

CRC Course
Vienna, 02 June 2017

www.efort.org/crc2017



EFORT SYLLABUS

The Comprehensive Orthopaedic Review Course

During the 18th EFORT Congress Vienna: 02 June 2017

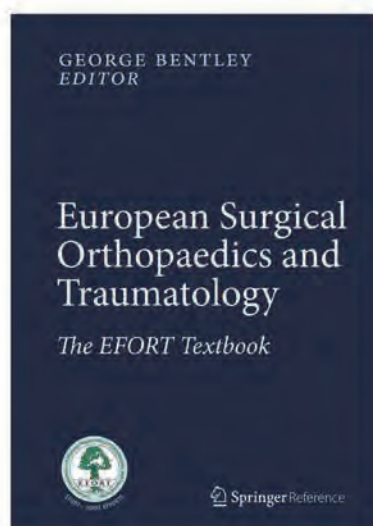
🐦 #CRC

Course highlights

- Basic Science
- Paediatrics
- Tumour
- Spine (incl. Trauma)
- Reconstruction
- Sports/knee
- Trauma
- Sport Activities & Orthopaedic Practice



European Surgical Orthopaedics and Traumatology



Edition 2014, 4983 pages.
3325 illus., 2033 illus. in color.
7 volumes, not available separately.

SPRINGER REFERENCE

- **Print (Book)**
549.00 € | £494.50 | \$749.00
*587.43 € (D) | 603.90 € (A) | CHF 731.00
- **eReference**
549.00 € | £494.50 | \$749.00
*653.31 € (D) | 658.80 € (A) | CHF 768.00
- **Print + eReference**
823.00 € | £612.00 | \$1,115.00
*913.20 € (D) | 921.76 € (A) | CHF 904.00



The EFORT Textbook

- Guides the reader through the total management of the patient, including surgical techniques
- Written by recognized international authorities
- Didactic style appropriate for those preparing for examinations
- Abundant illustrations highlighting the essentials of each clinical scenario

This important reference textbook covers the surgical management of all major orthopaedic and traumatological conditions.

The book will act as the major source of education and guidance in surgical practice for surgeons and trainees, especially those preparing for higher surgical examinations and the Board of Orthopaedics and Traumatology examinations within and beyond Europe.

The emphasis throughout is on the application of current knowledge and research to technical problems, how to avoid operative problems, and how to salvage complications if they occur. The didactic text is complemented by abundant illustrations that highlight the essentials of each clinical scenario.

The authors are all recognized international authorities active at congresses and workshops as well as in universities and hospitals across the world.

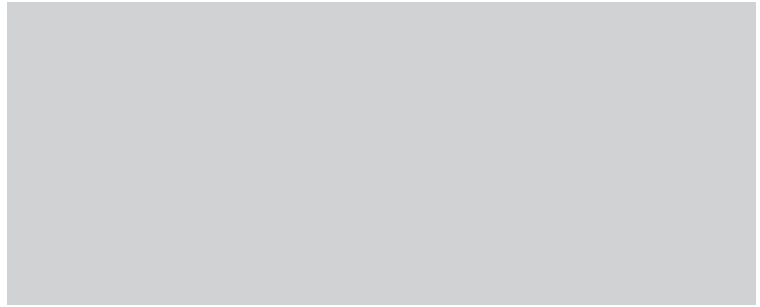
Editor: G. Bentley, Edition 2014

Order online at www.springer.com

- For outside the Americas call +49 (0)6221 345 4301 | email: orders-hd-individuals@springer.com
- For the Americas call (toll free) 1-800-SPRINGER | email: ordersny@springer.com

The first € price and the £ and \$ price are net prices, subject to local VAT. Prices indicated with * include VAT for books; the €(D) includes 7% for Germany, the €(A) includes 10% for Austria. Prices indicated with ** include VAT for electronic products; 19% for Germany, 20% for Austria. All prices exclusive of carriage charges. Prices and other details are subject to change without notice. All errors and omissions excepted.

Welcome



Welcome to this 9th EFORT Comprehensive Review Course (CRC) on Friday 2 June during the 18th EFORT Congress in Vienna.

This course is a quick analysis 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.

Year after year, the Programme summarizes in a few pages, the elements which are presented during the course in a brief and easy way to understand.

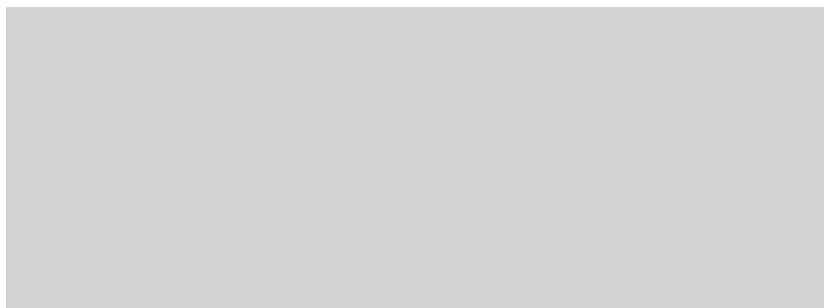
It starts with the fundamental knowledge of the basic sciences and general, such as tumour conditions, infections and metabolic diseases. Orthopaedics and trauma are reviewed by each subspecialty within the most important information from the fields of spine, paediatrics and then the key aspects of the trauma and orthopaedic in the upper and lower limb. Reconstructive surgery of bones and joints and sports medicine are treated succinctly without forgetting complications.

Experts in each field conduct the course and whom we would like to thank for their participation and effort in the preparation of this syllabus.

We wish to thank the collaboration of all the different authors of the CRC, the work of the Head Office staff and the EFORT Publications Committee in order to achieve these objectives.

Klaus-Peter Günther
Chairman EFORT Education Committee

Thierry Bégué
Chairman EFORT Science Committee



EYE OPENER	
Sport Activities Et Orthopaedic Practice.....	03
BASIC SCIENCE	
Biomechanics And Biomaterials For Musculoskeletal Application.....	05
Metabolic Bone Disease	11
PAEDIATRICS	
Classification And Principle Of Fractures Treatment In Childhood.....	18
Foot Disorders Of Newborns.....	21
Hip Diseases In The Childhood	27
RECONSTRUCTION	
Hip: Osteotomy And Arthroplasty	34
Knee: Osteotomy And Arthroplasty	39
Ankle Osteoarthritis, Adult Acquired Flatfoot Deformity and Hallux Valgus.....	47
Degenerative Diseases Of The Cuff, Shoulder And Elbow	54
SPORTS KNEE	
ACL, PCL, Collaterals And Meniscus.....	60
INFECTION	
Infections After Total Joint Arthroplasty.....	66
TUMOUR	
Diagnostic And Recognition Of Primary Bone Tumours.....	70
Diagnostic Algorithm And Treatment Options In Bone Metastasis.....	73
SPINE	
Paediatric Idiopathic Scoliosis.....	77
SPINE (INCLUDING TRAUMA)	
Degenerative Spine Diseases Et Spine Fractures	81
TRAUMA	
Fractures: Pelvic Ring And Acetabular Fractures.....	91
Fractures: Femur, Tibia And Open Fractures.....	94
Fractures: Pilon, Ankle, Talus And Calcaneus.....	98
Fractures: Hand And Wrist.....	103
Fractures: Shoulder, Arm, Elbow And Forearm	108



Dr José Neves Pinto

Orthopedic Surgery Department, Garcia de Orta Hospital,
Almada, Portugal

josenevespinto@gmail.com

Sport Activities & Orthopaedic Practice

Reading and evading a defense, organizing 15 men in attack, tackling, passing a rugby ball with 20.000 people cheering is not orthopedic surgery, but for both, excellence is due to dedication, preparation, training, teamwork and finally performance.

Aside from my parents, rugby has been the most formative experience of my life. Integrity, passion, solidarity, discipline and respect are core values shared between a lot of team sports, but the unique physicality of the game where you can run full speed against (or be hammered by...) your opponent creates a unique bond between present and past rugby players all around the globe.

In a time where sports, particularly contact sports, are under siege by the media due to their potential adverse effects to athlete's health, it is critical that we reflect on the broader meaning and positive values imparted on youth. I was easily the physically weakest person on the team and realized quiet early that success would not come easily. Each step-wise goal met, another goal set. Eventually I would exceed others' expectations, meet my own and even fulfill my dream of playing versus the All Black in a Rugby world cup match!

Lessons learned transcend practice fields, weight rooms, and stadiums and enable athletes to develop strategies for pursuing success and more importantly facing defeat to overcome adversity in future professional endeavors and interpersonal relationships.

From checklists to error-reporting, physicians and hospitals are implementing aviation safety techniques in health care. What about sports? What lessons from Medicine school did I take to the rugby field and made me a better player? What about surgical performance – what can we learn from sport? Personal and Professional success today as an orthopedic surgeon is driven by lessons learned on the rugby field.

Mobility as a vital part of being – its loss carries dire implications for patients. My mission today isn't earning playing time, rather it is restoring mobility. Returning an athlete to the field, enabling weekend warriors to play without pain, helping people return to work sooner, and allowing parents and grandparents enjoy time with their families. When challenged with a difficult clinical problem I draw on the drive that allowed me to become a Professional athlete. Every patient is a new line of scrum. Ready. Set. Go!

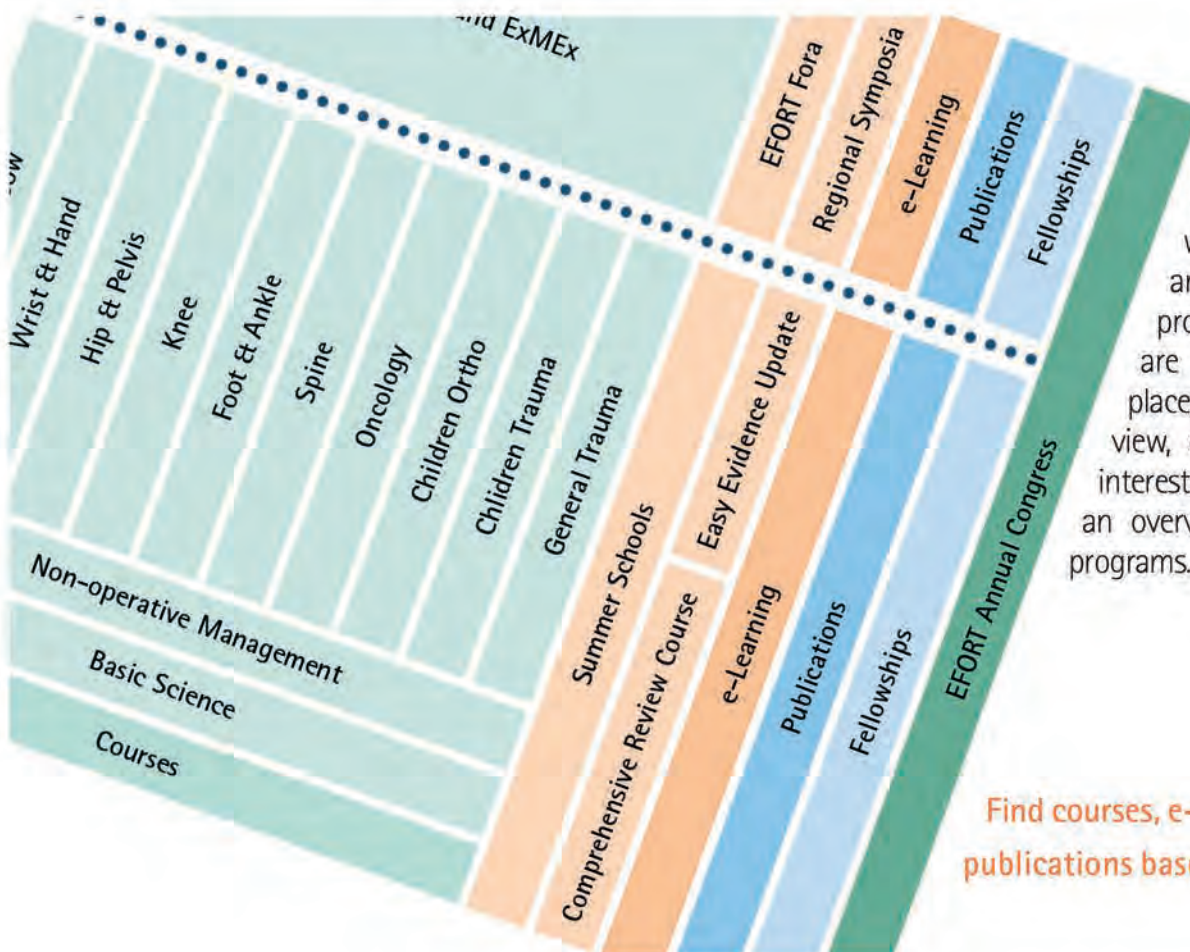
EFORT EOTEP Launch

www.efort.org/eotep



European Orthopaedics & Traumatology Education Platform

The European Orthopaedics & Traumatology Education Platform EOTEP proposes a new approach in the field of the lifelong learning for Orthopaedic & Traumatology Surgeons practicing in Europe and beyond.



The EOTEP offers a platform in form of a **roadmap** whereby different and already existing programs from Europe are compiled into one place, in a structured view, especially for those interested in looking for an overview of educational programs.

Find courses, e-learning activities, publications based on the syllabus!

"It was important for EFORT to let transpire the partnership vision with national and specialty societies, and to give both [...] an opportunity to join the dynamism of cross-border lifelong learning."

—Prof. Dr. Klaus-Peter Günther, chairman of the EFORT Education Committee



Prof. Elizabeth Tanner

University of Glasgow, Glasgow, Scotland
Skåne University Hospital, Lund, Sweden
elizabeth.tanner@glasgow.ac.uk

Biomechanics And Biomaterials For Musculoskeletal Application

1.0 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 enormous subjects in medicine and surgery, but only those areas of importance to orthopaedic surgery will be considered in this lecture.

2.0 Biomechanics

2.1 Newton's Laws

The mechanics of all stationary and 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 forces include those generated by gravity acting on the mass of the body and of individual elements (arms, legs etc). The mass of the person multiplied by the acceleration due to gravity is the force the person is applying to the floor. 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 100g is about 1N. The difference between weight and mass is that mass is the amount of material in something and usually measured in kg or g. On earth an 80kg astronaut on earth weighs 784.8N (Newtons), but in space there is zero gravity acting so their mass remains 80kg, but their weight is now 0N.

Someone standing still is "in a state of rest", so the forces acting on the body, both horizontally and vertically, must balance. However if they want to jump the force between them and the floor must increase, and how much greater than their weight this force is will control the acceleration, thus their take-off velocity. Once in the air the force they are exerting on the floor has gone, while gravity is

still acting so they return to the floor. To start walking a horizontal force, applied to the floor, is needed to move the person forward. The applications of these two laws to the generation of forces within 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 your elbow with nothing in your hand compared with the same action with a weight in your hand. The muscle is contracting by the same distance, but the force is generated by reacting against the mass in the hand is different. In the first case all it has to move is the weight of your forearm, in the second it is the weight of your forearm and the weight of 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.

2.2 Levers in Biomechanics

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 (Figure 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.

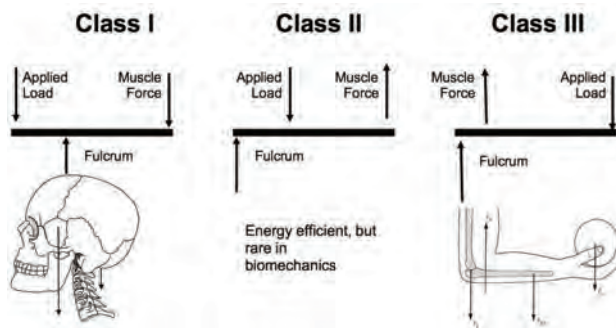


Figure 1: Types of Levers depending on the Relative Positions of the Loads and Fulcrum

2.3 Forces occurring in the body

To analyse 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 (Figure 2). The weight is acting vertically downwards and to be held still, the upward forces in the arm through to the body must equal the total weight 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 the distance to 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, nearly 4 times the total weight of the forearm and the weight being held.

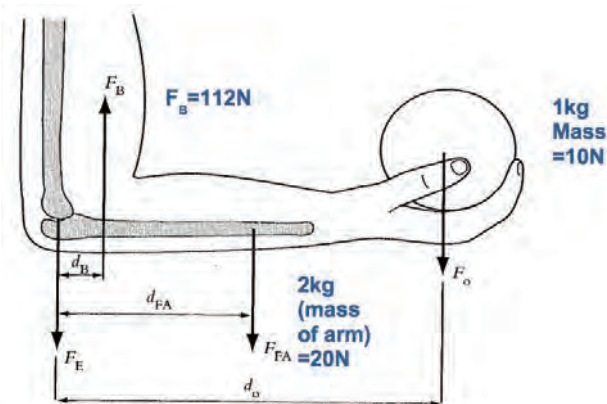


Figure 2: Calculation of forces in the forearm holding a mass

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 (Figure 3). So while gravity can be taken as being vertical most muscles do not act vertically thus care has to be taken in calculating the moment, to use the perpendicular distance.

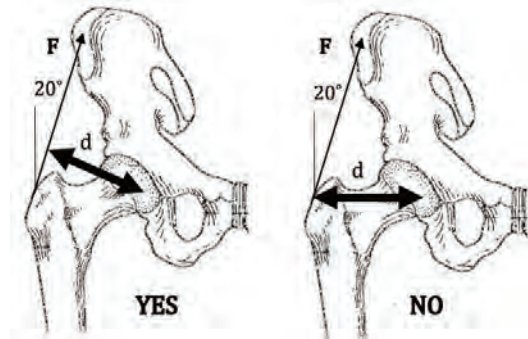


Figure 3: Calculating the moment exerted by a force F , requires the distance d to be along the line perpendicular to the direction of the force

If we apply these calculations to a person standing on one leg and making appropriate assumptions of distances in the body (Figure 4), then we can calculate that the load on the femoral head, by looking at the vertical and horizontal forces and the moments. The weight of the trunk, arms and head (F_{trunk}) will be acting vertically and through the centre of the pelvis, the weight of the contralateral leg (F_{leg}) will be acting down the centre of that leg. The moments produced by these forces will be counterbalanced by the force generated by the forces in the abductor muscles (F_{abd} , principally gluteus medius and gluteus minimus and can be assumed to act between the greater trochanter and their insertion into the pelvis) thus acting approximately at an angle of 20° to the vertical. These are mainly acting downwards and are balanced by the force through the hip joint (F_{hip}) acting at an unknown angle. F_{abd} and F_{hip} have horizontal components and these need to be equal and opposite. Thus we have three equations and three unknowns, F_{abd} , F_{hip} and ϕ the direction of the hip force so we can solve these.

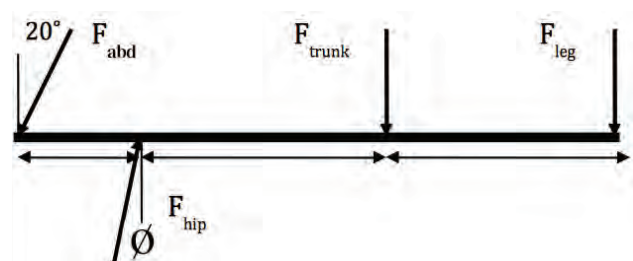


Figure 4: Forces at the hip in one legged stance

The vertical components of force give:

$$F_{hip} \cos \phi = F_{abd} \cos 20^\circ + F_{trunk} + F_{leg}$$

Horizontally we get:

$$F_{hip} \sin \phi = F_{abd} \sin 20^\circ$$

And taking moments about the hip joint gives:

$$F_{abd} \times 50 \cos 20^\circ = F_{trunk} \times 100 + F_{leg} \times 200$$

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. More complex computer based modelling using programs such as Anybody (www.anybodytech.com) can be applied which factor in a range of muscle actions and apply an optimising algorithm to "chose" how much force each muscle is assumed to apply.

The final factor to be considered is the number of load cycles applied during walking and other activities of daily living. (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 point in this paper was that they also measured some joint replacement patients and found they these people were applying more load cycles than typical for their age group. In the hand Joyce and Unsworth (2000) estimated similar number of load cycles for the fingers, but estimated that two types of loading occurred in the fingers, high range movement with low loads interspersed with limited range movement but high loads. In holding something, say a suitcase, the movement of the fingers is limited, but the loads are high, whereas in typing or playing a musical instrument, the movements are extensive, but the forces exerted are lower.

3.0 Biomaterials

3.1 Responses to Implantation of a Biomaterial

"A biomaterial is a non viable material used in a medical device, intended to interact with biological systems" according to (Williams 1999) and for an implant 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:

1. **Toxic**, that is it kills cells either in contact with the implant or away from it,
2. **Bioinert**, that is produces no response by the body and which never truly occurs, as there is always some response to implantation, but when the response is minimal the material is called bioinert.
3. **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
4. **Biodegradable** where the implant breaks down in the body to non-toxic components or compounds which can be excreted by the body, typically carbon dioxide and water.

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.

3.2 Defining the Mechanical Properties of Biomaterials

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, or change in length divided by the original length 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 much energy is needed to fracture a material. Finally **fatigue resistance** is a measure of the number of cycles at a given stress level are needed to fracture the implant or device. Which of these properties is most important will depend on the application. For a hip or knee replacement the strength and fatigue resistance are the most important properties, followed by the stiffness. In a suture the ductile and stiffness will control the easy of use, but as long as the strength is reasonably high, pull-out of the suture through the tissue being sewn is more likely to cause failure than actual fracture of the suture material.

When choosing a material for use in the load bearing areas of the body, a major consideration is the mechanical properties of the implant material compared to the body component being replaced. If two materials are being loaded together, how much load is taken by each will depend on the relative stiffness of the materials. So the steel stem of a joint replacement has a material stiffness about 10 times that of the bone, but the shapes of the implant and the bone are different, so the structural stiffness is probable 5 times that of the bone and it can be estimated that in this case 1/6 of the load will go through the bone and 5/6 through the implant. If the metal is changed to a low modulus alloy so the that stiffness of the implant is only 2 times that of the bone the loads will be divided 1/3 to 2/3 and the load on the bone will double, thus reducing the stress shielding, which leads to disuse atrophy and bone resorption (Mellon and Tanner, 2012).

3.3 Properties of Natural Materials

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 as described by Wolff's Law 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). The response of cancellous bone to compression is very different to tension, as the individual trabeculae fracture, but then pack together. This behaviour is essential around a joint replacement where the loading is compressive, Ligaments and tendons have non-linear mechanical properties with the stiffness increasing as the load increases.

3.4 Mechanical Properties of Biomaterials

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 the pure metal 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 low or 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 the newer TiNb and TiMo alloys and finally the shape memory alloys which are approximately 50:50 Ti:Ni, with the exact composition being used to control the temperature at which the shape memory effect occurs. Commercially pure Ti (CP Ti) and Ti-Al based alloys have a Young's modulus of 106 GPa. For all Ti the wear debris is black in the 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 are not as clinically 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 the newer TiNb and TiMo alloys have being developed which have yet lower Young's moduli, down to 42GPa, thus bringing their stiffnesses

closer to those of cortical bone (Hao, Li et al., 2007; Yang, Li et al., 2014). The importance of lower modulus is to bring the stiffness of the implant closer to that of the bone, reducing the unloading of the bone and thus reducing the "stress shielding". The use of magnesium alloys which degrade slowly in the body has been suggested for high load bearing degradable implants, however, at the moment none are in clinical application, but this is likely to change (Gu, Li et al. 2014).

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 Al_2O_3 was preferred as ZrO_2 can be morphologically unstable but now PSZ (Partially Stabilised Zirconia) is available. Al_2O_3 has been used by Sedel in Paris for more than 30 years as ceramic-on-ceramic hip replacements (Nizard, Pourreyron et al. 2008). In the initial implants the individual grains in the ceramics components were large and failures occurred, now grain size is reduced and failures have reduced to >1:2000. However, very close tolerances on head-cup dimensions are needed so matched pairs are supplied to reduce the fracture risk. It is essential that the matched pairs are retained to ensure that the head and cup fit each other as near "perfectly" as possible to reduce stress concentrations and fracture.

Bioactive ceramics are used in six major applications: bulk implants, that is space filling implants, porous implants when used as implants for ingrowth or scaffolds for tissue engineering, granules used to supplement or to replace autologous bone graft, coatings which can be plain hydroxyapatite ($HA - Ca_{10}(PO_4)_6(OH)_2$), tricalcium phosphate (TCP - $Ca_3(PO_4)_2$) or HA+TCP (also called biphasic calcium phosphate - BCP) and finally as injectable where the calcium phosphate, with or without some calcium sulphate (plaster of Paris - $CaSO_4$) and other additives, is mixed in the operating theatre, injected into the body and sets in situ. The degradation rate depends on the crystallinity of the ceramic phases and their relative amounts. In terms of increasing degradation rates the order is HA then TCP and finally calcium sulphate. The required degradation rate depends on the clinical application.

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

Material	Contents	Young's modulus /GPa	Strength /MPa	Comments
Bone (cortical)	40 vol% bone mineral in collagen. Approx. 3% porous	7-25	50-150	Properties depend on loading direction.
Bone (cancellous)	40 vol% bone mineral in collagen. Porosity 20-90%	0.1-1.0	1-10	Properties depend on porosity and loading direction.
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.
Ti-15Mo	15% Mo rest Ti	78	544-875	One of the simpler low modulus Ti alloys
Ti-24Nb-4Zr-7.9Sn	24% Nb, 4% Zr, 7.9%Sn rest Ti	55	570-755	One of the lowest modulus Ti alloys.
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	Polymethylmethacrylate plus opacifiers and antibiotics	2.4	40-60	

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 used 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) where the crosslinking is produced by heavily irradiating the PE are used to reduce the production of wear particles and finally Vitamin E containing PEs (Gu, Li et al. 2014). 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 material, 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 both the exotherm and shrinkage are 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 the joint and the presence of opacifier particles can lead to resorption of bone around the implant (Sabokbar, Fijikawa et al. 1997). Finally, antibiotics are added prophylactically to bone cement to reduce the risk of infection (Jiranek, Hanssen et al. 2006).

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 is excreted. 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 (or more) phase materials where the phases can be seen as separate either with the naked eye or using an

optical microscope, that is the two phases can be differentiated on the millimetre/submillimeter 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 an 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" for the required application, while 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 increasing (Tanner 2010). The earliest ones were bioinert, but now bioactive implants are beneficially interacting with the human body. By choice of non-degradable phases such as HA in PE non-degradable composites are produced, while combining TCP with PLLA a fully degradable composite can be produced, with stiffness approaching that of cortical bone, although at the moment the strengths are lower. However, the cellular response to some of these composites can be excellent (Talal, McKay et al. 2013) and can be used for drug release.

4.0 Conclusions

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

References

1. Ansari, F., M.D. Reis, et al. (2016) "Effect of processing, sterilization and crosslinking on UHMWPE fatigue fracture and fatigue wear mechanisms in joint arthroplasty." *Journal of the Mechanical Behavior of Biomedical Materials* 53: 329-340
2. Bergmann, G., F. Graichen, et al. (1993). "Hip-joint loading during walking and running, measured in 2 patients." *Journal of Biomechanics* 26(8): 969-990.
3. Bleach, N. C., S. N. Nazhat, et al. (2002). "Effect of Filler Content on Mechanical and Dynamic Mechanical Properties of Particulate Biphasic Calcium Phosphate Poly(lactide Composites)." *Biomaterials* 23(7): 1579-1585.
4. Cook, S. D., N. Thongpreeda, et al. (1988). "The effect of post-sintering heat treatments on the fatigue properties of porous coated Ti-6Al-4V alloy." *Journal of Biomedical Materials Research* 22(4): 287-302.

5. Currey, J. D. (1998). "Mechanical properties of vertebrate hard tissues." *Proceedings of the Institution of Mechanical Engineers: Part H Engineering in Medicine* 212-H(6): 399-412.
6. Currey, J. D. (2006). *Bones: Structure and Mechanics*, Princeton University Press.
7. Gibson, L. J. and M. F. Ashby (1999). *Cellular Solids*. Oxford, Pergamon Press.
8. Gu, X.-N., S. S. Li et al. (2014) "Magnesium based degradable biomaterials: A review." *Frontiers in Materials Science* 8(3): 200-218.
9. Hao, Y. L., S. J. Li, et al. (2007). "Elastic deformation behaviour of Ti-24Nb-4Zr-7.9Sn for biomedical applications." *Acta Biomaterialia* 3(2): 277-286.
10. Huttunen, M., P. Törmälä, et al. (2008). "Fiber-reinforced bioactive and bioabsorbable hybrid composites." *Biomedical Materials* 3(3).
11. Jiranek, W. A., A. D. Hanssen, et al. (2006). "Antibiotic-loaded bone cement for infection prophylaxis in total joint replacement." *Journal of Bone and Joint Surgery* 88-A(11): 2487-2500.
12. Joyce, T. J. and A. Unsworth (2000). "The design of a finger wear simulator and preliminary results." *Proceedings of the Institution of Mechanical Engineers: Part H Engineering in Medicine* 214-H(5): 519-526.
13. Li Y.H., C. Yang et al. (2014) "New Developments of Ti-Based Alloys for Biomedical Applications", *Materials* 7, 1709-1800.
14. Mellon, S. and K. E. Tanner (2012) "Mechanical Adaptability of Bone in Vivo and in Vitro – A Review", *International Materials Reviews* 25(5), 235-255
15. Nizard, R. S., D. Pourreyron, et al. (2008). "Alumina-on-alumina hip arthroplasty in patients younger than 30 years old." *Clinical Orthopaedics and Related Research* 466: 317-323.
16. Sabokbar, A., Y. Fijikawa, et al. (1997). "Radio-opaque agents in bone cement increase bone resorption." *Journal of Bone and Joint Surgery* 79B(1): 129-134.
17. Tanner, K. E. (2010). *Hard tissue applications of biocomposites*. Biomedical Composites. Ed L. Ambrosio. Cambridge, UK, Woodhead Publishers.
18. Talal, A., I.J. McKay, et al. (2013) "Effects of Hydroxyapatite and PDGF Concentrations on Osteoblast Growth in a Nanohydroxyapatite-Polylactic Acid (nHA-PLA) Composite for Guided Tissue Regeneration", *Journal of Materials Science: Materials in Medicine* 24, 2211-2221.
19. Wallbridge, N. and D. Dowson (1982). "The walking activity of patients with artificial hip joints." *Engineering in Medicine* 11(3): 95-96.
20. Wang, J. S., H. Franzen, et al. (1993). "Porosity of Bone Cement reduced by mixing and collecting under vacuum " *Acta Orthopaedica Scandinavica* 64(2): 143-146.
21. Williams, D. F. (1999). *The Williams Dictionary of Biomaterials*. Liverpool, Liverpool University Press.

QUESTIONS

1. What distance do you use when calculating the moment exerted by a force about a joint?
2. Why is the stiffness of the implant important in the loosening of implants and fracture fixation plates?
3. What type of interface will be produced by a bioinert material and how strong will be the interface?
4. What are the benefits of using a bioactive material?
5. What are the major components of bone cement and why are they used?

Answers

1. The perpendicular distance from the joint to the direction of the force (see Figure 3).
2. A stiff implant will carry most of the load, therefore reducing the load on bone and bone when the applied load is reduced starts to resorb (according to Wolff's Law). Thus a lower stiffness implant should reduce the rate at which bone resorbs around an implant.
3. A bioinert material leads to the body encapsulating the implant in a fibrous layer which has low mechanical properties.
4. In orthopaedics bioactive materials are generally used to encourage bone ongrowth on to the material or implant. Typical applications are HA coatings on the stems of non-cemented joint replacements (non-degradable) or in bone graft substitutes/extenders (as a degradable bioactive material).
5. PMMA powder so already polymerised and thus reduce polymerisation exotherm and shrinkage, MMA monomer which is responsible for the setting process, opacifier (zirconia or barium sulphate) to allow the cement to be seen on X-rays and antibiotic to reduce infections.



Prof. Dr. med. Karsten Dreinhöfer

Charité Universitätsmedizin Berlin, Berlin Germany

Karsten.Dreinhoefer@charite.de

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%).

1.2.1 Inorganic component

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

1.2.2 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 differentiated: 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

1.3. Cellular Structure

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

1.3.1 Osteoprogenitor cells:

Osteoprogenitor cells are pluripotent mesenchymal stem cells differentiating into osteoblast when stimulated

1.3.2 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.

1.3.3 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.

1.3.4 Osteoclast

Osteoclasts are responsible for bone resorption. They are large, multinucleated 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.

1.4 Remodeling

Remodeling or bone turnover is a constant process right from the embryonic age to the end of life [4] 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, 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-day 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).

1.5 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.

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

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

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

2. Metabolic Bone Disease

- Diseases Associated with Abnormal Matrix = Disorders of osteoblasts
- Diseases associated with Abnormal Remodelling = Disorders of osteoclasts
- Diseases Associated with Abnormal Mineral Homeostasis

2.1 Diseases Associated with Abnormal Matrix

2.1.1 Osteogenesis Imperfecta

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

Clinical expression:

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

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

Treatment:

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

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.

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."

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.

2.1.3.1 Pathophysiology

The underlying mechanism in all cases of osteoporosis is an imbalance between bone resorption and bone formation. 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 microarchitecture 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.

2.1.3.2 Risk Factors

The most important risk factors for osteoporosis are advanced age (in both men and women) and female gender ;

While these are non-modifiable 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

2.1.3.3 Falls risk

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

2.1.3.4 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.

An approach combining the assessment of bone mineral density, clinical risk factors for fracture with use of the FRAX, and bone turnover markers will improve the prediction of fracture risk and enhance the evaluation of patients with osteoporosis.

Patients who have had recent osteoporotic fractures are at particular high risk for additional fractures.

2.1.3.5 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. 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.

2.1.3.6 Prevention

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

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

2.1.3.7 Treatment

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

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. Deficient carbonic anhydrase might result in defective hydrogen ion pumping in osteoclasts. This might cause defective bone resorption, since an acidic environment is needed for dissociation of calcium hydroxyapatite from bone matrix and its release into blood circulation. If bone resorption fails while formation persists, excessive bone is formed.

Despite a diffuse symmetric skeletal sclerosis, bones are brittle and fracture frequently. Many bones do not develop a medullary cavity. Mild forms may cause no symptoms. However, serious forms can result in stunted growth, deformity and an increased likelihood of fractures. Bone marrow narrowing leads to extramedullary hematopoiesis, resulting in hepatosplenomegaly. Patients suffer from anemia and recurrent infections. Due to the increased pressure put on the nerves by the extra bone it can also lead to blindness, facial paralysis, and deafness.

The only durable cure for osteopetrosis is bone marrow transplant.

Table 1: Comparison of bone pathology

Condition	Calcium	Phosphate	Alkaline Phosphat	Parathyroid Hormon	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

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 hyper responsive reaction to vitamin D and RANK ligand might be the cause.

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.

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.

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. 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 feedback mechanisms leads to excessive Parathormone secretion with continuing PTH output. Increased parathyroid hormone are 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 to differentiate 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. There are two main components to renal osteodystrophy:

- 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.
- Secondary hyperparathyroid effects secondary to hyperphosphatemia and hypocalcemia due to phosphate retention and excess calcium loss in urine of the damaged kidney.

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

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

Blood tests will indicate decreased calcium and calcitriol 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. 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 is often restricted to the milder, adult form of the disease, while in children the disease is known as rickets.

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.

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

Treatment:

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

References:

1. Al-Rashid M, Ramkumar DB, Raskin K et al. (2015) Paget Disease of Bone. *Orthop Clin North Am* 46:577-585
2. Bhan A, Rao AD, Rao DS (2012) Osteomalacia as a result of vitamin D deficiency. *Rheum Dis Clin North Am* 38:81-91, viii-ix
3. Black DM, Rosen CJ (2016) *Clinical Practice. Postmenopausal Osteoporosis*. *N Engl J Med* 374:254-262
4. Bolland MJ, Cundy T (2013) Paget's disease of bone: clinical review and update. *J Clin Pathol* 66:924-927
5. Clarke LA, Hollak CE (2015) The clinical spectrum and pathophysiology of skeletal complications in lysosomal storage disorders. *Best Pract Res Clin Endocrinol Metab* 29:219-235
6. Datta HK, Ng WF, Walker JA et al. (2008) The cell biology of bone metabolism. *J Clin Pathol* 61:577-587
7. Elder CJ, Bishop NJ (2014) Rickets. *Lancet* 383:1665-1676
8. Fraser WD (2009) Hyperparathyroidism. *Lancet* 374:145-158
9. Galson DL, Roodman GD (2014) Pathobiology of Paget's Disease of Bone. *J Bone Metab* 21:85-98
10. Kanis JA, McCloskey EV, Johansson H et al. (2013) European guidance for the diagnosis and management of osteoporosis in postmenopausal women. *Osteoporos Int* 24:23-57
11. Kemper MJ, van Husen M (2014) Renal osteodystrophy in children: pathogenesis, diagnosis and treatment. *Curr Opin Pediatr* 26:180-186
12. Lampe C, Bellettato CM, Karabul N et al. (2013) Mucopolysaccharidoses and other lysosomal storage diseases. *Rheum Dis Clin North Am* 39:431-455
13. Laron D, Pandya NK (2013) Advances in the orthopedic management of osteogenesis imperfecta. *Orthop Clin North Am* 44:565-573
14. Lindahl K, Langdahl B, Ljunggren O et al. (2014) Treatment of osteogenesis imperfecta in adults. *Eur J Endocrinol* 171:R79-90
15. Pelletier S, Chapurlat R (2010) Optimizing bone health in chronic kidney disease. *Maturitas* 65:325-333
16. Poole KE, Compston JE (2006) Osteoporosis and its management. *BMJ* 333:1251-1256
17. Rachner TD, Khosla S, Hofbauer LC (2011) Osteoporosis: now and the future. *Lancet* 377:1276-1287
18. Sobacchi C, Schulz A, Coxon FP et al. (2013) Osteopetrosis: genetics, treatment and new insights into osteoclast function. *Nat Rev Endocrinol* 9:522-536
19. Tolar J, Teitelbaum SL, Orchard PJ (2004) Osteopetrosis. *N Engl J Med* 351:2839-2849
20. Tomatsu S, Almeciga-Diaz CJ, Montano AM et al. (2015) Therapies for the bone in mucopolysaccharidoses. *Mol Genet Metab* 114:94-109
21. Unnanuntana A, Gladnick BP, Donnelly E et al. (2010) The assessment of fracture risk. *J Bone Joint Surg Am* 92:743-753
22. Van Dijk FS, Sillence DO (2014) Osteogenesis imperfecta: clinical diagnosis, nomenclature and severity assessment. *Am J Med Genet A* 164A:1470-1481
23. Waugh EJ, Lam MA, Hawker GA et al. (2009) Risk factors for low bone mass in healthy 40-60 year old women: a systematic review of the literature. *Osteoporos Int* 20:1-21
24. WHO (1994) Assessment of fracture risk and its application to screening for postmenopausal osteoporosis. Report of a WHO Study Group. World Health Organization technical report series 843: 1-129

QUESTIONS

1. List the two most important non-modifiable and further three modifiable risk factors for osteoporosis.
2. What are the three most important risk factors for an osteoporotic fracture ?
3. Which are the cornerstones of osteoporosis prevention ?
4. 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 too low, e.g. the T-Score
 - a) is greater than -1.0
 - b) is between -1.0 and -2.5
 - c) is below -2.5
5. A slow progressive metabolic bone disease with abnormal osteoclast and osteoblast activity that leads to spinal stenosis, facet arthropathy, and spinal fractures, with elevated serum alkaline phosphatase, and urinary hydroxyproline
 - a) osteitis deformans (Paget's Disease)
 - b) osteochondritis desicans
 - c) osteogenesis imperfecta
6. A genetic disorder of abnormal collagen sythesis with fractures and deformity of weight bearing bones, thinning of bones, most likely:
 - a) osteitis deformans (Paget's Disease)
 - b) osteochondritis desicans
 - c) osteogenesis imperfecta
7. Decalcification of bones secondary to vitamin D deficiency, patient with severe pain, fractures, weakness, and deformities most likely:
 - a) osteomyelitis
 - b) osteoporosis
 - c) osteomalacia

ANSWERS:

1. i) **nonmodifiable:** advanced age and female gender
ii) **modifiable:** malnutrition, Vitamin D deficiency, physical inactivity, tobacco smoking, excess alcohol
2. i) low BMD
ii) high age
iii) propensity to fall
3. i) Achieving high peak bone mass in young age
ii) Avoidance of modifiable risk factors (immobility, smoking, tobacco)
iii) Proper nutrition incl. appropriate amount of calcium and Vitamin D
iv) Aerobics, weight bearing and resistance exercises

4c, 5a, 6c, 7c



Prof. Dr. med. Hakan Ömeroğlu

TOBB University of Economics and Technology, Faculty of Medicine,
Department of Orthopaedics & Traumatology,
Ankara, Turkey
omeroglu.h@gmail.com

Classification And Principle Of Fractures Treatment In Childhood

Epidemiology

1. About one out of four of children are injured per year and skeletal trauma accounts 10% to 25% of these injuries.
2. About 1% to 3% of children have at least one fracture per year. From birth to 16-17 years of age, about one third of children sustain at least one fracture. There is a linear increase in the annual incidence of fractures with age.
3. A considerable increase in incidence has been seen due to changes in life style, childhood obesity, more participation in sports activities and increased number of traffic accidents.
4. Children's fractures are 1,5 to 3 times more common in boys than in girls.
5. About two third of the children's fractures occur in upper extremity. The most common fractured bone in children is radius and the most common fractured site is the distal third.
6. Children's fractures are commonly due to falls, vehicular or pedestrian traffic accidents, sports injuries as well as non-accidental reasons including child abuse and pathologic fractures.
7. The rates of open and multiple fractures in children are about 3% and 4%, respectively

5. Non-union is rare in children's fractures.
6. Growth stimulation or overgrowth of the fractured bone may occur in children

Classification According to the Fracture Pattern

Transverse fracture: The applied force to the long bone at the fracture site is perpendicular to the long axis of the bone.

Oblique fracture: The applied force to the long bone is the compression force (axial loading).

Spiral fracture: This type of fracture is caused by torsional force.

Butterfly fracture: A combination of compression (axial loading) and perpendicular (angulation) forces produce this fracture.

Comminuted fracture: This type of fracture is due to high-energy trauma and rarely occurs in children.

Growth Plate (Physeal) Injuries and Classification

About one out of five fractures in children involve the growth plate. Such an injury can cause a growth disturbance and this is mainly due to avascular necrosis, crushing or infection of the growth plate, formation of a bone bridge between epiphysis and metaphysis. Most of the fractures occur through the hypertrophic zone of the growth plate. The rate of growth disturbance is not so high in growth plate injuries.

Salter-Harris classification is the universally accepted and the most commonly used classification system for growth plate injuries (Figure 1).

Type I: The fracture line passes through the hypertrophic zone of the growth plate. Such injuries are mostly due to shearing, torsional or avulsion forces especially in infants and young children. Growth arrest is rare. A secure closed reduction is possible in most of these injuries.

Type II: The fracture line passes through much of the hypertrophic zone of the growth plate and then extends through metaphysis. The metaphyseal fragment is called "Thurston-Holland fragment". This is the most common growth plate fracture type (about 75%). Long-term sequel is rare and secure closed reduction can be achieved in most of these injuries. Open reduction and internal fixation may be required in some locations such as distal femur.

Type III: Fracture line passes through the growth plate and then extends into the epiphysis and the adjacent joint. The most common site for this type of fracture is the distal tibia. Growth disturbance can occur and open reduction and internal fixation is frequently needed.

Differences Between Fractures in Children and in Adults

Children are not just small adults and their fractures differ from the ones in adults. The biology of musculoskeletal system in children makes the fracture healing process less complicated. The main differences are listed below;

1. Children have thicker, more vascular and osteogenic periosteum that allows their fractures to heal more quickly than the adults' one.
2. The Haversian canals are wider and bones are more porous in children. Thus, some specific pediatric age group incomplete fractures such as traumatic bowing, torus fracture and greenstick fracture can occur due to previously described special nature of bones in children.
3. Fractures in children have a considerable capability of remodeling which is more efficient in younger children, in fractures close to active growth plates and in fractures healed with residual angulation in the motion plane of the nearest joint.
4. Angular deformity and limb-length discrepancy can be seen as a result of permanent damage to the growth plate.

