



TISSUE SPECIFIC EXERCISES



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Tissue responses to various levels of exercise stress

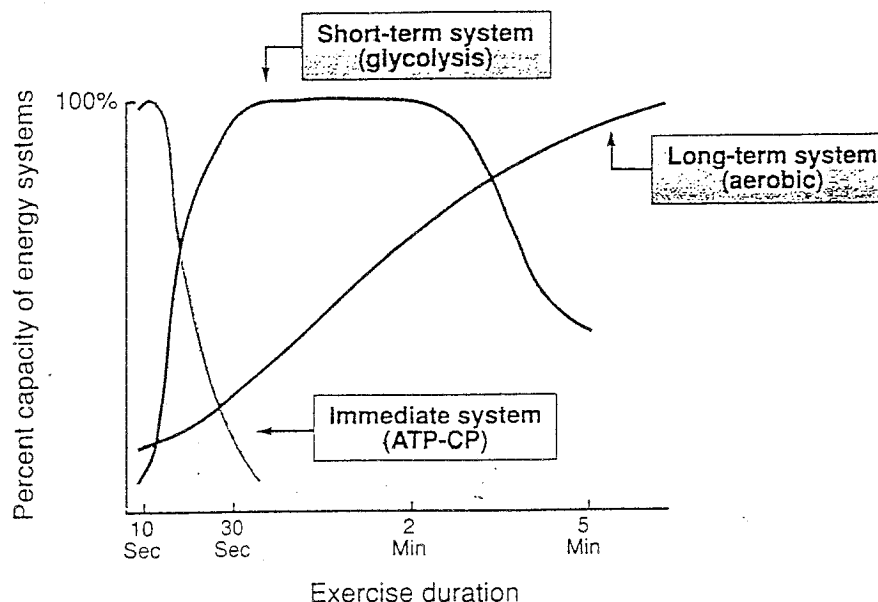
All tissues depend on exercises for vitality. Resistance, intensity and frequency must be optimal. Sufficient **rest** is vital. You must prescribe rest first. Depending on the health and fitness level of your patient, prescribed exercises may need to take ADL into account. Tissue repair, regeneration, and remodeling are dependent on repeated exercise performance and adequate rest.

The rest period must be:

- Up to 48 hours for muscle strengthening
- Several hours for muscle endurance training
- More frequent (every 30 seconds of exercise) and short (5-10 seconds) in order to avoid anaerobic metabolism
- Of excellent quality (it is not enough to do some other activity)

Recommended rest after depletion

Replenishing glycogen level in muscle	10-46 hours
Replenishing glycogen level in liver	12-24 hours
Replenishing O ₂ level	10 seconds-1 minute
Removal lactic acid from blood and muscle	30 minutes-2 hours
ATP/CP level	20-30 seconds allows 50% recovery 40 seconds allows 75% recovery 60 seconds allows 85-90% recovery 3 minutes allows 100% recovery



The various energy systems and their involvement during all-out exercise of different duration

The duration and intensity of the exercise will largely determine if an exercise is aerobic or anaerobic. The first few seconds of an exercise are anaerobic, you use ATP/CP stores. During minutes 1-4 (or greater than 5 if deconditioned) the exercise is a mixture of aerobic/anaerobic. As the duration increases the exercise becomes more aerobic.

When carbohydrates (CHO) are used, the body will increase its ability to metabolize CHO for short duration high intensity work. Endurance is not improved much. Conversely, when fat is used, the body will improve its ability and capacity to metabolize fat for fuel during exercise.

The relationship between bodyweight and O₂ consumption is linear. The more you weigh the greater the oxygen consumption.

With endurance training, there will be an increase in the vascularization of the tissues used. There is also an increase in the aerobic enzyme activity and collagen synthesis in tendons and ligaments.

People who are unfit have an anaerobic threshold of about 30%, therefore walking can become an anaerobic event. Turning on the Krebs cycle is trainable. Marathon runners dip in their Krebs cycle much faster. Increased enzyme activity and mitochondrial activity is required. To accomplish this you might have to start with interval training. At the end of approximately 8 minutes of exercise you have exhausted your ATP/CP and glycogen energy stores. So instead of shutting this down and relying on the Krebs cycle (which might be weak if untrained), you rest for approximately 1 minute to replenish some of your energy pools before continuing.

The Adaptation Syndrome

Depletion

In this phase, nutrients, vitamins and minerals are being used and amino acids related to enzymes are being depleted. Depending on the degree of depletion, a signal is sent to the tissues to move on into the next phase of adaptation

Replenishing or compensation

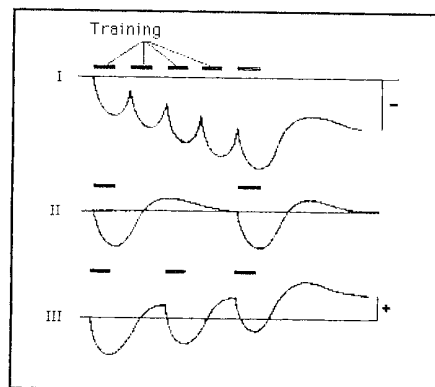
This phase is highly dependent on the amount of rest to the tissues and good nutrition in order for this to be successful and complete. The rest necessary varies widely from a few minutes to 48 hours or more. If rest is not induced, replenishing will be incomplete or fail completely. When replenishing is successful, the last phase of the adaptation syndrome can occur.

Supercompensation

When exercise depletes the tissue and replenishing compensates for the loss of tissue elements and nutrients, the adaptation syndrome leads to a higher level of tolerance to exercise. This is called supercompensation because the level of O₂ in the tissues is higher, the level of glycogen in the muscle and liver is higher, vascularity increases, the production capacity for ATP increases and thus tissue capacity and strength will increase.

The supercompensation principle will also give you some guidelines as how often you should treat the patient. In graph I there is a negative effect of training since the loading phase is too frequent. This results in overtraining and an overloading effect. Graph II shows a loading phase that is too infrequent. If training impulses are not forthcoming in sufficient time, it returns to its original condition. Graph III shows supercompensation where an optimal state exists between loading and resting. Here a new load is given when the body's overcompensation is at its maximum. Optimal improvement of the exercised functional qualities will be achieved.

You will find however, that adhering to this principle is easier said than done, as recovery time is different for individual functional qualities.

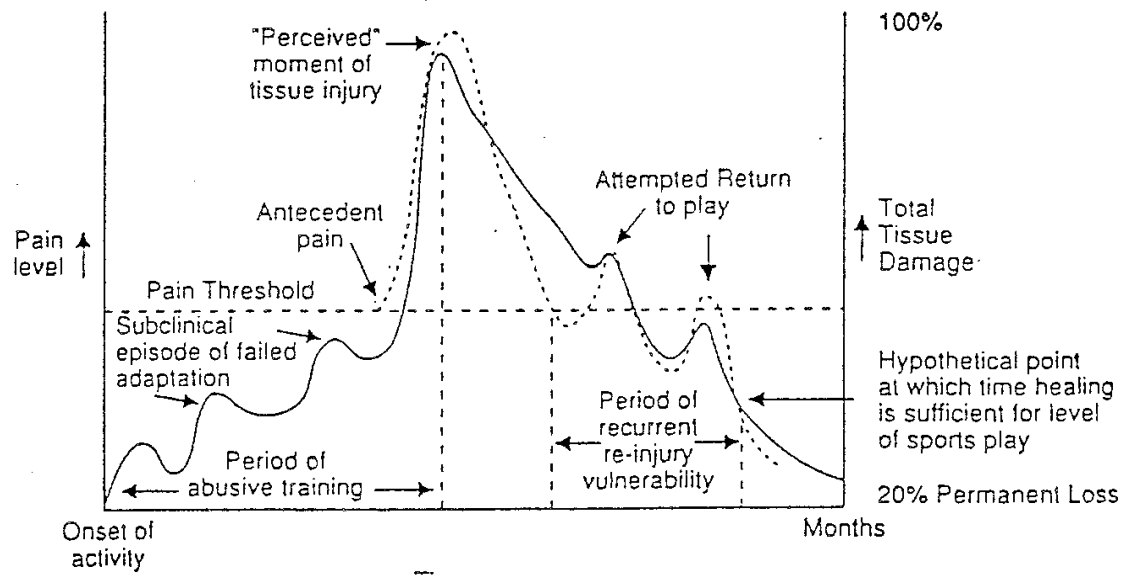


Exhaustion

When exercise bouts are repeated frequently, with very short or non-existent rest periods, adaptation might fail. The failure is usually caused by insufficient time for replenishing. When the compensation is absent in the tissues, repeated exercise stress may result in tissue injury and breakdown. At first the patient will experience this as fatigue due to lack of O₂ supply to the tissue. This will lead to anaerobic metabolism and excessive acidity in the local tissues. When fatigue is carried too far it will lead to inflammation. This occurs as the tissue attempts to remove damaged chemical compounds, amino acids and enzymes. The patient will have a sense of tightness, stiffness, soreness and pain. If this process is carried further, it will lead to increased vascular permeability, causing swelling in the soft tissues and joints. The final result of such tissue injury and breakdown will be experienced as pain.

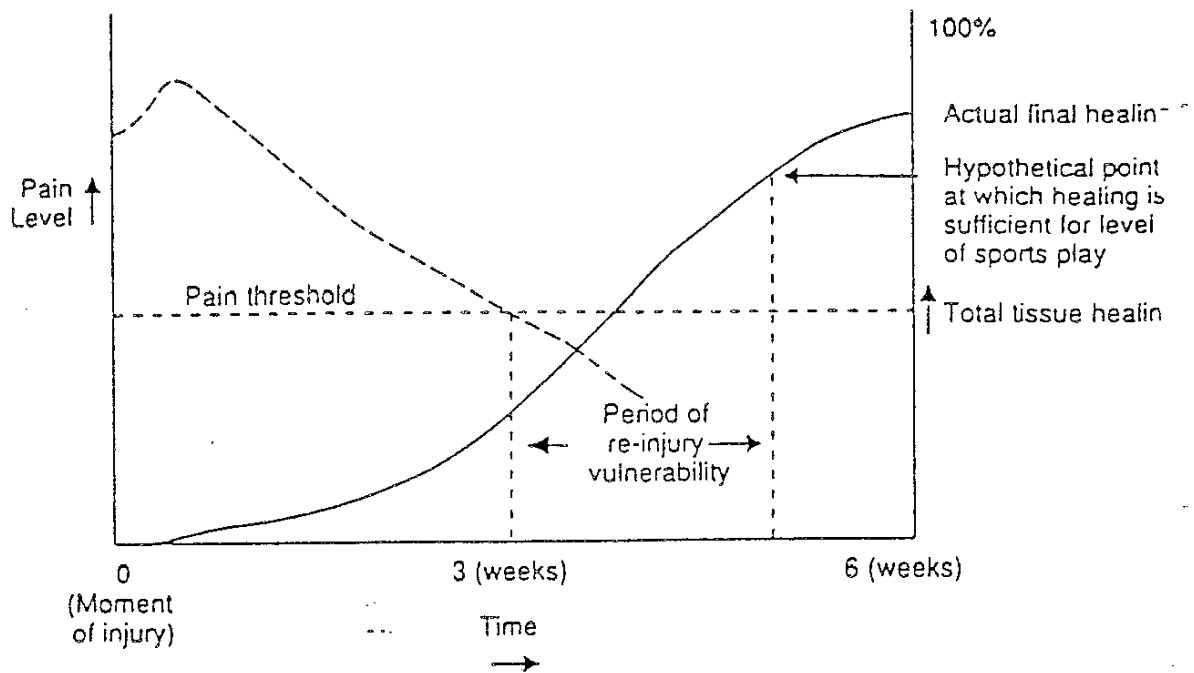
Overuse injury

When tendons are repeatedly exposed to high loads and without sufficient rest between load cycles and periods of repeated load cycles, there is exhaustion of the energy supplied to the tendon. The patient does not recognize this fatigue immediately. There are subclinical episodes of failed adaptation. There usually is insidious onset of pain, or a trivial injury mechanism. The overuse period has usually lasted for 2-3 months. The overuse is usually caused by change in joint biomechanics, exercise resistance, duration/ frequency, or a change in job activity.



