

Current Concepts in the Management of Muscle Tendon Unit Dysfunction

4th March 2006, Grange City Hotel, London
Jane's personal notes on the nine lectures attended

Muscle tendon unit stiffness – an etiological factor for muscle strain, Glenn Hunter, University of the West of England, Bristol, UK

Glenn is doing a PhD looking at the effect of stretching on the active stiffness of the hamstring muscles, funded by the FA.

We can only identify two risk factors to hamstring injury: (1) risk increases with age, (2) having had a previous injury. Neither of these is modifiable.

When a player reports the pain of an injury may not be at the time of injury, but afterwards. Injuries most commonly occur at the end of the first half, and the end of the second half of a football match. Therefore fatigue may be an important factor.

- Injuries mostly occur when a player is sprinting forward, weightbearing on a flexed knee, NOT in non-weight bearing knee extension. This movement occurs at the end of the swing phase, just at the beginning of the stance phase in the gait cycle.

- 80% of hamstring injuries occur in biceps femoris.

Interestingly, players with *stiff* hamstrings sustained *less* in injuries than those with less stiff hamstrings. Glenn's hypothesis is that stiff hamstrings result in less deformation of surrounding structures, whereas less stiff hamstrings lead to more deformation and therefore more injury.

- Injuries occur during *active* stiffness.

Glenn's researchers measured 2000 people for hamstring stiffness using a specially designed apparatus that mimicked the phase of the gait cycle, in which injuries occurred, i.e. hips flexed, knee flexed to 30 degrees.

- Female muscle stiffness decreases during menstruation.

- As we fatigue there is a *decrease* in muscle stiffness.

- We modify stiffness in response to different surfaces. For example, active muscle stiffness is different when we walk on carpet compared to when we walk on concrete.

- Stiffness prevents deformation at a joint and is regulated by the reflex mechanism. The stiffer the muscle, the more energy it can absorb, and therefore the more resistant you are to injury.

- Strength does *not* equate to stiffness. Therefore, strong people do not necessarily have stiffer muscles. In an investigation of a professional footballer with a history of 6 recurring injuries researchers found a decrease in muscle stiffness on the injured side.

Can you change stiffness values? We can boost strength but seem unable to alter stiffness.

The muscle tendon unit is not uniformly stiff. Standing and leaning forwards at the ankle has traditionally been thought to be brought about by eccentric contraction of the plantarflexors, i.e. gastrocnemius etc. lengthening whilst under load. Researchers (Kawakami et al, 2002) discovered that actually, there is a shortening of the plantarflexors (i.e. concentric contraction) but a lengthening of the *tendon*.

Before jumping to the conclusion that we should avoid stretching we need to ask whether stretching actually decreases muscle stiffness (see notes for lecture 4). Perhaps we need to be doing exercises that increase the pliability of the tendon?

The etiology and pathology of muscle strain – current evidence, Professor Tom Best, USA

Tom's research focuses on the pathophysiology and treatment of muscle strain injuries.

Tom showed us slides of injured muscle tissue and asks, "how do we define injury?" damage to muscle fibers is not always apparent light microscopy but show up under an electron microscope.



Not all injuries are the same: in a study of sprinters and dancers researchers found that sprinters sustained injury during sprinting and dancers sustained injury during stretching. The initial loss of strength and flexibility was greater in the sprinters. Are there different mechanisms of injury?

Injured people report feelings of “stiffness” in muscles in the tie leading up to the injury.

The timing of activation of the muscle-tendon unit may be critical. In other words, the motor nervous system may play a key role in preventing injury.

Neutrophils, a key inflammatory marker, are present in damaged muscles. They appear almost immediately after injury and produce free radicals. 24 hours post injury there are more damaged muscle fibers than at the initial time of injury. Interestingly, free radical production by neutrophils is at its highest 24hrs post injury. Neutrophils may therefore be responsible for this secondary muscle damage. It is not clear why this secondary muscle damage is important in the repair process.

If anti-inflammatory drugs are to be used in treating muscle damage they must be used early.

In one study, researchers blocked the neutrophil’s respiratory burst, preventing free-radical production.

There was less muscle damage following this intervention. However, in a study by Teixeira (2003)

depletion of neutrophils resulted in a deficient regenerative response in mice. Neutrophils may have a role to play in muscle fiber regeneration by activating satellite cells.

Macrophages are responsible for muscle regeneration.

In addition to neutrophils and macrophages calcium may also play a key role in muscle regeneration. If you block calcium channels you get less muscle damage.

It is possible that stretching a muscle may bring neutrophils into an area.

Muscles heal by scarring. Toms researchers are able to identify and quantify the extend of scarring.

Current evidence for the surgical and conservative management of Achilles tendonopathy, Professor Nicola Mafulli, Keele University, UK

Professor Mafulli pioneers minimally invasive surgical techniques in the treatment of tendon injuries and is an expert in ACL, foot and ankle surgery.

We do not know the risk factors associated with Achilles tendon problems.

