Knee Surg Sports Traumatol Arthrosc (2015) 23:636–639
DOI 10.1007/s00167-014-3323-8

EDITORIAL

Anatomic ACL reconstruction: The final answer?

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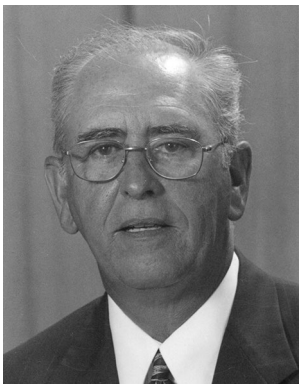
Published online: 30 September 2014



In the dark ages of knee instability, (1950–1970s), the majority of orthopedists thought that these were due to either medial or lateral rotatory instability and not due to injury to the anterior cruciate ligament injury and numerous procedures were developed to stabilize the knee. O'Donoghue [23], however, believed that the real culprit was a torn anterior cruciate ligament. Feagin and similarly Marshall later also came to the same conclusion and advocated direct repair of the ACL in acute cases [10, 19]. Our experience at the Naval Academy (1972–1974) with these various chronic reconstructive procedures as well as primary repair yielded very poor results.

In 1975, Eriksson [9] presented the Ivar Palmar procedure for ACL reconstruction at the AAOS Winter Injury Course in Aspen, Colorado. After this presentation, it was my belief that this procedure could be far more successful than what was presently being performed. After performing this procedure, it was evident that the patellar tendon graft if left attached to the tibia was too short to allow it to be placed in its anatomic insertion on the femur. A review of Scapinelli's study of the knee vascular revealed that there was no contribution of blood supply to the patellar tendon from its tibial insertion thus one could osteotimize that portion of the tibial tubercle with the patellar tendon graft producing a bone–patellar tendon–bone-free graft which would allow the graft to be placed anatomically both on the tibia and the lateral femoral condyle [27]. We also noted that for anatomic tunnels, eccentric K-wire placement on both the tibia and femur was necessary to compensate for the oval entrance tunnels created by drilling at an angle [5].

Our mechanical testing, histology and revascular studies in Rhesus monkeys and dogs revealed that the patellar tendon grafts were completely revascularized at 8 weeks mainly by the endosteal vessels in the tunnels, that the grafts histologically at 1 year presented as normal ligamentous tissue and that the grafts regenerated over 80 % of their initial strength [4]. Amiel and others further documented the process of biochemical metamorphosis of the tendon graft to that of a ligament which they termed "ligamentization" [1, 20]. Our clinical 2–8-year follow-up study in humans for both acute and chronic ACL reconstructions revealed an over ninety percent good to excellent overall results [5, 6]. Our criteria were that the highest overall score was that of the lowest score in any category of subjective, objective and functional evaluation. This concept was then adopted by the IKDC committee.

From the late 1970s to the late 1980s, the vast majority of the orthopedists in the world adopted this procedure. However, as my former history professor had taught me, "History is nothing more than taking two steps forward and then one step backwards". In the 1990s, over fifty percent of orthopedists in the USA started performing the

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transtibial endoscopic procedure where in the femoral tunnel was now drilled through a previously drilled tibial tunnel. The orthopedic journals over the ensuing years were filled with articles utilizing this technique routinely and claiming it to be anatomic. Our studies of this technique in cadaver knees revealed that it was rarely possible to achieve anatomic placement. In the late 1970s, we discovered that there is a bony ridge which demarcated the anterior and superior insertion of the ACL on the lateral femoral condyle and termed it the resident's ridge and it was this landmark we utilized to determine whether, indeed, the transtibial approach could achieve anatomic placement [14, 15]. Additionally, Girgis and Marshall documented that the ACL's anatomic insertion on the tibia was anterior to the base of the tibial spine [12]. The traditional transtibial approach required drilling through the tibial spine then upwards to the 11:00–12:00 o'clock femoral position.

Our plea of anatomic graft placement for the most part went unheeded during this period of time. As a result of this, we believed that a scientific study was necessary that would clearly document exactly where the anatomic insertions of the ACL were and document the bony landmarks that would delineate their boundaries. Utilizing CT, sophisticated computer software and methodology as described by Hounsfield, we were able to clearly define the exact anatomic insertion sites and the bony landmarks [13, 24]. This research also confirmed that the resident's ridge was the anterior superior insertion site of the ACL on the lateral femoral condyle as well as Girgis and Marshall's work on the tibial insertion of the ACL just proximal to the anterior edge of the tibial spine. Further studies by Ferretti et al. [11] confirmed our studies while adding a ridge perpendicular to the resident's ridge which he initially termed the bifurcate ridge which separates the ACL insertion into two parts referred to as the anteromedial (AM) and posterolateral (PL) bundles. In our studies, the anteriomedial bundle comprising 70–80 % of the entire insertional area.

