Presidential Guest Speaker's Address

What I Have Seen and Learned Since Looking Through an Arthroscope: 43 Years and Counting

I wish to relate what I have seen and learned since looking through an arthroscope, now some 43 years later. First and foremost there is the continuing resistance to arthroscopy. I always find it interesting to ask, "What orthopaedic subspecialty group was most against arthroscopy in the early days?" It is ironic that it was the sports medicine doctors of that day. The other group resisting arthroscopy was the open shoulder surgeons of that day, even publishing a letter to the editor of the *Journal of Bone and Joint Surgery* calling arthroscopy the "tool of the devil."[1] Their argument was as ridiculous then as today. They would argue, "Why would one look through a keyhole when one could open the door?" The implication was that surgical visualization was better via an arthrotomy than through an arthroscope. That was not so then and certainly not so now. The criticism continues, especially related to arthroscopy implementation for degenerative arthritis of the knee.[2,3] Subsequent publications have addressed what pioneer arthroscopists knew 30 years ago.[4] The meniscus is a component of a degenerative knee and not the cause, not the sole treatment, but perhaps a factor. Furthermore, meniscectomy is not curative for degenerative arthritis.[5,6] The literature asserts that arthroscopic knee surgery to "clean up" an arthritic knee is no better than fake surgery. The knee meniscus tears in middle-aged and elderly patients don't cause pain or problems with function. Arthroscopic knee surgery doesn't help middle-aged patients with meniscus tears and mild to moderate arthritis any better than physical therapy. Arthroscopic knee surgery for patients with a degenerative meniscal tear and no arthritis doesn't help patients any more than a fake surgery. The criticisms are now more wide spread and address much of orthopaedic surgery. Orthopaedic procedures have been called "scandalously poor" and "uneven evidence based."[7]

Are we to hold the following "truths as self evident?" When examining the medical literature, are we to assume that all doctors are the same, all surgeons have same skill level, all arthroscopic surgeries are the same, all surgeons have same level of knowledge, all surgeons have same level of judgment, all surgeons have the same technical skills, all surgeons have the same outcomes, and all authors are superb surgeons? I think not. To better understand the point I am making please consider the following statement: When Jordan Spieth, winner of the 2015 Masters Tournament, and Lanny Johnson are playing golf, are they doing the same thing?

We should have legitimate questions for authors, especially those reporting on arthroscopy for degenerative arthritis. Are they willing to respond to inquiry? Unfortunately I found it not so on two written attempts.[2] Do they have libraried videos for review? Looking at the video tapes would give insight to the expertise of the surgical technique. What was the patient selection? Was it possible that those subjects on or seeking disability might have poor outcomes? What was the exact diagnosis of every compartment? Were the posterior compartments inspected? What were the exact surgical techniques and the extent of the debridement? Was each compartment vacuumed of loose bodies; under and around each recess? What exactly was the postoperative joint protection compliance?

Before the advent arthroscopic surgery, it was known for sure that cartilage does not repair/heal. The surgeon must drill deep into bone to reach the subchondral blood supply. If any tissue responded, it would not endure. Certainly, there would be no clinical benefit.[8,9] Even reports some 18 years ago cited reservations about cartilage repair.[10]

These publications prompted me to have the subtitle for this presentation, "Just because something is known for sure, does not make it so!" In order to respond to these published criticisms, I would like to focus on the role of arthroscopic surgery in articular cartilage conditions and disease. I would like to review the FACTS and FACTORS and the FUTURE.

Such an effort requires a continuing pursuit to resolve the controversies. As arthroscopic surgeons, we must have data and evidence that cartilage tissue forms. I started computerized medical records in 1980 for data collection and documentation. We must show that the tissue endures. We must answer the question of whether fibrocartilage will endure. Is there a

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conversion in phenotype to hyaline cartilage over time? Is there any possible clinical benefit? Finally, we must address the possibilities for arthroscopic treatment of degenerative arthritis. The unique clinical observation provided by arthroscopy has a huge advantage with direct observation and libraried videos for review and biopsy.

