CRC Course

London, 5 June 2014

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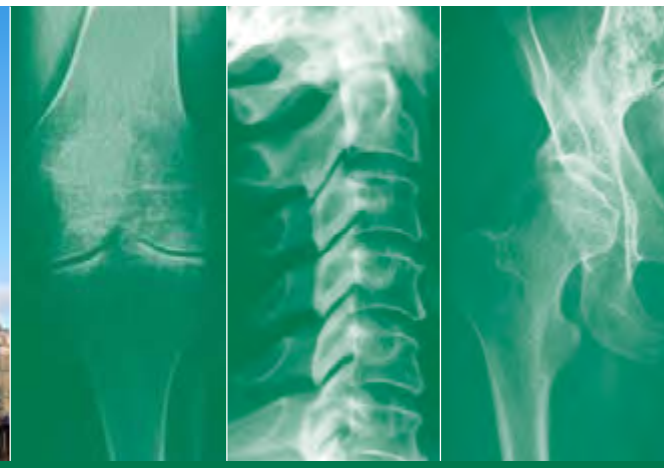

EFORT SYLLABUS

The Comprehensive Orthopaedic Review Course

During the 15th EFORT Congress London: 5 June 2014 #CRC

Course highlights

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

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Introduction
Welcome

Enric Cáceres Palou

Autonomous University of Barcelona,
Barcelona, Spain

Email: ecaceres@vhebron.net

The EFORT Comprehensive Review Course is a quick review of the essentials of our specialty for those who want to review in a single day the basic and fundamental knowledge of Orthopaedic surgery and Traumatology.
The information is presented in a concise and brief way, gathering knowledge of the orthopaedics and traumatology that currently have a high degree of evidence .
The syllabus, year after year, trys to summarize in a few pages the elements which are presented during the course in a brief and easy way to understand.
It starts with the fundamental knowledge of the basic sciences and general, such as tumour conditions , infections and metabolic diseases.
Orthopaedics and trauma are reviewed by each subspecialty within the most important information from the fields of spine, paediatrics, and then the key aspects of the trauma and orthopaedic in the upper and lower limb.
Reconstructive surgery of bones and joints and sports medicine are treated succinctly and without forgetting complications.
The course is conducted by experts in each field and to whom we appreciate your participation and effort in the preparation of this syllabus .
In the future our goal is to achieve in the different editions a clear , brief and concise text and in successive editions, attempt to improve the content and help prepare the essential knowledge necessary to overcome the different assessments of European countries and especially the EBOT Exam .
This is a result of the comments we receive each year from the help every year to improve the content of the Comprehensive Review Course of EFORT
We wish to thank the collaboration of all the different authors , the work of the Head Office staff and the EFORT Publications Committee in order to achieve these objectives

Enric Cáceres Palou
Scientific Chairman of EFFORT

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Eye Opener

P. HOFFMEYER, Geneva

University Hospital
Geneva, Switzerland
pierre.hoffmeyer@hcuge.ch

Patient safety is today's major health issue.

Patient safety is the foremost health issue that orthopaedic and trauma surgeons face and must tackle today. Over 50 wrong site operations are performed in Europe every week! Based on a conservative estimate, it may be surmised that 175,000 deaths are attributable to death every year in Europe*. Expenditures of these untoward events are staggering and numbered in the billions! Even though the problem is of a great magnitude, relatively simple measures involving method, discipline and low cost actions may help to effectively reduce the dimensions of the problem. These measures include the use of checklists, alcohol based hand washing, registries and flawless technique.

Clearly, checklists are of a huge benefit in the aviation world which boasts the lowest accident rates of all industries. For surgical interventions checklists have been shown to diminish wrong site surgery, to curb infections and unexpected readmission to the operating theatre and in some countries have even diminished mortality.

Alcohol based hand washing before and after each clinical encounter with a patient must be observed. It's effectiveness is proven by a multitude of studies but it still seems to be an uphill battle to persuade all health care givers to seriously and conscientiously apply this simple life saving measure.

Implant registries will help to detect early bad outcomes due to poor implants or insufficiently mastered techniques. This has been shown effective by the Nordic countries' registries which have now conquered practically every European nation.

Lastly and importantly surgical techniques matter and the pertinent anatomy must be mastered. Courses are available widely to perfect our techniques and indications.

All these measures have been shown to reduce the burden of iatrogenic and hospital acquired disease. We, the community of orthopaedic surgeons, need to act and to proactively implement these measures before some else does it for us!

**Guggenbichler J P et al. Incidence and clinical implication of nosocomial infections associated with implantable biomaterials – catheters, ventilator-associated pneumonia, urinary tract infections. GMS Krankenhhyg Interdiszip. 2011; 6(1): Doc18.)*

Prof. Dr. K. E. Tanner

University of Glasgow,
Glasgow, UK

e-mail address: elizabeth.tanner@glasgow.ac.uk

Biomechanics and Biomaterials

Introduction

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 so only those areas of importance to orthopaedic surgery will be considered in this lecture.

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 must act on it, secondly how fast it moves depends on the magnitude (size) 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. Think about bending an elbow with nothing the your hand compared with the same action with a weight in your hand. The muscle is contracting by the same amount but the force is generated by reacting against the mass in the hand is different. 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, in the body this is the muscle force. 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 (Fig 1). 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.

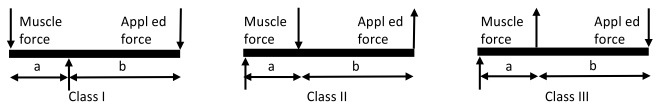

Fig 1 Types of Levers depending on the relative positions of the loads and fulcrum

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 small 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 upwards forces in the arm through to the body must be equal and the moments (calculated as force multiplied by distance) 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.

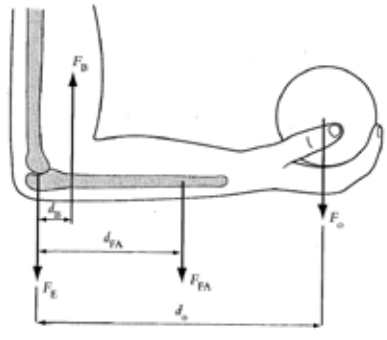

Fig 2 Calculation of forces in the forearm holding a mass

When calculating the moment produced by a force the distance between a point and the force has to be taken along a perpendicular to the line of action of the force (Fig 3). So while gravity can be taken as being vertical most muscles act at an angle thus care has to be taken in calculating the moment.

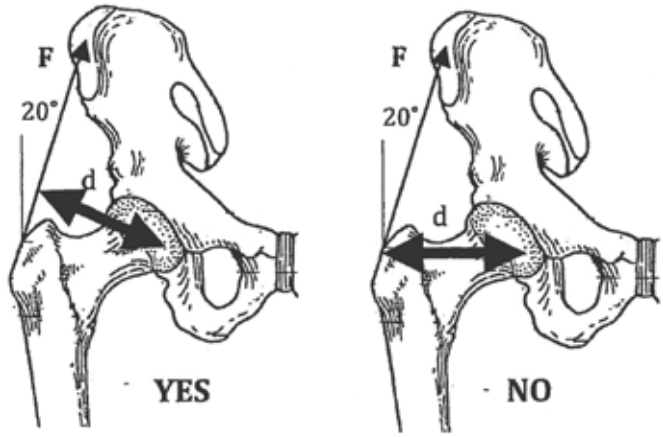

Fig 3 Calculating the moment exerted by a force F, requires the distance d to be along the line perpendicular to the direction of the force

If we apply these calculations to a person standing on one leg and making appropriate assumptions of distances in the body (Fig 4), then we can calculate that the load on the femoral head, by looking at the vertical and horizontal forces and the moments.

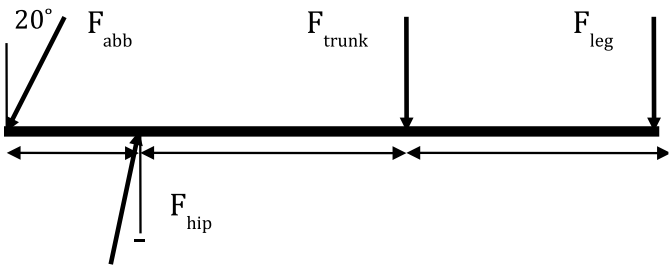

Fig 4 Forces at the hip in one legged stance

The vertical components of force give:

$$F_{hip}\cos\theta = F_{abb}\cos 20 + F_{trunk} + F_{leg}$$

Horizontally we get:

$$F_{hip}\sin\theta = F_{abb}\sin 20$$

And taking moments about the hip joint gives:

$$F_{abb}\,50\cos 20 = F_{trunk} \times 100 + F_{leg} \times 200$$

Remembering to take the perpendicular distance to the line of action of the abductors. The weight of each leg is approximately 16% of body weight, giving the trunk 68% of the body weight. So working through these calculations, the hip force is 2.58 times the subject's body weight and the forces in the abductor muscles is 1.77 times body weight. If these simplistic 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.

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 truely occurs as there is always some 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. Some of their properties are given in Table 1. In stainless steel 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, the fatigue properties are acceptable. Cobalt Chrome alloy contains 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. There are three major groups of titanium: commercially pure, Ti-6%Al-4%V 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, that 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 stiffnesses 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 and alumina and the bioactive mainly hydroxyapatite and tricalcium phosphate. The bioinert ceramics are principally used for articulating surfaces as either ceramic-on-polymer or ceramic-on-ceramic. Initially Al2O3 was preferred as ZrO2 can be morphologically unstable but now PSZ (Partially Stabilised Zirconia) is available. Al2O3 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 calcium phosphate - 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 2/3rds pre-polymerised and 1/3 monomer the exotherm and skrinkage 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. 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

Material	Contents	Young's modulus /GPa	Strength /MPa	Strength /MPa
Bone	40 vol% bone mineral in collagen	7-25	50-150	
Stainless Steel (316/316L)	18% Cr, 13% Ni, 2.5% Mo, rest Fe	210	520-680	Ductile, can be cold worked. Good fatigue reasonable wear
Cobalt chrome	27-30% Cr, 5-7% Mo, rest Co	230	600-1140	Contains no Ni. Very good fatigue and good wear properties
Commercially pure titanium	>99% Ti	110	170-500	Poor wear properties and debris is black in body
Ti-6Al-4V	6% Al, 4% V, rest Ti	110	880-950	Contains no Ni, lowish modulus, poor wear properties
Nitinol	50%Ti, 50%Ni	28-40 or 75-83	70-140 or 195-690	Shape memory alloy, properties depend on nitinol phase
Bone cement	Polymethylmethacylate plus opacifiers and antibiotics	2.4	40-60	

Table 1 Mechanical properties of bone and metals used in orthopaedic applications.

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 prophylatically to bone cement to reduce the risk of infection (Jiranek, Hanssen et al. 2006).

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 CO2 and H2O 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 were the two phases can be seen as separate either with the naked eye or using a microscope, that 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 is it being loaded and the second is what is it made of. Without appropriate interactions between both of these factors an implant will not be successful

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Prof. Dr. Karsten Dreinhöfer

Charité Universitätsmedizin Berlin
Berlin, Germany
karsten.dreinhoefer@efort.org

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 (Ca10 (PO4) 6OH2) 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 reabsorbtion 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 reabsorbtion 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 resorbtion 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 synthesis 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 affect 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 tonedue 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 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 or greater 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, ortheses 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 calcitrol) 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 a increased likelihood of fractures. Bone marrow narrowing leads to extramedullary hematopoesis, 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 und 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 ostrodystrophy:

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 ostrodystrophy therefore shows combination of excessive bone erosion by osteoclasts, failure of mineralisation of osteoid collagen (osteomalacia), osteosklerosis 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	▼	▼	▲ ø ▼	▲	soft bones
Osteitis fibrosa cystica	▲	▼	▲	▲	brown tumors
Osteoporosis	(▲)	ø	▲ ø ▼	ø	decreased bone mass
Osteopetrosis	ø	ø	ø	ø	thick dense bones
Paget's disease	ø	ø	▲	ø	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.

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Prof. Dr. Bjarne Møller-Madsen

Aarhus University Hospital
Aarhus, Denmark
bj.moma@gmail.com, www.dpor.dk

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).

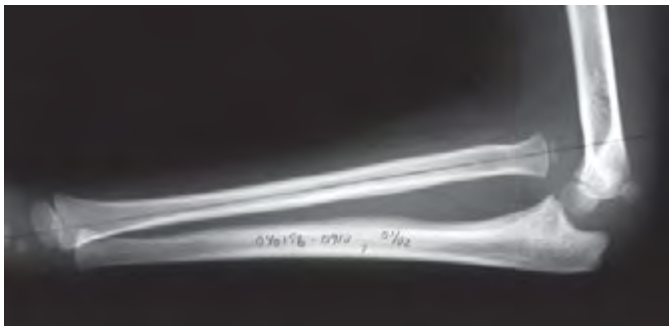
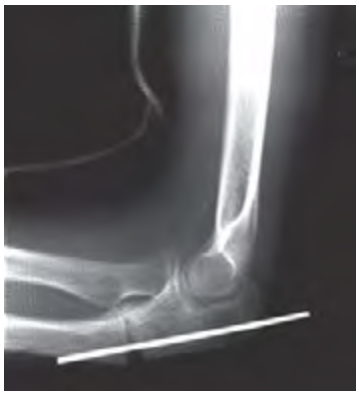


Fig. 1



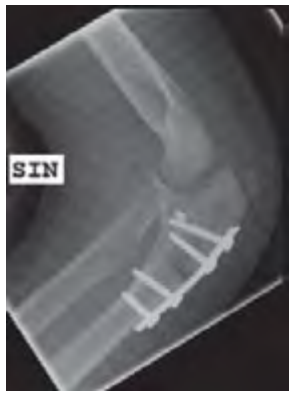


Fig. 2

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 capittum 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 throchlea (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).

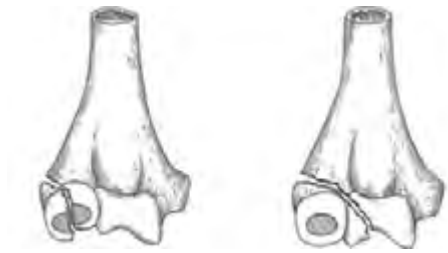


Fig. 3

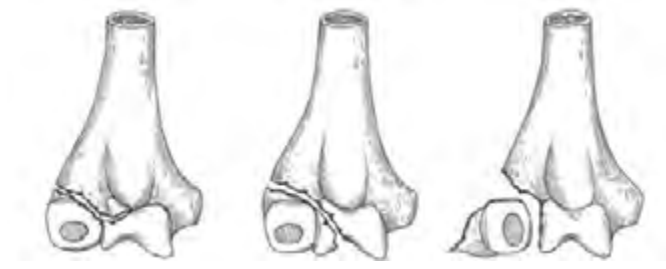


Fig. 4

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.

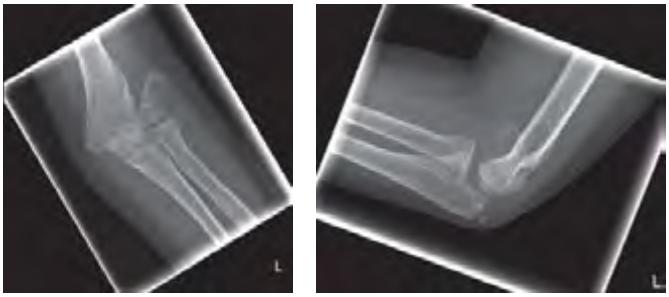


Fig. 5

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Assoc. Prof. Christof Radler, MD

Orthopaedic Hospital Speising
Vienna, Austria
E-mail: christof.radler@oss.at

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 [38].

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 [30].

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 [26,27].

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 [19]. 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 adductus (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.

Fig 1: Mild but persistent metatarsus adducts

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 [20]. 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 [18]. In rigid cases a follow up treatment with IPOS shoes or reverse-last shoes should be performed. A recent study suggests similar results of casting versus using an orthosis [14]. However, the orthosis seems to require more active parental cooperation and should only be recommended to very compliant families.

Most cases will correct well with the described treatment regimes. 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 usually 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 [11].

For the very rare case with severe persistent deformity and significant foot wear problem a minimal invasive procedure has recently been described [21]. However, 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.

1.2 Skewfoot

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 [12].

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 (Figure 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 (Figure 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 [25].

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 [10].

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

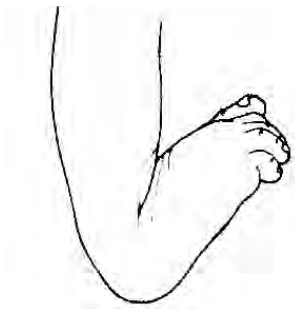

Fig 2: Schematic drawing of a talipes calcaneovalgus

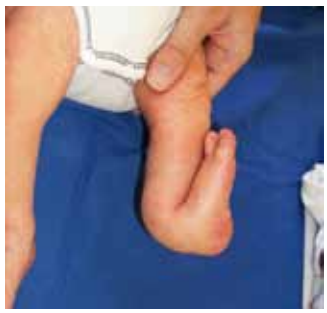

Fig 3: Posteromedial bowing. Note that the deformity is in the tibia only

neural and no sign of the rigid deformities seen in true clubfoot (Figure 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.

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.

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

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 polygenetic threshold model explaining the inheritance patterns [6].

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 (Figure 5 a,b).

Additionally to the displacements of the bones there are marked structural deformities like the talus dome which is mildly flattened [15] which might be increased during treatment by over- vigorous casting,

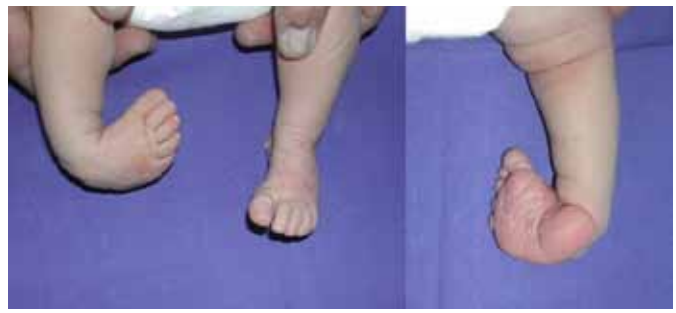

Fig 5a: Unilateral clubfoot in a 5 days old girl with Pirani 5.5.

the ligaments are less flexible and contracted, the muscles are more fibrotic and cells of the medial ligamentous tissue have myofibroblastic characteristics [16,34].

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 [33].

Treatment should be started within the first weeks of life.

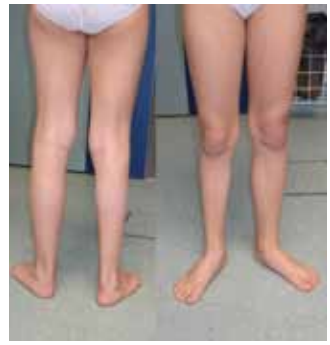

Fig 5b: Same patient at age 9 years after Ponseti treatment with additional tibialis anterior tendon transfer

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 [35].
In the last 10 years the Ponseti method has become the gold standard for the treatment of clubfoot worldwide [17]. 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 [28,29]. 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 [31]. 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 week peroneal muscles and an overpowering tibialis anterior muscle.
Mid- and longterm results are very good and superior to all other treatment regimens or surgical approaches reported in literature [3-5,32,36].

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 (Figure 6). Mostly cases are associated with
syndromes like arthrogryposis, trisomy 18, sacral agenesia 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 [22]. However, isolated CVT can be transmitted as an autosomal dominant trait with variable expression and incomplete penetrance [8].
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%) [23].
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 drorsiflexion 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 [7]. 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 [7,9].

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

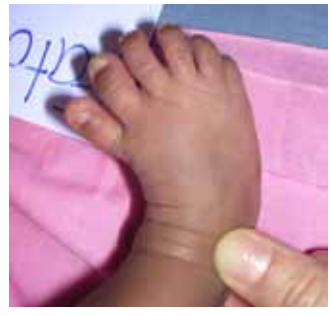

Fig 7: Tibial preaxial polydactyly with duplication of the first ray.

surgical correction flexor tenotomy has been shown to be effective. [13]

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 crafts 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 [2,37]. 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 (Figure 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 [24]. 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 [39].

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Dr. Rudolf Ganger

Orthopedic Hospital Vienna Speising
Vienna, Austria
rudolf.ganger@oss.at, www.oss.at

Hip Diseases in the Childhood

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

1.2.1. Neglected cases of hip dislocation

1.2.2 Residual dysplasia of the hip

1.2.3 Osteonecrosis of the hip after treatment of DDH
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].

1.2. DDH after walking age

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

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

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].

1.2.1 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.

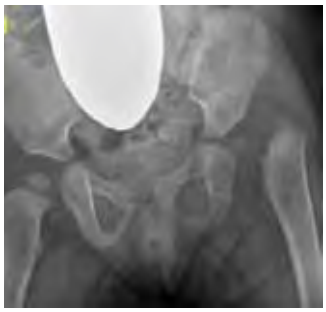

Figure 1a: Two-year-old girl with unilateral untreated dislocation of the left hip.

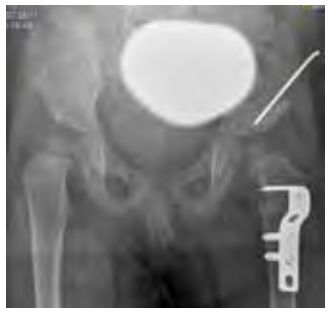

Figure 1b: The reduced left hip of the same girl. Six weeks after open reduction, Pemberton acetabuloplasty and femoral osteotomy (varus, shortening and derotation).

1.2.2 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).

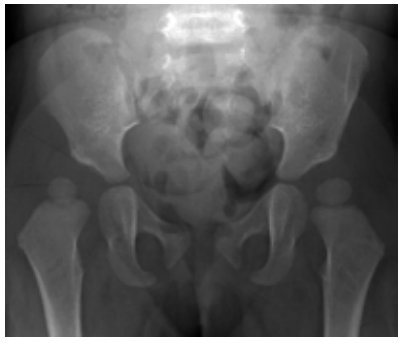

Figure 2: Three-year-old girl with bilateral treated dislocation of the hip at the age of five months (closed reduction of the right hip, open reduction of the left hip). At follow up both hips show residual acetabular dysplasia with subluxation of the right hip and disrupted Shenton lines on both sides.

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 ostetomy 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.

1.2.3 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-Odgen 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, b).

2. Transient (toxic) Synovitis

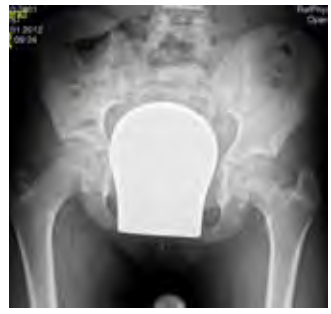

Figure 3a: 11-year-old girl with an closed reduction of the dislocated left hip at the age of five months. At follow up signs of osteonecrosis with a trochanteric overgrowth were evident.

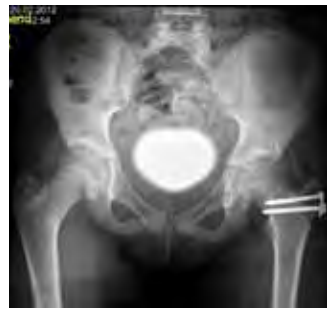

Figure 3b: Three months after the trochanteric advancement surgery. The greater trochanter is now below the level of the top of the femoral head.

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).

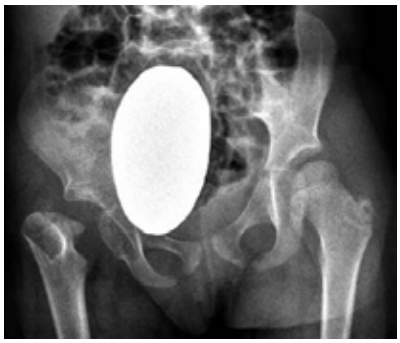

Figure 4: Radiograph of a three-year-old girl after neonatal septic arthritis of the right hip. Complete destruction of the femoral head and neck are visible.

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.

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

Table 2: Radiographic stages of Legg-Calve-Perthes Disease

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 resoprtion (Salter-Thomspon 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]

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 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.

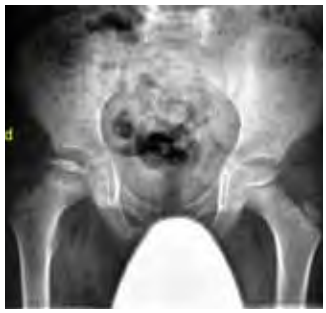

Figure 5a: Seven-year-old boy suffering from Perthes disease on the right hip. Radiological signs of "head-at-risk" with lateralization of the femoral epiphysis, horizontal physeal line and the Gage sign are evident.

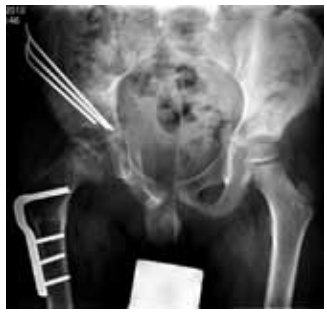

Figure 5b: Radiographs of the same boy, five months after Salter innominate and femoral varus osteotomy. The femoral head is well covered.

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 techniqually 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.

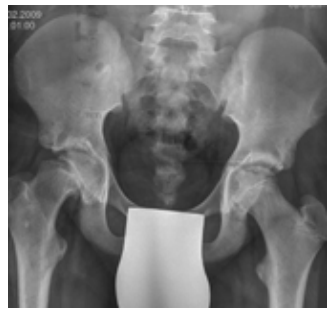

Figure 6a: 14-year-old boy with a severe, chronic slipped capital femoral epiphysis of 60 degrees.

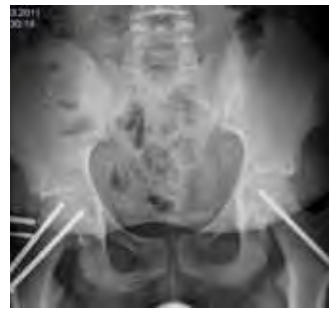

Figure 6b: Radiographs of the same boy two years after surgery. The right hip was treated with open reduction and subcapital osteotomy through a surgical dislocation approach. The left hip was fixed prophylactically with a cannulated single screw.

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Mr. Stephen Cannon

The Royal National Orthopaedic Hospital
Stanmore, UK
stephen.cannon@efort.org

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	Scinitig-raphy	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
Periostcal/ enosteal react	1.6	2.1			3.4

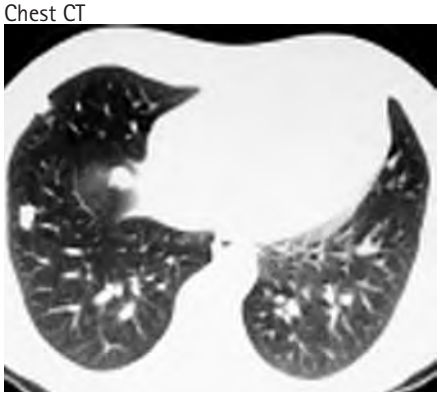
Local staging of the disease essentially wishes to draw out the local extent of the lesion and by use of an MRI of the whole bone define any local skip metastases. The local staging also requires an estimation of the extraosseus extent of the disease, the involvement of soft tissue muscle compartments, 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.