Profile of micro-traumatic soft tissue injury. This profile is typical of overuse tendon injury. The solid line indicates the percentage of tissue damage.

In the period of time where the exercise load is continued the cell matrix fails to adapt to the stresses being put on the tissue. Edema and hyperemia of the paratenon might appear, as well as tendon thickening. Sometimes this is palpable, either as a thickening of the tendon, or in the early phase mainly as tenderness. This process of overuse changes is best understood as failed adaptation and will eventually result in disruption of the tendon of a similar nature on a structural level with the traumatic dysfunction. The main difference is that the overuse syndrome will have an extreme low level of mitochondrial activity in the tissues surrounding the site of injury, whereas the traumatic injured tendon is likely to have much higher levels of mitochondrial activity. Aerobic energy production takes place in the mitochondria. The ability to use oxygen and produce ATP via oxidation depends on the number and size of the muscle mitochondria. Both increase with aerobic training. For this reason, it's much more difficult to treat an overuse injury compared to a traumatic tendon injury. The therapist must take caution and exercise pain-staking discipline in order to restore mitochondrial capacity at the same time that structural integrity is reestablished.



Hypothetical profile of acute macrotraumatic tissue injury. This profile is typical of an acute partial tendon strain or the pattern of healing in other acutely injured connective tissues such as lateral ankle sprains. Curved dashed line: tissue injury. Curved solid line: tissue healing.

Pain elimination takes shorter time than tissue healing and repair. When the pain is eliminated the patient is vulnerable for re-injury.

Cryotherapy

Blood supply to tissue and oxygen dissociation from hemoglobin decreases with a decrease in tissue temperature (4-7). For acute injuries, the first line of management is to stop the bleeding and decrease the swelling in the tissue. The application of ice has proven to be a very effective tool to accomplish this.

Depending on the extent of the trauma, the acute phase can last anywhere from 24 hours to 1 week. After the acute phase is over, is the use of ice beneficial in speeding up the healing process? The research is decidedly poor on this topic.

An RCT by Garra et al (2010) looked at 60 patients with acute back or neck pain in a hospital ER department. All patients received 400 mg of ibuprofen and then were randomized to 30 minutes of heating pad or cold pack applied to the strained area. The addition of a heating pad or cold pack to ibuprofen therapy resulted in a mild yet similar improvement in the pain severity. However, it is possible that pain relief was mainly the result of ibuprofen therapy. Their conclusion was that the choice of heat or cold therapy for acute back pain should be based on patient and practitioner preferences and availability.

A systematic review of the literature by Collins (2008) looked at the use of ice for soft tissue injuries. The relevant outcome measures were (1) a reduction in pain; (2) a reduction in swelling; (3) improved function; or (4) return to participation in normal activity. The results showed that most of the research was of poor quality, and the remaining two studies had inconclusive results. The conclusion was that there is insufficient evidence to suggest that ice improves clinical outcomes in the management of soft tissue injuries.

So what to do?

When research is lacking, we have to rely on clinical reasoning. For acute injuries, by all means ice. It stops the bleeding and the swelling in the tissue, and helps to control pain. The use of ice after workouts or therapy sessions should be questioned though. When there is increased swelling, decreased joint motion, or residual pain for longer than 45 minutes after the workout or treatment is over, ice is indicated. At that time the workout intensity has surpassed the tissue capacity, and there is worsening of symptoms. Ice is appropriate at that time. Keep in mind though, that the prime purpose for exercising is to increase oxygenation of the tissue. This is what stimulates the healing process. If you indiscriminately apply ice after each workout or therapy session, you will immediately decrease blood flow to the tissue, thereby decreasing oxygenation. In effect you have negated all the positive changes you had tried to achieve during the therapy session/workout.

As far as what helps best to modulate pain (after the acute phase is over), the research states that the choice in essence is yours, or the patient's.

And what about the use of whole body cryotherapy, which has increased in popularity recently?

Whole-body cryotherapy, which involves a single or repeated exposure to extremely cold dry air (below -100 °C) in a specialized chamber or cabin for two to four minutes per exposure, is currently being advocated as an effective intervention to reduce muscle soreness after exercise. However, there is insufficient evidence to determine whether whole-body cryotherapy (WBC) reduces self-reported muscle soreness, or improves subjective recovery, after exercise compared with passive rest or no WBC in physically active young adult males. There is no evidence on the use of this intervention in females or elite athletes. The lack of evidence on adverse events is important given that the exposure to extreme temperature presents a potential hazard. Further high-quality, well-reported research in this area is required and must provide detailed reporting of adverse events.

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Smoking

Oxygenation of the tissues may be altered because of smoking. It is documented that 15 minutes of smoking decreases oxygenation of the nucleus pulposus by about 50%. It takes 2 hours after smoking before oxygenation has reached pre smoking levels. Repeated exposure to carbon monoxide and nicotine will thus maintain a very low oxygenation level and inhibit replenishing. Smokers have a chronic decrease oxygen tension in the disc of 30%. The affinity of hemoglobin for CO is 250 times as much as that for oxygen. When a hemoglobin molecule binds with CO it cannot bind with oxygen. This results in a decrease in oxygen carrying capacity. Progression of exercise must be adapted to a slower pace for smokers. Smoking is the second most important risk for disc degeneration, only vibration is a greater risk factor.

Contract with the smoker

- Postpone smoking for more than 2 months.
- If that is out of the question, postpone 2 hours before treatment and postpone until 2 hours after treatment.

Soreness

When you go through an exercise bout and depletion is nearly complete, the person will experience some sense of soreness. It is very seldom beneficial to continue exercise in the presence of pain.

Acute muscle soreness

Pain felt during and immediately after exercise can result from accumulation of the end products of exercise, such as H^+ , and from tissue edema which is caused by fluid shifting from the blood plasma into the tissues. Edema is the cause of the pumped up sensation that people feel after heavy lifting. The pain and soreness usually disappears within a few minutes to several hours after the exercise.

Delayed onset of muscle soreness.

Almost all current theories acknowledge that eccentric contraction is the primary initiator of DOMS. Some investigators have suggested that an inflammatory reaction within the muscle causes the soreness. However, the link between the inflammatory reaction and muscle soreness has been difficult to establish.

The following sequence of events has been proposed in the onset of DOMS:

- High tension in the contractile system results in structural damage to the muscle and its cell membrane
- The damage disturbs calcium homeostasis in the injured fiber, resulting in necrosis that peaks about 48 hrs after exercise
- The products of macrophage activity and intra cellular contents (histamine, kinins and K^+) accumulate outside the cells. These substances then stimulate the free nerve endings in the muscle. This process appears to be accentuated in eccentric exercise, in which large forces are distributed over relatively small cross-sectional areas of the muscle.

PRICE vs POLICE, an update on management of acute soft tissue injuries

The acronym PRICE (protection, rest, ice, compression and elevation) has been central to acute soft tissue injury management for many years despite a paucity of high-quality, empirical evidence to support the various components or as a collective treatment package.

Rest, ice, compression, and elevation (RICE) are the basic principle of early treatment. Most research has focused on the analgesic effect of icing or the associated skin or intramuscular temperature changes. Clinical studies into compression are also lacking, and much of its rationale is extrapolated from research relating to deep venous thrombosis prophylaxis and lymphoedema management; there is little clinical research on elevation.

Protection and rest after injury are supported by interventions that stress protection, unloading and/or prevent joint movement for various periods. But, rest should be of limited duration and restricted to immediately after trauma. Longer periods of unloading are harmful and produce adverse changes to tissue biomechanics and morphology. Progressive mechanical loading is more likely to restore the strength and morphological characteristics of collagenous tissue. Indeed, early mobilization with accelerated rehabilitation is effective after acute ankle strain. Functional rehabilitation of ankle sprain, which involves early weightbearing usually with an external support, is superior to cast immobilization for most types of sprain severity.

Functional rehabilitation aligns well with the principles of manual therapy, whereby mechanical loading prompts cellular responses that promote tissue structural change.

The difficult clinical challenge is finding the balance between loading and unloading during tissue healing. If tissues are stressed too aggressively after injury, the mechanical insult may cause re-bleeding or further damage. Protection of vulnerable tissues therefore remains an important principle. But, too much emphasis creates a default mindset that loading has no place in acute management. Rest may be harmful and inhibits recovery. The secret is to find the 'optimal loading'.

Optimal loading means replacing rest with a balanced and incremental rehabilitation program where early activity encourages early recovery. Injuries vary so there is no single one size fits all strategy or dosage. A loading strategy should reflect the unique mechanical stresses placed upon the injured tissue during functional activities, which varies across tissue type and anatomical region.

POLICE, a new acronym, which represents **protection, optimal loading, ice compression and elevation**, is not simply a formula but a reminder to clinicians to think differently and seek out new and innovative strategies for safe and effective

loading in acute soft tissue injury management. Optimal loading is an umbrella term for any intervention and includes a wide range of manual techniques currently available. Paradoxically, crutches, braces and supports, traditionally associated with rest, may have a greater role in adjusting and regulating optimal loading in the early stages of rehabilitation.

POLICE should make us think more about designing rehabilitation strategies that are appropriate to the nature and severity of injury in different sports and activities. If the primary principle of treatment is to restore the histological and mechanical properties of injured soft tissue, optimal loading may indeed be sport specific. The challenge is in determining what is 'optimal' in terms of the dosage, nature, and timing.

POLICE is not just an acronym to guide management but a stimulus to a new field of research. It is important that this research includes more rigorous examination of the role of ICE in acute injury management. Currently, cold-induced analgesia and the assurance and support provided by compression and elevation are enough to retain ICE within the acronym.

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Tissue Specific Exercises

When tissues are exposed to repeated functional demands, O₂ is utilized and depleted at a local level. This depletion is a strong message for the tissues in question to increase vascularity and oxygenation. This process can best take place during a rest period after exercise loading. There is probably no better way to achieve this process. For this reason, it is not likely that ultrasound, cold laser, hot packs, or icing does much more than decrease the pain sensation. They do not stimulate tissue repair.

The best way to stimulate tissue repair is to construct an exercise program that will deplete O₂ levels in the tissues. If exercise is followed by rest, and maintenance of high tissue temperature, vascularization and oxygenation will be stimulated as a means of compensation for the relative depletion incurred during the exercise loading. Repeated exercise interrupted with relative long periods of rest, will first restore the biochemistry capacity and the mitochondrial function. After 1-2 weeks of such activity, tissue integrity will be restored. With an acute injury, this process may be shorter. With an overuse syndrome, it has been demonstrated that there is a significant decrease in pain over the first 2-3 weeks, but that tissue strength is not restored until 4-6 weeks after the exercises have been initiated.

