Vice-president's words
Dr Vikash Kapoor

Any society once formed takes a minimum of 5 years to mature and establish, catching the eye of the parent society and creating a meaningful impact.

In today's age and time when the market drives and dictates "To do or Not to do" in much the Shakespearean way, KASS with its grounded commitments to teaching and value based focused education for the cause of promoting Sports Surgery and Arthroscopy stands apart.

Representing our state and Kolkata at IASCON for past 14 years since 2002, I regretfully remember missing but one meeting.

As the time has flown the talent pool in East has not only grown but also got enriched by the leaders in the field of Arthroscopy and Sports Surgery.

The time has arrived when under the aegis of KASS a number of fellows are being offered to the young and eager, yearning to master the nuances of Arthroscopic Surgery.

The bar is being constantly raised as the team tests itself repeatedly by organizing seminars, guest lectures, meetings, live surgeries and some very exceptional cadaveric workshops.

With the constraints of time, space, finance and legalities the one thing that overshadows all is the phenomenal commitment, which the organizing team has shown consistently, delivering top quality educational interactions for past 5 years.

With the IASCON 2016 knocking doors it is indeed a proud moment for the society and for me personally to be writing this communication welcoming all of you to the City of Joy, which under watchful and prudent eyes of our society is now a city defining the art and science of Arthroscopy and Sports Surgery in its own innatural way.

Several institutions, which are cutting edge and world class in equipment and facilities, are now a part of this Victorian city's rich milieu merging seamlessly the values of tradition with the modern science of healthcare delivery to the common masses.

To this goal the contribution of all the KASS members and founders has been exemplary and it shows!

Best Wishes and looking forward for a great experience of learning and interactions at IASCON 2016.

Piece of Mind
Pursuit of Happiness
Dr M N Basu Mallick

In the two hundred thousand or so years that humans and their ancestors have arrived in this world, our brains have almost tripled in size, grossly disproportionate to the change in our physical stature during the same period. It's not only that size of brain changed, new areas appeared, and developed. One of these new areas is the frontal lobe, an area which gave us, among many other things, the power of pleasure and anticipation. Say for example, would you like to taste a brinjal and onion pudding? "Yuck." Why? Have you tasted it before? It's just this anticipation of bad taste that makes us "yuck".

Now take this example. Would you want to win the first prize or the 10th prize in our state lottery? The answer seems obvious. But if you look at all the people who won anything in that lottery, you'll find all of them happy. If you see them 1 month later, you'll find their mental states completely unrelated to that lottery result.

So what makes some people more happy than others. It's definitely not what they achieved in life, nor to that lottery. It's 'synthesis' of happiness. Meaning, they are synthesising or manufacturing, more happiness in their frontal lobes.

Now, the question arises, can we train our own frontal lobes to synthesise more happiness? There has been millions of hours spent by social scientists and psychologists researching this secret to synthesising happiness. And the obvious cliche answer has been "to be satisfied with what you get."

In a very popular experiment to this effect, isolation prisoners in a jail were given small and cheap items without being told that others were getting similar gifts. It made all of them happy and their behaviour improved. When similar gifts were distributed in mass cells of the same prison, people tended to compare with what others got, and many felt less happy. This was repeated again, wherein people were given the choice of gifts from a stack. And this time too the prisoners in general were less happy, and were troubled by the thought whether they made the right choice. It was the probability of better choices that made them less happy.

So, it seems, choice is what confuses us and makes us unhappy. Ability to isolate ones self from achievement of others, and being able to focus on what we have done indeed triggers the brain to synthesise happiness.

Think about it.
Tricks in tunnel preparation in anatomic ACL reconstruction

Dr. Sebham Gosh
MS Orth, Fellow – Arthroscopy and Sports Surgery

Proper placement of tunnels is crucial for achieving proper graft placement and good functional outcome in ACLR. Usually femoral tunnel is prepared prior to the tibial one to prevent irrigating fluid loss (except where trans-tibial femoral tunnel is made). As there has been a gradual shift from trans-tibial to trans-portal anatomic ACLR in view of higher chances of persistent rotatory instability with the former, certain difficulties in tunnel placement have emerged. And new tricks need to be learnt to get around those difficulties.