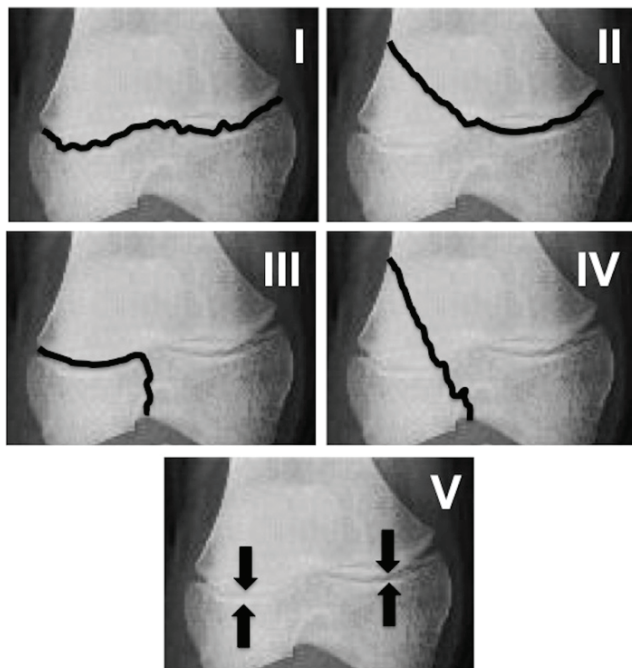


Figure 1: Salter-Harris growth plate injury classification system.

Type IV: The fracture line passes through metaphysis, entire growth plate and epiphysis, extends into the adjacent joint. Growth disturbance, joint stiffness and non-union can be seen. Open reduction and internal fixation is mandatory to restore both the joint and growth plate anatomy.

Type V: This is a compression injury of the growth plate.

Type VI: This very rare type is a peripheral growth plate injury at the level of perichondrial ring of Ranvier.

How to Approach Children's Fractures in the Emergency Room (ER)

The ER physician should first examine the child's overall health status. A careful initial radiographic examination is essential for making the proper diagnosis. Orthogonal AP and lateral views including the distal and proximal joints of the fractured bone are the initial mandatory steps for exact diagnosis. Sophisticated radiological diagnostic tools can be used if more information concerning the characteristics of the fracture is needed. Simple fractures, which do not need urgent Orthopaedic consultation can be splinted and directed to the Orthopaedics outpatient clinics. Several injuries including urgent, severe and open fractures as well as fractures with neurovascular injury should always be consulted with the Ortho team. This is followed by treatment of the fracture by Ortho team either in the emergency room or in the operating theatre following hospital admission.

Principles of Fracture Treatment in Children

The goals of fracture treatment are;

1. To provide fracture reduction by open or closed means and maintain fracture reduction by casts, splints, braces, traction, internal or external fixation

2. To protect the soft tissues
3. To facilitate the fracture healing
4. To allow early mobilization and to permit early range of motion
5. To avoid complications including malunion, non-union, premature physeal closure, infection, skin breakdown, joint stiffness, hardware failure, symptomatic hardware, compartment syndrome and iatrogenic neurovascular injury

Fractures in children are mostly treated by non-surgical methods. Age, body-mass index, fracture site, type and severity, coexisting injuries as well socioeconomic factors influence the choice of fracture treatment method. Surgical treatment is preferred in children with multiple injuries as well as in open fractures, in most of the pathologic fractures, in fractures with coexisting vascular injury, in fractures in which the initial conservative treatment has failed, in fractures in which the conservative treatment has no/little value such as femur neck fractures, some growth plate injuries (primarily Salter-Harris type III and IV fractures), displaced humerus supracondylar and lateral condyle fractures, femur, tibia and radius-ulna shaft fractures in older children and adolescents and in unstable pelvic and acetabulum fractures.

Splints and casts

Maintenance of the reduction may be achieved by splinting or casting in many pediatric fractures with a great success. Loss of reduction, skin problems, compartment syndrome due to tight casting or excessive swelling and inability to inspect wounds are the disadvantages of the casting.

Functional braces

This treatment method is indicated in stable fractures including certain phalangeal fractures (buddy taping) and humerus shaft fractures.

Traction

The use of skin or bone traction as a definitive treatment method is very limited in children's fractures. Skeletal traction is a useful method for temporary fracture fixation before the definitive treatment

Internal fixation

K-wires, flexible or rigid intramedullary nailing, plates and screws are the commonly used implants for the internal fixation of children's fractures. Most of the fractures in children can currently be reduced by closed means under image intensifier control before internally fixed. Optimal fracture stability, early mobilization and joint range of motion, reduced need for splints and casts can be achieved by internal fixation. Development of postoperative infection, the need for hardware removal, possible intraoperative damage to neighborhood neurovascular structures, growth plate, bone vascularity or joint surfaces are the disadvantages of the internal fixation.

External Fixation

Several external fixation devices are available in the market. External fixation is commonly preferred in contaminated wounds and provides rigid fixation, wound care, early mobilization and joint range of motion.

Special Conditions

Child Abuse

Child abuse is currently a significant pediatric problem and needs special attention as, about 1-3% of abused children die. About 25-50% of abused children have fractures. Abused children are frequently younger than 2 years of age. Humerus, femur and tibia are the most fractured bones. Corner (bucket-handle) fractures, humerus, femur and tibia shaft fractures and Salter-Harris type I growth plate injuries of the distal humerus are commonly seen in such children. Abused children may have head injuries and bruises, besides fractures. There may be multiple injuries over a period of time and history of previous injuries should be evaluated well. Parents commonly make evasive explanations about how the injury has happened and can be lack of tenderness. A high index of suspicion especially in children having the above-mentioned findings makes the exact diagnosis in most of the cases.

Open Fractures

Although it has been stated that the treatment of open fractures in children is similar to one of in adults, the level of evidence is low concerning this statement. Meticulous wound care, reduction and stabilization of the fracture and infection prophylaxis by antibiotics are the essential steps of the treatment. Severe open tibia fractures (grade III) in older children (>10 years) have a high complication rate and a poor prognosis like in adults.

Multiple Injuries

Fracture stabilization provides several non-orthopaedic benefits to a child with multiple injuries. Definitive treatment should be made when the child's general health condition is stabilized.

Pathologic Fractures

A pathologic fracture is defined as a fracture that occurs through abnormal bone. Pathologic fractures can occur due to a local bone disease (tumors, osteomyelitis) or due to a systemic disease that weakens the bone (marrow disease of bone, osteogenesis imperfecta, osteopetrosis, rickets, neuromuscular disorders). Surgical treatment is commonly preferred in most of these fractures.

References

1. Aygün U, Bolluk Ö, Ömeroglu H (2011) A regional epidemiologic study for children's fractures. *Acta Orthop Traumatol Turc* 45 Suppl 1:116
2. Beaty JH, Kasser JR (2001) *Rockwood and Wilkins' fractures in children*. 5th ed Lippincott Williams & Wilkins Philadelphia
3. Gougoulas N, Khanna A, Mafulli N (2009) Open tibial fractures in the paediatric population: a systematic review of the literature. *Br Med Bull* 91:75-85
4. Hedström EM, Svensson O, Bergström U, Michno P (2010) Epidemiology of fractures in children and adolescents: Increased incidence over the past decade: a population-based study from northern Sweden. *Acta Orthop* 81:148-153
5. Herman MJ, McCarthy JJ. The principles of pediatric fracture and trauma care. In: Weinstein SL, Flynn JM (2014) *Lovell and Winter's Pediatric Orthopaedics*. 7th ed Wolters Kluwer Philadelphia
6. Landin LA (1983) *Fracture patterns in children: Analysis of 8,682 fractures with special reference to Incidence, etiology and secular changes in a Swedish urban population 1950-1979*. *Acta Orthop Scand Suppl* 202:1-109
7. Musgrave DS, Mendelson SA (2002) *Pediatric orthopedic trauma: Principles in management*. *Crit Care Med* 30:S431-S443
8. Salter RB, Harris WR (1963) Injuries involving the epiphyseal plate. *J Bone Joint Surg* 45:587-622
9. Wenger DR, Pring ME (2005) *Rang's children's fractures*. 3rd ed Lippincott Williams & Wilkins Philadelphia

QUESTIONS:

1. Which one of the following is NOT a specific pediatric age group fracture type ?
a. Torus fracture
b. Physeal fracture
c. Metaphyseal fracture
d. Greenstick fracture
e. Traumatoc bowing
2. Which one of the following is NOT a common fracture in abused children ?
a. Distal humerus transphyseal fracture
b. Femur shaft fracture
c. Tibia shaft fracture
d. Humerus shaft fracture
e. Radius shaft fracture
3. Which one of the following fracture type is produced by a torsional force ?
a. Transverse fracture
b. Oblique fracture
c. Spiral fracture
d. Butterfly fracture
e. Comminuted fracture
4. Which one of the following factors do NOT influence the choice of fracture treatment in children ?
a. Age
b. Gender
c. Body-mass index
d. Fracture type
e. Fracture site
5. In which one of the following pediatric fracture scenarios in a 5 year old child (displaced & close fracture, no neurovascular injury, no coexisting injury) the surgical treatment is PRIMARILY indicated ?
a. Clavicle shaft fracture
b. Humerus metaphyseal fracture
c. Radius shaft fracture
d. Femur neck fracture
e. Tibia shaft fracture

Correct Answers:
1c, 2e, 3c, 4b, 5d



Assoc. Prof. Dr. med. Christof Radler

Orthopedic Hospital Vienna Speising
Vienna, Austria

christof.radler@oss.at

Foot Disorders of Newborns

Foot disorders 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 [41].

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 disorders 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 [32].

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 [28,29].

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 [20]. 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 adductus (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. (Figure 1) Clinical examination can differentiate between a flexible MTA and a rigid MTA. In flexible MTA the forefoot can passively be abducted to more than neutral, while rigid MTAs can not be abducted to a neutral position. Flexible MTAs usually resolve spontaneously. However, the mother can perform a stretching of the

medial foot and especially stimulation of the lateral foot and thigh which usually stimulate the peroneal muscle group and results in abduction, dorsiflexion and eversion of the whole foot and extension and abduction of the toes. In more resistant cases an abduction orthotic which is available in different forms of shoes like the IPOS shoe or reverse-last shoes might be used up to the age of 6-9 months.



Figure 1: Mild but persistent metatarsus adductus

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 [21]. 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 [19]. 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 [22]. 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 [27].

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].



Figure 2: Talipes calcaneovalgus in a newborn



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

1.4 Positional clubfoot (postural talipes equinovarus)

Positional clubfoot is a molding deformity which resembles clubfoot. The foot is in mild equinus, in adduction and is rotated inwards. However, the foot is passively fully flexible with dorsiflexion well above neutral and no sign of the rigid deformities seen in true clubfoot (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.



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

2. Structural deformities

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

2.1 Clubfoot

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

The etiology is still unknown; however there is a multitude of factors associated with an increased incidence of clubfoot. Genetic studies have favored the hypothesis of a heterogeneous disorder with a 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).



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

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, the ligaments are less flexible and contracted, the muscles are more fibrotic and cells of the medial ligamentous tissue have myofibroblastic characteristics [17,36].

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 [35].



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

Treatment should be started within the first weeks of life. However, there is no need to rush treatment as it was traditionally done in many countries. I recommend starting treatment after the infant and mother have been released from the hospital and feel well and strong enough for travelling. At presentation a thorough pediatric orthopaedic examination of the newborn must be performed which should include a hip ultrasound. Documentation should include digital pictures and a clinical scoring. The Pirani score is a very well reproducible and validated score that can even guide the treatment process [38].

In the last 10 years the Ponseti method has become the gold standard for the treatment of clubfoot worldwide [18,37]. 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 [30,31]. 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 tibio calcaneal angle [33]. The tenotomy is a full percutaneous tenotomy performed from medial to lateral to avoid bleeding complications. The last cast after the tenotomy stays for three weeks and is molded in full abduction and dorsiflexion. After cast removal a foot abduction orthosis (Denis Browne bar), with the clubfoot in 70 degrees abduction and the normal foot in 45 degrees abduction must be used for 22 hours for 3 months and for 3–4 years for nights and naps. A tibialis anterior tendon transfer is necessary in about 20 % of patients usually between age 3–6 in case of weak peroneal muscles and an overpowering tibialis anterior muscle.

Mid- and longterm results are very good and superior to all other treatment regimens or surgical approaches reported in literature [3-5,24,34,39].

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 agenesis or Larson's syndrome. The incidence is with approximately 1:10.000 about ten times lower than clubfoot.



Figure 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 [23]. 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%) [25].

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

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

Traditionally treatment was surgically with the most recent

approach being an open release through a Cincinnati approach, tendo Achilles lengthening, capsulotomy and medial release with reposition of navicular, reconstruction of capsule and transfixation with k-wires. A treatment based on serial manipulation and casting combined with a minimal surgical intervention was introduced by Dobbs [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 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 [2,40]. 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



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

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 [26]. 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 [42]

References:

1. Blauth W, Borisch NC (1990) Cleft feet. Proposals for a new classification based on roentgenographic morphology. *Clin Orthop Relat Res* (258):41-8.
2. Blauth W, Olason AT (1988) Classification of polydactyly of the hands and feet. *Arch. Orthop. Trauma. Surg* 107, 334-344.
3. Bor N, Coplan JA, Herzenberg JE (2009) Ponseti treatment for idiopathic clubfoot: minimum 5-year followup. *Clin Orthop* 467: 1263-1270
4. Church C, Coplan JA, Poljak D, Thabet AM, Kowtharapu D, Lennon N, Marchesi S, Henley J, Starr R, Mason D, Belthur MV, Herzenberg JE, Miller F (2012) A comprehensive outcome comparison of surgical and Ponseti clubfoot treatments with reference to pediatric norms. *J Child Orthop.* 6(1):51-9.
5. Cooper DM, Dietz FR (1995) Treatment of idiopathic clubfoot: a thirty-year follow-up note. *J Bone Joint Surg Am* 77-A: 1477-1489.
6. Dobbs MB, Gurnett CA (2012) Genetics of clubfoot. *J Pediatr Orthop B* 21(1):7-9.
7. Dobbs MB, Purcell DB, Nunley R, Morcuende JA (2006) Early results of a new method of treatment for idiopathic congenital vertical talus. *J Bone Joint Surg Am* 88(6):1192-200.
8. Dobbs MB, Schoenecker PL, Gordon JE (2002) Autosomal dominant transmission of isolated congenital vertical talus. *Iowa Orthop J* 22:25-7
9. Eberhardt O, Fernandez FF, Wirth T. [Treatment of Vertical Talus with the Dobbs Method.] *Z Orthop Unfall.* 2011;149(2):219-224.
10. Edwards ER, Menelaus MB (1987) Reverse club foot. Rigid and recalcitrant talipes calcaneovalgus. *J Bone Joint Surg Br* 69:330.
11. Farsetti P, Weinstein SL, Ponseti IV (1994) The long-term functional and radiographic outcomes of untreated and non-operatively treated metatarsus adductus. *J Bone Joint Surg Am* 76(2):257-65.
12. Hagmann S, Dreher T, Wenz W (2009) Skewfoot. *Foot Ankle Clin.* 14(3):409-34.
13. Hamer AJ, Stanley D, Smith TW (1993) Surgery for curly toe deformity: a double-blind, randomised, prospective trial. *J Bone Joint Surg Br* 75(4):662-3.
14. Herzenberg JE, Burghardt RD (2013) Resistant metatarsus adductus: prospective randomized trial of casting versus orthosis. *J Orthop Sci.* Nov 19. [Epub ahead of print]
15. Herzenberg JE, Carroll NC, Christofersen MR, Lee EH, White S, Munroe R (1988) Clubfoot analysis with three-dimensional computer modeling. *J Pediatr Orthop* 8(3):257-62.
16. Holt JB, Oji DE, Yack HJ, Morcuende JA (2015) Long-Term Results of Tibialis Anterior Tendon Transfer for Relapsed Idiopathic Clubfoot Treated with the Ponseti Method: A Follow-up of Thirty-seven to Fifty-five Years. *J Bone Joint Surg Am* 97(1):47-55.
17. Ippolito E, Ponseti IV (1980) Congenital clubfoot in the human fetus: A histological study. *J Bone Joint Surg Am* 62: 8-22.
18. Jowett CR, Morcuende JA, Ramachandran M (2011) Management of congenital talipes equinovarus using the Ponseti method: a systematic review. *J Bone Joint Surg Br* 93(9):1160-4.
19. Katz K, David R, Soudry M (1999) Below-knee plaster cast for the treatment of metatarsus adductus. *J Pediatr Orthop* 19(1):49-50.
20. Katz K, Naor N, Merlob P, Wielunsky E (1990) Rotational deformities of the tibia and foot in preterm infants. *J Pediatr Orthop* 10(4):483-5.
21. Kite JH (1967) Congenital metatarsus varus. *J Bone Joint Surg Am* 49(2):388-97.
22. Knörr J, Soldado F, Pham TT, Torres A, Cahuzac JP, Gauzy JS (2013) Percutaneous correction of persistent severe metatarsus adductus in children. *J Pediatr Orthop.* Nov 21. [Epub ahead of print]
23. Merrill LJ, Gurnett CA, Connolly AM, Pestronk A, Dobbs MB (2011) Skeletal muscle abnormalities and genetic factors related to vertical talus. *Clin Orthop Relat Res* 469(4):1167-74.

24. Mindler GT, Kranzl A, Lipkowski CA, Ganger R, Radler C (2014) Results of gait analysis including the Oxford foot model in children with clubfoot treated with the Ponseti method. *J Bone Joint Surg Am* 96(19):1593-9.
25. Ogata K, Schoenecker PL, Sheridan J (1979) Congenital vertical talus and its familial occurrence: an analysis of 36 patients. *Clin Orthop Relat Res* (139):128-32.
26. Opitz JM 1985 The developmental field concept. *Am J Med Genet* 21(1):1-11.
27. Pappas AM (1984) Congenital posteromedial bowing of the tibia and fibula. *J Pediatr Orthop* 4:525.
28. Paton RW, Choudry Q (2009) Neonatal foot deformities and their relationship to developmental dysplasia of the hip: an 11-year prospective, longitudinal observational study. *J Bone Joint Surg Br* 91(5):655-8.
29. Perry DC, Tawfiq SM, Roche A, Shariff R, Garg NK, James LA, Sampath J, Bruce CE (2010) The association between clubfoot and developmental dysplasia of the hip. *J Bone Joint Surg Br* 92(11):1586-8.
30. Ponseti IV (1996) *Congenital clubfoot. Fundamentals of treatment*. New York: Oxford University Press Inc.
31. Radler C. The Ponseti method for the treatment of congenital club foot: review of the current literature and treatment recommendations. *Int Orthop*. 2013;37(9):1747-53.
32. Radler C, Egermann M, Riedl K, Ganger R, Grill F (2010) Interobserver reliability of radiographic measurements of contralateral feet of pediatric patients with unilateral clubfoot. *J Bone Joint Surg Am* 20;92(14):2427-35.
33. Radler C, Manner HM, Suda R, Burghardt R, Herzenberg JE, Ganger R, Grill F (2007) Radiographic Evaluation of Idiopathic Clubfeet Undergoing Ponseti Treatment. *J Bone Joint Surg Am* 89: 1177-1183.
34. Radler C, Mindler GT, Riedl K, Lipkowski C, Kranzl A (2013) Midterm results of the Ponseti method in the treatment of congenital clubfoot. *Int Orthop*.;37(9):1827-31.
35. Radler C, Myers AK, Burghardt RD, Arrabal PP, Herzenberg JE, Grill F (2011) Maternal attitudes towards prenatal diagnosis of idiopathic clubfoot. *Ultrasound Obstet Gynecol* 37(6):658-62.
36. Sano H, Uhthoff HK, Jarvis JG, Mansingh A, Wenckebach GF (1998) Pathogenesis of soft-tissue contracture in club foot. *J Bone Joint Surg Br* 80(4):641-4.
37. Shabtai L, Specht SC, Herzenberg JE (2014) Worldwide spread of the Ponseti method for clubfoot. *World J Orthop* 5(5):585-90.
38. Shaheen S, Jaiballa H, Pirani S (2012) Interobserver reliability in Pirani clubfoot severity scoring between a paediatric orthopaedic surgeon and a physiotherapy assistant. *J Pediatr Orthop B* 21(4):366-8.
39. Smith PA, Kuo KN, Graf AN, Krzak J, Flanagan A, Hassani S, Caudill AK, Dietz FR, Morcuende J, Harris GF (2013) Long-term Results of Comprehensive Clubfoot Release Versus the Ponseti Method: Which Is Better? *Clin Orthop Relat Res*. Nov 19. [Epub ahead of print]
40. Watanabe H, Fujita S, Oka H (1992) Polydactyly of the foot: an analysis of 265 cases and a morphological classification. *Plast Reconstr Surg* 89:856-877.
41. Widhe T, Aaro S, Elmstedt E (1988) Foot deformities in the newborn--incidence and prognosis. *Acta Orthop Scand* 59(2):176-9.
42. Wood VE, Peppers TA, Shook J 1997 Cleft-foot closure: a simplified technique and review of the literature. *J Pediatr Orthop* 17(4):501-4.

QUESTIONS:

1. Molding deformities are deformities,
 - a. needing surgical treatment and follow-up.
 - b. connected to the position of the child in utero.**
 - c. which are the result of structural changes.
 - d. Connected to autosomal dominant diseases.
2. Which of the following deformities is a molding deformity?
 - a. clubfoot
 - b. talipes calcaneovalgus
 - c. congenital vertical talus
 - d. preaxial polydactyly
3. In clubfoot treatment using the Ponseti method the foot abduction brace should be used
 - a. for 3 months
 - b. for 2 years
 - c. until approximately 4 years of age
 - d. until the foot looks good
4. What is usually not part of clubfoot treatment using the Ponseti method
 - a. serial casting
 - b. abduction with counter pressure on neck of talus
 - c. a percutaneous Achilles tenotomy
 - d. an ankle foot orthosis (AFO)
5. The incidence of congenital vertical talus is approximately
 - a. 1:100
 - b. 1:1.000
 - c. 1:10.000
 - d. 1:100.000

ANSWERS:

1b, 2b, 3c, 4d, 5c



Prof. Dr. med. Rudolf Ganger PhD

Orthopedic Hospital Vienna Speising

Vienna, Austria

rudolf.ganger@oss.at

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

1.1 DDH in newborns (0 to 1 year)

Since the introduction of hip sonography and its standardization, results of treatment have improved due to early diagnosis of deformities at birth [3]. Every newborn should be screened for signs of DDH or instability. Clinical examinations include the Barlow and Ortolani techniques and require experienced examiners [4]. Ultrasound according to Graf as a screening method is independent to the examiner's experience and skills and provides a tool for early detection and early adequate therapy of DDH. Each sonographic type can be assigned to a specific phase of treatment correlated with a specific procedure effective in the given pathoanatomical situation (Table 1). Splinting therapy should be started up to the beginning of the sixth week of life in order not to miss the best time of treatment [5,6]. In cases, which are detected early, conservative

treatment can be completed before walking age. The α -angle according to Graf should be at least 60°. A radiograph should be performed in every treated child to exclude osteonecrosis of the hip.

Uncritical splinting which is not adapted to the given sonographic pathoanatomical situation of the hip or noncompliance of the parents during the splinting phase often leads to surgery with the need for open reduction even when early diagnosed [7,8].

1.2 DDH after walking age

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

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].

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

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

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.

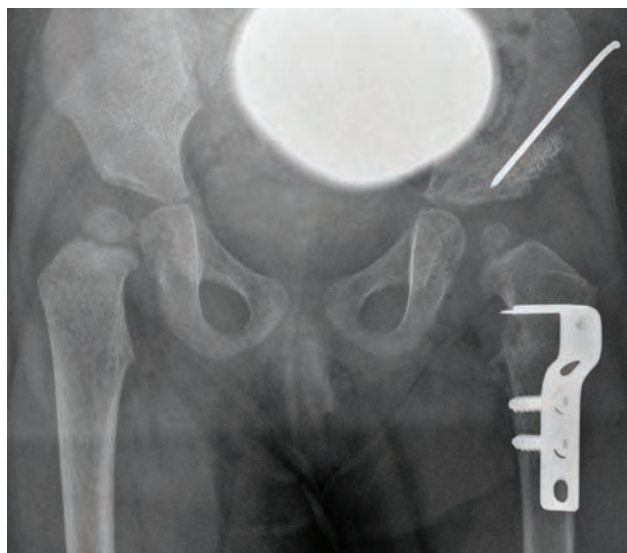


Figure 1b: The reduced left hip of the same girl. Six weeks after open reduction, Pemberton acetabuloplasty and femoral osteotomy (varus, shortening and derotation).

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 osteotomy is often the method of choice to correct hip dysplasia when spherical congruity is present. During this procedure the acetabulum becomes a free, rotating fragment, and overcorrection, impingement and retroversion of the acetabulum can occur. The triple pelvic osteotomy is a challenging procedure and requires an experienced surgeon. After closure of the triradiate cartilage, the periacetabular osteotomy (PAO) according to Ganz can be performed in cases with mild osteoarthritis (Toennis < 2). The PAO is even more challenging than the triple pelvic osteotomy.

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-Ogden system is most widely used to classify ON into four types (grade I to IV)[18].

The treatment goal is to achieve remodeling of the deformed femoral head by concentric reduction and pelvic osteotomy. Additionally, femoral valgus osteotomy, intertrochanteric double osteotomy with trochanteric advancement or greater trochanter transfer are often necessary (Fig. 3a, b).

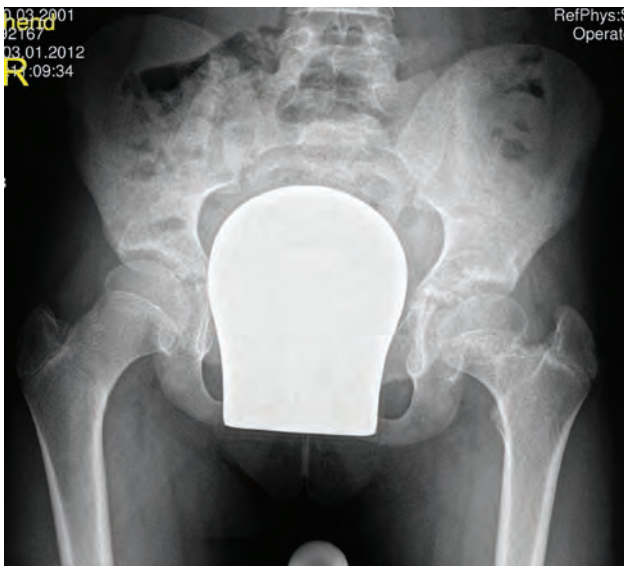


Figure 3a: 11-year-old girl with an closed reduction of the dislocated left hip at the age of five months. At follow up signs of osteonecrosis with a trochanteric overgrowth were evident.



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

2 Transient (toxic) Synovitis

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

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

drugs to expedite spontaneous resolution of the inflammatory synovitis. Clinical symptoms usually resolve completely over several days.

3. Septic Arthritis

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

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

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

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

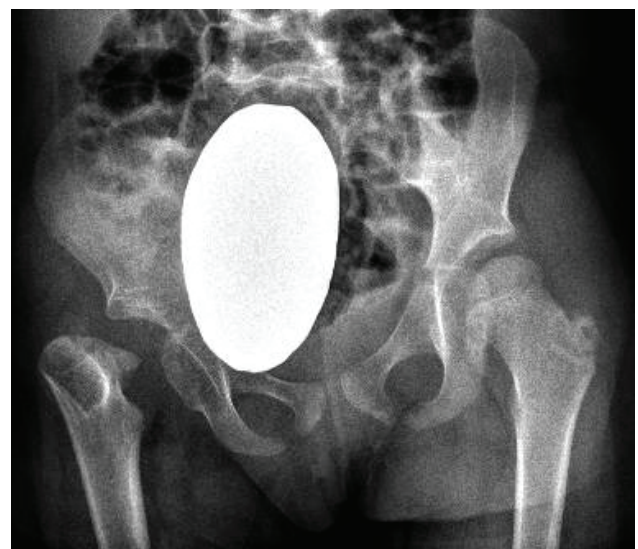


Figure 4: Radiograph of a three-year-old girl after neonatal septic arthritis of the right hip. Complete destruction of the femoral head and neck are visible. Legg-Calve-Perthes Disease (LCPD)

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

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

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):

Several radiographic classification systems are currently used:

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

1. Lateral subluxation of the femoral head
2. Gage sign: a radiolucency in the lateral epiphysis and metaphysis
3. Calcification lateral to the femoral epiphysis
4. Horizontal physal 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 incongruity 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 physal line and the Gage sign are evident.

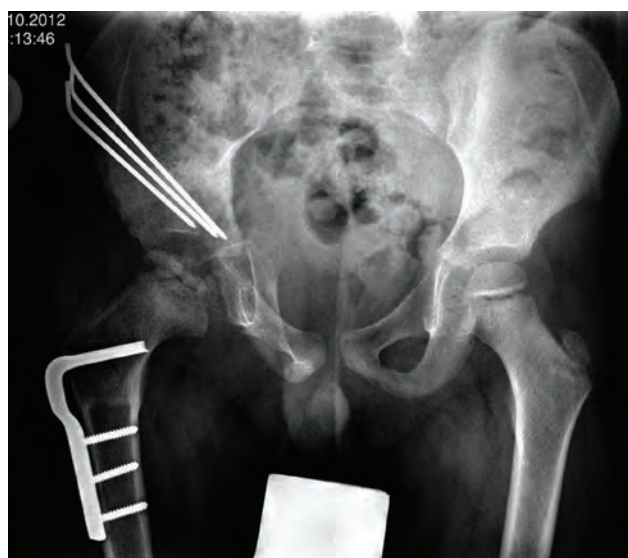


Figure 5b: Radiographs of the same boy, five months after Salter innominate and femoral varus osteotomy. The femoral head is well covered.5. Slipped Capital Femoral Epiphysis (SCFE)

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 metaphysis [25,26]. In early adolescence, the growth plate is weaker and vulnerable, as it carries about four times its body weight. If obesity or trauma is added to the physeal weakness, the growth plate may fail gradually or acutely. SCFE occurs in about 2 in 100 000, most commonly in obese boys [25,26]. It is bilateral in about one-fourth of cases, with possibly slight silent slippage in even more.

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

Treatment can be divided into two categories: treatment to prevent further slippage in mild and moderate cases, and treatment to reduce the degree of slippage in severe slips. Prevention of further slippage in mild and moderate slips can be accomplished by in situ pinning or screw fixation. In situ fixation allows a minimal invasive, percutaneously performed treatment with the goal to stabilize the femoral epiphysis to the femoral neck. The fixation device must be placed perpendicular to the plane of the proximal femoral epiphysis and must be of appropriate strength to avoid failure before physeal plate closure. Almost all SCFEs should be able to be stabilized with

percutaneous placement of a single 6.5- to 7.5-mm cannulated screw [30–32]. Because of the high prevalence of contralateral slip, prophylactic pinning is recommended, especially in patients who have SCFE associated with known metabolic and endocrine disorders [33]. Treatment methods that reduce the degree of slip, and lead to improved motion and function include open reduction and subcapital osteotomy through a Ganz surgical hip dislocation approach, and intertrochanteric osteotomy according to Imhaeuser/Southwick [34,29,35]. The choice of treatment to reduce the degree of slip is based on the surgeon's experience. Ganz surgical hip dislocation and open reduction is a technically challenging procedure (Fig. 6a, b). Intertrochanteric osteotomies remain the most frequently used procedures for realignment in SCFE. Two major complications, osteonecrosis and chondrolysis, are specifically associated with SCFE. After the diagnosis has been made, treatment must be directed at maintaining motion and preventing collapse including anti-inflammatory medication and relieved weight-bearing until healing occurs.



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.

References

- Toennis D (1984) *Die angeborene Hüftdysplasie und Hüftluxation*. Springer, Berlin Heidelberg New York
- Rosendahl K, Markestad T, Lie RT (1994) Ultrasound screening for developmental dysplasia of the hip in the neonate: the effect on treatment rate and prevalence of late cases. *Pediatrics* 94 (1):47-52
- Graf R (2006) *Hip Sonography. Diagnosis and Treatment of Infant Hip Dysplasia*. Springer, Heidelberg
- Barlow TG (1963) Early Diagnosis and Treatment of Congenital Dislocation of the Hip. *Proc R Soc Med* 56:804-806
- Matthiessen HD (1996) Forensic problems in the treatment of hip dysplasias and dislocations. *Z Orthop Ihre Grenzgeb* 134 (6):Oa10-12
- Matthiessen HD (1997) Dysplasia and therapy factors in hip developmental disorders. *Z Orthop Ihre Grenzgeb* 135 (1):Oa12-13
- Grill F, Muller D (1997) Results of hip ultrasonographic screening in Austria. *Orthopade* 26 (1):25-32
- von Kries R, Ihme N, Oberle D, Lorani A, Stark R, Altenhofen L, Niethard FU (2003) Effect of ultrasound screening on the rate of first operative procedures for developmental hip dysplasia in Germany. *Lancet* 362 (9399):1883-1887.
- Salter RB (1966) Role of innominate osteotomy in the treatment of congenital dislocation and subluxation of the hip in the older child. *J Bone Joint Surg Am* 48 (7):1413-1439
- Galpin RD, Roach JW, Wenger DR, Herring JA, Birch JG (1989) One-stage treatment of congenital dislocation of the hip in older children, including femoral shortening. *J Bone Joint Surg Am* 71 (5):734-741
- Lindstrom JR, Ponseti IV, Wenger DR (1979) Acetabular development after reduction in congenital dislocation of the hip. *J Bone Joint Surg Am* 61 (1):112-118
- Schoenecker PL, Strecker WB (1984) Congenital dislocation of the hip in children. Comparison of the effects of femoral shortening and of skeletal traction in treatment. *J Bone Joint Surg Am* 66 (1):21-27
- Kim HT, Kim JI, Yoo CI (2000) Acetabular development after closed reduction of developmental dislocation of the hip. *J Pediatr Orthop* 20 (6):701-708
- Albinana J, Dolan LA, Spratt KF, Morcuende J, Meyer MD, Weinstein SL (2004) Acetabular dysplasia after treatment for developmental dysplasia of the hip. Implications for secondary procedures. *J Bone Joint Surg Br* 86 (6):876-886
- Toennis D (1987) *Congenital dysplasia and dislocation of the hip in children and adults*. Springer, Berlin Heidelberg
- Pospischill R, Weninger J, Ganger R, Altenhuber J, Grill F (2012) Does open reduction of the developmental dislocated hip increase the risk of osteonecrosis? *Clin Orthop Relat Res* 470 (1):250-260.
- Firth GB, Robertson AJ, Schepers A, Fatti L (2010) Developmental dysplasia of the hip: open reduction as a risk factor for substantial osteonecrosis. *Clin Orthop Relat Res* 468 (9):2485-2494.
- Bucholz R, Ogden J. (1978) Patterns of ischemic necrosis of the proximal femur in nonoperatively treated congenital hip diseases. *The Hip: Proceedings of the Sixth Open Scientific Meeting of the Hip Society*. CV Mosby, St. Louis
- Morrey BF, Bianco AJ, Jr, Rhodes KH (1975) Septic arthritis in children. *Orthop Clin North Am* 6 (4):923-934
- Morrey BF, Peterson HA (1975) Hematogenous pyogenic osteomyelitis in children. *Orthop Clin North Am* 6 (4):935-951
- Catterall A, Pringle J, Byers PD, Fulford GE, Kemp HB, Dolman CL, Bell HM, McKibbin B, Ralis Z, Jensen OM, Lauritzen J, Ponseti IV, Ogden J (1982) A review of the morphology of Perthes' disease. *J Bone Joint Surg Br* 64 (3):269-275
- Salter RB, Thompson GH (1984) Legg-Calve-Perthes disease. The prognostic significance of the subchondral fracture and a two-group classification of the femoral head involvement. *J Bone Joint Surg Am* 66 (4):479-489
- Herring JA, Neustadt JB, Williams JJ, Early JS, Browne RH (1992) The lateral pillar classification of Legg-Calve-Perthes disease. *J Pediatr Orthop* 12 (2):143-150
- Catterall A (1971) The natural history of Perthes' disease. *J Bone Joint Surg Br* 53 (1):37-53
- Kelsey JL, Keggi KJ, Southwick WO (1970) The incidence and distribution of slipped capital femoral epiphysis in Connecticut and Southwestern United States. *J Bone Joint Surg Am* 52 (6):1203-1216
- Loder RT (1996) The demographics of slipped capital femoral epiphysis. An international multicenter study. *Clin Orthop Relat Res* (322):8-27
- Aronson J, Tursky EA (1996) The torsional basis for slipped capital femoral epiphysis. *Clin Orthop Relat Res* (322):37-42
- Loder RT, Richards BS, Shapiro PS, Reznick LR, Aronson DD (1993) Acute slipped capital femoral epiphysis: the importance of physeal stability. *J Bone Joint Surg Am* 75 (8):1134-1140
- Southwick WO (1967) Osteotomy through the lesser trochanter for slipped capital femoral epiphysis. *J Bone Joint Surg Am* 49 (5):807-835
- Goodman WW, Johnson JT, Robertson WW, Jr. (1996) Single screw fixation for acute and acute-on-chronic slipped capital femoral epiphysis. *Clin Orthop Relat Res* (322):86-90
- Herman MJ, Dormans JP, Davidson RS, Drummond DS, Gregg JR (1996) Screw fixation of Grade III slipped capital femoral epiphysis. *Clin Orthop Relat Res* (322):77-85
- Koval KJ, Lehman WB, Rose D, Koval RP, Grant A, Strongwater A (1989) Treatment of slipped capital femoral epiphysis with a cannulated-screw technique. *J Bone Joint Surg Am* 71 (9):1370-1377
- Jerre R, Billing L, Hansson G, Wallin J (1994) The contralateral hip in patients primarily treated for unilateral slipped upper femoral epiphysis. Long-term follow-up of 61 hips. *J Bone Joint Surg Br* 76 (4):563-567
- Imhauser G (1954) [Surgical treatment of pathological anteversion of the proximal femur]. *Z Orthop Ihre Grenzgeb* 85 (3):395-405
- Leunig M, Slongo T, Kleinschmidt M, Ganz R (2007) Subcapital correction osteotomy in slipped capital femoral epiphysis by means of surgical hip dislocation. *Oper Orthop Traumatol* 19 (4):389-410.

QUESTIONS

1. The appearance of an acute osteomyelitis in childhood
 - a. is mainly induced by a focus of the gastrointestinal area
 - b. is most often caused by Streptococcus
 - c. is typically located in the diaphyseal area of long bones
 - d. is most often located in the metaphyseal area of long bones**
 - e. is never penetrating to adjacent joints

2. Which criterion may be cause of an unfavorable results of Perthes disease?
 - a. Appearance before age 4
 - b. Initial joint effusion
 - c. Type Herring C**
 - d. Reduced abduction during fragmental period
 - e. Conservative treatment for type Catteral II

3. The Bernese periacetabular osteotomy for correction of hip dysplasia is recommended after maturity because of
 - a. Tendency to overcorrection
 - b. Involvement of the triradiate cartilage**
 - c. Tendency to undercorrection
 - d. Danger of impingement
 - e. To complicated during growth

4. Which condition CANNOT be found in cause of Femoroacetabular Impingement (FAI)?
 - a. Lack of acetabular coverage**
 - b. Acetabular retroversion
 - c. Protrusion of the femoral head
 - d. Cross-over sign
 - e. Acetabular over-coverage

5. What is the most common age group for Kingella kingae infection?
 - a. Infants less than 6 months of age
 - b. Children above 4 years
 - c. Children between 6 months and 4 years**
 - d. Teenagers
 - e. Adults

ANSWERS:

1d, 2c, 3b, 4a, 5c.



Dr. med. Moritz Tannast

Department of Orthopaedic Surgery, Inselspital,
Bern University Hospital, University of Bern,
Bern, Switzerland

moritz.tannast@insel.ch

Hip: Osteotomy and Arthroplasty

A. Osteotomies and Osteochondroplasties

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 (figure 1)[2, 3].

However long-term results 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.

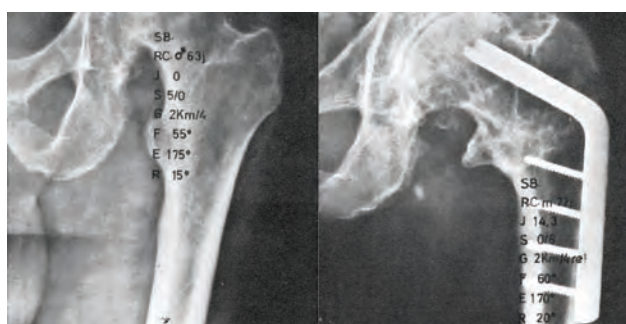


Figure 1) – Figure 1a) shows the radiograph of a 63 year-old farmer with an arthritic left hip joint. The patient was treated with an intertrochanteric varus osteotomy. Figure 1b) shows the radiographic result 14 years postoperatively.

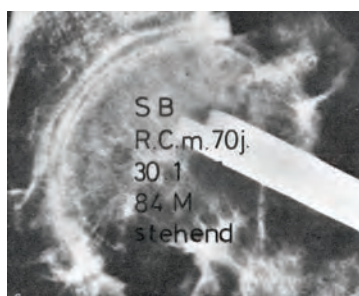


Figure 1c) demonstrates cartilage remodeling on the acetabular, as well as the femoral side at 7 years postoperatively. Image reprinted with permission from [2].

Osteochondroplasties around the hip

Nearly a decade ago the concept of femoroacetabular impingement (FAI) as 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

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

The treatment concept is based predominantly on:

- Trimming of abnormal bone at the femoral and/or acetabular side
- and/or
- 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:

- Open surgery via a safe surgical dislocation of the hip [7]
- Combined minimal anterior approach and hip arthroscopy [8]
- 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.

Osteotomies on both sides of the hip joint

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

- Spherical or rotational osteotomies [17, 18]
- Triple osteotomy [19]
- Bernese Periacetabular Osteotomy [20]

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

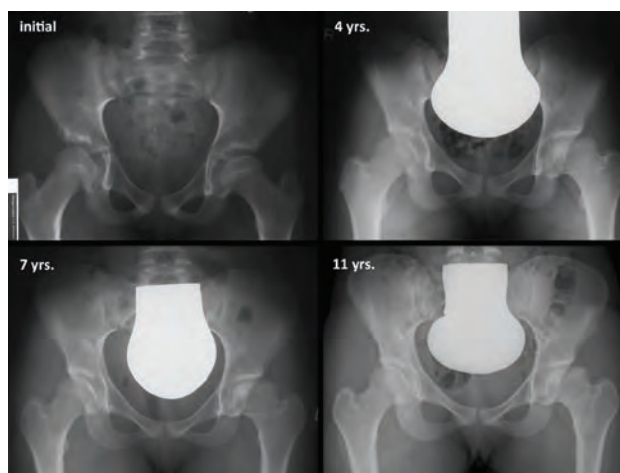


Figure 2a) A consecutive series of anteroposterior pelvis radiographs acquired over an 11-year period are shown. On initial presentation (upper left), this 10 year-old female patient presented with clinical symptoms and radiographic evidence of Perthes disease and concomitant marginal hip dysplasia on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

risers 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 (figure 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.

Table1.) 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.

Study (Year)	Surgical Technique	Number of Patients [hips]	Mean Patient Age [years]	Mean Followup [months]	Scores Employed	Score Improvement at Last Followup	Revision Surgery (other than THA)	Failure (Conversion to THA)
Naal et al. (2012)	Open	185 [233]	30 ± 9 (14–55)	60.7 ± 11.8 (24–120)	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)	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)	Combined	97 [100]	33 (16– 56)	4.9 (2.4–8.7)	NAHS	29.1 points	16 (16%)	11 (11%)
Clohisy et al. (2010)	Combined	35 [35]	34 (16–48)	2.2 (2–3)	mod. HHS NAHS UCLA	23.6 points 15.1 points 2.3 points	None	None
Byrd and Jones (2009)	HAS	200 [207]	33 (w/o range)	16 (12–24)	mod. HHS	20 points	3 (1.4%)	1 (0.5%)
Horisberger et al. (2010)	HAS	88 (105)	41 (17–66)	2.3 (1.3–4.1)	NAHS	27.8 points	None	9 (9%)

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

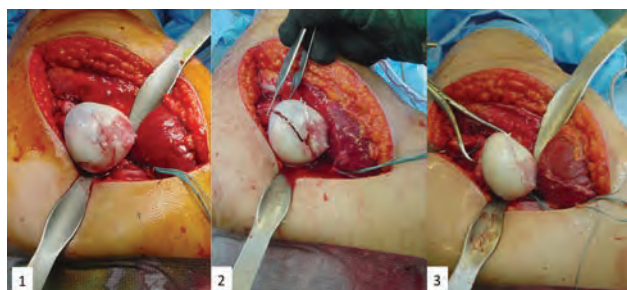


Figure 2b) Intraoperative images of the same patient introduced in figure 2a) are shown. At 22 years of age, a surgical hip dislocation with an extended retinacular soft-tissue flap was performed, allowing for inspection and remodeling of the deformed femoral head. In this case, the subsequent steps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



Figure 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.