From what has been stated above, one might believe that anatomic placement is the final answer to ACL reconstruction. However, one would be wrong to conclude this. There are other factors which must also be addressed to achieve a higher success rate. There are still a number of prominent surgeons who believe that they can achieve anatomic placement with a modified transtibial technique. They claim that they can achieve femoral anatomic placement which is some cases they can. However, to achieve this, they usually perform a significant notchplasty particularly on the medial wall of the lateral femoral condyle and drill their tibial tunnel too posterolaterally. This produces a graft that is shorter because the graft is now more lateral and posterior than it should be. When the knee is placed at or near full extension, the graft can be seen to almost abut the previously lateral notchplasty and a lateral roentgenogram will show that the posterior wall of the tibial tunnel enters the tibial spine. Further, this large notchplasty increases the space between the normal lateral femoral condyle and the lateral intercondylar ridge of the tibia which can produce more medial to lateral femoral sheer force on the articular surfaces.

Of far greater concern, however, is the decision on graft choices. The literature contains numerous conflicting reports on the choice of autografts, namely patellar tendon, hamstring tendon or quadriceps tendon. The consensus is, however, that there is no statistical difference among them with a very high success rate with all of these grafts when placed anatomically. Allografts, however, have produced multiple problems because of preparations including the use of ethylene oxide and high doses of irradiation. Presently, in the USA, over 50 % of surgeons utilize allografts.

At present, the use of allografts should be highly suspect. The animal research on autografts clearly documented that autografts in humans should regenerate graft strength to that of an ACL [4, 17]. However, animal studies utilizing allograft tendons have clearly documented that allograft tendons used in humans should not regenerate anywhere near the strength of an ACL particularly in those younger than 39 years of age. These animal studies have documented that allografts do revascularize and undergo "ligamentization", but their maturation is quite slow and that at 1 year, the graft strength is only 50 % of the control autograft [4, 7, 16, 28]. Animal studies have documented that autografts regenerate 80 % of their original graft strength; however, an allograft only regenerates 40 % of its original graft strength at 1 year [16, 28]. There have been no reports of tendon graft strength in either autografts or allografts at 18 or 24 months in the animal model.

Studies in animals have documented that allograft tissue, tendon, ligament, menisci do produce an immune response even if the tissue is deep frozen [2, 3, 26, 29, 32]. Vasseur found that synovial fluid from the knee joint of all six dogs who underwent allograft bone–ligament–bone ACL reconstruction tested positive for anti-dog leukocyte antigen, whereas the autografts were negative [32]. A study in humans having undergone deep frozen tendon allografts found a humeral immune response by noting lympocytic anti-donor IgG production to be present in 38 % of the allograft patients [29]. We have noted four patients with fulminent rejection reactions to allografts, two in the shoulder for rotator cuff reconstruction and two in the knee, one of which had been treated 2 years previously for idiopathic thrombocytopenic purpura. It is not unreasonable to suspect that a localized immune response may play a significant role in why allografts do not achieve the same tensile strength at least in the animal model.

In a review of the world's literature, there has been reported only 123 fresh frozen human cadaver human knees tested for ACL strength [17, 18, 21, 22, 25, 31, 33].

The average strength of the ACL in those knees 39 years and younger was 1,532 N, those 40–59 was 1,062 N, and those over 60 was 648 N. The average strength of a 10-mm patellar tendon in those under 35 was 2,860 N [8]. Stäubli's study of unconditioned and conditioned 10-mm patellar tendon in 16 specimens under the age of 30 years noted the unconditioned ultimate tensile strength averaged 1,965 N and the conditioned to average 2,410 N [30]. Theoretically, utilizing a fresh frozen 10-mm patellar tendon, allograft from an under 30-year old for a patient under 39 years would produce at 1 year a graft strength between 964 N and 1,144 N when the average ACL strength for this age group is 1,532 N. If this was truly to be the case, it would not justify the use of an allograft in this age group nor would it make sense logically or scientifically to utilize an allograft for a revision when autograft tissue is still available.

Anatomic ACL reconstruction has significantly advanced our success rate, but it is just a part of a still unsolved problem. Our clinical studies suggest that excessive physiologic posterolateral laxity may play an important role in predisposing females with femoral anteversion and concomitant external tibial torsion and over pronation of the foot to tear their ACL and repeat graft failure. Additionally, it is quite difficult to evaluate long-term arthritic knee changes in reconstructed knees due to concomitant meniscus tears, articular surface injuries and subtle capsular and other ligament injuries at the time of initial injury. The quest continues.

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