Femoral tunnel preparation:
Femoral tunnel is commonly done through a far-medial instrument portal. Femoral insertion is crucial for isometric placement of graft. Graft placement is assumed to be isometric when the distance between the tibial and femoral attachments changes by ≤2 mm when the knee is moved from full extension to 90 degree of flexion. Following are the areas of special consideration for perfect tunnel placement:

a) To reach the ever-the-top position, accessory medial portal has to be done at supra-menisical level. This is much lower and far medial than the usual trans-synovial meniscal portal, and grazes the top of the medial meniscus.

b) Using a spinal needle to access the reach and direction of the portal prior to giving the incision is prudent. One should make sure that the needle can reach the back of the femoral attachment of the ACL while simultaneously remaining off the medial femoral condyle.

c) Deciding the perfect site for femoral tunnel is debatable. The general consensus being tunnel should be prepared at the center of the femoral footprint of the ACL through the ridge separating the anteromedial and posterolateral bundles of the native ACL. Preserving the ACL footprint helps in this regard.

d) It is best to identify the entry point with the knee in 90 degree flexion as with higher degrees of flexion the orientation of the femoral condyles with respect to the tibial plateau changes.

e) The femoral entry point is slightly above the midpoint between the inferior edge and superior edge of the medial wall of the lateral femoral condyle. (With the knee at 90 degrees during arthroscopy this is slightly behind the midpoint of the anterior upper portion of the lateral femoral condyle.) Also 5-6 mm away from the articular cartilage of the posterior edge of the condyle (with the knee 90 degrees flexed during arthroscopy, this means 5-6 mm above the apparent inferior edge of the condyle designed by it).

f) Using the RF probe or a microfracture awl to mark the entry point prior to drilling helps maintain ideal position when the knee is flexed to 120 degrees.

g) It’s helpful to use an offset femoral guide to stabilize the drill pin during drilling, but it should not be used to guide the position of the femoral tunnel.

h) At 120 degrees flexion, the drill pin should be almost parallel to the tibial articular surface and the tunnel length around 35 mm.

i) If the femoral tunnel length is far more than 35 mm, it can be assumed that the tunnel mouth will be far too oblong instead of circular and the graft will misfit the tunnel mouth where its bony integration is critical.

j) Using threaded, stout, 2.4 mm guide wire rather than smooth and thin Kirschner wire; prevents inward deflection from lateral condylar cortex.

Tibial tunnel preparation:

a) Placing Tibial jig: The jig should be used through the anteromedial portal. Placement through the far medial portal hinders proper placement of tibial drill guide as it is deflected my medial femoral condyle.

b) Placing the stylus: Most common error during tibial tunneling is too anterior placement of tunnel. It has been shown that when placed too far anteriorly, 30% cases have deficiency due to chronic graft impingement. In sagittal plane there must be a distance of 2-3 mm between graft and the intercondylar roof and it should be placed behind the tibial plateau in fully extended knee. So, it is safe to put the stylus under direct vision anterior to the anterior circumference of PCL and in line with inner margin of anterior horn of lateral meniscus. There is a consistent distance of 7 mm between the centre of tibial attachment of ACL and anterior circumference of PCL in 90’ flexion of knee. In mediosagittal plane, it should be midway between medial and lateral extent of ACL footprint.

c) ACL stump: Some authors recommend preserving some remnant of the ACL which not only acts as a guide to proper placement of the stylus, it also; albeit theoretically; provides a neurologically active envelope of the graft for proprioceptive function during rehabilitation.

d) The centre of ACL stump is not a true guide for stylus placement. Having placed the stylus at the centre, one might have to undergo about 2-3 mm notchplasty to prevent impingement.

e) Upon introduction of the drill pin into the proposed tibial tunnel site, the knee should be taken through a full range of motion to see if there is any possible impingement against the roof or the walls.

f) The tibial jig should generally be 50-55 degrees. At higher degrees, the tibial tunnel is too vertical. At lower degrees it is too horizontal and might have a possibility of blowout.

g) Drilling technique: Sequential drilling allows for some degree of bone compaction and also tunnel correction. A 4.5 mm cannulated drill used over a 2.4 mm drill pin allows for readjustment of about 1 mm in any direction, and a 6 mm drill allows about 2mm readjustment. The initial drill pin should never be overdreilled right away to the definitive graft diameter.

h) Another important take on drilling is not to drill very haphazardly during penetration. It can cause serious damage to the bone and cartilage thereoriginating loose bodies within the joint. One may use a curette to cover the tip of the bone needle so that the drill doesn’t suddenly engage it and hit the femoral condyle.