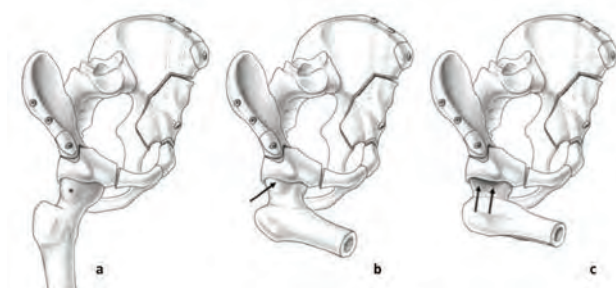


Figure 3.) The drawings depict a pelvis treated with bilateral periacetabular osteotomy for correction of acetabular dysplasia (a). With the acetabulum in a new, more anteriorly covered position, it becomes apparent that a coexistent femoral cam-type deformity leads to an antero-superior impingement 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).

B. Hip Joint Replacement

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].

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].

Resurfacing arthroplasty

Hip resurfacing arthroplasty can be considered a revival of a concept proposed and developed more than 30 years ago by Wagner [38]. The revival was based on the fact that new metal-on-metal bearings might solve the problems of the initial designs [38]. While the idea of bone preservation may be appealing, many advocates for revival of hip resurfacing arthroplasties wrongly advertised potential advantages of this type of joint replacement. The critical side in THAs is not the femoral bone, but the acetabular bone stock, which is not at all better preserved in resurfacing arthroplasties.

Hip resurfacing arthroplasty (HRA) does not lead to a increased range of motion compared to standard THA with 32 mm heads and thus HRA as such can not fulfill the promise of increased physical or athletic activities [39, 40]. Adverse tissue reactions on larger metal-on-metal bearings and femoral neck fractures, virtually unknown in conventional THA with standard bearings, have led to a decrease of indications and number of HRAs. Increasing reports on failures rates have led to the recommendation that HRA is not used in women any more and should only be considered in selected young men, then using a construct with larger femoral heads [32, 41–43].

Surgical approaches

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

References:

1. M.E. Müller, *DIE HÜFTNAHEN FEMUROSTEOTOMIEN. Unter Berücksichtigung der Form, Funktion und Beanspruchung des Hüftgelenkes.* 1957: Georg Thieme Verlag.
2. R. Schneider, *Die intratrochantäre Osteotomie bei Coxarthrose.* 1979, Berlin Heidelberg New York: Springer.
3. F. Pauwels, *Atlas zur Biomechanik der gesunden und kranken Hüfte.* 1973: Springer Verlag, New York.
4. R. Ganz, J. Parvizi, M. Beck, M. Leunig, H. Notzli, and K.A. Siebenrock, *Femoroacetabular impingement: a cause for osteoarthritis of the hip.* *Clin Orthop Relat Res*, 2003(417): p. 112–20.
5. N. Espinosa, D.A. Rothenfluh, M. Beck, R. Ganz, and M. Leunig, *Treatment of femoro-acetabular impingement: preliminary results of labral refixation.* *J Bone Joint Surg Am*, 2006. 88(5): p. 925–35.
6. C.M. Larson, M.R. Givens, and R.M. Stone, *Arthroscopic debridement versus refixation of the acetabular labrum associated with femoroacetabular impingement: mean 3.5-year follow-up.* *Am J Sports Med*, 2012. 40(5): p. 1015–21.
7. M. Lavigne, J. Parvizi, M. Beck, K.A. Siebenrock, R. Ganz, and M. Leunig, *Anterior femoroacetabular impingement: part I. Techniques of joint preserving surgery.* *Clin Orthop Relat Res*, 2004(418): p. 61–6.
8. J.C. Clohisy, L.P. Zebala, J.J. Nepple, and G. Pashos, *Combined hip arthroscopy and limited open osteochondroplasty for anterior femoroacetabular impingement.* *J Bone Joint Surg Am*, 2010. 92(8): p. 1697–706.
9. J.W. Byrd and K.S. Jones, *Arthroscopic management of femoroacetabular impingement.* *Instr Course Lect*, 2009. 58: p. 231–9.
10. J.C. Clohisy, L.C. St John, and A.L. Schutz, *Surgical treatment of femoroacetabular impingement: a systematic review of the literature.* *Clin Orthop Relat Res*, 2010. 468(2): p. 555–64.
11. C.L. Peters, L.A. Anderson, J.A. Erickson, A.E. Anderson, and J.A. Weiss, *An algorithmic approach to surgical decision making in acetabular retroversion.* *Orthopedics*, 2011. 34(1): p. 10.
12. K.A. Siebenrock, R. Schoeniger, and R. Ganz, *Anterior femoroacetabular impingement due to acetabular retroversion. Treatment with periacetabular osteotomy.* *J Bone Joint Surg Am*, 2003. 85-A(2): p. 278–86.
13. K.A. Siebenrock, S.D. Steppacher, P.C. Haefeli, J.M. Schwab, and M. Tannast, *Coxa valga et antetorta cause pain through posterior extraarticular impingement - Manuscript submitted to Clinical Orthopaedics and Related Research - under revision.* 2012.
14. R. Ganz, T.W. Huff, and M. Leunig, *Extended retinacular soft-tissue flap for intra-articular hip surgery: surgical technique, indications, and results of application.* *Instr Course Lect*, 2009. 58: p. 241–55.
15. K. Chiari, *[Pelvic osteotomy in hip arthroplasty].* *Wien Med Wochenschr*, 1953. 103(38): p. 707–9.
16. M. Lance, *Consitution d'une butrée ostéoplastique dans les luxationes subluxations congénitales de la hanche [in french].* *Presse Med*, 1925(33): p. 945–958.
17. S. Ninomiya and H. Tagawa, *Rotational acetabular osteotomy for the dysplastic hip.* *J Bone Joint Surg Am*, 1984. 66(3): p. 430–6.
18. H. Wagner, *Osteotomies for congenital hip dislocation.* In: *The hip Proceedings of the fourth open scientific meeting of the hip society.* 1976. St.Louis: CV Mosby.
19. D. Tonnies, K. Behrens, and F. Tscharni, *A modified technique of the triple pelvic osteotomy: early results.* *J Pediatr Orthop*, 1981. 1(3): p. 241–9.
20. R. Ganz, K. Klaue, T.S. Vinh, and J.W. Mast, *A new periacetabular osteotomy for the treatment of hip dysplasias. Technique and preliminary results.* *Clin Orthop Relat Res*, 1988(232): p. 26–36.
21. S.D. Steppacher, M. Tannast, R. Ganz, and K.A. Siebenrock, *Mean 20-year followup of Bernese periacetabular osteotomy.* *Clin Orthop Relat Res*, 2008. 466(7): p. 1633–44.

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



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 knee function. Patient history, physical examination, radiological and laboratory findings are the diagnostic criteria for knee OA. If, after a well-managed initial conservative treatment^{1,2}. The symptoms are not relieved, surgery should be considered and consists of many options such as: arthroscopic debridement, cartilage repair, osteotomies around the knee and unicompartmental knee arthroplasty (UKA) or total knee arthroplasty (TKA). From those surgical procedures, arthroscopy alone in the management of an arthritic knee has become controversial as a randomised publication in 2002 has shown only minimal benefit in patient outcome^{3,4}.

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 surgery⁵. 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 deformity⁶. 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^{7,8}. Later on, these procedures lost importance due to the success of knee arthroplasty. Also, they were considered as demanding procedures associated with significant complications such as compartment syndrome, peroneal nerve palsy or infection⁹. Still, the development during the last 10 years of new fixation devices^{10,11} (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 selection¹². The stage of OA shall be precisely addressed on radiological studies and if there already is a 4th grade wear (Kellgren–Laurence grading system¹³), only limited pain relief shall be expected. The range of motion is evaluated and at least 120° of flexion and no more than 10° 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. The patient shall be non-smoker. A BMI under 30 gives the best results. Also, the patient shall not suffer from inflammatory diseases such as rheumatoid arthritis¹⁴.

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 total 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 (\pm 2) mm medial to its center^{15–18}. This point is used to quantify the mechanical axis deviation (MAD)⁶ of the lower limb mechanical axis. It may be measured in millimetres from the center of the knee or like Fujisawa¹⁹ described, as a percentage of a medial or lateral compartment (Figure 1d). The anatomical axes of the femur and tibia correspond to the diaphyseal midline of these bones. The mechanical axis of the femur, running from its head to the center of the knee therefore forms an angle of $6 \pm 1^\circ$ with the anatomical axis. The tibia has a mechanical axis nearly identical to the anatomical axis. Under physiological conditions, the tangent line to the femoral condyles is almost parallel to the tangent to the tibial plateau ($0^\circ - 1^\circ$). The standard value of the mechanical medial proximal tibial angle (mMPTA) is of $87^\circ \pm 3^\circ$. On the other hand, the value of the mechanical lateral distal femoral angle (mLDFA) is also of $87^\circ \pm 3^\circ$. These latter values will be used to locate and address the deformity^{15,16} (Figure 1 a–c).

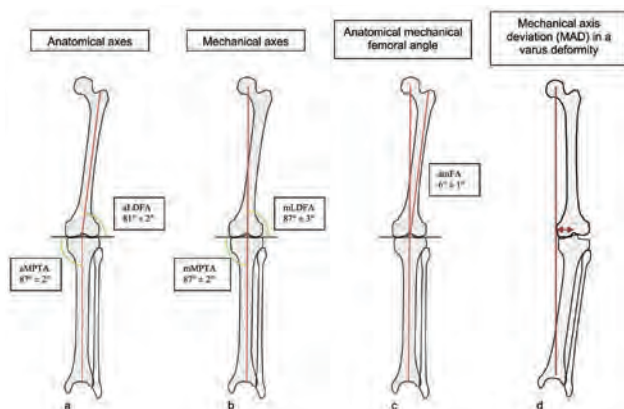


Figure 1 (a-c): Anatomical and mechanical angle values of the femur and the tibia. d Measurement of the MAD (mm) from the knee centre in a varus deviation. aMPTA: anatomical mechanical proximal tibial angle. aL DFA: anatomical lateral distal femoral angle. mMPTA: mechanical medial proximal tibial angle. mL DFA: 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 varus when the weight bearing axis of the lower limb runs medial to the center of the knee and valgus when it runs lateral to the center. The measures of the anatomical and mechanical angles of the distal femur and the proximal tibia are then necessary to point out the location of the deviation because axial deviations may exist due to isolated femur or tibia deformation, or due to a combination of both (Figure 2 a-b)^{6,15-17}. These more complex situations often need double osteotomies around the knee.

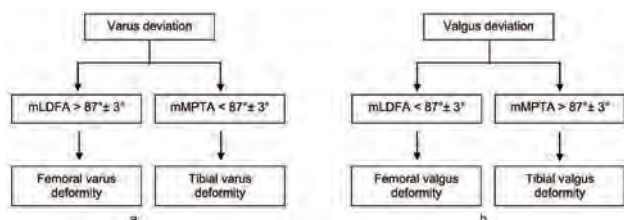


Figure 2 (a-b): Femoral and tibial analysis of varus and valgus deformity

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^{10,11}.

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 planes²⁰⁻²². 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 knee²³. 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. These specific torsional deformities mostly belong to congenital torsion deformities of the lower extremity and are a consequence of growth disorders of the acetabulum, femur, tibia and foot²⁴. These particular conditions are not addressed in this review.

1.6 Preoperative drawing

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 Miniaci¹⁵⁻¹⁹. 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^{19,21}. On the other hand, a valgus deformity is corrected up to neutral²⁵. 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 that is then reported in the proximal metaphyseal portion of the proximal tibia (Figure. 3 a-c).

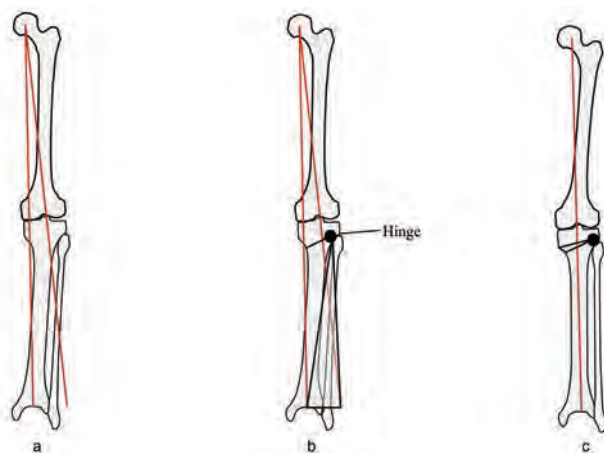


Figure 3 (a-c): Determination of the correction angle in high tibial osteotomy opening wedge (Miniaci)

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 (Figure 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.

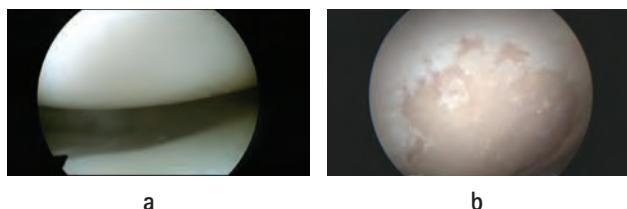


Figure 4: Arthroscopic views of a right knee.
a Lateral compartment.
b Medial compartment with cartilage wear

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-articular 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 of the postero medial corner of the proximal tibia to keep the tibial slope unchanged during the procedure. If necessary, at this stage, anterior or posterior knee instabilities can be treated by varying the tibial slope^{20-22,26,27}.

Once the planned correction is obtained, a definitive fixation is made by the insertion of a plate that is fixed with angular stability screws^{11,12} (Figure 5).



Figure 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. The Australian Orthopaedic Association National Joint Replacement Registry has reported that 43,543 unicompartmental knee procedures have been implanted in 2014 (increase by 4,6% compared to 2013). Osteoarthritis remains the principal diagnosis, accounting for 99% of primary unicompartmental knee replacement. These procedures are most frequently undertaken in patients aged between 55 and 74 years.

2.1 Unicompartmental knee arthroplasty

Unicompartmental knee arthroplasties (UKA) may be used to treat unicompartmental arthritis of either the medial or lateral knee compartment. In about 80% to 90% of patients, the medial compartment is involved and replaced³⁰ (Figure 6). For a successful UKA, the patient selection plays an important role^{28,29}. 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 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 and fewer short term complications³¹.

2.1.1 Outcome

Recent reports have described 10 years survival rates of 80.2% to 98%³². Although, these are respectable results, it is still generally accepted that total knee arthroplasties have a better long term survival. The Australian registry has recorded 4,874 revisions of primary unicompartmental knee replacements. The main reasons for revision are loosening/lysis (44.0%), progression of disease (28.4%) and persistent pain (9.9%). The main type of revision is to a total knee arthroplasty (86.4%)

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.

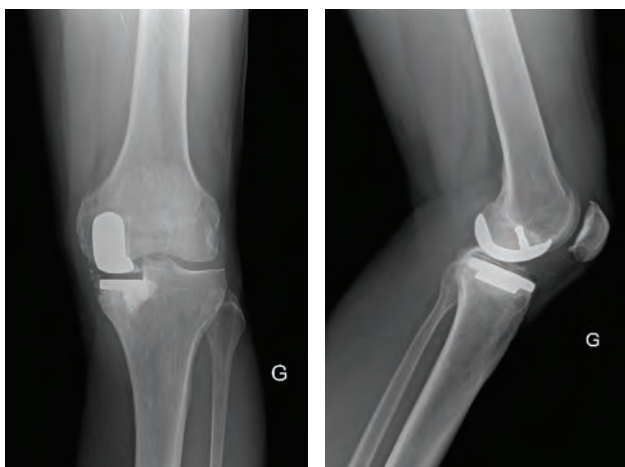


Figure 6: Medial KA

2.2 Patellofemoral arthroplasty

Isolated patellofemoral OA occurs in about 9% of patients over 40 years old and 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^{34,34}. Therefore, TKA should be considered as standard also for patellofemoral OA, especially for elderly patients.

3. Total knee arthroplasty

Total knee arthroplasty is a safe, cost-effective procedure for the management of advanced stage knee OA and results in a significant improvement of life quality. According to the Australian register the rate of implantation is rising (5.2% more in 2014 than in 2013) and the most common diagnosis for primary total knee replacement is osteoarthritis (97.5%), followed by rheumatoid arthritis (1.5%). Prosthetic survival reaches now 90% to 99% at 10 years. It seems to sink at 80% at 15 years and at about 75% after 20 years³⁵⁻³⁹.

Besides these very satisfactory results, some studies document that more than 50% revisions after TKA occur within the first five years^{39,40}.

The main indications for early revising a TKA are the loosening of components, femoropatellar pain, instability and infection. The Australian register reports loosening/lysis as the main reason for revision (28.7%), followed by infection (22.4%), patellofemoral pain (12.1%), pain (8.8%) and instability (6.3%). Infection, even if it is devastating as a complication, is not related to the implant. Also, femoropatellar pain and instability have to deal with errors in the surgical technique. So, as a matter of fact, only loosening may be related to implant selection but also to the surgical technique.

Late causes of revision, essentially polyethylene (PE) wear and osteolysis, are multifactorial in aetiology⁴¹.

Primary total knee replacement for osteoarthritic patients has the lowest revision rate according to the register. At 14 years, the cumulative percent revision for this indication is 7.2%. Age is considered as a major factor affecting the outcome of primary knee arthroplasties. The rate of revision increases with decreasing age. After three years, the register has noted that patients aged less than 55 years have over four times the rate of revision compared to those aged 75 years or older.

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 guides, 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^{42,43}.

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 (CR) and the posterior cruciate substituting. In that latter group, one can differentiate the postero-stabilized (PS) and the ultra-congruent (UC) implants.

3.1 Preoperative planning

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 (Figure 7, Table 1).

Table 1 Preoperative X- Rays

- Standing full-length AP view from 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)

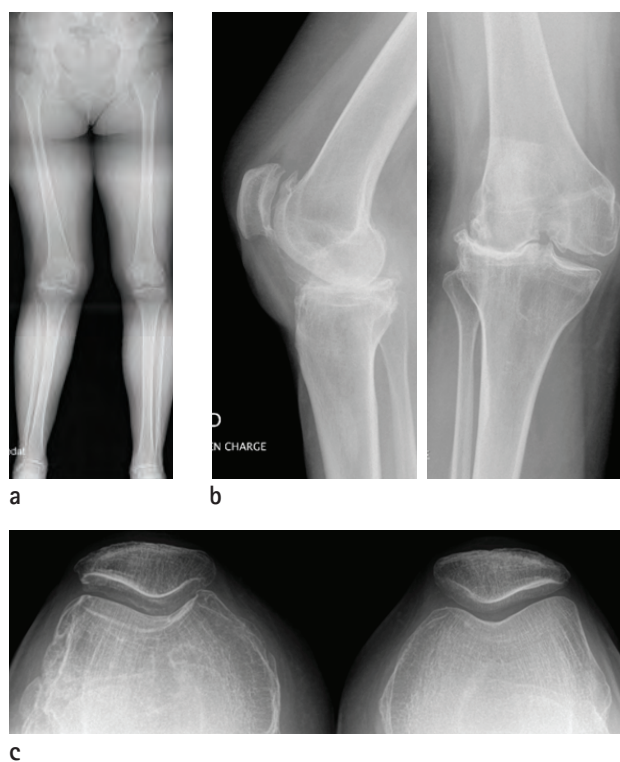


Figure 7 (a-c):

- a Standing antero posterior view
- b Frontal and lateral knee views
- c Patellofemoral view

3.2 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° (Figure 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. The recommended coronal alignment is within 0° to 3° of varus-valgus^{44,45}. Still, Parratte et al have found similar results between TKAs with more than 3° of coronal alignment compared to TKAs within the 3°⁴⁶. So, the debate is not over.

3.3 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. Figgie et al have found that the patella shall be within 10 to 30 mm above the joint line. 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 screw and cement whereas larger defects need metallic augmentation.

3.4 Soft tissue releases

It is probably the most fundamental step in TKA. During the degenerative process, ligaments and soft tissues will become contracted on the concave side of the deformity and stretched to lose on the convex side (Figure 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 and until an adequate balance in extension is reached⁴⁷. In the frontal plane, the anatomical structures to be released in a varus (Table 2) or valgus (Table 3) condition are listed below.

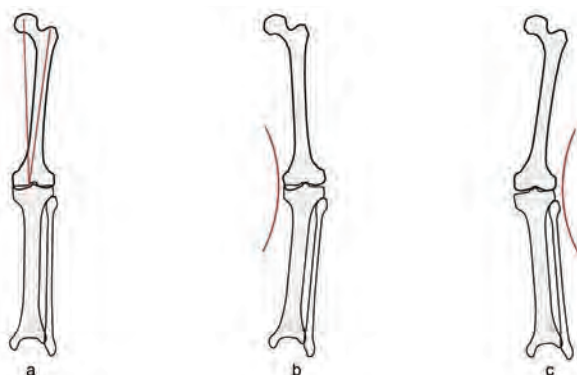


Figure 8 (a–c):

- a Measure of the femoral valgus cut angle
- b Medial contracted structures in varus deformity
- c Lateral contracted structures in valgus deformity

Table 2 Varus Deformity

MEDIAL RELEASE

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

Table 3 Valgus Deformity

LATERAL RELEASE

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

3.5 Exention – flexion gaps

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 perform a correct tissue release and proceed to an adequate amount of bone resection. The knee will be well balanced in the sagittal plane if the tibial insert remains stable during the full range of motion. As a general rule, if the gap problem is symmetric the tibia needs to be adjusted whereas if the gap problem is asymmetric the femur needs an adjustment (Table 4).

Table 4 Sagittal Plane Balancing

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

3.6 Patellofemoral alignment

To prevent patellofemoral maltracking^{50,51} 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 balancing⁴⁹ technique and the measured resection technique⁴⁸. 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 (Figure 9 a-b).



Figure 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.

3.7 Implant fixation

Cemented fixation of TKA (figure 10) is a standard procedure that is reliable and durable 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. An enhancement of the surgical techniques as well as more precise instrumentation has brought cementless implants back to light. Excellent results have been reported in different studies describing equal survivorship and outcomes in bilateral TKAs, one cemented, one non-cemented^{52,53}.

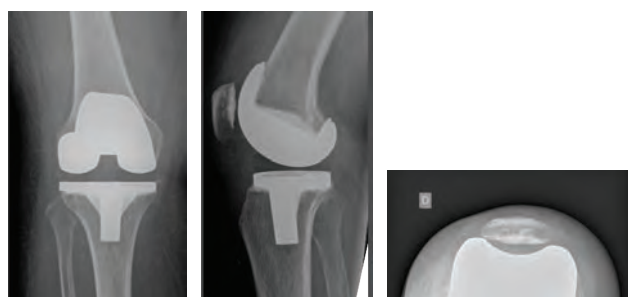


Figure 10: Total knee arthroplasty

4. Conclusion

The choice of a surgical option and the patient selection are the most challenging issues in the treatment of the knee osteoarthritis. It is the surgeon's duty to correctly analyse the stage of osteoarthritis, the ligamentary condition, the type of deformity and reducibility, the age, the range of motion and the expectations of the patient

before proposing a surgery. There is a place for osteotomies around the knee, not only for monocompartmental osteoarthritis but also to address specific knee instability or to protect an ACL reconstruction in younger patients. Unicompartmental knee arthroplasties give also good results but are to be considered as resurfacing surgery and need an optimal comprehension of the lower limb deformity and clinical status. Total knee arthroplasty remains the gold standard for the definitive treatment of knee osteoarthritis. Table 5 lists the ideal patient for each type of surgery.

Table 5 Indications		
OSTEOTOMIES	UKA	TKA
45 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

References

1. Hochberg MC, Altmann RD, April KT, et al: American College of Rheumatology 2012 recommendations for the use of nonpharmacologic and pharmacologic therapies in the osteoarthritis of the hand, hip, and knee. *Arthritis Care Res (Hoboken)* 2012;64(4):465-74.
2. Wilson B, Rankin HBarnes CL: Long-term results of an unloader brace in patients with unicompartmental knee osteoarthritis. *Orthopedics* 2011;34(8):e334-e337
3. Moseley JB, O'Malley K, Petersen NJ, et al: A controlled trial of arthroscopic surgery for osteoarthritis of the knee. *N Engl J Med* 2002;347(2):81-88.
4. Katz JN, Brophy RH, Chaisson CE, et al: Surgery versus physical therapy for a meniscal tear and osteoarthritis. *N Engl J Med* 2013;368(18):1675-84
5. Trousdale RT. Osteotomy. Patient selection, preoperative planning and results. In: Callaghan JJ, Rosenberg AG, Rubash HE, eds. *The Adult Knee*. Philadelphia, A: Lippincott Williams and Wilkins; 2002
6. Paley D, Pfeil J: Principles of deformity corrections around the knee. *Orthopäde* 2000;29(1):18-38.
7. Coventry MB: Osteotomy of the upper portion of the tibia for degenerative arthritis of the knee. A preliminary report. *J Bone Joint Surg* 1965;47:984-90.
8. Coventry MB: Upper tibial osteotomy. *Clin orthop Relat Res* 1983;182:46-52
9. Tunggal JA, Higgins GA, Waddell JP. Complications of closing wedge high tibial osteotomy. *Int Orthop*. 2010;34(2):255-61

10. Lobenhoffer P, Agneskirchner JD. Improvements in surgical technique of valgus high tibial osteotomy. *Knee Surg Sports Traumatol Arthrosc.* 2003;11(3):132-38
11. Staubli AE, De Simoni C, Babst R et al. TomoFix: a new LCP-concept for open wedge osteotomy of the medial proximal tibia—early results in 92 cases. *Injury* 2003;34(2):55-62.
12. Bonnin M, Chambat P. Current status of valgus angle, tibial head closing wedge osteotomy in medial gonarthrosis. *Orthopäde* 2004;33(2):135-42
13. Kellgren JH, Lawrence JS. Radiological assessment of osteoarthritis. *Ann Rheum Dis* 1957;16(4):494-502
14. Lobenhoffer P. Indications for high tibial osteotomy, unicompartmental knee arthroplasty, and total knee prosthesis. In: Lobenhoffer P, van Heerwaarden J, Staubli A, eds. *Osteotomies Around the Knee. Indications-Planning-Surgical Techniques using Plate Fixators.* US-New York, NY: Thieme New York; 2008.
15. Paley D, Herzenberg JE, Tetsworth K, et al. Deformity planning for frontal and sagittal plane corrective osteotomies. *Orthop Clin North Am* 1994;25(3):425-65.
16. Paley D, Tetsworth K. Mechanical deviation of the lower limbs. Preoperative planning of multiapical frontal plane angular and bowing deformities of the femur and tibia. *Clin Orthop Relat Res* 1992;280:65-71
17. Pape D, Seil R, Adam F, et al. Imaging and preoperative planning of osteotomy of tibial head osteotomy. *Orthopäde* 2004;33(2):122-34.
18. Miniaci A, Ballmer FT, Ballmer PM, et al. Proximal tibial osteotomy. A new fixation device. *Clin Orthop Relat Res* 1989;246:250-59.
19. Fujisawa Y, Masuhara K, Shiomi S. The effect of high tibial osteotomy on osteoarthritis of the knee. An arthroscopic study of 54 knee joints. *Orthop Clin North Am* 1979;10(3):585-608.
20. Noyes FR, Barber-Westin SD, Hewett TE. High tibial osteotomy and ligament reconstruction for varus angulated anterior cruciate ligament-deficient knees. *Am J Sports Med* 2000;28(3):282-96.
21. Reichwein F, Nebelung W. High tibial flexion osteotomy for revision of posterior cruciate ligament instability. *Unfallchirurg* 2007;110(7):597-602.
22. Marti CB, Gautier E, Wachtel AW, et al. Accuracy of frontal and sagittal plane correction in open-wedge high tibial osteotomy. *Arthroscopy* 2004;20(4):366-72.
23. Giffin JR, Vogrin TM, Zantop T, et al. Effects of increasing tibial slope on the biomechanics of the knee. *Am J Sport Med* 2004;32(2):376-83.
24. van Heerwaarden RJ, van der Haven, Kooijman M, et al. Derotation osteotomy for correction of congenital rotational lower limb deformities in adolescents and adults. *Surg Tech Orthop Traumatol* 2003;55-575-A-10:10
25. Johnson F, Leitl S, Waugh W. The distribution of load across the knee. A comparison of static and dynamic measurements. *J Bone Joint Surg Br* 1980;62(3):346-49
26. Jacobi M, Wahl P, Jakob RP. Basic principles of osteotomies around the knee. In: Lobenhoffer P, van Heerwaarden J, Staubli A, eds. *Osteotomies Around the Knee. Indications-Planning-Surgical Techniques using Plate Fixators.* US-New York, NY: Thieme New York; 2008
27. Stuart MJ. Tibial antiviral closing wedge osteotomy. *Oper Tech Sport Med* 2000;8(1):27-31
28. Pennington DW, wienckowski JJ, Lutes WB, et al. Unicompartmental knee arthroplasty in patients sixty years of age or younger. *J Bone Joint Surg Am* 2003;85:1968-73
29. Parratte S, Argenson JN, Pearce O, et al. Medial unicompartmental knee replacement in the under-50s. *J Bone Joint Surg Br* 2009;91:351-56
30. Hernborg JS, Nilsson BE. The natural course of untreated osteoarthritis of the knee. *Clin Orthop Relat Res* 1977;123:130-137
31. Noticewala MS, Geller JA, Lee JH, et al. Unicompartmental knee arthroplasty relieves pain and improves function more than total knee arthroplasty. *J Arthroplasty* 2012;27(8, suppl):99-105.
32. Heyse TJ, Khefacha A, Peersman G, et al. Survivorship of UKA in the middle-aged. *Knee* 2012;19(5):585-91
33. Fulkerson JP: Alternatives to patellofemoral arthroplasty. *Clin Orthop Relat Res* 2005;436:76-80
34. van Jonbergen HP, Werkman DM, Barnaart LF et al. Long-term outcomes of patellofemoral arthroplasty *J Arthroplasty* 2010;25(7):1066-71
35. Pavone V, Boettner F, Fickert S, Sculo TP: Total condylar knee arthroplasty: Along term follow-up. *Clin Orthop Relat Res* 2001;388:18-25
36. Rand JA, Ilstrup DM: survivorship analysis of total knee arthroplasty: cumulative rates of survival of 9200 total knee arthroplasties. *J Bone Joint Surg Am* 1991;73(3):397-409
37. Schai PA, Thornhill TS, Scott RD: Total knee arthroplasty with the PFC system: Results at a minimum of ten years and survivorship analysis. *J Bone Joint Surg Br* 1998;80(5):850-58
38. Ranawat CS, Flynn WF Jr, Saddler S, et al. Long-term results of the total condylar knee arthroplasty: A 15-years survivorship study. *Clin Orthop Relat Res* 1993;286:94-102
39. Gioe TJ, Killeen KK, Grimm K, et al. Why are total replacement revised? Analysis of early revision in a community knee implant registry. *Clin Orthop Relat Res* 2004;428:100-06
40. Sharkey PF, Hozack WJ, Rothman RH, et al. Why are total knee arthroplasties failing today? *Clin Orthop Relat Res* 2002;404:7-13
41. Lidgren L, Robertsson O: Annual report 2008: The Swedish Knee Arthroplasty Register. Lund, Sweden, Wallin & Dalholm, 2008
42. Lin WP, Lin J, Horng LC, et al. Quadriceps sparing, minimal-incision total knee arthroplasty: A comparative study. *J Arthroplasty* 2009;24(7):1024-32
43. Matsumoto T, Muratsu H, Kubo S, et al. soft tissue balance measurement in minimal incision surgery compared to conventional total knee arthroplasty. *Knee Surg Sports Traumatol Arthrosc* 2011;19(6):880-86
44. Fang DM, Ritter MA, Davis KE. Coronal alignment in total knee arthroplasty: just how important is it? *J Arthroplasty* 2009;24:39-43
45. Jeffery RS, Morris RW, Denham RA. Coronal alignment after total knee replacement. *J Bone Joint Surg Br* 1991;73:709-14
46. Parratte S, Pagnano MW, Trousdale RT, et al. Effect of postoperative mechanical axis alignment on the fifteen-year survival of modern, cemented total knee replacements. *J Bone Joint Surg Am.* 2010;92:2143-49

47. Verdonk PC, Pernin J, Pinaroli A, et al. Soft tissue balancing in varus total knee arthroplasty: an algorithmic approach. *Knee Surg Sports Traumatol Arthrosc* 2009;17:660-66
48. Hungerford DS, Krackow KA. Total joint arthroplasty of the knee. *Clin Orthop Relat Res* 1985;192:23-33
49. Dennis DA, Komistek RD, Kim RH, et al. Gap balancing versus measured resection technique for total knee arthroplasty. *Clin Orthop Relat Res* 2010;468:102-07
50. Berger RA, Rubash HE. Rotational instability and malrotation after total knee arthroplasty. *Orthop Clin North Am* 2001;32:639-47
51. Rhoads DD, Noble PC, Reuben JD, et al. The effect of femoral component on the kinematics of total knee arthroplasty. *Clin Orthop Relat Res* 1993;286:122-29
52. Park JW, Kim YH. Simultaneous cemented and cementless total knee arthroplasty in the same patients: A prospective comparison of long-term outcomes using an identical design of kexGen prosthesis. *J Bone Joint Surg Br* 2011;93-B(11):1479-86
53. Hofmann AA, Evanich JD, Ferguson RP, et al. Ten-to 14-years clinical followup of the cementless Natural Knee system. *Clin Orthop Relat Res* 2001;388:85-94

QUESTIONS

A fifty-six years old man is complaining of medial knee pain for over 12 months. He has undergone physical therapy without any improvement. He needs daily medication and complains also of night pain. He has undergone a sub total medial menisectomy of that knee 10 years ago. Clinical examination reveals a knee function of 130°-10°-0°, with light effusion. There is a fixed varus deformity. Femoropatellar joint slightly symptomatic.

Standard knee X-ray views show medial femoro-tibial heavy arthritis. Other compartments do not show many degenerative signs. The varus deformity is 9° on the long leg view. The mMPTA is about 83°.

1. If this patient is non smoker and heavy laborer what would be your surgical option?
 - a. High tibial valgisation osteotomy alone
 - b. High tibial valgisation osteotomy and arthroscopy of the knee joint
 - c. Medial unicompartmental knee arthroplasty
 - d. Total knee arthroplasty
 - e. Distal femoral valgisation osteotomy
2. If this patient suffers from an inflammatory disease (polyarthritis rheumatoid) for many years and is overweighted, what would be your surgical option?
 - a. High tibial valgisation osteotomy alone
 - b. High tibial valgisation osteotomy and arthroscopy of the knee joint
 - c. Medial unicompartmental knee arthroplasty
 - d. Total knee arthroplasty
 - e. Distal femoral valgisation osteotomy

3. If this patient would be a heavy smoker, thin and office worker, what would be your surgical option?
 - a. High tibial valgisation osteotomy alone
 - b. High tibial valgisation osteotomy and arthroscopy of the knee joint
 - c. Medial unicompartmental knee arthroplasty
 - d. Total knee arthroplasty
 - e. Distal femoral valgisation osteotomy

During the implementation of a postero stabilised (PS) total knee arthroplasty, you realize that there is an asymmetric gap between flexion and extension.

4. The extension is good and well balanced but the flexion is too tight. What is your optimal choice?
 - a. Decrease the size of the polyethylene insert
 - b. Decrease size of the femur, keeping the anterior reference
 - c. Cut more tibia
 - d. Cut more femur
 - e. Use a thicker polyethylene insert

During the implementation of a postero stabilised (PS) total knee arthroplasty, you realize that there is a symmetric gap between flexion and extension.

5. The flexion and extension are too loose. What is your optimal choice?
 - a. Decrease the size of the polyethylene insert
 - b. Decrease size of the femur, keeping the anterior reference
 - c. Cut more tibia
 - d. Cut more femur
 - e. Use a thicker polyethylene insert

ANSWERS:

1b, 2d, 3c, 4b, 5e



Prof. Dr. Xavier Crevoisier

CHUV, University Hospital of Lausanne, Switzerland
Lausanne, Switzerland
xavier.crevoisier@chuv.ch

Ankle Osteoarthritis, Adult Acquired Flatfoot Deformity and Hallux Valgus

ABSTRACT

Ankle osteoarthritis most commonly occurs as a consequence of trauma. Ankle arthrodesis and total ankle replacement (TAR) are the most common surgical treatments of end stage ankle osteoarthritis. Both surgeries result in similar clinical improvement at midterm. However, gait analysis has demonstrated the superiority of TAR over arthrodesis. There is a non union rate of 5–10% after arthrodesis and a 10–60% occurrence of osteoarthritis in the neighbor joints at long term. There is a trend towards less invasive approaches, especially arthroscopic ankle arthrodesis. TAR are associated with a 1% annual failure rate and a 3% annual revision rate. Main causes for failure are aseptic loosening and cyst formation. Today there is a trend towards implantation of fixed bearing prostheses, research about optimal design and coating, and design of revision implants. More recently conservative surgery (extra articular alignment osteotomies) around the ankle has gained popularity in treating early- to midstage ankle osteoarthritis.

There are two major categories of flatfoot: a) the constitutional flatfoot; b) the acquired adult flatfoot deformity (AAFD). Our purpose is to address the acquired adult flatfoot deformity (AAFD). In 80% of cases AAFD is the consequence of a posterior tibial tendon dysfunction (PTTD). Classification of the AAFD is based upon the function of the tibialis posterior tendon, the reducibility of the deformity and the presence or not of degenerative changes at the ankle joint. Conservative treatment includes orthotics for all stages and eccentric muscle training for the early stages. Functional surgery is indicated for the treatment of the early stages, i.e. as long as the deformity is flexible. In case of fixed deformity corrective and stabilizing surgery is performed.

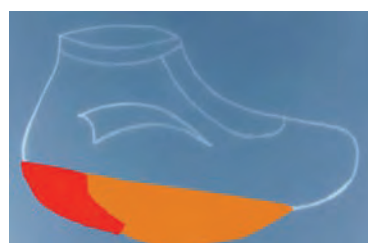
The hallux valgus (HV) deformity is the first reason to visit a F&A surgeon in Europe. It is more common in women (women:men = 8:1). The etiology and pathogenesis of hallux valgus are multifactorial. Analysis of HV requires subtle clinical and radiological examination. Conservative treatment can alleviate pain but is unable to correct the deformity. Surgical treatment must be adapted to the type and severity of the deformity. Success of surgical treatment ranges from 80 to 90% and complication rate ranges from 10 to 30%. The difficulty to fully understand the pathology partially explains these results and also the availability of multiple techniques to treat the same pathology.

ANKLE OSTEOARTHRITIS

Ankle osteoarthritis most commonly occurs as a consequence of trauma. In 2008, Glazebrook M. et al. have stated that "The mental and physical disability associated with end-stage ankle arthrosis is at least as severe as that associated with end-stage hip arthrosis". Conservative treatment includes medication, bracing and shoe modifications. Ankle arthrodesis and total ankle replacement (TAR) are the most common surgical treatments of end stage ankle osteoarthritis. More recently conservative surgery (extra articular alignment osteotomies) around the ankle has gained popularity in treating early- to midstage ankle osteoarthritis, especially in case of asymmetric osteoarthritis of the ankle joint.

CONSERVATIVE TREATMENT

Conservative treatment is mainly indicated for early stage ankle osteoarthritis, especially in young patients in whom we aim to avoid early definitive surgery like TAR or arthrodesis. Of course, this applies also to older patients whose general health condition doesn't allow surgery. Conservative treatment includes pain killers and non steroidal anti-inflammatory drugs (NSAID), intra articular corticosteroids injection, braces and shoe wear modifications. Shoe modifications typically include a rocker bottom sole and shock absorbing heel as well as a construct for medio-lateral and sagittal ankle stabilization (Figure 1).



A



B

Figure 1: Shoe modifications typically include a rocker bottom sole and shock absorbing heel (A) as well as a construct for medio-lateral and sagittal ankle stabilization (B).

ANKLE ARTHRODESIS

Ankle arthrodesis (AA) has been described by Albert in 1879. During almost one century it has been considered as the gold standard for surgical treatment of end stage ankle osteoarthritis. Relative contraindications include advanced osteoarthritis of the neighbor joints, rheumatoid arthritis, arteriopathy, and poor soft tissues conditions. In the two first cases a total ankle replacement should be discussed and, in the two last cases, a pluridisciplinary approach involving vascular and plastic surgeons is required.

Several techniques have been described, including closed, open, and arthroscopic procedures. Open procedures can be performed through lateral, anterior or posterior approaches. Having the choice between these different approaches is useful in case of fragile soft tissues conditions. There exist also many fixation techniques including, casting alone, external fixation, internal fixation with screws, plates, and/or staples (Figure 2). Positioning of the fused ankle is very important and should include 5-10° valgus of the hind foot, 5-10° external rotation, and a neutral position in the sagittal plane. Respecting this position allows the hind foot complex for optimization of compensatory motion resulting in more physiological gait pattern.



Figure 2: There exist many fixation techniques for ankle arthrodesis. (A) ap view of an arthrodesis of the left ankle with an external fixation using the Charnley frame. (B) ap and lateral views of an arthrodesis of the right ankle with crossed screws internal fixation.

Ankle arthrodesis has been demonstrated to result in significant pain alleviation and good functional results. In the past years there is a trend towards arthroscopic ankle arthrodesis because it has been shown to result in shorter hospital stay, lower initial complication rate and better early outcome. Performing arthroscopic ankle arthrodesis in case of severe deformity, however, may be challenging.

Major drawbacks associated with AA, as described in the literature over the last 40 years, are the 10-60% rate of occurrence of osteoarthritis in the neighbor joints at long term, a non-union rate of 10-20% (much closer to 5-10% with the modern fixation techniques), and an infection rate of 3-25% (much closer to 2-5% today).

TOTAL ANKLE REPLACEMENT (TAR)

The first ankle arthroplasty, rather encountered as total ankle replacement (TAR) in the literature, has been performed in the early 1970 and consisted in the implantation of an inverted hip prosthesis into the tibia and a cemented acetabular cup in the calcaneus. After a difficult start due to a high complication rate,

mostly associated with inadequate implants design and fixation, ankle arthroplasty has evolved during the past 20 years and is now challenging ankle arthrodesis as an alternative treatment for end stage ankle osteoarthritis.

Careful patient's selection is even more important for the success of TAR than the technical features. Indications for TAR include end stage osteoarthritis, rheumatoid arthritis, low to moderate functional demand, sufficient bone quality, healthy soft tissue envelop, and well aligned ankle – hind foot. Relative contra indications are high functional demand, diabetes, previous septic ankle arthritis. According to recent publications, overweight or malalignment (if causal adjunctive procedure is performed) do not impair the results. Absolute contra indications include active or recent septic ankle arthritis, talar or distal tibia necrosis, unstable soft tissue envelope, neuroarthropathy, and morbid arteriopathy.

Today, the concept including anatomical design, economic bone resection, non-cemented fixation, three components, and semi-constrained mechanics is generally accepted regarding the implants design (Figure 3). The advantages of fixed bearing versus mobile bearing implants are not yet established. However, since the American market is the more voluminous and since there exist no FDA approval for mobile bearing ankle prostheses in the USA there is a natural trend towards the use of fixed bearing implants. Furthermore, even if not definitively proven, fixed bearing implants are thought to reduce periprosthetic cysts formation and, thus, increase implant survival rate.

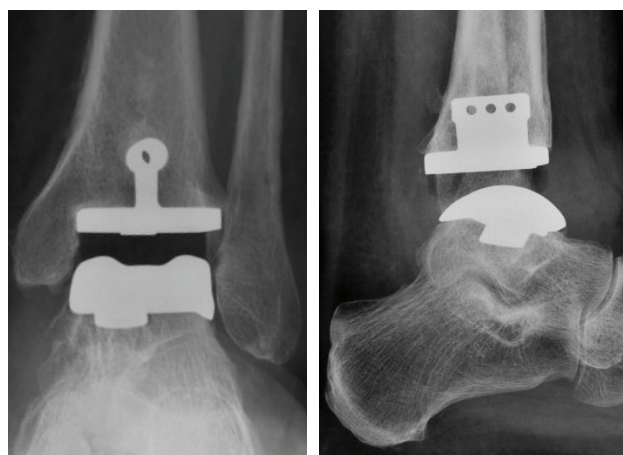


Figure 3: Ap and side views of a three components, semi-constrained, cementless, fixed bearing ankle prosthesis of the left ankle.

Preoperative procedure requires careful clinical examination of the foot and ankle as well as of the lower limbs, weight bearing x-rays of the ankle, and CT-scan. Implantation planning can be performed on x-rays or on CT-scan using 2D drawing templates or computer assisted 3D reconstruction. An anterior approach is usually performed, i.e. between the EHL and the tibialis anterior tendons. Few prostheses are designed to be implanted through a lateral, trans-malleolar approach. Postoperative care usually includes the application of a below knee walking cast for up to 6 weeks associated with partial weight bearing followed by a physiotherapy program to optimize ankle motion, strength, and proprioception.