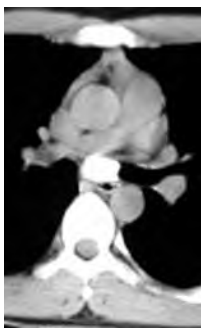
Distal staging is performed by the use of CT scan of the pulmonary lung fields as 95% of metastatic disease involves the pulmonary tissue, but a Technetium bone scan is also useful for picking up both soft tissue and bony metastases. If the bone scan picks up a distant metastasis in bone then further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

Distant Staging

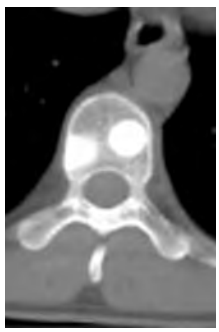
Chest CT



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 Enniking'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.

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Prof. Dr. Miklòs Szendröi

Semmelweis University
Budapest, Hungary
miklos.szendroi@efort.org

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 mulitplication occures in 1-3 years
- Pathological fracture in 20% of the cases
- Risk of pathological fractures: • more than 2 cm • lower limp (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).

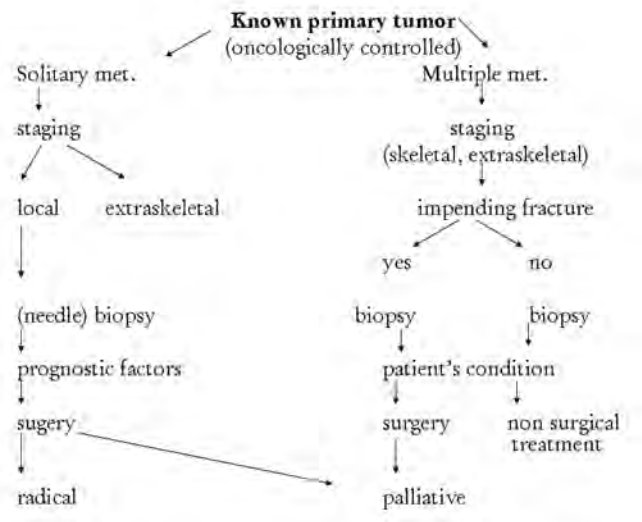

Fig. 1a Diagnostic algorythm at impending fracture (Known primary tumor)

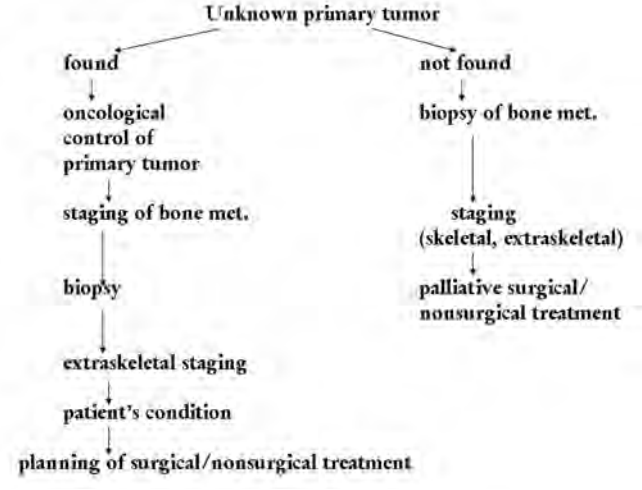

Fig. 1b Diagnostic algorythm at impending fracture (Unknown primary tumor)

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 multiorganic.

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

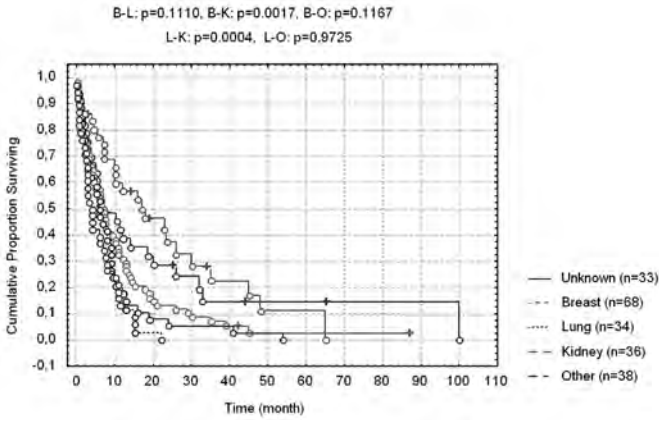

Fig. 2 Survival according to the primary site in 209 metastatic patients

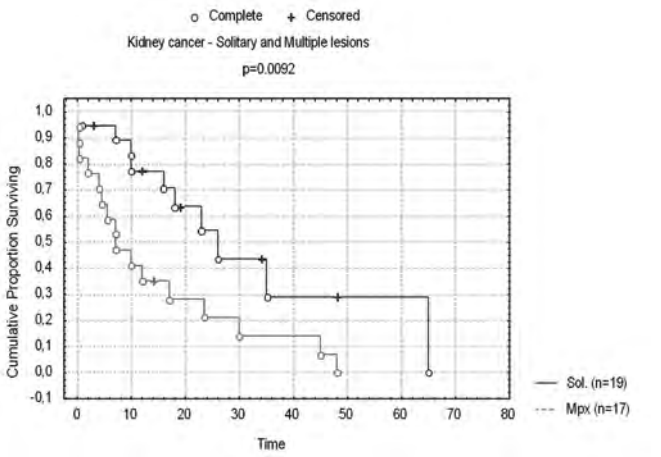

Fig. 3 Survival according to the solitary and multiple manifestations of bone metastases in kidney cancer

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 appearence 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 strenghten 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.

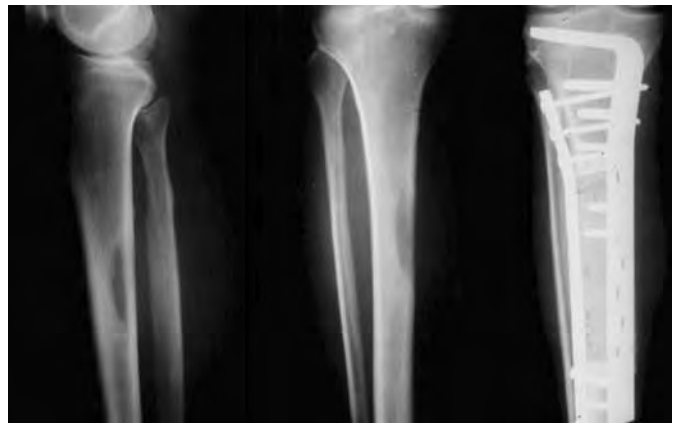

Fig. 4 Plating with cementation

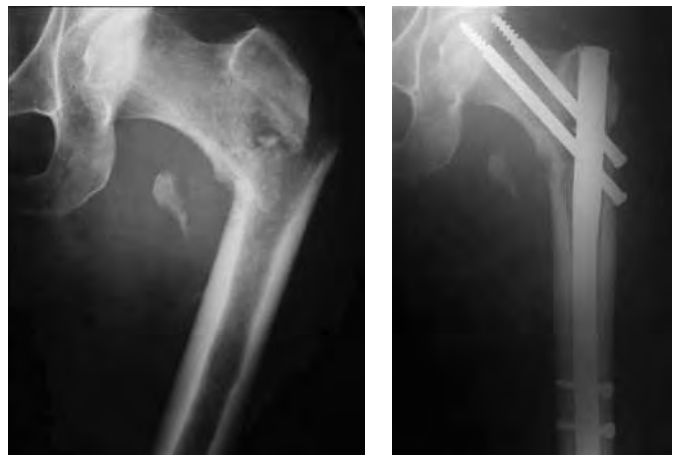

Fig. 5 Intramedullary nailing

Table 4

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

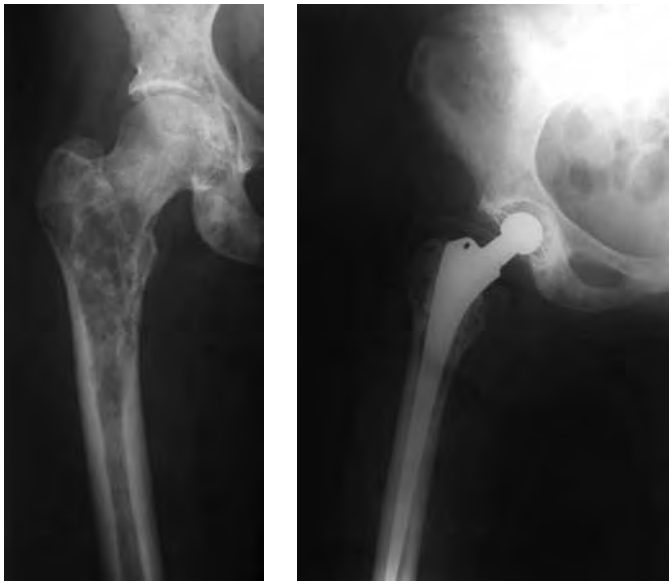

Fig. 6 Conventional cemented revision endoprosthesis

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Dr. Romain Dayer, Dr. Pierre Lascombes (PK)

University Hospitals of Geneva,
Geneva, Switzerland

e-mail: pierre.lascombes@hcuge.ch

Pediatric Idiopathic Scoliosis

Support

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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, 22}.

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 coro- nal 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 is 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{21}. 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}. None of these surgical techniques are associated with low complications rates and experienced surgical teams should use them accordingly.

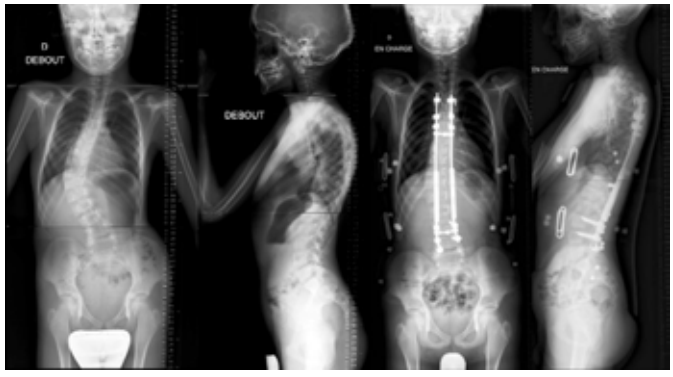

Fig. 1. Infantile scoliosis: dual growing rods technique

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 wit IS, as well as in general for pediatric deformity surgery. Preoperative oral iron and erythropoietin or autologuous 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{20}, 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. This surgical approach is clearly the most frequently favored for the surgical treatment of AIS together with aggressive derotation technique, with

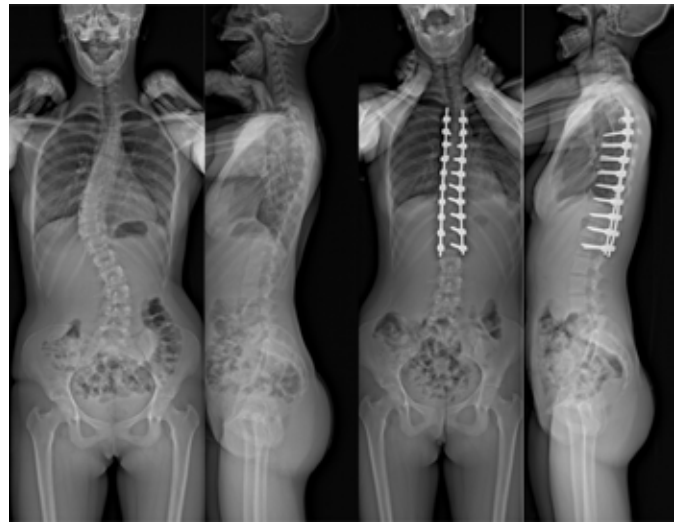

Fig. 2. AIS: posterior-only approach and instrumented fusion of a thoracic curve

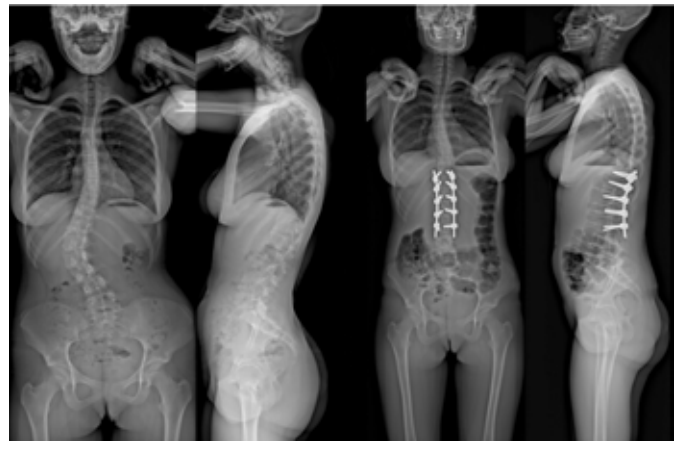

Fig. 3. AIS: posterior-only approach and instrumented fusion of a thoraco-lumbar curve

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}.

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.

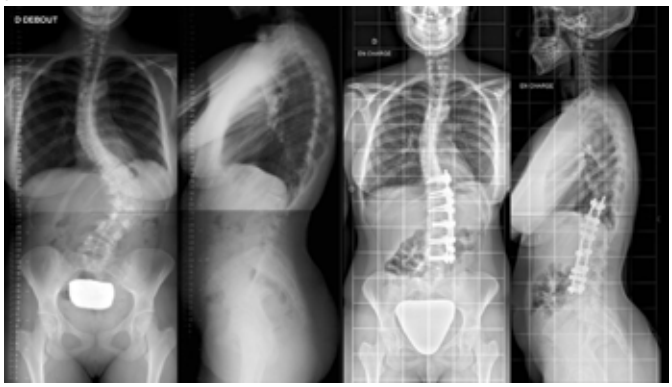

Fig. 4. AIS: anterior-only approach and instrumented fusion a thoraco-lumbar curve

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Maite Ubierna

Autonomous University of Barcelona,
Barcelona, Spain
maiteubi8587@gmail.com

Spine Fractures

1. Spinal cord injury

a. Background:

The annual incidence of SCI is approximately 40 cases per 1 million people in the United States, or 11.000 new cases per year.

1. 55% of SCIs occur in the cervical spine. The remaining injuries are equally distributed through-out the thoracic, thoracolumbar, and lumbosacral spine.
2. Motor vehicle accidents account for half of reported SCIs. Fall and recreational sport injuries are responsible for most of the remaining SCIs.
3. Neurologically, most patients sustain incomplete tetraplegia (34%), followed by complete paraplegia (25,1%), complete tetraplegia (22,1%) and incomplete paraplegia (17,5%).

b. Emergency department evaluation:

1. Respiratory pattern: SCI above C5 is more likely to require intubation, because of fatigue of the accessory respiratory muscles.º Complete tetraplegia is more likely to require intubation than incomplete tetraplegia .
2. Hemodynamic evaluation: neurogenic shock , defined as circulatory collapse resulting from neurologic injury, is caused by an interruption of the sympathetic output to the heart and peripheral vasculature. This collapse gives rise to the bradycardia and loss of vascular and muscle tone below the level of the SCI:

c. Neurological examination:

1. The American Spinal Injury Association (ASIA) standards of neurological testing provides a concise and detailed method for evaluating spinal cord and peripheral nerve root function. (Table 1)

ASIA A sensory and motor complete
ASIA B sensory incomplete, motor complete
ASIA C sensory and motor incomplete, motor function below the level of lesion in mean M3
ASIA D sensory and motor incomplete, motor function below the level of lesion in mean >M3
ASIA E no relevant sensorimotor deficit, minor functional impairments of reflex-muscle tone changes

Table 1. ASIA Impairment Scale

2. In traumatic cord injury the main classification distinguishes between paraplegia (impairment or loss of motor and/ or sensory function in the thoracic , lumbar or sacral neural segments T2-S5) and tetraplegia (impairment or loss of motor and / or sensory function in the cervical segments C0-T1).
3. A further differentiation is made with regard to the completeness of the lesion as: complete or incomplete. The distinction between complete and incomplete is based on the preservation of any sensory or motor function within the last sacral segments S4-S5.
4. The neurological examination should establish the level of SCI. Sensation is determined in all 28 dermatomes bilaterally by the patient's ability to detect the Sharp end of a pin. It is recorded as absent (0), impaired (1) or normal (2).Motor function or strength is documented and graded 1-5 based on resistance to physical manipulation or gravity.
5. Based and motor and sensory examination , the patient is further classified according the level of the lesion and graded using ASIA form (normal ASIA E to complete paralysis ASIA A).
6. Recognition of patterns of neurological deficits can help determine prognosis: Brown-Séquard carries the best prognosis, Central cord syndrome is the most common, Anterior cord syndrome carries the worst prognosis and Posterior cord syndrome.

d. Imaging evaluation:

1. Computed tomography (CT) scanning with coronal and sagittal reformatted images is useful to further define bony anatomy of the lesion.
2. Magnetic >Resonance imaging (MRI) is use dan all cases of neurological compromise or to better visualize soft tissue anatomy, that is , neural compressive lesions such as disk herniations, epidural hematomas or traumatic ligamentous injuries.
3. Radiological examination includes standard anteroposterior and lateral plain x-ray films of the cervical, thoracic and lumbosacral spine if patients conditions allow. Remember that 10%-15% of patients have non-contiguous spinal column fractures.

e. Pharmacologic intervention:

1. Respiratory, cardiac, and hemodynamic monitoring is necessary for SCVI patients. Hypotension (systolic blood pressure < 90mm Hg) should be avoided an a mean arterial blood pressure of 85 to 90 mm Hg should be maintained for the first 7 days.
2. To avoid deep venous thrombosis and pulmonary embolism, prophylactic use of low-molecular- weight heparing, a rotating bed, and pneumatic compression stockings or combination therapy are recommended.
3. The neurologic injury resulting from the primary event is generally understood to be irreversible.
4. The secondary processes include alterations in microvascular perfusion, elaboration of free radicals, lipid peroxidation, necrosis and apoptosis of the cell, and ionic imbalance.

5. Current treatment protocols attempt to attenuate these secondary pathophysiologic events.
6. Many clinicians believe there is insufficient evidence to support any pharmacologic therapies as a stand of care in the management of acute spinal cord injury .Criticism has recently been directed at the interpretation and conclusions of NASCIS II and III studies.
7. Methylprednisolone was indicated to improve the motor scores in post-traumatic SCI when patients were delivered within 8 hours of injury (NASCIS III).
8. Less than 3 hours after injury, a 30 mg/Kg bolus of methylprednisolone is administrated, followed by 5,4 mg/Kg/h for 23 hours.
9. Between 3 to 8 hours after injury, the 30 mg/Kg bolus is followed by 5,4 mg/Kg/h for the next 47 hours.
10. Gangliosides have both neuroprotective and neuroregenerative effect in the laboratory. After three clinical trials found no statistical improvement in the neurological outcomes but did show the drug to be beneficial in terms of earlier recovery of motor function and improved sensory, bowel, and bladder function.

f. Timing of treatment:

1. There are no accepted clinical treatment strategies or algorithms that provide neurological regrowth or regeneration of the injured spinal cord.
2. Treatment algorithms for the timing and method of treatment for traumatic spinal cord injury have not been standardized.

g. Timing of surgery:

1. Data for the timing of surgical treatment of spinal cord injury has not been shown conclusively to support either early or late intervention.
2. Proponents of early surgical decompression advocate timely normalization of the intracellular environment and recovery of capillar perfusion by removing external pressure from the spinal cord and establishing spinal stability.
3. There is substantial class 2 and 3 evidence (nonprospective, nonrandomized and uncontrolled) that surgical decompression provides better outcomes tan late or nonsurgical therapies.

2. Cervical fractures

a. Epidemiology:

1. Cervical spine injuries account for about one-third of all spine injuries. C2 was the most common level of injury, one –third of which were odontoid fractures. In the subaxial spine , C6 and C7 were the most frequent
2. A neurological injury occurs in about 15% of spine trauma patients
3. Functionally, the cervical spine is divided into the upper cervical (occiput C0-C1-C2) and the lower (subaxial) cervical spine (C3-C7). The C0-C1-C2 complex is responsible for 50% of all cervical rotation while 80% of all flexion/extension occurs in the lower cervical spine. The C5-C6 level exhibits the largest ROM.
4. The position of the spine at impact determines the fracture patterns.

b. Instability of the cervical spine:

1. One of the problem has been the absence of a clear definition based in reliable radiological criteria. Therefore White and Panjabi defined clinical instability of the spine as: The loss of the ability of the spine under physiological loads to maintain its pattern of displacement so that there is no initial or additional neurological deficit, no major deformity and no incapacitating pain.

c. Initial treatment:

1. Early recognition of injury begins in the field. A collar is placed and a spine board is applied.
2. Neurological examination should include the assessment of cranial nerves , motor and sensory function , reflexes , and rectal tone.
3. The level of neurological function is graded according to the ASIA classification and reflected in clinical history.
4. Avoid the "chin lift" method of the securing airway, it may decrease space available for spinal cord.

d. Imaging studies:

1. Symptomatic patients require radiographic studies to rule out the presence of a traumatic cervical spine injury before the cervical spine is cleared. A cervical spine injury is found in 2-6% of all symptomatic patients.
2. Radiograph remain the imaging modality of first choice. The lateral view should extend from occiput to T1. Do not miss injuries at the cervicocranial and the cervicotorathic junctions.
3. Passive flexion/ extension views in unconscious or sedated patients must not be done.
4. CT is the first choice for unconscious patients. Most large trauma centers now perform multislice CT scans for the assessment of polytraumatized patients.The reason why CT has surpassed radiography include the ease of performance , speed of study, and most importantly, the greater ability of CT to detect fractures other than radiography.
5. MRI is additional to CT for specific diagnostic assessments.
6. Magnetic resonance is the imaging study of choice to exclude discoligamentous injuries.Is the modality of choice for evaluation of patients with neurological signs or symptoms to assess soft tissue injury of the cord , disc and ligaments.

e. Upper cervical Trauma :

1. Atlas Fractures (C1):

- 7% of cervical spine fractures
- Neurological injury is rare because of the wide spinal canal at that level, but cranial nerve injuries are frequently observed.
- Classic Jefferson (burst) fractures are bilateral fractures of the anterior and posterior arches of C1 resulting from axial load.
- Long term stability depends on the mechanism and healing of the transverse ligament.
- If the combined lateral mass of C1 are laterally displaced more than 6,9 mm relative to the C2 lateral mass (CT first choice for diagnosis) the transverse ligament may be disrupted, making it a potentially unstable injury.
- Treatment: all stable fractures without transvers ligament injury can be treated non surgically, with 6 to 12 weeks of external immobilization
- Jefferson fractures with an intact transverse ligament are

considered stable fractures and can also be treated with external immobilization with halo.
- On unstable Jefferson fractures surgical options may be considered.

2. Axis fractures (C2):
- Odontoid fracture are the most common type of axis fracture
- Type 1 fractures are avulsion fractures of the tip of the odontoid
- Type 2 fractures occur through the waist of the odontoid process.
- Type 3 fractures extend into the C2 vertebral body.
- Treatment of Type 1 and Type 3 , tipically stable fracture, should be treated with a cervical orthosis for 6 to 12 weeks.
- Treatment of type 2 fractures , correlates with increased risk of non-union if there are greater than 6 mm of initial translation, failed reduction , age greater than 50 and angulation greater than 10º.
- These type 2 fractures should be considered for early C1-C2 fusion in elederly patients.
- In young people type 2 fractures nondisplaced could be treated with halo vest immobilization. Fractures in wich reduction cannot be achieved or maintained surgical treatment should be considered. Anterior odontoid screw placement is an option for minimally conminuted fractures or C1-C2 posterior stabilization and fusion.

3. Traumatic Spondylolisthesis of the Axis:
- This injury is characterized by bilateral fractures of the pars interarticularis (hangman's fracture)
- Most patient can be treated successfully with external immobilization in a halo vest or cervical orthosis for a 6 to 12 weeks.
- Surgical indications include fractures with severe angulation or with disruption C2-C3 disk and/or facet dislocation. Surgical options include C2-C3 interbody fusion, posterior C1-C3 fusion or bilñateral C2 pars interarticvularis screws.

f. Subaxial Cervial Trauma:

Apply the Allen and Ferguson classification of subaxial cervical trauma (Allen et al. 1982) for fractures and dislocations of cervical spine C3 through C7. (Table 2)

The classification system is based upon the mechanism of injury; there are six categories divided into stages. It provides probable deficiencies of bony and ligamentous elements.

The three most commonly observed categories are compressive flexion, distractive flexion and compression extension.

>8° axial rotation C0–C1 to one side
>1mm translation of basis on to dens top (normal 4–5 mm) on flexion/ extension
>7mm bilateral overhang C1–C2
>45° axial rotation (C1–C2) to one side
>4mm C1–C2 translation measurement
<13mm posterior body C2 – posterior ring C1
avulsion fracture of transverse ligamentv

Table 2. Criteria for C0-C1-C2 instability (According to White and Panjabi [206)

1. Compression-flexion:
- failure of anterior column compression and posterior column distraccion.
- are caused by axial loading in flexion with failure oif the anterior half of the body without disruption of the posterior body cortex and minimal risk of neurologic injury.
- there are five stages (Allen et al 1982): I- blunting of the anterior-superior vertebral body; II- Progression to vertebral body beaking; III- Beak fracture; IV- Cephaled vertebral retrolisthesis < 3 mm; V- Retrolisthesis > 3mm
- Treatment: stable undisplaced compression-flexion fractures can be treated conservatively with external inmobilization for 6 to 12 weeks with a rigid collar. Kyphosis deformity > 15º shloud be considered for operative stabilization with anterior cervical fusion.

2. Vertical compression injury:
- are caused by sever compressive load. These fractures, "burst fractures", are commonly associated with complete or incomplete SCI from retropulsion of fracture fragments into the spinal canal.
- Treatment: patients with neurological deficit are better treated by anterior decompression and reconstruction with strut grafts and platting. If there are a significant compromise of the spinal canal, can usually be reduced with traction.

3. Distraction- flexion:
- four stages (Allen et al 1982):
- stage 1- facet subluxation in flexion and widening of the interspinous distance. < 25% subluxation of facets
- stage II- unilateral facet dislocations
- stage III- bilateral facet dislocation with < 50% anterior vertebral body translation .
- stage IV- bilateral facet dislocation with 100% anterior translation of the vertebral body.
- Treatment: Rotational injuries are considered very unstable and are therefore usually treated operatively. Aware and alert patient can safely undergo closed reduction with progressive traction. Development of new or worsening neurologic deficits is an indication to cease closed reduction.
- Patients who have undergone successful awake reduction should undergo an MRI to verify that no disc material or hematoma reminds.
- A combined antero/posterior technique provides the best outcome although in selected cases (e.g. unilateral dislocation) either a single anterior or posterior approach may suffice.

g. Complications:
- Overall, 5% of patients with compressive injuries of the subaxial cervical spine had persistent instability after non-operative treatment.
- Kyphosis or subluxation develops in about 10% of patients who are treated with postertior fusion.
- Operative complications are more common in patients treated with posterior fusion procedures (37%) compared with anterior fusion procedures (9%).
- Graft displacements is the most common complication found in patients treated with anterior cervical fusion without anterior fixation (9%).