i) Proper drill bit selection: The femoral tunnel reamers have tendency to deviate from actual axis and more elliptical tunneling. They can cause inadvertent damage to the cancellous bone in upper tibia. So, it is better to use the classically cannulated tibial reamers in 1 mm increment. This also allows for compaction of tibial bone.

j) Using tunnel dilators: In very soft bone and also revision scenarios, it is preferable to use dilators of the preferred size after drilling tunnels of 1 mm under size. Improves screw purchase and pull out strength of graft.
Current concepts of treatment of Osteochondral Defects of the knee

Dr Mainak Chandra
Assistant Professor, Malda Medical College, West Bengal, India

Introduction
Articular cartilage lesions in weight-bearing joints often fail to heal on their own and may be associated with pain, loss of function and long-term complications such as osteoarthritis. Osteochondral injuries are both naturally and therapeutically irreversible with current treatment parameters. Inferior repair commonly occurs, but stable regeneration of hyaline cartilage has never been documented. Decisions about whether and how to treat an individual lesion are problematic. Regardless of the treatment method or the origin of repair factors, the end result is generally a fibrous repair tissue (fibrocartilage) which lacks the biomechanical characteristics necessary to withstand the compressive forces distributed across the knee during articulation. This fibrocartilage generally deteriorates over time, resulting in return of the original symptoms and occasionally reported progression to osteoarthritis.

Aetiology of osteochondral defects of the knee
There are two distinct chondral injury phenotypes according to attributing factors: focal lesions and degenerative lesions. Focal lesions are well delineated defects, usually caused by trauma, osteochondritis dissecans or osteonecrosis. Degenerative defects are typically poorly delineated and usually caused as a result of repetitive micro injuries, such as meniscal injuries, mal-alignment or osteoarthritis.

Diagnostic modalities of chondral lesions
For a good understanding of chondral lesions and suitable treatment policies, there is a need for a simple classification and quantification of the lesion.

The grading system devised by Outerbridge is simple and clinically useful in daily practice.

Grade 0 = Normal cartilage
Grade 1 = Softening, blisters or swelling of cartilage
Grade II = Partial thickness fissures and clefs <1mm
Grade III = Full thickness fissures and clefs extending within subchondral bone
Grade IV = Exposed subchondral bone

Radiological assessment of articular cartilage injury
Routine plain radiographs, including AP, LAT and standing PA flexion views, may reveal different findings, according to aetiology. Joint space narrowing, subchondral sclerosis or cysts will suggest an osteoarthritic origin. Osteochondritis dissecans defect can also be seen on plain radiographs, with or without loose body. Conventional radiography may reveal no change even with full-thickness cartilage lesions.

By using MRI, one can see bone structure, chondral lesions, meniscal or ligamentous pathology and bone marrow oedema (bone bruise). The optimal resolution for the articular cartilage surface is proton-density imaging of 0.4 mm sections and T2-weighted imaging with fat-saturation sequences.

Option specific treatment modalities of articular cartilage injury

The treatment of chondral lesions depends on patient selection, daily joint activity, articular activity, age, aetiology, grade and quality of the lesion. Treatment options range from conservative, through osteoarthroscopic or open surgical procedures.

Conservative treatment
The goal of conservative treatment is to reduce symptoms, not heal the lesion. It is considered in mild symptomatic cases or in cases with small lesions where surgery would do more harm than good. The appropriate approach for the asymptomatic patient with incidental articular cartilage injury is problematic. One approach is that of medications, such as NSAID, analgesics, and hormones (estrogen, growth hormone, etc). Next, there are several mechanical approaches including weight loss, rest, ice, canes, bracing, physical therapy, etc. Also, nutrition supply should be considered, with the use of supplements like chondroitin sulphate, MSM, Omega-3 and calcium and vitamins, as well as intra-articular injections such as steroids and visco-supplementation (Synvisc, Osteoïd, etc.).

To date, there has been no evidence of structural improvement with these conservative modalities.

Operative treatment
The various techniques available for surgical intervention result in reparative or restorative procedures. These procedures include the regeneration of ostechondral defects to ultrastructural and biomechanical competence of hyaline cartilage. Unfortunately, in all surgical techniques, the repair tissue is fibrocartilaginous in nature with little hyaline cartilage restoration. The basic idea is to adjust treatment to the individual patient, and to address related and/or contributing problems before or with the treatment of the chondral lesion, such as varus-valgus mal-alignment correction, patellofemoral tracking, stability of cruciate and collateral ligaments and meniscal lesions.

Surgical treatment for cartilage lesions is contraindicated in some cases as inflammatory arthropathy, unstable or mal-aligned joints, and contiguous lesions (bipolar), infection and obesity.

