ΕΛΛΑΣ ΛΕΠΤΑ 30

5th ICRS Surgical Skills Course

Larissa | Greece, October 23–25, 2014

HANDS ON COURSE IN CARTILAGE REPAIR

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Final Programme

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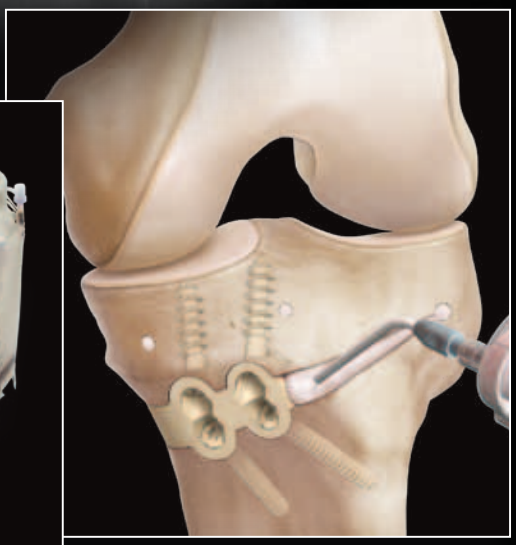

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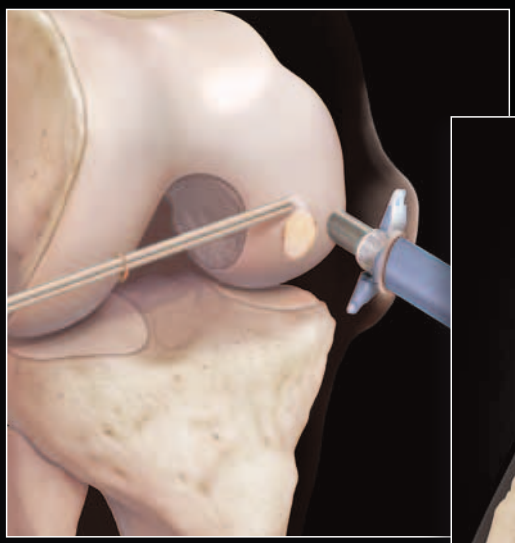
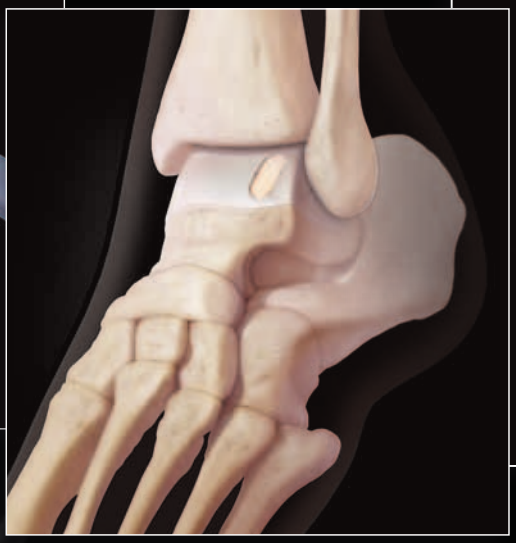


BioCartilage® Micronized Cartilage Matrix for Knee Defects



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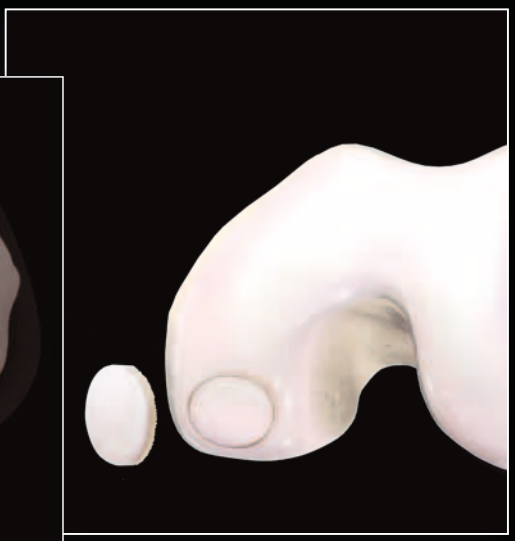

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Focus Meeting – The Knee

Konstantinos Malizos

Michael Hantes

Dear Colleagues & Friends

It gives us a great pleasure to welcome you to the 5th ICRS Surgical Skills in Cartilage repair, in Larissa, Greece from October 23 to 25, 2014 at the University of Thessaly.

Internationally, acknowledged surgeons and experts in the field of cartilage repair, will provide you with the newest information, tips and tricks of all options in the surgical treatment of chondral lesions. The Programme Committee has developed an outstanding academic agenda to include scientific lectures, live demonstrations, and hands-on workshops on human cadaver specimens. The course will include core lecture modules for cartilage and osteochondral repair. Each module will handle a specific area (cartilage repair techniques, meniscal repair replacement, patellofemoral joint problems management, osteotomies) in order that the trainee will acquire knowledge on the field of his/her training. We believe that this programme will cover the challenges of the cartilage repair field.

Larissa, the region where Hippocrates lived for many years, taught and died, is located in the crossroad of mainland Greece, offering many opportunities in early Spring for journeys to the eastern slopes of Olympus mountain or to the Greek Orthodox monasteries built on natural sandstone rock pillars at Meteora. For those who may stay in town, Larissa is going to challenge you with the modern city market, the impressive Ancient Theatre and the numerous taverns and coffee shops.

Please read over the information on our website describing the course program and other important details. We are looking forward to welcoming you in Larissa and wish you to have a nice stay with us.

Sincerely
Konstantinos Malizos, Professor & Chairman
Department of Orthopaedics & Musculoskeletal Trauma
University of Thessaly

Michael Hantes, Associate Professor
Department of Orthopaedics & Musculoskeletal Trauma
University of Thessaly

Organization

Course Director

Konstantinos K. Malizos, Prof. MD
University of Thessalia, School of Medicine
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Course Organizing Office

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International Faculty

Brittberg Mats, SE
Georgoulis Anastasios, GR
Gobbi Alberto, IT
Erggelet Christoph, CH
Hantes Michael, GR
Iosifidis Michael, GR
Malizos N. Konstantinos, GR
Nehrer Stefan, AT
Papacostas Emmanuel, GR
Saris Daniël, NL
Spalding Tim, UK
Tsezou Aspasia, GR
Petersen Wolf , DE
Peterson, Lars, SE
Vlychou Mariana, GR

ICRS Executive Board

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Venue & Lab Coordination

Michael Hantes, Assoc. Prof. MD
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Local Organizing Committee

Konstantinos N. Malizos, Larissa, Greece
Michael Hantes, Larissa, Greece
Emmanuel Papacostas, Thessaloniki, Greece
Michael Iosifidis, Thessaloniki, Greece
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Konstantinos N. Malizos, Larissa, Greece
Michael Hantes, Larissa, Greece
Emmanuel Papacostas, Thessaloniki, Greece
Michael Iosifidis, Thessaloniki, Greece

Scientific Programme

Thursday, 23 October 2014

13:00-15:00	**Registration**
14:30 - 14:45	**Welcome Addresses** *Ioannis Stefanidis (GR), George Petrakos (GR)* *Christoph Erggelet (CH), Konstantinos Malizos (GR)*
14:45 – 15.15	**Introduction** *Special Guest Speaker*

Session 1: Basic Science and Imaging in Cartilage Repair

Moderator: Alberto Gobbi (IT)

15:15 - 15:30	**1.1 Cartilage Special Ultra-Structural Anatomy & Composition** *Michael Iosifidis (GR)*
15:30 - 15:45	**1.2 Current Trends in Cartilage Imaging** *Mariana Vlychou (GR)*
15:45 – 16:00	**1.3 Cell Culturing for Tissue Engineering** *Mats Brittberg (SE)*
16:00 – 16:15	**1.4 Evaluation of Cartilage Repair Tissue** *Emanuel Papacostas (GR)*
16:15 – 16:30	**1.5 Treatment Algorithms** *Alberto Gobbi (IT)*
16:30 – 16:45	**Take Home Points** *Alberto Gobbi (IT)*
16:45 - 17:15	**Coffee Break / Exhibition**

Session 2: Cartilage Fate after Joint Injury

Moderator: Daniël Saris (NL)

17:15 – 17:30	**2.1 Biochemical Changes of the Subchondral Bone after Joint Injury** *Aspasia Tsezou (GR)*
17:30 – 17:45	**2.2 Impact of ACL Rupture on Cartilage stauts: Wait or Repair?** *Anastasios Georgoulis (GR)*
17:45 – 18:00	**2.3 Meniscus Lesion: How «far» do we have to try to repair it?** *Daniël Saris (NL)*
18:00 – 18:15	**2.4 Meniscus Replacement for Cartilage Protection** *Tim Spalding (UK)*
18:15 – 18:30	**2.5 Cartilage & Meniscal Status after ACL Injury** *Konstantinos Malizos (GR)*
18.30 – 18.45	**2.6 Ten toTwenty Years Follow-up of ACI with ACL Reconstruction** *Lars Peterson (SE)*
18.45 – 19.00	**2.7 ACL & PCL Reconstruction** *Wolf Petersen (DE)*
19:00 – 19:10	**Take Home Points** *Daniël Saris (NL)*
19:10 - 20:30	**Welcome Reception**

Scientific Programme

Friday, 24 October 2014

Session 3: Treatment Options for Cartilage Lesions

Moderator: Mats Brittberg (SE)

08:00 - 08:15 **3.1 Microfracture & Autologous Matrix-Induced Chondrogenesis (AMIC)**
Michael Hantes (GR)

08:15 – 08:30 **3.2 How Helpful is the Abrasion Technique for a Quick Return in the High Level Athletes?**
Emanuel Papacostas (GR)

08:30 – 08:45 **3.3 OATS - Osteochondral Autograft Transfer System**
Wolf Petersen (DE)

08:45 – 09:00 **3.4 Four generations of ACI; Mode of Actions, Techniques & Results**
Mats Brittberg (SE)

09:00 – 09:15 **3.5 Matrix-Induced Autologous Chondrocyte Implantation**
Christoph Erggelet (CH)

09:15 – 09:30 **3.6 MSCs for Cartilage Repair**
Daniël Saris (NL)

09:30 – 09:45 **3.7 Regulatory Matters**
Christoph Erggelet (CH)

09:45 – 10:00 **Take Home Points**
Mats Brittberg (SE)

10:00 - 10:45 **Coffee break / Exhibition / Changing into Scrubs**

Session 4: Hands-on Skills Lab: Cartilage Repair Techniques

Lab Leaders: Mats Brittberg (SE), Christoph Erggelet (CH)
Lab Instructors: All Faculty

10:45 – 11:15 **Live Demonstrations:**
15' MaioRegen / Finceramica – Mats Brittberg (SE)
15' ChondroCelect / SOBI – Daniël Saris (NL)

11:15 – 12:30 **Hands-on Experience: Cartilage Repair Techniques**
- Defect Preparation, Shouldering and Cartilage Biopsy
- Microfracture/AMIC Technique
- Autologous Osteochondral Transfer

For "Lectures Only" Participants
Video Demos & Case Discussions in the Auditorium

12:30 - 13:10 **Lunch Break**

Session 5: Reducing the Joint Surface Loading – Osteotomies

Moderator: Anastasios Georgoulis (GR)

13:10 – 13:30 **5.1 Minimally Invasive Technique for High Tibial Osteotomies: The Taylor Spatial Frame**
Konstantinos Malizos (GR)

13:30 – 13:50 **5.2 Distal Femoral Osteotomies**
Stefan Nehrer (AT)

13:50 – 14:10 **5.3 Combined Surgical Interventions for Instability& Cartilage Problems**
Mats Brittberg (SE)

14:10 – 14:15 **Take Home Points**
Anastasios Georgoulis (GR)

14:15 – 14:30 **Changing into Scrubs**

Scientific Programme

Session 6: Hands-on Skills Lab: Patellofemoral Joint/Osteotomies/Cartilage Repair

Lab Leaders: Konstantinos Malizos (GR), Lars Peterson (SE)
Lab Instructors: All Faculty

14:30 - 15:00 **Live Demonstration: MPFL Reconstruction**
15' Hyalofast / Anika – Stefan Nehrer (SE)
15' MPFL / Arthrex – Chris Erggelet (CH)

15:00 - 16:15 **Hands-on Experience: Patellofemoral Joint/Osteotomies**
- High Tibial Osteotomy
- Tibial Tubercle Osteotomy
- MPFL Reconstruction

For "Lectures Only" Participants
Video Demos & Case Discussions in the Auditorium

16:15 – 17:00 **Coffee break / Exhibition**

Session 7: Hands-on Skills Lab: Meniscal Repair/Replacement

Lab Leaders: Tim Spalding (UK), Daniël Saris (NL)
Lab Instructors: All Faculty

17:00 - 17:15 **Live Demonstration: Meniscal Repair**
15' Actifit / Orteq – Tim Spalding (UK)

17:15 - 18:00 **Hands-on workshop Meniscal Repair/Replacement**

For "Lectures Only" Participants
Video Demos & Case Discussions in the Auditorium

20:00 – 23:00 **Faculty/Course Dinner (50 Euros for Participants)**

19.45 Bus transfer from Hotel Imperial and Divani

Scientific Programme

Saturday, 25 October 2014

Session 8: Problems of the Patellofemoral Joint
Moderator: Christoph Erggelet (CH)

08:00 – 8:15 **8.1 Imaging of the Patellofemoral Joint**
Mariana Vlychou (GR)

08:15 – 8:30 **8.2 MPFL Reconstruction**
Michael Hantes (GR)

08:30 – 8:45 **8.3 Trochleoplasty**
Lars Peterson (SE)

08:45 – 9:00 **8.4 Tibial Tubercle Osteotomy**
Christoph Erggelet (CH)

09:00 – 9:15 **8.5 Patellofemoral Joint Cartilage Defects**
Alberto Gobbi (IT)

09:15 – 9:30 **8.6 Patella Dislocation & Chondral Lesions**
Michael Iosifidis (GR)

09:30 – 9:35 **Take Home Points**
Christoph Erggelet (CH)

Session 9 Case Presentations & Discussions

09:35 – 10:30 **What would You do? What would the Expert do?**
Moderator: Daniël Saris (NL)

10:30 - 10:45 **Closing Remarks**
Christoph Erggelet (CH)

10:45 - 11:30 **Coffee Break / Exhibition / Changing into Scrubs**

Session 10 Hands-on Skills Lab: ACL Reconstruction Techniques
Lab Leaders: Alberto Gobbi (IT), Michael Hantes (GR)
Lab Instructors: All Faculty

11:30 – 12:00 **Live Demonstration: ACL Anatomic Reconstruction Technique**
15’ ACL / Karl Storz – Wolf Petersen (DE)
15’ ACL / Arthrex – Alberto Gobbi (IT)

12:00 – 13:30 **Hands-on Experience: ACL Reconstruction Technique**
- Single Bundle Reconstruction
- Double Bundle Reconstruction
- Partial ACL Reconstruction

13:30 **Farewell Reception & Adjourn**

International Faculty

Mats Brittberg, Ass. Prof., MD, PhD
Kungsbacka Hospital, Göteborg University, Kungsbacka, Sweden

Mats Brittberg is a member of the Cartilage Research Unit at University of Gothenburg and an orthopaedic surgeon at Region Halland Orthopaedics at the Kungsbacka Hospital, Kungsbacka, Sweden. He received his MD at the University of Gothenburg in 1978 and completed a specialization in orthopaedics in 1985. In 1992 he passed the Swedish Orthopaedic Board Exam (S.O.B.E.), and in 1996 he earned a PhD. He is now a professor of orthopaedics connected to the Institution of clinical sciences and orthopaedic department, University of Gothenburg.

Mats Brittberg's research has been focused on cartilage repair and with main focus on cartilage regeneration with in vitro expanded autologous chondrocytes. Today the main interest is the European Connective Tissue Engineering centre (ECTEC) which is research collaboration between the Sahlgrenska Academy at University of Gothenburg with the institution of Polymer Technology, Chalmers Technical University. Mats Brittberg has also had research collaboration with Virginia Tech in USA on biotribology in cartilage and osteoarthritis as well as research collaborations with other centres in Europe and North America.