3. Thoracolumbar fractures

a. Epidemiology

1. The thoracolumbar spine is the most common site of spinal injuries.
2. Usually are the resukt of a significant- force impact, such as a motor vehicle accident or fall.
3. Most injuries (52%) occurs between T11 and L1 followed by L1 throgh L5 (32%) and T1 through T10 (16%).
4. The increased incidence of fractures of the thoracolumbar junction is the result of its location at the biomechanical transition zone between the rigid thoracic rib cage and the more flexible lumbar spine.
5. Contiguous and non-contiguous spine injuries are present in 6% to 15% of patients.
6. Associated injuries include intra-abdominal bleeding from liver and splenic injuries, vessel disruption, and pulmonary injuries (20% of patients).
7. In thoracolumbar fractures neurological deficiencies was reported between 22% and 35% . In the thoracolumbar transition (T10-L2) neurological deficiencies occur in 22-51% depending of the fracture type.

b. Radiologic Evaluation:

- Plain x-ray film is the initial screening modality with computed tomography (CT) scanning or magnetic resonance imaging (MRI) used as an adjunct depending upon whether the surgeon needs to further evaluate bony or soft tissue anatomy.

c. Classification Methods:

1. Denis (1983) chose to divide the anterior column into two making three columns in total, the middle of which was felt to be the biomechanical key, that is , disruption here was thought to render the fracture inestable.Major injuries include compression fractures, burst fractures, flexio-distraction injuries and fractures dislocations.
2. Magerl et al (1993) introduced a complex hierarchical classification system based on pathomorphologic criteria, of increasing injury and instability.This system divides thoracolumbar spinal fractures into three general groups. Type A compression injuries, Type B distraction injuries and Type C torsional injuries. The complexity os the system certainly enables researches to accurately compare fracture types in follow-up.
3. Vaccaro et al (2005) have proposed recently, a novel new Thoracolumbar Injury Classification and Severity Score (TLISS) based in three parameters: the morphology of the fractured vertebrae, the neurologic status and the integrity of the importance PLC now visible on MR.

d. Non operative treatment:

1. Most thoracolumbar spine fractures are stable and do not require surgery.
2. Non operative treatment with a well-molded brace or hyperextension cast has been shown in numerous studies to be very effective.
3. Simple compression or stable burst fracture without neuroligic complications can typically be treated with off-the-shelf braces or well-molded orthoses that permit early ambulation.
4. Upright radiographs of the patient, in brace or in caqst, should be obtained before discharge.
5. Significant increases in the fracture angle (>10°) or significant increases in pain have been suggested as an indication for consideration of operative treatment.

e. Operative treatment:

1. Operative treatment does offer a few advantages: immediate mobilization, earlier rehabilitation and may restore sagittal alignment more reliably in certain situations.
2. The benefits of surgical treatment must be carefully weighed against the potential morbidity associated with the operation.
3. Compression fractures: Coronal split type fractures frequently fail to unite and may be a source of painful non-union. Operative treatment is, more commonly considered, especially in the lower lumbar spine.
4. Burst fractures: instability should be considered whenever large degrees of axial compression (>50%) or more than 25° of angulation.

- The decision of surgery depends on the location of the fractures, the degree of vertebral destruction, any neurologic involvement, the degree of kyphosis, and the stability of the posterior column structures.
- It has been demonstrated in numerous reports that retropulsed bony fragments do resorb and the canal remodels up to 50% of the occlusion over time.
- Posterior pedicle screw fixation has been shown to be efficient reliable and safe for the reduction and stabilization of most traumatic fractures.
- The proportion of vertebral body damage, spread of the fracture fragments, and degree of kyphosis are tabulated to predict failure, that is, suggesting the need for additional anterior column support/surgery. In this situation anterior reconstruction with structural graft or plate instrumentation and short-segment posterior pedicle screw fixation has been shown to be effective.

5. Flexion-distraction injuries:

- Because the injury in these fractures is principally to the posterior osteoligamentous complex, it is best treated with a posterior compression type construct and fusion to restore the normal sagittal contour.
- Care must be taken however, not to overly compress or lordose the fracture site
- Most specialist advise postural reduction by positioning, gentle compression and lordosing rod.

6. Fractures-dislocation:

- Fracture-dislocation are often the results of very high-energy trauma and are the fracture type most often associated with neurological damage and associated skeletal injuries.
- Both bony columns and associated ligamentous structures are disrupted through combination of shear rotation and flexion-extension.
- Because of the severe nature of the bony disruption , realignment and fixation are best accomplished through posterior positioning, reduction, multilevel instrumentation, and fusion.

f. Complications:

The reported complications rate in the literature varies largely and ranges from 3,6% to 10%. Postoperative neurological complications range from 0,1% to 0,7%. Only honest and accurate assessment of complications will lead to scientific and clinical progress

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Enric Cáceres-Paluo

Autonomous University of Barcelona,
Barcelona, Spain

Email: ecaceres@vhebron.net

Degenerative Lumbar Spine

Prevalence of Lumbar Degenerative Disease

1. 70-80% of all individual will experience low back pain at some time in their lives; usually it resolves in some weeks

2. Low pain is the leading cause of disability in people younger than 50 years of age

3. Many sources of pain are described

3.1 facet joint arthropaty

3.2 discogenic pain or annular tears

3.3 spondilolisthesis

3.4 spinal stenosis

I Lumbar Disc Herniation

1. General overview

A. Incidence 80% of people has some episode of low back pain in your life, but only 2-3% has true sciatica

B. Age Average starting age 35 years old. Unusual before 20 and after 60 years. Less frequent in old people , more common associated with stenosis

C. Sex. Similar in both sex but delayed one decade in females

2. Anatomy

A. L4-L5 more frequent than L5-S1 less frequent in thoracic&high lumbar level

B. Frequently posterolateral

C. Central location will cause lumbar pain without sciatica

D. Foramina location more frequent in elderly people; high levels (L3-L4) E Intradural location very unusual

3. Clínical features Radiculopathy

A. Leg pain > lumbar pain

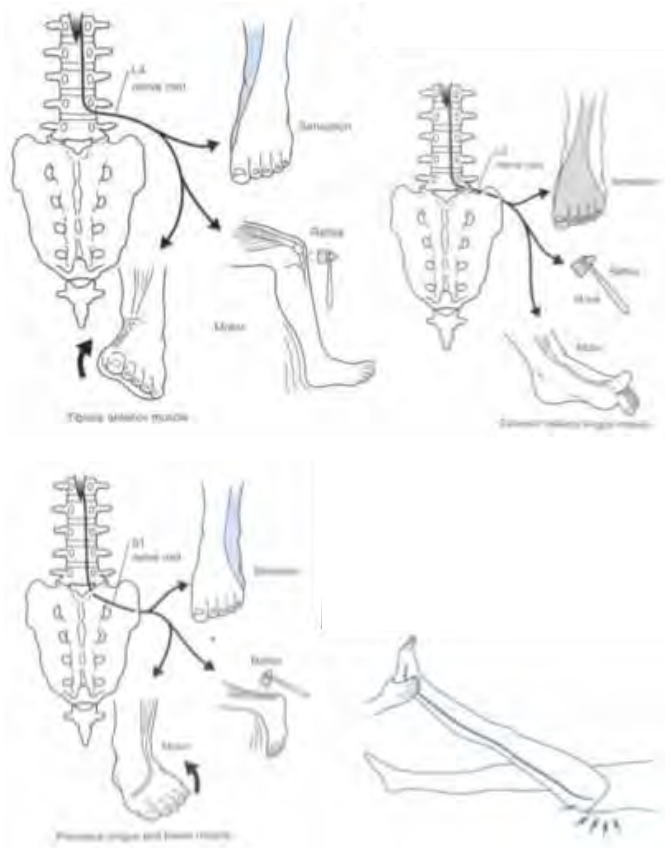
B. Dermatomal distribution

C. Increase sitting positions and forward bending

D. Improve with bed rest

4. Physical exam

4. Diagnostic imaging



4A. RNM

a. modality of choice for LHD
b. T2-weighted images most commonly used
c. T1-weighted + Gadolinium can differentiate between scar tissue and herniated disc material

4B. Discography + TAC useful tool in recurrence

5.Treatment

5.1 Nonsurgical treatment:
LDH has a favourable prognosis
90% report improvement of symptoms (natural history)

4. Short rest (3-5 days)
5. NSAIDs (more effective than placebo)
6. Physical therapy (extremely beneficial)
7. Epidural steroid injections (50% avoided surgery)

5.2 Surgical treatment: Conventional discectomy
Patient who failed to improve with nonsurgical treatment will probably need surgery.
Surgical treatment provided an increase in quality of life in comparison to continued nonsurgical treatment.

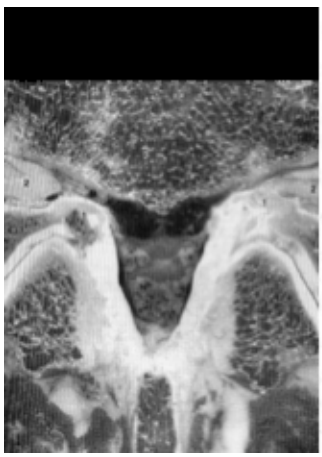
II Lumbar spinal stenosis

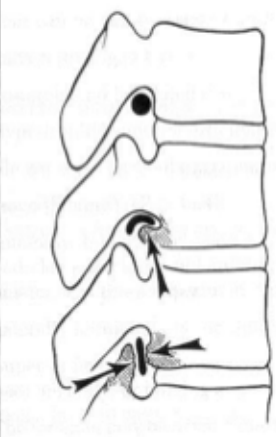
A. Definition.

- Spinal stenosis simply means a decrease in the space available for the neural elements, and, in the lumbar spine, the cauda equina. It can occur at different level: the central canal, the lateral recess or the intervertebral foramen causing neurological compression

B. Physiopathology
Is the final result of a cascade of events.

- The event that begins the process is thought to be the disk degeneration.
- As the disk height decreases, the loading characteristics of the facet joints are altered.
- Facet joints capsules become incompetent, leading to capsular, ligamentous flavum, and facet hypertrophy.
- The final result of this continuum of changes is a decrease in the diameter of the spinal canal.





C. Diagnostic imaging:

C1. Upright AP, lateral, and flexion–extension radiographs amount of lumbar degeneration , vertebral deformity and instability.

C2. EMG may be helpful to distinguish peripheral neuropathy from LSS. C3. Myelography: useful when deformity exists.

C4. CT scan: facet joints hypertrophy, disc vacuum, size of disc height and foramen height.

C5. MRI is currently the recommended advanced imaging modality to evaluate LSS. Non invasive technique.

D. Treatment

D1. Nonsurgical

1. Narcotics, NAIDs, anticonvulsants
2. LS ortosis
3. Physical therapy: flexion-based lumbar stabilization program
4. Steroid injections

D2. Surgical treatment

1. Indications:
   Caudal equina syndrome
   Severe neurologic deficit or impairment
   Failure to improve leg pain and neurogenic claudication after nonsurgical treatment. Persistent and severe decrease in patient quality of life.
2. Natural history
   Not well understood
   It is typically favourable with only 15% deteriorating clinically. Improvement occurs in 30% to 50% of patients.
3. Operative technique

- Preoperative medical evaluation
- Elderly patients
- Coexisting comorbidities
- Save blood preoperative techniques
- Self-saver postoperative
- Eritropoyetina
- Laminectomy
- Hemi laminectomy
- Resection of 50% of the cephalic and caudal lamina and ligamentous flavum
- Lateral decompression into the lateral recess and into the foramen
- Fusion if resection is > 50 % bilateral facets or complete unilateral facetectomy

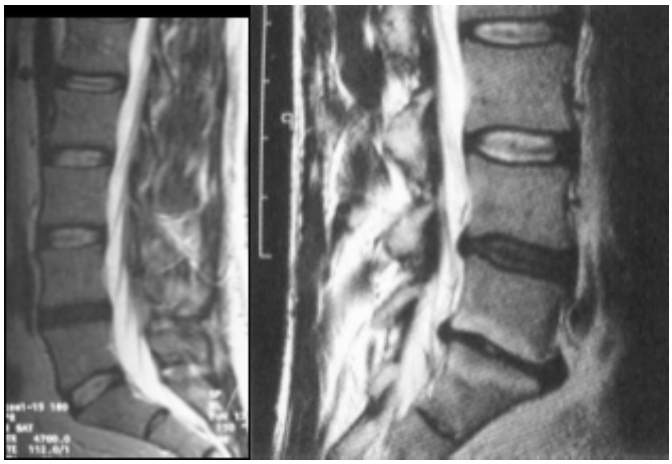
III Dark disc disease DDD

1. 1MRI image corresponding with a degenerative disk disease degree IV

2. Participates in the degenerative and physiologic cascade starting from 30 years old

3. When appear in a precocious time in young people could be symptomatic

4. Structural and biochemistry changes put up with alteration of biomechanics properties

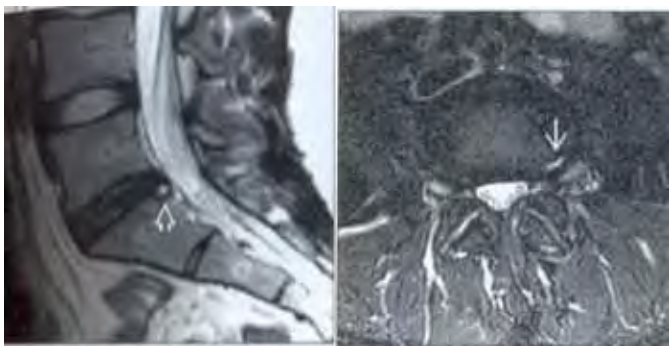


A. L4 –L5 selective DDD *B. Modic Changes in L5-S1*

5. Modic changes in MRI describes different situations around DDD

Modic type	T1 signal	T2 signal
Modic I	EDEMA decrease	Increase
ModicII	FAT DEGENERATION increase	Increase
ModicIII	ESCLEROSIS decrease	Decrease

6. HIZ changes: High Intensity Zone in T2 localized annulus posterior or posterolateral layer



Images of annular tears corresponding with HIZ in L5-S1

6.1 Disk tear lesion predictor

6.2 Contends vascularizated granulamotous tissue

6.3 Highlight with Gadolinium

6.4 Strong relationship whit positive discography...not always with clinical significance

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Prof. Dr. Klaus Arno Siebenrock (pic.), Dr. Timo Michael Ecker

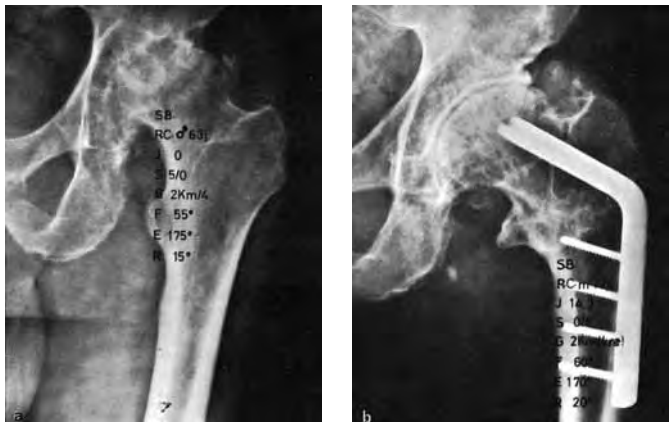
Inselspital, University of Berne
Berne, Switzerland
klaus.siebenrock@insel.ch, timo.ecker@insel.ch

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].



a b

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.

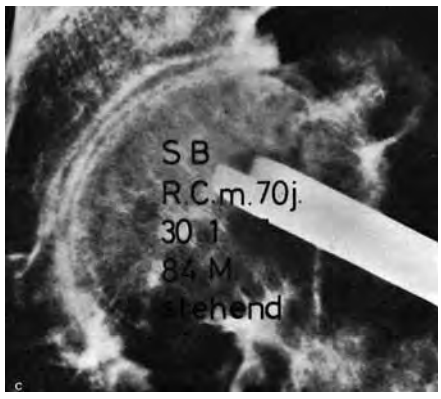


Fig. 1c The arthrogram demonstrates cartilage remodeling on the acetabular, as well as the femoral side at 7 years postoperatively. Image reprinted with permission from [2].

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 = Nonarthrtitic 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 antetorta 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]

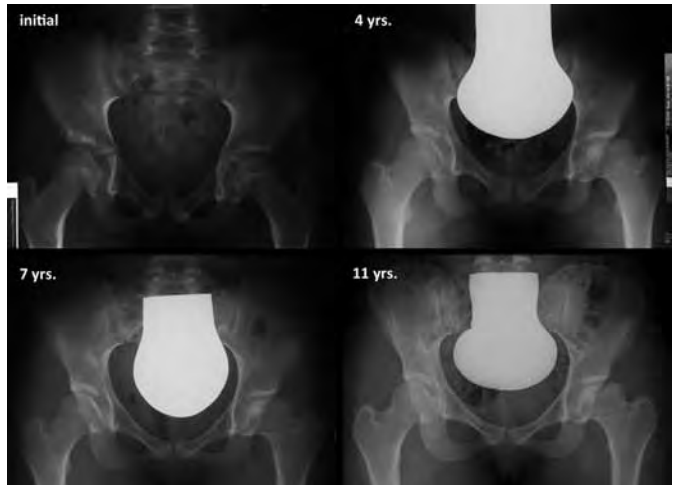


Fig. 2a A consecutive series of anteroposterior pelvis radiographs acquired over an 11-year period are shown. On initial presentation (upper left), this 10 year-old female patient presented with clinical symptoms and radiographic evidence of Perthes disease and concomitant marginal hip dysplasia on the 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.

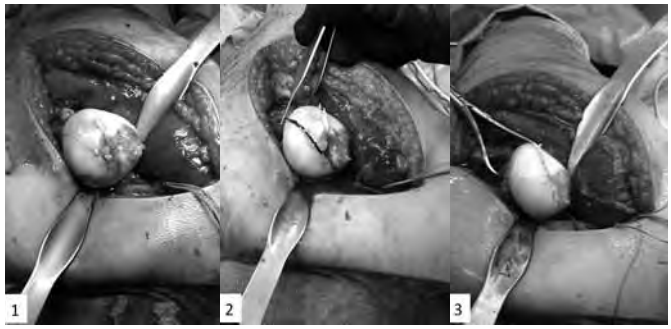


Fig. 2b Intraoperative images of the same patient introduced in figure 2a) are shown. At 22 years of age, a surgical hip dislocation with an extended retinacular soft-tissue flap was performed, allowing for inspection and remodeling of the deformed femoral head. In this case, the subsequent steps of 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.

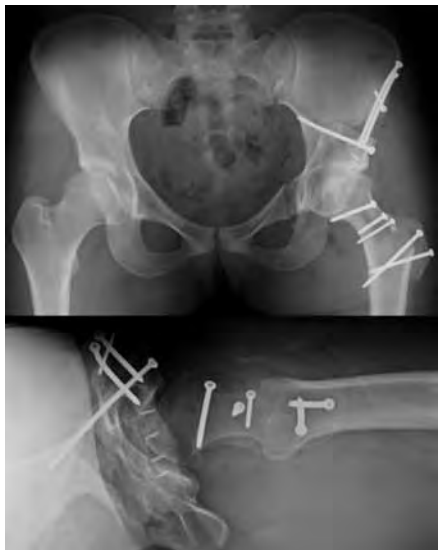
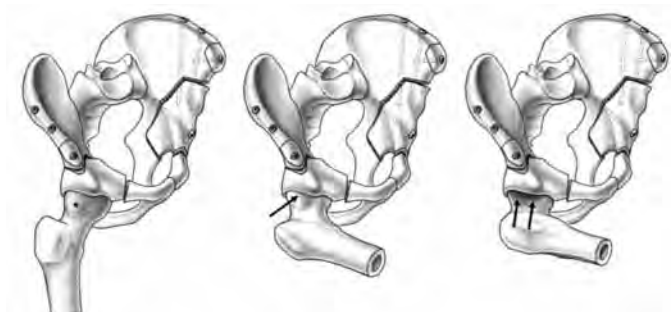


Fig. 2c The postoperative radiographs are shown. Apart from the head reduction-plasty, the patient received a periacetabular osteotomy of the left hip joint. The combined femoral and acetabular interventions significantly increased the containment of the femoral head and the congruity of the articulation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



a b c

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 actabular 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.

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Dr. Daniel Petek

District Hospital,
Fribourg, Switzerland
daniel.petek@h-fr.ch

Knee: Osteotomy and arthroplasty

Introduction

Symptomatic knee osteoarthritis (OA) is highly prevalent among people aged 50 years and over. 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 relived, 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 location of the deformity. 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 (Outerbrige) 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 ± 1° with the anatomical axis (Fig. 1 a-c). The tibia has a mechanical axis nearly identical to the anatomical axis.

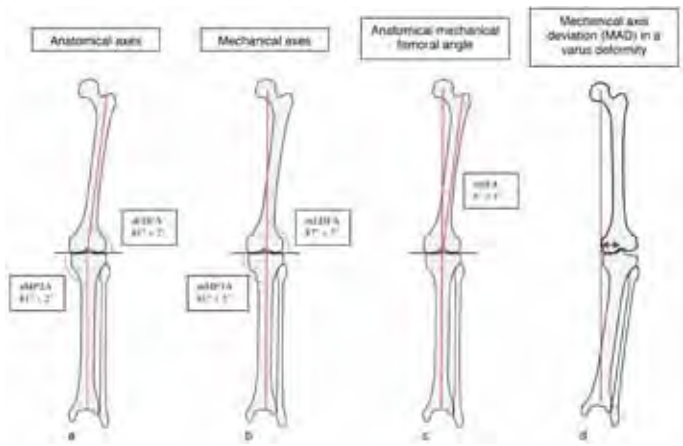

Fig. 1(a-c) Anatomical and mechanical angle values of the femur and the tibia. d Measurement of the MAD (mm) from the knee centre in a varus deviation. aMPTA: anatomical mechanical proximal tibial angle. aLDFA: anatomical lateral distal femoral angle. mMPTA: mechanical medial proximal tibial angle. mLDFA: 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. It is diagnosed as a varus when the weight bearing axis of the lower limb 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.

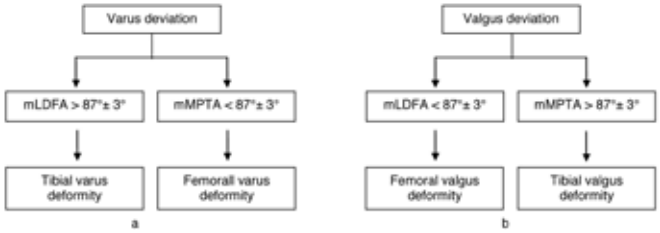

Fig. 2 (a-b) Femoral and tibial analysis of varus and valgus deformity

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).

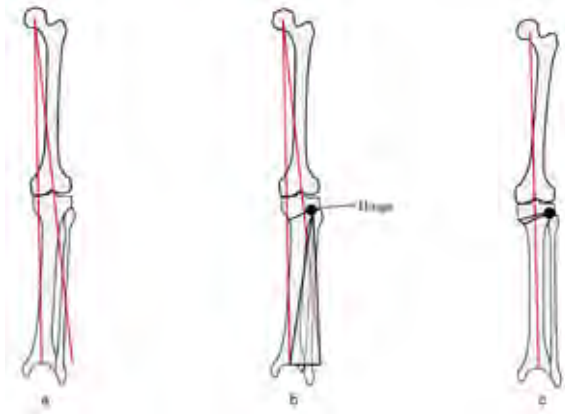

Fig.3 (a-c) Determination of the correction angle in high tibial osteotomy opening wedge (Miniaci),

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.

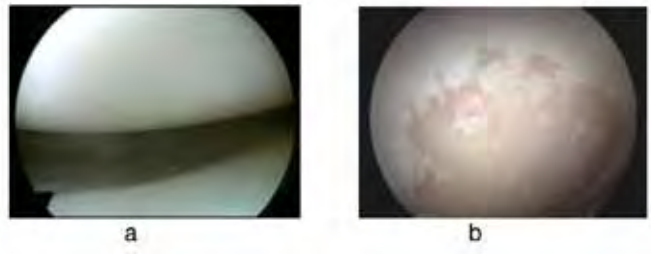

Fig. 4 Arthroscopic views of a right knee.
a. Lateral compartment.
b. Medial compartment with cartilage wear

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.

As the superficial portion of the medial collateral ligament inserts on the postero medial portion of the proximal tibia, 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).

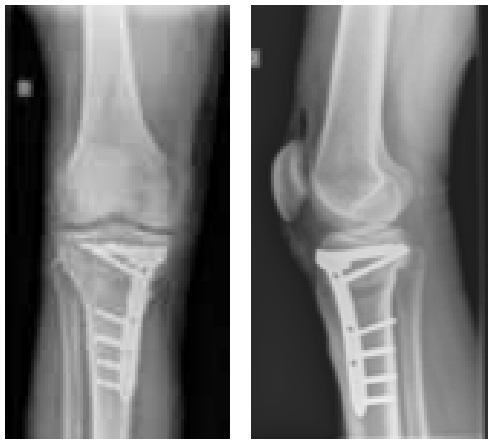

Fig. 5 Post-operative views of right knee after high-tibial opening-wedge valgisation osteotomy

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).

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.

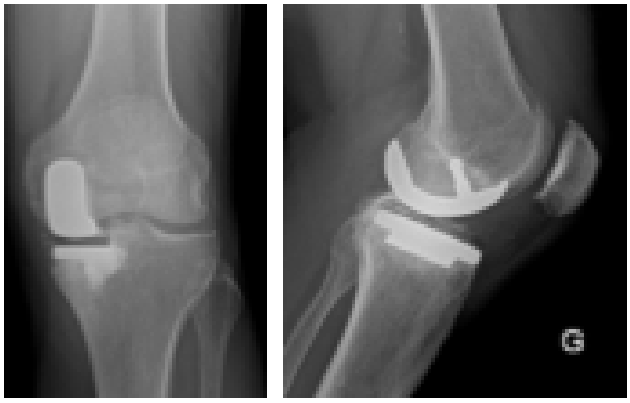

Fig. 6 Medial UKA

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 (Table 1, Fig. 7).

- 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)

Table 1 Preoperative X- Rays

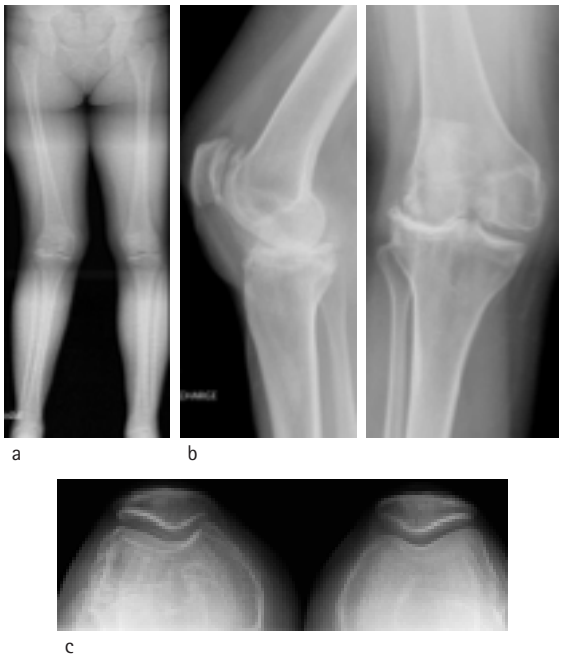

Fig. 7 (a-c)
a Standing antero posterior view
b Frontal and lateral knee views
c Patellofemoral view

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 starched 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.