There are some surgical treatment modalities depending on surface area of the lesion, grade of lesion, and financial capabilities. These modalities are:-

Arthroscopic lavage and debridement
First noted by Burman in 1935, washout of the injured synovial joints had been proven to be the best front line treatment of chondral lesions. Arthroscopic lavage washes out inflammatory mediators, lose cartilage and collagen fibres, but mainly lodge the synovium and cause synovitis and effusion. Debridement of cartilage (chondroplasty) removes loose caps or edges that mechanically impinge on the joint. However, longer follow-up is necessary to decide whether this treatment carries the longevity of the arthroscopic cartilage repair techniques.

Ablation artroplasty
Popularised in the early 1980s by Johnson, artroplasty is an indicated especially in large, deep defects. Ablation removes only the boundaries of the articular cartilage defect to sustain a uniformly contoured edge of fresh collagen, capable of adhering a fibrin clot. Then, the subchondral bone is breached, allowing blood to percolate into the defect forming a fibrin clot.

The outcomes of abrasion arthroplasty are that the fibrin clot hardens, and none exhibits consistent good or excellent results.

Arthroscopic Subchondral drilling After debridement of the lesion edges to a contained crater, the subchondral bone is drilled with a high speed drill through subchondral bone. Blood is allowed to percolate into the defect forming a blood clot and initiating defect repair. The repaired cartilage is seen to be a full-thickness repair and fibrocartilage. The main drawback of this technique is thermal necrosis.

Microfracture With the purpose of making a rough surface analogue to fibrin clot, but without the thermal effects of a drill, Steadman et al proposed the use of an arthroscopic needle to create several dozen holes up to 2 mm apart. In a series of more than 200 patients treated, with three to five years follow-up, they reported improvement in symptoms and in Tegner activity level. Microfracture stabilisation in 20% and deterioration in 5% Histological analysis of microfracture repair shows, as is the case with all marrow-apposing techniques, that a hybrid of fibrocartilage and fibrocartilage dominates the defect site. In a study of 85 patients with full thickness lesions treated with microfracture and 36 months follow-up, Kreuz et al found improvement in all patients during the first 18 months. Deterioration began after 18 months and was significantly pronounced in patients older than 40 years. They concluded that results of microfracture are age-dependent and the best prognostic factor is age 40 or younger. However, Alparsson et al reported good results with improvement of function and activity after microfracture of full-thickness chondral lesions in 20 patients, with average age of 44 years, after 3.8 years follow-up.

Osteochondral autografting (OATS)
For taking regeneration of osteochondral defects, autografting is an obvious approach, due to same tissue and antigenity, with a non-weight-bearing area as a donor. The optimal patient is young with a medium-sized lesion (2.5-4 cm sq). Effectiveness is limited to the repair of focal defects and inability to restore degenerative lesions.

Arthroscopic debridement is followed by removal of unstable cartilage, aiming for a stable contained crater, preferably circular. This is achieved by measurement of surface area and cylindrical removal of subchondal bone, as close as possible to the lesion border. The next stage is harvesting cylindrical subchondal plugs from the knee (preferably NWB trochlear edges of femur) to the same depth removed from the crater. Interposition of the plug is performed, apart, by a graduated tamp, allowing accurate depth. Post-operative rehabilitation starts with therapy to six weeks of non-weight bearing, according to location and size of the lesion.

CPM, passive and active ROM and muscle strengthening is crucial during this time.

Osteochondral allografting
Due to graft size limitation and donor site morbidity, it is not feasible to perform a “compliance-free” allogeneic osteochondral graft. This may be used for medium to large defects, particularly for the completion of other primary surgical procedures. Two types are used: shell (<1 cm subchondral bone based) and deep grafts. Fresh allografts, obtained from the donor site, provide higher chondrocyte availability but carry a high risk for disease transmission. On the other hand, cryopreserved frozen allografts have reduced immunogenicity and disease transmission, but low chondrocyte availability. The best candidates are monopolar defects with bone loss, or defects in the vicinity of a tumour or salvage situations. Worse results are with osteoarthritis, avascular necrosis or hip fractures.