It is important to realize that the exercise stimulus necessary to reach optimal potential is a high numbers of repetitions. So be careful to guard against overuse in the treatment sessions. Make sure to include frequent rest periods.

It is our experience that a patient's knowledge of this repair process is severely limited and therefore they tend to overdo exercises and disregard pain as a signal that may be helpful in preventing overuse. We have found it necessary to spend time in repeated instruction to impress upon the patient the necessity to regard pain and tightness as warnings, reminding them of potential tissue damage.

Ultimately, it comes down to increasing **tissue capacity**. This is defined as the ability of the tissue to tolerate the loads you want/need to place on it. Injury decreases tissue capacity, as does inactivity. Keep in mind that it is not just the injured tissue that experiences a decrease in capacity. The tissues around it usually loose their capacity as well, and therefore need to be included in the rehab process.

Tissue capacity will only be as great as the loads you put on them. Lots of tendon injuries happen when people perform an unusual activity for that body part, or in an athlete's case, they take a 2-3 week vacation, and then come back full activity. The tissue's capacity has adjusted to the decreased level of activity, and therefore is more prone to injury. For example, the half-life of the tendon ground substance is 1.9-9 days. Thus, with a decrease in the level of normal activity, the proteoglycans have little "knowledge" of the stresses that were once applied. After 3 weeks of immobilization, the capacity of tendon tissue can be reduced by as much as 25%.

The opposite of tissue capacity is tissue weakness. The use it or lose it phenomenon of tissue capacity has been extensively described for bone. While injury may be limited to one tissue, pain and reduced loading must reduce capacity in associated tissues within the kinetic chain. Failure to return to capacity in all these tissues may result in re-injury or subsequent injury. Understanding the biological mechanics of musculoskeletal tissue, and how loading and unloading affects the capacity of these tissues is critical to clinicians.

Tendon

The role of the tendon is to transfer muscle forces to bone. Tendons play an important role in the development of power and in the efficiency of muscle contraction through the storage and release of elastic energy.

The metabolic rate of tendon is less than that of muscle since tendon is less vascular, therefore healing times of tendons are typically longer than that of muscle tissue. The blood supply to tendons is also substantially decreased compared to muscles, nerves and bones. Certain parts of ligaments and tendons are almost avascular. This potential deficiency is compensated by a pumping action of synovial fluid. This route for nutrition seems to be very important and occurs with even small movements of approximately 4 mm. (like in massage and gentle cross frictions). Tendons are stronger per unit area than muscle.

A tendon has a wave like form, which allows for a certain amount of deformation. When a deforming tension is released the waveform permits return to its original length if the stretch was no more than 4%. If the stretch is greater than 8%, the tendon begins to fail. The largest physiologic loads fall within the 12% range.

Tendon Pathology

The repair of overuse tendon conditions appears to be compromised by processes that are not quite understood. There is strong evidence that the inflammation paradigm is inaccurate. Despite that, most clinicians still use the term “tendinitis,” thus implying that the fundamental problem is inflammatory. We advocate the use of the term “tendinopathy” as a generic descriptor of the clinical conditions in and around tendons arising from overuse.

Evidence is mounting towards to 2 reasons for tendinopathy:

1. A biochemical cause of pain
2. Breakdown of the collagen fibers that make up the tendon

Histological examination of tendinopathy shows disordered, haphazard healing with an absence of inflammatory cells, a poor healing response, non-inflammatory intratendinous collagen degeneration, fiber disorientation and thinning, and scattered vascular ingrowth. Frank inflammatory lesions and granulation tissue are infrequent and are mostly associated with tendon ruptures. Tendinopathy can be viewed as a failure of the cell matrix to adapt to the stresses being put on the tissue.

It is important to note that tendons may be painful to palpation (especially in athletes) in people who do not have tendinopathy, and that palpation pain does not predict diagnosis. Like other chronic conditions, there can be a disconnect between clinical presentation and abnormalities on imaging. That is, pain may be present without imaging change and vice versa.

There may be important differences between UE and LE tendinopathies in terms of the contribution from peripheral tissue or central drivers. While the role of central sensitization has been highlighted for rotator cuff and lateral elbow tendinopathy there is preliminary evidence that central sensitization does not occur in Achilles tendinopathy. The importance of UE function for self-care and occupational activities, might contribute to these differences in central sensitization between UE and LE tendinopathies. As all nociceptive input is evaluated in terms of threat, the increased threat associated with UE tendinopathy (e.g. negative thoughts and feelings arising due to fear of pain with daily activities, fear of tendon rupture, fear of unemployment) likely amplify the pain experience. We should keep these differences in mind and be careful of applying research findings from the rotator cuff to Achilles tendinopathy.

Three categories are used to describe the different stages of tendon injury, but there are no discreet boundaries between the 3 stages.

The 3 stages of tendon pathology:

1. Reactive tendinopathy
2. Tendon dysrepair or failed healing
3. Degenerative tendinopathy

The model describes 3 distinct stages but in reality it is a continuum where one flows into the next. The primary stimulus that drives tendons up or down the continuum is adding or removing load. This is especially true in early stages. Reducing load may allow the tendon to return to its previous level of structure and capacity. This model also proposes that the pathology and the response to treatment is different in each of the stages. The model can therefore better aid clinicians target their interventions.

Reactive tendinopathy

Reactive tendinopathy is proliferative response that occurs in the cells and the ground substance. It occurs following acute tensile or compressive overloading of the tendon. In the early stages of tendinopathy the cells are activated and probably proliferated. These activated cells produce more proteoglycans. The increase in proteoglycans is responsible for increased water uptake in the tendon that makes the tendon a bit thicker and protects the cells. The purpose of this adaption is to reduce stress on the tendon by increasing cross-sectional area, which will decrease the force per unit area. In other words it is a temporary solution, a way to buffer the

forces until long term changes in either tendon structure or mechanical properties can occur. The activated cells are responsible for producing nociceptive substances, which trigger the pain response. The integrity of the collagen is mostly maintained in this stage but there may be some longitudinal separation but no change in neurovascular structure. However, the activated cells also produce enzymes that increase the degradation of the matrix. The tendons become thicker, but with reduced energy-storing capacity, meaning that for the same load, their tendons exhibit higher strains than those of healthy individuals.

If this process is not reversed, it will ultimately result in degeneration and weakening of the collagen fiber structure, which is stage 2, the tendon disrepair/failed healing stage.

Keep in mind that although the stiffness (deformation in response to tensile load) of the tissue is, on average, lower in tendinopathic tendon, this phenomenon is unrelated to the sensation of increased “stiffness” often mentioned by individuals with tendinopathy, which is likely related to sensory and motor changes. These are separate phenomena with different definitions, but they happen to have the same term in the English language.

In summary the reactive response is a short-term adaption to tendon overload that thickens the tendon, reduces stress and increases stiffness. The tendon has the potential to revert back to normal if the overloading is reduced or if there is sufficient time between loading sessions to allow for tendon adaption.

Tendon Disrepair

The second stage is tendon disrepair. In some ways it is similar to reactive tendinopathy, but there is greater breakdown of the matrix. In this phase there is an overall increase in number of cells in the tendon, mainly chondrocytes and myofibroblasts. This results in a marked increase in protein production, primarily proteoglycan and collagen. This increase in proteoglycans causes a separation of the collagen and disorganization of the matrix. These changes are often localized.

Degenerative Tendinopathy

In this stage there has been a progression of both matrix and cell changes, including areas of cell death. Exhaustion of these cells causes areas absent of any cells. There are large areas of disorganization in the matrix with many blood vessels. You will also find very little collagen and the byproducts of matrix breakdown. It should be noted that the whole tendon is not degenerated. In fact it is more like islands of degenerative pathology surrounded by normal tendon and areas of tendon in other stages of pathology. Unfortunately for the areas of tendon in the degenerative stage there is little capacity to recover. However, degenerative tendons can show improved function despite not returning to normal size or structure.

Medications as a causal factor

Certain medications play a role in causing or exacerbating tendinopathy.

Statins

Statin-induced tendinopathy was first reported in the 2000s. The predominant musculoskeletal complaint with this class of drugs is myalgia, but tendinopathy (in the form of tendinosis and tenosynovitis) is also a side effect. The incidence of statin-induced tendinopathy is not known, but it is thought to be rare, accounting for approximately 2% of all complications in one study.

Fluoroquinolones

A class of synthetic antibiotics, including commonly used medications such as Cipro and Levaquin. The fact that tendinopathy can occur as a side effect of these drugs was not widely recognized until the early 2000s. The estimated rate of tendinopathy in response to fluoroquinolone treatment is 2%, with the Achilles tendon being the most commonly affected. The onset is usually acute (median duration, 8 days after beginning treatment).

Corticosteroids

Corticosteroids can impair local collagen synthesis, resulting in tendon atrophy, reduction of tensile strength, and hence decreased load to failure. Local steroid injections in the vicinity of a tendon and in the presence of severe tendinosis or a tear are discouraged due to the concerns with respect to rupture.

Imaging

Diagnostic imaging

Ultrasound, MRI, CT, X rays are all types of diagnostic imaging that are best used when:

- The case is complicated and chronic
- PT has failed
- Examination has identified differential diagnosis in need of exclusion

Diagnostic Ultrasound

Diagnostic ultrasound is good to establish a bottom line, and to look for involvement of paratenon, bursa and plantaris muscle. It is not good for imaging the fat pad. If you suspect fat pad involvement, you need to do an MRI.

Currently, there is not much evidence to demonstrate that the tendon improves structurally on imaging as a result of good PT. The relation between objective changes on imaging, and complaints of pain, is tenuous at best. Functionally they will improve, but minimal changes will be noted on imaging. One explanation for this phenomenon may be that the changes in the pain system take place faster than the restoration of the tendon structure. The healing process for the tendon structure is simply different from that of pain, but no less important. This puts even more importance on adherence to a tendon training home program long after they have been discharged from PT.

Coombes et al (2008) suggest that the clinical presentation of tendinopathy is not just defined by local tendon pathology (structure) but also by the integration of the pain system and motor impairments.

Differential diagnosis of Achilles tendon pain

When a patient has failed to improve with traditional approaches to treatment for Achilles tendinopathy pain and has symptoms of pain on the medial aspect of the Achilles tendon, you may need to consider the involvement of the plantaris tendon.

The plantaris muscle functions as a weak plantar flexor at the ankle and knee flexor. It originates from the lateral aspect of the femur and inserts onto the medial aspect of the calcaneus. Cadaver studies have shown that the insertion point of the plantaris muscle can vary with up to 9 different insertion points. And it is also possible to not have a plantaris muscle at all. For these reasons, it has long been considered a non-important muscle and has been given little attention other than as a good tendon to use for reconstruction-type surgeries. This changed though, when in 2011, a study from Alfredson et al. showed its involvement in “Achilles pain” The theory behind this anatomical variation is that with evolution, humans have transitioned from more of a quadruped gait to a bipedal gait and with this, the function and demands on the plantaris muscle have changed.

