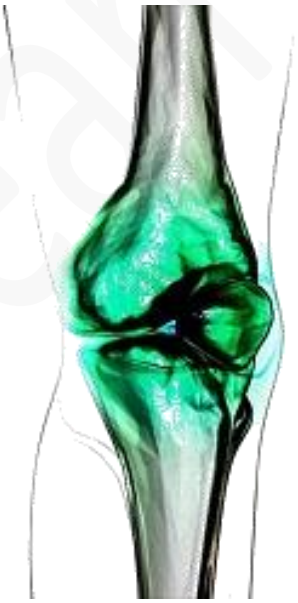
THE FINAL MORPHOLOGY OF ANTERIOR CRUCIATE LIGAMENT GRAFTS

ORTHOPAEDIC PRESENTATION



Dear colleagues and interested parties,

This document is part of a series of Orthopaedic Papers drawn from the past 40+ years of medical practice I have enjoyed, primarily focused on the treatment of knee injury and degeneration.

The series includes a mix of conference papers presented over the years, as well as general knee injury management reference documents covering some of the challenges and solutions developed during this time.

We needn't reinvent the wheel too often, so I hope these documents prove useful to my fellow surgeons and those interested in the treatment of knee injury, degeneration, recovery and patient care.

Thank you for taking the time to read these papers, and please do not hesitate to reach out to discuss any of the issues covered further.

Iain

Mr Iain D McLean
MBBS FRACS (ORTHOPAEDICS)
Orthopaedic Surgeon / Consultant

Contact

Mr Iain D McLean

MBBS FRACS (ORTHOPAEDICS)

W: www.iainmclean.com.au
E: iain@iainmclean.com.au

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Presented by:
Mr Iain McLean
Mr Owen Deacon
Mr Barry Oakes
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THE FINAL MORPHOLOGY OF ANTERIOR CRUCIATE LIGAMENT GRAFTS

ESSKA – FIRST WORLD CONGRESS IN SPORTS TRAUMA

MAJORCA – SPAIN

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THE FINAL MORPHOLOGY OF ANTERIOR CRUCIATE LIGAMENT GRAFTS

Mr Iain McLean
Mr Owen Deacon
Mr Barry Oakes

THE PURPOSE OF THIS PROJECT is to show that all biological materials used as intra-articular ACL grafts are replaced in the hostile environment of the knee by small diameter collage fibres.

With the FINAL STABILITY being seen as a spectrum, dependent on the individual's biological fibrous response to the injury and surgery, and not directly to the initial mechanical properties of the graft.

OUR ACL RESEARCH has encompassed a combination of clinical work, biopsies of human ACL grafts, and animal work.

But recognising the limitations of each.

Now, the STRENGTH OF ANY MECHANICAL SYSTEM, in this case the reconstructed ACL –

is dependent on its size, its shape, its attachments

and the materials it is made of and their organisation.

THE NEW ACL'S SIZE AND SHAPE is determined by the anatomical confines of the intercondylar notch,

As illustrated by excising the ACL in an animal, and placing a "blob" of play dough or bone cement in its place.

Provided we go through a full range of movement, then an exact replica in size and shape to the original ACL is formed. (It can be smaller, but not larger)

In the GOAT STUDIES, using a patellar tendon "over the top" method of reconstruction,

provided we are isometric so as to prevent graft breakage; and create a raw bone surface;

biological fixation would occur within 3 to 6 weeks, and the graft with time and motion would resemble a normal ACL with its exact anatomical insertion points.

However, when STUDIED MICROSCOPICALLY, there were differences –

we see on the left normal goat ACL in cross section, with the fascicular bundle formation,

and in the longitudinal section, the wavy collagen crimp pattern.

In patellar tendon grafts, by 3 weeks there is a massive cellular invasion and commencement of re-organisation.

But even AT 12 MONTHS, we see that although macroscopically it resembles a normal ACL, it has not formed the fascicular pattern, and remains less organised.

WITH ELECTRON MICROSCOPY – on the left upper frame is a normal goat patellar tendon,

and the lower frame, the normal goat ACL.

On the right, we see replacement of the large diameter fibres, by these small fibrils.

This ACL graft collagen remodelling is maximal in the first 3 to 6 weeks.

Greatest initially at the periphery, and the tibial end, and progresses into the centre of the graft and proximally.

The large patellar tendon fibrils >100 nm are rapidly depleted within 6 to 12 weeks,

with the small <100 nm predominating after 6 weeks.

But with remodelling occurring over the 12 month study period.

This remodelling can be summarised in this "whiz-bang computer graph".

But as many other workers have found in animal studies, the ULTIMATE TENSILE STRENGTH of the graft is only 30% to 50% that of the original ACL.