In terms of function and pain alleviation midterm results after TAR are equivalent to those following ankle arthrodesis. In terms of biomechanics neither the arthrodesis nor the TAR is able to

restore normal gait. Nevertheless, TAR is associated with a more physiological gait pattern than arthrodesis.

Drawbacks associated with TAR include, at first, and based upon several national registers, the low implant survival rate when compared to prosthetic replacement of other weight bearing joints. In fact, the average survival rate is 80% at 10 years, 90% in the best cases. Additionally, the revision rate is particularly high: 10-20% at five years and 20-40% at 10 years. Most frequent complications include primary or secondary malleolar fractures (0-10%), loosening (5-20%), and infections (2-4%). Periprosthetic cysts formation (Figure 4) has been recognized as a major problem in the past few years; they are encountered in up to 60% of cases and their origin is still not clearly established. Based upon the low survival rate and the shortcomings of TAR, optimizing implants design and also conception of revision implants have now become an important field of research. CT-based custom made resection guides also become an interesting development.

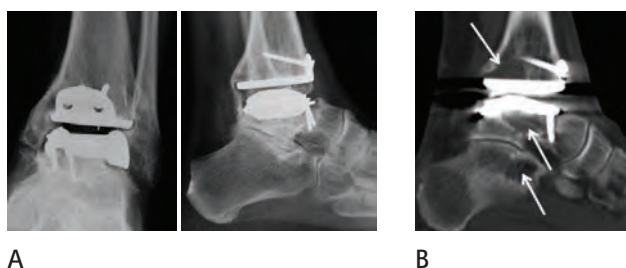


Figure 4: Periprosthetic cysts formation has become a major concern in total ankle replacement. They cannot always been recognized on conventional x-rays (A) and require a CT-scan to be quantified (B).

CONSERVATIVE SURGERY

Conservative surgery includes arthroscopic debridement, extra articular realignment osteotomies and ligament re-balancing.

Arthroscopic debridement is indicated in early stages, where the ankle alignment and the thickness of the cartilage are still quite preserved and where pain alleviation and optimization of motion can be attempted through resection of osteophytes and loose bodies. However, there is no evidence that arthroscopic debridement will stop the progression of osteoarthritis.

Extra articular realignment osteotomies are indicated in early stage ankle osteoarthritis and can address both osteoarthritis based on extra articular deformities (Figure 5) and osteoarthritis associated with intra articular deformities (Figure 6). In the second case they can be associated with ligament re-balancing and/or tendon transfers in order to aim not only anatomical but also dynamic correction. Recent publications have reported promising results associated with this surgery in terms of functionality and pain alleviation, some of them have even reported that it is able to stop the progression of osteoarthritis.

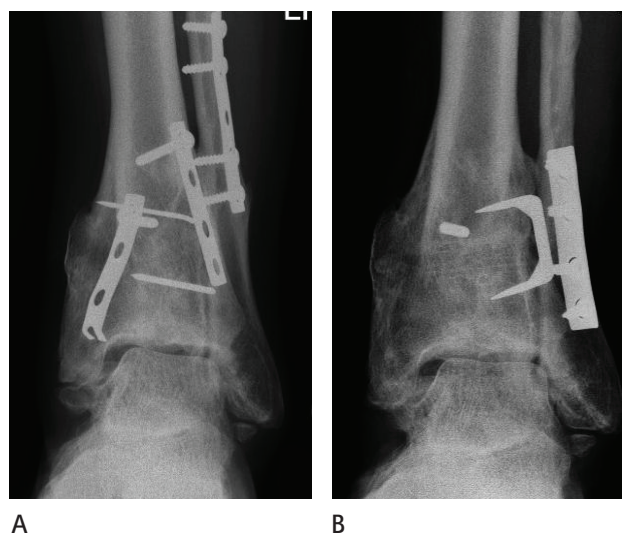


Figure 5: AP views of weight bearing x-rays of the left ankle of a 51 year old man before (A) and one year after (B) supramalleolar lateral closing wedge osteotomy and deltoid ligament release for early stage post traumatic varus ankle osteoarthritis based on extra articular deformity.

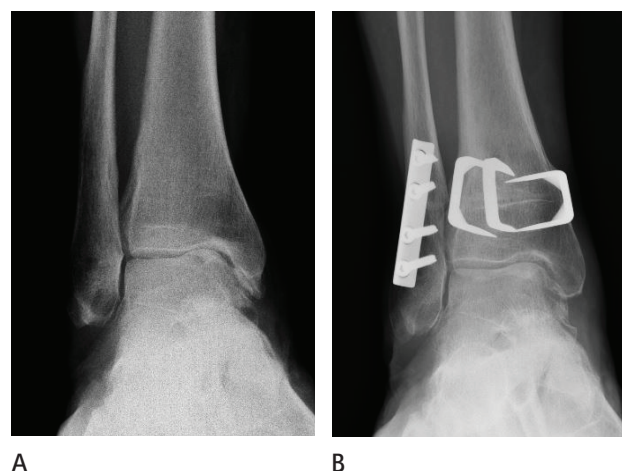


Figure 6: AP views of weight bearing x-rays of the right ankle of a 60 year old woman before (A) and two years after (B) supramalleolar lateral closing wedge osteotomy and deltoid ligament release for early stage varus ankle osteoarthritis associated with intra articular deformity. Note that, due to the lateral shift of the weight bearing axis, the narrowing of the medial joint space has been addressed successfully.

FLATFOOT

There are two major categories of flatfoot deformity: a) the constitutional flatfoot; b) the acquired adult flatfoot deformity (AAFD). The constitutional flatfoot is usually functional and asymptomatic while the acquired flatfoot is a pathologic condition. Our purpose is to address the acquired adult flatfoot deformity (AAFD).

The AAFD is a three dimensional deformity (Figure 7) and, in most of cases, the talo-navicular joint is the apex of the deformity and the key joint for its correction.

Reconstruction

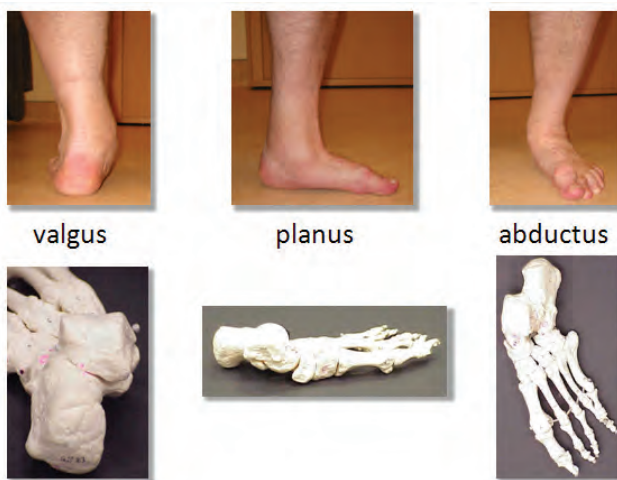


Figure 7: The acquired adult flatfoot is a 3D deformity including valgus of the hind foot, flattening of the longitudinal arch (planus) and abductus of the forefoot.

Conditions like rheumatoid arthritis (RA), trauma or neurologic disorders may result into an acquired flatfoot deformity. The most frequent cause of AAFD, however, is the posterior tibial tendon dysfunction, in 80% of cases. The pathology ranges from a flexible flatfoot with painful but functional posterior tibial tendon to a rigid deformity including osteoarthritis of the ankle. The most commonly used classification of AAFD has been established by Johnson and Strom in 1989 and completed by Myerson in 1996: It bases on the severity of the deformity, its reducibility, and the articular alterations (Figure 8). The pathology is not limited to the posterior tibial tendon but also includes ligamentous (especially the spring and delto-spring ligaments) and articular (i.e. the joints of the hind foot complex and the ankle joint) alterations.

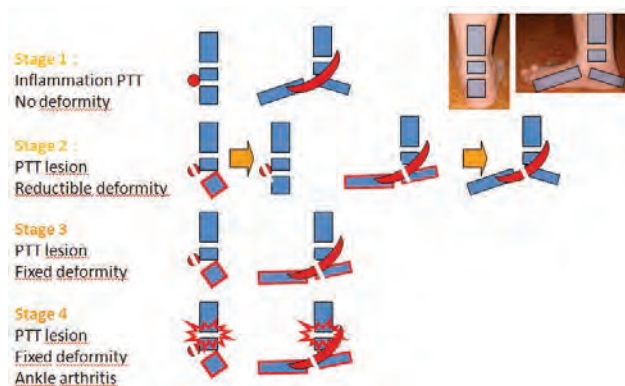


Figure 8: The acquired adult flatfoot deformity (AAFD) relies in 80% of cases on a posterior tibial tendon dysfunction (PTTD). It has been classified in three stages by Johnson and Strom in 1989 and, in 1996 a fourth stage has been added by Myerson.

Symptoms associated with AAFD include pain and swelling along the posterior tibial tendon, pain at the lateral aspect of the hind foot (sinus tarsi, fibulo-calcaneal conflict) and at the ankle in the late stages. Clinical signs include swelling and tenderness along the tibialis posterior tendon in the early stages, then increased valgus of the hind foot, flattening of the longitudinal arch, and abductus of the forefoot. Assessment of the function of the tibialis posterior tendon (single heel rise test), of the reducibility of the hind foot and of the midfoot deformities, and of the length of the

gastrocnemii (Silfverskiöld test) are the most important aspects of the functional clinical examination. Radiographic examination usually includes ap/lateral weightbearing x-rays of the foot and ap weightbearing x-rays of the ankle.

In early stages (I and II) conservative treatment should be attempted since specific eccentric programs associated with the prescription of orthoses have been shown to result in symptomatic and functional improvements, however without changes in tendon morphology or neovascularization. In late stages (III and IV), where the deformity is already fixed, conservative treatment will have poor corrective capability. However in cases of inoperability of the patient custom made shoes and orthoses can bring pain alleviation and functional improvement.

Depending upon the success or not of the conservative treatment surgical treatment can apply in all stages. In stage I a tibialis posterior tendon synovectomy can bring pain alleviation. In stage II functional surgery will be usually indicated and includes a soft tissue procedure (i.e. replacement of the diseased posterior tibial tendon by a flexor tendon, FHL or FDL, frequently associated with a gastrocnemius lengthening and, in some cases, with the spring ligament reconstruction) and a protective bony procedure (i.e. medial displacement osteotomy of the great calcaneal tuberosity or lengthening of the lateral column) (Figures 9&10). The above mentioned procedures are the most frequently described techniques in the literature. Neither for the soft tissues, nor for the bony procedures one technique has been proven to be superior to the other nor, also, even if gastrocnemius lengthening is performed in the majority of cases its absolute necessity has been established. In stage III stabilizing surgery includes double or triple corrective arthrodesis of the hind foot. In stage IV an additional procedure has often to be performed at the ankle (i.e. TAR or arthrodesis).



A

B

Figure 9: Functional surgery for flexible flatfoot deformity usually includes soft tissues and bony procedures. Figure 9A shows the harvest of the flexor digitorum longus tendon (FDL) at the node of Henry and its transfer into the navicular as replacement of the diseased posterior tibial tendon. Figure 9B explains that medial displacement calcaneal osteotomy realigns the hind foot and neutralizes the valgus traction of the triceps surae; it protects the transferred FDL.



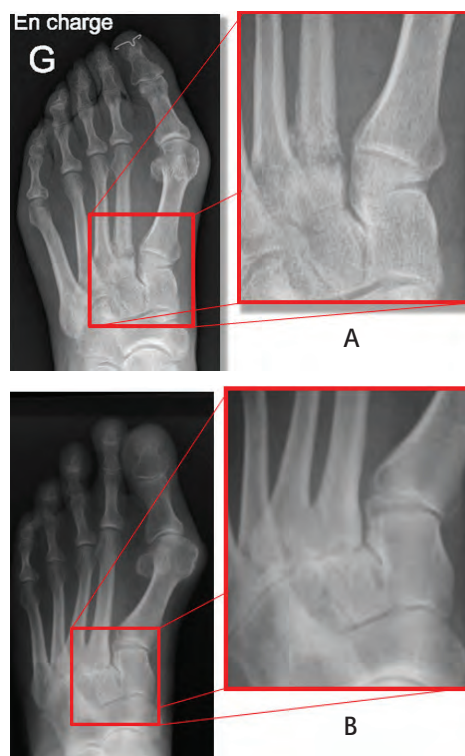
A

B
Figure 10: Radiographic appearance of a stage II flexible acquired flatfoot deformity before (A) and after (B) functional surgical correction including lengthening of the gastrocnemii, medial displacement osteotomy of the calcaneus and flexor digitorum longus transfer to the distal stump of the tibialis posterior tendon.

Results of functional surgery go along with pain alleviation and functional improvement. Midterm biomechanical evaluation has even demonstrated normalization of gait. Corrective stabilizing surgery also reduces pain and improves function but without full normalization of gait. Overall complication rate after flatfoot surgery ranges from 10–30%.

HALLUX VALGUS

The hallux valgus deformity is the first reason to visit a F&A surgeon in Europe. The deformity is more common under women than in men (8:1). Although a familiar predisposition has been identified the etiology and pathogenesis of hallux valgus still remain unclear and are thought to be multifactorial. The usually accepted radiological (weight bearing x-rays) definition of hallux valgus includes increased intermetatarsal ($IM > 10^\circ$) and metatarsophalangeal ($M1P1 > 10^\circ$) angles. However, the analysis of hallux valgus deformity is much more subtle than a simple radiographic angular evaluation. Clinically it should include the mobility of the first MTP joint, its reducibility, the intra articular pain, the presence of transfer metatarsalgia, and the presence of associated deformities (clawtoes, dislocation of the lesser MTP joints, flatfoot, etc.). Radiologically attention should be dedicated to the congruence or incongruence of the first MTP joint, orientation of the distal metatarsal articular angle (DMAA), degenerative changes of the joint, indirect manifestations of the insufficiency of the first ray, typically including hypertrophy of the second metatarsal, fatigue fracture of the second metatarsal, osteoarthritis of the second tarso-metatarsal joint (TMT2), dislocation of the lesser MTP joints (Figure 11). This careful analysis is mandatory for the choice of the most efficient treatment.



A
B
Figure 11: Indirect signs of first ray insufficiency include osteoarthritis of the second TMT joint (A), fatigue fracture of the second metatarsal (B), hypertrophy of the second metatarsal, and dislocation of the lesser MTP joints.

Conservative treatment includes the adaptation of footwear and the confection of insoles. It can bring pain alleviation but no conservative treatment has the faculty to correct the deformity. Surgical treatment includes the combination of bony and soft tissues procedures. Approaches can be performed open, minimal invasive or percutaneous. None of them has been demonstrated to be superior to the others and, in most of cases, the success rather depends upon the correct evaluation of the pathology and upon the mastering of a given technique by the surgeon.

Without entering into details, mild to moderate deformities (i.e. IM angle $< 18-20^\circ$, absence of degenerative changes, absence of significant indirect signs of insufficiency of the first ray) can be managed by a distal first metatarsal osteotomy (for example chevron or scarf osteotomy) associated or not with a basis phalanx medial closing wedge osteotomy (Akin) and a distal soft tissue procedure (Figure 12). Severe deformities require a proximal first metatarsal osteotomy or a TMT1 fusion (Lapidus) associated with a distal soft tissue procedure (Figure 12). In some cases (rheumatoid arthritis, arthritic MTP1, elderly patient, etc.), a MTP1 fusion may be the best choice. The juvenile hallux valgus, which includes a congruent first MTP and a pathological DMAA (Figure 13), requires not only the correction of the IM angle but also correction of the DMAA in order to prevent recurrence.



Figure 12: Mild to moderate deformities (A) can be managed by a distal first metatarsal osteotomy (B) and a distal soft tissue procedure. Severe deformities (C) require a proximal first metatarsal osteotomy or a TMT1 fusion (Lapidus) (D) associated with a distal soft tissue procedure.

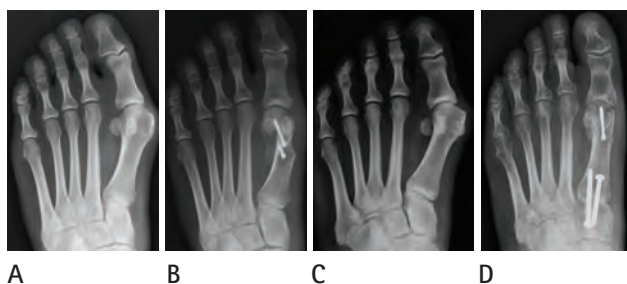


Figure 13: On these ap views of a juvenile hallux valgus we can appreciate the congruent joint and the pathologic DMAA (A) and (C). The correction has been performed by a derotating chevron osteotomy in the mild case (B) and by a modified Lapidus procedure + a derotating chevron in the more severe case (D).

Overall results following hallux valgus surgery are good or excellent in 80–90% of cases only. The complication rate ranges from 10–30% and includes recurrence, overcorrection, residual pain, non-union, chronic regional pain syndrome (CRPS).

References

Ankle osteoarthritis

1. Glazebrook, M et al (2008) Comparison of Health-Related Quality of Life Between Patients with End-Stage Ankle and Hip Arthritis. *JBJS* 90-A:499–505
2. Thomas RH & Daniels TR (2003) Ankle Arthritis *JBJS* 85-A 923–936.
3. Albert E (1879) Zur Resection des Kniegelenkes. *Wein Med. Press* 20:705–708.
4. Coester LM et al (2001) Long-Term Results Following Ankle Arthrodesis for Post-Traumatic Arthritis. *JBJS* 83-A (2):219–228.
5. Jordan Wet al (2014) Is End-Stage Ankle Arthritis Best Managed with Total Ankle Replacement or Arthrodesis? A Systematic Review. *Advances Orthop*:1–9.
6. Townshend D et al (2013) Arthroscopic versus open ankle arthrodesis: a multicenter comparative case series. *JBJS* 95-A(2):98–102.
7. Thomas R et al (2006) Gait Analysis and Functional Outcomes Following Ankle Arthrodesis for Isolated Ankle Arthritis *JBJS* 88-A (3):526–535.
8. Besse JL et al (2009) Clinical Evaluation and Radiographic Assessment of Bone Lysis of the AES Total Ankle Replacement. *F&A Int* 30(10):964–975.

9. Chopra S et al (2014) Outcome of Unilateral Ankle Arthrodesis and Total Ankle Replacement in Terms of Bilateral Gait Mechanics. *JOR* 32(3):377–384.
10. Haddad SL et al (2007) Intermediate and Long-Term Outcomes of Total Ankle Arthroplasty and Ankle Arthrodesis. A Systematic Review of the Literature. *JBJS* 89-A:1899–905.
11. Labek G et al (2011) Revision Rates After Total Ankle Arthroplasty in Sample-Based Clinical Studies and National Registries. *F&A Int* 32 (8):740–745
12. Piriou P et al (2008) Ankle Replacement versus Arthrodesis: A Comparative Gait Analysis Study *F&A Int* 29(1) :3–9.
13. Knupp M & Hintermann B (2012) Treatment of Asymmetric Arthritis of the Ankle Joint with Supramalleolar Osteotomies *Foot & Ankle Int* 33: 250–252.

Hallux valgus

14. Easley M & Trnka HJ (2007) Current Concepts Review: Hallux Valgus Part 1: Pathomechanics, Clinical Assessment, and Nonoperative Management. *F&A Int* 28 (5):654–659.
15. Easley M & Trnka HJ (2007) Current Concepts Review: Hallux Valgus Part II: Operative Treatment. *F&A Int* 28 (6):748–758.
16. Magnan B et al (2005) Percutaneous Distal Metatarsal Osteotomy for Correction of Hallux Valgus *JBJS* 87-A (6) 1191–1199.

Flatfoot

17. Johnson and Strom (1989) Tibialis Posterior Tendon Dysfunction. *CORR* 239:196–205
18. Kulig K et al (2009). Effect of Eccentric Exercise Program for Early Tibialis Posterior Tendinopathy. *F&A Int* 30(9):877–885.
19. Pinney & Lin (2006). Current Concept Review: Acquired Adult flatfoot Deformity. *F&A Int* 27(1):66–75.

QUESTIONS

1. What are the three typical features of a shoe designed for conservative treatment of ankle osteoarthritis?
2. What is the annual revision rate associated with total ankle replacement (TAR)?
3. Acquired adult flatfoot deformity (AAFD) has been classified in three stages by Johnson and Storm in 1989. For which stage is functional corrective surgery indicated?
4. Functional surgery for the correction of acquired adult flatfoot deformity (AAFD) typically includes replacement of the diseased tibialis posterior tendon by the flexor hallucis longus (FHL) or flexor digitorum longus (FDL) tendons. Is it true or false that the FHL transfer has been demonstrated to be superior to the FDL transfer?
5. Severe hallux valgus is frequently associated with functional insufficiency of the first ray. Please cite three typical alterations of the second ray that are considered as indirect signs of the first ray insufficiency.

ANSWERS:

1. Rocker bottom sole, shock absorbing heel, and ankle stabilization.
2. Annual revision rate after TAR is 3%.
3. Functional surgery is indicated for stage 2 AAFD.
4. It is false.
5. Cortical hypertrophy of the second metatarsal, fatigue fracture of the second metatarsal, osteoarthritis of the second tarso-metatarsal joint.



Dr. Nicolas Holzer
HUG, University Hospitals of Geneva,
Geneva, Switzerland
nicolas.holzer@hcuge.ch

Degenerative Diseases Of The Cuff, Shoulder And Elbow

ABSTRACT

Rotator cuff tears as well as osteoarthritis of the shoulder and elbow are frequently encountered when treating superior member pathologies. Degenerative disorders are a common etiology.

Rotator cuff pathology is a recognized health care problem with important economic implications. Diagnosis relies on thorough history and clinical examination confirmed by radiographic studies. Initial treatment of degenerative cases is usually conservative. Criteria for therapeutic decision are characteristics of the lesion, duration of symptoms, failure of conservative treatment, functional limitations of daily living and professional activities as well as presence of associated lesions (i.e. long head of the biceps and acromio-clavicular pathologies). Irreparable lesions may benefit from surgical debridement or tendon transfer procedures. Progression toward cuff tear arthropathy may be managed with reverse shoulder arthroplasty.

Shoulder osteoarthritis can be primary or secondary to trauma, instability or rheumatologic disorders. Diagnosis relies on radiographic evidence of cartilage damage. Correlation between radiographic features and functional impairment remains to be established and indication for surgical management depends on assessment of functional limitations. Conservative surgery may be warranted in presence of pain with preservation of range of motion. Joint replacement solutions comprise hemirathroplasty as well as total and reverse shoulder arthroplasty. Indication for each implant relies on determination of associated rotator cuff lesion as well as glenoid morphology.

Elbow osteoarthritis can present as a primary disease or be secondary to trauma or inflammatory arthropathies. Failure of conservative treatment with limitation of the joint's functional range of motion is an indication for surgical management. Non-arthroplasty procedures comprise open or arthroscopic debridement in early stages. Interposition arthroplasty may be undertaken in selected cases of end stage disease. Total elbow arthroplasty is seldom required and should be limited to patients with low functional demand.

in cadaver dissections ranges from 5 to 30%, increasing with age^{2,3}. Ultrasonographic assessment of a population of 638 individuals displayed presence of a rotator cuff tears in 36% of symptomatic shoulder and in 16.9% of asymptomatic ones⁴.

2. ETIOLOGY

Rotator cuff lesions have been theorized to occur as a normal degenerative process associated with aging. Development of tears has been historically linked to acromial morphology and impingement of the cuff on the antero-lateral acromial surface³. Recently role of the combination of acromial lateral covering with glenoid upward tilting, quantified by measurement of the critical shouder angle has been incriminated⁵. Rotator cuff lesions may yet be the consequence of micro and macro-trauma. Reported risk factors comprise smoking habit, hypercholesterolemia and genetic predispositions¹.

3. CLASSIFICATION

Multiple classifications of rotator cuff tears have been described to quantify extent of tendinous lesions as well as associated muscle degeneration and gleno-humeral joint damage.

3.1 Tendinous lesion

Rotator cuff lesions range from tendinopathy to complete ruptures, encompassing partial ruptures, articular or joint sided, as well as interstitial. Importance of complete rotator cuff ruptures is quantified by measuring retraction in the coronal plane and tear extension in sagittal plane. Retraction in the coronal plane may be classified according to Patte (Fig 1) as follows; stage I: proximal stump close to bony insertion; stage II: proximal stump at the level of humeral head and stage III: proximal stump at glenoid level^[6]. In the sagittal plane, tears have been classified by the same author according to the pattern of tendinous lesions. An alternative sagittal plane classification has been proposed by Snyder⁷ based on tear size and number of damaged tendons, introducing the concept of massive cuff tear when 2 or more tendon are involved.

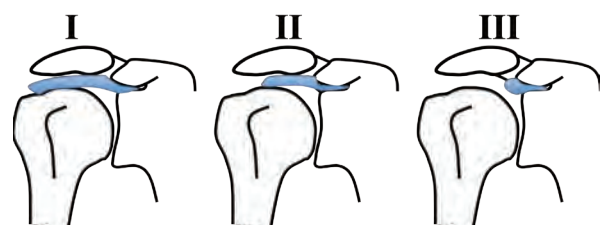


Fig. 1 Patte classification: cuff tear retraction

I. DEGENERATIVE CUFF LESIONS

1. INTRODUCTION

Rotator cuff disorder is the most common condition associated with shoulder disability¹. Prevalence of rotator cuff tears has been evaluated in cadaver and imaging studies, both in asymptomatic and symptomatic individuals. Presence of a full thickness tear

3.2 Muscle degeneration

Muscle fatty degeneration associated with cuff tearing has been classified based on computed tomography images by Goutallier et al.⁸ Five stages have been proposed: stage 0: normal muscle, stage 1: presence of fatty streaks, stage 3: fatty muscle atrophy less than 50%, stage 4: 50% fatty muscle atrophy, stage 5: fatty muscle atrophy more than 50%.

3.3 Cuff tear arthropathy

Radiographic study of conservatively treated massive cuff tears led Hamada et al to propose a grading of progression of the disease to its end stage, characterized by gleno-humeral joint destruction known as cuff tear arthropathy (CTA)⁹ (Fig. 2). In stage 1 and 2, acromio-humeral interval is of more and less than 6 mm respectively. Stage 3 is defined by association of stage 2 features with concave deformity of the acromial undersurface, termed acetabulization. Stage 4 is defined by association of stage 3 with narrowing of the gleno-humeral joint. Stage 5 is defined by bony destruction and collapse of the humeral head.

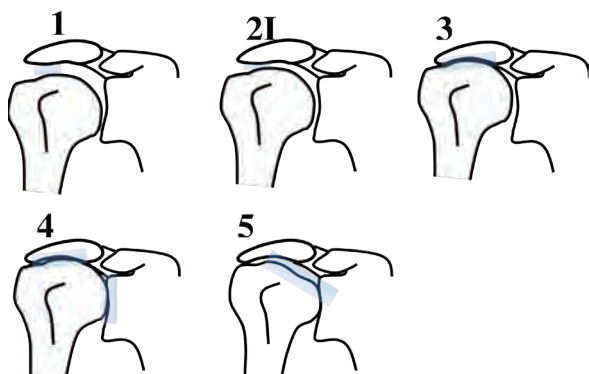


Fig. 2 Hamada classification: cuff tear arthropathy

4. DIAGNOSTIC ASSESSMENT

Shoulder pain may result from association of multiple lesions including rotator cuff tendons, long head of the biceps, capsulo-ligamentous tissues, acromio-clavicular and gleno-humeral joints as well as neurologic structures. Systematic correlation of reported patient's symptoms with clinical examination and imaging findings are required to establish all relevant diagnoses.

4.1 History and clinical examination

Pain, stiffness, weakness, instability and pseudoparalysis defined as inability to elevate the arm up to 90° are recorded as well as date and mode of onset of symptoms. All previous treatments including surgeries are listed. Self rated assessment of shoulder pain and disability¹⁰ is established. Clinical examination begins with inspection of muscle trophicity as well as any deformity. Palpation of the acromio-clavicular joint, greater tuberosity, bicipital groove, posterior glenoid-humeral joint line and neck allow for simple assessment of painful structures. Passive and active shoulder range of motion on both sides as well as strength of individual rotator cuff muscle is noted. Numerous provocative tests have been described for diagnosis of impingement syndrome as well as long head of the biceps and acromio-clavicular joint pathology. Although no consensus has been reached on which one to retain, they allow for precision of clinical diagnoses. Standardized use of functional scores for monitoring of therapeutic interventions is recommended¹¹.

4.2 Imaging

Acquisition of standard shoulder radiographs allow for simple assessment of indirect signs of rotator cuff lesion (fig 1) and associated pathologies including calcifying tendinitis as well as acromio-clavicular and gleno-humeral pathologies. It permits measurement of relevant radiologic indices such as acromio-humeral interval¹² and aforementioned critical shoulder angle.

Multiple complementary imaging modalities are available for diagnosis of rotator cuff tears. Magnetic resonance imaging with or without arthrography and ultrasound may be equally used for detection of full thickness tears¹³. Well-designed studies are warranted to compare sensitivity and specificity of each technique for diagnosis of tendinopathy, partial tears as well as muscle fatty atrophy.



Fig 3: Radiographic evaluation of rotator cuff tears.

Presence of acromial spur (S) and rounding of the greater tuberosity (T) indicates chronic cuff disorder. Acromio-humeral distance (AHD) narrowing indicates complete tear of supra and infraspinatus and progression toward CTA. Associated gleno-humeral osteoarthritis is diagnosed in presence of gleno-humeral osteophytes (O) (see also Fig 4).

5. NONOPERATIVE TREATMENT

Nonoperative treatment of rotator cuff tears may lead to satisfying results in 50 to 60% of patients¹⁴⁻¹⁶. Potential risks include progression of tear size, tendon retraction, and muscle fatty atrophy¹. Proposed contra-indications are full thickness tears, both acute and chronic, in patients less than 65 years old.

6. ROTATOR CUFF REPAIR

6.1 Indications

Indications for rotator cuff surgery have been shown to vary considerably between surgeons¹⁷. Criteria used for therapeutic decision are limitation of activities, history of trauma, age, duration of symptoms and failure of nonoperative treatment¹⁸. A recent study of societal and economical value of rotator cuff repair has reported lower societal costs for patients less than 61 years old and greater quality adjusted life years for all patients in comparison to nonoperative treatment¹⁹.

6.2 Repair techniques

Rotator cuff repair may be achieved by open, mini-open or arthroscopic approaches. Suture technique may be transosseous or transosseous equivalent using anchors in single row or double rows²⁰. Despite increasing publication numbers, meta-analysis of factors predictive of non-healing/retear and clinical outcome reports insufficient information to guide management²¹.

7. IRREPARABLE CUFF TEAR MANAGEMENT

7.1 Definition

Irreparability of cuff tears may be defined as the impossibility to achieve tendon to bone healing by surgical reinsertion. Factors associated with irreparability are tear size of more than 4–5 cm, fatty infiltration of grade 3 to 4 according to Goutallier²² as well as static superior humeral head migration with reduced acromio-humeral distance²³.

7.2 Non-arthroplasty solutions

Non-arthroplasty management of irreparable cuff tears includes nonoperative treatment, debridement, partial repair of lesions²⁴ as well as biceps tenodesis or tenotomy²⁵ and tendon transfer of the latissimus dorsi and pectoralis major^{26, 27}. Choice of treatment modality has to take into account needs and expectations of the patients as well as ability to follow intensive rehabilitation²⁸.

7.3 Reverse shoulder arthroplasty

Surgical management of massive irreparable cuff tears with or without associated cuff tear arthropathy and pseudoparalysis by means of reverse shoulder arthroplasty has been expanding substantially²⁹. Subjective improvement of symptoms and restoration of function have been described up to ten years follow up³⁰. Reported complication rate is important, even though they may be managed with limited compromise of final outcome. Proposed contraindications include isolated subscapularis tears and severely impaired deltoid function²⁹.

presence and size of osteophytes as well as joint space narrowing (Fig 4). The scale designed by Samilson and Prieto for post-instability gleno-humeral joint degenerative disease is frequently used³⁴ and has been adapted by several authors for other conditions. Although correlation between radiological grade of osteoarthritis and functional disability remains debated, good intra- and inter-observer reliability has been described for shoulder OA scales and their use advocated for clinical and scientific purposes³⁵.

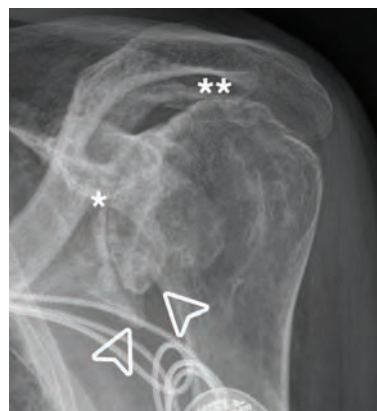


Fig 4: Radiographic features of shoulder OA.

End stage OA characterized by prominent osteophytes of inferior glenoid and humerus (arrows) associated with complete joint space width narrowing (*). Subacromial joint space width narrowing (**) to be correlated with glenoid morphology and degree of humeral head subluxation (see Fig. 5)

II. SHOULDER OSTEOARTHRITIS

1. INTRODUCTION

Shoulder osteoarthritis (OA) is characterized by shoulder disability associated with loss of gleno-humeral articular cartilage. It is a frequent cause of shoulder pain along with rotator cuff disorders and adhesive capsulitis³¹. Surgical treatment is usually indicated after failure of conservative treatment and includes conservative as well as prosthetic procedures. Arthroplasty solutions encompass unconstrained (hemiarthroplasty and total shoulder arthroplasty) and constrained implant types (reverse shoulder arthroplasty)³². Choice of use is based on assessment of function of the rotator cuff as well as alteration of the gleno-humeral bony anatomy.

2. DIAGNOSTIC ASSESSMENT

Classical reasons leading to seek medical advice are pain associated with catching, locking and crepitus during motion. Self assessment of pain and functional disability is advocated both for therapeutic decision making and scientific purposes³³. Duration of symptoms, prescribed treatments as well as history of previous trauma and instability of the shoulder are carefully recorded.

Clinical examination includes assessment of residual range of motion and rotator cuff function.

3. SHOULDER OA CLASSIFICATIONS

Criteria for therapeutic decision in shoulder OA comprise severity of articular cartilage and glenoid bone stock damage. Conventional radiology readily allows for establishing diagnosis. Several classification systems have been described. Assessed features are

Alteration of glenoid morphology is frequently observed in end stage shoulder OA. Widely used classification of Walch et al. comprises three stage according to positioning of the humeral head and pre-existing dysplasia³⁶. Centered humeral heads are classified as A1 in presence of minor central erosion and A2 with major wear. Subluxed humeral heads are classified as B1 when glenoid morphology is preserved and B2 when biconcave due to posterior wear. Type C is defined as glenoid retroversion superior to 25°. Asymmetric loading of glenoid components in presence of excessive retroversion, termed rocking horse phenomenon, may predispose to loosening. Careful preoperative assessment using computed tomography is thus advocated to ensure sufficient bone stock for stable implant positioning (Fig 5).

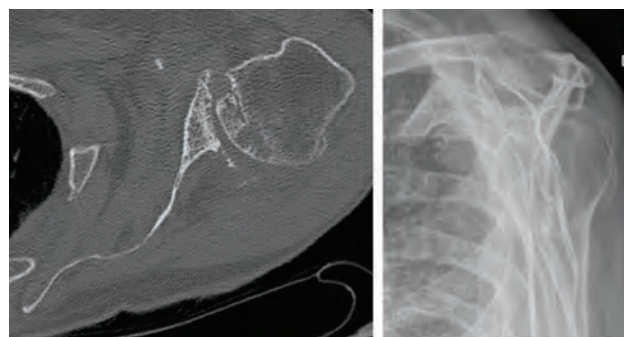


Fig 5: Glenoid morphology.

Computed tomography imaging of patient in fig. 4. Severe posterior glenoid wear and retroversion of 29° (Type B2 glenoid according to Walch et al.) associated with posterior subluxation of the humeral head readily demonstrated by conventional radiography. Note apparent subacromial joint space narrowing (Fig. 4) in presence of posterior subluxation.

Careful assessment of radiological signs of rotator cuff disorders is mandatory prior to joint replacement surgery (see Fig. 3). Impact of rotator cuff dysfunction on shoulder joint replacement outcome with first available unconstrained devices as been recognized early³⁷ and shoulder arthroplasty has been referred to as a mainly soft tissue procedure.

4. SHOULDER ARTHROPLASTY

4.1 Unconstrained shoulder arthroplasty

Use of unconstrained shoulder implants has been reported in all forms of shoulder disorders³⁷. It is generally admitted that indications are now restricted to situations displaying an intact or reparable rotator cuff³⁸. Use of hemiarthroplasty in shoulder replacement surgery for osteoarthritis remains advocated by some groups. Classical complication is glenoid wear by contact between the metallic humeral head and native glenoid cavity leading to shorter implant survival in comparison to total shoulder replacement³⁹.

Total shoulder arthroplasty for shoulder osteoarthritis with competent rotator cuff and preserved glenoid bone stock is the actual standard of care³⁸. Complications most frequently occur at the glenoid level with progressive loosening of the implant. Cemented, keeled, all polyethylene glenoid implant remains the reference standard, although pegged designs have been in use for more than a decade.

4.2 Constrained shoulder arthroplasty

Initial constrained "fixed fulcrum" implants designed to palliate cuff deficiency failed catastrophically due to glenoid component loosening⁴⁰. Pioneering work of Dr Grammont allowed for development of successful reverse shoulder arthroplasty implants to treat cuff tear arthropathy⁴¹. Indications have expanded to include management of severely comminuted proximal humerus fracture, fracture sequelae, revision arthroplasty and posterior glenoid bone defects⁴². Recent comparison of outcomes of total and reverse shoulder arthroplasty suggests increased final range of motion for the former. Caution should thus be taken not to over-indicate use of reverse shoulder arthroplasty.

III. ELBOW OSTEOARTHRITIS

1. INTRODUCTION

Osteoarthritis of the elbow although relatively rare can cause severe disability. It is characterized by pain associated with loss of range of motion and weakness as well as neurologic deficits. Important knowledge about its recognition and management has been gained in the last decades^{43, 44}. Treatment solutions have evolved and comprise conservative treatment, open and arthroscopic joint sparing techniques as well as arthroplasty.

2. ETIOLOGY

Pathophysiology of the disease is marked by varying degrees of cartilage damage, loose bodies generation, osteophyte formation and capsulo-ligamentous contracture. It may present as a primary disease or be secondary to trauma, rheumatologic conditions, osteochondritis dissecans, synovial chondromatosis, septic arthritis and hemophilia^{44, 45}.

Prevalence of symptomatic primary elbow OA is reported to be

about 2% of the general population⁴⁶. Association with strenuous work is described^{45, 47}. Male to female ratio is 4:1 with an average age of 50 years at presentation.

Simple dislocation of the elbow is not correlated with increased incidence of elbow OA. Unrecognized medial coronoid facet fracture on the contrary can rapidly evolve toward degeneration of the joint⁴⁸.

3. DIAGNOSTIC ASSESSMENT

3.1 History and clinical examination

Past medical history of trauma or surgery is carefully recorded as well as timing of onset of symptoms and self rated pain and functional scores.

The elbow is inspected in search of previous scars, swelling or deformation. Precision of localization of pain is achieved by palpation of the radio-capitellar joint, epicondyles and mediolateral gutter. Range of motion is assessed both actively and passively and pain at the end or throughout the arc of motion is noted as well as any catching or locking. Restriction of arc of motion may be characterized as minimal (<90°), moderate (61-90°), severe (31-60°) and very severe (<30°)⁴⁹. Ulnar neuropathy is frequently associated with elbow OA. Assessment of nerve irritability, subluxation and function is complemented by electro-neuro-myographic examination if positive.

3.2 Imaging

Conventional radiographic imaging assessing presence of osteophytes and joint space narrowing readily allows for diagnosis of elbow OA. Computed tomography with two- and three-dimensional reconstructions is helpful for surgical planning of osteophytes removal. Magnetic resonance has been reported not to add relevant information in elbow OA and some authors have recommended against its use⁵⁰.

4. CONSERVATIVE TREATMENT

Non-surgical management of elbow osteoarthritis includes anti-inflammatory medication, activity modification and corticosteroid injections. Aim is to facilitate mobilization of the diseased joint in early stages of elbow OA. Intra-articular injection of sodium hyaluronate has been shown to allow for pain relief limited to maximum 6 month follow up and use is not recommended⁵¹.

5. SURGICAL MANAGEMENT

Range of motion of the elbow inferior to 130-30-0° defined as the functional elbow range of motion by Morrey et al is generally accepted as threshold for indication to surgery⁵². Patients with pain at the end of range displaying preservation of joint space width can generally be treated with joint sparing surgery, open or arthroscopic, associating debridement, osteophyte excision and capsular release. Pain throughout the entire arc of motion associated with joint space narrowing and disruption of the joints architecture may be managed by interposition arthroplasty or total elbow arthroplasty.

5.1 Open joint sparing surgery

Joint debridement, capsular release and osteophytes resection has been referred to as ulnohumeral arthroplasty. It can be performed through a posterior approach and olecranon fossa fenestration, known as the Outerbridge - Kashiwagi procedure⁵³. Increased access to the anterior compartment and radio-capitellar joint may be gained by means of a lateral, column procedure⁴⁹. Postoperative splinting programs in flexion and extension is often introduced post-operatively.

5.2 Arthroscopic surgery

Soft tissue contractures as well as some features of osteoarthritis may be managed arthroscopically⁵⁴. Risk of neurologic complication in the proximity of neurovascular structures requires sound knowledge of local anatomy as lesions of about every nerve about the elbow have been reported⁵⁵⁻⁵⁷. Potential advantages include minimal disruption of soft tissue during surgical approach, less post-operative pain and faster recovery. Comparison of arthroscopic surgery with open ulnohumeral arthroplasty has shown better pain relief but decreased improvement in flexion⁵⁸.

5.3 Interposition arthroplasty

Severely damaged post-traumatic inflammatory elbow joints in young and active patients where total elbow prosthesis is contra-indicated may benefit from joint resurfacing by interposition of an Achilles allograft⁵⁹. The intervention is considered as a salvage procedure as it neither completely eliminates pain nor fully restores function.

5.4 Total elbow arthroplasty

Total elbow arthroplasty is seldom required in primary osteoarthritis⁴⁴. Indications have evolved from mainly management of rheumatoid arthritis to complex distal humerus fracture in the elderly patient⁶⁰. Use is limited to low functional demand patients as a definitive restriction of weight lifting to less than 2-5 kg as a single event and 0.5 to 1 kg repetitively is recommended⁶¹.