Due to the proximity with which the plantaris tendon and the Achilles tendon can be situated, peritendinous tissue can become irritated or compressed against the Achilles tendon, creating pain along the medial aspect of the Achilles.

So, if you have a non-responder (no improvement in 6 weeks of a solid tendon loading program), and the complaints of pain are mainly over the medial portion of the Achilles tendon, consider the involvement of the plantaris tendon. Ultrasound imaging can be used to confirm the painful structure.

Treatment for the involvement of plantaris irritation is surgical intervention to scrape the tendons, removal of the plantaris tendon or injection of a substance to help separate the tissues.

Treatment

Medication

The use of NSAIDs in the treatment of tendinopathies has been somewhat controversial. They reduce pain but have also been reported to slow tissue healing and therefore have a negative effect on tendon repair. However, in reactive tendinopathy, slowing the healing response is a desirable effect to slow cellular upregulation and excessive ground substance manufacture. It is recommended to take the NSAIDS for 2-4 weeks, 600mg TID, Ibuprofen has been proven to be most efficacious, followed by Naproxen and Cox II inhibitors.

Corticosteroids also decrease cell proliferation, protein production and pain, and therefore may be appropriate in the reactive phase. Peritendinous injections have been clinically accepted, but it is not known currently whether peritendinous injection actually induces cell and matrix changes within the tendon.

A systematic review by Andres and Murrell in 2008 showed that NSAIDs and corticosteroid injections provide short-term pain relief, but long-term effectiveness has not been established.

PRP can be beneficial for chronic degenerative tendons, where you are stuck in progressing. PRP can help to adapt the tendon structure.

Therapy

First and foremost, address the underlying biomechanical fault that causes the tendon to be a stress riser in the first place. If the biomechanical fault is not addressed, it is nearly impossible to create an ideal environment for tendon healing to take place. For example, in Achilles tendon problems, this is usually a combination of talocrural and subtalar hypomobilities in combination with soft tissue restrictions and motorcontrol problems in the mid/hindfoot.

Exercise is the best intervention for tendinopathy, and is the only stimulus with the capacity to positively affect tendomatrix. However, its application should vary according the presentation. Reactive tendinopathy requires relative rest from high tendon load activities (energy storage and release activities such as running) and prescription of isometric exercise for pain. In contrast, disrepair and degenerative pathology require progressive loading to moderate cell response and address functional deficits.

Adjunct treatments such as electrotherapy and massage have limited evidence for their effectiveness, however may be used as needed but should never form the basis of a tendon treatment. Furthermore, progression to surgery or invasive procedures should not be based on failure to improve from passive therapies. Patience of, and education for, the person with tendinopathy is essential and should be central to a PT program.

Tendons respond favorably to controlled loading after injury. For the tendon to respond to the exercise stimulus, the intensity of loading needs to be optimal. This brings on strain on the tendon, which fuels the healing response. The tendon will become stiffer and able to absorb tensile loads as a result of tendon rehab. Injured tendons tend to lose their capacity to absorb energy. For that to happen, rehab needs to be intense, and long (3-4 months)

However, what is the proper loading program for tendons? There is very little evidence to compare different loading programs. Evidence is limited in establishing treatment guidelines. It stands to reason though that we need to reduce the abusive load if the capacity of the musculotendinous unit has decreased because of injury.

It is vital to find the baseline training level that does not provoke the tendon and to take this as the point to start the rehab process. Don't get too preoccupied with the pain intensity the patient presents with. It is more important to pay attention to treatment response. You don't want any increased pain for longer than 30-45 minutes after treatment (or workout) is finished. The evidence suggests that during the rehabilitation process, any worsening of edema, morning stiffness, or delayed-onset pain should be closely monitored and controlled.

At the microscopic level, as a tendon heals, vessels and nerves regress; collagen fibers become stronger; and the tendon becomes less thickened, more resistant to load, and less prone to re-injury (i.e., recovering a more normal stress-strain curve).

However, there are no easy measures of tendon capacity. Even extensively pathological tendons appear to have the capacity to tolerate very high sporting loads. The mechanisms by which the tendon increases capacity in response to training loads are unclear, as the tissue pathology may not change. Perhaps we are merely increasing the capacity of the normal part of the tendon.

Some authors have advocated reinjuring the tendon through treatments such as intratendinous needling and injections, aggressive soft tissue therapy, etc. These approaches may improve the patient's symptoms in the short term, but could result in long-term damage to the tendon. In addition to clinical trials that show a lack of effect of this type of approach, the rationale is not well supported by current knowledge of pathology as outlined above.

Eccentric training is a common intervention when rehabilitating Achilles tendinopathy, however, systematic reviews have identified low methodological quality among studies on this intervention. Additionally, there is substantial variability in the protocol used for eccentric training.

There are unique characteristics of eccentric exercise that may have important implications for tendinopathy. These include modulation the neurological stretch response; perturbations of tendon force; increased shear forces between the tendon and paratenon structures; pain modulation, and adaption of mechanotransduction signaling in passive tendon structures. Despite these data, it is unclear whether it is important to isolate the eccentric component of the exercise rather than performing a concentric-eccentric exercise.

Eccentric exercise programs are not the be all end all, nor are they essential for tendon rehab. They are very useful as a part of the rehab process to maximize loading on the tendon, and improve the ability of the tendon to handle plyometric activities, but that cannot be all you do.

The proper exercise build up for tendon training:

1. Isometrics
2. Concentric/eccentric
3. Heavy load eccentric (where they cannot lift it concentrically)
4. Plyometrics (Stretch-Shortening-Cycle)

Tendinopathy Rehab Progression		
Phase of rehab	Aim	Treatments
1	Reduce pain	Isometrics in mid range. Reduce compressive load and use of SSC Ibuprofen (if reactive)
2	Improve strength	Heavy Slow Resistance training in non-compressive position
3	Build functional strength	Progress strength work into more functional tasks. Treat movement dysfunction
4	Increase Power	Reduce reps but increase speed of muscle contraction to build power
5	Develop Stretch-Shortening-Cycle (SSC)	Introduce plyometrics +/- graded return to running
6	Sport specific	Add drills specific to requirements of sport
Maintenance	Maintain optimal tendon health	Continue strength work. Gradual increase in loading. Avoid training error.

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In the reactive phase, emphasize unloading type activities. This includes activities like Total Gym, biking, using an unloading device to walk/run, but it also includes simple measures like using a heel lift to take the strain of a reactive Achilles tendon. In this phase you want to reduce the strain, as well as the compression on the tendon.

Tendon	Site of compression	Position of compression	Modification	Effectiveness
Achilles insertion	Superior calcaneus	Ankle dorsiflexion	Heel raise	Effective
Tibialis Posterior	Medial malleolus	Anatomically permanent pivot	Orthotics and heel raise	Limited
Long Head of Biceps	Bicipital groove	Shoulder extension		
Supraspinatus	Greater tuberosity	Shoulder adduction		
Proximal Hamstring	Ischial tuberosity	Hip flexion	Limiting sitting/ lunging	Moderate
Gluteus Medius and Minimus	Greater trochanter	Hip adduction	Lumbopelvic control, sleep supine	Effective
Adductor Longus/ rectus abdominus	Pubic ramus	Hip abduction/ extension	Limit loads in abduction/extension	Moderate
Peroneal Tendons	Lateral malleolus	Anatomically permanent pivot	Heel raise	Limited
Quadriceps	Femoral condyle	Deep knee flexion	Limit loads in deep knee flexion	Moderate
Pectorals	Humeral tuberosity	External rotation		

Low load isometric exercises work well in the reactive phase. Research shows that isometric exercises reduces tendon pain immediately for 45 minutes post-intervention and increases maximum voluntary muscle contraction. A much smaller decrease in pain and no change in cortical inhibition were found after isotonic exercises (Rio et al 2015). However, an RCT by van Ark et al (2015) showed no difference between isometric and isotonic exercises after four weeks. Besides decrease in tendon pain they also found a decrease in cortical inhibition, which is present at elevated levels in patellar tendinopathy.

The pain reduction observed following isometric exercise may be due to the cortical changes observed and motor neuron pool recruitment, and/or driven by changes at a tissue level. In the study, there were no non-responders to isometric exercise regardless of the pain severity or length of time of symptoms. The clinical implications are that isometric exercise may be used to reduce pain and motor inhibition in the early stages of rehabilitation and provide an important option for clinicians to offer for painful tendons that are difficult to load without aggravating symptoms or potentially prestrength training sessions in later stages. There also may be a role for using isometric exercise to reduce motor inhibition and improve responses to strength training, as it is currently known that deficits persist despite rehabilitation.

The current consensus is that the patient should be able to hold the isometric contraction for 30-60 seconds with 30%-40% bodyweight at midrange. Progress to

maintaining a 60 second contraction with full bodyweight. An RCT by van Ark et al (2015) used a high percentage of repetition maximum (80% 1RM) for isometric as well as isotonic exercises. Beneficial effects from rehabilitation require high load per repetition. This improves muscle strength and neural activation as well.

It is essential they master this before progressing to any type of eccentric activity. If they cannot, they are not able to go through the full range with proper muscle control, and they have no business progressing to eccentric activity. Low load isometrics are ideally suited to control pain, however, they do not affect the tendon tissue directly. For that you need high load isometrics, as only they are able to introduce sufficient strain to stimulate tendon healing.

Once the patient is out of the reactive phase, the training schedule will look as follows:

- 3x/week heavy
- 2x/week endurance

Progress to incorporating plyometrics 2x/week. Once that is tolerated well, you can truly do separate days of plyometric workouts alone.

The power phase of the program needs to be just as long as the slow phase of the program. So if the concentric/eccentric portion of the program is 10 weeks, the power phase needs to last 10 weeks as well. However, the phases can overlap, you don't need to be finished with the slow phase of the program before you start incorporating plyometric activities.

For endurance training, try to do calf raises until complete fatigue, up to 70 reps. For starters, 40-50 reps is good.

When progressing to the later phases of rehab, the tendon needs to be heavily loaded. Get them in the Gym. Exercises like jump squats in the Smith machine with >200#, or regular squats in the Smith machine with up to 300# are needed to properly load the tendon.

Heavy loading type exercises are emphasized only in athletes that are involved in explosive type activities, like sprinters. Nearly everybody else will do a combination of endurance and heavy loading.

For patellar tendon rehab, be careful not to go over 70-80 degrees of knee flexion with the heavy loading exercises. This includes squatting and leg press. The tensile forces on the patellar tendon become extremely high at that point, and the risk of damage becomes too much. You still need to work out the patellar tendon at that endrange of course, but you do that with functional activities, or with low loading. The risk benefit ratio with heavy load deep squatting is just not there.