Various open surgical debridement procedures were introduced for degenerative arthritis of the knee.[11,12] The result was a considerable morbidity including patient discomfort for up to 2 years as well as a positive bone scan from the large drill holes into bone. A similar procedure minus the drill holes was adapted to arthroscopy and facilitated by motorized instrumentation in 1975.[4] A minimal debridement of the articular surfaces and the degenerative meniscus was performed. In addition, a localized synovectomy was accompanied by vacuuming of all recesses for loose bodies. Unfortunately, vacuuming remains an overlooked technique by most arthroscopists. Experience with this debridement procedure revealed additional factors often overlooked in publications. These included minimal debridement and postoperative joint protection. It was also noticed that an immediate postarthroscopic blood clot formed on all injured and incised tissues would aid in healing.[13] Further it was recognized that a blood clot would spontaneously fill an articular cartilage defect. If the lesion was less than 2 cm in diameter and had normal articular cartilage integrity of the "shoulders" then a fibrin matrix would form and result in an articular cartilage repair. At an opportune second-look arthroscopy, such a finding was observed. In one such instance a biopsy showed articular cartilage being in the previous defect some 6 years after the index surgery.[4]

Today, an orthopaedic treatment cannot be limited to a single modality such as arthroscopy, or platelet-rich plasma, or stem cell injections. There are many more factors to be recognized. There are the patient demographics, general health, especially their expectations of outcome, and compliance with postoperative joint protection. The nature of the pathology must be considered. What is the alignment of the extremity? Is the joint stable? Is there articular congruity? What is the range of motion? What is the location and nature of the lesion? There are many biological factors. There is immediate postoperative bleeding and a fibrin clot matrix.[13] There is the bleeding and progenitor cell contribution from cancellous bone. Autogenous repair tissue and cells come from blood, remnant cartilaginous aggregates, which I will highlight shortly. There are stem cells heretofore not know from many sources; blood, vessels, fat, synovium, bone and cartilage.[14-18] Only the subchondral bone source of progenitor cells was known when microfracture was introduced.[19] In addition, the local environmental physical and chemical factors must be considered.

I would like to point out that the pathologic nature of the various degenerative lesions is often misunderstood. For instance, the Grade IV Outerbridge classification is used for 3 types of lesions; the end-stage degenerative lesion, the early degenerative loss of articular cartilage, and the traumatic lesion. They are very different in their pathological nature. The end-stage degenerative lesion is unique.[20] It has dead bone on the surface with empty osteones. The vascularity is at the surface has not been readily recognized by clinicians. There are spontaneous repair cartilaginous aggregates at and near the surface. The surface has multiple small pits due to erosion of cartilaginous aggregates, fissure bone, and ruptured blood vessels. These recesses may be the future home of therapeutics, i.e., stem cells.

The knowledge of the pathology of the end-stage osteoarthritic lesion conditions the nature and depth of the arthroscopic abrasion arthroplasty. The procedure is a superficial debridement of 1 mm so as to remove the dead osteones, expose the surface vascularity, and protect the cartilaginous aggregates. These are important surgical technique factors related to the nature of the lesion and necessary to enhance the articular repair.

The joint preservation factor is essential to postoperative tissue repair. In reports when patients were not compliant, the results were compromised.[21] When the joint protection factor was present and time factor was allowed, the patient undergoing this procedure had 50% wider joint spaces at 2 years.[4] Second-look arthroscopy with biopsy showed maintenance of fibrocartilage surface and even tide mark at 4 years. The emphasis again is on the time factor to facilitate healing.