BIBLIOGRAPHY

1. Tashjian, R.Z., *Epidemiology, natural history, and indications for treatment of rotator cuff tears*. *Clin Sports Med*, 2012. 31(4): p. 589-604.
2. Lehman, C., et al., *The incidence of full thickness rotator cuff tears in a large cadaveric population*. *Bull Hosp Jt Dis*, 1995. 54(1): p. 30-1.
3. Neer, C.S., 2nd, *Impingement lesions*. *Clin Orthop Relat Res*, 1983(173): p. 70-7.
4. Yamamoto, A., et al., *Prevalence and risk factors of a rotator cuff tear in the general population*. *J Shoulder Elbow Surg*, 2010. 19(1): p. 116-20.
5. Moor, B.K., et al., *Relationship of individual scapular anatomy and degenerative rotator cuff tears*. *J Shoulder Elbow Surg*, 2014. 23(4): p. 536-41.
6. Patte, D., *Classification of rotator cuff lesions*. *Clin Orthop Relat Res*, 1990(254): p. 81-6.
7. Snyder, S.J., *Shoulder arthroscopy*. 1994, New York: McGraw-Hill, Health Professions Division. xiii, 271 p.
8. Goutallier, D., et al., *Fatty muscle degeneration in cuff ruptures. Pre- and postoperative evaluation by CT scan*. *Clin Orthop Relat Res*, 1994(304): p. 78-83.
9. Hamada, K., et al., *Roentgenographic findings in massive rotator cuff tears. A long-term observation*. *Clin Orthop Relat Res*, 1990(254): p. 92-6.
10. Gilbert, M.K. and C. Gerber, *Comparison of the subjective shoulder value and the Constant score*. *J Shoulder Elbow Surg*, 2007. 16(6): p. 717-21.
11. Constant, C.R., et al., *A review of the Constant score: modifications and guidelines for its use*. *J Shoulder Elbow Surg*, 2008. 17(2): p. 355-61.
12. Flatow, E.L., et al., *Excursion of the rotator cuff under the acromion. Patterns of subacromial contact*. *Am J Sports Med*, 1994. 22(6): p. 779-88.
13. Lenza, M., et al., *Magnetic resonance imaging, magnetic resonance arthrography and ultrasonography for assessing rotator cuff tears in people with shoulder pain for whom surgery is being considered*. *Cochrane Database Syst Rev*, 2013. 9: p. CD009020.
14. Goldberg, B.A., R.J. Nowinski, and F.A. Matsen, 3rd, *Outcome of nonoperative management of full-thickness rotator cuff tears*. *Clin Orthop Relat Res*, 2001(382): p. 99-107.
15. Hawkins, R.H. and R. Dunlop, *Nonoperative treatment of rotator cuff tears*. *Clin Orthop Relat Res*, 1995(321): p. 178-88.
16. Bokor, D.J., et al., *Results of nonoperative management of full-thickness tears of the rotator cuff*. *Clin Orthop Relat Res*, 1993(294): p. 103-10.
17. Dunn, W.R., et al., *Variation in orthopaedic surgeons' perceptions about the indications for rotator cuff surgery*. *J Bone Joint Surg Am*, 2005. 87(9): p. 1978-84.
18. Marx, R.G., et al., *Indications for surgery in clinical outcome studies of rotator cuff repair*. *Clin Orthop Relat Res*, 2009. 467(2): p. 450-6.
19. Mather, R.C., 3rd, et al., *The societal and economic value of rotator cuff repair*. *J Bone Joint Surg Am*, 2013. 95(22): p. 1993-2000.
20. Duquin, T.R., C. Buyea, and L.J. Bisson, *Which method of rotator cuff repair leads to the highest rate of structural healing? A systematic review*. *Am J Sports Med*, 2010. 38(4): p. 835-41.
21. McElvany, M.D., et al., *Rotator cuff repair: published evidence on factors associated with repair integrity and clinical outcome*. *Am J Sports Med*, 2015. 43(2): p. 491-500.
22. Sugihara, T., et al., *Prediction of primary reparability of massive tears of the rotator cuff on preoperative magnetic resonance imaging*. *J Shoulder Elbow Surg*, 2003. 12(3): p. 222-5.
23. Nove-Josserand, L., et al., *The acromiohumeral and coracohumeral intervals are abnormal in rotator cuff tears with muscular fatty degeneration*. *Clin Orthop Relat Res*, 2005(433): p. 90-6.
24. Berth, A., et al., *Massive rotator cuff tears: functional outcome after debridement or arthroscopic partial repair*. *J Orthop Traumatol*, 2010. 11(1): p. 13-20.
25. Boileau, P., et al., *Isolated arthroscopic biceps tenotomy or tenodesis improves symptoms in patients with massive irreparable rotator cuff tears*. *J Bone Joint Surg Am*, 2007. 89(4): p. 747-57.
26. Gerber, C., G. Maquieira, and N. Espinosa, *Latissimus dorsi transfer for the treatment of irreparable rotator cuff tears*. *J Bone Joint Surg Am*, 2006. 88(1): p. 113-20.
27. Wirth, M.A. and C.A. Rockwood, Jr., *Operative treatment of irreparable rupture of the subscapularis*. *J Bone Joint Surg Am*, 1997. 79(5): p. 722-31.
28. Delaney, R.A., A. Lin, and J.J. Warner, *Nonarthroplasty options for the management of massive and irreparable rotator cuff tears*. *Clin Sports Med*, 2012. 31(4): p. 727-48.

29. Drake, G.N., D.P. O'Connor, and T.B. Edwards, Indications for reverse total shoulder arthroplasty in rotator cuff disease. *Clin Orthop Relat Res*, 2010. 468(6): p. 1526-33.
30. Ek, E.T., et al., Reverse total shoulder arthroplasty for massive irreparable rotator cuff tears in patients younger than 65 years old: results after five to fifteen years. *J Shoulder Elbow Surg*, 2013. 22(9): p. 1199-208.
31. Meislin, R.J., J.W. Sperling, and T.P. Stitik, Persistent shoulder pain: epidemiology, pathophysiology, and diagnosis. *Am J Orthop (Belle Mead NJ)*, 2005. 34(12 Suppl): p. 5-9.
32. Kim, S.H., et al., Increasing incidence of shoulder arthroplasty in the United States. *J Bone Joint Surg Am*, 2011. 93(24): p. 2249-54.
33. Provencher, M.T., et al., An analysis of shoulder outcomes scores in 275 consecutive patients: disease-specific correlation across multiple shoulder conditions. *Mil Med*, 2012. 177(8): p. 975-82.
34. Samilson, R.L. and V. Prieto, Dislocation arthropathy of the shoulder. *J Bone Joint Surg Am*, 1983. 65(4): p. 456-60.
35. Elsharkawi, M., et al., Reliability of radiologic glenohumeral osteoarthritis classifications. *J Shoulder Elbow Surg*, 2013. 22(8): p. 1063-7.
36. Walch, G., et al., Morphologic study of the glenoid in primary glenohumeral osteoarthritis. *J Arthroplasty*, 1999. 14(6): p. 756-60.
37. Neer, C.S., 2nd, K.C. Watson, and F.J. Stanton, Recent experience in total shoulder replacement. *J Bone Joint Surg Am*, 1982. 64(3): p. 319-37.
38. Walch, G., P. Boileau, and E. Noel, Shoulder arthroplasty: evolving techniques and indications. *Joint, bone, spine : revue du rhumatisme*, 2010. 77(6): p. 501-5.
39. Sperling, J.W., R.H. Cofield, and C.M. Rowland, Minimum fifteen-year follow-up of Neer hemiarthroplasty and total shoulder arthroplasty in patients aged fifty years or younger. *J Shoulder Elbow Surg*, 2004. 13(6): p. 604-13.
40. Neer, C.S., 2nd and R.M. Kirby, Revision of humeral head and total shoulder arthroplasties. *Clin Orthop Relat Res*, 1982(170): p. 189-95.
41. Boileau, P., et al., Grammont reverse prosthesis: design, rationale, and biomechanics. *Journal of shoulder and elbow surgery / American Shoulder and Elbow Surgeons ... [et al.]*, 2005. 14(1 Suppl S): p. 147S-161S.
42. Mizuno, N., et al., Reverse total shoulder arthroplasty for primary glenohumeral osteoarthritis in patients with a biconcave glenoid. *J Bone Joint Surg Am*, 2013. 95(14): p. 1297-304.
43. Cheung, E.V., R. Adams, and B.F. Morrey, Primary osteoarthritis of the elbow: current treatment options. *J Am Acad Orthop Surg*, 2008. 16(2): p. 77-87.
44. Gramstad, G.D. and L.M. Galatz, Management of elbow osteoarthritis. *J Bone Joint Surg Am*, 2006. 88(2): p. 421-30.
45. Stanley, D., Prevalence and etiology of symptomatic elbow osteoarthritis. *Journal of shoulder and elbow surgery / American Shoulder and Elbow Surgeons ... [et al.]*, 1994. 3(6): p. 386-9.
46. Ortner, D.J., Description and classification of degenerative bone changes in the distal joint surfaces of the humerus. *Am J Phys Anthropol*, 1968. 28(2): p. 139-55.
47. Lawrence, J.S., Rheumatism in coal miners. III. Occupational factors. *Br J Ind Med*, 1955. 12(3): p. 249-61.
48. Sanchez-Sotelo, J., S.W. O'Driscoll, and B.F. Morrey, Medial oblique compression fracture of the coronoid process of the ulna. *J Shoulder Elbow Surg*, 2005. 14(1): p. 60-4.
49. Mansat, P. and B.F. Morrey, The column procedure: a limited lateral approach for extrinsic contracture of the elbow. *The Journal of bone and joint surgery. American volume*, 1998. 80(11): p. 1603-15.
50. Sears, B.W., et al., Posttraumatic elbow arthritis in the young adult: evaluation and management. *The Journal of the American Academy of Orthopaedic Surgeons*, 2012. 20(11): p. 704-14.
51. van Brakel, R.W. and D. Eygendaal, Intra-articular injection of hyaluronic acid is not effective for the treatment of post-traumatic osteoarthritis of the elbow. *Arthroscopy*, 2006. 22(11): p. 1199-203.
52. Morrey, B.F., L.J. Askew, and E.Y. Chao, A biomechanical study of normal functional elbow motion. *The Journal of bone and joint surgery. American volume*, 1981. 63(6): p. 872-7.
53. Kashiwagi, D., Intra-articular changes of the osteoarthritic elbow, especially about the fossa olecranon. *Jpn Orthop Assn.*, 1978(1978;52:1367-82.).
54. Holzer, N. and S.P. Steinmann, The Stiff Elbow: Osteoarthritis and Arthrofibrosis Arthroscopic Management. *Operative Techniques in Sports Medicine*, 2014.
55. Haapaniemi, T., M. Berggren, and L. Adolfsson, Complete transection of the median and radial nerves during arthroscopic release of post-traumatic elbow contracture. *Arthroscopy : the journal of arthroscopic & related surgery : official publication of the Arthroscopy Association of North America and the International Arthroscopy Association*, 1999. 15(7): p. 784-7.
56. Papillon, J.D., R.S. Neff, and L.M. Shall, Compression neuropathy of the radial nerve as a complication of elbow arthroscopy: a case report and review of the literature. *Arthroscopy : the journal of arthroscopic & related surgery : official publication of the Arthroscopy Association of North America and the International Arthroscopy Association*, 1988. 4(4): p. 284-6.
57. Ruch, D.S. and G.G. Poehling, Anterior interosseus nerve injury following elbow arthroscopy. *Arthroscopy : the journal of arthroscopic & related surgery : official publication of the Arthroscopy Association of North America and the International Arthroscopy Association*, 1997. 13(6): p. 756-8.
58. Cohen, A.P., J.F. Redden, and D. Stanley, Treatment of osteoarthritis of the elbow: a comparison of open and arthroscopic debridement. *Arthroscopy*, 2000. 16(7): p. 701-6.
59. Larson, A.N. and B.F. Morrey, Interposition arthroplasty with an Achilles tendon allograft as a salvage procedure for the elbow. *The Journal of bone and joint surgery. American volume*, 2008. 90(12): p. 2714-23.
60. Gay, D.M., et al., Indications and reoperation rates for total elbow arthroplasty: an analysis of trends in New York State. *The Journal of bone and joint surgery. American volume*, 2012. 94(2): p. 110-7.
61. Sanchez-Sotelo, J., Distal humeral fractures: role of internal fixation and elbow arthroplasty. *The Journal of bone and joint surgery. American volume*, 2012. 94(6): p. 555-68.



Prof. Dr. med. Maurilio Marcacci
m.marcacci@biomec.ior.it

Dr. Giulio Maria Marcheggiani Muccioli
marcheggianimuccioli@sportsdoc.it

Istituto Ortopedico Rizzoli, University of Bologna
Bologna, Italy

ACL, PCL, Collaterals And Meniscus

ABSTRACT

All the structures of the knee are necessary to guarantee its functioning without pain and/or instability. To allow a correct function the aim of the modern orthopaedic surgery is to preserve as much as possible knee structures, to repair them when is it feasible or to replace them when is it needed.

There are many recent advances in arthroscopic instrumentation and techniques for knee ligament reconstruction. Nowadays soft-tissue knee surgery can rely on new biologic solutions, such as meniscal allograft transplantation and meniscal substitutes.

1. Meniscus

The primary function of the meniscus is to distribute the weight-bearing load across the knee joint.

The menisci transmit approximately 50% of the load with the knee in extension, and close to 90% of the load at 90° of knee flexion. With flexion past 90°, most of the force is transmitted through the posterior horns. The lateral meniscus has been shown to transmit a greater percentage of the load compared with the medial meniscus.

Menisci act also as secondary stabilizers in the knee joint, helping ligaments to restrain joint laxity.

A key aspect of the meniscal anatomy is its vascularity, which is one of the critical elements in healing of a meniscal repair/substitution/transplantation. The most peripheral 20% to 30% of the native medial meniscus and the peripheral 10% to 25% of the native lateral meniscus are consistent in vascularity. Because of its rich blood supply, this area is commonly referred to as the red zone, and it is an area that has greater healing potential than the inner portions of the meniscus with less or no vascular supply. The inner third of the meniscus is avascular and is referred to as the white zone. This area is nourished by synovial fluid diffusion and repairs usually do not heal well in this zone. The area (middle third) of meniscus between the red and white zones is known as the red/white zone. Because this area does have some blood supply, it has the potential for healing, particularly in the young patient.

1.1. Meniscus injury

When meniscal integrity is lost, abnormal articular contact stresses result, leading to potential increased wear of the articular cartilage and early degenerative changes. The more meniscal tissue that is lost, the greater the loss of contact surface area and the greater the increase in peak local contact stresses. Thus, the primary goal of treatment of a meniscal tear is to maintain as much healthy meniscus tissue as possible.

There are several anatomical patterns/types of meniscal tears: radial, flap, oblique, vertical longitudinal, bucket handle and complex tears. Another important distinction is between traumatic vs. degenerative and acute vs. chronic tears. Traumatic and acute tears have a great healing potential: they are suitable to repair if the geometry of the lesion allow it. Degenerative and chronic tears have low/no healing potential: they should be treated nonsurgically if possible or with meniscectomy if required.

Some traumatic meniscal tears, depending on the symptoms they incur, can be treated nonsurgically. These include: (1) longitudinal tears that are stable (displaced < 3 mm) and less than 5 to 8 mm in length; (2) partial tears that are stable; (3) shallow radial tears (< 3 mm in depth) and (4) tears with a favorable natural history, which includes small lateral meniscal tears with a concurrent ACL reconstruction.

A nonsurgical management should be attempted also for degenerative complex tears not causing a true locking of the knee.

1.2. Meniscectomy

A selective (partial) meniscectomy is indicated for tears in the avascular (white) zone and radial, oblique and flap tears. This procedure may also be performed for any tear that has caused significant injury to the body of the meniscus, because they damage the structural integrity of the meniscus and the vascularity may be in doubt. Additionally, degenerative and/or overload tears (at surgery yellowish degeneration of the meniscal core is found) are another indication for meniscectomy in case of failed nonsurgical management, because in these cases poor meniscal tissue and low cellularity greatly compromise the healing response.

If excision of a meniscal tear is performed, the surgeon should preserve as much viable meniscal tissue as possible.

Minimal tissue resection, leaving intact the meniscal rim, should be

the rule. Total/subtotal meniscectomy should be avoided. Care should be taken to resect what has been torn and remove meniscal tissue only to avoid any further impingement that may remain sensitive to rotational painful stress and may thus produce clinical symptoms.

Rehabilitation:

The patient is allowed to walk with crutches and partial load for the first two weeks after surgery. Afterwards full weight bearing is recommended. Knee passive mobilization can be started right after surgery, followed by active mobilization until the recovery of knee full range of motion (ROM). Exercises for muscle reinforcement of all lower limb muscles and stretching exercises are suggested. A connective tissue massage can be performed to avoid scar fibrous retractions.

Sport activities such as running are allowed 3 weeks after surgery, while contact sports can be performed 1 month after surgery.

1.3. Meniscus repair

Meniscal repair techniques are well established and allow surgeons to repair tears of different complexity and location.

If a repair is performed, it is important to consider the numerous factors involved in healing, such as location and vascularity of the tear, the type of the tear, and quality of the meniscal tissue. Red-on-red tears heal spontaneously within four to six weeks if the necessary immobilization is applied. The purpose of meniscal repair is to safely allow a mobilization of the knee in this period in order for the scar tissue to heal the lesion without losing the knee flexion. The stability of the knee is also important because repairs are more successful in a stable knee. Also, those repairs concurrently performed with an ACL reconstruction have a higher rate of healing, probably because of the various growth factors in the associated hemarthrosis. Chronicity is also a factor. In general, results of meniscal repair are better acutely after tear (< 8 weeks after injury). Younger patients have higher healing rates. Axial alignment is also important: patients with a varus alignment have a lower healing rate for medial repairs.

Meniscal repair can be done by either an open or an arthroscopic technique. Although the open technique has good results, most surgeons now prefer arthroscopic techniques. The implants developed in recent years allow for arthroscopic meniscal suturing with good long-term results in 80% of cases. There exist no universal technique, but rather several techniques which are adapted to different indications. Even if all-inside fourth-generation devices are now the gold standard in the majority of cases, inside-out, outside-in, and even open techniques are still indicated in selected cases. The ultimate goal is to achieve a strong repair.

In the future, the next step will be biological meniscus repair by introducing factors such as stem cells, growth factors, or cytokines at the site of the repair to enhance healing.

Rehabilitation:

The patient is allowed to walk with no weight bearing and a brace with extended knee for the first 3 weeks after surgery. Afterwards the brace can be removed during walking and progressive weight bearing is recommended. Knee passive mobilization can be started right after surgery within 60° of knee ROM during the first week. Afterwards active mobilization within 60° is allowed starting from the second week after surgery and increasingly to 90° at four weeks and subsequently to full knee ROM restoration.

Exercises for muscle reinforcement with open kinetic chain are allowed after 1 week, while exercises with closed kinetic chain can be started from the third week with knee ROM set at 45° and from 90° to full ROM starting 4 weeks after surgery.

Sport activities such as running are allowed 2 months after surgery, practice on field after 3 months while full sport resumption at competitive level is recommended 5 months after surgery.

1.4. Meniscus replacement: allograft transplantation and scaffolds

It is well accepted that complete or even partial meniscectomy will lead to degenerative changes and pain in the affected compartment (Fairbank).

A symptomatic (painful) patient with total/subtotal lack of a functional meniscus is a candidate for a meniscal allograft transplantation (MAT). Meniscal allografts have been used for over a decade with reasonable results. If the meniscus allograft is harvested in a sterile fashion, it can be used when the tissue bank has found the donor to be free of transmissible diseases. This avoids the use of irradiation to sterilize the graft, in order to not alter meniscal transplant structure. Deep-freezing (-80 °C) appears to be the most accepted method of preservation. Meniscus allograft cellularity is very low: the blood group of donor and receiver has not to be compatible and postoperative immunosuppressive therapy is not required. With proper technique, the grafts have been shown to heal in the peripheral repair site and at its insertion. Open or mini-open surgery is required for bone plug fixation. With improvements in meniscal fixation and suturing devices, arthroscopic MAT without bone plugs is nowadays possible with the invasiveness of an arthroscopic soft-tissue procedure (Fig. 1). The literature does not indicate whether one or the other technique is superior in the long-term. Nowadays MAT is performed also as a career salvage procedure in athletes with 75% of good results (Marcacci et al).

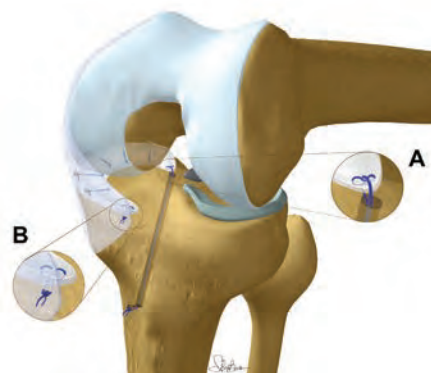


Figure 1: Arthroscopic meniscus allograft transplantation without bone plugs. (reproduced from Marcacci M, Zaffagnini S, Marcheggiani Muccioli GM et al. Meniscal Allograft Transplantation Without Bone Plugs : A 3-Year Minimum Follow-up Study. Am J Sports Med. 2012;40:395-403)

If a partial meniscectomy previously has been performed, a meniscal substitute is reasonable if the patient develops a painful knee. The first meniscus reconstruction device was developed in 1986: the Collagen Meniscus Implant (CMI). This scaffold was tested in animals

and subsequently in people and was found to successfully replace lost segments of meniscus tissue with good long-term follow-up results (Zaffagnini et al) (Fig 2). In animal studies, long-term assessment of a polyurethane scaffold showed that transformation into meniscus-like tissue took place as the implant slowly degraded. First human safety and efficacy studies showed statistically significant clinical improvements at two years (Verdonk et al, Kon et al).

Factors that contribute to the failure of a meniscal allograft/scaffold and thus are considered contraindications include patients with grade IV chondral changes, ligamentous instability, and knee malalignment.

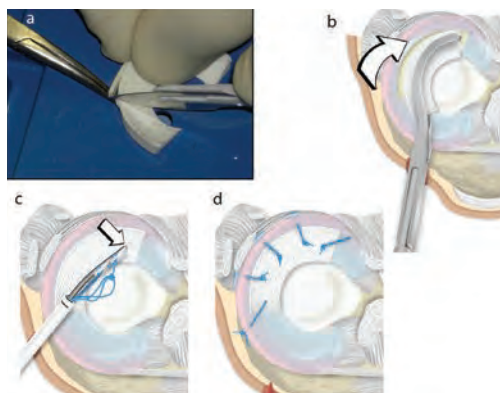


Figure 2: Arthroscopic collagen meniscus implantation. (reproduced from Zaffagnini S, Marcheggiani Muccioli GM, Grassi A et al. Arthroscopic lateral collagen meniscus implant in a professional soccer player. *Knee Surg Sports Traumatol Arthrosc.* 2011;19:1740–1743)

Rehabilitation:

The patient is allowed to walk with crutches and no weight bearing for the first month after surgery. Afterwards progressive load is suggested. Knee passive mobilization can be started right after surgery within 30° of knee ROM during the first week. Afterwards active mobilization within 60° is allowed starting from the second week after surgery and increasingly to 90° at 4 weeks and subsequently to full knee ROM restoration. Exercises for muscle reinforcement with open kinetic chain are allowed after 8 weeks with reduced knee ROM, while exercises with closed kinetic chain can be started from the fourth week with knee ROM set at 45° and from 90° to full ROM starting 8 weeks after surgery.

Sport activities such as running are allowed 4 months after surgery, practice on field after 4 to 5 months while full sport resumption at competitive level is recommended 6 months after surgery.

2. Collateral ligaments

2.1. Collateral ligaments injury

Injuries to the medial collateral ligament (MCL) occur much more frequently than to the lateral collateral ligament (LCL). The mechanism of MCL injury is a valgus force caused by lateral contact to the knee or lower leg, which either injures the distal femoral physis (in the skeletally immature patient), MCL (in the young to middle-aged adult), or the lateral tibial plateau (in the middle-aged to older adult or senior citizen). Careful physical examination

of the knee can confirm the presence of a Salter-Harris injury to the distal physis in the adolescent athlete; pain proximal to the normal insertion of the MCL and, more importantly, pain extending across the femur to the lateral side is common. Radiographs or MRI also can confirm diagnosis.

Physical examination determines whether treatment of MCL or LCL sprains is nonsurgical (majority of tears) or surgical (associated with cruciate and/or posteromedial corner tears).

Evaluation for growth plate injuries in adolescents, for instability in extension (indicating a posterolateral or medial capsular tear), and for complete cruciate ligament injuries is essential.

2.2. MCL injury treatment

Isolated injuries to the MCL are most common, with knee laxity at 20° to 30° of flexion; however, these injuries are stable with the knee in full extension (indicating intact posteromedial capsule). Isolated MCL injuries documented on physical examination are usually treated nonsurgically with protection from valgus. Isolated MCL injuries associated with ACL tears but with intact posteromedial capsule are usually treated nonsurgically at the time of ACL reconstruction. In the relatively rare case of MCL injuries with laxity in full extension, the posteromedial capsule is torn and usually one or both cruciates have been torn. In this circumstance, MRI to localize the site of the tear and fully define the associated injuries (especially of the cruciates) is helpful in surgical reconstruction of one or both cruciates and repair of the MCL and posteromedial capsule tears. MCL reconstruction (usually with allograft tendons) is very rare performed only in severe acute midsubstance injuries or chronic medial knee injuries (Laprade RF et al).

2.3. LCL injury treatment

Isolated injuries to the LCL are relatively rare but can occur with a contact mechanism that causes a complete ACL rupture. If laxity is present only in 20° to 30° of flexion, a palpable LCL in the figure-of-4 position is found, and no laxity is present in full extension, then a complete disruption of LCL is not present and the knee can be protected from varus stress with a brace in the healing phase before ACL reconstruction. When significant laxity is present in full extension, then a complete disruption of the LCL with associated posterolateral corner tears with involvement of a cruciate ligament (PCL or ACL). In the last case primary surgical repair is indicated in acute cases, usually with cruciate reconstruction. In combined chronic injuries to the LCL and posterolateral corner + ACL and/or PCL, surgical reconstruction of the injured ligaments is indicated (usually with allograft tissue) (Laprade RF et al).

Rehabilitation (for both MCL and LCL injury treatment)

Phase 1: from 1 to 4 weeks after surgery

Patient is allowed to walk with no weight bearing and a brace with extended knee.

Knee passive mobilization with Continuous Passive Motion (CPM) can be started 10 days after surgery.

Phase 2: from 5 to 8 weeks after surgery

Patient is allowed to walk with no brace and progressive weight bearing is allowed. Full knee ROM should be achieved.

Hidrokinestherapy and stationary bicycle can be performed. Muscular reinforcement of hip, knee and ankle by means of rubber bands and closed kinetic exercises within 45° of knee

ROM are suggested.

Phase 3: from 9 to 12 weeks after surgery

The patient can perform open kinetic exercises and closed kinetic exercises within 90° of knee flexion. Knee proprioceptive exercises are recommended.

Phase 4: from 13 to 20 weeks after surgery

Running in a straight line is allowed. Complete recovery of muscular strength of operated knee should be achieved.

Phase 5: from 21 to 24 weeks after surgery

Side cuts, jumps and complete aerobic activity can be performed

3. Cruciate ligaments

3.1. Cruciate ligaments injury

Noncontact (approximately 70%) and sports-related (approximately 80%) knee sprains are the most common mechanisms of injury for anterior cruciate ligament (ACL) tears. The majority of patients do not return to play in most sports after the injury without surgery. Contact injuries are more likely to involve MCL injuries. Patients injured while jumping have a significant increase in intra-articular injuries. Female athletes have a double risk of ACL tears when participating in the same sports and at the same levels as male athletes.

Physical examination remains the foundation in diagnosis of ACL (Lachman and Pivot-shift test) and PCL (posterior

drawer test) tears. Radiographs are normal in more than 95% of young, active athletes. The rare presence of a Segond fracture is pathognomonic for an ACL tear. MRI is particularly helpful when the diagnosis is in doubt. Antero-posterior laxity (Lachman test) measurement by arthrometer is the gold-standard instrumental examination. Quantitative pivot-shift analysis with inertial sensors are under research (Zaffagnini S, Lopomo N et al).

Complete posterior cruciate ligament (PCL) tears are extremely uncommon during sports activity, with the exception of occasional knee dislocations. However, PCL tears involving one or both collateral ligaments are common during motor vehicle crashes (higher-velocity and posterior-drawer or overextension mechanism of injury).

3.2. ACL reconstruction

The variables that need to be considered in the decision of whether to reconstruct the ACL-deficient knee include the presence of a repairable meniscus lesion, desired return to competitive sports activity, and involvement in sports/working/leisure activity involving cutting and pivoting. Whether ACL reconstruction prevents or delays knee osteoarthritis is unknown. Prerequisites to ACL reconstruction are nearly full range of motion, good quadriceps tone, normal gait, and limited effusion; all are signs that the knee has recovered from acute inflammatory trauma of hemarthrosis and that arthrofibrosis is less likely to develop postoperatively. Numerous surgical procedures have been described to stabilize a knee with a torn anterior cruciate ligament (ACL).

Regarding the graft choice, for many years the central third of the patellar tendon with bone plugs (bone-patellar tendon-bone: BPTB) autograft has been the most common type used because of excellent long-term results and strong bony fixation (Howe JG, Johnson RJ et al.). More recently the use of autogenous hamstrings (gracilis and

semitendinosus: Gr/St) tendons rise up due to some interesting advantages: small incision, large graft when gracilis and semitendinosus are sutured together, very similar biomechanical characteristics to ACL, rapid and safe harvest. New techniques for hamstring fixation have improved stability of the construct and currently the results achieved with BPTB or Gr/St are comparable (Mohtadi NG et al). Typically, autogenous grafts are harvested from the

ipsilateral knee. The use of allograft tissue for reconstruction of all knee ligaments is becoming more popular. It is appealing for multiligament injuries or revision cases in which autograft choices may be limited. Some studies have shown good results using allograft tissue for ACL reconstruction, although longterm results are lacking.

Regarding graft location, ACL reconstruction techniques can be divided in two major groups: intra-articular and extra-articular. The various intra-articular reconstructions repair the primary ACL lesion and nowadays are performed under arthroscopic control, without opening the knee joint. Randomized trials have not shown differences in clinical outcomes using different arthroscopic surgical techniques: two-incision (rear entry) versus single incision, double-bundle versus single-bundle (Mascarenhas R et al). The extra-articular techniques attempt to prevent the lateral tibial condyle anterior sub-luxation, controlling the most dynamic compartment of the knee. However, extra-articular procedures alone (as techniques by Lemaire or MacIntosh) do not directly repair the primary lesion that causes instability and have demonstrated a high percentage of degenerative changes at long-term (Vail TP et al). The first authors to combine the intra- and extra-articular reconstruction were Zarins and Rowe. The first arthroscopic procedure developed for combined intra- and extra-articular ACL reconstruction with hamstrings and used in clinical practice since 1992 is the one developed by the senior authors (Marcacci M, Zaffagnini S, et al.) (Fig. 3). From this starting point, after the first good preliminary results, many authors developed similar techniques.

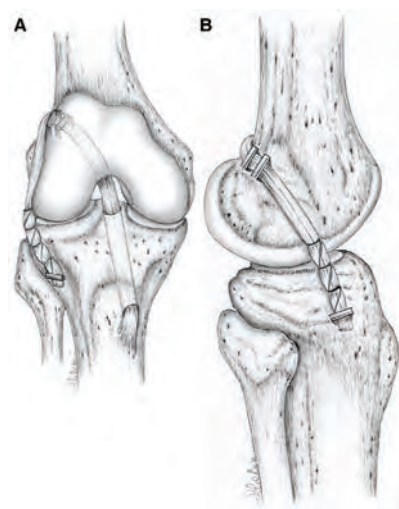


Figure 3 Anterior cruciate ligament reconstruction with hamstring tendons plus extra-articular plasty. a anteroposterior view; b lateral view. (reproduced from Marcacci M, Zaffagnini S, Giordano G et al. Anterior cruciate ligament reconstruction associated with extra-articular tenodesis: a prospective clinical and radiographic evaluation with 10- to 13-year follow-up. *Am J Sports Med.* 2009;37:707-714)

Rehabilitation:

Phase 1: from 1 to 4 weeks after surgery

Patient is allowed to walk with no weight bearing for the first 2 weeks after surgery, afterwards progressive weight bearing is recommended.

Knee passive mobilization can be started right after surgery followed by active mobilization until 90° of knee ROM within the first 2 weeks and full ROM restoration within 4 weeks after surgery. Isometric quadriceps reinforcement exercises must be performed.

Phase 2: from 5 to 8 weeks after surgery

Hidrokinestherapy and stationary bicycle can be performed. Muscular reinforcement of hip, knee and ankle by means of rubber bends and closed kinetic exercises within 45° of knee ROM are suggested.

Phase 3: from 9 to 12 weeks after surgery

The patient can perform open kinetic exercises avoiding the last 30° of knee extension and closed kinetic exercises within 90° of knee flexion. Knee proprioceptive exercises are recommended.

Phase 4: from 13 to 20 weeks after surgery

Running in a straight line is allowed. Complete recovery of muscular strength of operated knee should be achieved.

Phase 5: from 21 to 24 weeks after surgery

Side cuts, jumps and complete aerobic activity can be performed.

3.3. PCL reconstruction

PCL injuries with or without associated MCL/posteromedial capsular or LCL/posterolateral capsular injuries have been investigated using in vitro biomechanical studies. Key decision-making criteria in the clinical management of PCL injuries include magnitude of trauma (sports activity versus motor vehicle crashes), the degree of posterior laxity and associated collateral or ACL tears.

Partial PCL tears are more common in low-velocity injuries (especially from sports activities) and are treated nonsurgically. Patients with partial PCL injuries have shown healing over time on follow-up MRI. It has been shown that 1+ or 2+ (5 to 10 mm) posterior laxity is well tolerated even in the highly competitive athlete.

Complete PCL tears are more common from high-velocity injuries resulting from motor vehicle crashes and are often associated with collateral injuries and possible knee dislocation.

When PCL reconstructions are considered, the choice of surgical approach and grafts should be matched. Traditionally a single tibial tunnel plus single femoral tunnel approach was used to reconstruct the anterolateral PCL bundle. Another possible technique (developed to avoid the so-called "killer" curve at the exit of the tibial tunnel) is the tibial inlay technique. Tibial inlay requires a bone block and either a patellar tendon or Achilles tendon graft. If tibial inlay is preferred and autograft tissue is used, then a single femoral tunnel with interference screws is used. No comparative in vivo clinical studies have provided a preferred technique (May JH et al).

Nowadays new double-bundle techniques for PCL reconstruction, using tibial tunnel or tibial inlay approach, are developed by some authors using allograft tissue. Reproducible in vitro biomechanical studies have not identified the best approach or graft choice to date; however, research is ongoing (Zaffagnini et al). The surgeon should choose the safest, most reproducible approach and technique to improve posterior stability.

Rehabilitation:

Phase 1: from 1 to 4 weeks after surgery

Patient is allowed to walk with no weight bearing and a brace

with extended knee.

Knee passive and assisted active mobilization exercises can be started right after surgery within 60° of knee ROM.

Phase 2: from 5 to 8 weeks after surgery

Patient is allowed to walk with no brace and progressive weight bearing is allowed. Full knee ROM should be achieved.

Hidrokinestherapy and stationary bicycle can be performed. Muscular reinforcement of hip, knee and ankle by means of rubber bends and closed kinetic exercises within 45° of knee ROM are suggested.

Phase 3: from 9 to 12 weeks after surgery

The patient can perform open kinetic exercises and closed kinetic exercises within 90° of knee flexion. Knee proprioceptive exercises are recommended.

Phase 4: from 13 to 20 weeks after surgery

Running in a straight line is allowed. Complete recovery of muscular strength of operated knee should be achieved.

Phase 5: from 21 to 24 weeks after surgery

Side cuts, jumps and complete aerobic activity can be performed.

ABBREVIATIONS

MAT, meniscus allograft transplanatation

CMI, collagen meniscus implant

ACL, anterior cruciate ligament

PCL, posterior cruciate ligament

MCL, medial collateral ligament

LCL, lateral collateral ligament

REFERENCES

1. Jouve F, Ovadia H. Meniscal repair: Technique. In: Verdonk R and Beaufils PH (2010) *The Meniscus*.
2. Springer, Berlin Heildeberg
3. Howe JG, Johnson RJ, Kaplan MJ, Fleming B, Jarvinen M. (1991) Anterior cruciate ligament reconstruction using quadriceps patellar tendon graft. Part I. Long-term follow-up. *Am J Sports Med* 19:447-457
4. Fairbank TJ (1948) Knee joint changes after meniscectomy. *J Bone Joint Surg Br* 30:664-670
5. Kon E, Filardo G, Zaffagnini S, Di Martino A, Di Matteo B, Marcheggiani Muccioli GM, Busacca M, Marcacci M (2014) Biodegradable polyurethane meniscal scaffold for isolated partial lesions or as combined procedure for knees with multiple comorbidities: clinical results at 2 years. *Knee Surg Sports Traumatol Arthrosc* 22:128-134
6. Laprade RF, Wijdicks CA. (2012) The management of injuries to the medial side of the knee. *J Orthop Sports Phys Ther* 42:221-233
7. LaPrade RF, Wentorf F. (2002) Diagnosis and treatment of posterolateral knee injuries. *Clin Orthop Relat Res* 402:110-121
8. May JH, Gillette BP, Morgan JA, Krych AJ, Stuart MJ, Levy BA. (2010) Transtibial versus inlay posterior cruciate ligament reconstruction: an evidence-based systematic review. *J Knee Surg* 23:73-79

9. Marcacci M, Zaffagnini S, Giordano G, Iacono F, Presti ML (2009) Anterior cruciate ligament reconstruction associated with extra-articular tenodesis: A prospective clinical and radiographic evaluation with 10- to 13-year follow-up. *Am J Sports Med* 37:707-714
10. Marcacci M, Zaffagnini S, Marcheggiani Muccioli GM, Neri MP, Bondi A, Nitri M, Bonanzinga T, Grassi A (2011) Arthroscopic intra- and extra-articular anterior cruciate ligament reconstruction with gracilis and semitendinosus tendons: a review. *Curr Rev Musculoskelet Med* 4:73-77
11. Marcacci M, Zaffagnini S, Marcheggiani Muccioli GM, Grassi A, Bonanzinga T, Nitri M, Bondi A, Molinari M, Rimondi E (2012) Meniscal allograft transplantation without bone plugs: a 3-year minimum follow-up study. *Am J Sports Med* 40:395-403
12. Marcacci M, Marcheggiani Muccioli GM, Grassi A, Ricci M, Tsapralis K, Nanni G, Bonanzinga T, Zaffagnini S (2014) Arthroscopic meniscus allograft transplantation in male professional soccer players: a 36-month follow-up study. *Am J Sports Med* 42:382-388
13. Mascarenhas R, Cvetanovich GL, Sayegh ET, Verma NN, Cole BJ, Bush-Joseph C, Bach BR Jr. (2015) Does Double-Bundle Anterior Cruciate Ligament Reconstruction Improve Postoperative Knee Stability Compared With Single-Bundle Techniques? A Systematic Review of Overlapping Meta-analyses. *Arthroscopy*. doi: 10.1016/j.arthro.2014.11.014.
14. Mohtadi NG, Chan DS, Dainty KN, Whelan DB. (2011) Patellar tendon versus hamstring tendon autograft for anterior cruciate ligament rupture in adults. *Cochrane Database Syst Rev* 7;(9):CD005960
15. Vail TP, Malone TR, Bassett FH. (1991) Long-term functional results in patients with anterolateral rotatory instability treated by iliotibial band transfer. *Am J Sports Med* 20:274-282
16. Verdonk PC, Demurie A, Almqvist KF, Veys EM, Verbruggen G, Verdonk R (2005) Transplantation of viable meniscal allograft: survivorship analysis and clinical outcome of one hundred cases. *J Bone Joint Surg Am* 87:715-724
17. Verdonk PC, Verstraete KL, Almqvist KF, De Cuyper K, Veys EM, Verbruggen G, Verdonk R (2006) Meniscal allograft transplantation: long-term clinical results with radiological and magnetic resonance imaging correlations. *Knee Surg Sports Traumatol Arthrosc* 14:694-706
18. Verdonk R. Meniscal repair: Biomechanics. In: Verdonk R and Beaufils PH (2010) *The Meniscus*. Springer, Berlin Heidelberg
19. Verdonk P, Beaufils P, Bellemans J, Djan P, Heinrichs EL, Huyse W, Laprell H, Siebold R, Verdonk R; Actifit Study Group (2012) Successful treatment of painful irreparable partial meniscal defects with a polyurethane scaffold: two-year safety and clinical outcomes. *Am J Sports Med* 40:844-853
20. Zaffagnini S, Martelli S, Garcia L, Visani A. (2004) Computer analysis of PCL fibres during range of motion. *Knee Surg Sports Traumatol Arthrosc* 12:420-428
21. Zaffagnini S, Marcheggiani Muccioli GM, Lopomo N, Bruni D, Giordano G, Ravazzolo G, Molinari M, Marcacci M (2011) Prospective long-term outcomes of the medial collagen meniscus implant versus partial medial meniscectomy: a minimum 10-year follow-up study. *Am J Sports Med* 39:977-985
22. Zaffagnini S, Marcheggiani Muccioli GM, Grassi A, Bonanzinga T, Filardo G, Canales Passalacqua A, Marcacci M (2011) Arthroscopic lateral collagen meniscus implant in a professional soccer player. *Knee Surg Sports Traumatol Arthrosc* 19:1740-1743
23. Zaffagnini S, Lopomo N, Signorelli C, Marcheggiani Muccioli GM, Bonanzinga T, Grassi A, Visani A, Marcacci M. (2013) Innovative technology for knee laxity evaluation: clinical applicability and reliability of inertial sensors for quantitative analysis of the pivot-shift test. *Clin Sports Med* 32:61-70
24. Zaffagnini S, Grassi A, Marcheggiani Muccioli GM, Tsapralis K, Ricci M, Bragonzoni L, Della Villa S, Marcacci M. (2014) Return to sport after anterior cruciate ligament reconstruction in professional soccer players. *Knee* 21:731-735
25. Zarins B, Rowe CR. (1986) Combined anterior cruciate-ligament reconstruction using semitendinosus tendon and iliotibial tract. *J Bone Joint Surg Am* 68:160-177

QUESTIONS

1. Usually, meniscal repair performed combined with ACL reconstruction has:
 - a. Higher probability of healing
 - b. Lower probability of healing
 - c. Same probability of healing
 - d. It depends from the graft used for ACL reconstruction.
2. Which is the main indication for meniscal substitution?
 - a. Acute lesion of lateral meniscus posterior horn in a 25 years old recreational athlete
 - b. Acute bucket handle tear of medial meniscus in a 40 years old recreational athlete
 - c. Degenerative medial meniscus lesion in an 65 years old ex-athlete
 - d. Knee pain after a previous total/subtotal medial meniscectomy in a 35 years old recreational athlete
3. Which is the main indication for MCL reconstruction?
 - a. Acute isolate grade III lesion
 - b. Acute proximal MCL detachment combined with ACL and PCL injury
 - c. Acute midsubstance MCL grade III tear combined with ACL and PCL injury
 - d. Acute isolate grade II lesion
4. Which is the main aim of lateral extra-articular reconstruction combined to intra-articular ACL reconstruction?
 - a. Prevent anterior sub-luxation of lateral compartment
 - b. Prevent anterior sub-luxation of medial compartment
 - c. Prevent anterior sub-luxation of the whole tibia
 - d. Prevent anterior sub-luxation of the whole femur
5. Which is the main advantage of the inlay technique for PCL reconstruction?
 - a. Possibility to use a wide variety of grafts
 - b. Avoid an excessively acute angle between tibial tunnel and graft inclination
 - c. Less invasive surgical approach
 - d. More anatomical placement of the femoral tunnel

ANSWERS:

1a, 2d, 3c, 4a, 5b



Dr. Domizio Suva

HUG, University Hospitals of Geneva,
Geneva, Switzerland

Domizio.Suva@hcuge.ch

Infections After Total Joint Arthroplasty

Introduction

Total hip replacements (THR) and total knee replacements (TKR) substantially improve patients' quality of life by reducing pain and increasing mobility. However, despite advances in prevention these interventions are associated with a risk of infection of 0.5–1.5% for THR and 1–2% for TKR. After revision surgery the risks of infection are considerably higher, at approximately 10%. In addition to a substantial morbidity for patients due to a high number of surgical revisions, the occurrence of an infection triples the cost of a primary arthroplasty. Certainly this trend is not going to be reversed in the future, as it is estimated that in the U.S.A. in 2030 the number of THR and TKR will increase by 174% and 674%, respectively.

Pathophysiology of infection

There are three pathways for prosthetic contamination: direct inoculation, blood-borne contamination, and contamination by contiguity. Direct inoculation usually occurs in the operating room, but can also occur later in the presence of a wound dehiscence. Once in contact with the implant, bacteria leave their planktonic form to produce a biofilm, the latter composed of 30% bacteria and 70% adhesive and protective matrix. Bacteria in the biofilm are 1,000 to 10,000 times more resistant to antibiotics. From a clinical point of view, the result of biofilm formation is that cure of the infection will require mechanically removing the biofilm, which frequently involves removal of the implant. Removal of the implant will be even more required when bacteria produce a particular phenotype called "small colony variant." In summary, in cases of suspected prosthetic infection it is essential to make the diagnosis as early as possible in order to avoid biofilm formation. Thus the prosthesis can be preserved.

Diagnosis and microbiology

The diagnosis of prosthetic joint infection (PJI) can be very challenging. The latest consensus of the Society of Musculoskeletal Infection in the U.S.A. reported that the diagnosis of PJI requires the presence of one of two major criteria, or three of five minor criteria. (TABLE 1). From a microbiological viewpoint the most commonly involved germs are *Staphylococcus aureus*, coagulase negative staphylococci, and streptococci (TABLE 2). However, one must not forget *Propionibacterium acnes* in infections associated with shoulder prostheses.

Table 1 : Diagnostic criteria according to the Society of Musculoskeletal Infection in the U.S.A.