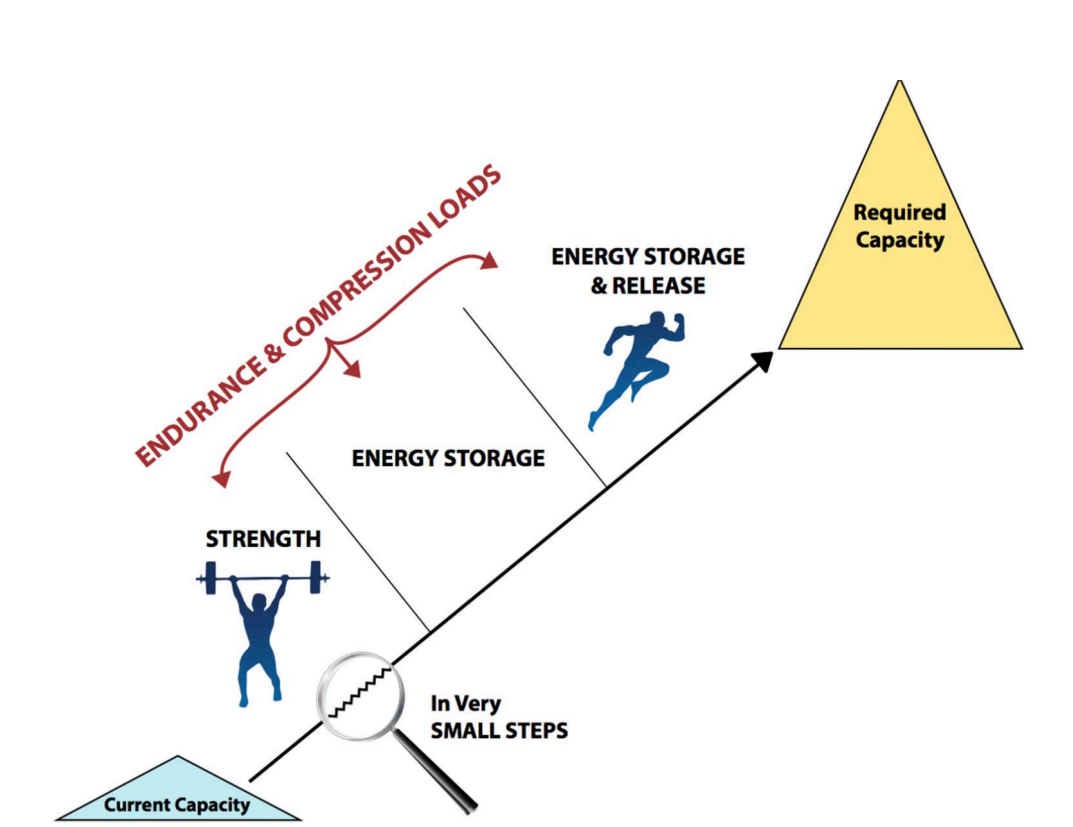
Leg extensions are a very good exercise for patellar tendon rehab. It is not a functional activity, but it is great to develop significant strain on the tendon tissue, necessary to provide stimulus for healing.

Once the patient is done with the supervised portion of the rehab, they need to be put on a maintenance program. This needs to consist of daily low load isometrics combined with endurance work for pain control. Heavy load isometrics are done 1x/week.

Length of training program

It is generally accepted that effective treatment for tendinopathy will result in significant (up to 80%) reduction in pain within 12 weeks, confirming the relative plasticity of the pain system. However, that does not mean that from a structural standpoint the tendon is better as well. This can take significantly more time.

Rehabilitation should be continued until the tendon regains full capacity to store and release energy without immediate or latent pain, and then be followed by a maintenance program if returning to competitive sport.



Schematic of tendon rehabilitation, improving tendon capacity with progressive loads. Introducing compressive loads are critical within each stage. The start and end points of rehabilitation will vary between each patient.

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Cartilage

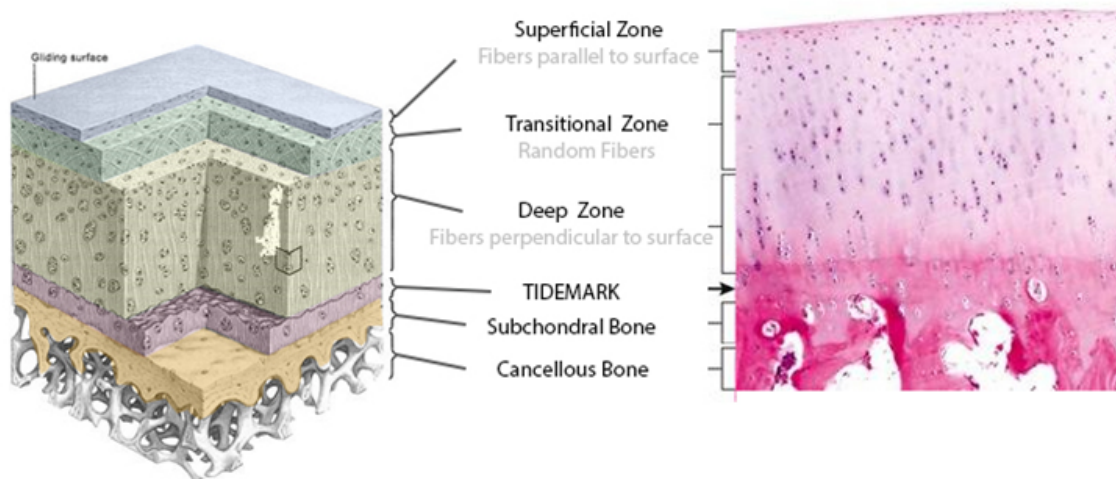
Articular cartilage has several functions:

1. It spreads the applied load to the subchondral bone
2. It provides the articular surfaces with low friction and lubrication
3. It is responsible for the mechanism of shock absorption

Cartilage is avascular, aneural and alymphatic tissue. The chondrocytes receive their nutrients by diffusion from the synovial tissue

Morphologically, articular cartilage can be divided into 4 zones.

1. Superficial zone (10-20% of full thickness)
2. Transitional zone (40-60% of full thickness)
3. Deep zone (30% of full thickness)
4. Calcified cartilage zone



At the border between the deep zone and calcified cartilage, there is a “tide mark” considered to be a crucial structure in load transfer from the cartilage to the underlying bone. At this level thick collagen fibers go across the tidemark, allowing a stable union between the deep layer and the calcified cartilage.

The biomechanical properties of articular cartilage are supported by the 2 main components of the extracellular matrix: proteoglycans and type II collagen fibers. They form a complex network that provides the articular cartilage with 2 characteristics: resistance to compressive stress and high elasticity.

The proteoglycans are made up of glycoprotein and glycosaminoglycans. The GAG's attract water in the tissue, which increases the volume of the tissue significantly.

The liquid in the tissue is the main factor responsible for load bearing: the incompressible water sustains the compressive stresses, ultimately protecting the solid components of the cartilage matrix, which is only partially involved in the biomechanical response. If the cartilage is damaged (as in OA) the water flows out

rapidly from the matrix. As a result, the solid components of the tissue become significantly involved in the biomechanical response. This may lead to rapid deterioration of the cartilage.

When shear force is applied, no interstitial fluid flow occurs and the tissue deforms because of the organized collagen fiber structure. In this case, the solid matrix is directly involved in the biomechanical response and is therefore exposed to deterioration. Synovial fluid and perfect architecture of the articular cartilage help to decrease the shear forces. Again, any type of damage, which causes an increase of the shear forces will lead to an increase of involvement of the solid matrix in the biomechanical response, and therefore to tissue deterioration.

Articular cartilage is metabolically active, but it has poor intrinsic healing potential when damaged. The repair process frequently results in fibrous tissue, which has inferior biomechanical properties, compared to hyaline cartilage.

Articular cartilage is able to repair, but not to regenerate. The cartilage response to trauma is limited for 2 reasons:

1. Cartilage is avascular tissue and therefore, bloodclot formation cannot take place and the inflammation response is absent. Because of this, migration of undifferentiated cells to the injury site is absent
2. Cartilage lacks undifferentiated cells.

The number of chondrocytes further decline with age, making the repair process harder in older patients.

Generally, cartilage lesions can be divided into 3 different lesions:

Superficial lesions

The permeability of the tissue increases and this increases the direct biomechanical response of the macromolecular framework during compression. If the damage progresses, fragments of articular cartilage may be released in the joint and the subchondral bone may be exposed. As a result the load is directly transmitted to the underlying subchondral bone, which responds by increasing its density and thickness.

Blunt trauma

This can cause a chondral lesion without the visible alteration of the superficial area. However, it does result in injury to the chondrocytes. This leads to disturbance of the matrix turnover and ultimately results in disruption of the cartilage structure. The trauma is transmitted all the way to the subchondral bone, which reacts by becoming thicker. This leads to a reduction in the shock absorbing effect at this level, eventually leading to deterioration of the above cartilage layers.

Osteochondral lesions.

This consists of full thickness cartilage defect extending into the underlying subchondral bone. These lesions are therefore accessible to bloodcells, macrophages and stemcells. Following damage, a fibrin clot is formed in the defect

and the bony part of the lesion. Stemcells then migrate in and start differentiating in chondrocytes and osteoblasts and in a few weeks will fill the defect. These cells will produce collagen, but these are less than those in normal cartilage, and are also poorly organized. So it does not restore the normal structure, composition and mechanical properties of healthy hyaline cartilage. As a result, in the long term a process of degeneration will start and leads to failure of the repair tissue. In addition the connection between the subchondral bone and the repaired cartilage is suboptimal, leading to decreased biomechanical properties as well.

The healing property of osteochondral lesions is influenced by the size and location of the damaged area and the age of the patient. Extensive defects or lesions in the weight bearing areas have a poor chance for successful healing.

Summary

The proper biomechanical composition and the morphological organization of the articular cartilage are fundamental for the correct biomechanical function of this tissue. Under normal conditions, this allows for a biomechanical response with practically no tissue deterioration. However when a lesion occurs, the remaining cells are not able to organize an adequate regenerative response. This leads to long term failure of the reparative tissue.

Treatment options

Initial management usually begins with analgesics and NSAIDs. However, the potential cardiovascular and GI toxicity, the large variation in individual response to the drug and the absence of clear data regarding the therapeutic value represent major limitations. Topical agents have only been proven useful for short term use in milder cases.

Cortisone injections in the joint are of short-term benefit for pain and function. They are also not able to alter the course of the disease and may have deleterious consequences on the joint structure.

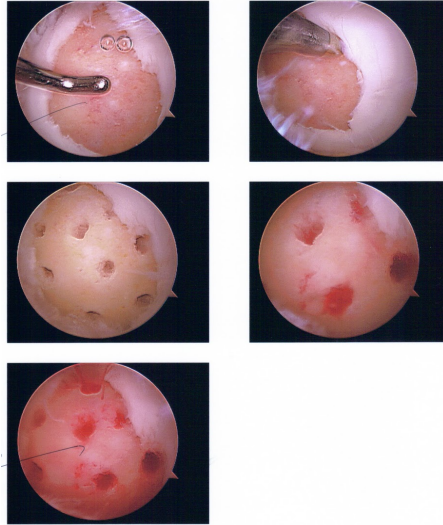
Glucosamine and chondroitin have not clearly demonstrated to be effective, and due to the continuing controversies and lack of common accepted evidence, cannot be considered ideal procedures for treatment of OA

Surgical procedures

Several repair techniques have been shown to be successful:

Microfracture is the most frequently used approach. Because it is a low cost and low morbidity procedure it should be the first line treatment for contained smaller defects. The minimal invasiveness of the technique allows early return to activity. However, it has been shown to offer mainly short-term benefits, tending to fail in providing long term results. This is probably due to the inferior mechanical properties of the newly formed fibrous tissue.