In September, 2010, Mats Brittberg received the ICRS Genzyme Lifetime Achievement Award in cartilage research and in 2012, the Shetty- Kim Foundation (SKF) Scientific award. In 2014, Mats Brittberg also received the SKF and UK Cartilage Club's Life time achievement Award.

Mats Brittberg has been on the board of TESi (Tissue engineering Society International) and has been chairing the Cartilage Committee of ESSKA 2006-08. Since the start 1997, he has been working with ICRS, as a secretary, Vice-president and President (2006-2008) and finally Past-President (2008-2009). He is since January 2013 Editor-in-Chief for the Sage journal ¨CARTILAGE. He is also associate editor with ESSKA journal as well as being on the editorial board of Osteoarthritis and Cartilage. Mats Brittberg is also involved in the Go:Life platform for scientific meetings in Gothenburg.

Christoph Erggelet, MD, Prof., PhD
Center for Biologic Joint Surgery, Zürich, Switzerland

Christoph Erggelet is an orthopaedic surgeon in Zurich/ Switzerland affiliated with the Department for Orthopaedic Surgery and Traumatology, University Medical Center, University of Freiburg/Germany. He received his MD in 1986 and passed the board exam for Orthopaedic Surgery in 1993. A PhD was granted by the University of Essen/Germany in 1987. Since 2002, he is faculty member of the University Medical School, University of Freiburg Germany. Research interests focus on biologic regeneration of joint function, e.g. culture of autologous chondrocytes, meniscus regeneration and ligament repair.

He served as a founding board member of the Bio Valley initiative, a tri-national tissue-engineering group, which enabled the setup of a licenced GMP laboratory at the University of Freiburg. International collaborations included board membership of the EU-funded EUROCELL program and the international Cartilage Repair Registry. Recent research has been done on stress loading of cartilage defects and stability of biodegradable scaffolds in collaboration with the Swiss Federal Institute of Technology Zurich/Switzerland. Christoph Erggelet is a member of the ICRS since the foundation in 1997 and served as a board member. He is currently the President of ICRS for the term office 2013 – 2015.

International Faculty

Anastasios Georgoulis, MD, Prof,
University of Ioannina Greece

Residency and Fellowship in Traumatology, Arthroscopy and Sports Medicine in Berlin, Universitaets Klinikum, Rudolf-Virchow and Doctor der Medizinischen Facultaet, Freies Universitaet Berlin , 1978-1990. Since 1990 Elected Member of the Medical School, University of Ioannina, in 2000 established the Orthopedic Sports Medicine Center. Since 2006 Elected Professor. He served as an Associate Editor in «Journal of Arthroscopy» and «KSSTA» for 5 years. He was the Chairman Program of ESSKA Congress in 2004 and is the Chairman Program of ISAKOS Congress 2015. He was Godfather of ESSKA travel Fellowship in Asia Pacific 2008. He is Member of the Herodicus Society.He is honorary Member of AANA .Visiting Professor in 6 Universities and invited Faculty Member in 18 international Societies. He has about 150 Publications, pub med papers, book chapters.

Alberto Gobbi, MD
Orthopaedic Arthroscopic Surgery International, Milano, Italy

Dr. Alberto Gobbi was born in Milan, Italy, he is a Board certified surgeon and specialist in the field of orthopaedics, traumatology and sports medicine. He has been performing highly skilled surgeries for decades, which coupled with his scientific and research oriented mind, have led to a tremendous growth in the field of arthroscopy and cartilage repair worldwide. He chairs OASI Bioresearch Foundation Gobbi Onlus, a No-Profit Organization, accredited by the Italian Ministry of Health and recognized as an International Teaching Center by International Society of Arthroscopy, Knee Surgery & Orthopaedic Sports Medicine (ISAKOS) and International Cartilage Repair Society (ICRS). The OASI Foundation promotes research on cartilage, joint aging and sports lesions and collaborates with surgeons from across the globe. He has innumerable publications to his name. He is the Associate Editor of 'Cartilage' since 2010 and the Chair of the Education Committee for ISAKOS. In 2012, he was awarded the Best International Publication in an American Journal (AOSSM). Serving ICRS as General Secretary, in 2014 he received an appointment as visiting professor at U.C.S.D. with Prof Amiel director of Connective Tissue Biochemistry Laboratories in San Diego USA. A talented sportsman himself, he is doctor to the Italian National Olympics Committee since 1983. He served the Medical Committees of the Italian Motorcycle Federation and the Motor-boating Federation for over 10 years. Dr. Gobbi's office is located in Milan.

Michael Hantes, Ass. Prof, MD, PhD
Department of Orthopaedic Surgery, University of Thessalia, Larissa, Greece

Michael Hantes received his medical degree at the Aristotelion University of Thessaloniki, Greece in 1990 and completed his residency in Orthopaedics in 1999, at the University Hospital (Orthopaedic Department) of Ioannina. He completed his training in Germany and United States (1999-2001) where he attended two fellowship programs for Arthroscopy and Sports Medicine. He received his PhD in 2001 at the University of Ioannina. Since 2008 he is faculty member of the University Medical School, University of Thessalia, Greece. He has been on the editorial board of the American Journal of Sports Medicine (AJSM) since 2010 and on the editorial board of Knee Surgery Sports Traumatology and Arthroscopy (KSSTA) since 2011. He is the past president of the Greek Arthroscopic and Sports Medicine Society (2012-2014). He is the current treasurer of the European Society for Sports Traumatology Knee Surgery and Arthroscopy (ESSKA). He is the founder of the Larisa Arthroscopy & Minimally Invasive Center (LAMILC) http://www.ortho-uth.org/lamilc/

His clinical and research interests are reconstructive surgery of the knee, shoulder, and ankle joint with special focus on ligament reconstruction, meniscal repair/replacement, and cartilage repair. He has published more than 55 peer-reviewed manuscripts in international journals and 11 book chapters.

International Faculty

Michael Iosifidis, MD, PhD
G.H. Papageorgiou, Thessaloniki, Greece

Dr. Iosifidis is an Orthopaedic Surgeon at 2nd Orthopaedic Department at "Papageorgiou" General Hospital of Thessaloniki, Greece since 2008. He studied at the Medicine School of the Aristotelian University of Thessaloniki, where he obtained his MD in 1992 and his PhD in 2006 completing his Doctoral Thesis with the title: "Elite athletes' sport activity as a predisposing factor for osteoarthritis". In 2000 completed his residency training programs in Surgery and Orthopaedic Surgery Deps. at Thessaloniki Greece. During 2004-2005 did a fellowship in arthroscopic surgery at Dep. of Orthopaedic Surgery and Center of Sports Medicine, DS Prof FH Fu, and also a research fellowship in basic science research on tendons problems (tendinopathy) and anatomic ACL reconstruction at Mechanobiology Laboratory, School of Medicine and Bioengineering School, Univ. of Pittsburgh. He followed fellowships' programs in arthroscopic surgery at Dep. of Orthopaedic Surgery Prof T Sculco, Hospital for Special Surgery, New York City (2007, 2008), and at TUM Univ. Klinik für Orthopädie und Sportorthopädie, Prof. Dr. A. B. Imhoff, Munich, Germany (2010).

He has published in national and international journals and book chapters, and presented at many national and international meetings. He is member of ICRS, ESSKA and ISAKOS, and served in the boards of Greek Orthopaedic and Arthroscopy Societies. In the last 5 years his research is focusing on MSCs use for cartilage lesions treatment and knee joint kinetics and kinematics.

Konstantinos Malizos, Prof, MD, PhD
University of Thessalia, Larissa, Greece

Konstantinos N. Malizos received his Medical Degree at Aristotle University of Thessaloniki, Greece, in 1978 and completed his residency in Orthopaedics in 1986 at the University Hospital (Orthopaedic Department) of Ioannina. He attended fellowship programmes at the: (a) Jewish Hospital, Orange, CA (05-07/1987), (b) Microsurgery and Surgery-of-the-Hand Center "SOS MAIN" in Strasburg, France (01-07/1988), (c) Clinique Longeraie, Lausanne, Switzerland (07-08/1988), and (d) Orthopaedic Department, Duke University Medical Center, Durham, NC, USA (1989-1991-33 months). He was appointed Lecturer (1991-1994) and Assistant Professor in Orthopaedics (1994-1998) at the University of Ioannina. He has been Professor and Chairman of the Orthopaedic Department, University of Thessalia, since November 1998. He is Founding Director of the Institute for Research & Technology-Thessaly (I.RE.TE.TH)/ Centre for Research & Technology-Hellas (CE.R.T.H), Larissa, Greece, and member of the Executive Council of CE.R.T.H. He is currently member of 16 scientific societies. He was President elect of the European Bone & Joint Infection Society (2005-2007) and he has been board member of EBJIS for 6 years. He is National Coordinator of the National Action Network for Bone and Joint Infection Society (2000-2010). He has been Ad Hoc Reviewer in 9 peer-reviewed journals, International Deputy Editor of JBJS Am, member of editorial board of 3 international peer-review journals, and Ad Hoc Reviewer in National Research Committee of Singapore. He is past Dean at the Faculty of Medicine, University of Thessalia (2007-2009, 2009-2011), past President of the Hellenic Association of Orthopaedic Surgery & Traumatology (HAOST) (2013), and member of the University of Thessalia council.

He has published 205 articles in International "Peer review" Journals and 34 in Greek journals, 151 published abstracts, 60 publications in Transactions and Proceedings of International Meetings and 29 book chapters. He has given more 500 lectures and instructional courses. He has been awarded several prizes from national and international Orthopaedic Societies and he has organized 16 International Congresses, 1 World Congress, 7 International Symposia and 2 European Congresses as Organizing Committee Chairman. He is also the editor of three books: 1. Malizos, KN (ed), Reconstructive Microsurgery. Georgetown, TX: Landes Bioscience, 2003. ISBN 1-57059-650-6. 2. Malizos KN, Soucacos PN (eds), Infections of the Hand and Upper Limb. Athens, Greece: Paschalidis, 2007. ISBN 978-960-399-593-7. 3. Malizos KN, Boukouvala V (eds) (2009). Acts of the Decade 1998-2008. Paschalidis Medical Publications Ltd., Athens, Greece. ISBN 978-960-399-888-4.

International Faculty

Stefan Nehrer MD, PhD
Donau University Krems, Austria

Univ.-Prof Dr. Stefan Nehrer is also Dean of Faculty Health and Medicine at Danube University Krems. Since 2013 he is the Head of the Department Health Sciences and Biomedicine. He studied at the Medical University Vienna where he obtained his MD in 1984 and his PhD in 1999. From 1995 to 1996, he was at the Harvard Medical School in Boston, USA, at Prof Myron Spector where he started his scientific work in cell-based therapies in cartilage regeneration. From 2000 to 2006, he was head of orthopaedic research at the Medical University Vienna and leading surgeon in sports medicine and paediatric orthopaedic. In 2007, he was appointed Professor for Tissue Engineering at Danube University Krems. Over the years, he has continued his research on experimental and clinical applications of chondrocyte transplantation and formed a group for tissue engineering research. Furthermore, his interests focused on mesenchymal cell differentiation and design/implementation of tissue culture bioreactors for automated and controlled manufacturing of cartilage, bone and osteochondral grafts, based on autologous cells and 3D porous scaffolds.

He has published more than 52 peer reviewed and 81 other articles in national and international journals and 14 book chapters. Prof Nehrer has presented at many national and international meetings and he is member of 10 national and international societies. Furthermore, he is since 2012 president of the Austrian Society of Orthopaedics and Orthopedical Surgery. Clinical and scientific interests are preserving knee and ankle joint surgery. Gian Salzmann has published numerous peer-reviewed articles on that topic.

Papacostas Emmanuel, MD
The MIS Orthopaedic Center, Thessaloniki, Greece

Dr Emmanuel Papacostas is the founder of Sports Clinic Thessaloniki and co-founder of The MIS Orthopaedic Center in Thessaloniki, leading private institutes in the field of Sports Orthopaedics in the country. He studied medicine in the Aristotle University of Thessaloniki receiving his medical degree in 1993. He received his orthopaedic specialization diploma in 2004 and works in the private sector ever since. During and after residency he attended several international meetings in the field of sports orthopaedics and cartilage repair being awarded with several national and international fellowships. His work as a young investigator on ankle ligament injuries was awarded with the Einjar Ericsson FIMS Award in 2002. Dr Papacostas is a PhD student in the University of Thessaly since 2012, working on treatment of large chondral and osteochondral knee lesions with ACI.

He became an ICRS full member in 2005 (one of the first two Greeks), being a committee member for two consecutive terms (2010 – 2014). His sports background (national and international level athletics) made him focus on sports injuries prevention and treatment and especially cartilage defects having being inspired by the ICRS, attending Surgical Skills Courses (Vienna, Miami, Rostock). He serves as head team physician of professional football teams and consultant surgeon for several clubs and national associations, having being actively involved in 2004 and 2008 Olympics. He is also a member of the International Ski Federation (FIS) Medical Committee and member of several societies in the field of sports and Sports Orthopaedics. Dr Papacostas is member of the board of the National Arthroscopic Association.

International Faculty

Wolf Petersen, Prof, MD
Martin Luther Krankenhaus, Berlin, Germany

Prof Dr. Wolf Petersen is chairman of the Department of Orthopaedic and Trauma Surgery at the Martin Luther Krankenhaus. Prof Petersen is specialized in knee surgery, especially in cartilage and ligament reconstruction. Over the years, he has continued his research on biomechanical research questions around the knee. Prof Petersen has published more than 150 peer reviewed papers (including award winning papers) and several other articles including book chapters etc. Prof Petersen has presented at many national and international meetings and he performed live surgeries around the world. He is member of 6 national and international societies. He holds several patents for surgical devices.

Prof Petersen was Associate Editor of the Arthroscopy Journal and he is co-editor of Oprat. Orthop. Trauma Surg. He is chairman of the ligament expert group of the German Knee Society (DKG) and board member of the German Knee Society. He is involved in the organization of several German workshops, courses and meeting. This year he is president of the annual congress of the German Knee Society.

Lars Peterson, Prof, MD, PhD
Gothenburg Medical Center, Sweden

Born in Dalarna, Sweden 1936. Studied medicine at the University of Uppsala and Gothenburg .MD in 1966 and Ph.D degree in 1974. Associate Professor of Orthopaedic Surgery in 1980. Worked as an assistant professor at the Department of Orthopaedic Surgery at Sahlgrenska University Hospital 1975-1983. Head of the Department of Orthopaedic Surgery at East Hospital, University of Gothenburg 1983-1988. Head of Gothenburg Medical Center, a sports medicine clinic in Gothenburg 1988- 2007. Appointed professor of Orthopaedics, Sahlgrenska Academy, University of Gothenburg in 2000.

Lars Peterson has worked in basic and clinical research in Sports medicine, Orthopaedics and Cartilage Repair and published in those subjects. Head physician of the Swedish National Ice hockey Team (1967-1990) and of the Swedish National Football Team(1985-1990) and been a member of the Medical Committee of FIFA (1979-) and a founding member of FIFA Medical Assessment and Research Centre(F-MARC ,1994-) He is a founding member of The International Cartilage Repair Society and was president 2001-2002.He is an Honorary Member of the Society (2007)

Lars Peterson has received Honorary Memberships in national orthopaedic societies around the world. He was elected “Member of The Hall of Fame” by the American Orthopaedic Society of Sports Medicine in 2007. He received the “Duke of Edinburgh Prize” in London 2010. He was promoted DOCTOR HONORIS CAUSA at the Medical Faculty of the University of Helsinki, Finland 2010. In 2011 he was promoted DOCTOR HONORIS CAUSA at the Medical Faculty of the Universidad Catolica San Antonio, Murcia, Spain.