Autologous chondrocyte implantation (ACI)
The use of human autologous chondrocyte implantation (ACI) is described by Mats Brittberg et al. In 1994. They reported on the success of deep cartilage defect treatment in 23 patients, using first generation ACI. Using second generation ACI, which uses a combinatorial approach to fibrocartilage and fibrocartilage, better results in femoral condylar repair and 73% demonstration of hyaline-like cartilage upon follow up. ACI still has a long way to go, and a long journey to a viable arthroscopy and biopsy. The process is performed in several stages. The first step is diagnosis, biopsy and identification of the depth and size. Second, chondrocyte cultivation is performed in a GMP laboratory for cell propagation for six weeks. Third, implantation surgery occurs, which is usually debridement and sizing of the cartilage defect, harvesting of the periosetal flap from the proximal tibia (ACI-P), suitable for the defect size, fixation of the flap and injection of the cultured chondrocytes before closing the last suture.

ACI is indicated for the younger (aged 20-50 years) active patients with isolated traumatic femoral condylar chondral, greater than 2.5 sq cm, with less than 5-6 mm depth, so that repair of the subchondral base is not necessary.

It is accepted that the results of ACI were less favourable in patellar femoral joint lesions.

Matrix-induced ACI (MACI)
Because of the demand of periosetal flap and surgical difficulties, researches continue looking for simpler procedures with less drawbacks. With ACI based surgery, a second generation of matrix-induced autologous chondrocyte implantation (MACI) was applied by Verijen company, as described by D. Paschalis et al. The procedure is based on two structures. The first is a collagen membrane (types I/III) seeded with autologous chondrocytes. The chondrocytes are seeded on the cambium side, which allows attachment and neomatris synthesis. The other side is a smooth, non-bioresorbable thin polyethylene-like sheet allowing the smooth glide of chondral surfaces. The second structure is fibrin glue, a mixture of fibrin and thrombin, which sticks the membrane to the chondral surface. It has been proven that the glue is a stable surface for chondral ingrowth, has potential for immunogenicity and allows chondral migration throughout the defect space.

In a randomised trial comparing ACI on MACI, Bentley et al. found good results in 60% of patients in both groups. However, at histology, the ICRS scores were marginally better in the ACI group. The main drawbacks of the MACI treatment option were the high costs and the high risk of non-union of the regenerative tissue.
Current concepts of treatment of Osteochondral Defects of the knee
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Artificial chondroplasty
Several kinds of implants are used today, most are experimental, and are for focal chondroplasty, especially as a salvage procedure in elderly osteoarthritic patients. Focal chondroplasty by Co-Cr metallic implants, for the management of full-thickness cartilage defects in an animal model, was reported by Kirker-Head et al. After one year follow-up, the chondral lesions were much reduced radiographs, related to their sizes at implantation day. Their results implied the safety, biocompatibility and functionality of the implant.

Future trends/research
As in other fields in surgical medicine, the future is being shaped by bioengineering and modification of stem cells. Several trends already exist and research continues to find simpler ways for treating all kinds of cartilage defects and produce hyaline or hyaline-like cartilage with biomechanical and biostructural properties similar to human cartilage.

In 1994, using a rabbit model, Wakisani et al. reported that pluripotent stem cells, isolated from bone marrow, synovium or periosteum, could repair osseous and chondral defects. In 1995, Grando et al. reported that mesenchymal stem cells repaired cartilage defects and subchondral bone.

Autologous matrix induced chondrogenesis (AMIC) is a single procedure, aimed at cartilage repair by the patient’s stem cells. The defect is prepared and followed by microfracture. Then, Chondrolog is sutured to the crater edge. Early good results were obtained by this technique, especially in bigger lesions and in early osteoarthritis. However, its superiority over microfracture alone is yet to be proven.

Growth factors act as three-dimensional templates for cell migration and proliferation. Most experimental interest is in transforming growth factor (TGF) beta (mainly 1 and 3), a potent chondrogenic with osteoinductive properties, and bone morphogenetic protein (BMP) (mainly 2 and 7), a potent osteogenic but which aids in chondral and osseous proliferation and differentiation.

Gene therapy concentrates on manipulation of progenitor cells and chondrocytes to locally express genes encoding growth factors to enhance osteochondral repair. Mason et al. reported complete bone and articular cartilage regeneration at 8 to 12 weeks. This was achieved by modification of mesenchymal stem cells retrovirally transferred and seeded with BMP-7 on polyglycolic acid scaffold in osteochondral defects of rabbit knees.

Bioscaffolds are biomaterials that act as three-dimensional templates for cellular propagation and growth factors seeding. These could be natural (collagens, hyaluronan, fibrin glue, etc.) or synthetic (carbon fiber, polyglycolic and polylactic acids, etc.).