The surgery was developed in the late 1980s and early 1990s by Dr. Steadman of the Steadman-Hawkins clinic in Vail, CO. The surgery is performed by arthroscopy, after the joint is cleaned of calcified cartilage. Through use of an awl, the surgeon creates tiny fractures in the subchondral bone plate.



Blood and bone marrow, which contains stemcells, seep out of the fractures, creating a blood clot that releases cartilage-building cells.

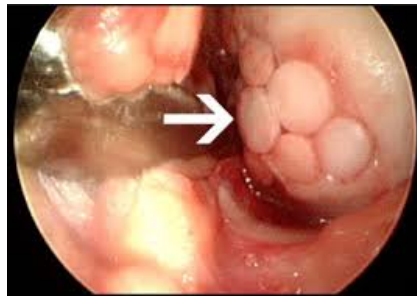


The microfractures are treated as an injury by the body, which is why the surgery results in new, replacement cartilage. The procedure is less effective in treating older patients, overweight patients, or a cartilage lesion larger than 2.5 cm. Studies have shown that microfracture techniques do not fill in the chondral defect fully, forming fibrocartilage rather than hyaline cartilage. Fibrocartilage is not as mechanically sound as hyaline cartilage; it is much denser and unable to withstand the demands of everyday activities as well as the original cartilage and is thus at higher risk of breaking down. The blood clot is very delicate after surgery and needs

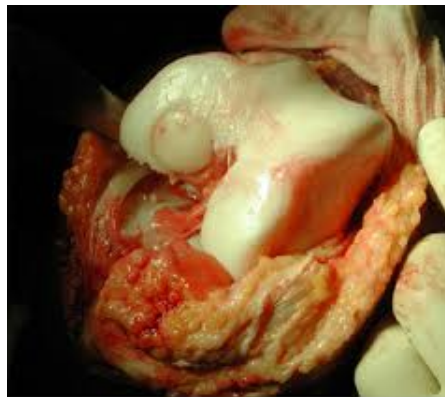
to be protected. In terms of time, the clot takes about 8 weeks to 15 weeks convert to fibrous tissue and is usually fibrocartilage by about four months post surgery, holding implications for the rehabilitation. Return to sports is reported at an average of 6.5-10 months.

Cartilage transfer procedures

There are 2 types of cartilage transfer procedures: osteochondral autograft transfer system (OATS procedure) and mosaicplasty. In mosaicplasty, cylinders of healthy cartilage and bone are taken from a donor site (typically a non-weight bearing surface of the knee joint) and moved to replace the damaged cartilage area of the knee. Multiple tiny plugs are used and once embedded, resembles a mosaic, hence the name. This technique is limited due to the availability of the cylinders at the donor site. In addition, restoration of the anatomic congruence of the cartilage surface is technically demanding and often not feasible.



With the OATS procedure, the plugs are larger. It is used more as a salvage procedure for larger osteochondral defects on the femoral condyle.



The technique is a good treatment option when there is subchondral bone damage as well, because it allows it to be repaired in combination with the cartilage, which is essential to obtain good results.

The literature shows encouraging results and a high ratio of return to sport activity. Return to full athletic activity was reported in 61-93% of athletes at an average of 6.5 months.

Donor site morbidity limits the indications of this procedure to lesions smaller than 2-3 cm.

Postoperative management

If the lesion is smaller than 2 square cm and the surrounding cartilage is stable, partial weight bearing (30#) can be implemented for the first 6 weeks. If the defect is larger, and the surrounding cartilage is unstable, non-weight bearing for 6 weeks should be performed. Passive motion of the knee starts on day 1 and is crucial for cartilage nutrition.

Autologous Cartilage Implantation (ACI)

This technique was developed in Sweden in 1987. The procedure takes place in three stages. In a first stage, between 200 and 300 milligrams cartilage is sampled arthroscopically from a less weightbearing area from either the intercondylar notch or the superior ridge of the medial or lateral femoral condyle of the patient. The matrix is removed enzymatically and the chondrocytes isolated. These cells are grown in vitro in a specialized laboratory for approximately four to six weeks, until there are enough cells to re-implant on the damaged area of the articular cartilage.

The patient then undergoes a second treatment, in which the chondrocytes are applied on the damaged area during an open-knee surgery. These autologous cells should adapt themselves to their new environment by forming new cartilage. During the implantation, chondrocytes are applied on the damaged area in combination with a membrane (tibial periosteum or biomembrane) or pre-seeded in a scaffold matrix. This is potentially the best procedure, but the problem traditionally has been the strength of the membrane or matrix in which the chondrocytes are placed. Essentially, the concept is based on the use of biodegradable polymers as temporary scaffolds for the in vitro growth of living cells and their subsequent transplantation onto the defect site. Various synthetic or natural materials such as hyaluronan, collagen, and fibrin glue have been developed and applied to cartilage tissue engineering.

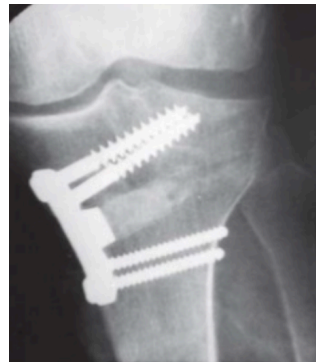
Much of the research the last few years has focused on developing stronger scaffolds. However, none have demonstrated a perfect regenerative potential yet. One of the disadvantages of ACI procedures is the requirement of multiple procedures due to the tissue harvest.

Encouraging outcomes, both clinical and radiological, have been reported in the literature at short- and mid term. Good to excellent results are reported in 72-96% of the patients. Best results were obtained with lesions of the medial femoral condyle.



Combined pathology such as malalignment, ligamentous instability, and meniscal injuries are frequently encountered by the surgeon treating cartilage deficits in the knee. Surgically addressing these concomitant pathologies is critical for effective and durable cartilage repair. Recent data demonstrate that combined procedures have no significant negative effect on the ability to return to athletics after any cartilage repair procedure.

The cartilage repair tissue has to be protected from excessive weightbearing to assure long-term success. This can be done through unloading the injured area, either through the use of unloader braces or through surgical means.



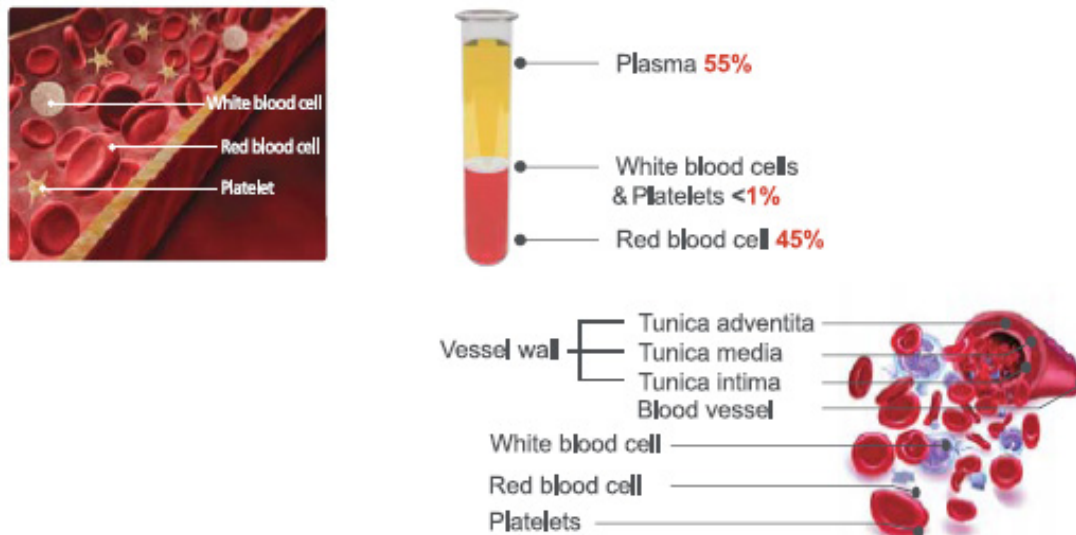
The proper timing for an unloading osteotomy depends on several factors such as lesion size, alignment, degree of instability and bodyweight.

Indications:

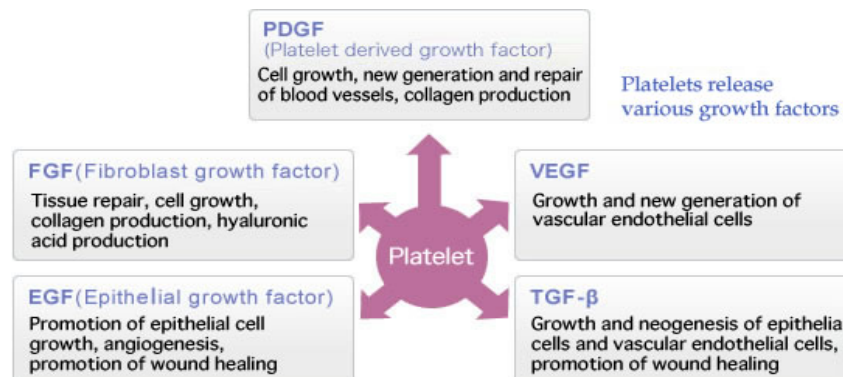
- Large defects without malalignment
- Small-large defects with malalignment
- All defects with surrounding poor quality cartilage

Platelet rich plasma injections.

The perspective of applying biological strategies for management of cartilage pathology has led to a growing interest in the field of growth factors and stem cells. The preeminent role is played by platelet rich plasma (PRP). Platelet derived growth factors (GF) contained in PRP are the most exploited way to administer a biological stimulus to damaged tissues such as cartilage, tendons and muscles. Platelets have many functions beyond simple hemostasis (stopping of the flow of blood). Platelets have been documented to act as a reservoir of many autologous GF (>6) as well as many bioactive and anti-inflammatory molecules. These factors regulate key processes involved in tissue repair, including cell proliferation, cell migration, cell differentiation, development of new blood vessels, and extracellular matrix synthesis. The rationale for the use of PRP is to stimulate the natural healing cascade and tissue regeneration by a “supraphysiological” release of platelet derived factors directly in the site of treatment.



Growth factors mediate the biological processes necessary for repair of soft tissues (muscle, tendon and ligament) following injury and animal studies have demonstrated a clear benefits in terms of accelerated healing. However, which growth factors are more beneficial under specific circumstances needs to be better understood.



PRP has been applied as a conservative approach, and as a biological augmentation during surgical procedures. Most of the research so far has dealt with knee application.

As a conservative approach, results of the studies suggest that the best responders were patients with lower grade OA. It demonstrates that PRP is a safe treatment, capable of improving articular function, reducing pain during daily activities and delaying more invasive procedures. Its effect is time dependent though, with results worsening over time. It is estimated that the median duration of PRP is approximately 9 months. Patients up to age 60 have a greater chance to benefit from PRP approach.

PRP is superior with respect to other traditional approaches such as viscosupplementation. This is the injection of hyaluronic acid in the joint space. The proposed effects are anti-inflammatory and inhibition of tissue nociceptors. Extensive research has been done to look for the efficacy, but not enough evidence is there to recommend Clinical Practice Guidelines. Lower level studies suggest it is safe to use, and provides short term relief in early stages of OA.

As an augmentation to surgical procedures, the findings are encouraging, but lacks RCT's to fully endorse PRP as a valid strategy to improve clinical outcomes.