International Faculty

Daniël Saris, Prof, PhD
University Medical Center, Utrecht, Netherlands

Daniël Saris (Past President of ICRS) is a specialized knee surgeon. He graduated from University of Amsterdam Medical School in 1992. During orthopaedic residency dr. Saris did a fellowship at the Mayo Clinic in Rochester MN USA under Prof Shawn O'Driscoll of the Cartilage and Connective Tissue Research Laboratory and Prof Kai-Nan An of the orthopaedic biomechanics institute. Dr. Saris completed a PhD thesis in 2002 at the University of Utrecht in the Netherlands that was titled "Joint Homeostasis in Tissue Engineering for Cartilage Repair". It first introduced the now generally accepted concept of joint homeostasis.
In 2000 Daniël joined as staff member in the department of Orthopaedics at the UMC Utrecht. In March of 2010 Dr. Saris was appointed as Professor of Reconstructive Medicine at the University of Twente. Prof Dr. Saris is director of the Biological Joint Reconstruction research program and head of the orthopaedic residency program at the University of Utrecht. As lead investigator he helped establish the first two registered cartilage cell therapy products in Europe and is currently actively pursuing one stage cell based repair in the IMPACT trial and accelerated soft tissue healing for orthopaedic sports medicine.

Tim Spalding Prof, MD
University Hospitals Coventry and Warwickshire NHS Trust, Warwick, UK

Tim Spalding treats all aspects of knee problems from sports related injuries to arthritis. His sub-specialist interest is in reconstructive knee surgery including meniscal transplantation, articular cartilage repair, osteotomy and ligament reconstruction. He is currently co-chairman of the program committee for ICRS 2015 conference in Chicago. Training took place in Oxford and at Royal Hospital Haslar, prior to an arthroscopy and knee surgery fellowship in Toronto, Canada in 1994-1995. He qualified in 1982 from Charing Cross Hospital, London and spent the first part of his medical career with the Royal Marines and the Royal Navy looking after service knees and seeing active service abroad. He joined Coventry in 2000 after 5 years as a Consultant in the armed forces.

He has a busy sports knee surgery practise, runs a knee fellowship program and continues to be very active in teaching and research, pioneering several new techniques. Through his hobby of sailing he was the medical advisor for the Volvo Ocean Race until 2012 and now sits on the Medical advisory committee for the RNLI.

Aspasia Tsezou, Prof, PhD,
University of Thessaly, Faculty of Medicine, Larissa, Greece

Aspasia Tsezou received her Ph.D. in 1991 in Medical Genetics at the University of Athens, Medical School. Since 2001 she is a faculty member at the University of Thessaly, Faculty of Medicine and since 2012 she is Professor of Medical Genetics at the same University. Since 2010 she is Director of the Laboratory of Cytogenetics and Molecular Genetics of University Hospital of Larisa. She is also Head of the Laboratory of Molecular Biology & Genetics in the Research & Technology Centre (Centre for Research and Technology-Thessaly, Greece). Prof Tsezou expertise is on gene expression studies and mechanisms controlling gene expression in multifactorial diseases, as osteoarthritis. Her most recent research interest is on mechanisms involved in host-implant interaction in musculoskeletal infections, with emphasis on host. Her research is supported by several competitive grants from European Commission (FP7 program: "Cooperation") and National sources, as well as by international biopharmaceutical industry. She collaborates as associate member with the FP7 TREAT-OA "Translational Research in Europe Applied Technologies for Osteoarthritis: consortium. Prof Tsezou serves as an advisor for scientific organizations and consults for industry. She is also a reviewer for high-impact scientific Journals and an evaluator for the European Commission since 2001.

International Faculty

Mariana Vlychou, Ass. Prof
University of Thessaly, Larissa, Greece

Marianna Vlychou is a radiologist with interest in musculoskeletal imaging, including diagnostic and interventional MSK ultrasound, CT and MRI. Her main topics of research include arthritis, bone and soft tissue tumours, osteoporosis and cartilage imaging. She is a member of European Society of Skeletal Radiology (ESSR) and International Skeletal Society (ISS). She qualified in 2001 from KAT Hospital, Athens Greece and finished her PhD in 2002. The main part of training in MSK radiology as an honorary fellow took place at Nuffield Orthopaedic Centre, Oxford UK. She also has experience in 3Tesla MRI imaging since 2008 and some basic skills in compositional imaging of the cartilage, after a four weeks visiting practice at the University Hospital of Vienna with Prof Trattnig.

She has full duties as a radiologist in the Department of MRI at the University Hospital of Larissa, Greece and is also very active in teaching and research at the Medical School of Thessaly. Her hobbies include travelling and classical music with emphasis on composers from the modern western period such as Sergei Rachmaninoff and Aram Khachaturian.

Extended Abstracts

Session 1 – Basic Science and Imaging in Cartilage Repair

1.1 Cartilage Special Ultra-Structural Anatomy & Composition – Michael Iosifidis (GR)

Articular cartilage is a living material composed of a relatively small number of cells known as chondrocytes surrounded by a multicomponent matrix without vessels and nerves (fig. 1). Chondrocytes vary in number, size, shape and orientation depending on the zone that they are belonged. They are highly specialized -mesenchymal origin- cells responsible for matrix production, sensing and signalling. The cells are anaerobic and receive their nutrition through diffusion from the synovial fluid and the subchondral bone. In adult humans, cell density is highest at the articular surface and decreases with increasing distance from the surface, as well as with advancing age[1].

Mechanically, articular cartilage is a composite of materials with widely differing properties. Approximately 70 to 85% of the weight of the whole tissue is water and is freely exchangeable with the synovial fluid. The remainder of the tissue is composed primarily of proteoglycans and collagen. Proteoglycans (90% aggrecans) consist of a protein core to which glycosaminoglycans (chondroitin sulfate and keratan sulfate) are attached to form a bottlebrush-like structure (fig. 2). These proteoglycans can bind or aggregate to a backbone of hyaluronic acid to form a macromolecule with a weight up to 200 million. Approximately 30% of the dry weight of articular cartilage is composed of proteoglycans. Proteoglycan concentration and water content vary through the depth of the tissue. Near the articular surface, proteoglycan concentration is relatively low, and the water content is the highest in the tissue. In the deeper regions of the cartilage, near subchondral bone, the proteoglycan concentration is greatest, and the water content is the lowest [2]. Collagen is a fibrous protein that makes up 60 to 70% of the dry weight of the tissue. Type II is the predominant collagen in articular cartilage, although other types are present in smaller amounts[3]. Collagen architecture varies through the depth of the tissue.

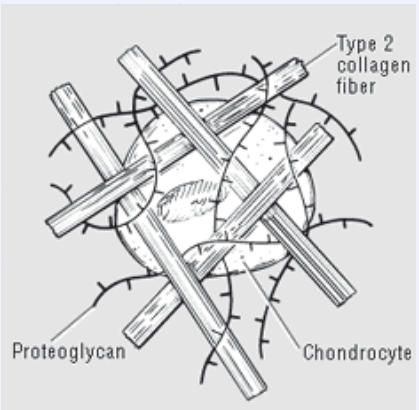

Figure 1. Chondrocytes are surrounded by a multicomponent matrix (mostly water, collagen type II and proteoglycans) without vessels and nerves.

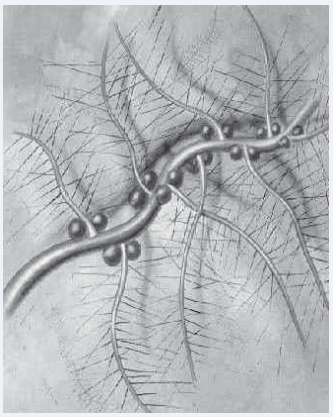

Figure 2. Proteoglycans (90% aggrecans) consist of a protein core to which glycosaminoglycans (chondroitin sulfate and keratan sulfate) are attached to form a bottlebrush-like structure.

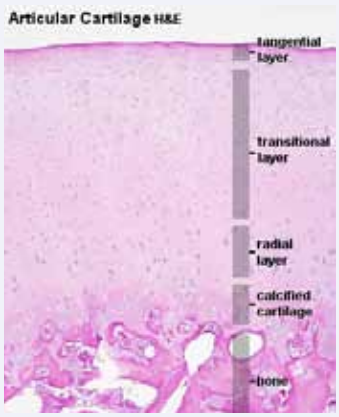

Figure 3. The structure of articular cartilage is consisted of four layers between the articular surface and the subchondral bone: the surface or superficial tangential layer, the intermediate or transitional layer, the deep or radial layer, and the calcified layer (Hematoxylin and Eosin/H&E staining histology picture)

The structure of articular cartilage is often described in terms of four layers between the articular surface and the subchondral bone: the surface or superficial tangential layer, the intermediate or transitional layer, the deep or radial layer, and the calcified layer (fig. 2). The calcified cartilage is the boundary between the cartilage and the underlying subchondral bone. The interface between the deep zone and calcified cartilage is known as the tidemark. Optical microscopy (e.g., polarized light), scanning electron microscopy, and transmission electron microscopy have been used to reveal the structure of articular cartilage[4,5,6]. While each of these methods suggests somewhat similar collagen orientation for the superficial and deep zones, the orientation of fibers in the middle zone remains controversial[7].

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Using scanning electron microscopy to investigate the structure of cartilage in planes parallel and perpendicular to split lines, Jeffery and coworkers have given some new insights into the collagen structure[8]. Split lines are formed by puncturing the cartilage surface at multiple sites with a circular awl. The resulting holes are elliptical, not circular, and the long axes of the ellipses are aligned in what is called the split line direction. In the plane parallel to a split line, the collagen is organized in broad layers or leaves, while in the plane orthogonal to the split lines the structure has a ridged pattern that is interpreted as the edges of the leaves. In the calcified and deep zones, collagen fibers are oriented radially and are arranged in tightly packed bundles. The bundles are linked by numerous fibrils. From the upper deep zone into the middle zone, the radial orientation becomes less distinct, and collagen fibrils form a network that surrounds the chondrocytes. In the superficial zone, the fibers are finer than in the deeper zones, and the collagen structure is organized into several layers. An amorphous layer that does not appear to contain any fibers is found on the articular surface.

The mechanical behavior of articular cartilage is determined by the interaction of its predominant components: collagen, proteoglycans, and interstitial fluid. In addition to the qualitative descriptions given above, quantitative correlations between the mechanical properties of cartilage and glycosaminoglycan content, collagen content, and water content have been established. The compressive stiffness of cartilage increases as a function of the total glycosaminoglycan content. In contrast, there is no correlation of compressive stiffness with collagen content[9].

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1.2 Current Trends in Cartilage Imaging – Mariana Vlychou (GR)

Imaging of the articular cartilage can pose immense challenges not only for early detection of cartilage lesions but also for monitoring surgical repair procedures. Magnetic Resonance Imaging (MRI) is the modality of choice for cartilage imaging and the advent of 3.0T scanners have led to improved diagnostic performance in clinical practice. This presentation aims to review conventional MRI protocols including two-dimensional and isotropic three-dimensional sequences that demonstrate mostly morphologic changes of cartilage and discuss newer techniques, which have been developed to map various cartilaginous parameters such as proteoglycan content, collagen content and orientation, water mobility, and regional cartilage compressibility. These include T2 mapping, T1rho mapping, delayed gadolinium-enhanced MRI of cartilage (dGEMRIC), diffusion-weighted imaging (DWI) and sodium MRI. The aforementioned techniques provide insight into the molecular composition of cartilage and offer a diagnostic path that can be further optimised for early and non-invasive detection of changes related to osteoarthritis.

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1.3 Cell Culturing for Tissue Engineering – Mats Brittberg (SE)

Cartilage has a very low cell to matrix quota and cartilage has no blood circulation. Within the cartilage matrix, cell recruitment is subsequently not possible via the vessel support as seen in other tissues.

A living tissue needs living cells. An injured tissue needs cells for the restoration. Cells need to be recruited with external help, either as a direct internal source, bone marrow derived or from externally handled or externally manipulated. The cells to be used could be divided into four subgroups;

1. True committed autologous chondrocytes
2. True committed allograft chondrocytes
3. Chondrogeneic autologous progenitor cells
4. Chondrogeneic allograft progenitor cells.

The most natural thing would be to use the cartilage own cells; the chondrocytes for the repair. From a small cartilage biopsy, those cells can be isolated and in vitro expanded in monolayer. In monolayer the cells dedifferentiate. However, when the cells are seeded on a 3D material, they re-differentiate.

The culture procedure takes time and to expand those cells, a two stage procedure is needed for a repair; first harvest of cartilage, followed by cell expansion and finally a second operation with implantation.

Due to such reasons, bone marrow mesenchymal stem cells (MSCs) are of interest as those cells can produce both the cartilaginous part as well as the chondral part when to repair osteochondral defects. Bone marrow cells may be used ¨off the shelf¨ as allogeneic stem cells for the repair.

However, it has been shown that chondrocytes and MSCs differentiate and form different subtypes of cartilage, the hyaline and a mixed cartilage phenotype, respectively.

Subsequently, I believe that to be successful in tissue engineering of cartilage injuries, one has to pay attention to both the osseous and cartilaginous part; use of chondrocytes for the cartilage layers and bone marrow stem cells for the bony part. At the same time, increased knowledge is needed of which type of matrices that permits the cells used to differentiate the way that the different layers of cartilage can be produced including the important calcified layer in between the chondrocyte repaired area and the subchondral bone. The aim is to tune the cells by cell recruitment, better cell to cell communication and finally cellular synergy to produce a tissue as near to regeneration as possible.

1.4 Evaluation of Cartilage Repair Tissue – Emanuel Papacostas (GR)

Cartilage repair procedures are widespread techniques used for symptomatic cartilage defects. Evaluating the result after such procedure is a difficult task as many parameters play a role.

On clinical grounds, pain and joint function are the two major components of the assessment. KOOS with its subscales has been validated for cartilage repair. IKDC, WOMAC, SF36 and Lysholm scoring scale are also useful as patient - reported outcome instruments.

Activity rating scales as Tegner - Wallgren activity scale and Marx activity rating scale have been helpful in the evaluation of sports population.

Besides clinical evaluation with the above mentioned outcome instruments, the result is also measured with interventional and non-interventional methods. The former include macroscopic and histological evaluation during second look arthroscopies and the latter MRI assessment.

ICRS proposes ICRS and Oswestry macroscopic cartilage evaluation scores which is validated for the macroscopic assessment after repair (table 1). Histological evaluation should follow ICRS Histology Endpoint Committee guidelines with ICRS I & II quantative scoring systems (table 2).

MRI evaluation of the repair tissue uses the MOCART score

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Criteria	Appearance	Points
I.A	Level with surrounding cartilage	4
	75% repair of defect	3
	50% repair of defect	2
	25% repair of defect	1
	0% repair of defect	0
I.B	100% survival if initially grafted surface	4
	75% survival if initially grafted surface	3
	50% survival if initially grafted surface	2
	25% survival if initially grafted surface	1
	0% survival if initially grafted surface	0
II. Integration	Complete integration with surrounding cartilage	4
	Demarcation border <1 mm	3
	75% integrated, 25% with notable border >1 mm	2
	50% integrated, 50% with notable border >1 mm	1
	0%-25% integrated	0
III. Appearance	Intact smooth surface	4
	Fibrillated surface	3
	Small, scattered fissures and cracks	2
	Small and large fissures	1
	Complete degeneration of graft area	0

Overall assessment and score:			
	Grade 1	Normal	12 points
	Grade 2	Nearly normal	8-11 points
	Grade 3	Abnormal	4-7 points
	Grade 4	Severely abnormal	0-3 points

Table 1 (from Mithoefer et al, Cartilage 2011 2:100-121)

Histological Parameter	Visual Analog Scale Score
1. Tissue morphology (polarized light)	0-100
2. Matrix staining (metachromasia)	0-100
3. Cell morphology	0-100
4. Chondrocyte clustering	0-100
5. Surface architecture	0-100
6. Basal integration	0-100
7. Tidemark formation	0-100
8. Subchondral bone abnormalities/marrow fibrosis	0-100
9. Inflammation	0-100
10. Abnormal calcification/ossification	0-100
11. Vascularization in repair tissue	0-100
12. Surface/superficial assessment	0-100
13. Midzone/deep zone assessment	0-100
14. Overall assessment	0-100

Table 2 (From Mainil-Varlet et al. AJSM 2010;38:880-90)

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1.5. Treatment Algorithms – Alberto Gobbi (IT)

The capability to repair chondral defects remains a challenging goal for orthopaedic surgeons. So far, most clinical approaches have been shown to have limited capacity to treat severe lesions. Current surgical repair strategies vary according to the nature and size of the lesion, and the preference of the operating surgeon. Not all the cartilage lesions that we can find in the articular joints are symptomatic; however an asymptomatic lesion could deteriorate over time and become symptomatic, either as a single or as multiple lesions with a risk of progression to osteoarthritis. Whether or not repairing such defects could be beneficial in relation to the risk of osteoarthritic development is still not demonstrated. As a doctor and surgeon we must treat the disabling symptoms such as pain, locking and swelling however we must also prevent degenerative arthritis. Nowadays the natural history of cartilage degeneration is not completely explained and there is a lack of clinical research to establish a reliable treatment algorithm for all types of lesions.