MAJOR CRITERIA	<ul style="list-style-type: none"> - Presence of 2 positive cultures on peri-prosthetic tissue with an identified germ - Fistula between the prosthesis and the skin
MINOR CRITERIA	<ul style="list-style-type: none"> - Elevated CRP and ESR - Elevated WBC in synovial fluid or positive leukocyte esterase test - Elevated percentage of neutrophils in the synovial fluid - Positive histological analysis of peri-prosthetic tissue - Positive culture of periprosthetic tissue

Table 2 : Germs distribution on prosthesis infection

GERMS	RATE
<i>Staphylococcus aureus</i>	21–43%
Coagulase-negative staphylococci	17–39%
<i>Streptococcus</i>	7–12%
Gram-negative bacilli	5–12%
<i>Enterococcus</i>	1–8%
Anaerobic bacteria	2–6%
No germ found	4–12%
* <i>Propionibacterium acnes</i> (shoulder, spondylodesis)	38%

Clinical presentation

There are basically two presentations of PJI, acute and oligosymptomatic. An acute infection is characterized by chills (bacteremia), local inflammation (pain, redness), joint effusion or draining fluid. When the origin of the infection is blood-borne, the clinical picture may initially be dominated by symptoms and signs associated with a systemic infection, such as endocarditis, pneumonia or urosepsis. Oligosymptomatic infection is more of an indolent nature and sometimes difficult to differentiate from aseptic loosening. The patient has chronic pain, perhaps low-grade fever, a joint effusion or radiological evidence of prosthetic loosening. The search for risk factors helps provide additional information to permit a diagnosis. Some are patient-related such as diabetes, obesity, rheumatoid arthritis or immunosuppression. Surgical risk factors include prolonged operative time, revision surgery, and lack of antibiotic prophylaxis. Finally, the post-operative period can also expose the patient to a higher risk of infection, in particular in the presence of wound problems, hematoma, or if there has been a bacteremia.

Laboratory tests

1. Blood tests

The presence of an erythrocyte sedimentation rate (ESR) ≥ 30 and C-reactive protein (CRP) ≥ 10 allow one to diagnose an acute PJI with a sensitivity of 91–97%, a specificity of 70–80%, and a negative predictive value of 96%. However, in chronic infections, the usefulness of these tests is considerably reduced, since the values can range between normal and elevated.

2. Joint aspiration

The likelihood of a PJI in the hip is high when the synovial fluid contains $>4,200$ leucocytes/ μL , or $>80\%$ granulocytes. Regarding the knee, these values are lower with $\text{WBC} > 1,700/\mu\text{L}$ or $>65\%$ PMN. However, these values only apply to infections diagnosed after the first 1–2 months postoperative; otherwise one must consider superior diagnostic values of $\sim 25,000$ leukocytes per μL .

3. Microbiological analysis

The sensitivity of synovial fluid culture is 45–95%, and that of periprosthetic tissue 65–95%. Periprosthetic tissue culture is the gold standard for diagnosing PJI. To ensure the highest sensitivity it is crucial to stop all antibiotic therapy two weeks before sampling. It is also recommended to take at least three samples that will be shared between bacteriology and histology. Gram staining of periprosthetic tissue has a very poor sensitivity (25%). Cultures of wound or fistula should be avoided.

4. Histopathology

There is a wide variability in the histological diagnostic criteria of infection. There is a consensus that ≥ 5 neutrophils per HPF (400 \times) in 5 different fields is strongly suspicious of PJI.

5. Imaging

Standards radiographs have minimal artifacts, but only low sensitivity and specificity. They may show osteolysis, loosening or signs of implant migration, or conversely, cortical and subperiosteal thickening. Computed tomography (CT) allows for a better understanding of the patient's bone stock, but is frequently distorted by metal artifacts. Finally, bone scan is very sensitive, but lacks specificity since the operative site can show persistent uptake for several months following surgery.

Treatment

There are 7 treatment options, as follows: (1) débridement and implant retention (DAIR); (2) one-stage prosthetic exchange; (3) two-stage prosthetic exchange; (4) removal of the prosthesis; (5) arthrodesis; (6) suppressive antibiotic therapy; or (7) the final option, amputation.

1. Debridement and implant retention

This treatment can be performed (a) when symptoms are present for less than 3 weeks (acute infection); (b) in the absence of loosening; (c) with no fistula or abscess; and (d) in the presence

of a bacteria that is sensitive to antibiotics. As regards the latter, an infection due to *Staphylococcus aureus* is associated with a lower healing rate than a streptococcal infection. Furthermore, it is recommended to change the modular components as this improves the chances of success, which overall are approximately 70–80%. The advantage of this option is that it is a relatively “minimally invasive” surgery, allowing better early mobilization. However, it must be pointed out that in cases of failure, the success rate of a prosthetic exchange is lower.

2. One-stage exchange

This is indicated (a) when symptoms last more than three weeks; (b) in the presence of good soft tissues without abscess or fistula; and (c) when the infection is caused by bacteria susceptible to antibiotics. One-time surgery has the advantage of a better functional recovery. However, the risk of failure and the surgical difficulty still encourage most surgeons to favor a two-stage exchange.

3. Two-stage exchange

This is the preferred treatment of PJI, and is indicated (a) when symptoms last >3 weeks; (b) in the presence of resistant organisms; (c) in the presence of poor soft tissues; and (d) in the presence of a fistula or abscess. The first stage consists of débridement and implant removal followed by a delay that can be short (2–4 weeks) or long (6 weeks). When the interval is short, the new prosthesis is implanted immediately at the end of this time and no antibiotic window or sampling is performed. When the interval is long, the second stage is usually performed after 8 weeks (6 weeks of antibiotics + 2 weeks of window). The 2nd stage includes prosthetic reimplantation associated with antibiotic therapy of either six weeks after a short interval, or for a variable duration (according to the results of culture) during a long interval.

Conclusion

The infection of a prosthetic joint replacement is a major complication, resulting in substantial morbidity for the patient and increased cost of the primary arthroplasty. Currently, the gold standard for diagnosis is the microbiological analysis of tissue biopsy, and the treatment of choice is the two-stage prosthesis exchange. A rapid and accurate diagnosis is the key pillar of management that requires a multidisciplinary collaboration between surgeons and infectious disease specialists to define the most appropriate strategy for each particular patient. The résumé and decision-making analysis are summarized in Figure 1.

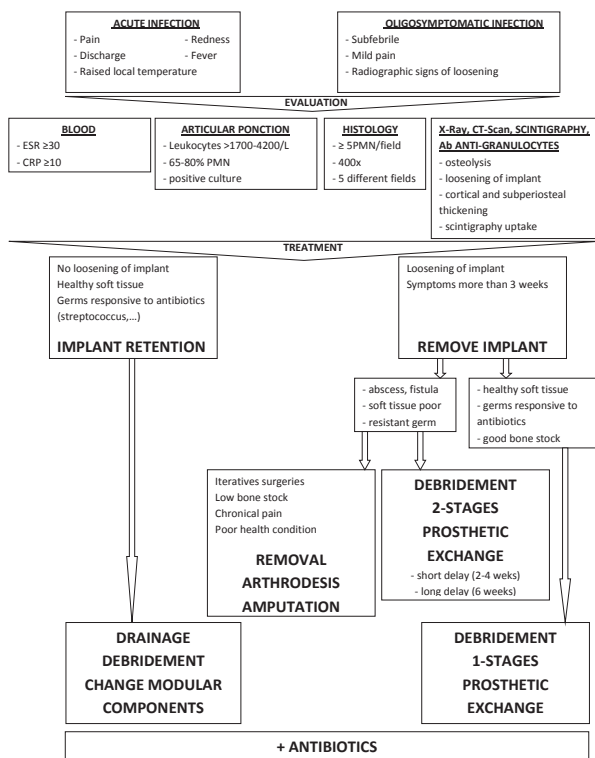


Figure 1: decision making in the presence of a PJI

References

1. Pivec R, Johnson AJ, Mears SC, Mont MA. Hip arthroplasty. *Lancet*. 2012 Nov 17;380(9855):1768-77.
2. Kurtz SM, Lau E, Ong K, Zhao K, Kelly M, Bozic KJ. Future young patient demand for primary and revision joint replacement: national projections from 2010 to 2030. *Clinical orthopaedics and related research*. 2009 Oct;467(10):2606-12.
3. Mont MA, Issa K. Updated projections of total joint arthroplasty demands in America. Commentary on an article by Steven M. Kurtz, PhD, et al.: «Impact of the Economic Downturn on Total Joint Replacement Demand in the United States. Updated Projections to 2021». *The Journal of bone and joint surgery American volume*. 2014 Apr 16;96(8):e68.
4. Lidgren L. Joint prosthetic infections: a success story. *Acta orthopaedica Scandinavica*. 2001 Dec;72(6):553-6.
5. Corvec S, Portillo ME, Pasticci BM, Borens O, Trampuz A. Epidemiology and new developments in the diagnosis of prosthetic joint infection. *The International journal of artificial organs*. 2012 Oct;35(10):923-34.
6. Public Health England. Protocol for the Surveillance of Surgical Site Infection. www.hpa.org.uk. 28 June 2013, date last accessed.
7. Gristina AG, Naylor PT, Webb LX. Molecular mechanisms in musculoskeletal sepsis: the race for the surface. *Instructional course lectures*. 1990;39:471-82.
8. Parvizi J, Gehrke T. International consensus on periprosthetic joint infection: let cumulative wisdom be a guide. *The Journal of bone and joint surgery American volume*. 2014 Mar 19;96(6):441.
9. Zimmerli W, Trampuz A, Ochsner PE. Prosthetic-joint infections. *N Eng J Med*. 2004 Oct 14;351(16):1645-54.

10. Poss R, Thornhill TS, Ewald FC, Thomas WH, Batte NJ, Sledge CB. Factors influencing the incidence and outcome of infection following total joint arthroplasty. *Clinical orthopaedics and related research*. 1984 Jan-Feb(182):117-26.
11. Jansen E, Nevalainen P, Eskelinen A, Huotari K, Kalliovalkama J, Moilanen T. Obesity, diabetes, and preoperative hyperglycemia as predictors of periprosthetic joint infection: a single-center analysis of 7181 primary hip and knee replacements for osteoarthritis. *The Journal of bone and joint surgery American volume*. 2012 Jul 18;94(14):e101.
12. Schinsky MF, Della Valle CJ, Sporer SM, Paprosky WG. Perioperative testing for joint infection in patients undergoing revision total hip arthroplasty. *The Journal of bone and joint surgery American volume*. 2008 Sep;90(9):1869-75.
13. Trampuz A, Steinrucken J, Clauss M, Bizzini A, Furustrand U, Uckay I, et al. [New methods for the diagnosis of implant-associated infections]. *Revue medicale suisse*. 2010 Apr 7;6(243):731-4.
14. Betz M, Abrassart S, Vaudaux P, Gjika E, Schindler M, Billieres J, et al. Increased risk of joint failure in hip prostheses infected with *Staphylococcus aureus* treated with debridement, antibiotics and implant retention compared to *Streptococcus*. *International orthopaedics*. 2014 Sep 4.
15. Zurcher-Pfund L, Uckay I, Legout L, Gamulin A, Vaudaux P, Peter R. Pathogen-driven decision for implant retention in the management of infected total knee prostheses. *International orthopaedics*. 2013 Aug;37(8):1471-5.
16. Bernard L, Legout L, Zurcher-Pfund L, Stern R, Rohner P, Peter R, et al. Six weeks of antibiotic treatment is sufficient following surgery for septic arthroplasty. *The Journal of infection*. 2010 Jul;61(2):125-32.
17. Wu CH, Gray CF, Lee GC. Arthrodesis Should Be Strongly Considered After Failed Two-stage Reimplantation TKA. *Clinical orthopaedics and related research*. 2014 Nov;472(11):3295-304.

QUESTIONS

1. Which one of the following statements is true regarding periprosthetic joint infection (PJI)?
 - a. PJI increases the risk of one-year mortality
 - b. The risk of infection is ~5% after primary THR
 - c. Early infections are associated with less virulent bacteria
 - d. Remote infections increase the risk of seeding a THR
 - e. Histopathology analyses are currently the gold standard
2. Among the following patient characteristics, which one is a contra-indication to débridement and implant retention of an infected THR?
 - a. Patient with slightly damaged tissues
 - b. Patient with several comorbidities
 - c. Patient with symptoms lasting more than 2 weeks
 - d. Patient with a *S.epidermidis* infection
 - e. Patient with a *S.aureus* infection

3. Which one of the following statements is true in cases of an infected TKR?
- a. Débridement and retention is increasingly recommended
 - b. One-stage revision is the gold standard for an infected TKR
 - c. Two-stage revision is indicated in the presence of a sepsis
 - d. There is a place for partial prosthetic exchange (i.e., only femur or tibia)
 - e. In unclear clinical situations it is best to wait and see

ANSWERS:

- 1a PJI increases ~5x the risk of death within 12 months.
- 2e *S. aureus* infections are significantly associated with a higher rate of failure after débridement and implant retention.
- 3c Two-stage revision remains the gold standard for the most severe clinical situations, i.e. patients with sepsis.



Mr. Stephen Cannon

The Royal National Orthopaedic Hospital,
Stanmore, United Kingdom
cannon.frcs@gmail.com

Diagnostic And Recognition Of Primary Bone Tumours

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

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

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

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

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

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

5. The radiological appearances of the lesion itself.

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

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

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

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

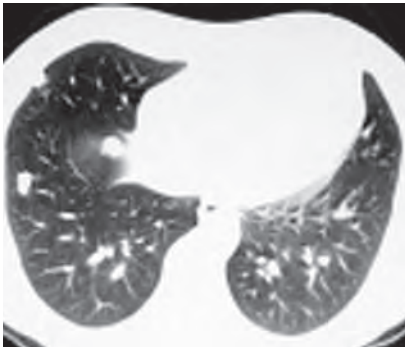
Local staging of the disease essentially wishes to draw out the local extent of the lesion and by use of an MRI of the whole bone define any local skip metastases. The local staging also requires an estimation of the extraosseous extent of the disease, the involvement of soft tissue muscle compartments,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

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

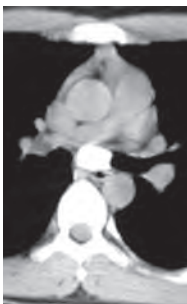
Distal staging is performed by the use of CT scan of the pulmonary lung fields as 95% of metastatic disease involves the pulmonary tissue, but a Technetium bone scan is also useful for picking up both soft tissue and bony metastases. If the bone scan picks up a distant metastasis in bone then further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

Distant Staging

Chest CT



Lung metastases



Mediastinal nodes



Vertebral metastases

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

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

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

References

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

QUESTIONS

1. Bone Sarcoma:
 - a. Have an incidence of 5 per million population
 - b. Have no variability in aggressiveness
 - c. Occasionally present as a fracture
 - d. Usually present below the age of 5 years
 - e. May be indistinguishable from infection
2. Name the six appropriate investigations utilised in the diagnosis and staging of a suspected bone sarcoma:

3. In Imaging tools:

- a. The MRI Scan is best for assessment of ossification of the lesion
- b. The Tech, Bone Scan gives an accurate assessment of extra osseous extent.
- c. Intraosseous extent is best assessed by MRI Scan
- d. PET Scanning has a very low false +ve rate
- e. CT is poor in the recognition of cortical destruction

4. A radiological narrow zone of transition is found in:

- a. Giant Cell Tumour
- b. Osteosarcoma
- c. Ewings Sarcoma
- d. Primary Lymphoma of Bone
- e. Metastasis

5. Which of the following tumours are classified as ENNEKING Grade 1

- a. Osteblastoma
- b. Parosteal Osteosarcoma
- c. Synovial Sarcoma
- d. Ewings Sarcoma
- e. Angiosarcoma of bone

ANSWERS:

- 1. 1a = T, 1b = T, 1c = T, 1d = F, 1e = T
- 2. 1) Plain X-ray in 2 dimensions, 2) MRI Scan, 3) CT SCAN, 4) Tech Bone, 5) Biopsy, 6) Haematological Investigations
- 3. 3a = F, 3b = F, 3c = T, 3d = F, 3e = F
- 4. 4a = T, 4b = F, 4c = F, 4d = F, 4e = F
- 5. b

T=True and F=False



Prof. Dr. Miklós Szendrői

Semmelweis University Budapest, Budapest, Hungary

szenmik@gmail.com

Diagnostic Algorithm And Treatment Options In Bone Metastasis

Introduction

Oncological management is becoming an increasingly serious task in orthopaedic and trauma surgery.

In the past without well-established follow-up protocols metastases were recognized late and mostly palliative treatments, radiotherapy and pain killing were favoured. Nowadays, modern diagnostic tools (PET-CT, whole body MRI, etc.) are included in the follow up protocols allowing for early detection of metastases. Besides improvements in the field of chemo-, and radiotherapy new targeted therapeutic agents such as bisphosphonates and denosumab (antigen against RANKL) reduce skeletal events. A broad spectrum of procedures are available to reconstruct defects, osteosynthesis may be performed, often by employing minimal invasive techniques. All of this results in significant longer survival rates for metastatic patients even those who have multiple metastases. Data from the Scandinavian Skeletal Metastasis Registry regarding the past decade supports this:

- incidence of cancer has increased by 18%
- mortality of cancer has increased by 2%

Epidemiology and characteristics of skeletal metastases
- Cancer - 2nd most frequent causative factor for death
- Every fourth person dies of cancer
USA: 1,2 million new tumour cases annually
- 300.000 metastases in the skeletal system
- 20 % will develop a pathological fracture (Capanna and Campanacci 2001)
- In 65-75 % of bone metastases the primary site is: lung, breast, kidney and prostate
- Imaging: lytic, mixed or sclerotic lesions
- Periosteal reaction is usually absent
- 10-20% are solitary at recognition but multiplication occurs in 1-3 yrs
- Pathological fracture in 20% of the cases
Risk factors (Mirel, 1989):
- Size (more than 2 cm)
- Location (lower limb, proximal femur)
- Blastic/lytic
- Pain

Metastases are 80-100 times more common than the skeletal system than primary malignant bone tumours! The bones most frequently involved listed according to decrease in prevalence 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.

Clinical signs, symptoms:
- deep intermittent pain, independent of movement
- often presents weeks or months before x-ray changes are detected
but: there are many reports on painless skeletal metastases in the early stages
- pathologic fracture as first episode in 10-30% of cases
- primary cancer in the case history in cc. 60-70% of cases
- laboratory tests can be useful for recognising the primary site, especially in case of prostate, thyroid cancer or myeloma.
- lytic metastases of kidney or lung cancer fracture frequently, while osteoplastic metastases of prostate cancer rarely break and have a good propensity to heal
- SRE (skeletal related events like fracture, cord compression, radiotherapy for bone metastases) is present in 10 to 20% at diagnosis of lung, breast and prostate cancer, increase, however, up to 50% during the follow-up

Imaging

In suspected cases, e.g. when there is local bone pain following a tumorous history, an X-ray is taken of the area in question and CT, MR (occasionally PET-CT) scans are added if necessary. In the spine, in contrast to spondylitis the tumor involves single vertebral bodies invading the intervertebral space in later stages only. In the long tubular bones the lesion may be central, though it is more often eccentric, and involves the cortex. Periosteal reaction is absent in most cases. A bone scan is also extremely important in determining whether the process is solitary or multiplex (Figure 1a and b).

Kidney and lung cancer usually produce lytic metastases in the bones, prostate cancer usually sclerotic ones, breast, thyroid and gastrointestinal cancers have most often mixed sclerotic-lytic metastases.

Diagnostic algorithm for skeletal metastases

(Flow chart for establishing the diagnosis and treatment of skeletal metastases)

When the patient is admitted with a lytic lesion or impending fracture that raises suspicion of a skeletal metastasis, systematical evaluation is necessary (Figure 1).

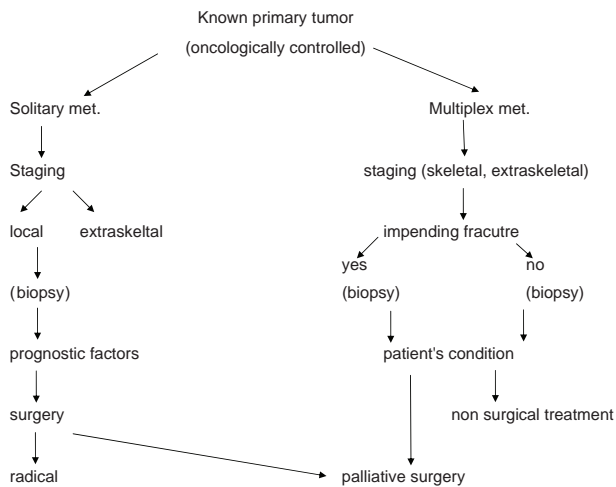


Figure 1: Diagnostic algorithm for impending fracture

In the case when the primary tumour is unknown, finding the primary site and staging of the primary tumour is of major importance. It determines the treatment of the metastasis whether the primary tumour can be oncologically controlled, excised, irradiated etc. or not. Clinical history, laboratory tests, MRI, bone scan, CT for chest, abdomen and pelvis and x-ray are helpful to screen the four-five most frequent primary tumours.

The treatment policy differs in pathologic fracture and in traumatic fracture of the extremities!!!

If x-ray/MRI/CT raise the diagnosis of pathologic fracture:

- Surgery is semi-urgent
- Set up the right diagnosis (primary or secondary bone tumour, tumour-like lesion)
- Look for the primary site
- Evaluate the prognostic factors than decide upon the right surgical intervention

Trauma: osteosynthesis, healing of the fracture

Metastasis: stabilization, weight bearing, mobility

Prognostic factors

Prognostic factors are dependant on the primary tumour, the metastasis and the patient's condition.

- Type of primary tumour is still the most sensitive prognostic factor (Table 1)
- Is the primary tumour oncologically controlled or not (inoperable, etc.)
- Is the primary site known or does it remain unknown despite careful examination?
- Number of skeletal metastases (soliter versus multiplex)
- Presence of visceral metastases
- General condition of the patient (Karnofsky's index)
- Pathological fracture and age have controversial roles as prognostic factors.

Table 1. Average survival of metastatic patients according to the type of tumour

Type of primary tumour	Average survival (in months)
Breast cancer	34 months
Prostate cancer	24 months
Cervical cancer	18 months
Kidney cancer	12 months
Melanoma	3,5 months
Lung cancer	3-6 months

Significant differences can certainly occur regarding these time frames depending on oncological stage, histological type, grade, radio- and chemotherapy-sensitivity, presence of hormone-receptors, etc. In some studies patients for example. with solitary kidney metastases (interval between primary tumour and appearance of the skeletal metastases more than 4 years) had an average survival more than 30 months. It is also known, that the histological grade, hormone receptor status etc. can also change in the metastasis concerning the primary tumour.

The unfavorable prognostic factors are summarized in Table 2.

Table 2

Unfavourable prognostic factors
- Primary tumor: unknown, or oncologically uncontrolled
- Primary site: lung, liver, pancreas or melanoma
- Axial location
- Multiple/multiorganic metastases
- Short doubling time of metastases
- Radio-, chemotherapy resistency
- Synchron or metachron appearance of metastases
- Poor general condition of patient, hemoglobin less than 7 mmol/l

Surgical treatment

Surgery is rarely the first option in the treatment of skeletal metastases. It is the decision of the oncoteam to recommend radiotherapy, chemotherapy, isotope- or hormon treatment or targeted therapy (bisphosphonates, denosumab, etc.) as the first line. Surgery is usually necessary if there is an impending or pathological fracture, and indications for curative or ablative surgery are present.

Aim of surgical treatment:

- To alleviate the pain
- To prevent the imminent fracture
- To perform an osteosynthesis and strenghten the bone in case of a pathological fracture
- Decompress and stabilize the vertebral column in case of neurological complications and instability of vertebra due to pathological fracture
- Allow the patient to regain his mobility
- Radical excision of solitary metastases
- Improve the quality of life

There is a broad range of the possible surgical procedures for reconstruction of the defect, i.e. plating (Figure 2), intramedullary nailing (Figure 3), curetting the defect and filling it with bone cement or insertion of a normal (Figure 6) or tumor endoprosthesis. Intramedullary nailing is advantageous for it allows stable weight-bearing and even if

the tumor progresses, loosening of the implant is unlikely. In 10-20% of the cases a curative-type radical tumor excision (Table 4) is warranted and limb-saving surgery and reconstruction of the defect by modular tumor endoprosthesis or allograft may be performed.

The most common area for the metastatic lesions is the spine.

Main indications for surgery are:

- Pain
- Instability
- Pathological fracture
- Cord compression, neurological deficit

Cord compression accompanied by a neurological deficit may occur in an unexpected and rapid fashion. Urgent surgery (Figure 4.) is mandatory, a few hours of delay may result in a definitive neurologic complication such as plegia.



Figure 2: Lymphoma of the femur with pathological fracture. Plating and augmentation by bone cement was performed.



Figure 3: Breast cancer metastasis in the femur with pathological fracture (a). Intramedullary nail inserted by minimal invasive technique was performed as an osteosynthesis (b).

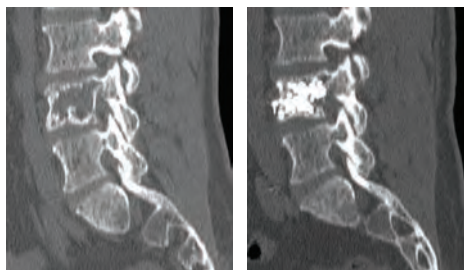


Figure 4: Percutaneous vertebroplasty in a metastatic vertebra with impending fracture. Lytic lesion in vertebral body (a), postoperative x-ray following the vertebroplasty (b).

Summary

- Change in surgical activity for metastatic patient
- Resection of the whole tumor if possible
- Life-long solution by minimal invasive palliative surgery
- Quality of life is an important

Literature:

1. Breast Specialty Group of the British Association of Surg. Oncol.: The management of metastatic bone disease in the United Kingdom. *Eur J Surg Oncol.*, 25: 3-23, 1999.
2. Capanna R., Campanacci D.: The treatment in metastases in the appendicular skeleton. *J Bone Joint Surg.* 83-B: 471-481, 2001.
3. Fizazi K., Carducci M., Smith M. et al: Denosumab versus zoledronic acid for treatment of bone metastases in men with castration-resistant prostate cancer: a randomised, double-blind study. *Lancet* 377(9768): 813-822, 2011.
4. Ford J.A., Jones R., Elders A. et al: Denosumab for treatment of bone metastases secondary to solid tumours: Systematic review and network meta-analysis. *Eur. J. Cancer*, 49: 416-430, 2013.
5. Greco C, Forte L, Erba P, Mariani G. Bone metastases, general and clinical issues. *Q J Nucl Med Mol Imaging.* 55:337-52, 2011.
6. Katagiri H, Okada R, Takagi T et al. New prognostic factors and scoring system for patients with skeletal metastasis. *Can Med* 2014;3(5): 1359-1367.
7. Kirkinis MN, Lyne CJ, Wilson MD et al. Metastatic bone disease: A review of survival, prognostic factors and outcomes following surgical treatment of the appendicular skeleton. *EJSO* 2016;42:1787-1797.
8. Ratasvuori M., Wedin R., Keller J. et al: Insight opinion to surgically treated metastatic bone disease: Scandinavian Sarcoma Group Skeletal Metastasis Registry report of 1195 operated skeletal metastasis. *Surg. Oncol.* 22: 132-138, 2013.
9. Szendrői A., Dinya E., Kardos M. et al: Prognostic factors and survival of renal clear cell carcinoma patients with bone metastases. *Pahology Oncology Research*, 16: 29-38, 2010.
10. Willeumier JJ, van de Linden YM, van de Sande MA et al. Treatment of pathological fractures of the long bones. *EOR* 2016;1:S28-S37.

QUESTIONS

1. Type of cancer which gives more frequent metastases into the bone with the exception:
 - a. kidney
 - b. prostate
 - c. gastrointestinal
 - d. lung
 - e. breast
2. How frequent occur pathologic fracture in skeletal metastases?
 - a. 1%
 - b. 10%
 - c. 20%
 - d. 30%
 - e. 50%
3. Unfavorable prognostic factors with exception:
 - a. breast cancer
 - b. lung cancer
 - c. multiple metastases

- d. unknown primary tumor
 - e. presence of visceral metastases
4. Frequently affected bones by metastases with exception:
- a. vertebra
 - b. scapula
 - c. pelvic bones
 - d. short tubular bones of hand and feet
 - e. long tubular bones
5. Consequence of a delayed operation in case of pathologic fracture of the vertebra
- a. permanent pain
 - b. instability
 - c. progression of the tumorous process
 - d. irreversible neurologic complicaiton, plegia
 - e. catastrophoal bleeding

ANSWERS:

1c, 2c, 3a, 4d, 5d



Dr. Pierre-Yves Zambelli

CHUV, University Hospital of Lausanne, Lausanne, Switzerland
 pierre-yves.zambelli@chuv.ch

Dr. Romain Dayer

HUG, University Hospitals of Geneva, Geneva, Switzerland
 romain.dayer@hcuge.ch

Paediatric Idiopathic Scoliosis

Disclosure: Authors do not have any conflicts of interest relevant to this chapter to report.

Support: None of the authors received financial support for this chapter.

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 ("Scoliosis Research Society (SRS) "). 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) is a crucial associated finding during patients and parents interview and create an absolute necessity to exclude a specific etiology. 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 (Ramirez, Johnston, & Browne, 1997; Sato et al.).

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 (Schulte et al., 2008; Wybier & Bossard).

Additional workup with MRI should be done, especially when contemplating surgical treatment, in selected cases of skin abnormalities along the spine, a left main thoracic curve (i.e. atypical curve), a rapidly progressing curve (Cobb angle progression > 1° per month), idiopathic scoliosis in a child under 10 years (juvenile-onset), 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 +/-1
- Juvenile scoliosis - presenting from age 3 through age 9 +/-1

- Adolescent scoliosis - presenting from age 10 through age 17 +/-1
 - 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 (Karol; "Scoliosis Research Society (SRS) ").

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) ("Scoliosis Research Society (SRS) ");

- 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 (King, Moe, Bradford, & Winter, 1983). It has three main disadvantages:

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

In 2001, Lenke proposed a new classification system for AIS meeting almost all of the above-mentioned criteria. It is based on four radiographs of the spine (standing long-cassette coronal and lateral as well as right and left supine side-bending) which will define 6 curve types. These 6 curve types are determined by the localization, degree, and flexibility of the manifested curves. Curve

types are further classified according to the degree of associated lumbar deformity (lumbar spine modifier) for thoracic curves and according to the sagittal alignment of the thoracic spine (sagittal thoracic modifiers). With these additional parameters, this complex classification system enables the categorization of 42 subtypes of AIS. Its inter- and intraobserver reliability was shown to be superior to the King classification (Lenke et al., 2001). This useful clinical tool is progressively becoming established as a standard worldwide. Although its complexity makes it difficult to use clinically for the general orthopaedic surgeon, it procures a differentiated instrument to the experienced pediatric spine surgeon, facilitating and standardizing the surgical treatment strategy.

The need for follow-up or treatment, and choice treatment for IS are mainly dictated by the risk of curve progression. In this setting, knowledge of the natural history and long-term prognosis associated with untreated IS is mandatory. There are clearly significant differences of natural history between the different types of pediatric IS. Untreated patient with AIS have been shown to be productive and functional at a high level at 50-year follow-up. Although the prevalence of back pain in this patient population is likely to be higher than in the general population, many studies tend to show that this issue is not a significant problem for these patients. On the other hand, cosmetic concerns associated with the development of significant deformity should not be underestimated in patient with untreated AIS (Weinstein et al., 2003). 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 (Pehrsson, Larsson, Oden, & Nachemson, 1992; Pehrsson, Nachemson, Olofson, Strom, & Larsson, 1992). 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 (Karol). 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 bracing, casting or growth-friendly surgical treatment based on distraction (dual spine-based and rib-based growing rods (Fig. 1), vertical expandable prosthetic expandable rib (VEPTR), and remotely expandable devices), compressions (applying compressive forces to the convexity of the curve, inhibiting growth, like vertebral staples and tethers) or guided growth (Shilla procedure and modern Luqué-Trolley) (Cheung et al., 2012; McCarthy et al.; Ouellet; Skaggs et al., 2014; Smith; Tis et al.). None of these surgical techniques are associated with low complications rates and experienced surgical teams should use them accordingly.

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 20° or for skeletally mature patient with a curve measuring less than 45°.

Bracing is proposed for growing adolescent with curves from 20° to 40°. This treatment has been associated with success (i.e. non progression to a degree of curvature necessitating surgery) in a multicenter prospective study, with a significant positive association between hours of brace wear and rate of treatment success (Weinstein,

Dolan, Wright, & Dobbs, 2013). Surgery is generally offered for growing AIS patients with curves exceeding 40° or for skeletally mature patient with curve beyond 45°. 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 (Dayer, Haumont, Belaieff, & Lascombes, 2013). 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 (Ogilvie; Ogilvie et al., 2006).



Figure 1. Infantile idiopathic scoliosis: dual growing rods technique.

The whole perioperative strategy for the surgical treatment of IS should be directed at preventing complications. A blood management protocol including preoperative evaluation, intraoperative cell salvage, topical hemostasis, antifibrinolytics, and hypotensive anesthesia is generally used for the surgical treatment of the pediatric patient with IS, as well as in general for pediatric deformity surgery. Preoperative oral iron and erythropoietin or autologous blood predonation can be used in this setting. Implementation of such a protocol is associated with a low perioperative transfusion rate together with preoperative diagnosis of patients with coagulopathy (Hassan et al.). 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 (Pahys et al., 2009). Intraoperative computed tomography-guided navigation is a promising but irradiating tool to further assist in the accuracy and safe placement of pedicle screws (Ughwanogho, Patel, Baldwin, Sampson, & Flynn), 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 the potential benefit to decrease the number of fused levels and increase postoperative patient satisfaction regarding cosmesis (Cuartas, Rasouli, O'Brien, & Shufflebarger, 2009). 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 (3 incisions technique with muscle splitting 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 (Sarwahi et al.).

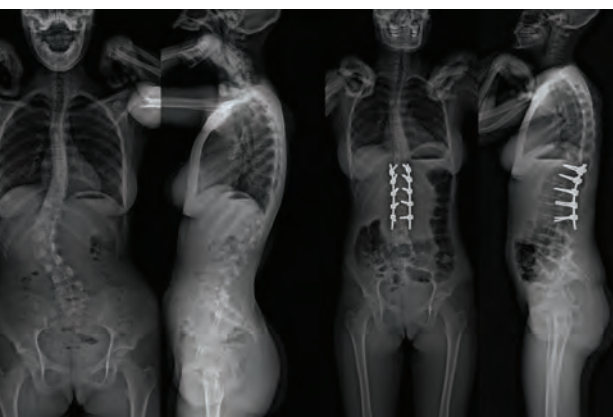
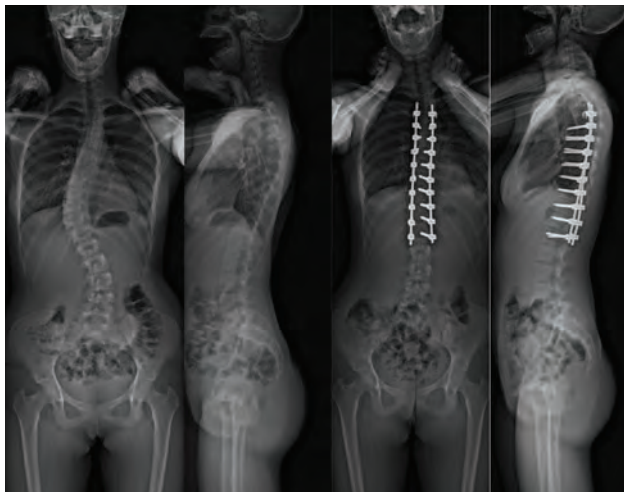


Figure 2 and 3. AIS: posterior-only approach and instrumented fusion.



Figure 4. AIS: anterior-only approach and instrumented fusion.

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.

References

1. Cheung, K. M., Cheung, J. P., Samartzis, D., Mak, K. C., Wong, Y. W., Cheung, W. Y., . . . Luk, K. D. (2012). Magnetically controlled growing rods for severe spinal curvature in young children: a prospective case series. *Lancet*, 379(9830), 1967-1974. doi:10.1016/S0140-6736(12)60112-3
2. Cuartas, E., Rasouli, A., O'Brien, M., & Shufflebarger, H. L. (2009). Use of all-pedicle-screw constructs in the treatment of adolescent idiopathic scoliosis. *J Am Acad Orthop Surg*, 17(9), 550-561.
3. Dayer, R., Haumont, T., Belaieff, W., & Lascombes, P. (2013). Idiopathic scoliosis: etiological concepts and hypotheses. *J Child Orthop*, 7(1), 11-16. doi:10.1007/s11832-012-0458-3
4. Hassan, N., Halanski, M., Wincek, J., Reischman, D., Sanfilippo, D., Rajasekaran, S., . . . Cassidy, J. Blood management in pediatric spinal deformity surgery: review of a 2-year experience. *Transfusion*, 51(10), 2133-2141.
5. Karol, L. A. Early definitive spinal fusion in young children: what we have learned. *Clin Orthop Relat Res*, 469(5), 1323-1329.
6. King, H. A., Moe, J. H., Bradford, D. S., & Winter, R. B. (1983). The selection of fusion levels in thoracic idiopathic scoliosis. *J Bone Joint Surg Am*, 65(9), 1302-1313.
7. Lenke, L. G., Betz, R. R., Harms, J., Bridwell, K. H., Clements, D. H., Lowe, T. G., & Blanke, K. (2001). Adolescent idiopathic scoliosis: a new classification to determine extent of spinal arthrodesis. *J Bone Joint Surg Am*, 83-A(8), 1169-1181.
8. McCarthy, R. E., Sucato, D., Turner, J. L., Zhang, H., Henson, M. A., & McCarthy, K. Shilla growing rods in a caprine animal model: a pilot study. *Clin Orthop Relat Res*, 468(3), 705-710.
9. Ogilvie, J. W. Update on prognostic genetic testing in adolescent idiopathic scoliosis (AIS). *J Pediatr Orthop*, 31(1 Suppl), S46-48.
10. Ogilvie, J. W., Braun, J., Argyle, V., Nelson, L., Meade, M., & Ward, K. (2006). The search for idiopathic scoliosis genes. *Spine (Phila Pa 1976)*, 31(6), 679-681.
11. Ouellet, J. Surgical technique: modern Luque trolley, a self-growing rod technique. *Clin Orthop Relat Res*, 469(5), 1356-1367.
12. Pahys, J. M., Guille, J. T., D'Andrea, L. P., Samdani, A. F., Beck, J., & Betz, R. R. (2009). Neurologic injury in the surgical treatment of idiopathic scoliosis: guidelines for assessment and management. *J Am Acad Orthop Surg*, 17(7), 426-434.
13. Pehrsson, K., Larsson, S., Oden, A., & Nachemson, A. (1992). Long-term follow-up of patients with untreated scoliosis. A study of mortality, causes of death, and symptoms. *Spine (Phila Pa 1976)*, 17(9), 1091-1096.
14. Pehrsson, K., Nachemson, A., Olofson, J., Strom, K., & Larsson, S. (1992). Respiratory failure in scoliosis and other thoracic deformities. A survey of patients with home oxygen or ventilator therapy in Sweden. *Spine (Phila Pa 1976)*, 17(6), 714-718.
15. Ramirez, N., Johnston, C. E., & Browne, R. H. (1997). The prevalence of back pain in children who have idiopathic scoliosis. *J Bone Joint Surg Am*, 79(3), 364-368.
16. Sarwahi, V., Wollowick, A. L., Sugarman, E. P., Horn, J. J., Gambassi, M., & Amaral, T. D. Minimally invasive scoliosis surgery: an innovative technique in patients with adolescent idiopathic scoliosis. *Scoliosis*, 6, 16.

17. Sato, T., Hirano, T., Ito, T., Morita, O., Kikuchi, R., Endo, N., & Tanabe, N. Back pain in adolescents with idiopathic scoliosis: epidemiological study for 43,630 pupils in Niigata City, Japan. *Eur Spine J*, 20(2), 274-279.
18. Schulte, T. L., Hierholzer, E., Boerke, A., Lerner, T., Liljenqvist, U., Bullmann, V., & Hackenberg, L. (2008). Raster stereography versus radiography in the long-term follow-up of idiopathic scoliosis. *J Spinal Disord Tech*, 21(1), 23-28.
19. Scoliosis Research Society (SRS) Retrieved from <http://www.srs.org/>
20. Skaggs, D. L., Akbarnia, B. A., Flynn, J. M., Myung, K. S., Sponseller, P. D., Vitale, M. G., . . . Scoliosis Research Society Growing Spine Study, C. (2014). A classification of growth friendly spine implants. *J Pediatr Orthop*, 34(3), 260-274. doi:10.1097/BPO.0000000000000073
21. Smith, J. T. Bilateral rib-to-pelvis technique for managing early-onset scoliosis. *Clin Orthop Relat Res*, 469(5), 1349-1355.
22. Tis, J. E., Karlin, L. I., Akbarnia, B. A., Blakemore, L. C., Thompson, G. H., McCarthy, R. E., . . . Southern, E. P. Early onset scoliosis: modern treatment and results. *J Pediatr Orthop*, 32(7), 647-657.
23. Ughwanogho, E., Patel, N. M., Baldwin, K. D., Sampson, N. R., & Flynn, J. M. Computed tomography-guided navigation of thoracic pedicle screws for adolescent idiopathic scoliosis results in more accurate placement and less screw removal. *Spine (Phila Pa 1976)*, 37(8), E473-478.
24. Weinstein, S. L., Dolan, L. A., Spratt, K. F., Peterson, K. K., Spoonamore, M. J., & Ponseti, I. V. (2003). Health and function of patients with untreated idiopathic scoliosis: a 50-year natural history study. *Jama*, 289(5), 559-567.
25. Weinstein, S. L., Dolan, L. A., Wright, J. G., & Dobbs, M. B. (2013). Effects of bracing in adolescents with idiopathic scoliosis. *N Engl J Med*, 369(16), 1512-1521. doi:10.1056/NEJMoa1307337
26. Wybier, M., & Bossard, P. Musculoskeletal imaging in progress: The EOS imaging system. *Joint Bone Spine*.
3. In a skeletally immature 12 years old girl with an adolescent idiopathic scoliosis of 28° (major curve), typical curve, and normal neurological examination, the recommended treatment option is:
 - a. surgery
 - b. bracing
 - c. nothing (no need to follow-up)
 - d. observation
 - e. additional workup with MRI first
4. In a skeletally mature 17 years old boy with an adolescent idiopathic scoliosis of 65°, typical curve, and normal neurological examination, the recommended treatment option is:
 - a. surgery
 - b. bracing
 - c. nothing (no need to follow-up)
 - d. observation
 - e. additional workup with MRI first
5. In a 7 years old boy with progressive and severe idiopathic scoliosis (major curve 70°) despite bracing and casting, the most reasonable treatment option would be:
 - a. physiotherapy
 - b. early definitive spinal fusion (posterior approach)
 - c. early definitive spinal fusion (anterior approach)
 - d. early definitive spinal fusion (combined anterior and posterior)
 - e. growth-friendly surgical treatment

ANSWERS:

1d, 2e, 3b, 4a, 5e

QUESTIONS

1. In a growing (skeletally immature) girl with an adolescent idiopathic scoliosis of 13° (major curve), typical curve, and normal neurological examination, the recommended option is:
 - a. surgery
 - b. bracing
 - c. nothing (no need to follow-up)
 - d. observation
 - e. additional workup with MRI
2. In a 7 years boy with an idiopathic scoliosis of 20° (major curve), and normal neurological examination, the recommended treatment option is:
 - a. surgery
 - b. bracing
 - c. nothing (no need to follow-up)
 - d. observation
 - e. additional workup with MRI first



Prof. Dr. med Enric Cáceres

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

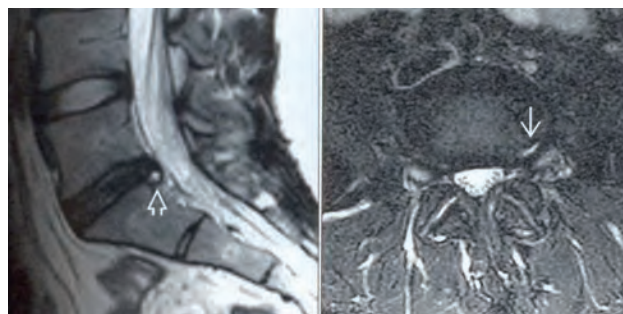
Dr. Maite Ubierna

Spine Unit Hospital Germans Trias i Pujol of Badalona, Barcelona, Spain
maiteubi8587@gmail.com

Degenerative Spine Diseases & Spine Fractures

I Pevalence of Lumbar Degenerative Disease

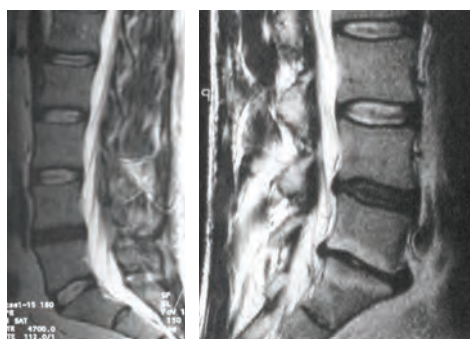
1. 70-80% of all individual will experience low back pain at some time in their lives; usually it resolves in some weeks
2. Low pain is the leading cause of disability in people younger than 50 years of age
3. Many sources of pain are described
 - 3.1 facet joint arthropathy
 - 3.2 discogenic pain or annular tears
 - 3.3 spondylolisthesis
 - 3.4 spinal stenosis



Images of annular tears corresponding with HIZ in L5-S1

II Dark Disc Disease DDD

1. MRI 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
5. Modic changes in MRI describes different situations around DDD



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

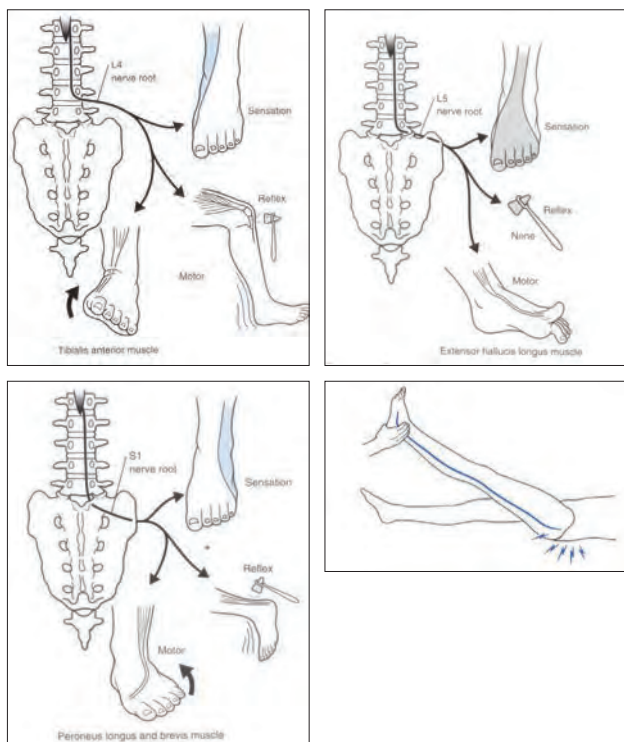
Modic type	T1 signal	T2 signal
Modic I	EDEMA decrease	Increase
ModicII	FAT DEGENERATION increase	Increase
ModicIII	ESCLEROSIS decrease	Decrease

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

Disk tear lesion predictor, contends vascularized granulomatous tissue, highlight with Gadolinium Strong relationship with positive discography...not always with clinical significance

III 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. Caudal segments are affected more commonly L5-S1 discreetly more frequent than L4-L5 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. Clinical features Radiculopathy
 - a. Leg pain > lumbar pain
 - b. Dermatome distribution
 - c. Increase sitting positions and forward bending
 - d. Improve with bed rest
4. Physical exam



5. Diagnostic imaging

5A. RNM

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

5B. Discography + TAC useful tool in recurrence

6. Treatment

6.1 Nonsurgical treatment:

- LDH has a favourable prognosis
- 90% report improvement of symptoms (natural history)

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

6.2 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. The Paraspinal splitting approach (Wiltse approach) is recommendable for extraforaminal disk herniation. Recurrent lumbar disc herniation has been reported in widely varying incidences between 3% and 18% of the patients and depends on the duration of the follow-up.