Stem cells

A stem cell is an “immature” or undifferentiated cell that is capable to differentiate into other types of body cells. This property may be perpetuated over many generations, resulting in considerable amplification of their numbers. Adult stem cells are found in specific niches or tissue compartments. The best are derived from the bone marrow.

Currently procedures are developed utilizing autologous bone marrow stem cells combined with various scaffolds for the treatment of full thickness cartilage defects in a single step procedure. The advantage of a single step procedure is that there is no need for culture, thereby avoiding the expense for an extra procedure to retrieve chondrocytes from chondral biopsy. This decreases the total cost as well as donor site morbidity.

Therapy

Articular cartilage healing is different since cartilage is avascular, aneural and alymphatic. Even though articular cartilage has no blood supply, chondrocytes show a high level of metabolism. Chondrocytes derive their nutrition mainly from synovial fluid and to a lesser extent from the underlying bone. The high level of metabolism is mainly due to proteoglycan turnover. The turnover time for proteoglycans is 2-3 months. The absence of blood vessels and a very tight matrix prohibit chondrocyte migration from adjacent healthy cartilage towards the wound. Both these factors exclude cell based repair and cartilage regeneration.

Current evidence on PT after cartilage repair is inadequate. For ACI procedures, the evidence has been described as “in its infancy” which is ominous. The role of PT is therefore underestimated.

One of the main factors that need to be addressed is the control of shear forces in the joint. Cartilage does not tolerate shear forces well, and this needs to be controlled by restoring proper joint mechanics and motor control. Joint mechanics are optimized through joint mobilizations. This will bring the instantaneous axis of rotation back to where it needs to be, thereby distributing the load evenly through the joint.

Assess for movement impairments around the involved joint. Keep in mind that in knee problems, the movement impairment will likely involve hip and foot/ankle.

Modifying joint loads is a key factor in cartilage rehabilitation. This can be achieved through surgery, bracing, joint mobilizations and strengthening/ coordination exercises.

Research shows that high repetition, low loading joint loading exercises are essential in maintaining articular cartilage health, and supports the nutrition of cartilage by improving diffusion of synovial fluid.

The first 12 weeks after cartilage repair are very important. Treatment guidelines are all based on protection of the repaired tissue and an optimal functional gain without jeopardizing the cartilage repair.

In the initial phase after surgery, weightbearing activities should be limited and controlled. If the lesion is located in the weightbearing area of the femoral condyle, load should be limited by using crutches for at least 3 weeks. If the lesion is in the patellofemoral joint, ROM needs to be limited by using a brace

Since chondrocytes derive their nutrition mainly from synovial fluid, low loading/high repetition type exercises are key in stimulating joint circulation. Synovial fluid gets stimulated and diffusion between cartilage and synovial fluid is facilitated. Activities that achieve this are CPM, cycling, rowing (both with minimal resistance), and total gym.

Bracing is often used to protect the repair site. Post op bracing is used for patellofemoral repairs, where it is important to control the flexion angle of the knee. Unloader braces are used for repairs on tibia and femur. It is also used if the patient displays significant AGMR on weight bearing.

To prevent re-injury, try to identify then underlying cause of the problem. Educating the patient about the possible risk factors for re-injury and the need to exercise to overcome the risk is hugely important.

Prescription for cartilage training

How many repetitions/sets/intensity for the exercises?

Research is sorely lacking in this aspect. Therefore we still fall back on the work of the Norwegian manual therapists who were the first to realize that different tissues needed different stimulus for optimal effect.

- Training load at 20% of 1 RM
- Slow speed
- 1000 repetitions or more
- Minimal to no muscle fatigue should occur
- Always exercise with optimal proximal control
- Change directions by pivoting the feet in semi flexion
- Avoid sustained postures over the repaired site
- Avoid weight bearing on the leg when moving within the joint angles mentioned.
- The use of crutches is advised as long until swelling and pain are not controlled.

Signs of overload of articular cartilage:

- Joint stiffness, especially in the AM
- Swelling
- Loss of ROM
- Aching

After ACI procedures

As the graft is most vulnerable within the first 12 weeks postoperatively, weightbearing (WB) activities have to be reduced to a minimum. Therefore, a controlled rehabilitation program incorporating a structured exercise program and partial WB is recommended. However, little information is available about the duration of partial WB and the progression to full WB.

It has been reported that longer periods of non-WB lead to the loss of functional properties of joint structures, and therefore, unloading during the postoperative period might be detrimental to the healing of repair tissue because of the reduced chondrocyte stimulus. On the other hand, caution must be used when applying high and excessive loads to avoid the risk of cell damage, early degeneration, or graft failure associated with the ACI technique.

A pilot study by Wondrasch et al in 2009 demonstrated that accelerated WB (full WB after 6 weeks) compared with delayed WB (full WB after 10 weeks) in patients undergoing ACI of the femoral condyle resulted in good clinical and MRI- based outcomes without harming the graft 2 years postoperatively. Although a tailored rehabilitation process has been described as one of several factors influencing the graft and clinical outcomes after ACI, the evidence base of this rehabilitation process is considerably deficient. In particular, the resumption of WB activities seems to be very critical regarding graft protection in the early postoperative phase, when the graft is most vulnerable. A RCT by Wondrasch et al in 2015 also showed no significant difference between the AWB and DWB groups 5 years postoperatively.

Patellofemoral joint lesions

It is important that you know where exactly the lesion occurs in the joint, and how large it is. Contact areas of the patellofemoral joint differ at different knee angles and knowledge of this is essential for describing the right exercises. This also applies to lesions on the femur or tibia. It makes you understand why some exercises at specific knee angles are indicated and others not

The following table gives some guidelines as to how to adapt training post surgery for repairs of the patella, in order to avoid excessive stress and control loading of the repair site. Three factors are taken into account: exact size and location, and the condition of the borders of the repair (contained or not contained).

Location on		Repair size (cm ²) and borders	Avoid shear-stress between	Intermittent controlled loading in CKC between
Patella	Trochlea			
<i>Inferior</i> 	<i>Superior</i> 	< 2.5	0° to 30° flexion	0° to 30° flexion
		> 2.5, contained	0° to 40° flexion	0° to 40° flexion
		> 2.5, uncontained	0° to 40° flexion	0° to 60° flexion
<i>Central</i> 	<i>Central</i> 	< 2.5	20° - 75° flexion	20° - 75° flexion
		> 2.5, contained	10° - 85° flexion	10° - 85° flexion
		> 2.5, uncontained	10° - 85° flexion	5° - 90° flexion
<i>Superior</i> 	<i>Inferior</i> 	< 2.5	45° - 130° flexion	35° - 140° flexion
		> 2.5, contained	45° - 130° flexion	35° - 140° flexion
		> 2.5, uncontained	45° - full flexion	35° - full flexion

The following table gives some guidelines as to how to adapt training post surgery for repairs of the femur condyle, in order to avoid excessive stress and control loading of the repair site. Three factors are taken into account: exact size and location, and the condition of the borders of the repair (contained or not contained)

Location on femur	Repair size (cm ²) and borders	Avoid shear-stress between	Intermittent controlled loading in CKC between
 Anterior	< 2.5	0° to 30° flexion	0° to 40° flexion
	> 2.5, contained	0° to 60° flexion	0° to 70° flexion
	> 2.5, uncontained	0° to 110° flexion	0° to 120° flexion
 Central	< 2.5	20° - 80° flexion	10° - 90° flexion
	> 2.5, contained	20° - 110° flexion	10° - 120° flexion
	> 2.5, uncontained	10° - 120° flexion	0° - 130° flexion
 Posterior	< 2.5	45° - 130° flexion	35° - 140° flexion
	> 2.5, contained	45° - 130° flexion	35° - 140° flexion
	> 2.5, uncontained	45° - full flexion	35° - full flexion

To summarize, the following factors need to be taken into account before rehab can start:

- Exact location of the repair
- Size of the repair (smaller/larger than 2.5 cm square)
- Condition of the borders of the repair
- Duration of symptoms before the surgery (more/less than one year)
- Pre injury activity level (professional/non-professional, competitive/non-competitive, high/low impact, high/low training volume)
- Movement impairment syndromes
- Changes in body weight

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Muscle

Strength

Strength is defined as the maximum force that a muscle can generate. Someone who can bench 220lbs has twice the strength as someone who can bench 110#.

Strength development is optimized by moderate to high resistance and low to moderate repetitions (60-80% of 1RM and 6-12 reps).

Endurance

Endurance is defined as the capacity to sustain repeated muscle contractions (as in running or cycling) or the capacity to sustain static muscle contraction (as when trying to pin an opponent in judo)

Endurance is optimized by low to moderate resistance and moderate to high repetitions (30-70% of 1 RM and 10-25 reps).

Power

Power is defined as the product of force and velocity. It is the explosive aspect of strength.

Power development is optimized by alternating low to moderate resistance and low repetitions (30-60% of 1RM and 3-6 reps) at an explosive velocity, with the traditional strength training recommendations of moderate to high resistance and low to moderate repetitions (60-80% of 1 RM and 6-12 reps).

Muscle size

When the training objective is to increase muscle size, an important objective for bodybuilders, moderate to high resistance with low to moderate repetitions should be used (70-100% of 1 RM and 1-12 reps)

Selecting the appropriate number of sets

It generally has been assumed that a minimum of 3 sets of each exercise is needed to provide the greatest gains in muscle strength and size. In the 1990s scientists started to challenge this assumption. Studies began to show that a single set might be just as effective as multiple sets for increasing muscle size and strength. Single sets are likely appropriate for untrained people for the first 6-12 months, but multiple sets are important for further gains in strength, endurance, power and hypertrophy. These findings have significant implications for designing training programs, as it would substantially reduce the total work out time. It also allows for the inclusion of a greater variety of exercises, instead of multiple sets within the same time period for those who are just beginning resistance training. This likely also applies to those who want to maintain a basic level of fitness and who are not interested in further improvement.

Emphasis	Training level	Loading	Volume	Velocity	Frequency (times per week)
Strength	Novice	60-70% 1RM	1-3 sets, 8-12 reps	Slow, moderate	2-3
	Intermediate	70-80% 1RM	Multiple sets, 6-12 reps	Moderate	2-4
	Advanced	1RM ^b	Multiple sets, 1-12 reps ^b	Unintentionally slow to fast	4-6
Hypertrophy	Novice	60-70% 1RM	1-3 sets, 8-12 reps	Slow, moderate	2-3
	Intermediate	70-80% 1RM	Multiple sets, 6-12 reps	Slow, moderate	2-4
	Advanced	70-100% 1RM; emphasis on 70-85% ^b	Multiple sets, 1-12 reps, with emphasis on 6-12 reps ^b	Slow, moderate, fast	4-6
Power	Novice	>80% 1RM—strength; 30-60% 1RM—velocity ^b	Train for strength	Moderate	2-3
	Intermediate	>80% 1RM—strength; 30-60% 1RM—velocity ^b	1-3 sets, 3-6 reps	Fast	2-4
	Advanced	>80% 1RM—strength; 30-60% 1RM—velocity ^b	3-6 sets, 1-6 reps ^b	Fast	4-6
Endurance	Novice	50-70% 1RM	1-3 sets, 10-15 reps	Slow—moderate reps Moderate—high reps	2-3
	Intermediate	50-70% 1RM	Multiple sets, 10-15 reps or more	Slow—moderate reps Moderate—high reps	2-4
	Advanced	30-80% 1RM ^b	Multiple sets, 10-25 reps or more ^b	Slow—moderate reps Moderate—high reps	4-6

Summary recommendations, derived by the American College of Sports Medicine .