As is common with ACL injuries having concomitant meniscal injuries and cartilage lesions, such patients may be more important to focus on when deciding upon whether or not to undertake cartilage repair.

A major point of note is that an asymptomatic joint where a detection of cartilage lesions has been done, normally do not need to be treated. As mentioned above, in the untreated lesions of cartilage and subchondral bone show pathological changes and there is risk of a general loss of cartilage volume. Subsequently, such joint damage warrants observations and guidelines being given to the patient

When we have a patient with joint pain, at the time of arthroscopy:
First of all, we need to classify the lesions to:
1. Location and grade (use the ICRS classification system).
2. Size.
3. Morphology/character.

Furthermore, the surgeon should try to find out the etiology of the lesion.
Is it a lesion due to trauma and is it acute or an elderly, chronic lesion?
Is it a developing osteoarthritic lesion? The cartilage lesions should also be evaluated in relation to concomitant injuries such as ligament injuries, meniscal damage and synovitis.

When the surgeon decides upon a treatment for a patient with cartilage lesions, the decision depends on variables such as:
1. Patient's age and activity level.
2. The degree of pain and disability that the patient is experiencing.
3. Location of cartilage lesion, size and depth of cartilage lesions.
4. Co-existing joint pathology such as loss of meniscus, ligament insufficiency, bone loss and malalignment.
5. Other concomitant diseases.

Furthermore we should consider:
1. Body weight or body mass index (BMI)
2. Demand and functional need.
3. Expectation.
4. Ability to comply with rehabilitation.
5. An increased BMI (greater than 30) may have an adverse effect on some cartilage repair procedures.
6. Smoking may impair cartilage repair processes.

The most appropriate treatment option for an individual patient should be based on the pathologic characteristics of the lesion and the patient's symptoms and expectations.

Defects to treat: Patients with symptoms of pain, swelling and catching/locking and isolated cartilage defects ICRS Grade 2-4 > 1cm2 on weight bearing surfaces. Early treatment of those lesions might be important for a more successful outcome.

ICRS grade 2 lesions are often unstable, with partly detached fragments that need to be debrided to form stable lesions. The prognosis for ICRS-2 partial-thickness lesions seems good with diminished mechanical symptoms following

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a simple debridement that involves excision of the unstable cartilage fragments back to smooth edges and leaves the base intact. In the literature, the deep to bare-bone lesions seem troublesome. Lesions that extend through > 50% of the cartilage thickness are classified as ICRS 3. While debridement of unstable edges (as is suggested for ICRS-2 lesions) is suitable for ICRS-3 lesions, further treatment is recommended for these more extensive lesions. All the cartilage lesions that are found in combination with other injuries have to be related to the severity of the other injury/injuries, if the cartilage lesions are suspected of being part of the symptomatology, they are treated the same way as the isolated lesions. If instability is the major symptom from an ACL deficient knee with small size cartilage lesion without subchondral reaction, such a lesion may be left untreated.

If however, the joint besides the ACL injury also has a major lack of the medial or lateral meniscus and cartilage injury with instability and pain, the evaluation and decision is more difficult. Even though the major disability could be due to the cartilage lesion, a repair may be endangered due to the loss of meniscal protection and more or less instability due to the ACL injury leading to fraying and increased pressure on the cartilage repair with dangerous shearing forces; the cartilage lesion treatment may need to be supported by the meniscal and ACL grafting at the same time for maximal protection of the repair tissue.

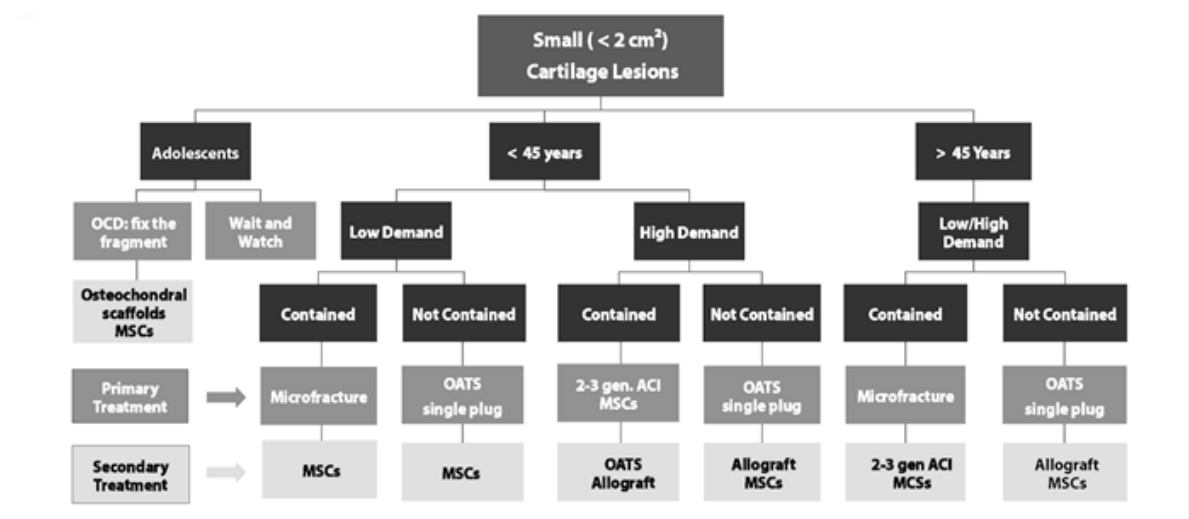
Even though the risk of lesion progression to osteoarthritis is multifactorial, rim stress concentration and altered loading may cause degeneration of adjacent cartilage. Unloading such a lesion is of importance when malalignment exists.

Conclusion

There is not enough research to be absolutely sure of the best treatment for cartilage lesions injuries, isolated or combined. A combination of imaging evaluation together with the evaluation points mentioned above have to be weighed against each other for a final decision. The global economic crisis has also influenced surgeons' possibilities and permission to use new technologies for cartilage repair. Many countries' health systems apply evidence-based approaches for the coverage of their decisions of payment.

Systematic reviews of information about the effectiveness of medical interventions are used as guidelines, however, the cost effectiveness analysis is limited by the paucity of the effectiveness data when comparing chondrocyte/chondrogenic cell implantation with other cartilage repair techniques, especially now cells are used in different scaffolding materials, implantation techniques vary from open to trans arthroscopic techniques and as the rehabilitation differs substantially. Many of the new techniques also lack long-term follow-ups. The orthopaedic surgeon needs to be familiar with both the existing and the newly emerging cartilage treatment techniques in order to best educate patients and meet their expectations for long-term benefits. In this book, you will find clinical approaches to cartilage repair, indications and techniques.

Treatment Algorithm of Chondral Lesions



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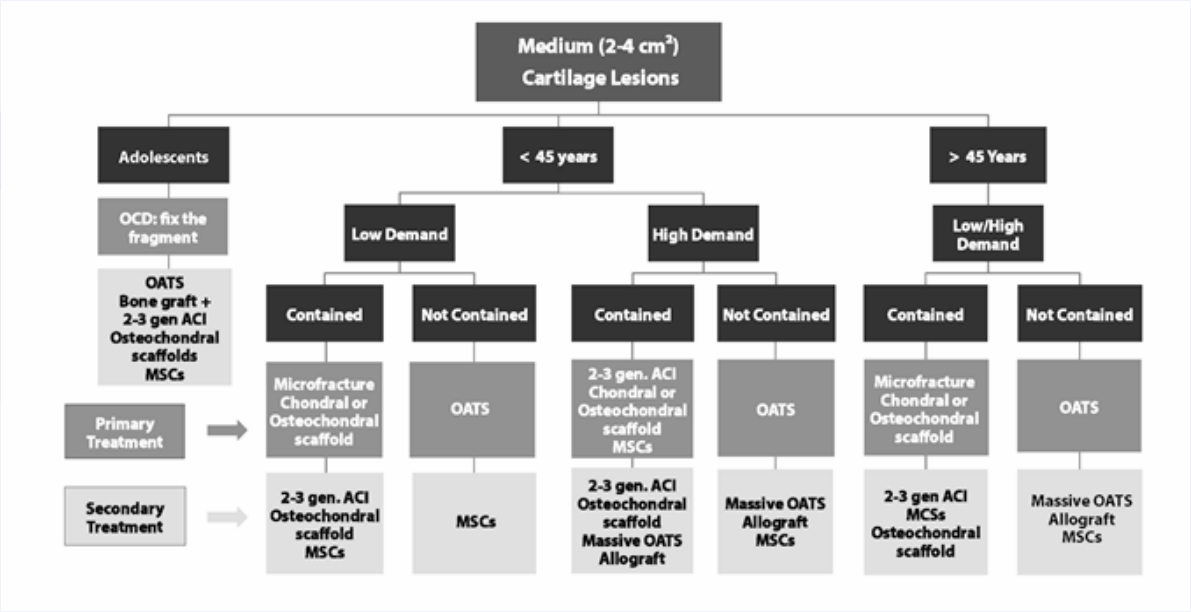

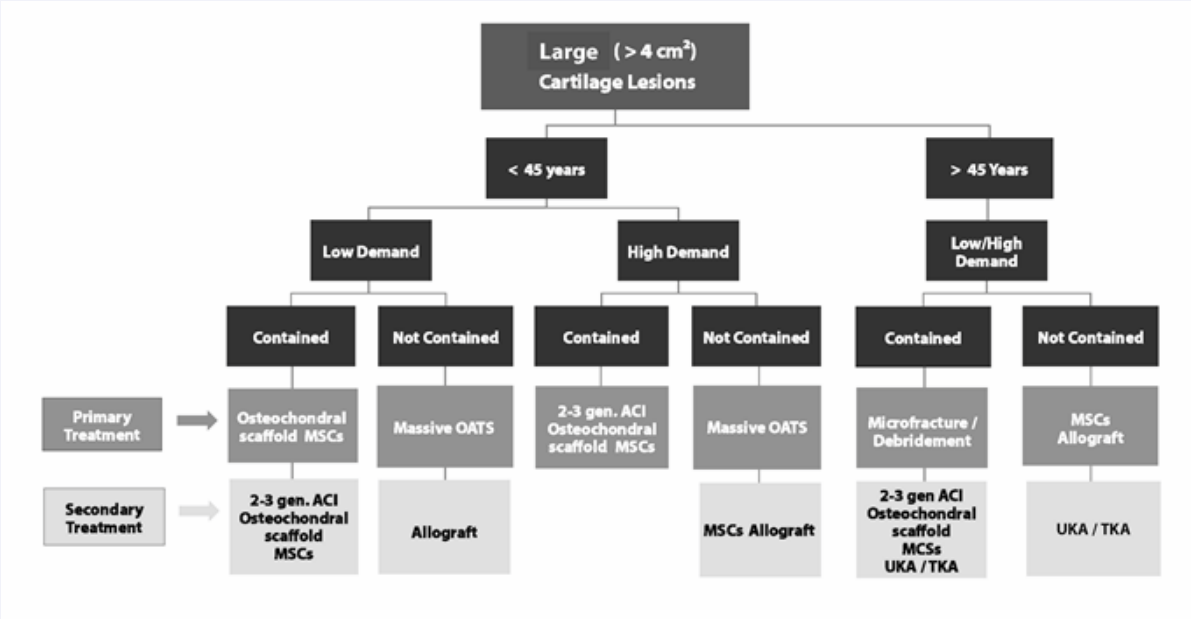

Session 2 – Cartilage Fate after Joint Injury

2.1 Biochemical Changes of the Subchondral Bone after Joint Injury – Aspasia Tsezou (GR)

Joint injuries including meniscal, ligament and joint capsule tears, joint dislocations and intra-articular fractures are known to markedly increase knee osteoarthritis risk. Joint injury triggers a lengthy remodeling process in the cartilage and surrounding tissues that has adverse biomechanical and biochemical implications that encourage joint degeneration. The pathogenesis of post-traumatic osteoarthritis (PTOA) is related to inflammatory processes and alterations to the articular cartilage, menisci, muscle and subchondral bone that are initiated in the acute post-injury phase, with important risk factors being time from injury and age. After knee injury, increased synovial fluid markers of type II collagen and aggrecan turnover, as well as increased concentrations of catabolic enzymes are present at various phases. In addition, elevated levels of pro-inflammatory cytokines (IL-1b, IL-6, IL-8 and TNF-a) are present up to 1 year after ACL injury. Gene expression of matrix metalloproteinase-1 (MMP-1) is up-regulated in articular cartilage, ligament and synovium after ACL injury, while MMP-13 and aggrecanase (ADAMTS-4) are upregulated in the synovium and ligament, suggesting that cartilage changes seen after ACL injury, may be mediated not just by chondrocytes, but also by other tissues of the joint. Cell necrosis occurs beyond the initial area of injury due to apoptotic mechanisms. In meniscal cells, there is differential gene expression between young (<40 years) and old (>40 years) patients. In patients over 40 years with an injured meniscus there is decreased expression of extracellular matrix genes and increased expression of inflammation-related genes, suggesting that in the older population there is a risk of meniscal degeneration and meniscus tear that may accelerate meniscus and cartilage matrix degradation with progressive inflammation, ultimately leading to the development of OA. The poor healing potential of ACL after injury which often results in knee instability, pain and even progressive degeneration of other joint structures has been attributed to specific cell-ECM interactions that may lead to defective fibroblasts self renewal and to their weak regenerative capacity. Recently, non-coding RNAs have emerged as potential biomarkers for cartilage damage associated with ACL injury. Specifically, small nucleolar RNA (snoRNAx) U38 and U48 were shown to be significantly elevated in the serum of patients developing cartilage damage at one year after ACL injury. Furthermore, the serum levels of U38 are substantially elevated in those patients who develop cartilage damage after ACL injury suggesting snoRNA U38 as a serum biomarker for early cartilage damage.

The identification of biochemical markers following a joint injury will help to stratify patients on the likelihood of their developing clinical disease and will guide future interventions, targeting elements of the inflammatory response within the joint, molecular abnormalities related to cartilage matrix degradation, chondrocyte function and subchondral bone remodeling.

2.2 Impact of ACL Rupture on cartilage status: Wait or Repair? – Georgoulis Anastasios (GR)

In the normal knee there is a co-ordination of the function of ACL and PCL providing a good function of the knee in terms of Stability during flexion-Extension and External –Internal Rotation (Screw Home Mechanism). This is after ACL injury disrupted, the “harmonic gliding and rolling” of the Lateral Femoral Condyle after ACL injury is not guaranteed and during pivoting abruptly movements like jerking or displacements can occur with a high risk of concomitant cartilage lesions. Additionally in the daily life there is an Anterior Tibial Translation and an abnormal Tibial Rotation. There are changes in the contact areas of femur and tibia and this can lead also to additional chondral damage. This subluxation of the knee can be seen in MRI with the increased curvature of the PCL measured by the deformity index. To protect the knee from this cartilage damage an anatomic ACL reconstruction has to be performed as early as possible .After an anatomical ACL reconstruction the ATT and abnormal Rotation have to be diminished and the PCL deformity have to be normalized in MRI.