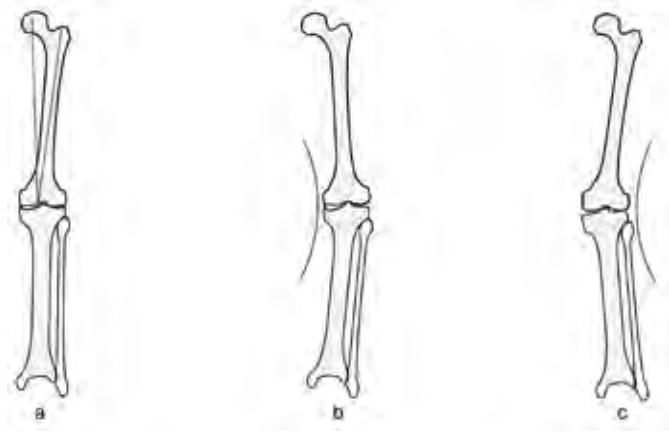

Fig. 8 (a-c)
a Measure of the femoral valgus cut angle
b Medial contracted structures in varus deformity
c Lateral contracted structures in valgus deformity

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

Table 2 Varus Deformity

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

Table 3 Valgus Deformity

3.4 Bone Resection

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).

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

Table 4 Sagittal Plane Balancing

3.5 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).

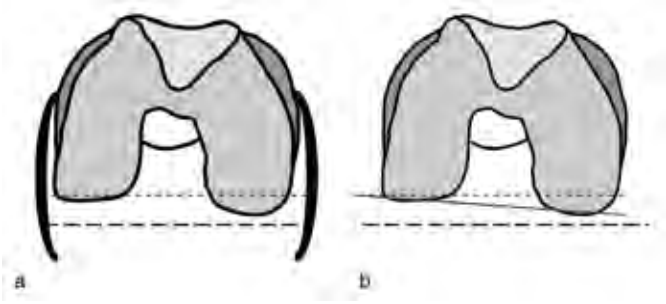

Fig. 9 (a-b) Optimal rotational positioning of femoral implant

— — — 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. Also, a sagittal lateral patellectomy (<10mm) may be performed to avoid a lateral patella femoral conflict.

3.6 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. Non-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 over the years. Still, for a successful outcome of a TKA, a well-balanced implant with a good patellofemoral tracking is essential (Fig. 10).

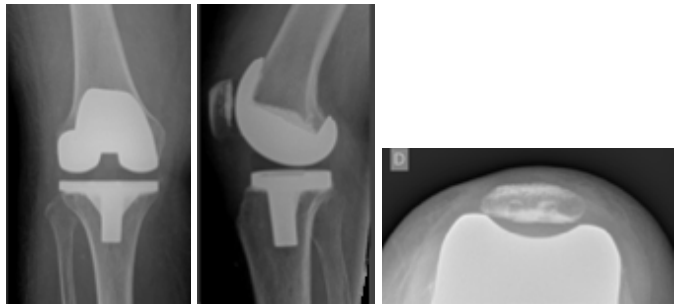

Fig.10: Total knee arthroplasty

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. Unicondylar 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 definitive treatment of knee osteoarthritis. Table 5 lists the ideal patient for each type of surgery.

OSTEOTOMIES	UKA	TKA
55 to 70 years (biological age)	Older than 55 years	Older than 70 years
May have extraarticular deformity	Must have correctable deformity	May have fixed axis deviation
Monocompartmental osteoarthritis	Monocompartmental osteoarthritis	Generalised osteoarthritis
May have extension deficit	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	Must have intact ACL/ PCL	May have ACL / PCL deficiency

Table 5 Indicatzions

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Prof. Dr. Victor Valderrabano

University Hospital of Basel
Basel, Switzerland
vvalderrabano@uhbs.ch

Ankle Arthrodesis-Arthroplasty, Hallux Valgus & Flat Foot

Preface: Reconstructive Foot and Ankle Surgery

Authors:
Dr. Alexej Barg
Prof. Dr. Victor Valderrabano

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 al[10] 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.

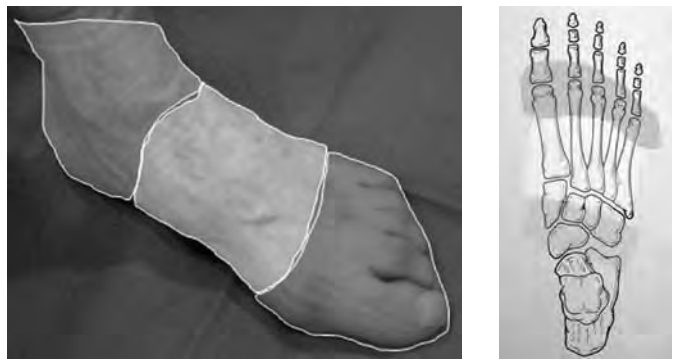

Figure. 1 Classification of foot and ankle disorders depending on its localization: hindfoot midfoot, and forefoot.

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Ankle Osteoarthritis: Ankle Arthrodesis and Total Ankle Arthroplasty

Authors:
Dr. Alexej Barg
Dr. Geert Pagenstert
Prof. Dr. Dr. Victor Valderrabano

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 ligamental 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 ligamental 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.)

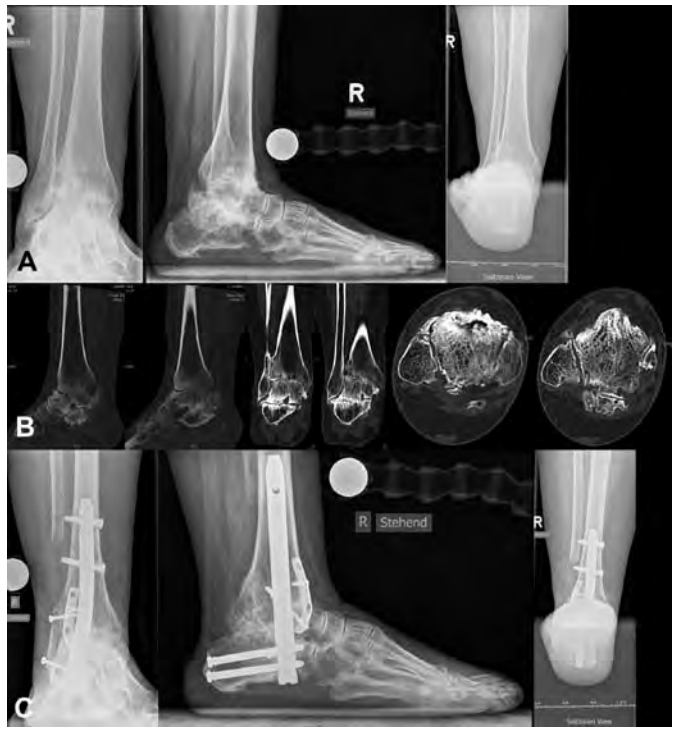

Figure 1 Tibiotalocalcaneal arthrodesis. A: 59-year old female with severe end-stage osteoarthritis of the tibiotalar and subtalar joints. Saltzman view shows substantial valgus deformity of the hindfoot. B: CT-scans confirm severe osteoarthritis with substantial destruction of the tibiotalar and subtalar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

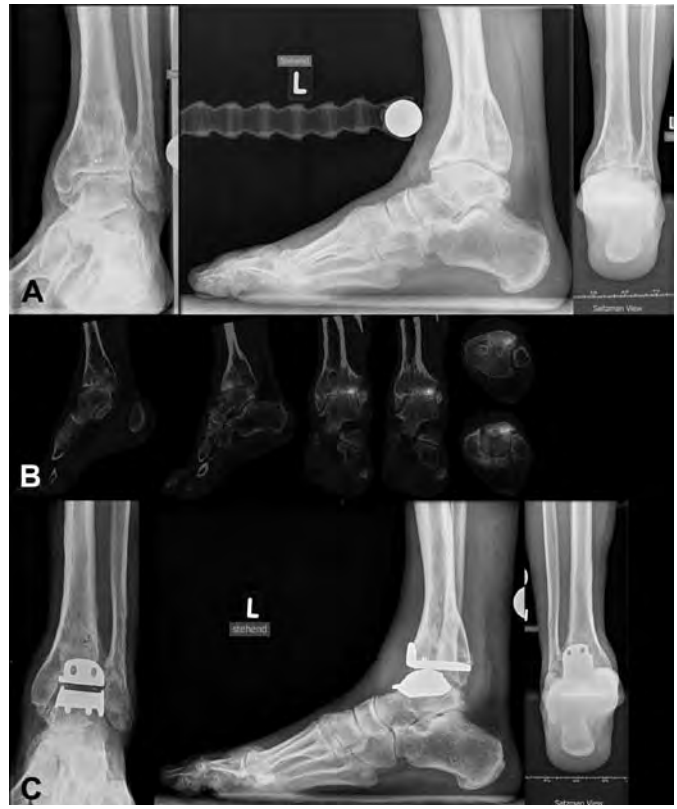

Figure 2 Total ankle arthroplasty. A: 73-year old male with symmetric osteoarthritis of the tibiotalar joint. Saltzman view shows no concomitant deformity of the hindfoot. B: SPECT-CT shows exact localization and biologic activity of osteoarthritic changes. C: Total ankle arthroplasty has been performed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

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Adult Flat Foot

Authors:
Dr. Jochen Paul
Dr. Sebastian A. Müller
Dr. Alexej Barg
Prof. Dr. Dr. Victor Valderrabano

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 of 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 iontopheresis, 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 iontopheresis 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]

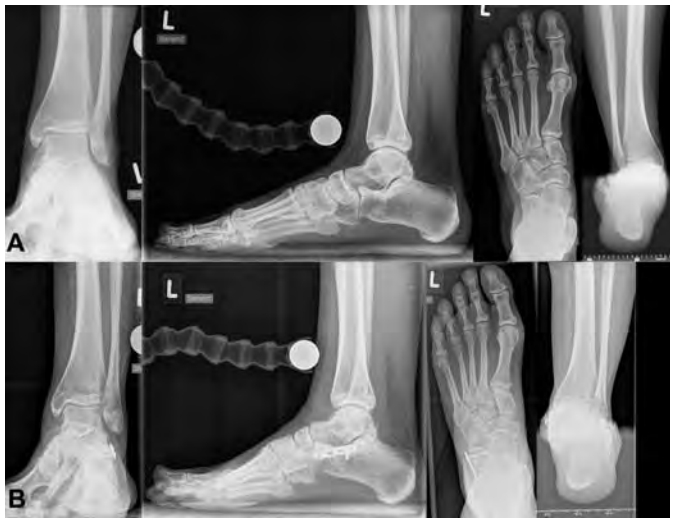

Figure 1 Lateral column lengthening osteotomy of the calcaneus. A: 42-year old female with substantial abductus deformity of the midfoot and valgus malalignment of the hindfoot. B: Lateral column lengthening osteotomy of the calcaneus with use of a spongy allograft block and a FDL on PTT transfer were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

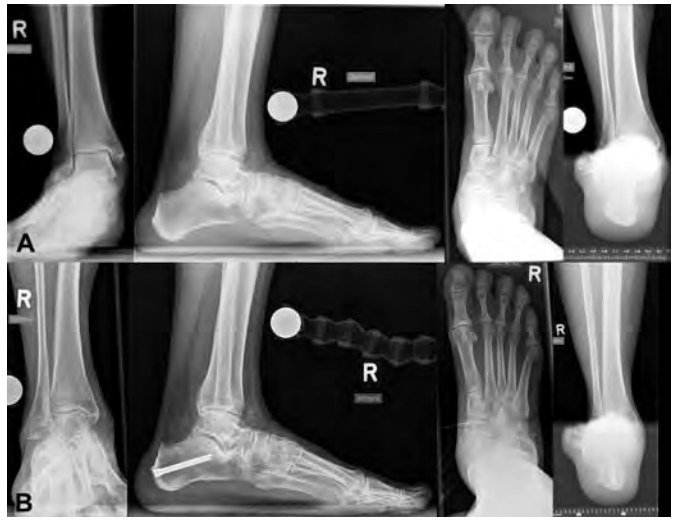

Figure 2 Medial sliding osteotomy of the calcaneus. A: 59-year old male with severe valgus malalignment of the hindfoot and substantial flattening of the medial arch. B: Medial sliding osteotomy of the calcaneus was performed resulting in physiological alignment of the hindfoot.

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]

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Hallux Valgus

Authors:
Dr. Monika Horisberger
Dr. Alexej Barg
Prof. Dr. Dr. Victor Valderrabano

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

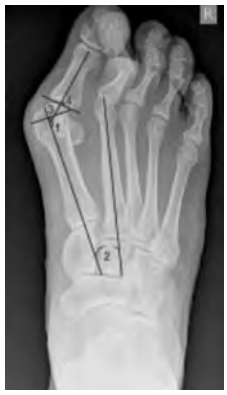
Development of hallux valgus is a multifactorial development and details are still unknown and a matter of debate [3]. Several etiologies for hallux valgus deformity have been suggested. These can basically be divided into primary and secondary causes. Primary causes include hallux valgus deformity due 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 abnormities 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



Figgure 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 oligosymtomatic 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 angle1
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

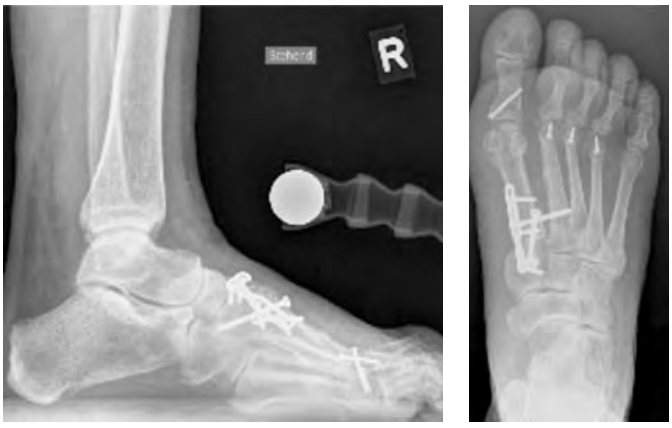


Figure 2 Lapidus arthrodesis. 56-year female with incongruent hallux valgus deformity, TMT-I instability and metatarsalgia MTP-II/IV

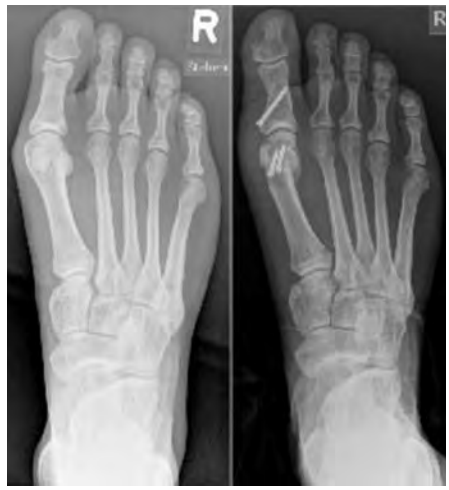


Figure 3 Chevron and Akin osteotomy. 31-year old female with moderate hallux valgus deformity.

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Mr Surya Gandham, MRCS, Orthopaedic Registrar
Mr Anthony McGrath MRCS, Orthopaedic Registrar
Mr Philip M Ahrens, FRCS (Tr & Orth), Orthopaedic Consultant(pic)

The Royal Free Hampstead NHS Foundation Trust, London, UK
philip.ahrens@nhs.net

Shoulder Degenerative Disorders

I. Introduction

1. With an increasing ageing population, incidence of both gleno-humeral joint (GHJ) osteoarthritis (OA) and rotator cuff disease is on the rise

2. Shoulder problems represent 2.4% of all Primary Care consultations in the UK1

3. Arthritis of the shoulder can be caused by

3.1 OA

3.2 Trauma

3.3 Avascular necrosis

3.4 Infection

3.5 Cuff tear arthropathy

4. OA is the most common cause of shoulder arthropathy and has been linked to age and chronic overuse

5. The rate of shoulder arthroplasty is rapidly increasing and will lead to increased costs and societal burdens2

6. Rotator cuff disease encompasses a spectrum of age related degeneration which include:

7.1 Rotator cuff tendinopathy

7.2 Partial thickness tears

7.3 Full thickness tears

7. Yamamoto et al demonstrated that the incidence of rotator cuff disease in a Japanese village was 20.7% for its general population3. Risk factors include:

8.1 Trauma

8.2 Arm Dominance

8.3 Age (Moosmayer et al and Sher et al have shown that tears are associated with increased age in asymptomatic shoulders4,

8. Enlargement of tears with time leads to muscle tendon unit retraction which in turn leads to:

10.1 Changes in muscle architecture

10.2 Joint mal-alignment

10.3 Altered biomechanics

10.4 Secondary Arthritis

9. In this review article we will discuss these two major degenerative disorders including aetiology and pathology, diagnosis and treatment

II. Rotator Cuff Disease

IIA. Aetiology & Pathology

1. Anatomical considerations

1.1 Acromion Shape : Three types of acromion shape5

a. Type 1 Flat
b. Type 2 Curved
c. Type 3 Hooked

1.1.1 The Type 2 acromion is the most prevalent in the general population and type 3 associated with rotator cuff disease

1.2 Tendon Insertion: Accurate understanding of rotator cuff anatomy will help in surgical reconstruction.

1.2.1 Mochizuki et al found in his cadaveric study that the infraspinatus (ISP) tendon footprint was found to cover a large area of the postero-lateral greater tuberosity whereas the supraspinatus (SSP) tendon attached to only a discrete anterior portion of the greater tuberosity which is less than previously described6

1.3 Vasculature and Rotator Cuff Disease: The anterior and posterior humeral circumflex arteries with assistance from the suprascapular, thoracoacromial and subscapular arteries perfuse the rotator cuff

1.3.1 Several studies have shown diminished blood flow at the level of the distal insertion of the SSP. a hypovascular pattern is found in intratendinous

tissue compared with the subacromial bursa, an age-related decrease in intratendinous vascularity, and a hypovascular pattern in the tendon, regardless of rupture of the tendon7

1.3.2 Laser Doppler flowmetry analysis has shown a difference between the vascularity of the normal and the pathological rotator cuff. Current research is unable to demonstrate a functional hypoperfusion area or so-called 'critical zone' in the normal cuff. The measured flux decreases with advancing impingement, but there is a substantial increase at the edges of rotator cuff tears

1.4 The Role of the Subacromial Bursa in Rotator Cuff Disease

1.4.1 The medial aspect of the bursa provides blood supply to the rotator cuff tendons and may accelerate tendon healing

1.4.2 The lateral bursa is a pain generator in subacromial pathology

1.5 Bone Quality:

1.5.1 Patient age and greater tuberosity bone density are inversely proportional

1.5.2 Use of the medial footprint on the tuberosity, near the articular cartilage for anchor placement is warranted

1.6 Suprascapular nerve and Rotator Cuff Disease

1.6.1 The suprascapular nerve may be compressed at the scapular notch by a thickened transverse ligament or a retracted cuff tear

1.6.2 A paralabral cyst can cause compression in the spinoglenoid notch

2. Biomechanical Considerations

2.1 The shoulder is a prime example of a closed chain system organised to provide bio-mechanical stability and mobility to the GHJ. It regulates the transfer of energy from the trunk and allows the hand to be placed into space

2.2 The rotator cuff provides a significant amount of GHJ stability. Therefore, defects can cause problems with activation, compression, tension, or motion

2.3 The scapula plays an important role in GHJ function as it provides a stable base for muscle activation and load transfer within the closed kinetic chain. Any abnormality in scapula position or movement will affect the functionality and stability of the rotator cuff, biceps and superior labrum8

3. Histological and Molecular changes

3.1 Histological changes rotator cuff disease (RCD) include:

3.1.1 A reduction in overall collagen content with an increase in type II and type III collagen

3.1.2 Tissue in RCD is more fibrocartilagenous in phenotype than healthy tendon

3.1.3 Collagenase matrix metalloproteinase are

3.1.4 increased in RCD showing a catabolic trend

3.2 Molecular changes in RCD include9:

3.2.1 Cytokine change are generally pro-inflammatory

3.2.2 Increase in Tumour Necrosis Factor, cyclo-oxygenases, nitric oxide, several growth factors and p53. These maybe important in promoting cell apoptosis and decrease cuff regeneration

4. Diagnosis

4.1 A thorough history documenting mechanism of injury, pain, clinical dysfunction and patient demands contribute to an accurate diagnosis

4.2 Impingement signs and / or scapular dyskinesis signs are very sensitive but not specific.

4.3 Lag signs such as drop-arm test, external rotational lag sign, and internal rotational lag sign are quite specific but not sensitive

4.4 The addition of a subacromial injection of lidocaine to assess cuff integrity increases the sensitivity of impingement signs and specificity of cuff specific tests to >90%

4.5 With regards to imaging, MR arthrography is the most sensitive and specific technique for diagnosing both full- and partial-thickness rotator cuff tears. Ultrasound and plain MRI are comparable in both sensitivity and specificity10 Overall U/S is most accurate for rotator cuff pathology and MRI for intra-articular pathology.

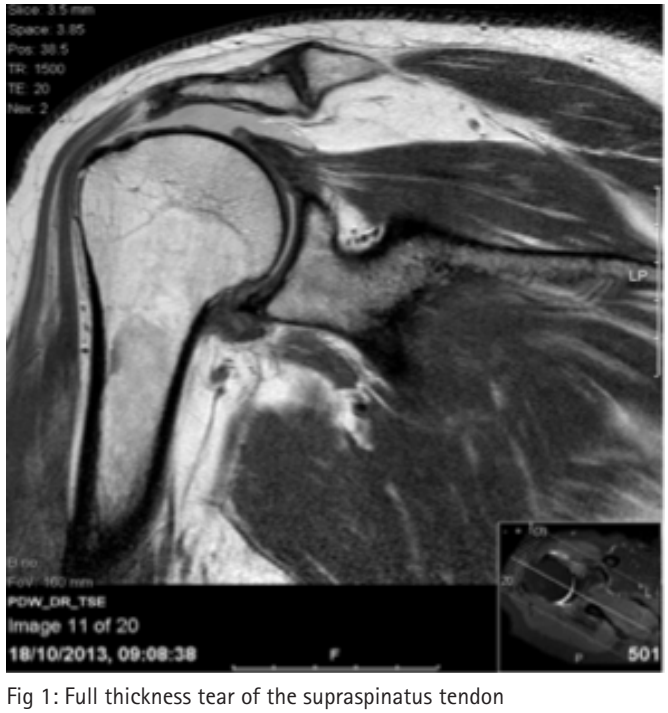


Fig 1: Full thickness tear of the supraspinatus tendon

5. Treatment

5.1 Impingement Syndrome

5.1.1 Impingement syndrome covers the spectrum of rotator cuff disease from bursitis, tendinitis, tendinopathy and tendon tears and describes antero-lateral shoulder pain with a painful arc of motions – classically 600-1200 of abduction.

5.2 Tendinopathy

5.2.1 Approximately 90% of simple impingement syndrome can be successfully treated with rest, activity modification, NSAID's and physiotherapy.

5.2.2 Subacromial steroid injections are also helpful for diagnostic confirmation, analgesia and symptom control.

5.2.3 Arthroscopic subacromial decompression (acromioplasty) is indicated in resistant or recurrent impingement, and is a common procedure with high rates of patient satisfaction, though there is limited quality evidence supporting its use.

5.3 Partial Rotator Cuff Tears

5.3.1 Conservative treatment: Conservative management (physiotherapy and activity modification) is successful in most patients but this should be based on the level of activity, tear thickness and symptoms.

5.3.2 Surgical management: The decision to operate is normally based on the failure of conservative management. Oh et al has highlighted the need for further research and the difficulty in designing such a study. Surgical options include:

a. Arthroscopic debridement +/- arthroscopic subacromial decompression
b. Arthroscopic conversion to a full thickness tear with repair (single/double-row)
c. Transosseous / transtendon repairs

6. Full-thickness Tears

6.1 Conservative

6.1.1 Asymptomatic Tears: 4% of asymptomatic patients aged < 40 years and 54% of patients aged ≥ 60 years, have partial or complete tears of the rotator cuff on a MRI scan. Conservative management with physiotherapy, if needed, is normally utilised. Studies have shown that asymptomatic tears can progress into symptomatic tears after 3 years

6.1.2 Symptomatic tears: Khun et al performed a study involving 452 patients and showed that nonoperative treatment using a physical therapy protocol is effective for treating atraumatic full-thickness rotator cuff tears in approximately 75% of patients that were followed up for 2 years20

6.2 Surgical treatment:

6.2.1 Non –reparative surgery such as Subacromial decompression and debridement have been used with satisfactory clinical results.

6.2.2 Rotator cuff repair is a common procedure and published success rates in terms of patient satisfaction are good

6.2.3 Tendon healing rates following surgical repair however vary for 40% to 95%.

6.2.4 The main factors in predicting tendon healing are patient age, size of tear, chronicity of tear, muscle atrophy and fatty infiltration

6.3 Arthroscopic vs Open:

6.3.1 In clinical practice guidelines published by the AAOS, the committee could not recommend a modality of surgical repair (eg, arthroscopicvsopen) as a superior method, citing a lack of comparative studies

6.3.2 However, other studies have shown a better visualisation and understanding of the disease with arthroscopic repair and have also been proven to be better for smaller tears, preserves the deltoid, lowers infection rates and postoperative pain and shortens hospital inpatient stay11

6.4 Single vs Double Row Repairs

6.4.1 The optimum technique for tendon repair is unknown but a meta analysis done by Xu et al has shown that a double row repair
rotator cuff repair has a significantly lower re-tear rate, higher American Shoulder and Elbow Surgeons Score (ASES) and a greater range of motion and internal rotation compared to a single-row repair technique12

7. The Biceps Tendon In RCD

7.1.1 The biceps-pulley anatomy plays a major role as a pain generator, in terms of concurrent tendonopathy and structural lesions such as partial tears, instability and intra-articular entrapment "the hourglass biceps"

7.1.2 Diagnosis: This can be made with both pre-operative palpation of the biceps groove and with a MRI/ultrasound scan. However, provocative tests are important because MRI or ultrasound scans cannot establish the exact location for tendon pathology.

