggia fracture-dislocation. *Am J Emerg Med.* 2001 May;19(3):225-8.
2. AO Principles of Fracture Management. Second expanded edition, TP Rüedi, RE Buckley, CG Moran. Georg Thieme Verlag; 2 Har/Dvdr edition (2007).
3. Gustilo RB, Mendoza RM, Williams DN. Problems in the management of type III (severe) open fractures. A new classification of type III fractures. *J Trauma* 1984, 24:742-746.
4. Gustilo RB, Anderson J. Prevention of infection in the treatment of one thousand and twenty-five open fractures of long bones: Retrospective and prospective analyses. *JBone Surg. Am* (1979).

Clinical presentation of forearm fractures

A deformed extremity is present. Look for neurovascular injuries (radial nerve) carefully before any manipulation of the injured extremity. Plain X-rays including the shoulder and elbow are generally sufficient in acute traumatic cases. MRI, CT, Bone scintigraphy are useful in special situations such as chronic infection, metastatic or primary tumors.

Conservative treatment

There is practically no place for conservative treatment in adult both bone forearm fractures. Isolated fractures of the ulnar shaft may be treated by functional bracing but the rate of non-union remains high and many authors recommend immediate plate fixation.

1. Mackay D, Wood L, Rangan A. The treatment of isolated ulnar fractures in adults: a systematic review. *Injury.* 2000;31(8):565-70.

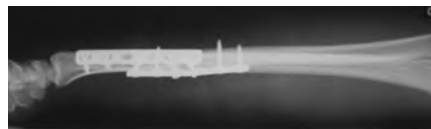
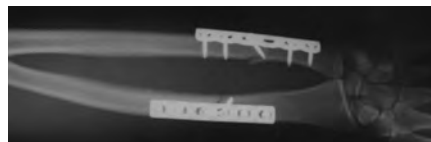
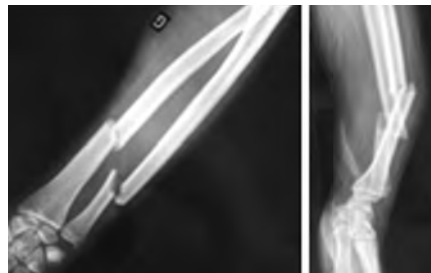
Surgical approaches

- Anterior (Henry) approach
Anatomic approach but with some soft tissue stripping. Allows exposure of the whole radius.
 - Dorsal Thompson approach
Danger to the Posterior Interosseous Nerve (PIN).
1. http://www.wheelessonline.com/ortho/dorsal_approach_thompson

- Direct approach

The direct approach is best suited for the ulna.

1. Mekhail AO, Ebraheim NA, Jackson WT, Yeasting RA. Vulnerability of the posterior interosseous nerve during proximal radius exposures. *Clin Orthop Relat Res.* 1995 Jun;(315):199-208. Erratum in: *Clin Orthop* 1997;(334):386.



Operative treatment indications:

The forearm constitutes a joint and in the adult the treatment is anatomical reduction and fixation

- IM Nailing
Difficult to guarantee stable fixation and anatomic fixation with these devices.
 - Anterograde
Nails for the ulna are in use and being developed
 - Retrograde
Nails for the radius may be used
- Plating
3,5 mm plates should be used and never semi or third tubular type plates. 6 cortices on each side of the fracture should be used.

Both bones fracture of the forearm fixed anatomically with rigid compression plating

External fixation

In case of open fractures an external fixation may be applied. For the ulna the pins may be applied closed but for the proximal radius an open approach allowing to identify the pertinent neurovascular structures should be performed.

1. Hertel R, Pisan M, Lambert S, Ballmer FT. Plate osteosynthesis of diaphyseal fractures of the radius and ulna. *Injury.* 1996;27(8):545-8.
2. Chapman MW, Gordon JE, Zissimos AG. Compression-plate fixation of acute fractures of the diaphyses of the radius and ulna. *J Bone Joint Surg Am.* 1989;71(2):159-69.
3. Lindvall EM, Sagi HC. Selective screw placement in forearm compression plating: results of 75 consecutive fractures stabilized with 4 cortices of screw fixation on either side of the fracture. *J Orthop Trauma.* 2006;20(3):157-62; discussion 162-3.



EPOS – EFORT Instructional Course – 09 to 11 October, Vienna

Paediatrics: Basic Course II

- Disorders of the upper limb
- Disorders of the knee
- Musculoskeletal infections
- Spine



EFORT Instructional Course – 15 & 16 November, Basel

Osteoarthritis: Joint preserving surgery (JPS) of the lower extremity

- Joint preserving surgery around the hip
- Joint preserving surgery around the knee
- Joint preserving surgery around the ankle

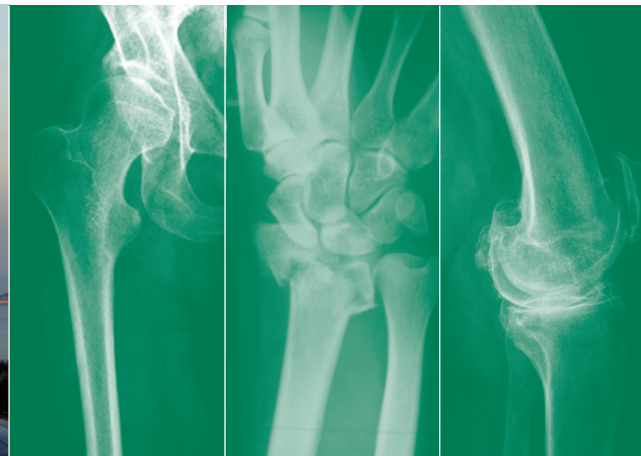
Hands-on Workshops:

- Hip: Osteotomies, arthroscopy, impingement surgery, chondral repair
- Knee: Osteotomies, arthroscopy, chondral repair, ligament repair
- Ankle/Hindfoot: Osteotomies, arthroscopy, chondral repair, ligament repair

CRC Course

Istanbul, 6 June 2013

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EFORT SYLLABUS

The Comprehensive Orthopaedic Review Course (CRC)

During the 14th EFORT Congress Istanbul: 6 June 2013

Course highlights

- Basic Science
- Tumour
- Paediatrics
- Spine (incl. Trauma)
- Reconstruction
- Sports knee
- Trauma