

SCIENTIFIC BASES IN ACCELERATED PHYSIOTHERAPY IN THE RECONSTRUCTION OF LCA WITH PLASTIA H-T-H. SECTION II

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• REGARDING MOBILITY: Consequences of Immobilisation:

1) ON THE CARTILAGE:

-Experimentally, joint immobilisation produces different modifications on the cartilage:

1) Cartilage softening (chondromalacia)

2) Joint space reduction.

3) Irregularities on the joint surface.

-There will also exist a reduction in glycosaminoglicanes (up to a 48%, mostly on the articular surface)

Kiviranta, 1987.

-**Troyer, 1975** : has demonstrated that there are modifications on the cartilage hydration.

-**Evans, 1960** : Demonstrated the existence of subchondral osteoporosis.

-These changes start quickly and become permanent after 8 weeks of immobilisation.

-It has also been confirmed that there are necrotic areas on the cartilage-cartilage contact points when an axial compression is applied (**Soltem, 1960**).

-After some time, fibro-adiposity adherences are confirmed on the cartilage surface.

-**Salter, 1982** : he verified with rabbits that the cartilage defects scar better when the joint is applied to a

Passive Continuous Mobilisation (MPC). The worst results were obtained when the joint was immobilised.

2) ON THE SYNOVIAL :

-The continuous immobilisation favours the increasing of the fibro-adipose tissue in the joint and, therefore, the creation of adherences in the bottom of the synovial sac (**Evans, 1960**).

-He verified also that Force Mobilisation produces superficial abrasions at cartilage level in animals.

-**Paulos, 1987** : He has described an infrapatellar contracture syndrome related to fibrosis on the knee's front side (Patella infera syndrome due to arthrofibrosis), and comes along with flexion and extension loss.

3) ON THE TRANSPLANT :

-Immobilisation doesn't guarantee a normal movement of synovial fluid which is essential for the neoligament nutrition, specially during the revascularisation stage along the first post-operative weeks (Mc. Donough).

-**Amiel, 1983** : He demonstrated that there exists knee peripheral ligament atrophy besides the fact that the collagen degradation increased. If immobilisation goes from 9 to 12 weeks, collagen degradation is respectively from 14 to 28%, more elevated than the NON immobilised rabbit group. An exponential degradation continues as long as immobilisation persists.

-**Akeson, 1984** : He confirms a collagen cellular and fibrillar lack of organization. The immobilised ligament becomes less rigid and less resistant. For a same weight, the ligament shows an increased deformation

-The resistance to side ligament or LCA rupture shows a clear reduction of resistance around 1/3 in the immobilised group, besides the energy which has been absorbed before the rupture (**Noyes, 1977**).

-The phenomenon is increased by the fact that there is a weakness on the bone anchorage, which becomes

osteoporotic (**Woo, 1984**), and he verifies besides that the recovering after a long immobilisation is slow and incomplete.

-**Hart, 1987** : in a study with animals, he shows that active mobilisation encourages a more solid mechanically. The immobilisation seems to generate neosynthesized collagen which organises itself in such a way to resist better traction forces (following the force lines)

-**Woo, 1984**. This author points the effect of capsule injuries. When the capsule is intact, mobilisation has a positive effect on the ligament scarring. But if the capsule is broken, mobilisation will have a negative effect.

• HOW SHOULD YOU TREAT MOBILITY PROTECTING THE PLASTY:

-First, you should fight the joint effusion: drainage, ice, antiinflammatories, compressive cryotherapy.

-**Noyes, 1987** : In a prospective study, he demonstrated that early mobilisation does NOT increase the joint effusion percentage.

The hyperextension produces tension or overload in the transplant. For **Markolf**, these overload goes from 50 to 500N for 5° of hyperextension. This value is less than the transplant mechanical value and the fixation systems. Tensions on LCA increase after 120° flexion, but staying beneath the reasonable limits. FIG-1:

-**Wascher** : He demonstrated on 18 cadavers that there is a medium tension of 180N for a 10Nm in hyperextension on LCA.

- At LCP level, tensions during mobilisations are less important. In this case, there is LCP tension starting from a flexion of 60°.
 - Knee mobilisation is another key in the recovering joint amplitudes in the immediate postoperative stage. If there is a lack of knee mobility on the front and vertical plane due to a quadriceps retraction, it will slow down the flexion and extension recovery, producing femur-patellar pain.
 - The complete extension recovery and the fight against flexum are a priority without danger for LCA neoplasty. The passive flexion recovery beneath the pain threshold is also very important.
 - The 0° extension must be achieved before the 14th postoperative day and a flexion of 120° in 6 weeks time.
 - Lerat, 1982** : He foresights that an early passive mobilisation beneath the pain threshold from 0-120°.
- Monitoring apparatus play an important role (MPC).