7.1.3 If degeneration of the long head of the biceps triggers pain, this should be treated with either a tenotomy or tenodesis

7.1.4 Biceps tenodeses will preserve elbow supination and flexion when compared with tenotomy

8. Asymptomatic tears

8.1.1 Most asymptomatic tears respond to non operative management and remain asymptomatic for 2 years

9. Augments

9.1 Tendon Transfer

9.1.1 Normally used in younger patients with massive irreparable cuff tears where reverse shoulder arthroplasty is not indicated

a. Latissimus dorsi transfer has been utilised where the patient has an intact subscapularis, acceptable passive flexion, and a large posterior superior tear and severe atrophy
b. In a patient with an intact infraspinatus and a torn, atrophic subscapularis, a split pectoralis major transfer is another option

9.2 Reverse Shoulder Arthroplasty

9.2.1 Cuff tears in the presence of cuff tear arthritis is the primary indication for a reverse shoulder arthroplasty

9.2.2 This has been proven to provide pain relief and active elevation13

9.2.3 This operation can also be successfully performed in massive rotator cuff tears with pseudoparalysis but no arthritis, or in inflammatory arthritis

10. Future Therapies

10.1 Scaffolds and Grafts

10.1.1 Scaffolds have been used to augment the tendon to bone interface during repairs

10.1.2 However, further work needs to be done to optimise scaffold properties

10.1.3 The ideal scaffold would be an inductive template and recreate the natural gradation between the compliant tendon and stiff bone

10.2 Biological aids

10.2.1 Growth factors have been used to aid cartilage and tendon-to-bone healing with limited results

10.2.2 Scaffold embedded with growth factor has been attempted in animal models

10.2.3 Mesenchymal Stem Cells (MSC) transfected with a transcription factor necessary for tendon development showed positive results in rats. Transfection with type I matrix metalloproteinase also provided hopeful results

10.2.4 Platelet Rich Plasma (PRP) has shown no clear evidence of clinical benefit in patients undergoing rotator cuffrepair14

III. Glenohumeral Osteoarthritis (OA)

IIIA. Aetiology & Pathology

1. OA is defined as non-inflammatory degeneration of cartilage and narrowing of the GHJ space

2. OA is associated with a decrease in glycosaminoglycans, including chondroitin sulphate, hyaluronic acid, and keratin sulfate; increased water content as water diffuses into the cartilage as glycosaminoglycans are lost; and increased enzymatic activity from MMPs. The MMPs play an important role in degeneration of the extracellular matrix of cartilage

3. Degradation of cartilage in OA is attributed to mechanical stress that leads to multiple microfractures. Subsequently, altered chondrocyte metabolism and production of MMPs lead to gradual loss of cartilage, altered joint architecture, and osteophyte formation

4. GHJ OA can be divided into primary and secondary OA

4.1 Primary GHJ OA is increasingly being seen as a cause of pain and disability in the shoulder. It is generally of an idiopathic origin

4.2 Secondary GHJ OA may be caused by:

4.2.1 Trauma

4.2.2 Cuff tear arthritis

4.2.3 Increased glenoid retroversion/dysplasia

4.2.4 Avascular necrosis

4.2.5 Surgical complications such as post Capsulorraphy (Over tightening of soft tissues) and mal-positioned and migrated hardware

IIIB. Diagnosis

1. History:

1.1 Symptoms

1.1.1 Pain at night

1.1.2 Pain with activities of daily living

2. Physical examination

2.1.1 Tenderness at GHJ

2.1.2 Crepitus

2.1.3 Functional limitations at GH joint caused by decreased range of motion

2.1.4 Pain at end of range of motion

3. Imaging

3.1 Radiographic findings from true AP and axillary views are:

3.1.1 Joint space narrowing

3.1.2 Osteophyte formation ("goat's beard")

3.1.3 Subchondral cysts

3.1.4 Posterior wear or glenoid bone loss

3.1.5 Superior migration of head to indicate cuff tear arthropathy (acetabularization)

3.1.6 Evidence of previous surgery

3.2 Classification[15]
Walch classification of glenoid wear associated with primary OA (table 1)

IIIC. Treatment

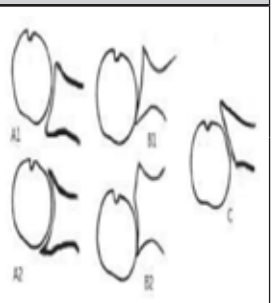
Walch classification of glenoid wear associated with primary OA		
Type A	Concentric wear, no subluxation, well centered A1 minor erosion A2 deeper central erosion	
Type B	Biconcave glenoid, asymmetric glenoid wear and head subluxated posteriorly B1 narrowing of posterior joint space, subchondral sclerosis, osteophytes B2 posterior wear, biconcave glenoid	
Type C	C Glenoid retroversion of more than 25 degrees (dysplastic in origin) and posterior subluxation of humerus	

Table 1

1. Conservative management:

1.1 Pharmacological treatment such as nonsteroidal-anti-inflammatory drugs (NSAIDs) (oral/topical), paracetmol, opioids and over the counter supplements have been used with varying degrees of success. A meta-analysis done by the AAOS of recent studies concerning pharmacotherapy use in GHJ OA showed that there were no studies of sufficient quality to recommend or disregard their use16

1.2 Intra-articular steroid injections are given regularly in an outpatient setting. However, there is no evidence that supports or refutes their use in the treatment of GHJ OA16

1.3 Intra-articular visco supplementation injections have limited use as there is insufficient evidence to support this treatment in the literature.

1.4 Physiotherapy is used to increase joint range of movement and strengthen scapular girdle muscles. It also combats stiffness which is a source of pain. However, there is insufficient evidence to make a conclusion in favour of or against the efficacy of physiotherapy16

2. Surgical Options: When non operative measures still do not alleviate pain and cause reduced activity, surgical intervention is often considered:

2.1 Arthroscopic debridement +/- capsular release

2.1.1 Considered in younger patients due to the concerns of prosthetic loosening and decreased implant survivorship with the need for revision surgery

2.1.2 Patients with mild to moderate OA may not have the severity of symptoms which warrant replacement, therefore arthroscopy is an option

2.1.3 Reports have shown that arthroscopic debridement +/- capsular release provides short term symptom relief in the osteoarthritic shoulder and provides a temporal bridge to arthroplasty

2.1.4 LHB tenotomy and removal of loose bodies may also be indicated

2.1.5 However, there is still a lack of quality evidence for or against the use of arthroscopic treatment

2.2 Biologic resurfacing with soft tissue interposition +/- humeral head replacement

2.2.1 Biologic resurfacing with knee meniscus, Achilles allograft, anterior shoulder capsule and other tissues have all been reported. Early reports were favourable, but mid-term results have shown a high rate of failure. Currently, glenoid resurfacing with biologic interposition is only recommended for young patients

2.2.2 Partial Humeral head prosthetic replacements with stem-less implants have been considered in small focal lesions, isolated avascular necrosis and in younger patients as it preserves proximal humeral bone stock for future revision surgery. It is not indicated for generalised forms of OA

2.3 Total Shoulder Arthroplasty (TSA)

2.3.1 TSA is the gold standard treatment for severe, symptomatic GHJ OA16

2.3.2 A TSA involves replacement of the humeral head and prosthetic glenoid re-surfacing

2.3.3 TSA offers the patient pain relief, increased function and improves quality of life

2.3.4 The main concern with TSA is glenoid component loosening, as this represents the most common complication

2.3.5 However good long-term survivorship with the use of polyethelene glenoid resurfacing has also been reported. Component material and engineering is an ongoing subject of research

2.4 Hemiarthoplasty (HA)

2.4.1 HA are used in select cases such as:

a. Rheumatoid arthritis
b. OA in younger patients
c. Severe bone loss
d. Avascular necrosis of the humeral head

2.4.2 The major early complication of HA implants is excessive wear of the glenoid

2.5 Resurfacing

2.5.1 Resurfacing hemiarthroplasty has been used with similar results to conventional stemmed prostheses, and are proposed as bone conserving, but are more technically demanding as TSR, and there is no proven advantage in the literature

2.6 Short stem

2.6.1 Short stem prosthesis, predominatly uncemented, have been recently introduced, are easier to use as TSR, but as yet are of unproven advantage

2.7 Cemented/Uncemented stems

2.7.1 Humeral stem survival has been shown to be good (>95% 10 yr survival) for both cemented and uncemented prosthesis

2.8 TSA vs HA

2.8.1 A systematic review of literature done by van den Bekerom et al has advocated the use TSA due to lower revision rates but incurs higher complication rates17

2.9 Patient outcomes and range of movement have generally been reported superior in TSR than Reverse Total Shoulder Arthroplasty (RTSA)

2.9.1 The RTSA is indicated primarily in the setting of rotator cuff insufficiency and rotator cuff arthropathy. It is also used in fracture treatment and in TSA and HA revision surgery

2.9.2 Reverse TSA has a constrained ball-in-socket design, with a hemispherical ball implanted in the glenoid and a stem with a concave polyethelene cup implanted in the humerus

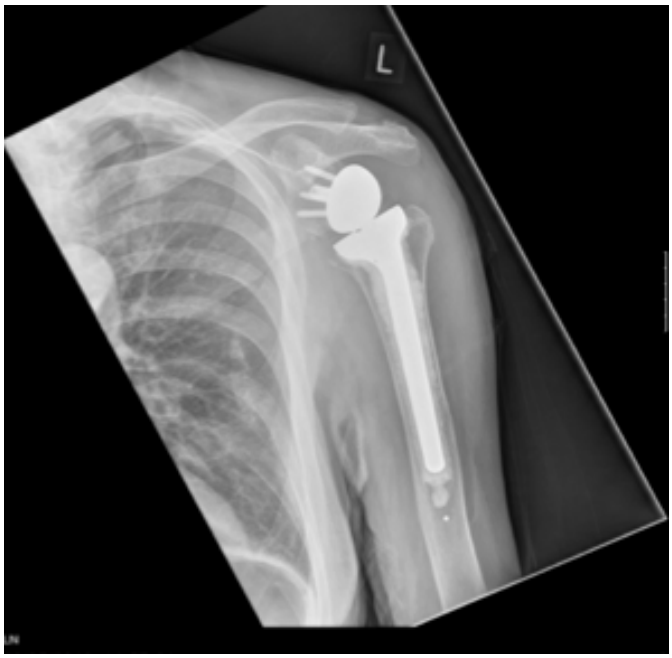


Figure 2: Reverse Total Shoulder Arthroplasty

2.9.3 Reversal of the components changes the biomechanics, medialising and distilising the centre of rotation and improving deltoid lever elevation in the absence of a rotator cuff

2.9.4 However, a competent deltoid is critical for a successful outcome.

2.9.5 Recently, RTSA have also been used in the treatment of irreparable rotator cuff tears without GHJ OA and rheumatoid arthritis with favourable results

Figure 2: Reverse Total Shoulder Arthroplasty

IV. Rheumatoid Arthritis (RA)

IVA. Aetiology and Pathology

1. RA is a chronic inflammatory disease with a reported prevalence of 0.5% to 1% and the shoulder joint is affected in approximately 60% of hospitalised patients with RA

2. The pathology of RA differs from both RCTA and OA. RA patients usually have a long history of their systemic disease, with medical treatment causing general osteoporosis, higher incidence of rotator cuff tears and a compromised immune system

IVB. Diagnosis

1. History and examination are similar to that of normal OA but there are differences in the radiographic images:

a. Peri-articular erosions
b. Osteopenia
c. Severe wear of the glenoid and the coracoid process
d. Cuff pathology also affecting infraspinatus and teres minor

IVC. Treatment

1. Conservative Treatment follows a similar path to the treatment given for OA with the addition of optimum medical treatment of the patients RA

2. Surgical Treatment presents challenges in performing shoulder arthroplasty due to osteopenia, rotator cuff disease, glenoid erosion and immunosuppression

2.1 Total Shoulder Arthroplasty VS Hemiarthroplasty

2.1.1 TSA has been shown to fail due to early glenoid loosening and rotator cuff failure

2.1.2 HA are known to have poor functional outcomes and provide partial pain relief

2.1.3 Barlow et al has shown TSA to be superior in patients with an intact rotator cuff in terms of function and pain

2.2 Reverse Shoulder Arthroplasty (RSA)

2.2.1 RSA have been used In RA patients with severe rotator cuff tears

2.2.2 Initial data are encouraging but longer term quality studies are needed

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Dr Domizio Suva

University Hospital of Geneva,
Geneva, Switzerland
email: domizio.suva@hcuge.ch

Osteoarthritis of the Elbow

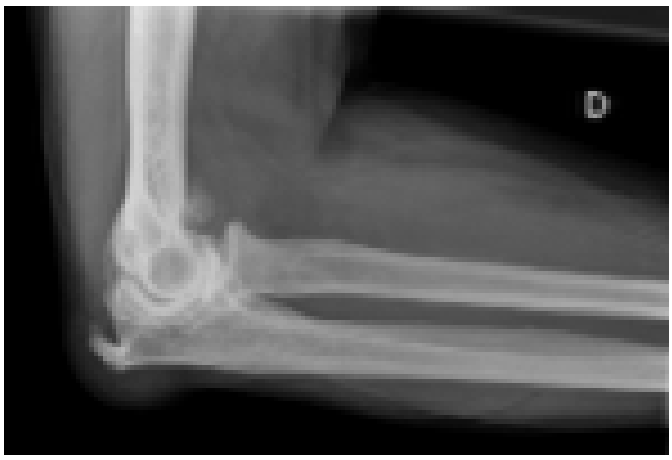
Primary osteoarthritis (OA) of the elbow is relatively rare, affecting approximately 2% of the population. The average age of patients at initial presentation is 50 years old, and the male-to-female ratio is 4:1. The etiology is unclear, but primary elbow OA is usually associated with a history of heavy 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.

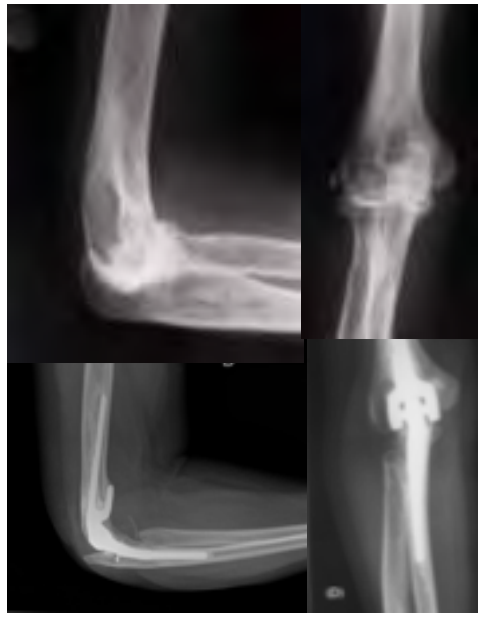
The classic Outerbridge-Kashiwagi (OK) procedure includes a posterior approach to the elbow with a triceps split, removal of the tip of the olecranon, and osteophyte removal through olecranon fossa trephination. Limitations of this procedure are an incomplete anterior release and inability for osteophyte 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.



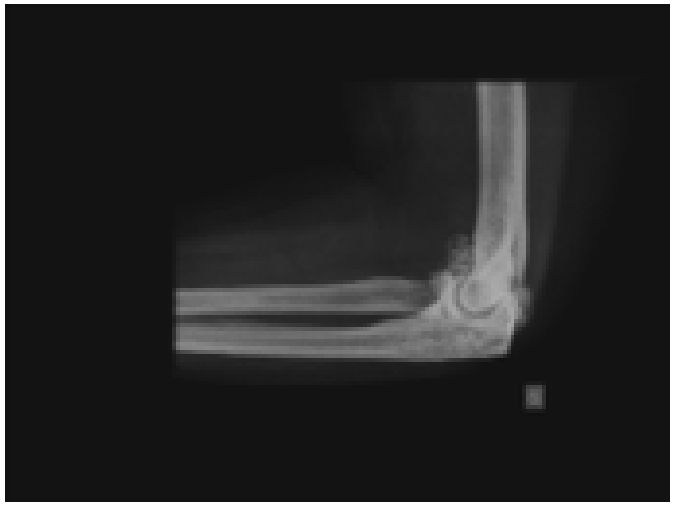
The Rheumatoid Elbow

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.

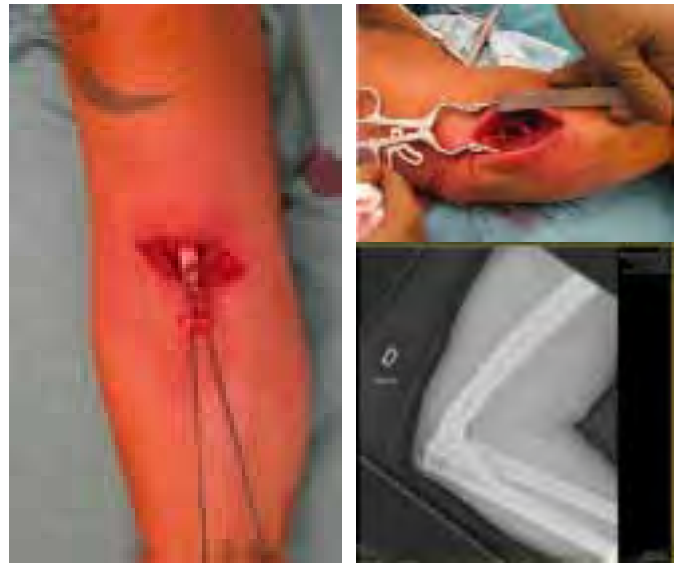
Synovial chondromatosis



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

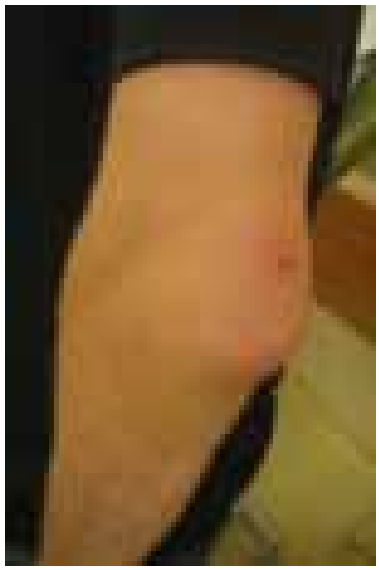
This usually affects men between 40 and 60 years of age, and generally occurs in the dominant extremity. The mechanism is a single traumatic event in heavy workers or weight lifters. Patients report pain and weakness in flexion and supination. Clinical examination reveals an absent distal biceps tendon, with proximal retraction of the biceps muscle. Most often the diagnosis is clear, but in cases of 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 canaland 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) exercices 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, andafter several days secondary closure can be performed. During the treatment period appropriate antibiotics are administered intravenously.

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Prof. Dr. Philippe Neyret

Hôpital de la Croix Rousse
Lyon, France
philippe.neyret@chu-lyon.fr

ACL, PCL, COLLATERALS

Authors:
Victoria B.Duthon
Sébastien Lustig
Elvire Servien
Prof. Dr. Philippe Neyret

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 differencial laxity with the controlateral knee can be evaluated with an arthrometer, for example KT-1000®. More thant 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 weigthbearing radiographs, or on stress radiographs (Telos®) with a 50N anterior tibial drawer.

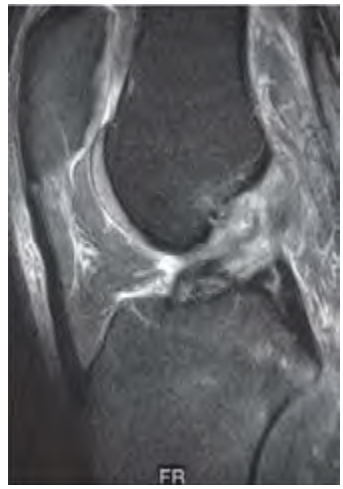

Fig. 1 ACL rupture.

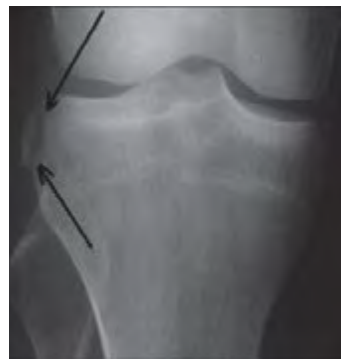

Fig. 2 "Segond" fracture.

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 strenghten 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 positionning 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 (occasionnaly 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 wich sometimes is longuer 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 throught 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 positionning of the graft may lead to persistant 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 Endobuton®, or may also be impacted in the tunnel (press-fit). Double tibial fixation is prefered 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 trafic road accident with dash-board injury, which is the most frequent trauma mechanism. Accidental kneeling with ankle plantar-flexion 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 controlateral side.

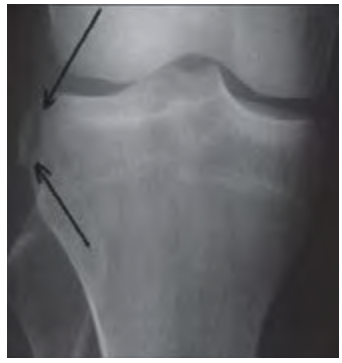

Fig. 3 PCL rupture.

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 strenghtening 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 quadricipital 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 interferences screws, or outside with an Endobuton® for example. If arthroscopic reconstruction is not technically possible, tibial PCL graft fixation can be done by a posterior knee approach with a stample.

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.

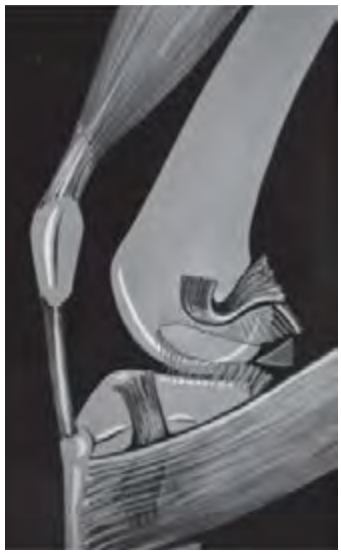

Fig. 4 MCL rupture

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 longuer 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 asymetric 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 asymetric lateral compartement 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 largerly 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.

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Prof. Rene Verdonk

Gent state university,
Gent, Belgium
email: rene.verdonk@uqent.be

The sports knee

INTRODUCTION

The knee anatomy evolved over millions of years from crawling to standing position without much of a change in its anatomic relationship between femur , tibia, patella and ligament fixation and menisci. Every single element has its place regarding form and function.

THE MENISCI

Well known medial C and lateral O shape presentation protecting the weight bearing cartilage of the femur and tibia. The menisci have 4 essential functions

- Stabilizing
- Load bearing
- Shock absorption
- Lubricating

When partially or totaly removed pressure increases leading to cartilage degeneration.

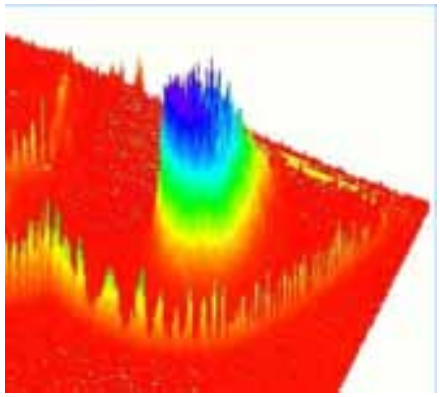

Fig 1 meniscal resection will dramatically increase pressure on the weight bearing cartilage (Amis et all)

Therefore meniscal repair must be performed in every possible case and more specifically in children. meniscal repair results are highly satisfactory and have an average succes rate of 75% when performed in the red on red and red on white zone.

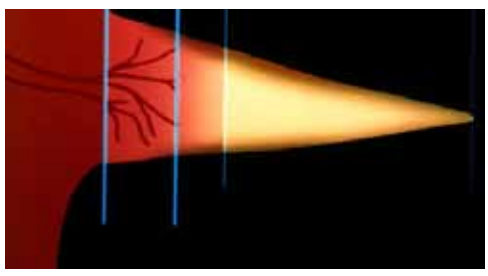

Fig 2 : mensical suture result are optimal in red on red and red on white zones. White on white zone sutures give disapointing results

Imaging results are somewhat less obvious depending on the evaluation technique (MRI vs CT and arthro CT vieuwing)
Statistics suggest better average healing in the lateral vs the medial meniscus. The surgeon needs to take the risk of failure when confronted with a meniscal suture indication !
indeed bucket handle tears may heal less well but then resection of the meniscus will inevitably lead to early degenerative cartilage.
This approach leads to 10 Commandments in meniscal repair

1. perform a preop MRI
2. use all inside or combined hybrid sutures
3. perform peroperative debridment of the leasion
4. obtain a a primary stable construct
5. in stable knees the periferal lesions should be reapaird
6. in athletes the horizontal tears should be repaired
7. in ACL deficient knees the medial meniscus needs repair. The lateral meniscus can be repaired or left alone
8. in ACL deficient knees the ACL should be repaired in conjunction with meniscal repair
9. the indication for repair should be pushes in children
10. by all means save the meniscus

Meniscal resection will lead to

- decreased capacity to distribute load
- higher peak stress on cartilage
- cartilage degeneration
- which may bring the surgeon to suggest meniscal replacement
- in case of TOTAL meniscectomy a meniscal ALLOGRAFT is indicated more specifically in the lateral compartment
- in specific cases of partial meniscectomy a meniscal substitute may find an indication using a CMI implant (collagen meniscal implant) or preferably an ACTIFIT (Orteq) implant in polyurethane.

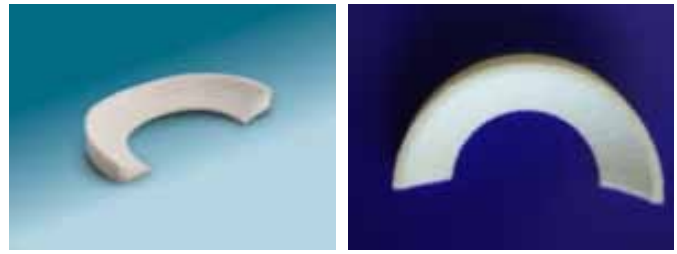

Fig 3 CMI implants (left) and ACTIFIT implant (right) are most valuable alternative to replace partially remove menisci with good succes

As a conclusion in the approach to meniscal lesions we suggest that

- PAINFULL partial meniscectomy R/ partial replacement? .....
- PAINFULL total meniscectomy / meniscal allograft .....

- But only in
- Correct alignment
- Stable knee
- MINIMAL cartilage deficit
- < 50 y

The Central Pivot Ligaments

OA predictors at 24.5 y suggest , after proper ACL repair to be

- medial cartilage lesion X 5
- medial meniscectomy X 3
- higher age at time of intervention X 1.15
- longer surgical delay X 1.15

- showing that when a medial meniscal cartilage lesion is found ab initio chances are X5 to develop OA
- a medial meniscectomy ab initio leads to 3 X more OA at 24.5 y
- higher age ab initio and a longer surgical delay originally do not compromise OA development as dramatically

- so , if ab initio, one can approach cartlage lesions and meniscal lesions appropriately chances are degenerative arthritis will not appear as readily as today

THE PATELLA

Anterior knee pain , in which the patella may be involved may find several causes such as

- Patellar syndrome
- Patellar dislocation
- Paterllar subluxation
- Patellar instability
- Osgood schlatter diseaseHyperpression
- Any vascular issue
- Patellar tendinitis
- Pschychiatric background

Fig 4: the patella presents with various forms of pathology

In fact, regarding instability, Dejour H and Walsch G defined 4 major reasons that lead to true instability being

- Trochlear dysplasia
- A high riding patella
- A TT –TG > 20°
- A patellar tilt > 20°

To stabilze the patella one should proceed as diagnostically as possible looking for

- Reducing the TT-TH to less than 15mm
- To address trochlear dysplasia in grade B en D
- In case of High riding patella to index 1
- In case of patellar tilt using VMO plasty techniques and MPFL repairing techniques when indicated

ADRESSING FRESH CARTILAGE LESIONS

Agreat number of cartilage lesions are found in case of " routine " arthroscopies

According to Aroen 6% of these lesions are ICRS grade 3 – 4 and extending to mere than 2cm square

A basic approach to these sometimes symptomatic lesions is the Microfracture technique as developed by Steadman. The awl should perforate the subchondral lamina every 2 to3 mm, go deep (to 4mm) and expect bleeding to accur in situ.