We should be using the term “tendinopathy” to describe these conditions and tendinitis has traditionally been used to describe inflammation and there is almost no inflammation in problems associated with the Achilles.

The Achilles may take 100 days to synthesis its main structural protein. Therefore it is slow to heal.

Tendons can sustain up to 17 x our bodyweight

There are no validated conservative management protocols for the treatment of Achilles tendonopathy.

Mr Mafulli will only operate after there has been 3-6 months of conservative treatment

The Achilles tendon is only 5mm thick. Even in large, elite football players it is only 7mm.

The paratendon should be exceptionally thin, not a second skin. Mafulli showed us slides of a thickened incised paratendon and asks whether such a structure could compress the Achilles tendon, disrupting its physiology.

The Achilles is made form type I collagen. Tenocytes from tendonopathies produce collagen type III.

We don’t have a word for the failed healing response of the Achilles tendon so was describe it as “degradation”.

A common way to help the healing process is eccentric strength training of the plantarflexors of the ankle. However, eccentric heel drops don’t always work.

The role of stretching in the prevention of muscle tendon unit injury, Glenn Hunter, UK

- Whilst animals stretch after inactivity, they do not stretch prior to exercise/physical activity. Why do we think we ought to be stretching before a burst of physical activity? Glenn suggests that it is the muscles of respiration that we stretch instinctively after a period of inactivity.

- Is “non-contractile” tissue really non-contractile? The non-contractile element in skeletal muscle is titin and perimysium.

- Tissues may stiffen with inactivity and become thixotropic with stretching.

- It does not matter how you stretch, all forms are equal (with PNF being ever so slightly superior)

- It is very easy to change flexibility. Best way is to do 5 repetitions each lasting 15-30 seconds.

- If we stop stretching we return to our pre-stretch values within about 3-6 weeks. There is a genetic point to which we return.
- Note after stretching *stiffness stays the same* but ROM increases.
- Glenn suggests that we desensitize the response to stretch. In other words, we get an increase in ROM due to increased stretch tolerance not as a result of increased compliance. The neural component to stretching is therefore significant.
- Stretching has a minimal (5%) effect on reducing the risk of injury.
- However, we should stretch and mobilize injured tissue in order to return it to normality.
- Stretching seems to make DOMS worse. In one study, his researchers got subjects to do step-ups for an hour in order to induce DOMS. The subjects were then put into two groups and one group given stretches to do. All subjects were tested at intervals for their response to pressure on the calves. The group who did the stretching reported *more* pain in response to calf pressure than the group who did no stretching. (Pressure applied was obviously the same in both groups and was applied mechanically).
- We store energy in non-contractile tissue.
- Immobilization leads to contractures in non-contractile tissue and an increase in stiffness.
- Non-contractile tissue contains myofibroblasts. When you have a wound the myofibroblasts (containing smooth muscle tissue) bring the edges of the wound together. Unfortunately, when they are in the perimysium this leads to conditions such as frozen shoulder and Dujptren's Contracture).
- Tibialis posterior is *not* a pronator. It decelerates tibial rotations and is therefore an *anti*-pronator.

Scientific guidelines for the rehabilitation of muscle strain injury – current evidence, Professor Tom Best, USA

Hamstring injuries occur during the initial ground contact when knee flexion/hip extension moments are largest.

Best hypothesises that muscles would be damaged if they have to undergo a large peak stretch and that by increasing tendon compliance you increase elastic energy storage and reduce peak muscle stretch. In other words, the tendon and muscle work together and by improving the function of one you may improve overall function and perhaps decrease the likelihood of injury.

Peak stretch in biceps femoris occurs in the late swing phase.

When you vary the length of the tendon this has a huge effect on the length of the muscle and on muscle power.

Muscle injury produces short-term neurological alterations that must be overcome in a successful rehabilitation programme. In his research on injured sportspeople one group incorporated core stability exercises and one group used stretching. The stretching group took longer to get back to sport and some of them were re-injured compared to the core stability group who returned to sport more quickly and sustained no further injuries during the study period. He concluded that “progressive dynamic neuromuscular control and core stabilization drills appear to be effective at secondary injury prevention”.

The conservative management of rotator cuff tendonopathy – current evidence, Julia Church, UK

Julia is the physiotherapist for the Harlequins Rugby Club currently doing a PhD in rotator cuff impingement syndrome.

The mechanism for impingement is both extrinsic and intrinsic. Extrinsic factors are sub-acromial impingement and internal impingement of soft tissues under the acromion, Intrinsic is tendon degeneration.

Dissection of cadavers has shown acromial spurs, so there is therefore some mechanically induced microtrauma resulting in inflammation of supraspinatus and infraspinatus tendons.

Most acromioplasties fail. This is because the tear is not on the bursal side. There must therefore also be some tendon degeneration, i.e. intrinsic factors responsible for this condition.

Tendon degeneration reduces the tensile strength of the tendon.

At the insertion point, there is less type I collagen in the tendon, plus poor vascularity. (Type I collagen also decreases with ageing, hence age being a risk factor).