IN THE HUMAN – Owen Deacon and myself, with some assistance from colleagues, have now biopsied well over 100 ACL grafts

- from 4 months to 20 years post-surgery
- grafts of patellar tendon, hamstrings, iliotibial tract and allografts
- from different surgeons
- different operative techniques
- and different postoperative rehabilitation programs

MACROSCOPICALLY there is a SPECTRUM

- from a small flimsy lax graft
- to those resembling a normal ACL
- through to poorly defined grafts with almost complete intercondylar fibrosis

After clearing the debris, and synovium if necessary, the fibres on probing separate more easily and are less tightly packed than the normal ACL.

ON ELECTRON MICROSCOPY

- all grafts show identical unimodal small diameter collagen fibres; as seen at the top of the left slide

- compared to the normal ACL in the centre with medium fibres
- and the patellar tendon at the bottom, with bimodal large fibres

The accompanying FIBROBLASTS show

- active rough endoplastic reticulum
- but do not appear to be native tendon cells
- they are most likely of synovial stem cell origin

COLLAGEN TYPING – was performed on 20 ACL grafts.

This, taken in conjunction with the EM studies, confirms the creeping substitution or ligamentisation of the human graft tendons;

- from the tendon, consisting of bimodal larger collagen fibres and being approximately 100% type I collagen,
- to the final ACL structure, being small diameter collagen fibres with 70% type I and 30% type III collagen
- type III collagen is seen in healing tissues, and indicates repair and turnover within these ligaments.

SO WHAT!

The ACL graft then:

Is simply an avascular denervated band of collagen,

The cells and collagen of which may have no ability to survive "long-term", or reproduce, in the knee.

It appears to act purely as a scaffold and STIMULUS TO SYNOVIAL FIBROBLASTIC ACTIVITY.

The tendon graft undergoes biological change, but

QUALITY COLLAGENISATION and maturation of fibres does not occur in the presence of inflammatory cellular activity.

The "LIGAMENTISATION" is dependent on the balance between

inflammatory cellular activity with macrophage breakdown

and the fibroblastic production of collagen.

CLINICALLY, we see a spectrum of "BIOLOGICAL RESPONSES" to injury and to our intra-articular surgery.

1. Some knees show NO RESPONSE
   - with very little pain
   - with almost immediate muscle control
   - rapid range of movement

but with progressing stretching and laxity

- the graft arthroscopically is small, flimsy and lax.

2. If we have a significant INFLAMMATORY RESPONSE
   - there is an effusion, with increased cellular activity
   - with soft, boggy, stretchable tissues
   - and with the articular surfaces at risk from lysosomal activity.
3. A DRY FIBROUS REACTION results in synovial fibroblastic activity
   - and collagen deposition into, onto, and around the graft.

(a) With "QUADRICEPS INHIBITION" and lack of patellar and joint movement

- we have poorly organised deposition in the intercondylar notch, suprapatellar pouch and lateral gutters.

The fat pad fibroses, and the patellar tendon contracts

- leading to patella infera, and a block to extension
- with resulting patellofemoral problems and degeneration

This is seen more commonly in –

the acute knee

with patellar tendon grafts

with arthrotomy

the older age group

and those not adequately "prepared" for surgery

(b) If, on the other hand, we GAIN ACTIVE QUADRICEPS FUNCTION

- with proximal patellar movement, and good joint motion
- the collagen deposition is moulded in the intercondylar notch to form a functionally stable knee
- that can be more rapidly rehabilitated back to active sports.

SO FAR,

OUR CLINICAL AND ANIMAL STUDIES show that the only correlation with "STATIC JOINT STABILITY" –

is the "final" intercondylar collagen mass (represented by the straight line graph)

and its organisation.

This does not appear to correlate directly with the initial graft size and strength.

IF WE SUPERIMPOSE this graph on our Normal Histiogram Curve we can appreciate our arthroscopic and clinical findings.

IN CONCLUSION

- we have studied over 100 human ACL graft biopsies, plus goat work
- this indicates that all grafts act purely as a scaffold for "synovial" fibroblasts, and are replaced in the "hostile" environment of the knee, by small diameter collagen fibres.
- The collagen typing confirms a turnover of collagen, and a constant repair pattern.

The FINAL STATIC STABILITY appears to depend on our ability to trigger or initiate the fibrous reaction in the joint

- and to mould this within the confines of the intercondylar notch.

THE "FUNCTIONAL" STABILITY IS MULTIFACTORIAL, WITH SURGEONS NEEDING TO CONSIDER THE BIOLOGICAL, AS WELL AS THE TECHNICAL, ASPECTS OF ACL RECONSTRUCTION; AND THE FINAL OUTCOME BEING RELATED TO THE MATURATION OF A "COMPOSITE" OF GRAFT AND FIBROUS/SCAR TISSUE.

Please visit https://www.iainmclean.com.au/ for further information and links to reputable online orthopaedic resources.