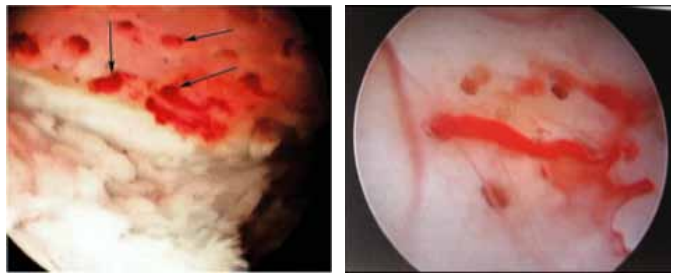

Fig 5 : Microfracture techniques require rigourous surgery (Steadman et all)

In larger surface lesions the Microfracture approach may be insufficient as one has to move on to Mosaicplasty preferably using single bone carrots to obtain better fixation and ingrowth bringing better mechanical results

The ideal case for cartilage surgery remaining

- Young patient < 35- 40 y, activ in free time sports
- traumatic Lesion, recent < 3 m
- Normo axial knee , no associated lesions
- BMI < 30 kg/m2
- size : < 3 cm2

as a conclusion

- to this chapter one should approach
- the meniscus by saving it
- the ligaments by repairing them
- the patella by stabilizing
- the cartilage by taking care of it

Olivier Borens

University Hostpital Lausanne
Lausanne, Switzerland
olivier.borens@chuv.ch

Infections after TJA

Introduction

Implants are used in most surgical specialties. Despite advances in material design and biocompatibility, implant-associated infections remain a difficult to treat complication, causing high morbidity, considerably mortality and consume healthcare costs, result in patient restriction from work and compromise their social life. The number of total joint replacements infections is expected to increase along with the growing number of joints implanted and about 2 to 4 percent of TJA will need treatment for infection even if adequate prophylactic measures are taken. The ultimate aim in treating infected TJA is to regain a pain free and functional joint. Whenever possible, the joint should also be free of infection. In order to achieve these goals, a clear treatment concept is needed.
The treatment concept is based on five strong pillars: teamwork, understanding biofilm, proper diagnostics, proper definition and classification and patient-tailored treatment.

Teamwork
A multidisciplinary approach is needed in treating TJA-infections since the combination of good diagnosis, surgery and antibiotic treatment has been shown to have the highest success rate in eradication of infection. Nonsurgical treatment alone with suppressive antibiotics will not be able to eradicate infection. The surgeon will decide the best among the possible surgical options and the infectious disease specialist together with microbiologist who will decide on the best option among the antibiotic arsenal. The multidisciplinary team for treating TJA infection can be reinforced further by a plastic surgeon, a vascular surgeon or an internal medicine specialist.

Understanding the "biofilm"
TJA, like other foreign bodies are prone for colonization by microorganisms. Microorganisms that colonized TKR cause formation of biofilms. Biofilms can be defined simply and broadly as communities of bacteria attached to surface. Unlike their freeliving (planktonic) counterparts, bacteria's in biofilms live in an organized structure at a liquid interface. The formation of biofilms is a rapid process. Within minutes to hours bacteria attach to surface of an implant. In the following stages, these bacteria become embedded in a matrix (glycocalix) and they enter a stationary stage. Problems associated with biofilm in TJA result from the characteristics of biofilms. Firstly, biofilms are difficult to clear by immune killing or by treatment with antibiotics. Bacteria's in biofilm are difficult to attack because of the compact structure of biofilm and because they enter stationary state. Secondly, biofilms might be capable of shedding individual bacterial and sloughed pieces of biofilm into surrounding tissues that cause infection.

Diagnostics

Up to date, there is no single set of accepted criteria for TKR infection that can be considered as gold standard. Recently, American Academy of Orthopedic Surgeons (AAOS) published their proposed criteria to define prosthetic joint infection (PJI). In this proposal, diagnosis can be made based on signs only, or microbiological culture of tissue only, or combination of laboratory- and pathological findings. According to this proposal, a definite diagnosis of PJI can be made when several conditions are met (Table 1).

1.	A sinus tract communicating with the prosthesis; or
2.	A pathogen is isolated by culture from two separate tissue or fluid samples obtained from the affected prosthetic joint; or
3.	For the following six criteria exist: Elevated serum erythrocyte sedimentation rate (ESR) or serum C-reactive protein (CRP) concentration (CRP can be normal in low grade infections!) Elevated synovial white blood cell (WBC) count Elevated synovial neutrophil percentage (PMN%) Presence of purulence in the affected joint Isolation of a microorganism in one culture of periprosthetic tissue or fluid Greater than five neutrophils per high-power field in five high-power fields observed from histologic analysis of periprosthetic tissue at 400 times magnification.

Table 1: American Academy of Orthopedic Surgeons (AAOS) published their proposed criteria to define prosthetic joint infection

Several novel diagnostic methods are not included in the proposal such as sonication and polymerase chain reaction (PCR) but are worth mentioning. Sonication is a method used in the microbiology laboratory to dislodge microorganisms from the biofilm formed on the surface of the prosthesis (16). This technique has been shown to have higher sensitivity for the microbiologic diagnosis of PJI than tissue culture. Sonication is particularly helpful in detecting bacteria in patients treated recently with antibiotic.

Definition and classification

PJI can be classified as: early, delayed and late (Table 2). Early infections occur within 3 months after implantation. The germs caused early infections are usually acquired during the implantation of the TKR. Delayed infections occur between 3 and 24 months after implantation. The germs caused delayed infections are also acquired during surgery but they are usually less virulent like Propionibacterium acnes or Staphylococcus

epidermidis. Late infections are generally hematogenous and occur later than 24 months after implantation surgery. Staphylococcus aureus is the most frequent cause of hematogenous TKR infection.

Classification	Beginning of the infection after implantation	Pathogenesis	Typical germs
Early infection	< 3 months	During surgery	Highly virulent germs like S. aureus or Gram negative bacilli
Delayed infection	3-24 months	During surgery	Less virulent germs like S. epidermidis or P. acnes
Late infection	>24 months	Mainly hematogenous	Typically highly virulent germs like S. aureus, streptococci or Gram negative bacilli

Table 2: Types of infected TKR according to when the symptoms start after implantation

After the classification, the next step is to differentiate whether the infection of TKR is acute or chronic. This differentiation is important for the choice of the treatment option. In acute infection the beginning of the symptoms should be less than 3 weeks before the diagnosis. Infection is considered to be chronic when the beginning of the symptoms occurs longer than 3 weeks. Acute infection can occur in the early postoperative phase, and is usually due to highly virulent germs like Staphylococcus aureus. Acute infection can also occur in the late postoperative phase. The symptoms start out of the blue after infection of the knee by the microorganisms from a distant infectious focus. Chronic infection can also happen in early (0 to 3 months postoperative) and in late (more than 24 months postoperative) phase. The patients have typically persistent knee pain.

Patient-tailored treatment

Once the diagnosis has been made and the type of infection has been determined, the orthopaedic surgeon together with the infectious disease specialist must make a treatment plan to achieve painless, functional and infection free PJI.

Solely medical treatment will not lead to eradication of the infection. Therefore, it should be only used in patients with multiple co-morbidities (e.g. patients too sick to endure surgery), in patients who refuse surgery or when surgery is technically challenging. In all other situations, surgery in combination with appropriate antibiotics is the way to reach the treatment goal. The choice of surgical treatment is determined by several factors: The type of infection (acute versus chronic), the state of the soft tissue, the type of pathogens and their susceptibility to antibiotics, and patient's expectation. There are five surgical options (Table 3).

1.	Débridement and retention
2.	One-stage exchange
3.	Two-stage exchange a. with short interval (two to four weeks) b. with long interval (six to eight weeks)
4.	Arthrodesis
5.	Amputation

Table 3: Five surgical options in treating PJI

Débridement and retention with exchange of mobile parts

Adequate débridement and retention with good results can only be done through an open procedure and not by arthroscopy. It consists of arthrotomy, extensive synovectomy, irrigation with 9 litres of saline and debridement of all infected soft tissue followed by exchange of the mobile parts. Intraoperative cultures are taken and mobile parts are sent for sonication. The patients are further treated with antibiotics for 3 months.

Compared to one- or two-stage exchange, there are several potential advantages of this method. Firstly, many patients can return to full activity quickly as the surgical aggression and the blood loss are minor than in exchange procedure. Secondly, surgical time and costs (only one surgery is performed and only mobile parts are exchanged) are lower. Thirdly, the risk of intraoperative fracture is low since the bone stock is left intact unlike during removal of a well fixed prosthesis where bone loss occurs. Lastly, there will be no soft tissue distension or retraction unlike in two-stage exchange where spacers are implanted.

There are several requirements for a successful outcome when débridement and retention is performed. Firstly, the implant should be stable. Secondly, the infection should be acute (i.e. less than 3 weeks). Thirdly, the soft tissue should be intact. Lastly, the pathogens should be sensitive to biofilm active antibiotics (i.e. rifampin, ciproxin, fosphomycin).

One –stage revision

This surgical option consists of removal of the prosthesis, débridement of infected soft tissue, reimplantation of the prosthesis.

The theoretical advantage of this procedure compared to two-stage exchange is the elimination of the demanding second surgery that could result in complications, prolonged hospital stay and increased patient morbidity related to the spacer. A disadvantage is the need to do more aggressive debridement and often the need for more constrained implants.

There are several requirements in performing one-stage exchange. Firstly, the soft tissue condition should be satisfactory. If soft tissues are not sound, the risk of wound breakdown and persistence of infection or super-infection are increased. Secondly, like in débridement and retention, the pathogens should be sensitive to biofilm active antibiotics (i.e. rifampin, ciproxin, fosphomycin).

Two–stage revision

This procedure is considered the gold-standard in the United States and appears to be the most effective surgical management for infected TKR with an success rate of 88% to 96% if débridement and retention are not possible.

In the first stage, the prosthesis is removed. All macroscopically infected bone must be removed together with cement fragments as they are covered with biofilm and might become a reservoir of infection and increase the risk of persistent infection. Intraoperative cultures are taken and the removed prosthesis is sent for sonication. A cement spacer can be implanted at that moment.

Among the general benefits of cement spacers are: prevention of abundant scar tissue formation, maintenance of joint space, prevention of contraction of the collateral ligaments, facilitation of exposure and revision, and improvement of patient comfort between the stages. In current practice, cement spacers for two-stage exchange are impregnated with antibiotics since they can deliver local antibiotics in much higher dose than using intravenous administration.

In general, we can differentiate the time of reimplantation in short and

long interval. The short interval is when the reimplantation is performed 2 to 3 weeks after the first stage. In long interval, the reimplantation is performed at 6 to 8 weeks after the first stage. Short interval is preferred over long interval in situations where the soft tissues are in good state and the pathogens are not difficult to treat (i.e. sensible to rifampin or ciprofloxacin). Among the benefit of short above long interval are: less scarring or contracture and shorter intravenous antibiotic therapy needed and subsequently less cost for monitoring this therapy.

Arthrodesis

Arthrodesis can be done by external fixation, plating or nailing and will help relieving pain and restoring some function. Achieving consolidation of the arthrodesis can be difficult and the chances of good bone healing and stability are directly related to large surfaces of bleeding bone and good bone contact if possible under compression.

Amputation/Girdlestone

In cases of recurrent infection, difficult to treat organisms and bad soft tissues or an incompetent extensor apparatus above-the-knee amputations can be considered. For the hip Girdlestone procedure can be a viable option.

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Prof. Dr. Ulrich Stöckle

Klinikum rechts der Isar der TU München
Munich, Germany
stoeckle@uchir.me.tum.de

Fractures: Pelvic ring and 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 lumbal 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 then 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.

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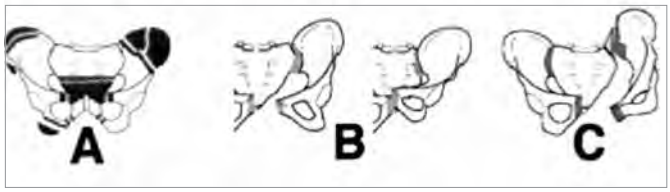
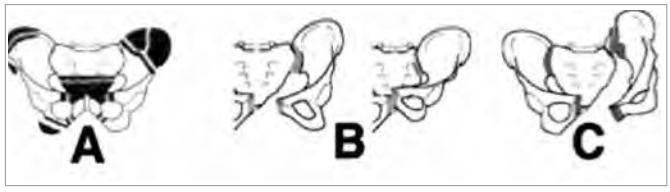
Figures

AO- Classification



a

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



b

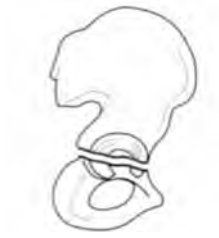
A Conservative treatment
B Predominantly operative treatment
C Operative treatment

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



a

Combined fractures

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

b

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			

c

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

Dr. Olivier Borens

University Hospital Lausanne
Lausanne, Switzerland
olivier.borens@chuv.ch

Fractures: Hip, femur, tibia & open fractures

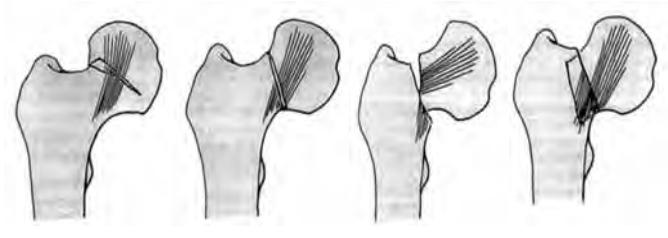
1. Intracapsular hip fractures

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.B.3.3.



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

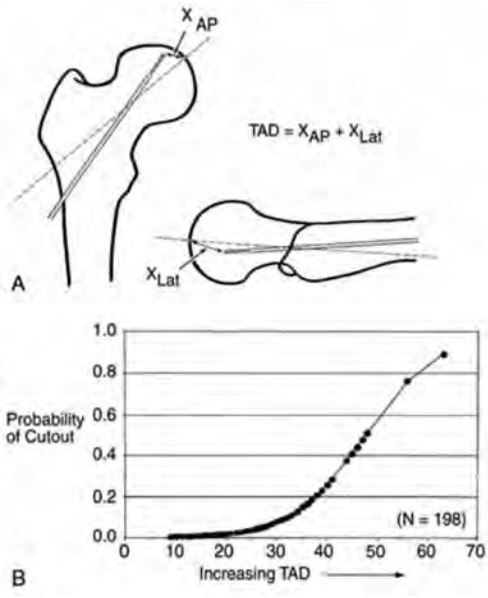
Treatment: A femoral neck fracture in a young patient should be considered an orthopaedic emergency, elderly patients should be operated as soon as possible once they are medically cleared and ideally within 48h. Young patients with a displaced femoral neck fracture should benefit from anatomic reduction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

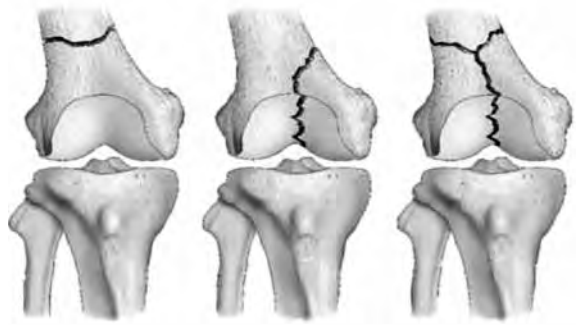
Treatment: Nonsurgical treatment with only traction can be used but comes with shortening, rotational malunion and knee stiffness and is thus not the treatment of choice. EX FIX is used in severe open fractures as well as for the initial stabilization in hemodynamically unstable poly- trauma patients 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.

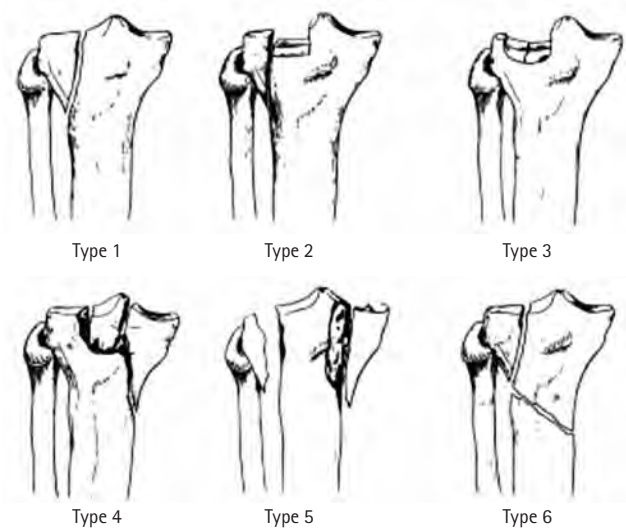
Classification: Usually the AO classification is being used.

Treatment: For successful treatment 4 conditions must be fullfilled: 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 metapyseal-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.

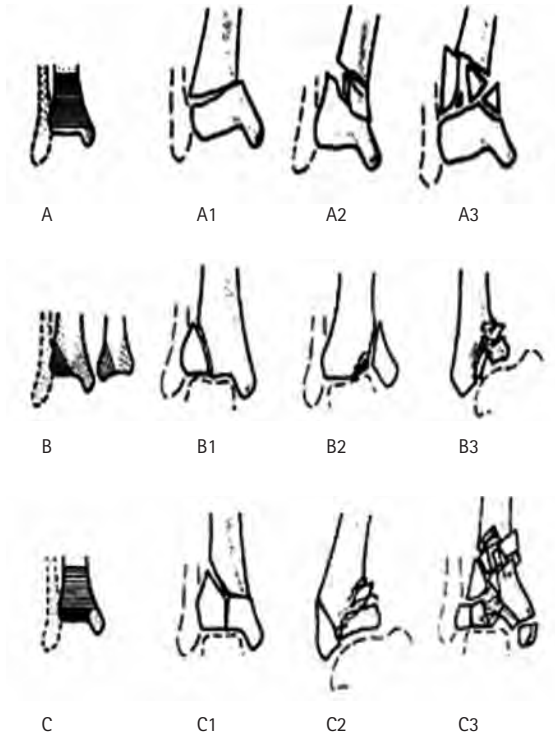
Treatment: All tibia shaft fracture needs treatment. This can go from simple splinting to amputation. Acceptable displacement for conservative treatment with a cast or brace are: angular malalignment of ≤ 5° in all planes, rotational malalignment within 10°, and < 10-12mm of shortening [Brumback]. Plate 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 malalignmemt 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

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Dr. Jordi Tomás Hernández

Trauma Unit. Hospital Vall d'Hebron.
Barcelona, Spain.
jotomas@vhebron.net

Fractures: Pilon, Ankle, Talus & Calcaneus

1. Ankle Fractures:

1.1. Epidemiology:

- Most common injuries requering orthopaedic care.
- Highest incidence in elderly women.

1.2. Mechanism of injury:

Most ankle fractures result from low-energy rotational forces. Injuries compromise bony and soft tissue structures.

1.3. Anatomy:

Medial malleolus, lateral malleolus (fibula), posterior malleolus. Ligaments: deltoid, talofibular and calcaneofibular, syndesmotic complex (anteroinferior tibiofibular ligament (AITF) and postero-inferior tibiofibular (PITF) ligament) and the interosseous ligament.

1.4. Clinical Evaluation:

- Mechanism, timing, soft-tissue injury, bone quality, comorbidities (diabetes), associated injuries
- Skin (open medial wound?), circulatory and neurologic exam (superficial/deep peroneal, sural and posterior tibial nerves), pain and deformity.
- Fracture- dislocations and soft-tissue swelling should be reduced and assessed because it will affect surgical timing.

1.5. Radiographic Evaluation:

- Standard 3 views: AP, lateral and Mortise. Considerations:
- Subchondral bone of the tibia and fibula should form a continuous line around the tallus in all views.
- AP view: Tibio-fibular overlap around 10mm; talar tilt 83º +/- 4º.
- Mortise view: Tibio-fibular clear space <6mm; Medial joint space <4mm and equal to the superior clear space between the talus and the tibia.
- Lateral view: talar subluxation; distal fibula translation/ angulation, syndesmotic relationship; occult hindfoot injuries.

1.6. Classifications:

- AO/Weber classification: fractures classified based on the location of the fibular fracture. Weber A (44-A): Infrasyndesmotic and less likely to result in instability.
  Weber B (44-B): fracture at the level of the ankle syndesmosis. Most common type.
  Weber C (44-C): Suprasyndesmotic and generally unstable. A very high Type C fracture is also called Maissoneuve fracture.
- Lauge-Hansen classification: Describes four major fracture types based on the mechanism of injury; first word: position of foot/ second word: force applied to foot relative to tibia.

1. Supination-adduction (SAD). Infrasyndesmotic fibula fracture+/- vertical medial malleolar with medial plafond impactation.
2. Supination-external rotation (SER). Most common, 70% of ankle fractures
   SER Stage-1: Disruption of the AITF ligament or Tillaux-Chaput fragment or Wagstaffe avulsion (distal anterior fibula).
   SER Stage-2: Spiral fibula fracture at syndesmotic level.
   SER Stage-3: Disruption of the PITF ligament or posterior malleolus fracture (Volkman)
   SER Stage-4: Fracture of the medial malleolus or deltoid ligament disruption.
3. Pronation-external rotation (PER).
   PER Stage-1: Medial malleolus fracture or deltoid disruption.
   PER Stage-2: Disruption of the AITF ligament or Tillaux-Chaput fragment or Wagstaffe avulsion.
   PER Stage-3: Suprasyndesmotic fibular fracture with interosseous membrane disruption.
4. Pronation-abduction (PAB).
   PAB Stage-1: Medial malleolus fracture or deltoid disruption.
   PAB Stage-2: Disruption of the AITF/PITF ligaments
   PAB Stage-3: Short Oblique fibula fracture at joint level laterally comminuted.

1.7. Treatment:

Nonsurgical:

- In stable fibular fractures without associated medial injury.
- Beware of a single fibular fracture with a normal medial clear space: "Gravity" test or manual stress test.
- Short leg cast or functional brace for 4-6 weeks.
- Weight bearing is permitted when symtoms allow.

Surgical:

- Goals: To achieve an stable, reduced, healed ankle fracture to allow ambulation.
- Initial management: A "good" closed reduction and well-splinted ankle to maintain the reduction. In case of severe instability or skin problems: External Fixation is an option.
- Definitive management: "Timing" (skin)
  - Lateral Malleolus : Must be restored to normal length and rotation.
  - Options: lag compression screw and 1/3 tubular plate or precontoured plate . Posterior antiglide plate , lag screw or tension band wire in very distal and transverse fractures.

Avoid injury to the superficial peroneal nerve.

- Medial Malleolus : Oblique fractures can be stabilized with two 4.0mm partially threaded cancellous screws. Vertical shear fractures with articular impactation: ORIF and bone grafting and antiglide or buttress plate fixation with a 1/3 tubular plate. Tension band for small fragments. o Posterior malleolus : Requires anatomic reduction if fragment size is >20% of articular surface. Difficult to accurately image on plain radiographs, CT can be very useful. Options: One or two lag screws from either anterior to posterior or posterior to anterior. Posterior antiglide plate is another option in osteoporotic bone (posterolateral approach).
- Syndesmosis : Check stability after ankle is fixed ("hook" test or manual stress test). Options: 3,5 vs 4,5mm screws, 3 cortices vs 4 cortices, retain vs remove and metallic vs suture…No consensus. In cases of Maissoneuve fractures is recommended to use 3,5mm cortex screws through 4 cortices and consideration given for two screws.

1.8. Diabetic Ankle Fracture:

- Beware the diabetic ankle fracture. Treatment goals are the same.
- Complications much higher regardless of treatment modality chosen.
- "Double everything": Fixation, time to suture removal, time to weight bear, frequency of office visits for wound checks, cast changes, etc.

1.9. Postoperative Management:

- Posterior splint for 6 weeks (prevent ankle equinus and allow for soft tissue healing).
- Ankle range of motion exercices are allowed during first 6 weeks.
- At 6 weeks: Progressive weight-bear if radiograph is satisfactory.

1.10.- Complications:

- Non-union , rare. Usually involves medial malleolus.
- Mal-union.
- Wound problems.
- Infection.
- Postraumatic arthritis.
- Reflex sympathetic dystrophy.
- Compartment syndrome of foot.
- Tibiofibular sinostosis.
- Loss of reduction.
- Loss of ankle range of motion.

2. Pilon Fractures(Plafond):

2.1. Epidemiology:

- A plafond fracture is an intrarticular fracture of the weight bearing surface of the distal tibia.
- Less than 10% of lower extremity injuries.
- Males about 35-40 years of age.

2.2. Mechanism of Injury: Two types.

- Rotational type: low energy injury with little conminution and few articular fragments. Soft tissue less affected.
- Axial compression: High energy trauma with extensive bony and joint conminution. Soft tissue with severe damage.

2.3. Clinical Evaluation:

- Mechanism, timing, soft-tissue injury, bone quality, comorbidities (diabetes), associated injuries
- Skin (open medial wound?), circulatory and neurologic exam (superficial/deep peroneal, sural and posterior tibial nerves), pain and deformity.
- Fracture- dislocations and soft-tissue swelling should be reduced and assessed because it will affect surgical timing.

2.4. Radiographic Evaluation:

- 3 ankle views and a full length tibia to check the joint above and below. Fibula is usually fracture in the rotational types and may not be fracture in the axial types.
- CT scan after external fixation is mandatory in every case to fracture analysis prior the surgery .

2.5. Classifications:

- AO classification: 43-A or extrarticular : should be considered as distal tibia fractures and not pilon fractures. 43-B or partial articular and 43-C complete articular fractures.
- Rüedi-Allgöwer classification: Type I: Non-displaced. Type II: Displaced but minimally comminuted. Type III: Highly comminuted and displaced.
- Tscherne classification: Treatment largely depends upon the soft tissue envelope. The most vulnerable skin for tibial plafond fractures is the anteromedial side of the tibia and its injury should be classified according to Tscherne classification (Grade 0 to Grade 3).

2.6.- Treatment:

Nonsurgical:

- Less common for tibial plafond than for ankle fractures.
- Stable patterns without displacement or nonambulatory patients or patients with significant neuropathy.
- Long leg cast for 6 weeks followed by a fracture brace to progressive weight-bear and range-of-motion exercices.

Surgical:

- Goals: To achieve an stable, reduced, healed ankle to allow ambulation .
- Initial management:
  - Rotational injuries (low energy): splint and elevation.
  - Axial compression injuries (high energy): Ankle-spanning external fixator (delta or rectangular frame). No consensus on fixing the fibula acutely (ensure >5cm between incisions).
- Timing for surgery: Until resolution of soft tissue injury (2-3 weeks normally). "Wrinkle" Test. Blisters epithelialized.
- Surgical Approaches: Many options: Anteromedial, Direct anterior,
- Anterolateral, Posterolateral and posteromedial. Choose one or another or combine depending the fracture pattern and the soft tissues (fracture "personality").
- Surgical sequence:
  - 1) Usually fix the fibula first to get the length. An intraoperative distractor or an external fixator could be a useful device to assist the surgery.
  - 2) Restore articular congruity by reduction of impacted articular fragments working superior to the impacted area and bringing the fragments down to the joint level.

- 3) Bone graft or bone graft substitute to support the articular surface.
- 4) Temporary K-wires to maintain the reduction of the fragments
- 5) Plate fixation (precontoured plates usually). Many options and possibility to combain (depending on fracture pattern): Anterolateral plate, anterior plate, medial plates, antiglide posterior plates.
- Soft tissue management during surgery requires very careful handling to avoid wound/skin problems.

- In some cases, this fractures could be treated definitively with external fixators.

2.7. Postoperative Management:

- Posterior splint for 2 weeks (prevent ankle equinus and allow for soft tissue healing).
- Removable Boot until 12 weeks.
- Ankle range of motion exercices are allowed after skin is healed.
- At 12 weeks: Progressive weight-bear if radiograph is satisfactory.