Purpose
The purpose of this study was to test if the PCL deformity is a good sign of in vivo rotational kinematics and joint laxity measures in knees of patients after single-bundle ACLR.

Study Design
Controlled laboratory study.

Methods
At a mean of 18.1 months postoperatively, the PCL index was defined on MRI in 16 patients prospectively followed up after anatomic single-bundle ACLR and in 16 matched controls. The patients were evaluated with 3-dimensional mo-

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tion analysis during (1) descending and pivoting as well as (2) landing and pivoting tasks. The side-to-side difference in tibial rotation range of motion between the reconstructed knee and the contralateral intact knee was calculated. The side-to-side difference in anterior tibial translation was measured with a KT-1000 arthrometer. Linear regression models were used with the PCL index as a predictor of the side-to-side difference in tibial rotation for each task and the side-to-side difference in anterior tibial translation.

Results
The PCL deformity index of the reconstructed knees was significantly lower compared with that of the control knees ($P < .001$). The index was predictive of the side-to-side difference in tibial rotation during both tasks ($R(2) = 0.472$ and 0.477, $P = .003$), with a lower index being indicative of increased rotational laxity. It was not predictive of anterior tibial translation (at 134 N: $R(2) = 0.13$, $P = .17$; at maximum force: $R(2) = 0.009$, $P = .726$).

Conclusion
The PCL deformity index after anatomic single-bundle ACLR using a bone-patellar tendon-bone graft is predictive of rotational kinematics during in vivo dynamic pivoting activities. The results show that the PCL index is correlated with the postoperative ability to control rotational kinematics of the knee joint.

This study provides evidence regarding the interplay between restoration of the native ACL's anatomy and the PCL's appearance and suggests that the effective restoration of tibiofemoral alignment after ACLR that is reflected in the PCL deformity index translates into better functional outcomes as measured by tibial rotation during pivoting activities.

Not just an ACL reconstruction is indicated to protect the knee from further cartilage damage but a very anatomic one which restores the anatomy , biomechanics and MRI appearance of the knee joint.

2.4 Meniscus Replacement for Cartilage Protection – Tim Spalding (UK)

Introduction
The anatomy and microstructure of the menisci allow effective distribution of load across the knee. Menisectomy results in an altered biomechanical environment and is a potent risk factor for osteoarthritis. Despite the trend towards meniscal preserving surgery, many tears are irreparable and many repairs fail. Meniscal allograft transplantation has been primarily been performed for pain in patients with a history of meniscectomy. Numerous case series have reported significant improvements in patient reported outcomes following surgery, and the procedure is no longer considered experimental. There are still however many unanswered questions.

Over 130 meniscal transplantations have been performed at University Hospital Coventry and Warwickshire NHS Trust and current research is directed at determining the chondroprotective effect of meniscal allograft transplantation.

Meniscus structure and function
The menisci are two fibrocartilage structures that sit between the tibio-femoral articulations of the knee. In the axial plane they are crescentic and cross-sectionally they are wedge shaped.

It is generally accepted that the primary role of the menisci is load distribution. The concave upper surface of the menisci and flat lower surface increase the congruency of the otherwise incongruent tibio-femoral joint. In the loaded knee, the lateral meniscus transmits 70 per cent and the medial meniscus 50 per cent of the load through the knee. Biomechanical studies have shown that meniscectomy decreases the tibio-femoral contact area by 50 to 75 per cent and increases the peak contact pressure by 200 to 300 per cent. The menisci have also been shown to provide secondary constraint to the knee. Further roles proposed include joint lubrication and proprioception. Shock absorption is commonly stated as a function of the menisci, although this is probably not true.

Consequences of menisectomy
The consequences of meniscectomy have been well documented. It is thought that damage to the meniscus results in changes to the biomechanical and biochemical environment of the knee, which may lead to OA progression in sus-

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ceptible patients. Baratz et al. performed a cadaveric study showing that total meniscectomy decreased tibio-femoral contact area by 75 per cent and increased peak contact stresses by 235 per cent. Similar findings have been reported in other studies. More recent studies have shown local biomechanical changes to the articular cartilage and knee joint following meniscectomy. These changes in the articular cartilage have also been seen after partial meniscectomy. Animal model studies have also reported articular cartilage softening and fibrillation, swelling, fissures and cytokine mediated responses following meniscectomy.

Meniscal allograft transplantation

In response to reports of the clinical consequences of meniscectomy, meniscal substitution and replacement began to emerge in the 1980's. Canham and Stanish reported the results of using a Teflon net as a meniscal substitute in dogs[63]. Three years later a study with successful implantation of medial meniscal allografts in dogs was reported. This showed for the first time that a completely detached meniscus could be implanted successfully. In 1989, Milachowski, Weismeier and Wirth reported a case series of meniscal transplantation in 30 sheep and then 22 patients. The first human meniscal allograft transplantation was performed by this group in May 1984. The authors concluded that meniscal allograft transplantation was a reasonable procedure and observed no adverse immunological reactions.

Since the first reported human meniscal transplantation, there have been numerous case series reported in the literature. A recent systematic review reported over 1600 cases, although the total number performed is likely to be many more. In 2003 it was estimated that over 4000 transplantations had been performed in the USA alone, with a yearly rate approaching 800. It is primarily performed for relief of compartmental pain in patients with a history of meniscectomy. These symptoms are thought to result from a biomechanically 'overloaded' knee]. The patient reported outcomes from case series studies have been encouraging, but no randomised controlled trials (RCT) have been performed. A recent systematic review reported that the mean weighted Lysholm score (score 0 to 100, with 100 being the best) improved from 55.5 to 82.7 pre-operatively to final follow up (weighted average follow up of 4.5 years) respectively. They also found that all other functional outcome measures were improved at final follow up, which has been supported by other systematic reviews. Pooled complication rates for isolated meniscal transplantation have ranged from 6 to 11 per cent, although this is likely to be an underestimate. The average graft survival time has been reported to be between 10 and 16 years. Disease transmission and immune rejection are also possible, although extremely unlikely complications. The meniscal allograft is thought to be immuno-privileged and there has only been one reported case of possible rejection.

Chondroprotection

It has been stated that the most important question in meniscal allograft transplantation is whether it can preserve knee cartilage. This question is also vital to the scientific plausibility that it is an effective treatment. Despite this, there is currently little definitive evidence that this is the case. Sekiya et al. compared joint space changes following meniscal allograft transplantation to the contralateral limb, finding no significant differences between the groups. Other studies have found no joint space loss following meniscal transplantation, but others that have found statistically significant joint space losses.

Biomechanical studies support the chondroprotection hypothesis as they have shown that meniscal transplantation improves peak contact stresses and total contact area compared with meniscectomy. One study found that peak contact stresses in the native and transplanted knee were not significantly different. Animal model studies have also demonstrated chondroprotective effects of meniscal allograft transplantation compared to meniscectomy. Szomor et al. reported significant protection of the articular cartilage at four months in the meniscal transplantation group of a sheep model compared to the meniscectomy group. Kelly et al. performed a similar study using MRI outcome tools and found a significant decrease in articular cartilage degeneration compared to the meniscectomy group.

Conclusions

The shape and microstructure of the menisci allow it to effectively distribute load across the knee. Meniscal tears and meniscectomy result in an altered biomechanical and biochemical environment, and are potent risk factors for OA of the knee. Meniscal allograft transplantation has been performed for over thirty years and multiple case series' have consistently reported improved patient reported outcomes, with a reasonable complication and survival rate.

References available on request

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2.5 Cartilage & Meniscal Status after ACL Injury – Konstantinos N Malizos (GR)

Knee injury with ligament tears, and the subsequent symptomatic instability have been associated with a variable percentage of symptomatic knee osteoarthritis development(~50%) 10 - 15 years from initial trauma.Cruciate ligament tears are rarely isolated, and are commonly combined in more than onethird of the cases with rupture of the menisci, bone marrow edema, cartilage injuries, subchondralplate ruptures or compressive fractures. ACL tears commonly occur with a high incidence in adolescents & young physically active adults in sports & recreational activitythat involves pivoting. More severe ligamentous instability with concomitant meniscal and chondral damage carry much higher risk for less optimal outcome after reconstruction.

With the majority of acute ACL tears in individuals younger than 30 years, early onset osteoarthritis with pain and disability would be anticipated in their forties and fifties. Delays in the repair of joint instability after knee injuries are often neglected. Individuals with an unstable knee who maintain a high level activity will develop a meniscal tear and/or cartilage damage in more than 90% of the cases, triggering the "cruciate ligament injury cascade" which leads to osteoarthritis prematurely. A number of risk factors and mechanisms are responsible for further intra-articular damage which may accelerate joint degeneration. Following the ligament rupture the tibio-femoral contact location is shifting and the cartilage deformation increases with more wear and tear from increased shear forces. The chondrocytes' response is an altered cell metabolism, a higher rate of cell death and apoptosis in combination with disruption of cartilage matrix. Intra-articular injuries and bleeding also activate inflammatory pathways and synovial reaction, with increased cytokine excretion, IL-1β, IL-8, IL-6, PGE-2, TNF-α, and nitrus oxide, leading to a catabolic effect of link proteins by Metallo-proteinases (MMPs). MMP-13 is very strong against type II collagen and causes degradation of the collagen network of the articular cartilage. An elevated expression of catabolic markers such as IL-1β, ADAMTS, MMP-1, MMP-9, MMP-13 and NFκB2 has been revealed in patients with meniscal and cruciate ligament tears, leading more likely to arthritis. There is an urgent need to devise effective strategies to prevent knee ligament injuries and reduce the risk of osteoarthritis, through understanding of the pathogenesis of the disease at the cellular and the molecular levels both in the acute post trauma phase and the subsequent long clinically asymptomatic chronic phase with minimal structural changes, until the establishement of the final symptomatic phase with pain and dysfunction. These findings are suggesting that factors responsible for articular cartilage homeostasis combined with appropriate surgical treatment may yield more effective therapies for post-traumatic OA. Apoptotic, inflammatory and catabolic activity is associated also with time from injury. Cell death and the breakdown of the extracellular cartilage matrix are gradually degrading the hyaline cartilage leading to joint degeneration.

2.6 Ten to twenty years follow-up of ACI with ACL reconstruction – Lars Peterson (SE)

Articular cartilage lesions have been reported to occur in between 40 to 70% in acute and chronic Anterior Cruciate Ligament(ACL) injuries. Does ACL reconstruction prevent or delay further deterioration of articular cartilage or injuries to it to progress into posttraumatic osteoarthritis? Several studies have been published with different results but no convincing findings have been presented for that ACL reconstructions in early or late stages after the injury have any preventive effect on progression into osteoarthritis. The question is: Does ACL reconstruction in combination with Autologous Chondrocyte Implantation stop or delay the progress into Osteoarthritis over time?

Since 1987 ACI has been in clinical use and the first results were published in 1994 showing G/E results of injuries on the femoral condyles and biopsies showing "hyaline –like" microscopic repair. During the following years the indications were widened and the concept of addressing all background factors to create an optimal environment(or homeostasis) for the short and long term survival of the repair tissue was established. Correction of Instability (ACL,PCL,MCL,LCL) , Patellar Malalignment and Instability as well as KNEE VARUS and VALGUS Deformities, Bone Defects and Pathology, Meniscus Deficiency or any Background Factors of Importance are considered mandatory.

The background to the actual surgical ACL technique used.
In a biomechanic study of the strength of the medial and lateral retinaculae initiated after a proximal dislocation of the patella in a teenage boy where the patellar ligament was avulsed at the apex along with one bone fragment from the medial proximal margin of the patella and one fragment from the lateral margin of the patella. In testing a cadaver preparation in loading over 50 N was needed to rupture the medial and lateral longitudinal retinaculae. In dissection studies we also found that the anastomosis connecting the superior and inferior medial genicular arteries running in

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the spatium between the medial longitudinal retinaculum and the patellar tendon(ligament) gives vascular supply to those structures. In a following study we used the medial retinaculum as vascularized autologous augmentation of the acute repair of ACL showing that the subjective and objective results were superior to repair alone. These results inspired to an other biomechanic cadaver study dissecting the medial third of the patellar ligament en bloc with the medial longitudinal retinaculum preserving the connective tissue and arterial supply at the base in between and with bone from the patella in the proximal ends of the bloc. This vascularized anatomic graft was replacing the posterolateral bundle with the medial third of the patellar ligament, the connective tissue with the vascular supply replacing the intermediate bundle and the medial retinaculum replacing the anteromedial bundle. The anatomic position of the bundles was achieved by using oval drillholes in the tibial and femoral anatomic ligamentous landmarks. The graft ends with bone were fixed by pullout sutures over a 4 hole botton on the outer lateral cortical bone of the femur and tied after conditioning of the graft in 90 degrees of knee flexion for the anteromedial / intermediate bundles and the posterolateral bundle tied close to extension. On testing the stability there was no sagittal or rotational instability. The first patient was operated with this graft in 1983 and after preparing the graft the tourniquet was released and there was a significant bleeding from the free ends of bone and soft tissue showing a pres erved vascularization of the graft. All patients were operated with this ACL technique and ACI in a one step procedure. A brace was used with 0-60-90 degrees for the first 3 weeks and then 0 – 130 of motion for the following 3 weeks. Weightbearing was adapted to the actual cartilage surgery, but in most case full WB was reached at 12 weeks along with physiotherapy and functional training.

Results

The ACI/ACL results have been reported at 2-9 years, 5-11 years ,10-20 years follow ups. The results have been durable and at the last follow up 82% were G/E. No sagittal or rotational instability reported. Macroscopic assessment of the repair tissue according to ICRS after ACI/ACL showed a filling of 10.9 (8-12)points out of 12 maximal evaluating FILLING 4 p,Integration 4p and surface appearance 4 p.Tegner - Wallgre´ns Activity scores(max 15 p) showed 7-9 points.Biopsies taken showed “hyalin-like” microscopic appearance in 80%.

In summary ACI/ACL patients have been followed up repeatedly up to 20 years and over 80% reported G/E results with objectively supported data . Ninety two % wanted to have the ACI/ACL operation again .

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2.7 ACL & PCL Reconstruction – Wolf Petersen (DE)

ACL Reconstruction
For anatomical ACL reconstruction the semitendinosus tendon is harvested via a 3 cm skin incision approximately 1 cm distal and medial to the tibial tuberosity. A four stranded tendon graft is prepared with a minimum length of 6.5 cm. Alternative grafts for this technique are the patellar tendon, quadriceps tendon, and allografts.

The femoral tunnel for the ACL graft is drilled via a deep anteromedial portal under arthroscopic control. For precise placement of the guide wire a specific offset aimer is used. For drilling the knee must be flexed more than 110 °. Landmarks are the intercondylar line and the cartilage-bone interface. The position of the guide wire is always controlled by the medial portal (medial portal view). The guide wire is overdrilled with a cannulated drill (4.5 mm when a flip tack is used). The drill diameter for the 30 mm long blind tunnel is choosen according to the graft diameter. A gentle tunnel preparation may be achieved with the use of dilators. At the tibia, the anterior horn of the lateral meniscus is used as a landmark in the absence of ACL stump. The guide wire is first overdrilled with a 6 mm drill. Slight adjustments to the tibial tunnel location can be archieved when the guide wire is overdrilled eccentrically with a larger drill. At the femur an extrakortikal fixation technique with a flip button is preferred. At the tibia, a hybrid fixation with absorbable interference screw and button is used.