IV Lumbar Spinal Stenosis

A. Definition.

- Spinal stenosis simply means a decrease in the space available for the neural elements, and, in the lumbar spine,

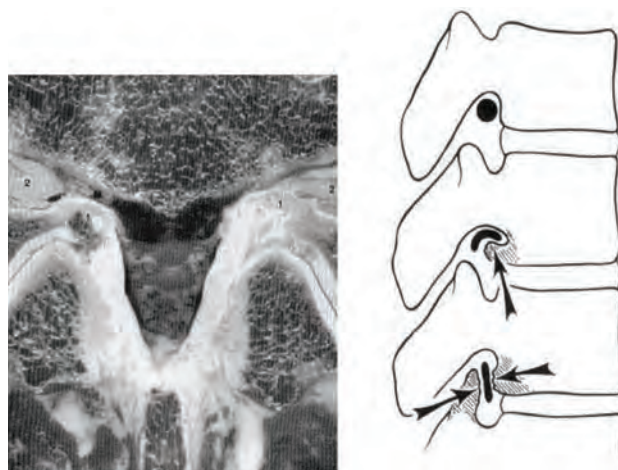
the cauda equina. It can occur at different level: the central canal, the lateral recess or the intervertebral

- 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:

- Upright AP, lateral, and flexion-extension radiographs amount of lumbar degeneration, vertebral deformity and instability.
- EMG may be helpful to distinguish peripheral neuropathy from LSS.
- Myelography: useful when deformity exists.
- CT scan: facet joints hypertrophy, disc vacuum, size of disc height and foramen height.
- MRI is currently the recommended advanced imaging modality to evaluate LSS. Non invasive technique. If there is discordance between clinical and lumbar MRI cervical spine should be reviewed. The association between cervical and lumbar stenosis is common

D. Treatment

D1. Nonsurgical

- Narcotics, NAIDs, anticonvulsants
- LS orthotics
- Physical therapy: flexion-based lumbar stabilization program
- Steroid injections

D2. Surgical treatment

- Indications:
 - Caudal equine syndrome
 - Severe neurologic deficit or impairment
 - Failure to improve leg pain and neurogenic claudication after non surgical treatment.
 - Persistent and severe worsening in patient quality of life.
- 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

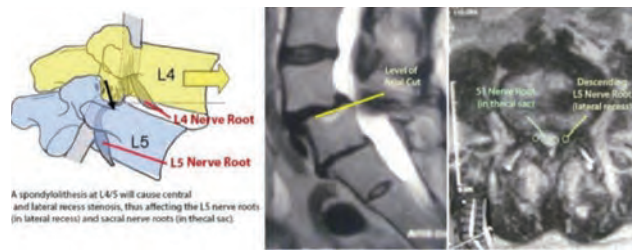
Save blood preoperative techniques

Self-saver postoperative

Laminectomy

Lateral decompression into the lateral recess and into the foramen

Fusion if resection is > 50 % bilateral facets or complete unilateral facetectomy. Incidental dural tear primarily repaired not change the clinical outcome.



○ caused by slippage, hypertrophy of ligamentum flavum, and encroachment into the spinal canal of osteophytes from facet arthrosis

• foraminal stenosis

○ a degenerative slip at L4/5 will affect the L4 nerve root as it is compressed in the foramen

○ vertical foraminal stenosis (loss of height of foramen) caused by

○ loss of disk height

○ osteophytes from posterolateral corner of vertebral body pushing the nerve root up against the inferior surface of the pedicle

○ anteroposterior foraminal stenosis (loss of anterior to posterior area) caused by

○ degenerative changes of the superior articular facet and posterior vertebral body

V. Degenerative Spondylolisthesis

Overview

A condition characterized by lumbar spondylolisthesis without a defect in the pars

absent of pars defect differentiates from adult isthmic spondylolisthesis

- Epidemiology

• prevalence

○ 5% in men and 9% in woman

• demographics

○ more common in African Americans, diabetics, and woman over 40 years of age

○ 8 times more common in woman than men

○ increase in prevalence in women postulated to be due to increased ligamentous laxity related to hormonal changes

• location

○ degenerative spondylolisthesis is 5-fold more common at L4/5 than other levels

○ this is different that isthmic spondylolisthesis which is most commonly seen at L5/S1

- Pathoanatomy

• forward subluxation (intersegmental instability) of vertebral body is allowed by

○ facet joint degeneration

○ facet joint sagittal orientation

○ intervertebral disc degeneration

○ ligamentous laxity (possibly from hormonal changes)

• degenerative cascade involves

○ disc degeneration leads to facet capsule degeneration and instability

○ microinstability which leads to further degeneration and eventual macroinstability and anterolisthesis

○ instability is worsening with sagittally oriented facets (congenital) that allow forward subluxation

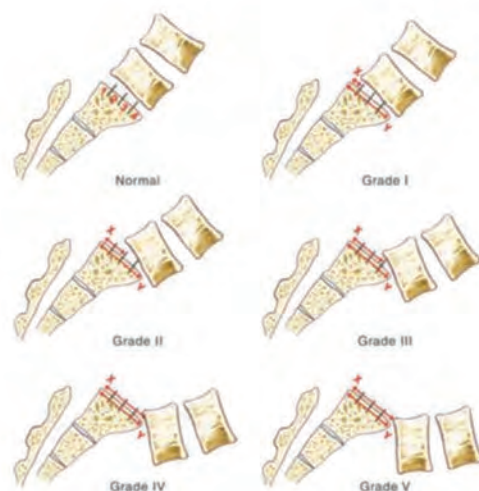
• neurologic symptoms caused by

○ central and lateral recess stenosis

○ a degenerative slip at L4/5 will affect the descending L5 nerve root in the lateral recess

Myerding Classification

Grade I	< 25%
Grade II	25 to 50%
Grade III	50 to 75% (Grade III and greater are rare in degenerative spondylolisthesis)
Grade IV	75 to 100%
Grade V	Spondyloptosis



Classification

Presentation

- Symptoms

• mechanical/ back pain

○ most common presenting symptom

- usually relieved with rest and sitting
- neurogenic claudication & leg pain
 - second most common symptoms
 - defined as buttock and leg pain/discomfort caused by upright walking
 - relieved by sitting
 - not relieved by standing in one place (as is vascular claudication)
 - may be unilateral or bilateral
 - same symptoms found with spinal stenosis
- cauda equina syndrome (very rare)
- Physical exam
 - L4 nerve root involvement (compressed in foramen with L4/5 DS)
 - weakness to quadriceps
 - best seen with sit to stand exam maneuver
 - weakness to ankle dorsiflexion (cross over with L5)
 - best seen with heel-walk exam maneuver
 - decreased patellar reflex
 - L5 nerve root involvement
 - weakness to ankle dorsiflexion (cross over with L4)
 - best seen with heel-walk exam maneuver
 - weakness to EHL (great toe extension)
 - weakness to gluteus medius (hip abduction)
 - provocative walking test
 - have patient walk prolonged distance until onset of buttock and leg pain
 - have patient stop but remain standing upright
 - if pain resolves this is consistent with vascular claudication
 - have patient sit
 - if pain resolves this is consistent with neurogenic claudication (DS)
 - hamstring tightness
 - commonly found in this patients, and must differentiate this from neurogenic leg pain

Imaging

- Radiographs
 - recommended views
 - weight bearing lumbar AP, lateral neutral, lateral flexion, lateral extension
 - findings
 - slip evident on lateral xray
 - flexion-extension studies
 - instability defined as 4 mm of translation or 10° of angulation of motion compared to adjacent motion segment
- MRI
 - indications
 - persistent leg pain that has failed nonoperative modalities
 - best study to evaluate impingement of neural elements
 - views
 - T2 weighted sagittal and axial images best to look for compression of neurologic elements

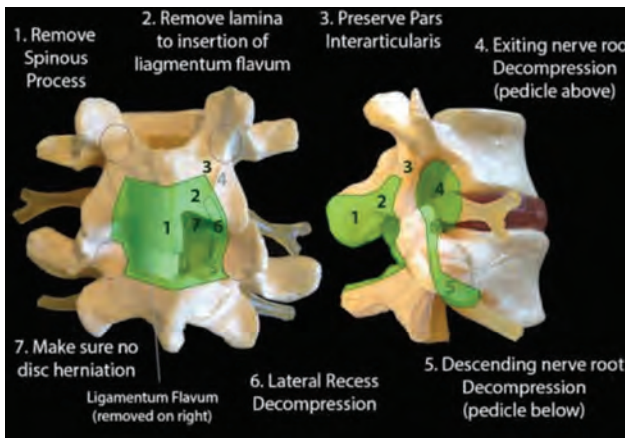


- CT
 - useful to identify bony pathology
- CT myelogram
 - helpful in patients in which a MRI is contraindicated (pacemaker)

Treatment

- Nonoperative
 - physical therapy and NSAIDS
 - indications
 - most patients can be treated nonoperatively
 - modalities include
 - activity restriction
 - NSAIDS
 - PT
 - epidural steroid injections
 - indications
 - second line of treatment if non-invasive methods fail
- Operative
 - lumbar wide decompression with instrumented fusion
 - indications
 - most common is persistent and incapacitating pain that has failed 6 mos. of nonoperative management and epidural steroid injections
 - progressive motor deficit
 - cauda equina syndrome
 - outcomes
 - 79% have satisfactory outcomes
 - improved fusion rates shown with pedicle screws
 - improved outcomes with successful arthrodesis
 - worse outcomes found in smokers
 - posterior lumbar decompression alone
 - indications
 - usually not indicated due to instability associated with spondylolisthesis
 - only indicated in medically frail patients who cannot tolerate the
 - increased surgical time of performing a fusion
 - outcomes
 - 69% treated with decompression alone are satisfied
 - 31% have progressive instability
 - anterior lumbar interbody fusion (ALIF)
 - indications

- reserved for revision cases with pseudoarthrosis
- outcomes
 - injury to superior hypogastric plexus can cause retrograde ejaculation
 - '



Surgical techniques

- Posterior decompression and posterolateral fusion (+/- instrumentation)
 - approach
 - posterior midline approach
 - multiple parasagittal incisions for minimally invasive approaches
 - decompression
 - usually done with laminectomy, wide decompression, and foraminotomy
 - fusion
 - posterolateral fusion with instrumentation most common
 - TLIF/PLIF growing in popularity and may increase fusion rates
 - and decrease risk of postoperative slip progression
 - reduction of listhesis
 - limited role in adults

Complications

- Pseudoarthrosis (5-30%)
 - CT scan is more reliable than MRI for identifying failed arthrodesis
- Adjacent segment disease (2-3%)
 - incidence is approximately 2.5% a year
- Surgical site infection (0.1-2%)
 - treat with irrigation and debridement (usually hardware can be retained)
- Dural tear
- Positioning neuropathy
 - LFCN
 - seen with prone positioning due to iliac bolster
 - ulnar nerve or brachial plexopathy
 - from prone positioning with inappropriate position
- Complication rates increase with
 - older age

- increased intraoperative blood loss
- longer operative time
- number of levels fused

REFERENCES

Degenerative Disk Disease

1. Lindstrom I, Ohlund C, Eek C, Wallin L, Peterson LE, Fordyce WE (1992) The effect of graded activity on patients with subacute low back pain: a randomized prospective clinical study with an operant-conditioning behavioural approach. *Physical Therapy* 72: 279-293
2. Frost H, Klaber Moffett JA, Bergman JA, Spengler D (1995) Randomised controlled trial for evaluation of fitness programme for patients with chronic low back pain. *Br Med J* 310:152-154
3. Van Tulder M, Koes B, Malmivaara A (2006) Outcome of non-invasive treatment modalities on back pain: an evidence-based review. *Eur Spine J* 15:S64-S81
4. Abenham L, Rossignol M, Valat JP, Nordin M, Avouac B, Blotman F, Charlot J, Dreiser RL, Legrand E, Rozenberg S, Vautravers P (2000) The role of activity in the therapeutic management of back pain. Report of the International Paris Task Force on Back Pain. *Spine* 25:1S-3S

Lumbar Disk Herniation

1. Williams RW (1978) Microlumbar discectomy: a conservative surgical approach to the virgin herniated lumbar disc. *Spine* 3:175-82
2. Atlas SJ, Keller RB, Wu YA, Deyo RA, Singer DE (2005) Long-term outcomes of surgical and non-surgical management of sciatica secondary to a lumbar disc herniation: 10 year results from the Maine Lumbar Spine Study. *Spine* 30:927-935
3. Balague F, Nordin M, Sheikhsadeh A, Echegoyen AC, Brisby H, Hooge- woud HM, Fred- man P (1999)
4. Weber H (1983) Lumbar disc herniation. A controlled, prospective study with ten years of observation. *Spine* 8:131-140
5. Weinstein JN, Lurie JD, Tosteson TD, et al. (2006) Surgical vs nonopera- tive treatment for lumbar disk herniation. The Spine Patient Outcomes Research Trial (SPORT), a randomized trial. *JAMA* 296:2441-2450
6. Weinstein JN, Lurie JD, Tosteson TD, et al. (2006) Surgical vs nonoperative treatment for lumbar disk herniation. The Spine Patient Outcomes Research Trial (SPORT) observational cohort. *JAMA* 296:2451-2459

Spinal Stenosis

1. Amundsen T, Weber H, Nordal HJ, Magnaes B, Abdelnoor M, Lilleas F (2000) Lumbar spinal stenosis: conservative or surgical management? A prospective 10-year study. *Spine* 25(11):1424-35
2. Grob D, Humke T, Dvorak J (1995) Degenerative lumbar spinal stenosis. Decompression with and without arthrodesis. *J Bone Joint Surg Am* 77:1036-41
3. Herkowitz HN, Kurz LT (1991) Degenerative lumbar spondylolisthesis with spinal stenosis. A prospective study comparing decompression with decompression and inter-transverse process arthrodesis. *J Bone Joint Surg Am* 73:802-8

Degenerative Spondylolisthesis

1. Mark D. Miller and Stephen R Thompson Elsevier Miller's Review of Orthopaedics ISBN 978-0-323-35517-9 Philadelphia 2016
2. An. Howard S. Synopsis of Spine Surgery Williams & Wilkins ISBN 0-683-18097 Pennsylvania 1998
3. Vincent J Devlin Spine Secrets Hanley & Belfus, Inc ISBN 1-56053-358-7 Philadelphia 2003
4. Orthobullets.com. Orthopaedic Review <http://www.orthobullets.com/Lineage Medical Cambridge, MA 02142>.

QUESTIONS

1. What is the risk of recurrent herniation's at the same level in surgically treated patients at 5-year follow-up?
2. What is the most common location for lumbar disk herniation?
3. How are extraforaminal disk herniations ideally approached?
4. A 65-year-old man has low back pain and leg pain with standing. Walking endurance is limited to two blocks due to leg cramping. He has a wide-based, unsteady gait and hyperflexia. Lumbar radiographs reveal a degenerative spondylolisthesis at L4-L5, and an MRI scan shows moderate spinal stenosis at this level. The next step in his care should include...
 - a. lumbar epidural steroid injections.
 - b. lumbar decompression with fusion.
 - c. a lumbar epidurogram.
 - d. interspinous distraction.
 - e. cervical MRI.
5. An incidental dural tear was primarily repaired with a watertight closure during an otherwise uncomplicated laminectomy. After surgery, the patient should be informed that...
 - a. the chance of resolution of the preoperative symptoms will be decreased.
 - b. there is a greater risk of a wound infection.
 - c. the clinical outcome will be unaffected.
 - d. strict bed rest for 2 weeks is recommended.
 - e. a compression dressing must be maintained for 7 days.

ANSWERS:

1. 1. 3-18%
2. Caudal segments are affected more commonly, L5-S1 discretely more frequent than L4-L5. Less frequent in thoracic and high lumbar level
3. Wiltse approach
4. a
5. c

Maite Ubierna

Head Spine Unit Hospital Germans Trias I Pujol
Barcelona, Barcelona, Spain
Maiteubierna8587@gmail.com

Spine Trauma

1. SPINAL CORD INJURY

a. Background:

1. The annual incidence of SCI is approximately 40 cases per 1 million people in the United States, or 11,000 new cases per year.
2. 55% of SCIs occur in the cervical spine. The remaining injuries are equally distributed through-out the thoracic, thoracolumbar, and lumbosacral spine.
3. Motor vehicle accidents account for half of reported SCIs. Fall and recreational sport injuries are responsible for most of the remaining SCIs.
4. 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. 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) (table 1) standards of neurological testing provides a concise and detailed method for evaluating spinal cord and peripheral nerve root function. (table 1)

Table 1. ASIA Impairment Scale

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 sensor/motor deficit, minor functional impairments of reflex-muscle tone changes

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. 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 reformat images is useful to further define bony anatomy of the lesion.
2. Magnetic Resonance imaging (MRI) is used in all cases of neurological compromise or to better visualize soft tissue anatomy, that is, neural compressive lesions such as disk herniation, 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 patient 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 < 90 mm Hg) should be avoided and 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 heparin, a rotating bed, and pneumatic compression stockings or combination therapy are recommended.
3. 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.
4. Methylprednisolone was indicated to improve the motor scores in post-traumatic SCI when patients were delivered within 8 hours of injury (NASCIS III).
5. Less than 3 hours after injury, a 30 mg/Kg bolus of methylprednisolone is administered, followed by 5.4 mg/Kg/h for 23 hours.
6. 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.

f. 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 capillary perfusion by removing external pressure from the spinal cord and establishing spinal stability.
3. There is substantial class 2 and 3 evidence (non prospective, nonrandomized and uncontrolled) that surgical decompression provides better outcomes than late or nonsurgical therapies.

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.

b. Instability of the cervical spine:

1. One of the problems has been the absence of a clear definition based in reliable radiological criteria. Therefore White and Panjabi Table 2 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.

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

>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 ligament

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. Radiography remains the imaging modality of first choice. The lateral view should extend from occiput to T1. Do not miss injuries at the cervicocranial and the cervicotoracic junctions.
3. 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.
4. MRI is additional to CT for specific diagnostic assessments.
5. Magnetic resonance is the imaging study of choice to exclude

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,

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.
- 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 , typically stable fracture, should be treated with a cervical orthotics 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 10°.
- These type 2 fractures should be considered for early C1-C2 fusion in elderly patients.
- In young people type 2 fractures no displaced could be treated with halo vest immobilization. Fractures in which reduction cannot be achieved or maintained surgical treatment should be considered. Anterior odontoid screw placement is an option for minimally comminuted 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 bilateral C2 pars interarticularis screws.

f. Subaxial Cervical 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.

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.

1. Compression-flexion:

- failure of anterior column compression and posterior column distraction.
- are caused by axial loading in flexion with failure of the anterior half of the body without disruption of the posterior body cortex and minimal risk of neurologic injury.
- Treatment: stable undisplaced compression-flexion fractures can be treated conservatively with external immobilization for 6 to 12 weeks with a rigid collar. Kyphosis deformity > 15° should 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 plating. 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 displacement 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 result 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 through 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 unstable. Major injuries include compression fractures, burst fractures, flexion-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 of 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 neurologic 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 cast, should be obtained before discharge.
5. Significant increases in the fracture angle ($>10^\circ$) 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.
 - 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.
 - 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

References:

Book chapters

1. Heinzelmann M, Wanner GA. Thoracolumbar Spinal Injuries. In: Boss N, Aebi M (2008) *Spinal Disorders*. Springer – Verlag. Berlin Heidelberg.
2. White AA, 3rd, Panjabi MM: *Practical biomechanics of spine trauma*. In: White AA, 3rd, Panjabi MM (eds) *Clinical Biomechanics of the spine*. JB Lippincott, Philadelphia 1990, pp169–275

Journals:

1. Allen BL, Ferguson RL, Lehmen TR, O'Brien RP: A mechanistic classification of closed, indirect fractures and dislocation of the lower cervical spine. *Spine* 1982; 7:1–27.
2. Anderson PA, D'Alonzo RT. Fractures of the odontoid process of the axis. *J Bone J Surg* 1974; 56A(8):1663–1674
3. Levine AM, Edwards CC. Fractures of the atlas. *J Bone J Surg* 1991; 73A:680–691.
4. Denis F. The three column spine and its significance in the classification of acute thoracolumbar spine injuries. *Spine* 1983; 8:817–31.
5. Effendi B, Roy D, Cornish B et al. Fractures of the ring of the axis – A classification based on the analysis of 131 cases. *J Bone J Surg* 1981; 63B:3
6. Harris MB, Kronlage SC, Carboni PA, et al.: Evaluation of the cervical spine in the polytrauma patient. *Spine* 2000; 25:2884–2891.
7. Magerl F, Aebi M, Gertzbein SD et al. A comprehensive classification of thoracic and lumbar injuries. *Eur Spine J* 1994; 3:184–201.
8. Vaccaro AR, Lehman RA, Hulbert RJ, et al. A new classification of thoracolumbar injuries: the importance of injury morphology, the integrity of the posterior ligamentous complex and neurologic status. *Spine* 2005; 18:209–15.
9. Wood K, Buttermann G, Mehbod A, Garvey T, Jhanjee R, Sechriest V: Operative compared with nonoperative treatment of a thoracolumbar burst fracture without neurological deficit. *J Bone Joint Surg Am* 2003; 85-A:773–781.

c. BSS, ACS, CCS

d. BSS, CCS, ACS

e. ACS, CCS, BSS

3. Which is the most common site of spinal fractures?

4. Which mark be fitting with a incomplete sensory deficit and complete motor deficit in ASIA Impairment Scale

5. Which classification of thoracolumbar fractures MRI is used to identify PCL injury Posterior Ligament Complex?

ANSWERS:

1. b

2. d

3. Thoracolumbar spine,

4. ASIA B

5. TLICS Thoracolumbar injury classification and severity score

QUESTIONS

1. A 44-year-old farmer involved in a rollover accident on his tractor sustained a L1 burst fracture with 20% loss of vertebral body height, 30% canal compromise, and 15° of kyphosis. He remains neurologically intact. The preferred initial course of action should consist of
 - a. posterior spinal fusion with instrumentation
 - b. a thoracolumbarsacral orthosis (TLSO) extension brace and early mobilization.
 - c. Bed rest for 6 weeks followed by immobilization in a cast
 - d. Anterior L1 corpectomy and fusion with instrumentation
 - e. Anterior corpectomy followed by posterior fusion with instrumentation
2. What is the prognosis for ambulation, from best to worst, for patients with an incomplete spinal cord injury? CCS = Central Cord Syndrome. ACS Anterior Cord Syndrome, BSS = Brown-Sequard Syndrome
 - a. CCS, ACS, BSS
 - b. CCS, BSS, ACS.



Prof. Dr. med. Ulrich Stöckle

BG Klinik Tübingen
Tübingen, Germany
ustoeckle@bgu-tuebingen.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 lumbar nerve plexus are running in front of the sacrum. Major bleeding can be caused by disruptions of the posterior pelvic ring, caused mainly by bleeding out of the sacral venous plexus and fracture sites. In only 20% arterial bleeding is the reason for hemodynamic instability.

1.2. Diagnostics

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

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

1.3. Classification

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

C-type injuries

A-type injuries, stable lesions:

- A1: iliac wing fractures, apophyseal fractures
- A2: anterior pelvic ring fractures
- A3: transverse sacral fractures

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

- B1: open book injuries
- B2: lateral compression type injuries
- B3: bilateral B-type injuries

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

- C1: unilateral vertical instability
- C2: one side vertical instability, other side B-type injury with horizontal instability
- C3: bilateral vertically unstable lesion

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

1.4. Treatment

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

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

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

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

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

The alternative method for external stabilisation of the pelvic ring is

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

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

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

1.5. Results

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

2. Acetabular fractures

2.1. Anatomy

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

2.2. Diagnostics

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

2.3. Classification

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

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

2.4. Treatment

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

2.5. Results

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

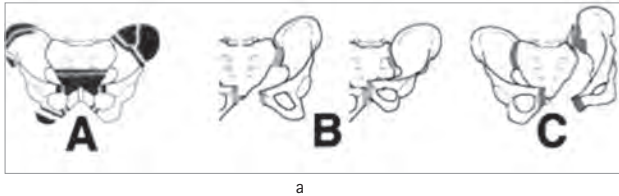
References

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

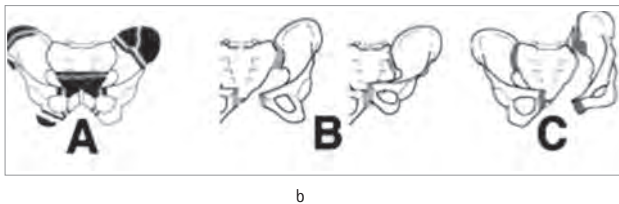
9. H Tscherne, T Pohlemann, Unfallchirurgie 1998. Buch. XVI, 498 S.: 800 s/w-Abbildungen, 30 s/w-Tabellen. Hardcover Springer Berlin ISBN 978-3-540-62481-3

Figures

AO- Classification



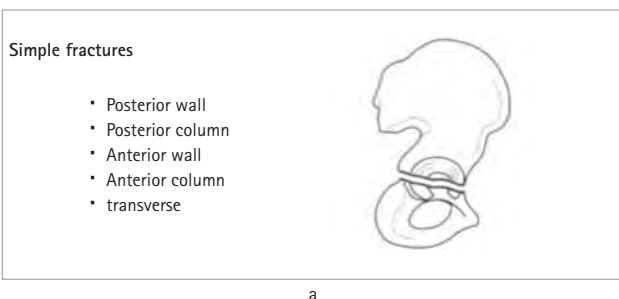
- A** Anterior and Posterior Arch Intact
Stable Lesions
- B** Anterior Complete,
Posterior Incomplete Disruption
Rotational Instability
- C** Anterior and Posterior Complete Disruption
Rotational and Vertical Instability



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

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

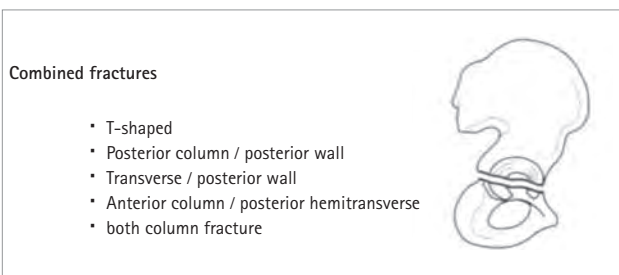
Letournel Classification



Simple fractures

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

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.

QUESTIONS

- Pelvic B-type injuries are
 - stable
 - horizontally unstable
 - vertically unstable
 - horizontally and vertically unstable
- Bleeding in pelvic ring injuries
 - is neglectable
 - is mainly arterial bleeding
 - is mainly venous bleeding and out of the fracture sites
 - needs always surgical bleeding control
- The key question in the treatment of pelvic ring injuries is
 - the displacement in the anterior pelvic ring
 - the stability of the posterior pelvic ring
 - the fracture of the ischial bone
 - the iliac view X-ray
- Acetabular fractures
 - need always operative fixation
 - should be operated on an emergency base
 - need anatomic reconstruction
 - are suffered by patients in an average age of 30 years
- The ilioinguinal approach
 - is a posterior approach for acetabular fractures
 - provides good insight into the joint
 - can be used for most both column fractures
 - is mainly used for posterior wall fractures

ANSWERS:

1b, 2c, 3b, 4c, 5c.



Dr. med Cecilia Rogmark

Skåne University Hospital, Malmö, Sweden

Cecilia.Rogmark@skane.se

Fractures: Femur, Tibia and Open Fractures

General

Fractures in the lower extremities occur in all ages. Osteopenia or osteoporosis increases the risk of fracture even after low-energy trauma, and both hip fracture, distal femoral and proximal tibial fractures are considered fragility fractures. The aim of the treatment should be unrestricted mobilization as soon as possible. This is particularly important in elderly or otherwise frail patients, who cannot cope with restricted weight bearing. Therefore fixation should be stable, if possible. The use of hip replacement instead of internal fixation in hip fracture is a good example of immediate stability resulting in better functional outcome.

Always consider the risk of deep vein thrombosis and/or pulmonary embolism, and follow the local guidelines for prophylaxis. Usually antibiotics are given perioperatively to prevent infection, at least in hip fracture cases and other fractures treated with implants. What type to choose depends on the mix of pathogens within the region, resistance pattern, any allergy etc. Generally, antibiotics should be given immediately (<30-60 min) before surgery and should not exceed the 24-hour post-operative period. A single dose is preferred, but if surgery is prolonged or major bleeding occurs, a repeated dose should be given.(1)

Hip fractures

General

Hip fractures are confirmed via conventional x-ray. If ambiguity, MRI will be useful (2).

Surgery should be performed within 24 hours in order to decrease complications and mortality (3).

Care should be given in close cooperation with geriatricians (4).

Postoperative rehabilitation, during several months, is necessary in order to regain function. Multidisciplinary teams may be useful when providing rehabilitation (5).

See NICE Guidelines Hip Fractures - <https://www.nice.org.uk/guidance/cg124>

Femoral neck fracture

Undisplaced femoral neck fracture

These are usually treated with internal fixation and fixed in situ (6). That is, a valgus impaction is accepted. Non-surgical treatment

is not recommended due to the risk of secondary displacement and therefore the need of frequent check-ups. Nevertheless, in case of contraindications for surgery it may be an alternative.

Displaced femoral neck fracture

Treatment options are internal fixation (after closed or open reduction), total hip arthroplasty (THA) or hemiarthroplasty (7). Choice is guided by patient's biological age, activity level, health and supposed remaining life span. For a majority, those with functional and/or psychological limitations, hemiarthroplasty is the most suitable choice. The benefits are "forgiving" surgery (can be performed by surgeons with less experience, i.e. emergency procedure), shorter surgery time, less bleeding and lower risk of dislocation. In active individuals on the other hand, hemiarthroplasty carries an unacceptable high risk of acetabulum erosion (8). In those cases, THA is a better option. THA in fracture cases puts higher demands on the surgeon, as malpositioned implant parts increase the risk of dislocation and pain. The surgery time is longer, and altogether THA procedures may be demanding to cope with within an emergency setting. Arthroplasty in fracture patients should be cemented (less risk of periprosthetic fracture) and inserted via direct lateral approach (less risk of dislocation) (9-13). Increased risk of perioperative death in connection with the cementation process can be limited by proper anesthesiologic care and cementing technique (thorough lavage, no excessive pressure etc)(14). In addition, uncemented stems lead to a higher risk of death after the first postoperative week (15, 16). This may be due to their higher risk of periprosthetic fracture.

Internal fixation may be suitable for younger individuals, even though they have the same high risk (30-50%) of pseudarthrosis and avascular necrosis as elderly patients have. But, if healthy, the young patients can better withstand a secondary procedure if needed. And if they heal, they maintain their native hip joint. The age limit for primary prosthesis is controversial. 60, 65 or 70 years are suggested. The important thing is to understand the concept of biological age. For example a 55 year old with pre-senile dementia and functional limitation may benefit from hemiarthroplasty. Regarding reduction of the fracture, there is no clear evidence that open reduction add any benefit compared to closed ditto, but studies are very scarce.

Extracapsular hip fractures

Basicervical fractures are not well studied. Mostly sliding hip screw (SHS), with or without an extra anti-rotational screw, is recommended.

Stable, or two-fragment trochanteric fractures, should be fixed with SHS (17). Using intramedullary nail (IM nail) in these cases brings on unnecessary complications, mainly peri-implant fractures.

In unstable trochanteric fractures, both SHS and IM nails can be used (18). To understand the "personality" of the fracture and to obtain a good fracture reduction and implant position, is more important than implant choice (19). A sliding hip screw with biaxial sliding function is a proven alternative, whereas the trochanteric supporting plate lacks scientific support. Locking plates have no role in proximal femoral fractures (20).

Subtrochanteric fractures can be treated with IM nails or sliding hip screw with biaxial sliding function. Fixed angle plates are not an option nowadays (18).

Femoral shaft fractures

General

Femoral shaft fractures occurs mostly due to high energy trauma. An important subgroup, as well, is periprosthetic fractures in the presence of hip and/or knee replacement. When an earlier implant inserted in the femur, preoperative considerations are even more important. Any loosening of a joint prosthesis must be evaluated, and a loose implant should be revised. Use – and understand – the Vancouver classification and always cooperate with an arthroplasty surgeon!

Femoral fractures can lead to great amount of blood loss. In high energy trauma, muscular injury and subsequent necrosis can lead to renal insufficiency.

Treatment

ER room – temporary stabilization with traction or long posterior splint. In case of severe instability – both generally and/or locally – an immediate external fixation will serve as damage control. This leaves time for other life-saving interventions, and thereafter final internal fixation can take place.

Check vital parameters continuously as blood loss can be substantial, oxygen is often needed. Pain treatment, in which femoral blocks may be an option. Monitoring regarding compartment syndrome is important in high energy-trauma cases.

Surgical treatment with intramedullary nailing is the most common method for diaphyseal fractures, and plating (with locking screws) for metaphyseal fractures, as well as periprosthetic fractures.

Tibial shaft fractures

General

Common fracture localization, open fracture quite common due to thin layer of soft tissue. Higher impact equals higher risk of complications – anamnesis important.

Compartment syndrome – devastating if not diagnosed and treated!

Treatment

ER room – temporary stabilization with long posterior splint, pain treatment and monitoring regarding compartment syndrome.

Nonoperative treatment suitable for closed fractures with minimal displacement or stable reduction; in a long leg cast.

Encourage ambulation with weight-bearing as tolerated in simple transverse fractures but check with frequent radiographs fracture alignment. Long leg cast may lead to increased joint stiffness and a change to functional bracing or patella tendon bearing lower leg cast at appr. 6 wks is recommended. Compared with surgical fixation, the risk with nonoperative treatment is noted increased nonunion and healing time, in particular in displaced fractures. On the other hand, the risk of surgically related infections is absent.

Surgical treatment is nowadays used in most unstable fractures. Methods used are intramedullary nailing, plating and external fixation. In extremely severe cases (see open fractures) even amputation might be necessary. IM nailing with locking screws is the most frequent option, as it leads to fewer non-unions and mal-unions, compared to other methods. It also admits immediate weight-bearing in most cases, leading to a return to activity sooner. The major question is whether tibia should be reamed prior to insertion or not. The evidence is not clear, but suggests some advantage for reaming in closed fractures.

Open fractures

Open fractures of the long bones are increasing, now estimated to occur in 30 per 100,000 persons annually.

Leads to contamination with pathogens, damaged blood perfusion, decreased immune defense. Risk of infection (six-folded compared to internal fixation of closed fractures) and healing disturbances.

Treatment should aim at avoiding infection, improve healing and restore function.

Classification

Gustilo-Anderson

- i. Wound <1 cm and minimal/clean soft tissue injury and minimal fracture comminution
 - ii. Wound >1 cm and moderate soft tissue injury/contamination and moderate fracture comminution
 - iii. Extensive soft tissue damage including muscle, skin, neurovascular structures or Open segmental fracture, irrespective of the wound size or Open fractures over 8 hours old
- a. Adequate soft tissue coverage, including segmental/severely comminuted fractures
 - b. Extensive loss of soft tissue, periosteal stripping, bony exposure
 - c. As B including major arterial injury requiring repair for limb salvage

Treatment

Antibiotics intravenously preferably within 1 hour postinjury (can be given in pre-hospital setting) – observe local guidelines for type of antibiotic drug. Initial pathogens may be less of a problem than secondary nosocomial infections. Protect the wound! Antibiotics given for one day only might be as effective as courses of three to five days, one rule-of-thumb is to give antibiotics for 24–48 hours after the last debridement.

Consider local antibiotics (bead pouches, sponges etc.) after debridement. See Cochrane review 2009; Antibiotics for preventing infection in open limb fractures, and NICE guidelines complex fractures 2016 (22).

Tetanus prophylaxis should be considered

Immediate simple reposition and temporary fixation; wound covered in sterile dressings

Do not irrigate open fractures of the long bones, hindfoot or midfoot in the emergency department before debridement. Initial bacterial culture probably of no use.

Further treatment

Immediate treatment of highly contaminated open fractures – otherwise time frames as below

Gustilo III – surgery within 12 hrs; exposure, debridement, low pressure lavage, temporary or final fixation, coverage (primary suture/NWPT/sterile dressing/graft). Assistance by vascular and plastic surgeon might be needed. Soft tissue grafting can be undertaken as a second procedure, preferably within 72 hrs.

Gustilo II – surgery within 24 hrs; treatment as Gustilo III

Gustilo I – as similar closed fracture

The historical "6-hour rule" – study on guinea pigs by Friedrich in 1898 – has been questioned in recent papers (21). Debridement can supposedly be performed within the first 12–24 hours without any increased risk of infection. This information is useful when transfer may be needed before surgery.

Amputation – may be primary treatment in certain cases, such as "a limb is the source of uncontrollable life-threatening bleeding, or a limb is salvageable but attempted preservation would pose an unacceptable risk to the person's life, or a limb is deemed unsalvageable after orthoplastic assessment" (NICE guidelines (22)).

References:

1. Antibiotic prophylaxis in surgery. SIGN publication no.104. Edinburgh2008 [updated 2014]. Available from: <http://www.sign.ac.uk>.
2. Lubovsky O, Liebergall M, Mattan Y, Weil Y, Mosheiff R. Early diagnosis of occult hip fractures MRI versus CT scan. *Injury*. 2005;36(6):788–92.
3. Simunovic N, Devereaux PJ, Sprague S, Guyatt GH, Schemitsch E, Debeer J, et al. Effect of early surgery after hip fracture on mortality and complications: systematic review and meta-analysis. *CMAJ*. 182(15):1609–16.
4. Grigoryan KV, Javedan H, Rudolph JL. Orthogeriatric care models and outcomes in hip fracture patients: a systematic review and meta-analysis. *J Orthop Trauma*. 2014;28(3):e49–55.
5. Donohue K, Hoevenaars R, McEachern J, Zeman E, Mehta S. Home-Based Multidisciplinary Rehabilitation following Hip Fracture Surgery: What Is the Evidence? *Rehabilitation research and practice*. 2013;2013:875968.
6. Handoll HH, Parker MJ. Conservative versus operative treatment for hip fractures in adults. *Cochrane Database Syst Rev*. 2008(3):CD000337.
7. Parker MJ, Gurusamy KS, Azegami S. Arthroplasties (with and without bone cement) for proximal femoral fractures in adults. *Cochrane Database Syst Rev*. 2010(6):CD001706.
8. Baker RP, Squires B, Gargan MF, Bannister GC. Total hip arthroplasty and hemiarthroplasty in mobile, independent patients with a displaced intracapsular fracture of the femoral neck. A randomized, controlled trial. *J Bone Joint Surg Am*. 2006;88(12):2583–9.
9. Enocson A, Hedbeck CJ, Tidermark J, Pettersson H, Ponzer S, Lapidus LJ. Dislocation of total hip replacement in patients with fractures of the femoral neck. *Acta orthopaedica*. 2009;80(2):184–9.
10. Enocson A, Tidermark J, Tornkvist H, Lapidus LJ. Dislocation of hemiarthroplasty after femoral neck fracture: better outcome after the anterolateral approach in a prospective cohort study on 739 consecutive hips. *Acta orthopaedica*. 2008;79(2):211–7.
11. Leonardsson O, Garellick G, Karrholm J, Akesson K, Rogmark C. Increased risk of re-operation for bipolar and uncemented hemiarthroplasty. 23,509 procedures after femoral neck fractures from the Swedish Hip Arthroplasty Register 2005–2010. *Acta orthopaedica*. 2012;Accepted April 2012.
12. Rogmark C, Fenstad AM, Leonardsson O, Engesaeter LB, Karrholm J, Furnes O, et al. Posterior approach uncemented stems increases the risk of reoperation after hemiarthroplasties in elderly hip fracture patients. An analysis of 33,205 procedures in the Norwegian and Swedish national registries. *Acta orthopaedica*. 2014;In press.
13. Inngul C, Blomfeldt R, Ponzer S, Enocson A. Cemented versus uncemented arthroplasty in patients with a displaced fracture of the femoral neck: a randomised controlled trial. *Bone Joint J*. 2015;97-B(11):1475–80.
14. Membership of the Working P, Griffiths R, White SM, Moppett IK, Parker MJ, Chesser TJ, et al. Safety guideline: reducing the risk from cemented hemiarthroplasty for hip fracture 2015: Association of Anaesthetists of Great Britain and Ireland British Orthopaedic Association British Geriatric Society. *Anaesthesia*. 2015;70(5):623–6.
15. Costa ML, Griffin XL, Pendleton N, Pearson M, Parsons N. Does cementing the femoral component increase the risk of peri-operative mortality for patients having replacement surgery for a fracture of the neck of femur? Data from the National Hip Fracture Database. *J Bone Joint Surg Br*. 93(10):1405–10.
16. Costain DJ, Whitehouse SL, Pratt NL, Graves SE, Ryan P, Crawford RW. Perioperative mortality after hemiarthroplasty related to fixation method. *Acta orthopaedica*. 82(3):275–81.
17. Matre K, Havelin LI, Gjertsen JE, Espehaug B, Fevang JM. Intramedullary nails result in more reoperations than sliding hip screws in two-part intertrochanteric fractures. *Clinical orthopaedics and related research*. 2013;471(4):1379–86.
18. Parker MJ, Handoll HH. Gamma and other cephalocondylic intramedullary nails versus extramedullary implants for extracapsular hip fractures in adults. *Cochrane Database Syst Rev*. 2008(3):CD000093.
19. De Bruijn K, den Hartog D, Tuinebreijer W, Roukema G. Reliability of predictors for screw cutout in intertrochanteric hip fractures. *J Bone Joint Surg Am*. 2012;94(14):1266–72.
20. Streubel PN, Moustoukas MJ, Obremskey WT. Mechanical failure after locking plate fixation of unstable intertrochanteric femur fractures. *J Orthop Trauma*. 2013;27(1):22–8.