2.8. Complications:

- Non-union and delayed union
- Mal-union.
- Wound problems.
- Infection.
- Postraumatic arthritis.
- Reflex sympathetic dystrophy.
- Loss of reduction.
- Loss of ankle range of motion.

3. Talus Fractures:

3.1. Epidemiology:

Second most common bone fractured of all tarsal bones, compromise about 0.3% of all fractures.

30% of all talar fractures involve the neck . 1% of talar fractures involve the body

3.2. Anatomy of the talus:

- 3 parts: Body, neck and head.
- Mostly articular, 70% covered by cartilage.
- Limited blood supply: Artery of the tarsal canal and deltoid artery (body), artery of the tarsal sinus (head and neck).

3.3. Mechanism of Injury:

Mechanisms of injury of neck fractures

a. Hyperdorsiflexion of the foot on the leg ("aviator's astragalus")
b. Axial load on plantar surface of fixed talus
c. Direct blow on the dorsum of the foot.

Mechanisms of injury of body fractures: Axial compression of the talus between the plafond and the calcaneus.

3.4. Radiographic Evaluation:

- 3 ankle views (AP, lateral and mortise)
- AP, lateral and Canale views of the foot.
- CT: essential to understand fracture morphology.
- MRI can be used postoperatively to detect osteonecrosis.

3.5. Classifications:

Classifications of talar neck fractures: Hawkins

a. Type I: Nondisplaced
b. Type II: Subluxation or dislocation of the subtalar joint
c. Type III: Dislocation of the subtalar and ankle joints
d. Type IV: Types II or III with subluxation or dislocation of the talonavicular joint

Classification of talar body fractures: Best method is to describe the location of the fracture.

3.6. Treatment:

Talar Neck or Body

Nonoperative care : Useful only for Type I or nondisplaced body fracture but need to make certain there is no displacement.

Preferred method of treatment is open reduction internal fixation. Urgent surgical treatment when subluxation/dislocation leads to soft tissue compromise.

Surgical Approaches:

a. Anteromedial: Good for body fractures, often requires osteotomy of medial malleolus to treat body fracture
   i. Use lag screw technique to compress fragments
   ii. Need to countersink the screw heads
b. Anterolateral: Good for neck and lateral body fractures
c. Ollier: For lateral body, lateral process or fusions
d. Posterior: Good for fusions and posterior process fractures

Surgical implants

a. Need small and mini-fragment implants (titanium) along with biofix pins
b. Don't be afraid to use a plate especially when dealing with neck comminution.
c. Be sure that you have obtained the correct length and rotation before applying definitive fixation.
d. If adjacent joints are unstable (talonavicular joint), don't be afraid to apply a pin across the joint to provide additional stability. Pins can be pulled at 3-5 weeks.

3.7. Results of neck fractures and complications:

a. Union rates about 94%
b. 100% of type III and IV develop arthritis
c. Neck fracture AVN rates: Highest risk is comminution and open injury
   i. Type II: 40%
   ii. Type III: 40-65%
d. No correlation between time of injury and time to surgery for closed injuries as far as arthritis, AVN, nonunion or AOFAS scores.
e. Hawkins sign: Good prognostic sign. Subchondral osteopenia seen at 6-8 weeks on plain radiographs and indicates revascularization of the body.

4 . CALCANEUS FRACTURES:

4.1. Epidemiology:

- Most frequently fractured tarsal bone (60% of all tarsal fractures).
- 75% of calcaneus fractures are intrarticular.

4.2. Mechanism of injury:
Intraarticular fractures: Axial load applied directly to the heel (falls from height, motor vehicle accidents.
Extraarticular fractures: Twisting or avulsions.

4.3. Radiographic Evaluation:
Lateral view of the foot and ankle: Böhler angle (normal 20° to 40°)
AP and oblique views of the foot: can visualize the calcaneocuboid joint
Broden view: useful intraop to evaluate reduction of the posterior facet
Axial Harris view: widening, shortening and varus of the tuberosity fragment
CT scan is mandatory to understand the fracture morphology.

4.4. Classifications:
a. Essex-Lopresti
b. AO (81.2) A: extrarticular. B: intrarticular. C: fracture-dislocation
c. Sanders: Type I: nondisplaced. Type II: posterior facet is in two fragments.
Type III: posterior facet is in three fragments. Type IV: comminuted, more than 3 fragments.

4.5. Treatment:
Nonsurgical:
- Type I fractures
- Should be considered in diabetic patients, smokers, peripheral vascular disease and patients unable to understand the injury.
- Do not weight-bear for 10-12 weeks. Range-of-motion as soon as soft-tissue swelling allows.

Surgical:
- Goals: Anatomic restoration of joint surface and anatomic or near anatomic overall calcaneal morphology to restore height, width and biomechanics of hindfoot.
- Initial management:
  - Bulky dressing and splint and limb elevation. Beware of compartment syndrome (in up to 10% of patients).
- Timing for surgery: Until resolution of soft tissue injury (2-3 weeks normally). "Wrinkle" Test. Blisters epithelialized.
- What needs to be addresses acutely
  1. Skin at risk
     a. Tongue type
     b. Displaced Tuberosity: Joint Depression
  2. Open Injuries: Irrigate debride acutely. Wait for soft tissue envelope to mature before treating definitively
- Definitive treatment:
  - ORIF.
    - Extensile lateral L-shaped approach (full thickness flap)
    - No-touch retraction techniques (K-wires)
    - Lag-screws and bridging plates +/- bone graft or bone graft substitutes.
  - Percutaneous techniques (Essex-Lopresti maneuver) / less invasive sinus tarsi approach
    - May be advantageous in poor candidates for ORIF or in patients with poor soft-tissue envelope.
    - Best used for the Essex-Lopresti tongue-type
  - External Fixation
  - Primary subtalar arthrodesis
    - For the more several comminuted fractures (Sanders type 4)
    - Better results after previous ORIF.

4.6. Results and complications:
- Outcomes correlate with:
  a. Accuracy of reduction
  b. Severity of injury
- Complication rate up to 40%:
  a. Wound-related complications: Soft tissue and bone infection.
  b. Malunion: Widened heel, hindfoot varus, loss of heel height and lateral impingment with peroneal tendon pathology
  c. Post-traumatic subtalar arthritis

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Dr. Philippe Kopylov

Ortho Center Skåne
Malmö, Sweden
philippe.kopylov@med.lu.se

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 Head. 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 percutaneuos 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 radials, 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" in2004. 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).

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 evocated 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 evocated 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 examinated 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 Botelheiro the diagnosed scaphoid fracture has to be classified essentially to define the correct treatmenrt. 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 god 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 prolongated. 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

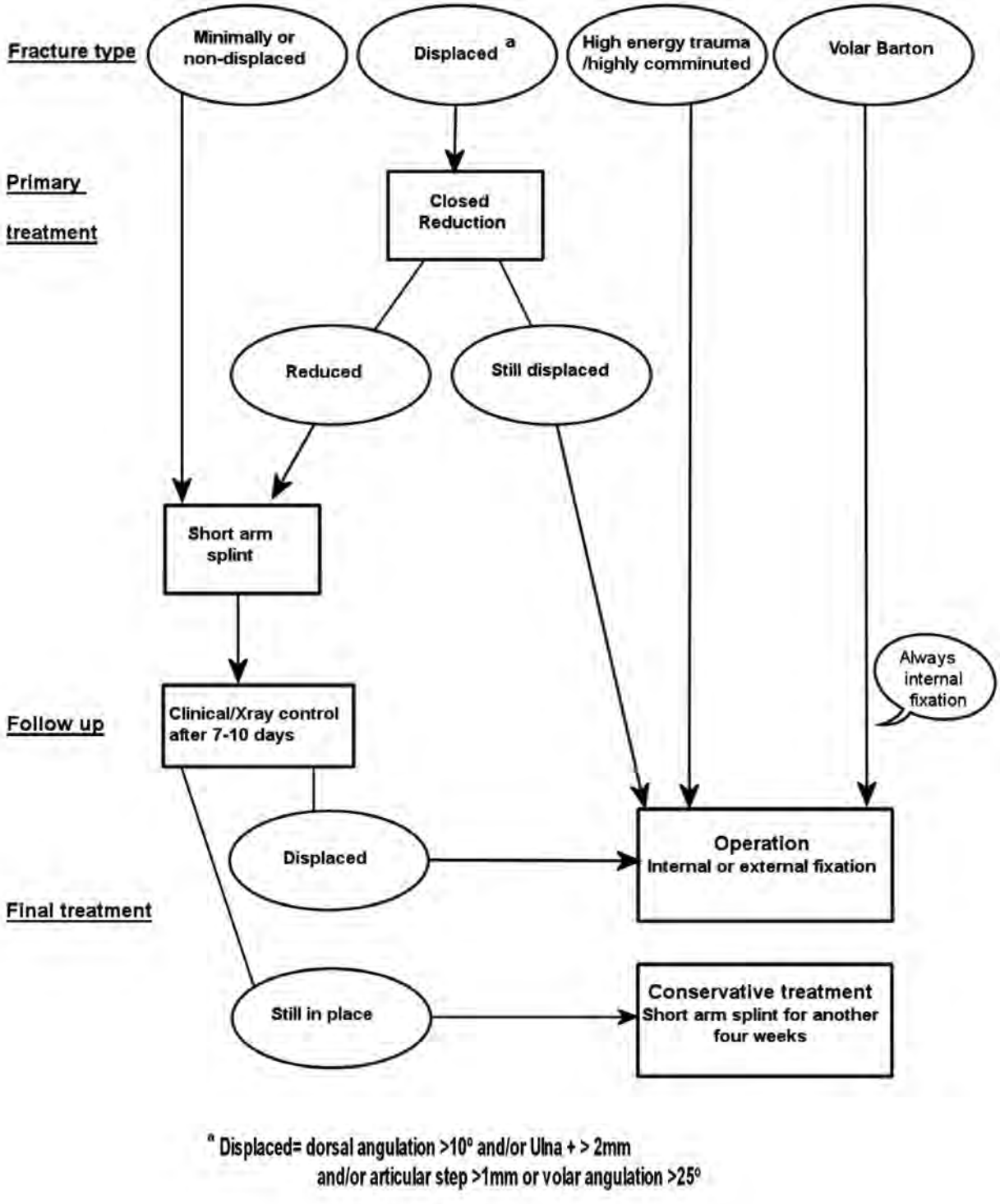

Fig. 1 The southern Sweden treatment protocol for DRF. When selecting different treatments the patient's age and demands also has to be accounted for.

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 are 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 theses cases, the scaphoid fracture be denied (Fig. 1).

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.

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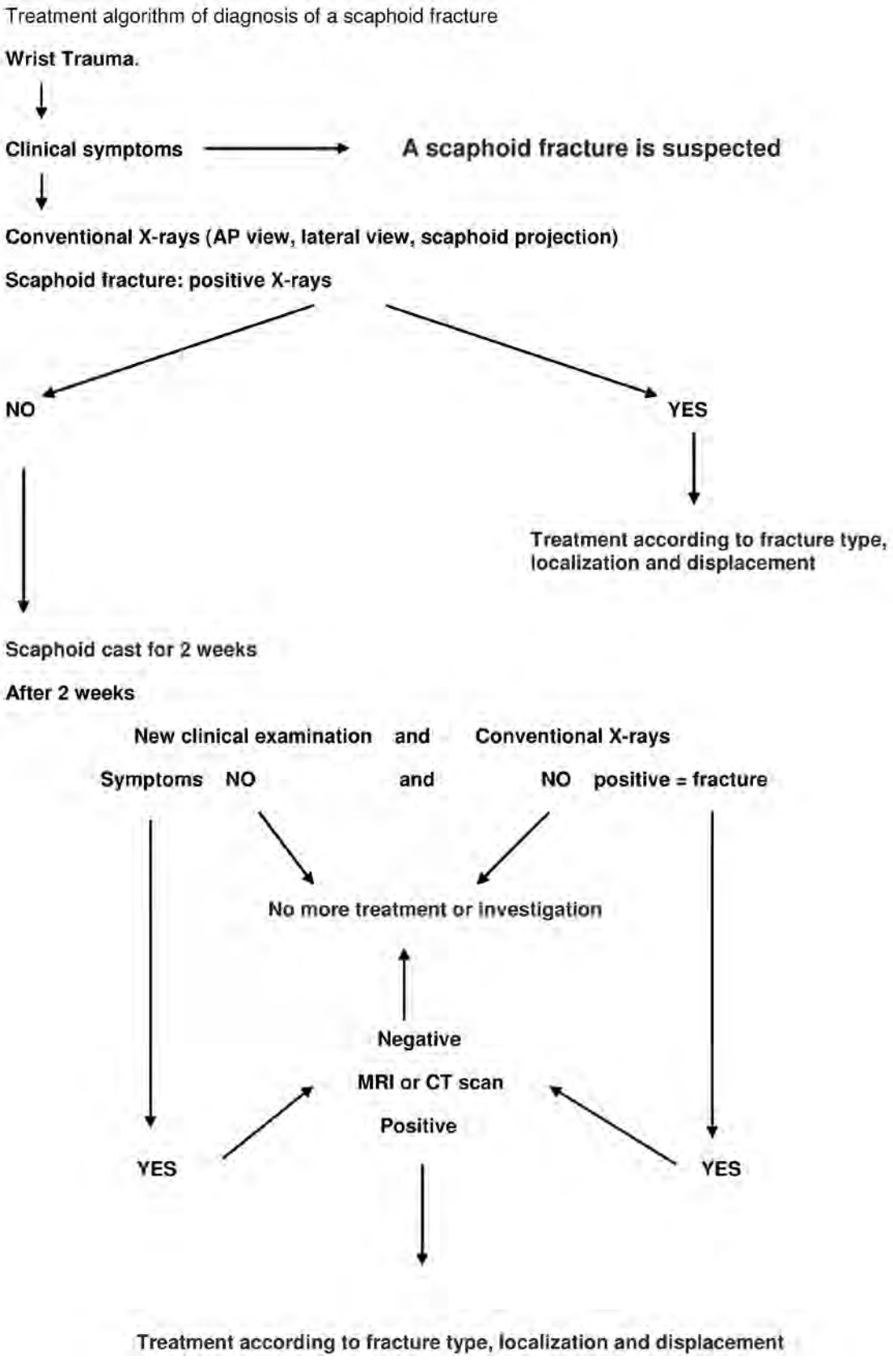

Fig. 2 Treatment algorithm of diagnosis of a scaphoid fracture

P. HOFFMEYER, Geneva

University Hospital
Geneva, Switzerland
pierre.hoffmeyer@hcuge.ch

Upper Limb Trauma

Shoulder Girdle Trauma

Clavicle fractures

Epidemiology: Clavicular fractures: 3-5% of all fractures and 35% to 45% of shoulder girdle injuries.
Mechanism: Falls, direct trauma, secondary to metastatic disease.
Clinical: Deformity, shortening of the shoulder girdle; neurovascular status assessed.
Diagnosis: AP X-ray of the clavicle and AP view of shoulder girdle. Rarely CT.
Treatment: depends on location

Mid-third

Undisplaced: Sling or figure of 8.
Relative indications for fixation: Displacement >100% in superior-inferior; > 2 cm of shortening; professional cyclist.
Absolute indications for fixation: Flail chest, scapulothoracic dissociation, fractures menacing skin or open fractures
Fixation: 3.5 mm reconstruction, dynamic compression anatomical plates with or without locked screws. Nails of different types have been advocated and reported to be successful by many authors. [1-4]

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Distal third

Neer I: Displacement > 100% superior-inferior; skin menace; open fracture: surgical fixation. Small distal fragment: simple excision. Large: Figure of 8 wiring with pins, osteosutures, specific plates or hook plates.
Neer II : Coracoclavicular fixation (sutures or screws).
Neer III: Symptomatic treatment, may need distal clavicle excision if arthritis sets in.

Proximal Third

Fixation: Major displacement, neurovascular or functional (tracheal or oesophageal) compromise [5-10]

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Complications of Clavicle fractures

Infections, nonunions (up to 30%), neurovascular compromise, reoperations >50% for hardware removal. [11-12]

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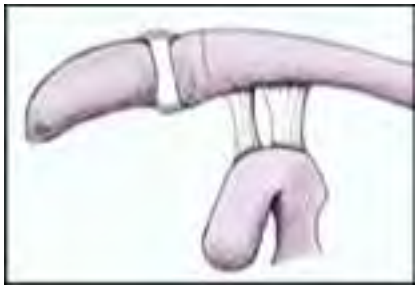
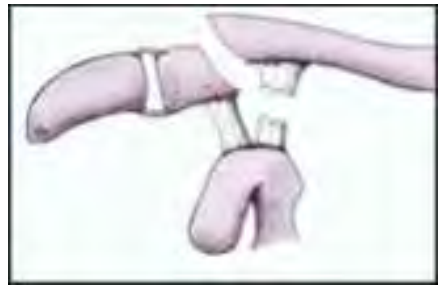
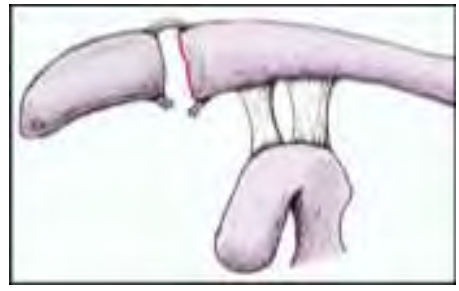


Neer Type I



Neer Type II



Neer Type II

Sternoclavicular dislocations

Incidence: 0.2% of all joint dislocations
Anatomy: Constraints ligamentous: Anterior capsular ligament posterior capsular ligament, meniscus. Costoclavicular and interclavicular ligaments: adjunct stability. [13-15]
Types:

- Antero-superior dislocation: avoid surgery, reassure.
- Postero-inferior dislocation: potentially life threatening (dyspnea, dysphagia, vascular compromise or thrombosis). CT for diagnosis. Closed reduction under general anaesthesia or surgical fixation (No pins or K-wires! Migration) t[13]

13. *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

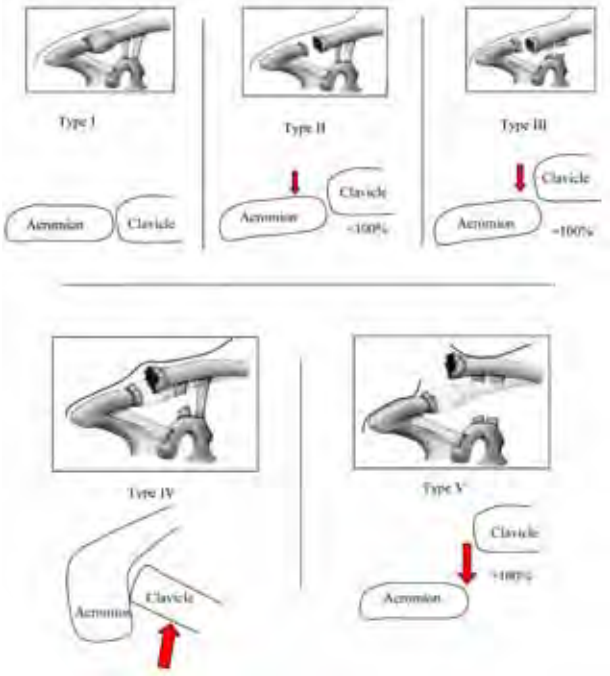
Incidence: 5.6% of all dislocations
Mechanism: Fall on tip of shoulder with acromion displaced inferiorly.
Clinical: Epaulette deformity, dermal abrasions.
Diagnosis: AP X-rays of the shoulder, Zanca views (10°-15° cephalic tilt) and axillary views. Stress views are not necessary.
Classification (Rockwood):

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% sup-inf displacement with tearing of AC and CC ligaments and overlying trapezius muscle.

Treatment:

- Conservative: Types I and II.
- Surgical: Types IV and V.
- Controversial: Type III.

Surgical techniques: Coraco-clavicular screws, CC and AC heavy sutures or tapes, transarticular pinning, endoscopically assisted CC and AC repairs.
Chronic cases: Weaver-Dunn procedure, Hook plates (Need removal), Dewar Barrington (Osteotomized coracoid fixed to distal clavicle) [14-16]



14. *The Shoulder, Fourth Edition. Editors CA Rockwood, FA Matsen, MA Wirth, SB Lippitt. 2009, Philadelphia, Saunders Elsevier.*
15. *AAOS Comprehensive Orthopaedic Review. Jay R. Lieberman, MD, Editor 2009 Rosemont, IL, American Academy of Orthopaedic Surgeons*
16. *Hindle P, Davidson EK, Biant LC, Court-Brown CM. Appendicular joint dislocations. Injury. 2013;44(8):1022-7. 21.*

Scapulothoracic dissociation

Mechanism: Major high energy injury.
Clinical: Loss of muscular attachments of the scapula to the thoracic wall. Internal amputation with neurovascular damage. Injury to major neurovascular structures, and thorax.
Diagnosis: Lateral displacement of scapula on AP chest X-Ray.Fracture or dislocation of shoulder girdle (AC, Clavicle, SC);
Outcome: Loss of the upper extremity or death. [17]

17. *Ebraheim NA, An HS, Jackson WT, Pearlstein SR, Burgess A, Tscherne H, Hass N, Kellam J, Wipperman BU. Scapulothoracic dissociation. J Bone Joint Surgery Am, 1988;70,428-432,*

Scapula fractures

Incidence: 0.3% of all fractures
Mechanism: High energy trauma with 80 to 95% incidence of associated trauma 50% of which are thoracic trauma. Mortality is 10% to 15% due to associated thoracic and cranial injuries.

Body Fractures

Mechanism: Direct trauma
Clinical: Neurovascular assessment, thoracic examination
Diagnosis: Plain X-Ray, CT with 3D reconstruction.
Classification: Ideberg
Clinical: Shoulder pain
Treatment: Conservative as a rule [18-21]

18. *Ideberg R, Grevsten S, Larsson S. Epidemiology of scapular fractures. Incidence and classification of 338 fractures. Acta Orthop Scand. 1995;66(5):395-7.*
19. *Schofer MD, Sehrt AC, Timmesfeld N, Störmer S, Kortmann HR. Fractures of the scapula: long-term results after conservative treatment. Arch Orthop Trauma Surg. 2009.*
20. *Lapner PC, Uhthoff HK, Papp S. Scapula fractures. Orthop Clin North Am. 2008 ;39(4):459-74, vi.*
21. *Lantry JM, Roberts CS, Giannoudis PV. Operative treatment of scapular fractures: a systematic review. Injury. 2008;39(3):271-83..*

Glenoid Fractures

Mechanism: High energy trauma.
Clinical: Pain on mobilisation, glenohumeral dislocation
Diagnosis: Plain X-Rays, CT, Occasionally MRI for soft tissues.
Treatment: Absolute indication if humeral head dislocated or subluxed. For displaced glenoid neck fractures clavicle fixation aids to reduce and stabilize. [22-24]

22. Goss TP. Fractures of the glenoid cavity. J Bone Joint Surg Am. 1992;74-A:299-305.
23. *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

24. *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.*

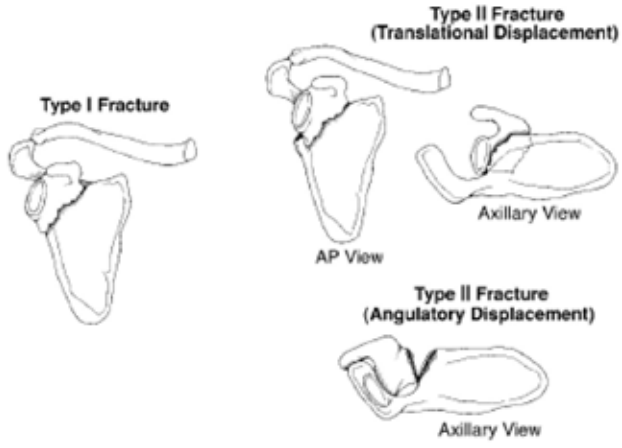
Acromion and Spine Fractures

Mechanism: Direct trauma after a fall or impact.

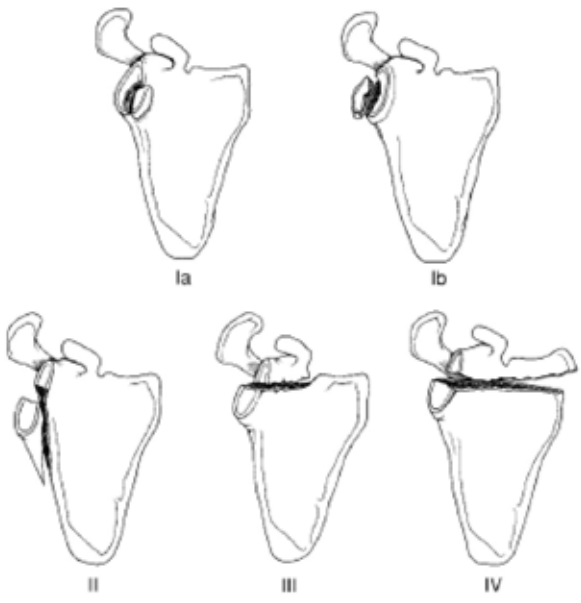
Clinical: Tenderness and pain on palpation and mobilisation of the shoulder

Treatment: For displaced fractures fixation or tension band fixation to counteract deformity due to deltoid tension and pull. [25]

25. *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: TP Goss. J Am Acad Orthop Surg 1995;3:22-33



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

Incidence: 32.5% of all joint dislocations, which represents 50/10000 population/year.

Dislocation : Complete loss of contact between two joint surfaces.

Subluxation: Partial loss of contact.

Laxity: Clinical examination with sulcus and drawer signs accompanied by excessive passive range of motion.

Instability: Subjective sensation of joint giving way.

Pathoanatomy:

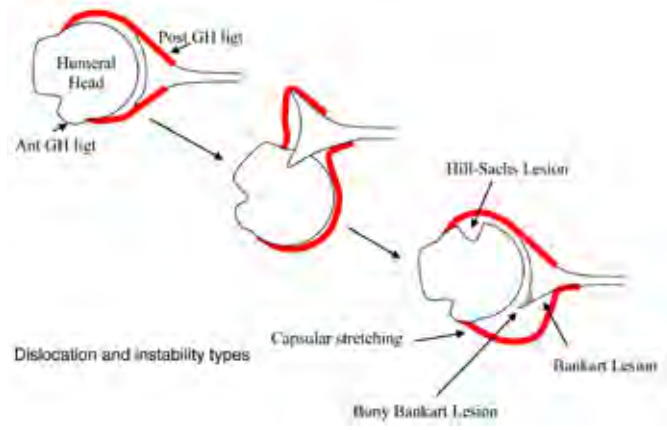
- Traumatic anterior instability

- Stretching, tear or detachment of the antero-inferior glenohumeral ligament.
- Cartilaginous damage to antero-inferior glenoid surface
- Bankart lesion: Detachment with or without a bony fragment of the antero-inferior capsulo-ligamentous complex.
- Hill-Sachs lesion: Impaction fracture of the greater tuberosity
- Stretching injury to the axillary nerve and plexus.
- Damage (rare) to the axillary artery.

- Traumatic posterior instability

- Tear or detachment of the inferior glenohumeral ligament.
- Cartilaginous damage to antero-inferior glenoid surface.
- Stretching of the posterior capsulo-ligamentous complex.
- Reverse Bankart lesion: Detachment with or without a bony fragment of the posterior capsule.
- Reverse Hill-Sachs impaction anteriorly at the region between articular surface and lesser tuberosity (may lead to a head-split fracture).
- Stretching injury to the axillary nerve and plexus.
- Damage (rare) to the axillary artery.