PCL Reconstruction
For PCL reconstruction hamstring grafts are used. The tendons are folded to a 4 or 5 stranded graft with a minimum length of 10 cm. The femoral tunnel for the graft is drilled via a deep anterolateral portal under arthroscopic control. For drilling of the tibial tunnel a posteromedial portal is needed. The tibial insertion of the posterior cruciate ligament is debrided with a shaver and a specific raspatorium. For tibial tunnel placement a specific closed aimer is used (KARL STORZ Tuttlingen) and a K wire is placed in the center of the tibial insertion. This K wire is overdrilled with a cannulated drill with a diameter according to the graft size. After femoral fixation the graft is tensioned in 90° flexion with 80 N. At the femoral and tibial side a hybrid fixation is performed with a button (Flipp tack) and a resorbable interference screw. If there are any signs for posterolateral instability a posterolateral corner reconstruction is performed before tensioning and fixation of the PCL graft.

Session 3 – Treatment Options for Cartilage Lesions

3.1 Microfracture & Autologous Matrix-Induced Chondrogenesis (AMIC) – Hantes Michael (GR)

It is known that cartilage lesions do not heal spontaneously and may predispose the joint to the subsequent development of secondary osteoarthritis. A variety of surgical techniques that aim for resurfacing and regenerating of the articular cartilage are available today. The Autologous Matrix-Induced Chondrogenesis (AMIC) technique combines the microfracture method with matrix-based techniques that utilizes a collagen (cell free) membrane to serve as a scaffold for new cells, allowing effective reconstruction of a damaged cartilage surface. Similar to other bone marrow stimulation techniques, after microfracture, stem cells are simply released into the joint rather than being contained at the site of the cartilage defect. The AMIC technique developed by Behrens, in order to overcome this potential limitation.

Indications for AMIC are symptomatic lesions grade 3 and 4 according to ICRS, with a defect size between 2 and 4 cm2 situated at the femoral condyle, the patella or the trochlea. Exclusion criteria are advanced osteoarthritis, kissing lesions, underlying rheumatic disease, massive overweight (BMI>35) and deviation of the mechanical axis to the affected compartment. Osteochondritis dissecans, is also another indication for the procedure. In these cases, filling of the osseous defect with impaction of autologous bone graft (harvested from the tibial head, femoral condyle or iliac crest) is performed as the first step, and then the membrane is attached on the defect. The scaffold is like a sponge, which holds the blood clot and progenitor cells within the defect, inducing haemostasis and protecting the underlying tissue. Commonly used membranes are, Chondro-Gide (Geistlich Biomaterials, Wolhusen, Switzerland), consisting of a bilayer porcine collagen I/III matrix, and Hyalofast (Fidia Advanced Biopolymers Laboratories, Abano Terme, Italy) a non-woven pad made of Hyaff-11, which is a semi-synthetic derivative of hyaluronic acid.

An all-athroscopic or a mini-open approach can be used for the procedure. As a first step, a standard arthroscopic examination is performed using standard anteromedial and anterolateral portals in supine position. After comple-

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te inspection of the joints and assessment of the chondral defect, including size, depth, and location, the defect is carefully debrided down to the bed with a curette until healthy-looking bone and healthy cartilage edges appeared. Subsequently, microfracture is performed according to the technique described by Steadman with a microfracture awl. Alternatively, a 1.1-mm K-wire can be used to perforate the subchondral bone since many recent studies reported that the subchondral stroma is better reached by drilling.

For an open approach, a mini arthrotomy is performed according to the location of the defect. A template is used to determine the size of the defect and to facilitate trimming of the membrane in terms of size and shape. Commercially available fibrin glue or suturing is used for fixation of the membrane. The stability of the matrix, is assessed by extending and flexing the knee. No drains are used in order to avoid the membrane from being dislodged from the lesion. For an arthroscopic procedure, placement of the membrane should be performed after microfracture under dry arthroscopic conditions. The size of the defect is assessed by a circular sharp punch and corresponding numbers of circles are cut from the rectangular membrane in order to fill the defect.

Finally, the so-called AMIC plus technique, have been developed by some surgeons which is an AMIC procedure combined with platelet-rich plasma (PRP) gel in order to enhance the healing response. The PRP gel is applied beneath the membrane. PRP has already shown to have a positive effect on cartilage and probably could enhance the induction of chondrogenic cells. However, there are no comparative studies to test this hypothesis. Postoperatively, patients use crutches to maintain partial weight-bearing for 6-8 weeks. Passive range of motion begin one week postop from 0 to 90° for 3 weeks, and then gradually increased. Unrestricted weight-bearing and range of motion is permitted after 8 weeks. Participation in sports activities is permitted after 12-14 months.

Since cell-free scaffolds, and subsequently the AMIC technique, have been developed recently, few results and clinical studies have been published, with a short-term follow-up. Furthermore, the AMIC technique has not been evaluated with randomized controlled trials. According to published studies the IKDC, Lysholm, Tegner, KOOS, and VAS scores improved significantly when preoperative and postoperative values were compared at 12 and 24 months postoperatively. Adverse reactions to the matrix were not observed. The MRI follow-up showed that results are inconsistent, with some patients demonstrating good defect filling while others demonstrated no filling or hypertrophy. Integration to the border zone was generally good, but abnormalities of subchondral bone and lamina were common.

Definitely, the AMIC procedure is a cost-effective option for the surgeon treating cartilage defects. It is easy to apply, and good to excellent results are expected in the vast majority of cases. However, prospective randomized trials are needed to compare the results of the AMIC procedure to microfracture alone and MACI.

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3.2 How Helpful is the Abrasion Technique for a Quick Return in the High Level Athletes? – Emanuel Papacostas (GR)

Cartilage defects in athletes are historically considered as a career ending injury. After the introduction of cell based treatment options and the improvement of other techniques (mosaicplasty, AMIC, MSCs etc) the results, in terms of returning to sports, are improved. Treatment algorithms have been already proposed for the treatment of chondral and Osteochondral defects in general population and football players, from ESSKA, ISAKOS and ICRS groups.

Besides considering the indications and deciding for the proper procedure, high level professional athletes and their background consider time the major influencing factor. That is the reason for which suboptimal treatment options are usually undertaken at this level. There is no doubt that staying with the indications and personalizing the treatment will result in improved outcome, return to the same level of participation and maintenance of the function for longer period. On the other hand patient prefferal has to be heavily considered as long as he/she is thoroughly informed. In this matter, arthroscopic debridement is, maybe, the most common first line treatment option for high level athletes. The advantages are listed below:

- Low morbidity
- Symptoms relieving procedure
- Faster rehab and return to sports
- Low cost
- Does not “burn bridge”

As disadvantages one can consider
- Short term clinical improvement
- Need for second operation

The use of debridement of cartilage lesions for the high level athlete is therefore a reality, with results that cannot be reproducible. We believe that this option is valid and can help this special population but, of course, cannot be supported by hard scientific evidence. In general terms abrasion and removal of loose cartilage bodies and smoothening of fibrilled cartilage ends can relieve mechanical symptoms of locking and catching. Bone oedema is usually the cause of discomfort for these patients, and if this is present, and arthroscopic debridement is chosen for treatment, retrograde drilling could be considered as decompression method.

3.3 OATS – Osteochondral Autograft Transfer System – Wolf Petersen (DE)

Introduction
Osteochondral grafting is a treatment option for stage IV osteochondral defects of the talus. Several techniques for osteochondral grafting have been described in the literature . All techniques have in common that cylindrical osteochondral grafts are harvested from the trochlea or notch to reconstruct a defect in the load bearing zone of the femoral condyles or at the trochlea.

Most authors use the femoropatellar joint as donor site for the osteochondral grafts. In our experience the harvesting of osteochondral grafts from the femoropatellar joint may be associated with donor site morbidity such as anterior knee pain. Several other authors could confirmed and described significant donor site morbidity after graft harvesting from the femoropatellar joint. An alternative donor site for osteochondral grafts with low donor site morbidity are the posterior aspects of the femoral condyles. With this graft harvesting technique good to excellent clinical results with no donor site morbidity have been described for the treatment of osteochondral defects of the knee. A disadvantage of this technique was that the cylindrical grafts were harvested with the patient in prone position. That means, the patient has to be turned around with new skin disinfection and draping to implant the graft into the defect.

Technique
For osteochondral cylinder harvesting and defect preparation the Diamond twin system® (Karl Storz, Tuttlingen, Germany) is used. This system consists of various diamond coated bone cutters and extractors. The mutually adjusted

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sizes of the bone cutters allow a press fit implantation to the defct. The donor cylinder is 0.05 mm larger in diameter than the defect cylinder.

The operation starts with harvesting of the osteochondral donor cylinder from the posterior femoral condyles. To reach the donor site the patient is placed in a prone or lateral position. After application of a thigh tourniquet the skin was disinfected and draped.

The skin incision is performed in the popliteal fossa. The fascia is opened with a scissor and the posterior joint capsule of the lateral femoral condyle was exposed with four Langenbeck retractors after blunt preparation with the index finger. The lateral gastrocnemius must be pushed to the side with a periosteal elevator. Then, the capsule is opened with a scalpel and a Hohmann retractor is placed medially and laterally behind the femoral condyle. The cartilage is cutted with an appropriately sized extractor and then the bone is cutted with a diamond coated hollow grinder (diamond bone cutter). The size of the donor cylinder shold be determined on the preoperative MRI. The largest grinder used for this technique has the following dimensions: External diameter 16,3 mm, internal diameter 15,25 mm. The smallest grinder has an external/internal diameters of 8,35/7,45 mm. The hollow grinder is connected to a conventional drilling machine by an adapter. To prevent heat damage to the bone the cutter has to be cooled permanently with NaCl. The depth of the donor cylinder should be between 15 and 20 mm according to the depth of the defect. The depth of the cylinder can be read on the tiller. The donor cylinder is finally extracted with an appropriately sized extractor by a quick rotation.

After rinsing the donor defect is filled with a cylindrical bone graft substitute optimized for use with the instruments (Syntricer® - biodegradable β-tricalcium phosphate ceramic, Karl Storz, Tuttlingen). The cylindrical bone graft substitute is gently pressed into the donor defect until the cylinder surface is one or two millimeters below the surrounding cartilage. The donor site is rinsed with NaCl and the capsule is closed by resorbable sutures. If a second cylinder is needed, the same procedure has to be repeated at the medial side. In most cases two larger cylinders are used. If a third cylinder is needed, it must be harvested from the femoral trochlea or the intercondylar notch.

To reach the medial or lateral femoral condyle a small medial or lateral approach was performed. The defect is prepared by excising the necrotic sequestrum with a bone cutter one size smaller as used for harvesting of the donor cylinders. The technique for defect preparation is the same as for harvesting the donor cylinder. First the cartilage is cutted with the extractor. Then the defect cylinder is excised with the diamond coated grinder and the extractor. The depth of the defect hole has to be measured and in case of a mismatch the donor cylinder should be shortened. After accurate defect preparation th donor cylinders are pressed gently into the recipient site to fill the defect.

Rehabilitation protocol
The patients are mobilized with partial weight-bearing for six weeks and with early physiotherapy to both the ankle and the knee. Unprotected weight-bearing is allowed after a radiological control. Jogging is allowed after six months and impact activities after screw removal after one year.

Conflict of interest: Dr. Petersen reports personal fees from Karl Storz, personal fees from Otto Bock, personal fees from AAP implants.

Session 3 – Treatment Options for Cartilage Lesions

3.4 The Four generations of ACI; Modes of action, techniques and results – Mats Brittberg (SE)

In October 1987, 24 years ago, the autologous chondrocyte implantation (ACI) technique was used for the first time to treat patients with chronic disabling symptoms of the knee joint with cultured cartilage cells from their own cartilage. The first 23 patients were presented in the New England Journal of Medicine in 1994. The technique appeared to be most successful in patients that had injuries on the femoral surfaces producing a single, localized deep cartilage lesion. Since then more than 30000 patients worldwide have been treated with this or similar techniques and several new generations of ACI have appeared. A generation can be referred to different stages of successive improvement in the development of a technology.

- The first generation of autologous chondrocyte implantation (ACI) consisted of cells in suspension injected in under a sutured membrane of a living tissue; periosteum.
- The second generation ACI was an exchange of the periosteum with a collagen inert membrane
- The third generation of ACI is the technology where cells are grown prior to implantation in or on different scaffolding materials.
- The fourth generation of ACI are either one stage operation with cartilage fragments either autologous and allogenous implanted in fibrin glue under a synthetic membrane or directly isolated chondrocytes mixted with directly isolated mesenchymal stem cells.

The ACI was the first example of musculoskeletal tissue engineering used clinically and it has forced the surgeons to learn more about the healing processes of living tissues. An implantation of a high number of living cells in suspensions or in different scaffolds has led to collaboration between surgeons, bio-, material and chemical engineers to improve the cells viability, integration and matrix production in order to induce repairs with high quality. We have during those 27 years learned that cell repair involves a lot of difficult processes that may influence the final results. Surgeons need to know that cartilage repair with cell implantation is a slow process with a progressive maturation of the repair tissue and that such a maturation is influenced by mechanical forces such as by malalignment and instability. Also smoking may impair the cartilage repair. We have shown that with ACI generation I up to 20 years post-surgery (mean 12.8 years) still 74% of the patients are in the category of good-excellent. In 97 biopsies, the quality of the repair tissue after ACI showed a hyaline like or a mixture of fibro hyaline and hyaline like tissue. The normal distribution of the cells was not restored. However in some cases the normal columnar distribution was restored over time. Similar findings have been shown also from other groups where by time a maturation of the repair is seen with a significant zonal stratification. The long-time follow up of the different ACI generations is discussed in relation to clinical, radiologic and histological findings.

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Session 3 – Treatment Options for Cartilage Lesions

3.6 Allogeneic MSCs for cartilage repair: our experience with cellular communication – Daniël Saris (NL) (De Windt TS, Vonk LA)

The technovolution of articular cartilage repair procedures has resulted in a variety of cell-based therapies that use both autologous and allogeneic mesenchymal stromal cells (MSCs). As these cells are increasingly available and show promising results both in vitro and in vivo, cell-based strategies, which aim to improve ease of use and cost-effectiveness, are progressively explored. The use of MSCs in cartilage repair makes it possible to develop single-stage cell-based therapies. However, single-stage procedures rely on one intervention, which will limit cell sources to fraction-concentrates containing autologous MSCs such as bone-marrow aspirate concentrates or the stromal vascular fraction of adipose tissue. In recent years, we have focused on using cellular communication through cocultures between chondrocytes and allogeneic MSCs to provide reproducible cartilage regeneration and allow a single-stage approach. Indeed, when MSCs are mixed with chondrocytes, reproducible chondrogenesis has been shown.[1,2] The idea behind this coculture approach is that allogeneic MSCs can stimulate patient own cells to produce cartilage. Others have found that when mixed with chondrocytes, MSCs stimulate their proliferation while slowly disappearing from the culture (chondroinduction).[3,4] We have recently confirmed these findings in vitro and found direct cell-cell contact and communication through gap junctions to be a key player in this system. A preclinical model that we explored earlier, already showed autologous chondrocytes with their pericellular matrix (chondrons) and allogeneic MSCs mixed at a 10:90 ratio capable of regenerating cartilage in both a small and large animal model.[5] This has supported he initiation of a first in man study that is currently ongoing (NCT02037204). In the Instant MSC Product accompanying Autologous Chondron Transplantation (IMPACT) trial, a cell combination of 10% autologous chondrons and 90% allogeneic MSCs are mixed in a fibrin glue scaffold and applied to a focal cartilage defect within one surgical procedure. Recently the inclusion of all 35 patients has been completed. To date, no serious adverse events have been encountered as monitored by an independent rheumatologist and data safety monitoring board. The first patients that have completed one-year follow-up (n = 4) have all shown significant improvement in clinical outcome scores as well as in MRI evaluated defect fill. Second-look arthroscopy in these patients showed complete defect fill and histology indicated good integration with the subchondral bone and positive proteoglycan staining.