Jack Bert's recent article in *Arthroscopy* challenged the use of traditional microfracture.[22] We owe a debt to Drs. Steadman and Rodkey for the advancement of cartilage repair.[19] Their initial indication was for traumatic articular lesions of incomplete depth. Subsequently they expanded their technique to degenerative lesions. However, their traditional technique made use of multiple punctures often over a small area. Potential complications were bone collapse and cysts.[21]

I consider the original technique one of **impaction and compaction**. The smooth awl used to create the wound was hit with a hammer and the method physically compacted bone on the margin of the bore. Such a surgical lesion has compromised healing.[23] An alternate procedure that incises and removes bone results in faster and more complete and mature articular cartilage healing in an experimental animal (M. Spector, written communication, January 2004). Therefore, if one is to do microfracture, use of instruments for **incision and excision** would be preferred over the traditional **impaction and compaction** method. Instruments to perform what I would refer to as the "new microfracture" have been introduced; a rake chisel and a

triangular awl (Medical Products Resource, Burnsville, MN).

A major factor often overlooked is the protection of the joint following treatment. The resulting noncompliance fails to protect the integrity of the matrix and results in biological and clinical failure. The potential benefit of unloading the joint has been known following hip or knee osteotomy. It is well known that an osteotomy of the hip or knee will result in a wider joint space, even without intra-articular surgery.[24,25] What has not been generally recognized is the potential for end-stage osteoarthritic joints to spontaneously regenerate.[26] The article by Guyton and Branch is most amazing. They reported 2 patients who had severe bilateral hip degenerative arthritis. The patients underwent total hip on one side and were scheduled for the other side in 3 months. When the patients returned, they said their other hip was better and they did not want surgery. Radiographs at 7 and 11 years showed not only a regenerated joint space, but remodeling of the bone. Eventually both patients came to surgery on the remaining degenerative hip. The articular cartilage showed hyaline cartilage. The question remains, how could this be possible? The patients without instruction or ambulatory aid spontaneously unloaded their affected hip over to the painless total hip. Over time there was regeneration. The likely cause was the cartilaginous aggregates on and in the surface of the end-stage degenerative lesion.[27] Zhang et al. reported the cartilaginous aggregates have all the histochemical nature of articular cartilage.[27] Further support for such a rationale is give by Milgram, who reported the pathology of 535 femoral heads from total joint surgery.[28] He showed abundant cartilaginous aggregates covering the surface, most likely for the same reasons as Guyton and Branch's patients. Those in Milgram's series were unloading a painful hip some months prior to surgery. There is even more circumstantial evidence that the aggregates have potential to regenerate a joint.[29] They reported that regeneration occurred after tibial osteotomy in Grade IV lesions but none in Grades II and III. The latter were not end-stage lesions with cartilaginous aggregates. With all this evidence, one certainly has to consider the factor of postoperative joint protection over a period of time.

Wedged insoles have been considered for unloading a medial or lateral compartment of the knee. The literature remains controversial. In my opinion, the controversy exists because no author to date has considered testing the potential compliant subtalar motion or the compliance of the knee joint collateral ligaments as an inclusion criterion.[30] Wedged insoles have been shown to be effective in independent testing on subjects with experimental total knees with load sensors. They may be considered as protective treatment of knee joint medial or lateral compartment conditions.

The question remains in many sectors concerning the nature and formation of articular tissue following arthroscopic surgical intervention. It may be argued that if it does form, it will not endure. To answer this question I refer you to a report on a long-term follow-up series of the most unlikely candidates for articular cartilage healing.[31] These were patients who had large 3-dimensional osteochondral defects of the medial femoral condyle. They underwent autogenous bone grafting of the defects. They were followed for 13 to 21 years with physical exams, radiographs, MRI, plus second-look arthroscopy and biopsy. The biological response was bone-to-bone healing and an early fibrocartilage surface regeneration that survived for many years as shown by MRI. At 20 years, a biopsy on one patient showed mixed hyaline and fibrocartilage at tide mark, and lubricin on the surface. These patients surgical knee was protected for 2 months following the index operation. Yes, there is evidence of biological repair and regeneration. It is now known for sure that articular cartilage will repair, tissue will form, and tissue will endure. The tissue was initially fibrocartilage and subsequently over time become hyaline in nature. Note that the factors are multiple and include arthroscopy, autogenous bone graft matrix, presence of stem cells in bone and elsewhere, joint protection, and the passage of time.