Anterior dislocation injuries



- Anterior dislocation

Mechanism: Related to sports activities (soccer, skiing etc.) or falls.

Recurrence: Recurrence rates are high in patients below 20 yrs (up to 90%), between 20 and 40 yrs 60% recurrence rates, above 40 yrs 10%.

Clinical :

Acute: pain and inability to move upper limb. Axillary nerve injury in 5%.

Chronic: Apprehension in abduction and external rotation.

Generalized laxity: Antero-posterior drawer, inferior sulcus sign, joint hyperlaxity (fingers, thumb, elbow).

Imaging: AP and axillary views. Arthro-CT: Bony lesions, capsular detachment. MRI: tendon and soft tissue injuries.

Treatment acute dislocations
AFTER diagnostic X-Rays and neurovascular testing.
Reduction techniques:
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: Intra-articular injection of lidocaïne or equivalent.
Davos: Patient sitting on a stool, joins hands with crossed fingers around his flexed knee, elbows extended, slowly leans backwards.
Hippocrates technique: Anaesthesia, traction on the arm with foot in the axilla or using folded sheets.
Postreduction treatment: Immobilisation 2 to 4 weeks, strengthening exercises. (NB: No proof that external rotation is superior)

Treatment for recurrent dislocations
Surgical indication: One episode of dislocation too many, severe apprehension.
Post-op recurrence rates: 5% to 30%.
Techniques: Capsulorraphy, Bankart refixation, bony augmentation (Latarjet).
Arthroscopic techniques: Traumatic Bankart lesions, remplissage for major Hill-Sachs lesions.
Open techniques: Capsular stretching, bony augmentation, remplissage for large Hill-Sachs lesions.
Rehabilitation: 3 to 6 weeks immobilization in internal rotation; muscular strengthening; range of motion exercises.
Avoid contact sports for one year.

- Posterior dislocation

Incidence: 5-10% of all glenohumeral dislocations.
Mechanism: Fall on outstretched hand, seizures or electrical shocks are the main causes.
Diagnosis: Beware. Many are primarily missed. Cardinal sign: active and passive limitation of external rotation.
Imaging: AP and axillary X-rays, CT.

Treatment for acute posterior dislocations
Small reverse Hill-Sachs: gentle traction and immobilize in an external rotation splint for three to 6 weeks with a rehabilitation programme to follow.
Large reverse Hill-Sachs lesion: Reduction under anaesthesia. If unstable: McLaughlin procedure (Anterior deltopectoral incision the head is levered out and reduced; Subscapularis or osteotomized lesser tuberosity sutured or screwed into bony defect). External rotation immobilisation 4 to 6 weeks followed by a rehabilitation programme.

Treatment for recurrent posterior dislocations
No major Hill-Sachs lesion: Posterior approach with a cruciate capsulorraphy and fixation of the reverse Bankart lesion is performed with or without bony augmentation.
Major Hill-Sachs lesion: McLaughlin procedure and if insufficient an adjunct posterior procedure capsulorraphy or bony augmentaion.

Multidirectional dislocation
Clinical: Young patients painful, lax and unstable in more than one direction, i.e. anterior and posterior or posterior and inferior or all three.
Signs: hyperlaxity, sulcus sign and anterior and posterior drawer signs causing discomfort or apprehension.
Diagnosis: Clinical, Standard X-rays, arthro-CT or MRI
Treatment: One year of muscle strengthening and stretching.
Surgery: Only after failure of conservative treatment. Capsular shift (Neer) through an anterior deltopectoral approach may need an adjunct posterior approach.
Aftertreatment: 6 weeks immobilisation in neutral (handshake) rotation followed by muscle strengthening.

Chronic dislocation
Debilitated patients: Abstention, reverse prosthesis, arthrodesis.

Recurrent dislocation in the elderly patient
Massive rotator cuff tear present.
Treatment: Tendon repair, reverse prosthesis, arthrodesis.

Wilful dislocation.
Rehabilitation, psychiatric help, avoid surgery. [26-29]

26. *The Shoulder, Fourth Edition. Editors CA Rockwood, FA Matsen, MA Wirth, SB Lippitt. 2009, Philadelphia, Saunders Elsevier.*
27. *AAOS Comprehensive Orthopaedic Review. Jay R. Lieberman, MD, Editor 2009 Rosemont, IL, American Academy of Orthopaedic Surgeons.*
28. *Shoulder Reconstruction. CS Neer. W.B. Saunders Company (January 1990)*
29. *Hindle P, Davidson EK, Biant LC, Court-Brown CM. Appendicular joint dislocations. Injury. 2013 Aug;44(8):1022-7.*

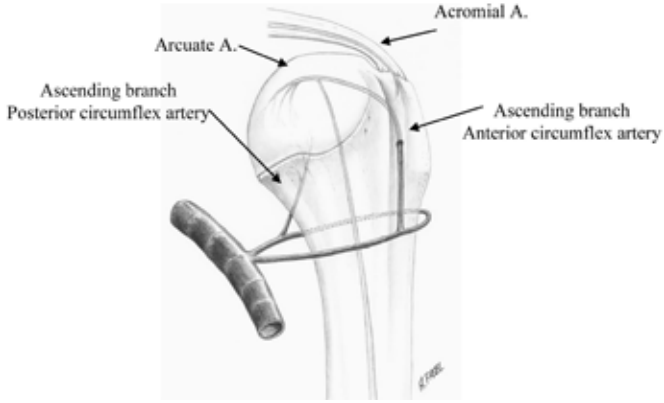
Proximal humerus fractures

Epidemiology: Proximal humerus 5% of all fractures or 60/10000 population/year.
Mechanism: High energy in young adults, low energy fall in osteopenic patients.
Surgical anatomy: Loss of sphericity of the humeral head, Impairment of rotator cuff to fine tune motion, scarring and stiffness of capsulo-ligamentous structures, interruption of terminal circulation of the humeral head leading to necrosis.
Clinical: Painful deformity, hematoma, neuro-vascular status
Diagnosis: AP and axillary views. CT and 3D CT useful, MRI useful to assess the cuff.
Classification: Neer, AO, Hertel, Duparc.
Treatment:
Conservative: Immobilisation for three to six weeks in a shoulder immobilizer or a Velpeau type bandage followed by physiotherapy.
Surgery indicated: Displacement of >1cm and 45°, neurovascular involvement.
Technique:
2 and 3 part factures: Osteosynthesis (nail, locking plate, osteosuture)
4 part and head-split: Prosthesis (Anatomic or reverse controversy)
Complications: Avascular necrosis, mal or non unions, stiffness and postoperative sepsis plague the treatment results. [30-36]

30. *Neer CS II: Displaced proximal humeral fractures: Part I. Classification and evaluation. J Bone Joint Surg Am 1970;52:1077-1089*
31. *AO Principles of Fracture Management. Second expanded edition, TP Rüedi, RE Buckley, CG Moran. Georg Thieme Verlag; 2 Har/Dvdr edition (2007).*
32. *Hertel R. Fractures of the proximal humerus in osteoporotic bone. Osteoporos Int. 2005;16 Suppl 2:S65-72.*

33. *Duparc J. Classification of articular fractures of the upper extremity of the humerus. Acta Orthop Belg. 1995;61 Suppl 1:65-70.*
34. *The Shoulder, Fourth Edition. Editors CA Rockwood, FA Matsen, MA Wirth, SB Lippitt. 2009, Philadelphia, Saunders Elsevier*
35. *AAOS Comprehensive Orthopaedic Review. Jay R. Lieberman, MD, Editor 2009 Rosemont, IL, American Academy of Orthopaedic Surgeons.*
36. *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.*

Vascular anatomy of the Humeral Head



Humeral shaft fractures

Epidemiology: 2-3% of fractures distributed in a bimodal mode. High energy fractures in younger patients and low energy fractures in the elderly osteoporotic individual.

Mechanism: Torsion, blunt trauma, falls, gunshot wounds, hang gliders, arms wrestling, pathologic fractures metastases (8% of humeral fractures), chronic osteomyelitis. [37-39]

37. 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.
38. 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.
39. Frassica FJ, Frassica DA. Metastatic bone disease of the humerus. J Am Acad Orthop Surg. 2003;11(4):282-8.

Surgical anatomy: Radial nerve runs 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. [40-41]

40. *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.*
41. *AAOS Comprehensive Orthopaedic Review. Jay R. Lieberman, MD, Editor 2009 Rosemont, IL, American Academy of Orthopaedic Surgeons.*

Classification : AO classification
type A (simple, transverse or spiral)
type B (wedge with a butterfly fragment)
type C (segmental or comminuted fragments).
Open fractures (Gustilo and Anderson): Type I inside-out (< 1 cm)
Type II outside-in (> 1cm)
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).
[42-44]

42. *AO Principles of Fracture Management. Second expanded edition Thomas P Rüedi, Richard E Buckley, Christopher G Moran. Georg Thieme Verlag; 2 Har/Dvdr edition (2007).*
43. *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.*
44. *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: Deformed extremity, neurovascular injuries (radial nerve).

Imaging: Plain X-rays including the shoulder and elbow are generally sufficient in acute traumatic cases. MRI, CT, Bone scintigraphy for chronic infection, metastatic or primary tumors.

Conservative treatment: Immobilisation in Velpeau type bandage after two to three weeks functional brace. Planar angulations of 20° sagittally and 15° frontally, malrotations up to 15°, and shortening up to 3 cm acceptable. Varus angulation:16%>10°-20° most common complication. [45-46]

45. *Sarmiento A, Latta LL. Humeral diaphyseal fractures: functional bracing. Unfallchirurg. 2007;110(10):824-32.*
46. *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 treatment: 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.

Antero-lateral approach: radial nerve identified between brachialis and brachioradialis. [47]

47. *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: Radial nerve 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. [48]

48. *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

IM Nailing

Minimally invasive, control of rotation is and locking bolts. Non-unions and shoulder pain are more common complications.

- Anterograde
  Adequate point of entry through the cartilaginous surface of the head minimizing injury to rotator cuff.
- Retrograde
  Entry point above olecranon fossa to avoid fragilizing distal humerus. Not suited for distal fractures.

Plating

Lateral or posterior approach, plates adapted to anatomy (broad plates

in a large bone, narrow plates in a small bone). Minimally invasive approaches with incisions proximally and distally (radial nerve) allowing closed plate insertions. Locked screws useful in osteoporotic bone. Union rates > 94% with plating.

External fixation

Polytrauma, open fractures. Avoid injuring nerves: Open placement of pins distally. [49-51]

49. *Bhandari M, Devereaux PJ, McKee MD: Compression plating versus intramedullary nailing of humeral shaft fractures—A meta-analysis. Acta Orthop 2006;77:279-284.*
50. *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*
51. *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.*

Elbow fractures

Surgical pathoanatomy

Arch held by two columns.

Trochlea: Cartilage covered in a 300° arc.

Distal humerus: Flexed 30° anterior, tilted 6° in valgus and rotated medially 5°.

Capitellum : Half sphere covered anteriorly with cartilage.

Radial head: Articulates with proximal ulna and distal humerus. Head -neck angle 15° in valgus; head asymmetric with 240° coverage of articular cartilage leaving 120° of non cartilage covered area amenable to hardware fixation.

Surface of coronoid process equivalent to radial head.

Carrying angle: 11°-14° in men and 13°-16° in women.

Elbow range of motion: 0° extension, 150° flexion, pronation 75°, supination 85°.

Primary static stabilizers: Ulno-humeral articulation, collateral ligaments.

Secondary static stabilizers: Capsule, radiohumeral articulation, common flexor and extensor tendon origins.

Dynamic stabilizers: Muscles crossing the elbow (Anconeus, triceps, brachialis).

All forces that cross the elbow joint are directed posteriorly. [52]

52. *The Elbow and Its Disorders, Editor BF Morrey, 4th Edition, Elsevier 2008.*

Fractures of the distal humerus

Incidence: 2% of all fractures, but represent a 1/3 of all elbow fractures. 6th decade and associated with osteoporosis.

Clinical: Deformity, neurovascular, compartment syndrome.

Imaging: Plain X-ray, Traction x-rays (Anesthetized patient), CT, 3D CT.

Classifications AO:

Type A: Extra-articular

Type B: Partial intra-articular

Type C: Complete intra-articular

Treatment: Unstable frequently comminuted fractures needing surgical stabilisation as a rule.

AO surgery reference: http://www.aofoundation.org.

Surgical 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 supinatorus 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 with extensile exposure of the distal humerus (Fractures distal humerus, arthroplasty, stiff elbow):
  - Bilaterotricipital approach (Alonso-Llames) 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. [53-57]

53. *The Elbow and Its Disorders, 4th Ed, Elsevier 2008 Ed. Morrey BF.*
54. *Fracture of the Anteromedial Facet of the Coronoid Process. Surgical Technique. Ring D, Doornberg JN. J Bone Joint Surg Am. 2007;89:267-283.*
55. *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.*
56. *AO surgery reference: http://www.aofoundation.org.*
57. *Surgical Exposures in Orthopaedics: The Anatomic Approach. Hoppenfeld S, deBoer P,Buckley R.Lippincott Williams & Wilkins; Fourth Edition edition 2009.*

- Capitellum fractures: Lateral approach. Diagnosis on lateral X-Ray, repositioned and fixed with two posterior to anterior small fragment 3.5 mm lag screws or with Herbert type screws.

- Epitrochlea: Direct medial approach. Isolated fractures and fixed with a lag screw. The ulnar nerve must be protected.
- Lateral column: 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 extra-articular : Posterior approach. Can be addressed through a bilaterotricipital Alonso-LLames or TRAP approach. Both columns are identified and fixed to the articular epiphysis using a lateral 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 intra-articular: Posterior approach. 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. One the joint surface has been reconstructed it is then possible using various types of implants to fix both columns. In general a 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. [58-64]

58. *Pollock JW, Faber KJ, Athwal GS.Distal humerus fractures. Orthop Clin North Am. 2008;39(2):187-200, vi*
59. *Bryan RS, Morrey BF: Extensive posterior exposure of the elbow. Clin Orthop Relat Res 1982;188-192. [58-62]*
60. *The Elbow and Its Disorders, 4th Ed, Elsevier 2008 Ed. Morrey BF.*
61. *Fracture of the Anteromedial Facet of the Coronoid Process. Surgical Technique. Ring D, Doornberg JN. J Bone Joint Surg Am. 2007; 89:267-283.*
62. *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.*
63. *AO surgery reference: http://www.aofoundation.org.*
64. *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. [65]

65. *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. Debride and wash out the wound and proceed with internal fixation as if it were a closed injury. If not possible Ex-Fix and ORIF when soft tissue situation appropriate.

Fractures of the radial head

Incidence: 2 % of all fractures and 33% of all elbow fractures.
Mechanism: Fall on the slightly flexed outstretched elbow with the hand in supination.
Clinical: Immediate pain after a fall. Effusion with palpable fluctuation bulging outwardly through the radio-humeral joint. Prono-supination is painful or impossible. After intra-articular injection with lidocaïne smooth, non-grating, active or passive prono-supination signifies minimal displacement and non-operative treatment.

Classification:
Mason Classification:
Type I: Non-displaced
Type II: Displaced marginal fracture
Type III: Comminuted fracture
Type IV: Associated with elbow dislocation
Hotchkiss modification:
Type I: No surgery
Type II: Displaced but fixable
Type III: Displaced and unfixable

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: begun early and motion should be started within days of the intervention in case of operative treatment or diagnosis in case of conservative treatment. An articulated brace is useful for protection in cases of instability.
The Essex Lopresti injury: Associates comminuted radial head fracture with tearing of the interosseous membrane and disrupting the DRUJ. The radial head fixed or replaced with a prosthesis and the DRUJ stabilized with cross pin left in situ for 4 to 6 weeks. [66]

66. *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.*
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

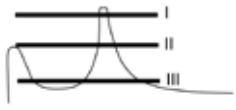
Incidence
Clinical: Fall on the tip of the elbow, palpable gap, unable to actively extend.
Imaging: Plain x-rays
Classifications

Mayo
Type I: Undisplaced
Type II: Displaced but stable elbow (Noncomminuted: A / Comminuted: B)
Type III: Displaced and unstable elbow (Noncomminuted: A / Comminuted: B)

AO
A: Extra-articular fractures
B: Intra-articular fractures
C: Fractures of both olecranon and radius

Treatment: Surgical.
Technique: Direct approach, Tension band wiring, plate reconstruction.
Rehabilitation: backslab at 80° of flexion and gentle active flexion and extension exercises are started as tolerated for 6-8 weeks.

Coronoid fractures



Clinical: Associated with elbow dislocations.
Imaging: Plain x-rays, ct, 3D CT
Classification (Regan and Morrey)

Type I: Fracture of the tip
Type II: < 50% of the height of the coronoid
Type III: > 50%
Type IV: Fracture of the sublime tubercle.
A and B types signify no or associated dislocation.

Treatment: Type I and II stable no fixation. Types III and IV need fixation: Medial collateral ligament attaches to medial coronoid and risk of instability .
O'Driscoll has modified this classification

Type 1: Tip fracture
Type 2: Anteromedial fracture
Type 3: Base of coronoid fracture

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. [67-69]

67. *Regan W, Morrey B. Fractures of the coronoid process of the ulna. J Bone Joint Surg Am 1989;71:1348–1354.)*
68. *O'Driscoll SW, Jupiter JB, Cohen MS, Ring D, McKee MD. Difficult elbow fractures: pearls and pitfalls. Instr Course Lect 2003;52:113–134.*
69. *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.*

Dislocation of the elbow

Incidence: 3.5% of all joint dislocations
Mechanism: Fall on the slightly flexed elbow and wrist in supination.
Clinical: Pain and deformity. Median nerve injury frequent.
Imaging: Plain x-rays.
Treatment: Reduction with sedation or anesthesia. Surgical exploration if interposed fragments or soft tissues.
Rehabilitation: Place elbow in splint. No immobilisation longer that 7 days. Start at 7 days with active-assisted flexion extension and at three weeks with prono-supination.
Complications: Heterotopic bone, stiffness, neurovascular injury. [70]

70. *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

Clinical: Throwing athletes, pain and "pop" medially while throwing. Tinel's sign for ulnar nerve.
Imaging: Stress x-rays, MRI, dynamic ultrasound. [71]

71. *Treatment: Medial collateral ligament reconstruction (Fascia lata, Palmaris longus), - Safran M, Ahmad CS, El Attrache NS. Ulnar collateral ligament of the elbow. Arthroscopy 21:1381, 2005.*

Postero-lateral rotatory instability of the elbow

Clinical: After elbow dislocation, instability sensation. Clinical testing : Elbow stressed.
Diagnosis: Stability test in in valgus and supination.
Imaging: Plain x-ray, MRI, CT Arthrographoy.
Treatment: Tendon graft uniting the humerus to the supinator crista of the ulna and passing under the radial head. [72]

72. 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.

Distal Biceps Tendon Ruptures

Clinical: Weakness in prono-supination, palpable muscle ball above elbow crease.
Imaging: Ultrasound, MRI.
Treatment: Young and active tendon suture. Two incision Morrey technique, suture to brachialis.
Rehabilitation: 6 weeks in a back-slab. Gentle Flexion-extension excercises. Full return to function after 6 to 8 weeks.
Complications: Suture failure, radial nerve damage, infection.
Rehabilitation: Gentle flexion-extension exercises follow the surgery and at 6 weeks a full return to activity is permitted. [73]

73. Papandrea RF: Two-incision distal biceps tendon repair, in Yamaguchi K, King GJWt, McKee O'Driscoll SW : Advanced Reconstruction Elbow. Rosemont, IL, American Academy of Orthopaedic Surgeons, 2006, pp 121-128.

Brachialis and triceps rupture
Clinical: Rare injuries, palpable gap, flexion or extension deficit.
Imaging: Ultrasound, MRI.
Treatment: Surgical suture repair indicated.

Lateral Epicondylitis (Tennis Elbow)

Mechanism: Overuse causing chronic tearing of the extensor carpi radialis brevis tendon at its distal humerus insertion.
Clinical: Pain lateral aspect elbow at rest and on wrist extension.
Diagnosis: Wrist or long finger extension against resistance.
Differential diagnosis: Radio-humeral arthrosis, radial nerve entrapment, carpal tunnel, cervical disc hernia.
Imaging: Plain x-ray, MRI.
Treatment: Conservative, surgical if non responding.
Technique: Open or arthroscopic surgical excision of the ECRB tendon origin, situated under the Extensor Carpi Radialis Longus tendon.
Rehabilitation: Protective splint followed by gentle motion as tolerated with full function possible 6 to 8 weeks postoperatively. [74-75]

74. 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.

75. Nirschl RP, Pettrone F. Tennis elbow: The surgical treatment of lateral epicondylitis. J. Bone Joint Surg Am. 1979; 61:832.

Medial Epicondylitis

Incidence: Rare
Mechanism: competitive athlete overuse of the flexor-pronator complex.
Clinical: Pain on activity and at rest.
Imaging: X-ray, MRI.
Treatment: Conservative, Surgical excision of the diseased part of the medial conjoint tendon of the flexor-pronator complex with transposition of the ulnar nerve. [76]

76. 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

Incidence: Rare, mostly skeletally immature patients, throwing sports,
Clinical: Pain, flexum deformity or catching and locking
Classification: Simple cartilage fissures (I) to detachment of large fragments (IV)
Imaging: Plain x-rays, CT, MRI.
Treatment: conservative, simple drilling of the lesion, complex mosaicplasty. [77]

77. 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.

Forearm fractures

Introduction: Forearm a whole functioning joint pronation of 75°,supination of 85°. Interosseous membrane major stabiliser.
Incidence: 1.2% of fractures
Mechanism: Direct trauma (nightstick fracture), fall, polytrauma.
Classification: AO

- type A (simple, transverse or spiral)
- type B (wedge with a butterfly fragment)
- type C (segmental or comminuted fragments).

Open fractures: Gustilo and Anderson:

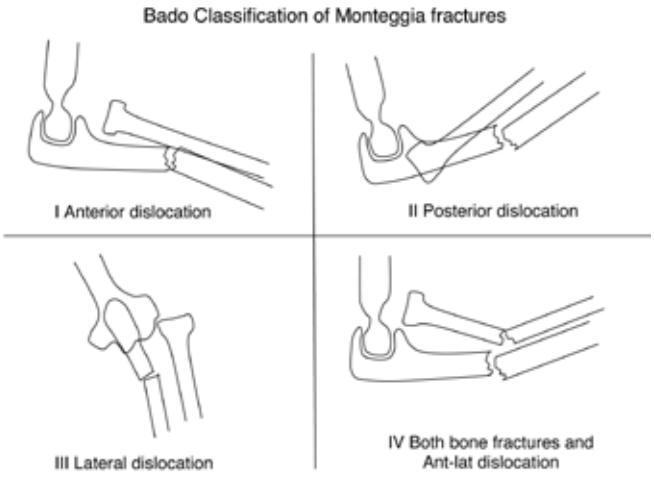
- Type I inside-out (< 1 cm).
- Type II outside-in (> 1cm).
- Type III A (open but osseous coverage possible).
- Type III B (open necessitating a local or free flap).
- Type III C any open fracture with vascular injury.

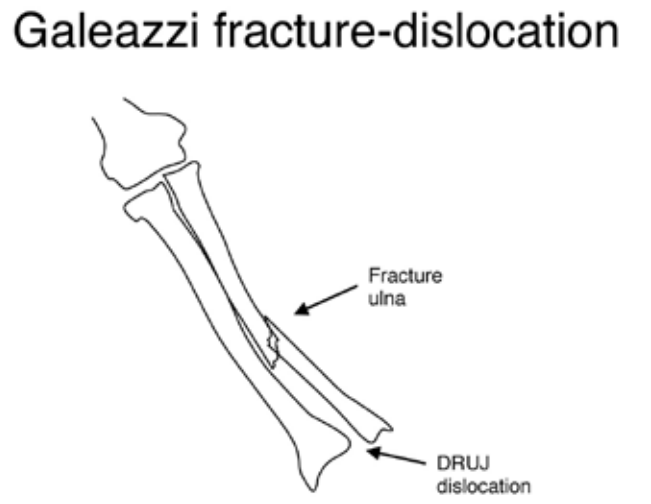
Monteggia fracture pattern (Fracture of the ulna with dislocation of the radial head):
Bado classification:

- 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)

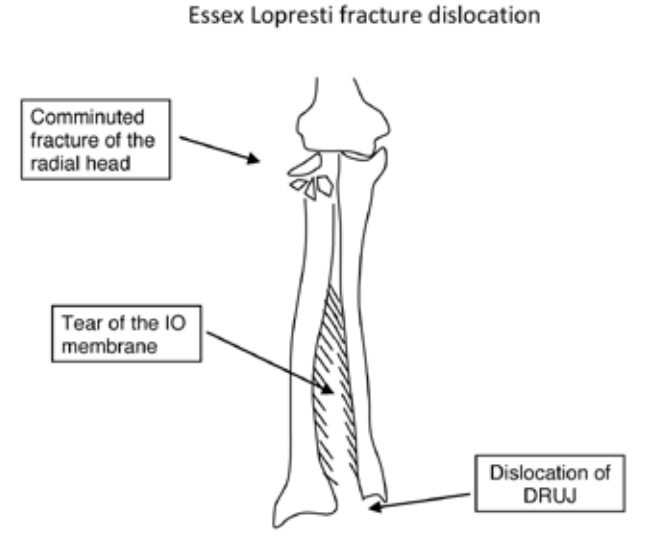
[78]

78. Konrad GG, Kundel K, Kreuz PC, Oberst M, Sudkamp NP. Monteggia fractures in adults: long-term results and prognosticc factors. J Bone Joint Surg Br. 2007;89(3):354-60.





Galeazzi fracture pattern: radius shaft fractured with dislocation of the distal radio-ulnar joint (DRUJ).



Essex-Lopresti fracture dislocation.
Combination of a comminuted radial head fracture and a tor interosseous membrane.
Imaging: Plain x-rays, CT, 3D CT, MRI for interosseous membrane injury.
Treatment: In all cases ORIF of radius and ulna. (Isolated fractures of the ulna: attempt at conservative, recommende ORIF).
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.

- Surgical approaches to the forearm:

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). [79]
79. www.wheelessonline.com/ortho/dorsal_approach_thompson

Direct approach
The direct approach is best suited for the ulna. [80]
80. 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.

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.

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. [81-83]
81. Hertel R, Pisan M, Lambert S, Ballmer FT. Plate osteosynthesis of diaphyseal fractures of the radius and ulna. Injury. 1996;27(8):545-8.
82. 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.
83. 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.
Complications: Non-union, malunion, compartment syndrome, infection. [84]
84. Rettig ME, Raskin KB. Galeazzi fracture-dislocation: a new treatment-oriented classification. J Hand Surg Am. 2001;26(2):228-35.

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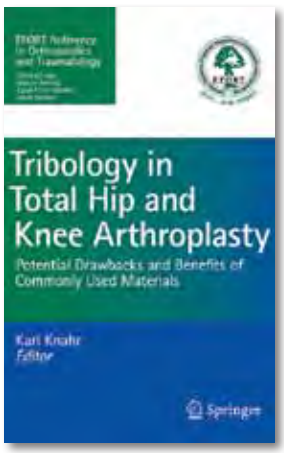

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