IMPACT video Youtube

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Session 5 – Reducing The Joint Surface Loading – Osteotomies

5.1 Minimally Invasive Technique for High Tibial Osteotomies: The Taylor Spatial Frame
Konstantinos Malizos (GR)

Introduction
The success of High Tibial Osteotomy (HTO) in patients with medial compartment osteoarthritis (OA) is known to be depended on the accuracy of Mechanical Axis correction. We present the short term results of gradual correction of HTO in OA patients with varus deformity using a minimal invasive technique with the use of the Taylor Spatial Frame (TSF).
Materials and Methods: Fifty-Five patients with medial compartment arthritis of the knee and varus mal-alignment underwent an HTO using a TSF and gradual correction of the mechanical axis. Mean age was 51 years (range:40-58y). A low energy osteotomy was performed with a small 2 cm skin incision distal to the tibial tubercle in 46 patients. In the remaining nine a biplane osteotomy was performed with the tibial tubercle at the distal segment in order to decompress the patello-femoral joint. Gradual correction of the mechanical axis was performed using the system's software starting 4-6 days post-op. Mean follow-up time was 4 years (2-6 years).

Results
The osteotomy was fully consolidated within an average of nine weeks (7-11) and frame removed in all patients 10 to 14 weeks post-op. The average correction was 12 degrees (5-18) and the average time of distraction was 11 days. A residual deformity correction was performed after the end of initial correction in 7 patients. There was no loss of correction after frame removal and during the follow-up period in all but one patient. 10% of patients had pin-tract infections that were treated conservatively with no need of pin removal. All patients gained significant functional improvement regarding knee pain and flexion and there was no deterioration of the functional results during the follow-up of 2- 9 years. No revision to TKA.

Conclusions: HTO in patients with medial compartment OA and varus deformity, carried out with a minimal incision & corrected gradually with TSF leads predictable deformity correction and an excellent clinical outcome, with the burden of wearing the Ex fix for 10-14 weeks.

5.2 Distal Femoral Osteotomies – Stephan Nehrer (AT)

Biomechanics of joints of the lower limb is the determining factor for the development of degenera-tive changes especially in the knee joint. The restoration of an orthograde anatomic alignment repre-sents the basis for every recovery of the joint function. The ligamental stability, meniscal injuries and the cartilage damage are important factors for the knee joint but the biomechanics of the entire lower limb is the most important factor of progression joint diseases; therefore osteotomies are cru-cial operative corrective actions, to restore correct alignment. In the last years osteotomies have become more important, after years of being almost out of fash-ion. The reasons for the positive trend are the development of angle stable screw fixation of the im-plant, which enables to initiate an immediate mobilization after osteotomy and by the use of the open wedge technique, which allows a more physiological adjustment of joint axis: the angle of rota-tion of correction is closer to the actual rotation center of deformity and no leg length is lost.

The open wedge technique of the femor is characterized by spreading of the osteotomy gap by chis-els or spreader and intraoperative correction according to intraoperative measurements. Closing wedge osteotomies do not allow corrections after cutting out the bonewedge, especially in overcor-rection and lead to an asymmetric deformity of the femor head. However, openwedge techniques need a high experience in stepwise spreading the osteotomy, otherwise your risk a fracture of the opposite corticalis. In more severe cases of correction and osteoarthritis as well as in overlenth clos-ing wedge osteotomy is still preferable. Especially patients between 40 and 60 years with early onset osteoarthritis in a joint compartment are candidates for an osteotomy. The selection of the right patients and a detailed planning of the osteotomy before surgery are the most important factors for an optimal clinical result.

For the planning longleg-standing X-rays to evaluate the mechanical axis are mandatory on which the mechanical axis of the whole extremity is determined by drawing a line from the hip center to the center of the ankle joint (Mikulic line). Than the joint line is created and the angles between mechan-ical axis and joint line are measured to determine whether the deformity is in the femur or in the tibia. About 30% of varus deformities also have some amount of femoral deformity. Femoral correc-tions are nowadays performed as closing wedge osteotomies on the medial side with angle stable implants. Intraoperative control of correction angles and alignment is key to successful surgery. Navigation systems or an image intensifier are used for confirmation of the achieved correction. An arthroscopy to directly assess the intraarticular structu-

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res and address concomitant pathology is performed in all cases. Mensicustears, ACL ruptures and cartilage defects have to be addressed at the same time, furthermore is has to be secured that the medial compartment and the femoropatel-lar joint is free of sever degenerative diseases. In the case of cartilage defect we either perform a two-stage procedures with a biopsy for celltransplantation and the second surgery with osteotomy and celltransplantation.

With the biological treatment options in cartilage repair on the affected side osteotomies have be-come a wider indication in early onset osteoarthritis and are in our hands competing with hemi-protheses or partial joint replacement. However the ongoing studies and controlled trials will clarify the role of osteotomy in cartilage repair.

5.3 Combined Surgical Interventions for Instability & Cartilage Problems
Mats Brittberg (SE)

Cartilage lesions are often seen combined with other injuries such as menisci, cruciate ligaments and patellar instabilities and as with any cartilage repair method, good results should not be expected if coexisting knee pathology is not carefully taken care of. Biomechanical misalignment and ligamentous insufficiency can lead to excessive forces and abnormal compressive loads that can destroy the induced repair tissue. Therefore, it is critical that any associated knee pathology responsible for or contributing to the chondral defect is identified and corrected prior to or in conjunction with the cells and scaffold are being implanted.

Biomechanical malignment
If the mechanical axis on long standing x-rays passes through the compartment in which the chondral injury is located, an unloading osteotomy is recommended to shift abnormal forces away from that compartment . Unloading osteotomies should also be considered when the lesions on the condyles are large even without a malalignment or as an alternate use, protect weight bearing with the use of a custom-made unloader brace.Also important is to evaluate any abnormal patellar tracking and if needed unload the patellofemoral joint by realignments procedures shifting the distal patella tendon insertion medial/lateral and or anteriorly.

Ligamentous insufficiency
Ligamentous insufficiency produces excessive shear forces in the knee, which may negatively influence the maturing process of the repair tissue. If performed concomitantly, cruciate ligament reconstruction should precede ACI and should be performed in standard fashion with the desired technique of the surgeon and patient.

Meniscal deficiencies
The importance of a functional and intact meniscus when to consider cartilage repair cannot be overstated. Whenever possible, the meniscus should be preserved or repaired In the presence of a total meniscectomy, or when the meniscal function is equivalent, meniscus transplantation may be considered. The meniscal allograft will help reduce the concentrated forces in the involved compartment and help protect the newly formed repair tissue. When performing a meniscal allograft concomitantly with an ACI, the meniscal allograft should be placed and secured, after which the ACI can be completed.

Osteochondral defects
Bone grafting can be done at the time of arthroscopic evaluation and chondral biopsy. Another option is to do the procedure as a 1-stage procedure with ACI in combination with bone grafting via the so called “sandwich” technique (Jones and Peterson, 2007) in which the bony defect is filled with bone grafts, periosteum is on top of the bone grafts level with the subchondral bone plate and periosteum is also on top of the cartilage defect with the cells in between the both cell layers.

Conclusion
Additional treatments when doing cartilage repair are quite often needed. One-stage procedures are also becoming more popular and that is why all causes of the disability should be investigated and evaluated when planning your surgery. To restore the joint homeostasis is of uttermost importance in order to reach maximum success with your cartilage repair procedure. It is important to remember that cartilage alone is most often not the lonely producer of the disability of the joint but combined causes. Seek and ye shall find.....

Session 8 – Problems of the Patellofemoral Joint

8.1 Imaging of the Patellofemoral Joint – Mariana Vlychou (GR)

A wide range of disorders can affect the patellofemoral joint (PFJ) due to congenital, traumatic and non-traumatic pathologies that may lead to anterior knee pain, instability and degeneration. Various imaging modalities including radiographs with dedicated views, Ultrasound (US), Computed Tomography (CT) and Magnetic Resonance Imaging (MRI) have a distinct role in detection and characterisation of PFJ disorders and their combined use may be of clinical importance. The clinical history and the physical examination of the PFJ provide the appropriate context to pathology revealed on imaging. The integrity of patellar cartilage can be assessed by both conventional MRI sequences that appreciate and classify morphological defects and quantitative MRI techniques such as T2 mapping, in order to detect subtle biochemical changes of cartilage composition that may lead to osteoarthritis. The above applications require high-field MR scanners and appropriate optimisation of imaging protocols.

8.2 MPFL Reconstruction – Hantes Michael (GR)

Patella instability after patellar dislocation is a relatively common problem in adolescents, and correlates with trochlear dysplasia, patella alta, and femoral or tibial torsional malalignmet. The primary restraining structure against patellar lateralization and dislocation is the medial patella-femoral ligament (MPFL), which contributes up to 70% of the restraining forces against lateral patellar displacement. According to MRI studies, the MPFL is disrupted after patellar dislocation, in the vast majority of cases (90-100%). Therefore, it has been suggested that reconstruction of the MPFL will provide stability and prevent re-dislocation of the patella. Indeed, during the past decade isolated reconstruction of the MPFL has become the primary surgical treatment for recurrent patellar instability.

The MPFL is a thin ligament located in the second layer in the medial knee and inserts at the proximal two thirds of the medial border of the patella. Some of the fibers fuse with the vastus medialis obliquus (VMO) aponeurosis also. At the femur, the ligament inserts medial at the adductor tubercle (AT) proximal to the insertion of the medial collateral ligament and distal to the insertion of the adductor magnus tendon. The length of the MPFL is 57.7 mm (range 47–65 mm), its width at the femoral insertion is 12.2 mm (range 8–16 mm), and at the patellar insertion 24.4 mm (range 17–32 mm).

Numerous techniques for MPFL reconstruction have been described, and they all aim to supply tendon tissue from the medial aspect of the patella to the femoral insertion site of the MPFL. Numerous graft sources for reconstruction are described in the literature, including semitendinosus, gracilis (the most common), quadriceps, patella tendon, allografts or artificial tendons. In addition, multiple techniques for fixation of the graft at the patella are in clinical use at present. Although some authors use suture anchors or interference screws for fixation of the graft at the patella, others describe techniques whereby the graft is passed through 2 bone tunnels in the patella, or a bone bridge is created on the medial margin of the patella to hold the graft in place. However the key point to MPFL reconstruction is its positioning in the femoral insertion. This is because it dictates how the graft will function and a non-anatomical placement will create non-physiological graft conditions. This will cause knee motion problems and subsequent graft failure. According to Schöttle et al. a reproducible radiographic point to verify the femoral insertion for MPFL reconstruction intraoperatively, is1.3 mm anterior to the posterior cortex extension and 2.5 mm distal to the posterior origin of the medial femoral condyle, just proximal to the level of the posterior point of the Blumensaat line on a lateral view with the posterior condylar margin overlapped.

We use the gracilis tendon for MPFL reconstruction in our institution, and a transosseous suture technique for graft fixation at the patellar site and a bioresorbable interference screw was to secure the graft within the medial condyle tunnel with the knee flexed to 30°.

Recent studies have shown that reconstruction of the MPFL provides significant improvements in all outcome scoring systems, and good patient satisfaction. According to the literature, the percentage of patients returning to their preoperative levels of sports activity after surgery varies from 32% to 76%. However, complications like patellar fracture, persistent instability, loss of knee flexion, pain and wound problems have been reported after this procedure.
Finally, in some cases with severe trochlear dysplasia and increased tuberosity-trochlear groove (TT-TG) distance additional procedures to MPFL reconstruction, like trochleoplasty and tistal tuberosity transfer should be performed also.

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8.3 Trochleoplasty – Lars Peterson (SE)

Patellofemoral Stability

Skeletal and ligamentous (passive) and muscular(active) stabilizers of the patellofemoral joint work together for maintaining the joint stability. Other factors affecting joint function are genu valgum, increased Q-angle, femoral anteversion , tibial torsion causing increased lateral tracking and insufficiency of the MPFL and VMO . The geometry of the trochlea and patellar surfaces is of the highest importance for the patellar stability during the first 30 degrees of flexion(or extension)

Patellofemoral dysplasia is a common background factor to acute patellar dislocation after minor trauma, to knee pain and to patellar malalignment and instability. It is most common in teenagers and young women and is probably a developmental abnormality due to patella alta and the lack of mechanical stimulation (compare to the dysplastic hip in childhood) between the patellar and trochlear surfaces in 0-30 degrees flexion(30-0 extension) resulting in the development of dysplasia on both sides. The trochlea dysplasia is the most important factor to restore the skeletal /geometric stability .

The MPFL and LPFL (or medial or lateral transverse retinaculae) are passive stabilizers to the patella throughout the whole ROM. They work also as active tendinous extensions to VMO and VL in the same way as the medial and lateral longitudinal retinaculae with their insertions in the proximal tibia. The patellar tendon(ligament) also has a double role in stabilizing the patella proximal as ligament and actively as a tendon to the quadriceps muscle.

Trochlear Dysplasia

Trochlear dysplasia can be classified into 3 grades. Grade I: A flat or slightly shallow trochlea. Grade II: A convex trochlea with proximal extension .Grade III: Flat lateral trochlea with flat articulating lateral patella facet . No trochlear proximal sulcus, dysplastic medial trochlea.

Trochlea dysplasia is seldom a single cause of knee pain or patellofemoral instability, and for a successful and optimal treatment all background factors to patella malalignment and instability need to be identified and addressed.

Trochleoplastie

Bereiter et al.(1994) reported on lateral trochlea wedge osteotomy with bone graft. Dejour reported(2010) on deepening trochleaplasty for patellar Instability with staple fixation. Peterson et al reported (1988, 2010) on proximal trochleoplasty in recurrent patellar dislocation with 14 years follow up. Later on proximal trochleoplasty and the importance of correcting all background factors for long term success. Biedert (2006,2010) reported on trochlear lengthening ostetomy with or without elevation of the lateral trochlear facet and bone grafting.

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Proximal Trochleoplasty(Grooveplasty)
The aim of this technique is to stabilize the patella during the initial 0-30 degrees of flexion(extension30-0) without too much interfering with the patella-trochlea congruity during the remaining patella-trochlea contact to full flexion. Other trochleoplasties interfere with this congruity and may cause progressive cartilage erosion on the joint surfaces.

Preoperative Examinations
The aim should be to assess the dysplasia and identify other(all) background factors. Based on these findings the surgery will be planned for correcting important factors, in combination with cartilage repair techniques.
History and clinical examination should be carefully performed including background factors lateral tracking, patellar tilt,subluxation and dislocation , using apprehension tests.

Standing X-rays-hip-knee-ankle for malalignment,CT-scan in extension and 20 degree flexion with and without q-ceps contraction for evaluation of degree of dysplasia and maltracking-instability.MRI can be added to evaluate cartilage , other pathology.

Surgical Technique
Open trochleaplasty is used in almost all cases when ACI and other concomitant procedures such as proximal and distal realignment are indicated . Arthroscopic trochleoplasty can be used in cases of failed operated instability when trochlea dysplasia has not been corrected or in combination with arthroscopic reconstruction of the MPFL and/or lateral release.
Use central skin incision and dissect medial and lateral to the patella patellar tendon. Medial arhrotomy starting 2 cm proximal to the patella and incise between the rectus femoris and VMO tendons, continue 5-7 mm medial between VMO,MPFL and capsule down to the infrapatellar bursa which is opened medially. Identify the type of dysplasia and evaluate patellofemoral cartilage lesion(s). Release the synovial lining from the articular border of the trochlea by proximal dissection 4-5 cm close to the cortex of the femur.

Draw an imaginary horizontal line across the trochlea .Than draw an imaginary line from the top of the intercondylar notch perpendicular to the horizontal line .This is the mid point of the new trochlea .Use a curved osteotome to remove cartilage and bone 10 mm distal and extending 15 mm to the sides to create a concave groove. If the bone is flat or convex on the femur shaft this bone should be removed making a coninuous groove. Use a burr to smoothen the bone. Adjust the groove if necessary.Usually this is done after eventual osteotomy of the tibial tuberosity(TT) for correcting the Q-angle Now test the patella sliding through the groove and use 3-0 resorbable sutures to fix the synovial lining back to the cartilage edge. . Use 4-5 mattress sutures for good adaptation. Inject fibrin glue between the bone and the synovial lining to make a smooth surface and limit bleeding.