21. Schenker ML, Yannascoli S, Baldwin KD, Ahn J, Mehta S. Does timing to operative debridement affect infectious complications in open long-bone fractures? A systematic review. *J Bone Joint Surg Am.* 2012;94(12):1057-64.
22. Fractures (complex): assessment and management NICE guideline [NG37] 2016 <https://www.nice.org.uk/guidance/ng37/chapter/recommendations>

QUESTIONS:

1. What is correct about undisplaced femoral neck fracture (one alternative)
 - a. The fracture should always be reduced to an anatomical position
 - b. Internal fixation is the most common treatment
 - c. Non-surgical treatment is regarded equally good as surgical treatment in a majority of patients
2. Which type of hip complication is more common after uncemented femoral stems in fracture related hip arthroplasty, compared to cemented stems?
3. What is correct about antibiotics in emergency treatment of open fractures (one alternative)
 - a. 1. Antibiotics should be given intravenously within 1 hour from injury
 - b. Antibiotics should not be given until bacterial cultures shows the type of pathogen
 - c. Antibiotics should only be given if dirt is seen in the wound
4. Why is it important to have knowledge upon the timeframes within open fractures can be safely operated on?
5. Mention one benefit and one risk with non-surgical treatment of tibia shaft fractures, compared to surgical treatment:

ANSWERS:

1. b
2. Periprosthetic fracture
3. a
4. To better organize the care chain, to have the possibility to transfer a patient to another hospital with proper competence (i.e. vascular surgeons, plastic surgeons, angiography facilities etc)
5. Benefits: absence of surgical complications such as infection, no need for subsequent hardware removal, may be less expensive
Risks: increased risk of nonunion and longer healing time



Dr. Jordi Teixidor Serra
jteixidorsera@gmail.com

Dr. Jordi Tomás Hernández
jotomas@vhebron.net

Vall d'Hebron University Hospital,
Barcelona, Spain

Fractures: Pilon, Ankle, Talus and Calcaneus

1. ANKLE FRACTURES:

1.1. Epidemiology:

- Most common injuries requiring 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 (antero-inferior 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 talus in all views.
- AP view: Tibio-fibular overlap around 10mm; talar tilt 83° +/- 4°.
- 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 Maissonneuve 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 impaction.
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 symptoms 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 4.0 mm 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.
 - 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 Maissonneuve 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 exercises 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.
- Posttraumatic 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 comminution and few articular fragments. Soft tissue less affected.
- Axial compression: High energy trauma with extensive bony and joint comminution. 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 exercises.

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:

- 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.
- Restore articular congruity by reduction of impacted articular fragments working superior to the impacted area and bringing the fragments down to the joint level.
- Bone graft or bone graft substitute to support the articular surface.
- Temporary K-wires to maintain the reduction of the fragments
- 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.
- Posttraumatic 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 talo-navicular 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

- Need small and mini-fragment implants (titanium) along with biofix pins
- Don't be afraid to use a plate especially when dealing with neck comminution.
- Be sure that you have obtained the correct length and rotation before applying definitive fixation.
- 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:

- Union rates about 94%
- 100% of type III and IV develop arthritis
- Neck fracture AVN rates: Highest risk is comminution and open injury
 - Type II: 40%
 - Type III: 40-65%
- No correlation between time of injury and time to surgery for closed injuries as far as arthritis, AVN, nonunion or AOFAS scores.
- 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:

- Essex-Lopresti
- AO (81.2) A: extrarticular. B: intrarticular. C: fracture-dislocation
- 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 addressed acutely
 - 1. Skin at risk
 - Tongue type
 - 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 severe comminuted fractures (Sanders type 4)
 - Better results after previous ORIF.

4.6. Results and complications:

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

Bibliography:

1. *Master Techniques in Orthopaedic Surgery, Fractures*, DA. Wiss (Editor), 2nd Ed, Lippincott Williams & Wilkins, Philadelphia, 2006.
2. *Surgical Treatment of Orthopaedic Trauma*. James P. Stannard, Andrew H. Schmidt, Philip J. Kregor. Thieme, New York/Stuttgart, 2007.
3. *Emergency Orthopedics: The Extremities*. Robert R Simon, Scott C Sherman, Steven J Koenigsnecht, 5th Ed, McGrawHill, 2007.
4. *AO Principles of Fracture Management*, T Ruedi, R Buckley and C Moran, AO Publishing, Thieme, Stuttgart, 2007.

QUESTIONS:

1. A 28-year-old man in a motor vehicle accident sustains a hyperdorsiflexion injury to his foot. A radiograph reveals a Hawkins type II talar neck fracture with dislocation of the subtalar joint. Open reduction with internal fixation is performed, and the patient returns for follow-up radiographs 8 weeks later. A radiograph shows well-placed hardware and a radiolucent band in the dome of the talus (Hawkins sign). Based on these findings, which of the following is LEAST likely to occur in this patient?
 - a. Ankle stiffness
 - b. Avascular necrosis (AVN) of the talus
 - c. Subtalar and talonavicular arthritis
 - d. Talus nonunion or delayed union
 - e. Varus malunion
2. A 63-year-old man lands awkwardly while skiing and hears a loud pop in his left ankle. He cannot bear weight on the ankle, and edema ensues soon after the injury. Which of the following fractures would be missed if only radiographs of the ankle are taken without knee examination or radiographs of the entire lower leg?
 - a. Calcaneal fracture
 - b. Maisonneuve fracture
 - c. Pilon fracture
 - d. Pott fracture
 - e. Talar neck fracture
3. In an isolated ankle syndesmotic injury, the fibula is unstable in the incisura fibularis of the tibia. In what direction is the fibula most unstable?
 - a. Anterior-posterior
 - b. Medial-lateral
 - c. Proximal-distal
 - d. Rotational
 - e. Equivalent instability in all axes
4. In the Lauge-Hansen classification system, a pronation-abduction ankle fracture has what characteristic fibular fracture pattern?
 - a. Transverse fracture below the level of the syndesmosis
 - b. Short oblique fracture running from anteroinferior to posteriosuperior
 - c. Short oblique fracture running from posteroinferior to anterosuperior
 - d. Comminuted fracture at or above the level of the syndesmosis
 - e. Wagstaff fracture
5. The talocrural angle of an ankle mortise x-ray is formed between a line perpendicular to the tibial plafond and a line drawn:
 - a. Perpendicular to the medial clear space
 - b. Parallel to the talar body
 - c. Between the tips of the malleoli
 - d. Perpendicular to the shaft of the fibular
 - e. Parallel to the subtalar joint

ANSWERS:

1b, 2b, 3a, 4d, 5c.



Dr. med. Ariane Gerber Popp

University of Basel,
Liestal, Switzerland
ariane.gerberpopp@ksbl.ch

Fractures: Hand And Wrist

Fractures of the wrist

Fractures of the distal radius

Epidemiology

- Bimodal distribution: young patients/high energy trauma vs elderly patients/insufficiency fractures or high energy trauma
- Incidence: 37/10'000 person-years in women >50 years, 6/10'000 person-years in men >50 years.
- Risk of sustaining a distal forearm fracture: 6% in 80 years old women, 9% in 90 years old women.
- Medical cost worldwide \$2 billion, \$250 million associated with implant costs.

Clinical diagnostic features/Classification/Indication

- Contemporary classifications: Conney, Fernandez, AO Classification
- Generic classification considering following characteristics on an ap and lateral view of the wrist
 - Location (intraarticular, extraarticular)
 - Configuration (simple, comminuted)
 - Displacement
 - Ulnar styloid involvement
 - Integrity of the distal radioulnar joint
 - Stability
 - Associated injuries (carpal injuries, acute compressive neuropathy, compartment syndrom, complex injuries with soft tissue involvement)
 - Bone quality
- Fracture is considered potentially unstable if one or more of the following criteria is present
 - >10° loss of angulation
 - >5mm axial radial shortening
 - >2mm intraarticular incongruity
 - comminution of one cortex across the mid axis line on lateral X-Ray
 - comminution of both dorsal and palmar cortices
 - irreducible fracture
 - loss of reduction after closed reduction
- A distal radius fracture can be considered as minimally displaced (acceptable deformity) if:
 - <10° dorsal angulation
 - >15° radial inclination
 - <3mm shortening of the radius
 - >1mm step-off at the articular surface
 - see also tear drop angle, lateral radius width

- elderly low-demand patients tolerate higher degrees of deformity
- consider oblique views or CT scan if assessment of deformity is no possible on an ap and lateral view of the wrist
- Treatment
 - Goals of treatment:
 - Restoration of anatomy within the acceptable range
 - Stabilization until fracture is healed
 - Quick recovery of function
 - Addressing concomitant injuries
 - Treatment options
 - Closed reduction, plaster
 - Technique
 - Advantages: no surgery,
 - Closed reduction, pinning/external fixator
 - Advantages: no open approach
 - Open reduction, plate fixation
 - Advantages: stable fixation with locking plate design
 - Adjuvant therapy
 - Vitamine C to prevent chronic reflex pain syndrom
 - Occupational therapy, Ultrasound
 - Outcome/Complications
 - At the present time, there is no evidence showing a superior treatment modality for the treatment of distal radial fractures
 - Extraarticular deformity (as defined above) influences clinical outcome
 - The relative risk of a bad clinical outcome in the presence of an unacceptable deformity decreases with age.
 - Deformity is best tolerated in patients >65 years old with low functional demand
 - Intraarticular step-off >2mm leads to osteoarthritis. However most of the patients with osteoarthritis remains satisfied and show a good wrist function over time.
 - Tendon problems:
 - Rupture of the long extensor of the thumb in conservative treatment
 - Rupture of the flexor tendons (flexor pollicis longus) after volar plating
 - Scaring and consecutive limitation of motion (wrist and fingers) after dorsal plating

Fractures of the scaphoid

Epidemiology

- 2-7 % of all fractures, 80% of carpal bone fractures
- Incidence 5/10'000 person-years, young adult males

Clinical/diagnostic features

- Clinical suspicion if history a fall on the outstretched hand and tenderness in the anatomic snuff-box and the scaphoid tubercle
- Initial ap and lateral X-ray of the wrist detects around 70% of scaphoid fractures
 - Repeated radiograph exam 10-14 days after trauma has low sensitivity and is not indicated to detect occult fractures
 - The average sensitivity/specificity of additional diagnostic tools are 100%/90% for bone scintigraphy, 80%/100% for MRI and 93%/99% for CT scan
- Immobilisation in a cast without thumb inclusion is recommended until a scaphoid fracture has been ruled out.

Classification/Indication

- Clinical relevant classifications
 - Herbert: Fracture stability
 - Unstable fractures: displacement >1mm, angulation more than 15°, all fractures of the proximal pole, multi-fragment fractures, additional fracture of the wrist, transscaphoid-perilunate dislocations
 - Mayo: distal tubercle, distal articular surface, distal third, middle third, proximal third
- The risk of non-union in the absence of adequate immobilisation is 30%

Treatment

- Goals of treatment
 - Fracture union in anatomic position for recovery of function
- Treatment modalities:
 - Stable fractures
 - Under the elbow cast, wrist in 10° dorsal extension, without thumb inclusion
 - The duration of immobilisation depends upon the site of the fracture: fractures of distal third heal in 6 to 8 weeks; fractures of middle third heal in 8 to 12 weeks; fractures of proximal third take 12 to 24 weeks.
 - No available data showing that the overall outcome after screw fixation is better than casting for stable fractures
 - Unstable fractures:
 - Anatomic reduction and screw fixation (volar approach for distal and middle third fractures, dorsal approach for proximal third fractures)
- Complications
 - Delayed union/Non-union
 - 5%-10%, (highest prevalence in displaced fractures)
 - Necrosis
 - 13-50%, (with highest prevalence for fractures within the proximal 1/5 of the scaphoid)

Fractures of the thumb

Epidemiology

- occurs commonly in children and elderly patients
 - in the elderly 20% of all fractures of the hand
 - in young adults 12%

Clinical diagnostic features/Classification/Indication

- Thumb axis located at the trapeziometacarpal joint
 - Flexed and pronated 80° compared to the long fingers
 - Enables circumduction, opposition, prehension
- X-rays:
 - True ap (Robert's view) and lateral view mandatory to assess the deformity
- Phalangeal Fractures
 - Bony mallet lesion
 - >30% of the articular surface usually leads to instability of the IP joint and must be treated surgically
 - Shaft fractures
 - <20° Deviation in the frontal plane and <30° in the sagittal plane can be compensated
 - Osseous avulsion of the ulnar collateral ligament of the MP joint
 - Conservative treatment if: 1) <20% of the joint surface, 2) no significant displacement, 3) no substantial instability
- Extraarticular metacarpal fractures
 - Shaft fractures
 - Rare
 - Epibasal fractures (Winterstein)
 - Typical flexion deformity
 - <30° is well tolerated
 - >30° leads to compensatory hyperextension of the MP joint
- Intraarticular metacarpal base fractures
 - Bennett
 - intra-articular fracture separating the volar ulnar aspect of the metacarpal base from the remaining thumb metacarpal.
 - The volar-ulnar fragment is held in place by its ligamentous attachment to the trapezium
 - Associated with fractures of the trapezium and injury of the MP joint
 - Gedda classification (Type 1: large fragment and subluxation, Type 2: impaction fracture without dislocation, Type 3: small fragment with metacarpal dislocation)
 - Requires surgical treatment, goal: anatomic articular reduction
 - Rolando
 - Y- or T-pattern fractures that include the volar-ulnar Bennett fragment in addition to a dorsal radial fragment
 - is applied to many comminuted fractures of the base of the first metacarpal
 - more difficult to treat than Bennett fracture, outcome less predictable
 - Requires surgical treatment

Treatment

- Bony mallet lesion
 - Conservative treatment

- Dorsal splint over the IP joint, Stack splint allowing mobilization of the IP joint
- Fractures heal within 4 weeks
- Operative treatment
 - Closed reduction and transfixation of the IP joint with 1.25mm K-wire
 - Pinning the fragment may be necessary
 - Open reduction and screw fixation of large fragments
- Complications
 - Splint related skin problems
 - Injury of the nail matrix with consecutive nail growth disturbance
 - Woundhealing problems in open reduction techniques
- Extraarticular metacarpal fractures
 - Conservative treatment
 - Closed reduction accomplished through axial traction, extension, and pronation with direct pressure over the fracture dorsally.
 - Requires immobilisation of the IP joint
 - Operative treatment
 - Skeletal fixation is performed with similar techniques as those used in finger metacarpal fractures
 - Open reduction and stable plate fixation allows early mobilisation
 - Complications
 - Stiffness of the IP and MP joint
 - Scarring of the extensor mechanism in dorsal plating requiring hardware removal
 - Neuroma of the dorsal sensory branches of the radial nerve
- Intraarticular metacarpal base fractures
 - Bennett
 - Closed reduction (axial traction, palmar abduction and pronation of the thumb) and pinning to the 2. Metacarpal bone with 2x1.25mm K-Wire
 - Open reduction and pinning, or screw fixation if articular congruity (step-off > 1mm) can not be achieved
 - Rolando
 - Two large fragments: open reduction, fixation with K-wires or/and screws, additional pinning to the second metacarpal to protect the articular reconstruction
 - Comminuted fractures: Reduction of the joint plane through ligamentotaxis, external fixation
 - Complications
 - Posttraumatic osteoarthritis
- a flexion-extension arc of motion of 15–25° at the carpometacarpal joint
- The ring finger is the only metacarpal without a proximal tendon attachment.
 - The traction exerted by the insertion of the extensor carpi ulnaris at the base of the fifth metacarpal acts as a deforming force and leads to ulnar and proximal displacement of fifth metacarpal base fractures (reversed Bennett fracture or Baby Bennett)
- In extension, the collateral ligaments are lax and thus the joints may deviate radially and ulnarly.
- In flexion, the cam structure puts the collateral ligaments under tension which stabilizes the joint allowing minimal motion to radial and ulnar directed forces.
- Every 2 mm of metacarpal shortening results in 7° of extension lag.
 - Shortening of up to 6 mm is tolerable with neutral MCP extension (the MP joint hyperextend by about 20°)
- Following deformities are acceptable and non-operative management can be recommended:
 - 0°–50° of apex dorsal angulation in the small finger, more than 30° of dorsal angulation can lead to weakness of grip
 - 30° at the ring finger,
 - 20° at the middle finger
- A step off of > 1 mm or involvement of more than 25 % of the articular surface are indications for operative fixation to align the joint and minimize the risk of subsequent arthrosis.
- Rotational deformity is not well tolerated and is an indication for surgical treatment
 - 1° degree of rotation at the metacarpal results in 5° of rotation at the finger tip, and 1.5 cm of digital overlap in the closed fist.
- Clinical examination:
 - Flexion of the head, shortening leads to loss of knuckle
 - Complete neurovascular examination (ulnar motor branch) is mandatory in fractures of any of the bases of metacarpals 2 through 5 (proximity of the course of the deep branch of the ulnar nerve)
 - To assess rotation, the examiner should compare the affected and contralateral hands. Normally, all fingers point to scaphoid tubercle
- Radiographic investigation:
 - Ap, oblique and lateral view
 - CT scan especially for fractures of the base of the metacarpal bones

Fractures the finger metacarpal

Epidemiology

- 88% of non-thumb metacarpal fractures
- Fifth metacarpal most commonly involved

Clinical diagnostic features/Classification/Indication

- General considerations
 - The index and middle finger metacarpals are fixed to the carpus, and form the central pillar of the hand, which is rigidly fixed by both the bony and ligamentous anatomy of the carpometacarpal articulations
 - The ring and small finger metacarpals are mobile with

Treatment

- Goals: restore alignment within the acceptable range, correction of any rotational deformity, Correction of intraarticular deformity, proper reinsertion of avulsed tendon insertion
- Modalities
 - Shaft fractures
 - Conservative treatment: wrist splint and immediate mobilization of the fingers
 - Surgical treatment: Lag screws, intramedullary nailing, plate fixation depending on the morphology of the fracture
 - Subcapital fractures of the fifth ray

- Conservative treatment: no immobilization, buddy taping to the fourth ray
- Operative treatment: intramedullary nailing
- Head fractures
 - Open reduction with screw/plate fixation
 - Replacement arthroplasty in comminuted fractures
- Base fractures
 - Closed reduction with intermetacarpal pinning or open reduction and plate/screw fixation,
 - Fusion in comminuted fractures of the second and third rays
- Complications non-operative
 - Malunion

Deformity	Tolerable limit of deformity	Exam findings	Possible complications
Apex dorsal angulation	Neck: Index and middle fingers 10–15° Ring finger: 30° Small finger 50–70° Shaft: Index and middle fingers 10° Ring and small fingers 20°–30° Up to 6 mm	Dorsal prominence	Pseudoclauing, grip weakness, malunion
Shortening	Up to 6 mm	Loss of prominence of the MCP joint in closed fist	Extension lag, grip weakness
Rotation	No tolerable limit	Extension lag Malaligned nail beds Finger overlap/scissoring in closed fist	Scissoring, grip weakness

from Carpenter et al, Hand (2014), 9:16–23

- Complication operative treatment
 - Stiffness
 - Hardware problems requiring removal (4–32%)
 - Infection (1%)

Phalangeal fractures of the fingers

Epidemiology

- occurs commonly in adults and machinerie is a dominant cause of injury

From Carpenter et al, Hand Clin (2013) 29:519–34

Table 1
Characteristics predictive of phalangeal fracture stability

	Stable	Unstable
Anatomic location	Distal phalanx	Subcondylar proximal phalanx
Fracture characteristics	Simple, transverse Impacted	Short oblique, spiral Comminuted
Displacement	None or minimal	Displaced, malrotated
Articular incongruity	None or minimal	Incongruous surface
Soft tissue injury	Minimal	Severe

Treatment

- Goals: restoration of anatomy, minimizing soft tissue injury and enabling mobilization of the injured digit as soon as fracture stability permits.
- Middle and proximal shaft fractures
 - Conservative treatment: stable fractures
 - Buddy tape, early mobilisation
 - Operative treatment
 - Stable fixation for early mobilization
 - Periosteum repair to protect gliding surfaces and repair of the extensor tendon mechanism are critical
 - Closed reduction with K-Wire fixation, Open reduction with lag screws (spiral) or plate (transverse) fixation
- Proximal phalanx base fractures: ligament avulsion
 - Conservative treatment: stable joint, small fragment
 - Buddy tape, early mobilisation
 - Operative treatment: instability (especially on the radial side: pinch!)
- Proximal phalanx base fractures: epibasal fractures (typical extension deformity, usually unstable)
 - Operative treatment:
 - Closed reduction, crossed K-wire fixation
 - Open reduction, plate fixation
- Middle phalanx base fractures: intraarticular dorsal avulsion
 - Closed reduction, dynamic splint
 - Open reduction and fixation of the central slip if closed reduction fails
- Middle phalanx base fractures: intraarticular volar avulsion
 - Small avulsion with stable joint are treated with a 30° dorsal splint fixed at the middle phalanx, allowing full flexion of the PIP joint and limiting extension.
 - Fragment >40% of the articular surface leads to subluxation of the PIP joint and should be treated with open reduction and screw fixation.
- Middle phalanx base fractures: intraarticular pilon lesion
 - Must be treated surgically. If open reduction and fixation is not possible, external fixator or arthroplasty may be alternative options
- Distal phalanx base fractures
 - See osseous mallet lesion of the thumb
- Complications (nonoperative or operative management)
 - delayed union, nonunion, malunion
 - soft tissue adhesions, joint contractures, infection, posttraumatic arthritis, tendon rupture.
 - hardware issues

References (1–15)

1. Lafontaine M, Hardy D, Delince P. Stability assessment of distal radius fractures. *Injury*. 1989;20(4):208–10.
2. Dias JJ, Wildin CJ, Bhowal B, Thompson JR. Should acute scaphoid fractures be fixed? A randomized controlled trial. *J Bone Joint Surg Am*. 87. United States 2005. p. 2160–8.
3. Bushnell BD, Draeger RW, Crosby CG, Bynum DK. Management of intra-articular metacarpal base fractures of the second through fifth metacarpals. *J Hand Surg Am*. 2008;33(4):573–83.

4. Carlsen BT, Moran SL. Thumb trauma: Bennett fractures, Rolando fractures, and ulnar collateral ligament injuries. *J Hand Surg Am.* 2009;34(5):945-52.
5. Rozental TD, Blazar PE, Franko OI, Chacko AT, Earp BE, Day CS. Functional Outcomes for Unstable Distal Radial Fractures Treated with Open Reduction and Internal Fixation or Closed Reduction and Percutaneous Fixation. 2009.
6. Buijze GA, Ring D. Clinical impact of United versus nonunited fractures of the proximal half of the ulnar styloid following volar plate fixation of the distal radius. *J Hand Surg Am.* 35. United States: 2010. Published by Elsevier Inc.; 2010. p. 223-7.
7. Diaz-Garcia RJ, Oda T, Shauver MJ, Chung KC. A systematic review of outcomes and complications of treating unstable distal radius fractures in the elderly. *J Hand Surg Am.* 2011;36(5):824-35 e2.
8. Rhemrev SJ, Ootes D, Beeres FJ, Meylaerts SA, Schipper IB. Current methods of diagnosis and treatment of scaphoid fractures. *Int J Emerg Med.* 2011;4:4.
9. Carpenter S, Rohde RS. Treatment of phalangeal fractures. *Hand Clin.* 2013;29(4):519-34.
10. Hammert WC, Kramer RC, Graham B, Keith MW. AAOS appropriate use criteria: treatment of distal radius fractures. *J Am Acad Orthop Surg.* 2013;21(8):506-9.
11. Buijze GA, Goslings JC, Rhemrev SJ, Weening AA, Van Dijkman B, Doornberg JN, et al. Cast immobilization with and without immobilization of the thumb for nondisplaced and minimally displaced scaphoid waist fractures: a multicenter, randomized, controlled trial. *J Hand Surg Am.* 2014;39(4):621-7.
12. Carpenter CR, Pines JM, Schuur JD, Muir M, Calfee RP, Raja AS. Adult scaphoid fracture. *Acad Emerg Med.* 2014;21(2):101-21.
13. Daneshvar P, Chan R, MacDermid J, Grewal R. The effects of ulnar styloid fractures on patients sustaining distal radius fractures. *J Hand Surg Am.* 2014;39(10):1915-20.
14. Kollitz KM, Hammert WC, Vedder NB, Huang JI. Metacarpal fractures: treatment and complications. *Hand (N Y).* 2014;9(1):16-23.
15. Mallee WH, Henny EP, van Dijk CN, Kamminga SP, van Enst WA, Kloen P. Clinical Diagnostic Evaluation for Scaphoid Fractures: A Systematic Review and Meta-Analysis. *Journal of Hand Surgery.* 2014;39(9):1683-91.e2.



Prof. Dr. med. Pierre Hoffmeyer

HUG, University Hospitals of Geneva,
Geneva, Switzerland

pierre.hoffmeyer@hcuge.ch

Fractures: Shoulder, Arm, Elbow And Forearm

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, Distal third Proximal third

Mid-third

- Undisplaced:
Sling or figure of 8 bandage.
- Displaced:
Relative indications for fixation:
 - >100% displacement clavicle diameter;
 - > 2 cm of shortening;
Professional cyclist.
Absolute indications for fixation:
Flail chest, scapulothoracic dissociation, fractures menacing skin or open fractures

Technique

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.

European Surgical Orthopaedics and Traumatology. The EFORT Textbook. Ed: G Bentley. Springer Berlin, 2014

Distal third



Neer Type I



Neer Type II



Neer Type III

Neer I:

Displacement > 100% superior-inferior; skin menace; open fracture:
Surgical fixation:

Small distal fragment: simple excision.

Large fragment: 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

Surgical fixation: Major displacement, neurovascular or functional (tracheal or oesophageal) compromise

Complications of Clavicle fractures

Infections, nonunions (up to 30%), neurovascular compromise, reoperations >50% for hardware removal.

European Surgical Orthopaedics and Traumatology. The EFORT Textbook. Ed: G Bentley. Springer Berlin, 2014

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.

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)

European Surgical Orthopaedics and Traumatology. The EFORT Textbook. Ed: G Bentley. Springer Berlin, 2014

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.

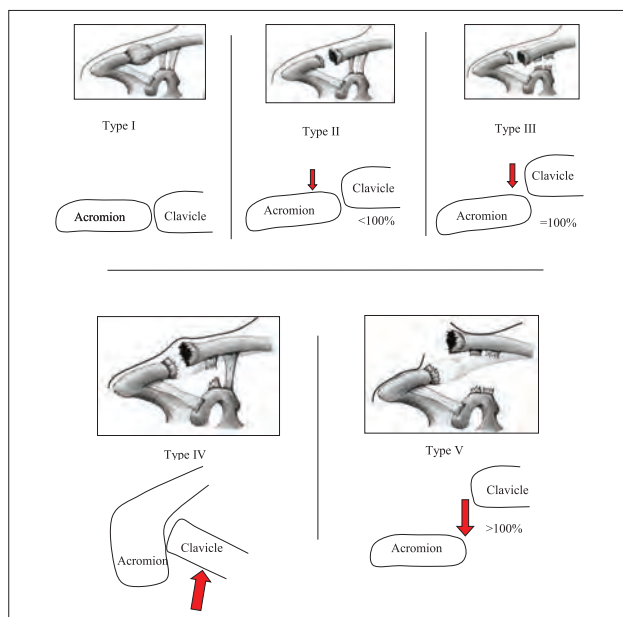
Treatment:

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

Surgical techniques:

Acute cases: 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)



European Surgical Orthopaedics and Traumatology. The EFORT Textbook. Ed: G Bentley. Springer Berlin, 2014

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.

European Surgical Orthopaedics and Traumatology. The EFORT Textbook. Ed: G Bentley. Springer Berlin, 2014

Scapula fractures

Incidence:

0.3% of all fractures. Mortality 10% to 15% due to associated thoracic and cranial injuries.

Mechanism:

High energy trauma with 80 to 95% incidence of associated trauma 50% of which are thoracic trauma.

Body Fractures

Mechanism:

Direct trauma

Clinical:

Neurovascular assessment, thoracic examination

Diagnosis:

Plain X-Ray, CT with 3D reconstruction.

Classification:

Ideberg

Clinical:

Shoulder pain, neurovascular signs

Treatment:

Conservative as a rule

European Surgical Orthopaedics and Traumatology. The EFORT Textbook. Ed: G Bentley. Springer Berlin, 2014

Glenoid Fractures

Mechanism:

High energy trauma.

Clinical:

Pain on mobilisation, glenohumeral dislocation

Diagnosis:

Plain X-Rays, CT, Occasionally MRI for soft tissues.

Treatment:

Conservative: If less than 35% of surface involved and humeral head centered.

Surgical fixation:

- Absolute indication if humeral head dislocated or subluxed.
- Lateral scapular fragment impinging on the humeral head
- Medially displaced glenoid neck fractures associated with clavicle fracture: Clavicle fixation aids to reduce and stabilize.

European Surgical Orthopaedics and Traumatology. The EFORT Textbook. Ed: G Bentley. Springer Berlin, 2014

Acromion and Spine Fractures

Mechanism:

Direct trauma after a fall or impact.

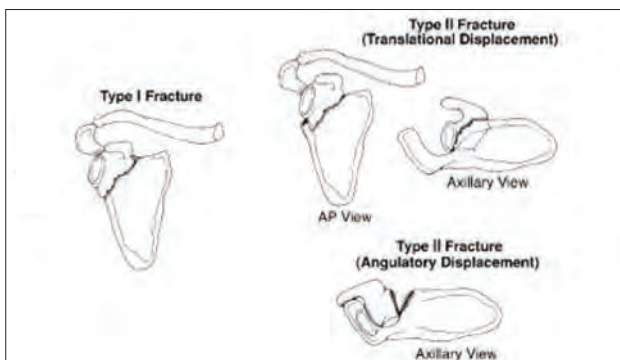
Clinical:

Tenderness and pain on palpation and mobilisation of the shoulder.

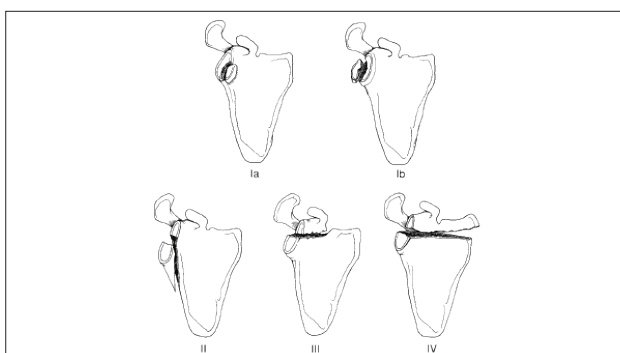
Treatment:

For displaced fractures screw/plate fixation or tension band fixation to counteract deformity due to deltoid tension and pull.

European Surgical Orthopaedics and Traumatology. The EFORT Textbook. Ed: G Bentley. Springer Berlin, 2014



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.

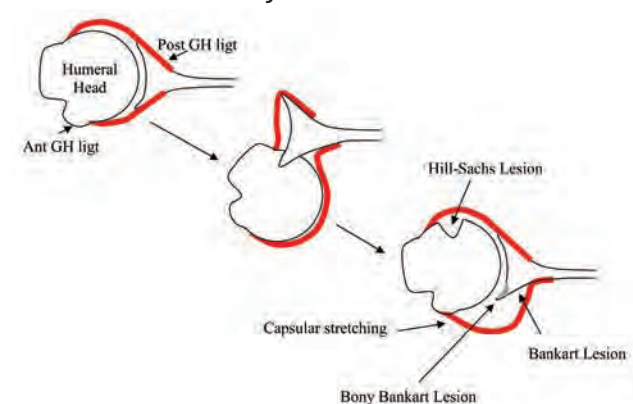
Instability:

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



Dislocation and instability types

- 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 lidocaine 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 documented evidence 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:

Open techniques:

Capsulorraphy, Bankart refixation, bony augmentation (Latarjet).

Arthroscopic techniques:

Traumatic Bankart lesions, remplissage for major Hill-Sachs lesions, closed Latarjet technique

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 or "handshake 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 augmentation.
- 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

Massive rotator cuff tear present.

Recurrent dislocation in the elderly patient

Treatment:

Tendon repair, reverse prosthesis, arthrodesis.

Debilitated patients:

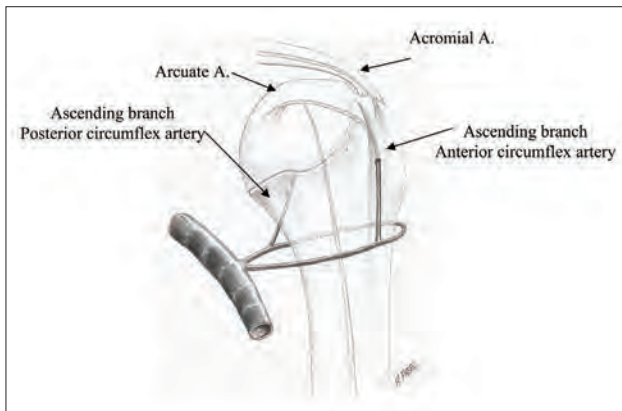
Abstention, reverse prosthesis, arthrodesis.

Wilful dislocation.

Rehabilitation, psychiatric help, avoid surgery.

European Surgical Orthopaedics and Traumatology. The EFORT Textbook. Ed: G Bentley. Springer Berlin, 2014

Vascular anatomy of the Humeral Head



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 fractures: 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.

NB: Surgical treatment in the elderly is controversial because equivalent results may be obtained by either conservative or surgical treatment. No evidence is available definitively favouring one or the other method.

European Surgical Orthopaedics and Traumatology. The EFORT Textbook. Ed: G Bentley. Springer Berlin, 2014

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.

European Surgical Orthopaedics and Traumatology. The EFORT Textbook. Ed: G Bentley. Springer Berlin, 2014

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.

European Surgical Orthopaedics and Traumatology. The EFORT Textbook. Ed: G Bentley. Springer Berlin, 2014

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)

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.

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.

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.

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.

European Surgical Orthopaedics and Traumatology. The EFORT Textbook. Ed: G Bentley. Springer Berlin, 2014

Elbow fractures

General surgical pathoanatomy

Distal humerus is an 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: Ulna-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.

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 supinatoris of the ulna is released, and the entire muscle flap is retracted radially, exposing the radiohumeral joint. The posterior interosseous nerve is protected in the substance of the supinator, which must be gently retracted

Posterior approach with extensile exposure of the distal humerus (Fractures distal humerus, arthroplasty, stiff elbow):

- Bilateral tricipital 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.

European Surgical Orthopaedics and Traumatology. The EFORT Textbook. Ed: G Bentley. Springer Berlin, 2014

- 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 intra-articular fractures: 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. Once 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.

European Surgical Orthopaedics and Traumatology. The EFORT Textbook. Ed: G Bentley. Springer Berlin, 2014

- 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.

- 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.

European Surgical Orthopaedics and Traumatology. The EFORT Textbook. Ed: G Bentley. Springer Berlin, 2014

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 lidocaine smooth, non-grating, active or passive pronosupination 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 disruption of 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.

European Surgical Orthopaedics and Traumatology. The EFORT Textbook. Ed: G Bentley. Springer Berlin, 2014

Fractures of the olecranon

Incidence:

10% of fractures of the adult elbow.

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.

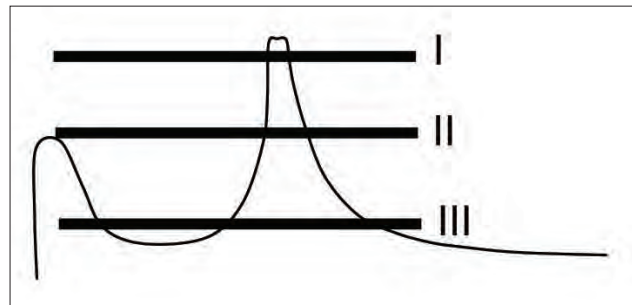
Coronoid fractures

Clinical:

Associated with elbow dislocations.

Imaging:

Plain x-rays, ct, 3D CT



Classification:

Regan and Morrey

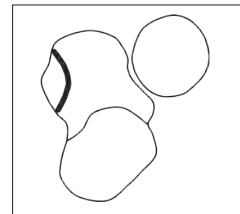
Type I: Fracture of the tip

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:

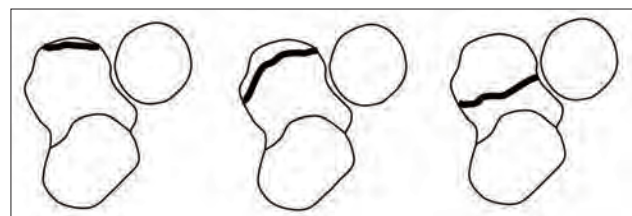
Type I and II stable no fixation. Types III and IV need fixation: Medial collateral ligament attaches to medial coronoid and risk of instability.

O'Driscoll has modified this classification

Type 1: Tip fracture

Type 2 : Anteromedial fracture

Type 3 : Base of coronoid fracture



NB: 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.

European Surgical Orthopaedics and Traumatology. The EFORT Textbook. Ed: G Bentley. Springer Berlin, 2014

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 than 7 days. Start at 7 days with active-assisted flexion extension and at three weeks with pronosupination.

Complications:

Heterotopic bone, stiffness, neurovascular injury.

European Surgical Orthopaedics and Traumatology. The EFORT Textbook. Ed: G Bentley. Springer Berlin, 2014

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 exercises. 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.

European Surgical Orthopaedics and Traumatology. The EFORT Textbook. Ed: G Bentley. Springer Berlin, 2014

Medial instability of the elbow

Clinical:

Throwing athletes, pain and "pop" medially while throwing. Tinell's sign for ulnar nerve.

Imaging:

Stress x-rays, MRI, dynamic ultrasound.

Treatment:

Medial collateral ligament reconstruction (Fascia lata, Palmaris longus)

European Surgical Orthopaedics and Traumatology. The EFORT Textbook. Ed: G Bentley. Springer Berlin, 2014

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:

Stretching, braces, injection, immobilization

Surgical:

Technique:

Open or arthroscopic surgical excision of the ECRB tendon origin, situated under the Extensor Carpi Radialis Longus tendon.

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 Arthrography.

Treatment:

Tendon graft uniting the humerus to the supinator crista of the ulna and passing under the radial head.

European Surgical Orthopaedics and Traumatology. The EFORT Textbook. Ed: G Bentley. Springer Berlin, 2014

Distal Biceps Tendon Ruptures

Clinical:

Weakness in pronosupination, palpable muscle ball above elbow crease.

Rehabilitation:

Protective splint followed by gentle motion as tolerated with full function possible 6 to 8 weeks postoperatively.

European Surgical Orthopaedics and Traumatology. The EFORT Textbook. Ed: G Bentley. Springer Berlin, 2014

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 (Stretching, braces, injection)

Surgical excision of the diseased part of the medial conjoint tendon of the flexor-pronator complex with transposition of the ulnar nerve.

European Surgical Orthopaedics and Traumatology. The EFORT Textbook. Ed: G Bentley. Springer Berlin, 2014

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 anterior)

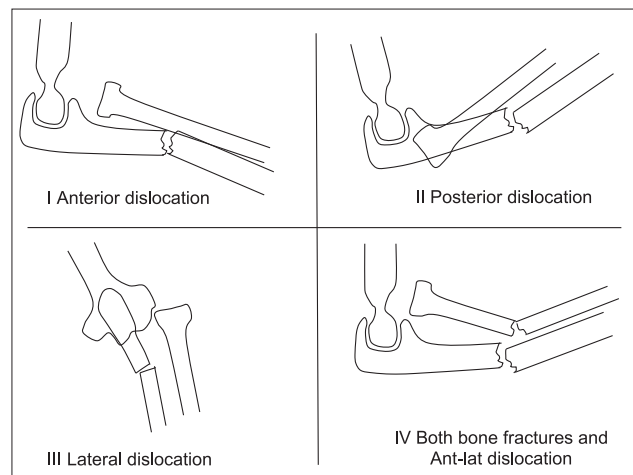
II Posterior or postero-lateral radial head dislocation and proximal ulnar shaft fracture (apex posterior)

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)

European Surgical Orthopaedics and Traumatology. The EFORT Textbook. Ed: G Bentley. Springer Berlin, 2014

Monteggia fractures: Bado classification



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.

European Surgical Orthopaedics and Traumatology. The EFORT Textbook. Ed: G Bentley. Springer Berlin, 2014

Forearm fractures

Introduction:

Forearm a whole functioning joint pronation of 75°, supination of 85°. Interosseous membrane major stabiliser.

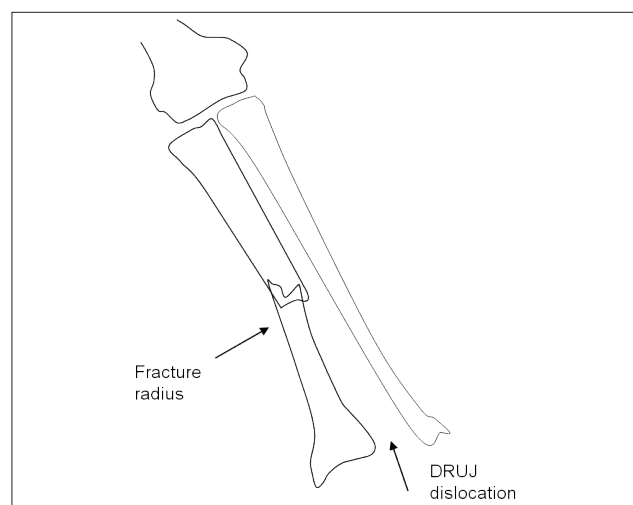
Incidence:

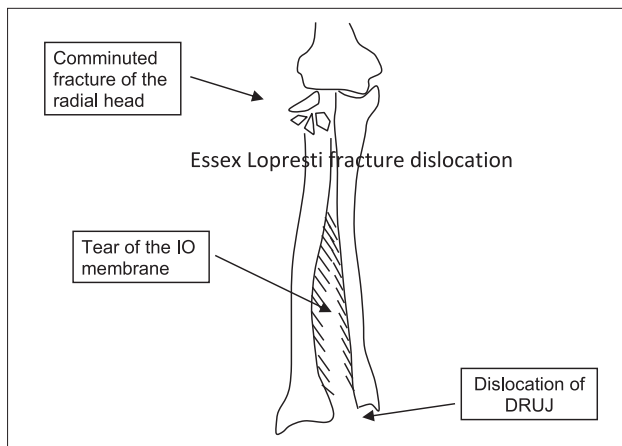
1.2% of fractures

Mechanism:

Direct trauma (nightstick fracture), fall, polytrauma.

Galeazzi Fracture





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 torn 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, recommend 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).

http://www.wheelsonline.com/ortho/dorsal_approach_thompson

Direct approach

The direct approach is best suited for the ulna.

- 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.

European Surgical Orthopaedics and Traumatology. The EFORT Textbook. Ed: G Bentley. Springer Berlin, 2014

Complications:

Non-union, malunion, compartment syndrome, infection.

European Surgical Orthopaedics and Traumatology. The EFORT Textbook. Ed: G Bentley. Springer Berlin, 2014

QUESTIONS

- The best x-ray view to diagnose a posterior dislocation of the shoulder is:
 - axial View
 - Neer view
 - AP view
 - Zanca view
 - Transthoracic view
- The most specific clinical sign of a posteriorly dislocated shoulder is:
 - fullness beneath the coracoids
 - limited elevation
 - limited external rotation
 - Limited internal rotation
 - Weakness in abduction
- The axillary nerve does not supply which muscle:
 - Anterior deltoid
 - Posterior deltoid
 - Middle deltoid
 - Teres major
 - Teres minor
- The platysma muscle is innervated by:
 - The axillary nerve
 - The trigeminal nerve
 - The facial nerve
 - The accessory nerve
 - The hypoglossus
- A displaced four part proximal humerus fracture with an acute axillary nerve palsy is best treated by:
 - Hemiarthroplasty
 - Reverse arthroplasty
 - Axillary nerve reconstruction followed by reverse arthroplasty
 - Axillary nerve reconstruction treated by hemiarthroplasty
 - Axillary nerve reconstruction

ANSWERS:

1a, 2c, 3d, 4c, 5a



 #CRC

EFORT Head Office

ZA La Pièce 2
1180 Rolle, Switzerland

Phone +41 (0)21 343 4400

Fax +41 (0)21 343 4411

office@efort.org

www.efort.org



www.efort.org/linkedin



www.efort.org/facebook



www.efort.org/twitter



www.efort.org/youtube