The question of potential clinical benefit remains in the minds of some.[10] Many patients undergoing arthroscopic abrasion arthroplasty were doing so as an alternative to total knee surgery. Their expectations were met in a high percentage of patients.[4] When the more severe pathological articular disruption of large-volume femoral defects of the knee were treated by arthroscopy, the results were similar regarding avoidance or delay of total knee surgery.[32] Even more amazing was one patient with the largest of bilateral femoral lesions. At 17 years following surgery, he had a normal range of motion of both knees and unaided normal ambulation. Notice the duration of the clinical benefit: 17 years. Yes there is evidence of clinical benefit following arthroscopic intervention for articular cartilage injury and disease.

There are many factors to consider when considering the role of arthroscopy in degenerative joints. Present day understanding has realized that degenerative arthritis is an inflammatory disease.[33] That being recognized, in retrospect, the original arthroscopic treatment for degenerative joints was unknowingly pathologically appropriate for an inflammatory condition.[4]

A factor yet to be addressed in the treatment of synovial joints is the optimization of the biochemical environment. The future treatment of the articular cartilage injury or disease, independent of the method, will have to include a means of optimizing the biochemical synovial joint environment. There is

evidence that certain phytochemical reagents will induce the human synovial explants to turn the gene on for IGF-1, long know to have a role in cartilage healing (US patent #8,263,069; September 11, 2012). There is unpublished animal experimental evidence that the same process will occur by injection and or pill. There is evidence that a phytochemical food supplement pill of cyanidin-3-glucoside and or its main metabolite, protocatechuic acid, will alter the biomarkers in a rabbit synovial joint. The animal had undergone a surgically induced degenerative arthritis of the knee. The synovial genetic makeup was changed to increase the gene of IGF-1 and decrease that of MMP-3. The synovial fluid anabolic/catabolic hemostasis is disrupted by the surgery. However the treatment reduces the amount of catabolic cytokines (IL-1, IL-6, TNF-α, MMP-3) and increases the amount of the anabolic cytokines (IGF-1, IL-4, IL-10). At the same time there was a decrease in the inflammation biomarkers in the blood of C-reactive protein and in the joint with decreased white blood cell counts. There was protection of the articular cartilage in the presence of the severe degenerative arthritis. The International Cartilage Research Society scoring was better in the treated group than in the controls. There was more aggrecans and type II collagen in the cartilage. There was more lubricin on the surface of the treated cartilage. The MMP-3 and IL-1β were decreased in the treated cartilage. Both chondronutrition and chondroprotection were observed in the treated cartilage. This evidence would suggest that cyanidin-3-glucoside and or its main metabolite may have a role in altering the joint chemical environment when articular cartilage is to be treated. Certainly this or something like this will be necessary to optimize the healing of the affected synovial joint.

Many important factors have been considered; the surgeon, the exact surgical technique, the patient selection, the understanding of the pathology, the understanding of the biological response of bleeding, blood and fibrin clot matrix, the appreciation of physical factors such as unloading the joint, and the future potential for optimizing the biological chemical factors in the joint with C-3-G and or protocatechuic acid. Last but not least is the time factor for recovery and maturation of the regenerative tissue.

With data, evidence, and factor recognition, I believe we as arthroscopic surgeons can meet the challenge. In summary, what I have seen and learned since I started looking through an arthroscope is the following:

Just because something is known for sure, does not make it so!

Arthroscopy has a role in cartilage injury and disease!

We now have relevant factors for success.

It is time for everyone to contribute to the advancement of the art and science of arthroscopy. This will best accomplished with everyone's participation in the AANA data collection initiative.

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