Results. In total 92 patients had patella or trochlea or patello –trochlear lesions,mean size /lesion 5.5 cm2. Twenty-two had trochleo plasty along with reconstruction of the extensor mechanism, another 2 had a trochleoplasty ,1 with medial plication and 1 with a lateral release. Tegner Wallgre´n score improved by 1 from 7.1 to 8.1 ,and the Lysholm score was improved by 9 points to 69.3 . Patients with malalignment and instability had comparable results and stable patella. Ninety two to 94 % would have the surgery again.
Concomitant procedures will be shortly discussed..

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8.5 Patellofemoral Joint Cartilage Defects – Alberto Gobbi (IT)

Patellofemoral chondral lesions are difficult-to-treat entities often affecting a young and active patient population and remain challenging, especially the large full-thickness lesions. Moreover, cartilage with its inherent isolation from systemic regulation, lack of vessels and nerve supply, contributes in difficulty in healing. Chondral or osteochondral lesions are frequently found during knee arthroscopy. In a study of 1000 patients who underwent arthroscopy the prevalence of osteochondral defects was 61% while 17% of them was located in the patellofemoral joint (11% patella 6% trochlea)[1.] Furthermore, patellofemoral maltracking and instability often acts as an undiagnosed background factor for articular cartilage lesions in the patella and trochlea[2].

Microfracture in long term studies has shown poor results in patella femoral lesions.[3] Since years, autologous chondrocyte implantation (ACI) has managed to sustain as a good treatment option to deal with the large full-thickness chondral lesions.4, 5 The first generation ACI technique is complex, requires periosteal tissue harvest and a meticulous sewing of the patch over the defect to ensure a "water-tight" closure preventing spillage of the chondrocytes; furthermore, it is associated with donor site morbidity related to biopsy.[4-6]

Second generation ACI was named matrix-induced autologous chondrocyte implantation (MACI), because of the use of a matrix seeded with chondrocytes. The use of a three-dimensional scaffolds for chondrocyte culture was developed with the aim to improve both the biological performance of chondrogenic autologous cells as well as renders the surgical technique easier, avoiding the use of the periosteal flap. Previous studies showed good results with various scaffolds in patella femoral chondral lesions, improvement at medium term follow-up with a repair tissue similar to normal cartilage, mechanically and histologically stable.7 Hyaluronic Acid (HA) is a naturally occurring molecule present in all soft tissues that plays an essential role in the maintenance of the normal extracellular matrix structure. The use of this molecule in cartilage lesion treatment has been shown to provide good results, since it favored the formation of new cartilage tissue. Specifically, scaffold based on HYAFF®11, a derivative of HA, has been shown to provide successful tissue-engineered repair of cartilage. MACI technique provides a friendly use and application of graft and shorter surgical time as there is no need for periosteal tissue harvest; however, MACI still requires two surgical procedures.[7, 8]

Research moves toward the possibility of performing a single-step procedure; in this regard, the use of bone marrow aspirate concentrate (BMAC), which contains multipotent stem cells (MSCs) and growth factors, could represent a possible solution.[9] The easy availability, coupled with the self-renewal capacity and multi lineage differentiation potential of MSCs to cartilage tissue offer a promising option in cartilage surgery.[10, 11] MSCs interaction with a non-woven scaffold was found suitable for tissue repair; HYAFF®11 scaffold supported the adhesion, migration and proliferation of MSCs, as well as the synthesis and delivery of extracellular matrix components under static culture conditions. Additionally, the specific ability of MSCs on HYAFF®11 scaffold to differentiate into chondrocytes has been observed by the expression and production of specific extracellular matrix molecules. On contrary to MACI, MSCs isolation is devoid of the need to harvest healthy cartilage tissue and therefore bypasses the first surgical step required for cartilage biopsy and subsequent chondrocyte cell. Recent studies reported that one-step technique with bone marrow-derived MSCs implantation could be an efficient alternative for cartilage repair, permitting marked improvement of functional scores and near hyaline cartilage repair, while overcoming the drawbacks of previous techniques.[12, 13]

In a recent study (Gobbi et al, unpublished data) were compared the clinical outcomes of two similar groups of patients with patella femoral full-thickness cartilage lesions, treated with MACI or BMAC, utilizing the same scaffold thirty-seven patients with patellofemoral chondral lesions were prospectively followed up, for a minimum of 3 years; 19 patients were treated with MACI and 18 with BMAC. Radiographs, MRI and IKDC, KOOS, VAS and Tegner scores were collected preoperatively, at 2- year and final follow-up. No adverse reactions or complications were noted and both groups showed significant improvement in all scores, from pre-operative to final follow-up (P=0.001), but there was no significant difference in improvement between the two groups. Age was not statistically significant prognostic factor in current study groups, although patients younger than 45 years of age showed better results. Lesion size and number were important predictors of outcome in both groups. Reported better results in subgroup of patients with lesion size ≤10cm2 and single lesions in both groups correspond to previous reports; however, literature lacks the evidence of outcome results after MACI for such big lesions. Trochlear lesions showed better improvement than patellar group in MACI group at 2-year and less deterioration at final follow-up scores, in accordance to other reports. On the other hand, patients presenting with patellar and trochlea lesions showed nearly same results in all scores in BMAC group.

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MRI showed complete filling of the defects in about 89 % of the patients treated with the 2 techniques and histological analysis revealed hyaline-like features and ligamentous insufficiency, in a previous or concomitant cartilage repair procedure, is widely recognized. Concomitant correction of tibiofemoral axis malalignment, when articular cartilage restoration techniques are applied, provides greater survival at medium and long-term follow-up but further to decreasing the stress on the load-bearing, only partial remodeling to fibrocartilage tissue has been reported. Similarly, concomitant patellofemoral maltracking correction reduces overloading of the lateral patellofemoral joint and therefore reduces the risk of future cartilage injuries.

Histological examination of the performed biopsies revealed the regeneration of new tissue with hyaline-like cartilage features. MRI evaluation showed complete filling of the defect in 76% of the MACI group and 81% of the patients in BMAC group with no signs of hypertrophy in either group, similarly to previous reports

MACI procedure has shown advantages over first generation ACI in terms of easy handling and application of graft material via minimally invasive or arthroscopic techniques, shortened surgical time, avoiding the need for periosteal tissue harvest and periosteal hypertrophy and considerably low failure rate (2.5%), compared to 5% to 13% reported with first-generation ACI. However, MACI still remains a 2-surgery technique and is an expensive solution. Increasing interest in MSCs research is due to the widespread availability of MSCs and the possibility to isolate them from various sites, making them easy targets for harvesting. Furthermore, the finding that bone marrow–derived MSCs have a better proliferation rate than chondrocytes and that MSCs transplantation has comparable outcomes to ACI portends positive trends for the use of these cells in cartilage surgery. The single-step technique with BMAC implantation is a bedside technique, with no need for culture thereby avoiding the expenditure for an extra procedure to retrieve chondral biopsy decreasing the total costs of the procedure and donor site morbidity that studies on MACI have shown. The use of a point-of-care device provides a sufficient concentration of total nucleated cells and platelets included in the aspirate that further contribute to the stimulation of progenitor cells acting as a chemoattractant the utilized HYAFF®11 scaffold provides both the suitable environment to maintain cell in situ into the defect; in addition, it has been reported to support the adhesion, migration and proliferation of MSCs, as well as the synthesis and delivery of extracellular matrix components under static culture conditions. Both MACI and MSCs implantation techniques have been proven viable and effective for the treatment of large patella femoral chondral defects at medium term follow-up. MSCs implantation is a new technique with potential advantages including a single-step surgery, no need of cartilage biopsy and cells cultivation, thus reducing the total cost. However, a larger number of patients and randomized comparative trials with longer follow-up are essential to establish conclusively about the comparative efficacy of these procedures in the treatment of patellofemoral lesions.

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8.6 Patella Dislocation & Chondral Lesions – Michael Iosifidis (GR)

Patellofemoral (PF) dislocations are commonly associated with chondral injury. Chondral and osteochondral lesions are often associated with traumatic (high-energy) PF dislocations, whereas atraumatic (low-energy) PF dislocations in patients with significant PF risk factors have a much lower incidence of osteochondral damage. This is because the amount of energy required for a dislocation to occur is inversely proportional to the amount of PF dysplasia. It was estimated that patients with normal patellar stability who suffered traumatic dislocations for the first time were at a 2.5 times greater risk of articular surface damage compared with the patients "hypermobile" patella[1]. Once the first dislocation has occurred, recurrent dislocations may lead to additional injury to the articular surfaces. The treatment planning for recurrent dislocation has to think about the cartilage "protection" and cope with patellofemoral joint as "whole issue".

The first traumatic dislocation regards mostly young active patients (children-adolescents) and it has been reported that is 2-3% of all knee injuries (the second cause for haemarthrosis)[2]. A historical review of the literature suggests that acute PF dislocations result in predominantly lateral femoral condylar and medial patella facet lesions (Fig. 1.). The incidence is high and ranges from 70% to 93%. Bone bruises of the medial patella border and the lateral femoral condyle are present in 100% of all patients undergoing a first-time traumatic patella dislocation, whereas in patients with recurrent or chronic patella dislocations, the incidence of bone bruising is markedly lower. The latter might be because the medial patellofemoral ligament (MPFL) was injured with the first dislocation, and thus, less energy is required for subsequent dislocations to occur[3]. It is also reported that possibly 30-40% of the lesions are missed[4,5,6].

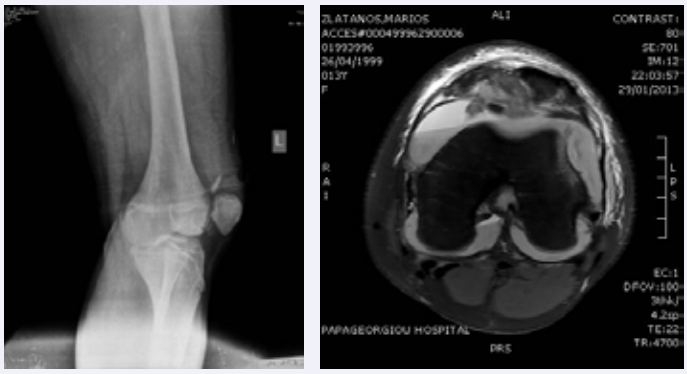

Figure 1. Acute PF dislocations result in predominantly lateral femoral condylar and medial patella facet lesions. (x-ray and MRI from personal data)

It is important to evaluate not only the mechanism of the dislocation after an initial occurrence, but also to define the extent of chondral or osteochondral injury. With the low sensitivity of radiographs in detecting chondral and even osteochondral injury, it is necessary to obtain an MRI after the acute (first) patellar dislocation[7,8]. Further knowledge about important anatomic parameters help to determine the extent of the potential damage suffered during the initial injury. It is also important to analyze each parameter of the known risk factors for PF stability: patella alta, excessive lateral position of the tibial tuberosity (TT-TG), trochlear anatomy (dysplasia), and femoral/tibial axial rotation. There is a broad continuum from normal anatomy to the extreme of pathoanatomy. Just because a patient might be classified as having suffered a traumatic patella dislocation does not preclude a full evaluation of underlying risk factors for PF instability that may lead to chronic redislocations and unfavorable non-operative treatment[3].

The treatment of focal chondral and osteochondral lesions varies extensively depending on defect size, damage to the osteochondral fragment, and acuity of the injury. In a first-time acute patella dislocation, the fragments can range in size from small comminuted pieces of debris to hemicondylar avulsions. If there is a fragment that allows for technical fixation, then the fragment should be fixed in situ, mostly with absorbable pins or nonabsorbable cannulated screws that may require hardware removal (Fig. 2). Chronic lesions need to be addressed from a more restorative point of view. Partial thickness lesions <1cm2 may be potentially asymptomatic and may require only chondroplasty. In small full-thickness lesions (1 cm^2), microfracture may be appropriate, which is technically difficult to perform and the results have been unfavorable9. Small to medium sized lesions 1 to 4 cm2 can be addressed with osteochondral autografts, which is also a demanding technique with doubtable results[10]. For the treatment of larger lesions (2 to 10 cm2), autologous chondrocyte implantation is the most commonly reported technique11. These patients, ideally, are younger, present with a focal defect, have a short onset of symptom duration (Fig. 3). Alternatively, large deep lesions with significant subchondral bone erosion may require reconstruction of the osteochondral unit. As for the treatment of patella instability there should be careful preoperative planning according to type of dislocation: first acute or recurrent. For example, it may exist increased danger for joint stiffness if MPFL reconstruction is performed in acute dislocated knee.

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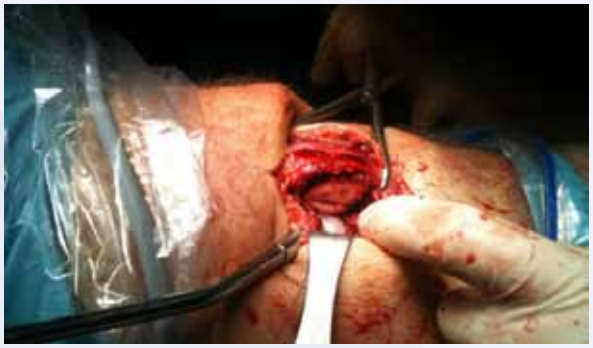

Figure 2. A quite big fragment after 1st time patella dislocation was fixed in situ, with absorbable pins (photo from personal data)

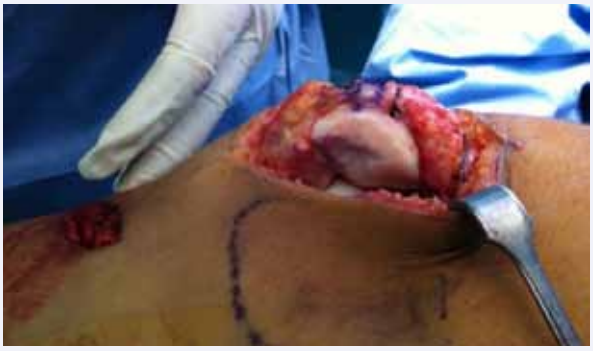

Figure 3. ACI for patella cartilage lesion performed along with MPFL reconstruction (photo from personal data)

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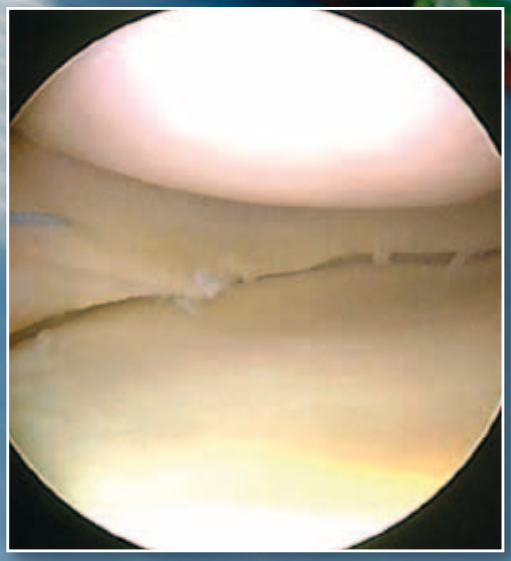

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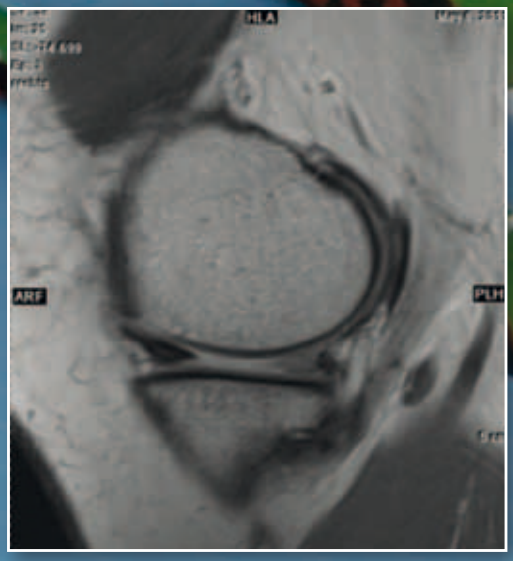

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