Progressive loading increases collagen synthesis, improves fiber alignment and increases proteoglycan content.

When and how should we load? Continuous Passive Movement (CPM) mitigates strength and collagen



losses. Eccentric loading normalizes tendon structure, reduces pain and improves function. Ice is beneficial at reducing pain.

Treatment should progress from passive exercises, to active assisted to active.

Julia showed us some excellent video footage of players performing rotator cuff strengthening whilst resting/sitting on an unstable surface.

The role of orthotics in managing muscle tendon unit dysfunction – current evidence, Alison Middleditch, Surrey Physiotherapy Clinic, UK

Alison is a bioengineer, researcher and has just written 2nd Edition of *Functional Anatomy of the Spine*.

In a study of 58 healthy women, only 17% had “normal” alignment. There is no such thing as “normal” foot alignment.

There is a weak correlation between foot pronation and overuse injuries.

Our foot angles change throughout the day.

Podiatrists take a measurement of our feet in bilateral stance. Yet foot position changes enormously in single stance.

There is no evidence that orthoses significantly change rearfoot kinematics.

The plantar fascia reproduces the windlass mechanism.

Foot orthoses reduce the force needed to establish the windlass mechanism, but do not change the calcaneal angle.

“Prefabricated orthoses increase activation of vastus medialis and gluteus medius muscles during squat and step-down exercises”

In a study of Canadian military soldiers chose orthotics based on comfort and for the next 4 months wore those they preferred. Those wearing orthoses had 40% fewer injuries. The authors concluded that comfort was of clinical significance.

In a study of 523 subjects given orthoses for foot condition 63% had experienced complete resolution in symptoms at 14 weeks post issue; 93% said their condition was completely or partially resolved.

It seems to make no difference whether orthoses are custom made or prefabricated.

The role of NSAID's in the management of muscle tendon unit dysfunction – current evidence, Dr Stan Wyngaerden, UK

Stan is musculoskeletal medicine consultant at Eastbourne District General Hospital. His special interest is in myofascial disorder and spinal pain.

The stages of muscle injury are (1) degeneration, (2) inflammation, (3) regeneration, and (4) fibrosis.

Cells present during the process are platelets, endothelium, neutrophils, macrophages, T-lymphocytes, fibroblasts, mast cells and neurons.

A) Platelets are responsible for coagulation, inflammation and repair and release factors signalling to other cells to repair tissue.

B) Endothelium is found on the inside of blood vessel walls and is an endocrine organ weighing 1800g. Is neovascularization (the growth of new blood vessels) a contributory factor in pain in tendonopathies?

C) Neutrophils are highly destructive and kill bacteria through phagocytosis as well as producing oxygen free radicals. They exacerbate mechanical damage and have a negative role in exercise induced muscle injury (DOMS). They are *absent* in tendonopathies.

D) macrophages appear in later stages of inflammation. There are resident macrophages and invading monocytes. All are versatile and produce up to 100 chemicals. They are *scarce* in “tendinosis” biopsies.

E) T-Lymphocytes are found in the remodelling phase (14 days). Reduction of T-lymphocytes delays healing, reduces collagen production and leads to the formation of a fragile scar.

F) Fibroblasts secrete inflammatory mediators in response to mechanical loading.

G) mast cells act like security alarms and activate nearby cells.

H) Neurones influence all of the above cells as they release a number of chemicals. Denervation leads to reduced healing.



There are 2000 deaths every year in the UK from NSAIDs, due to gastrointestinal bleeding. There are 12,000 hospitalizations each year in the UK from NSAIDs. When used with muscle injury, there is only modest inhibition of the initial inflammatory response and its symptoms. They have no significant long-term benefit in the management of tendinopathy.

Clinical reasoning and the management of muscle tendon unit dysfunction – putting it into practice – the views of the faculty

Summary

The panel took comments from the floor. Overall guidelines were not to use NSAIDs nor corticosteroids in the treatment of tendonopathies; lower limb rehab may be different to upper limb rehab; eccentric strength training still useful; ice useful for pain; frictions are useful; conservative surgery recommended (e.g. needling); role of neutrophils uncertain but definitely not present in tendonopathies; only two risk factors (age and previous injury) for muscle/tendon injuries; recovery is 12 weeks without aggravation; do not use ultrasound on the Achilles tendon.

Notes on the overall symposium

Good points:

- * Well-qualified, well-informed speakers
- * Notes provided of eight of the nine power point presentations
- * Certificate of attendance provided (valued at 6 hours).

A feedback form was handed out at the end for comments.

Great venue: few minutes walk from Tower Hill tube. Circular tables for approx. 150 attendees

10 textbooks given away free by the organizers to attendees who found a sticker under their chair!

Overall, great value for money. £98. All teas, coffees, morning patisseries, afternoon cakes, and lunch provided. Organized by Wellbeing CPD.

Bad points:

- * None

Improvements:

None

Jane Johnson MCSP, MSc, BSc, BA(Hons)

Co-Director and esteemed author of *Soft Tissue Release: Hands-on Guides for Therapists*