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Stretching

We all know that stretching is an important part of a therapy treatment. However, for such a common treatment, there is much controversy. Many theories exist, but there is not much consensus as to what needs to be stretched, how long, how hard, how often etc.

What are we trying to achieve with stretching? The following objectives can be found:

- Injury prevention
- Increase flexibility
- Lengthening of muscles
- Increase joint ROM
- Prevent delayed onset of muscle soreness
- Increasing mechanoreceptor activity, which in its turn will help to improve coordination

Before we stretch though, there is a question that need answering.: is it useful to have long muscles everywhere in the body? Every muscle has its optimal length-tension ratio and our lifestyles (prolonged sitting), passions and hobbies can disrupt the “normal”. In high-level athletes, muscles have adapted optimally to function at that athletes highest level. That also means that some muscles have actually shortened functionally. For example, cyclists and speedskaters have functionally short hip flexors, as they both those sports are performed in a sustained flexion type posture. Specialists in butterfly have functionally short pectoral muscles, which makes it harder for them to do freestyle.

You have to wonder if it is a good idea to stretch those functionally shortened muscles, as you will basically negate their functional adaptations for that specific activity.

Stretching is also frequently done to decrease the intensity of delayed onset of muscle soreness. Usually the muscle soreness is pretty severe for 2-3 days after the activity and then starts to decrease although muscle fibers take about 2 weeks to completely recover. Stretching is frequently done to decrease the tightness and soreness that occurs after activity. However, if the cause of the muscle soreness is tissue damage, it's somewhat hard to see how stretching right after the activity can have a stimulating effect on the healing process.

Research show that prolonged static stretching of the hamstrings after activity can lead to an increase of the muscle soreness. Prolonged stretching will decrease the lumen of the capillaries, which decreases the ability of the muscle to get rid of the excess metabolites. Also hard stretching can lead to further damage to already impaired muscle tissue.

Cooling down with light dynamic muscle contractions is probably a better way to restore aerobic activity in the muscle. It has been shown that normally it takes about 1 hour to get rid of the excess lactic acid in the muscle. Low intensity cooling down decreases the time it takes to get rid of metabolites by 50%. It also helps to get rid of excess fluid build up in the muscle, which can cause shortening/tightening of the muscle. Stretching right after working out probably does not help with any of this and can possibly lead to more tissue damage when done too hard.

There is moderate to strong evidence that routine static stretching does not reduce overall injury rates (Small et al., 2008). There is preliminary evidence, however, that static stretching may reduce musculotendinous injuries (Small et al., 2008). Zakaria (2015) looked at using stretching as a method for injury prevention in high school soccer players and found no difference between dynamic stretching and static stretching in the prevention of lower-extremity, core, and back injuries. Overall, the study showed that static stretching did not provide any added benefit to dynamic stretching in the prevention of injury in this population before exercise.

From these two studies it would appear that stretching has no protective value in preventing injuries.

Research by Sekir et al. (2015) disproved the loss of power theory by showing that there is no decrease in power output in the functional hamstring to quadriceps ratio following a static stretching routine. This result supports the idea that athletes can confidently perform static stretching during their warm-up routines and not worry that they are negatively affecting the functional strength of their muscles. A study by Clark and colleagues (2014) showed that while dynamic stretching is beneficial in decreasing presynaptic inhibition, it did not lead to the hypothesized increase in power output. From these two studies, it would appear that stretching has no positive or negative effect on power output.

Food for thought

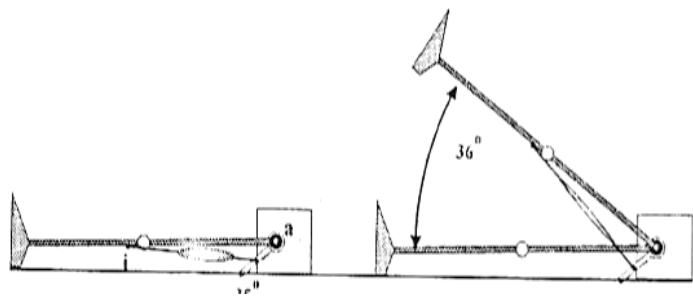
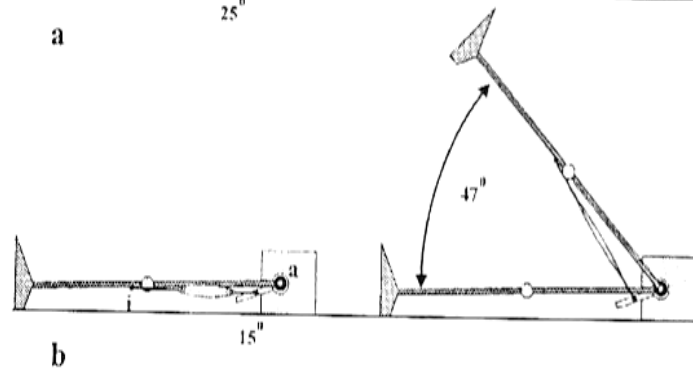
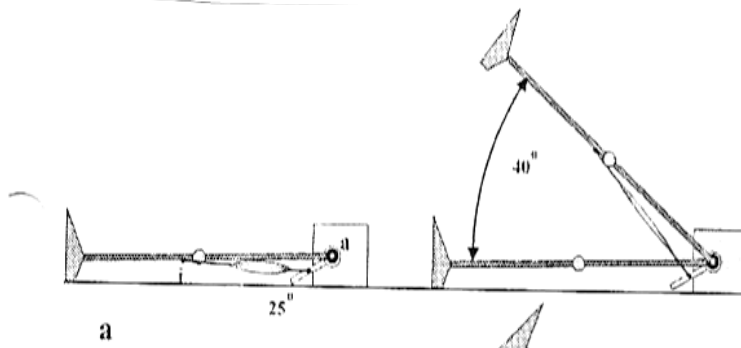
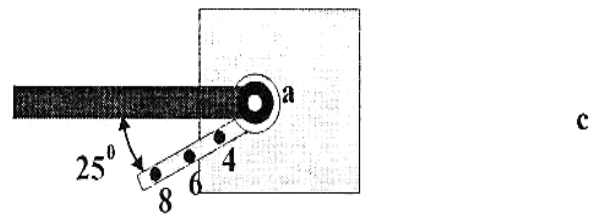
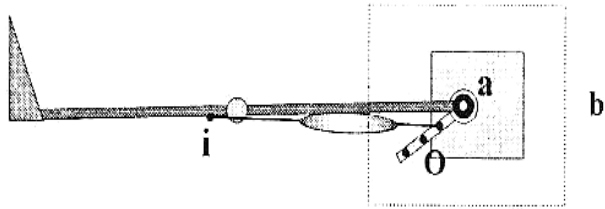
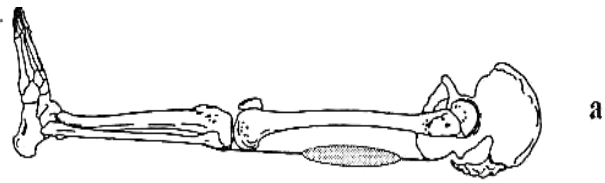
The limiting factor of the stretching capability of a muscle is the elastic component of the muscle tissue. From its resting position, a sarcomere can shorten 41% maximally, and lengthen 52% maximally. Percentage wise, this is the same for every muscle throughout the body. This is not trainable. So why, for example, can some people have a straight leg raise of 120 degrees, while other people only have 60 degrees? A possible explanation is the exact insertion point of the tendon. It appears that small differences in the angle of insertion, and the exact spot where the tendon inserts on the greater tubercle (more proximal/distal) have great influence on the straight leg raise score, while the stretching ability of the muscle has remained the same.

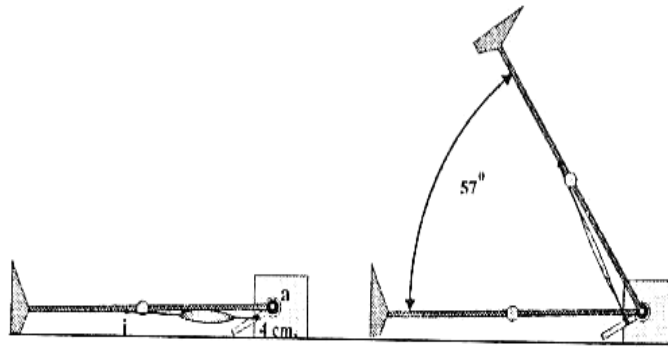
It also appears that small changes in the concavity/convexity of ball and socket have a great influence on the amount of movement available in a joint. If both these

arguments are true, it is very possible that true flexibility in a joint is not really trainable.

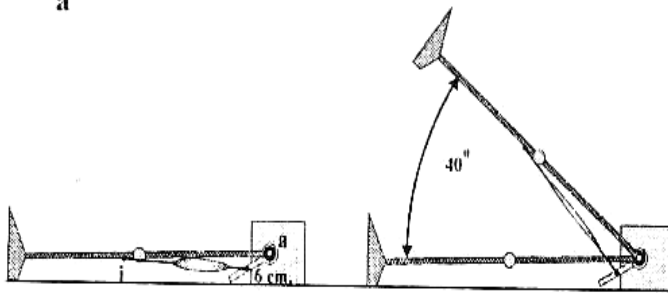
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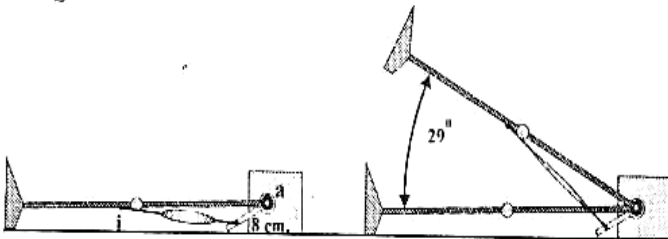




a



b



c

Stretching of non contractile tissue

Collagen fiber elongation of 1-1.5% for < 1 hour causes no permanent deformation.

1.5-2% elongation will cause more permanent lengthening.

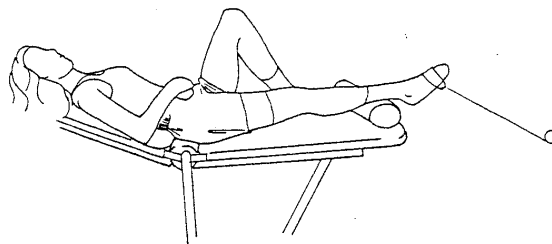
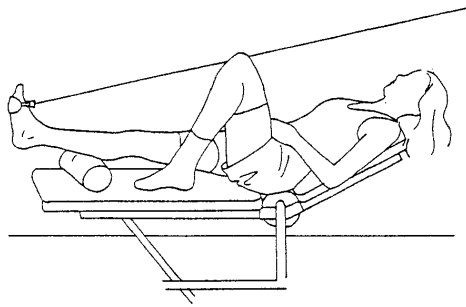
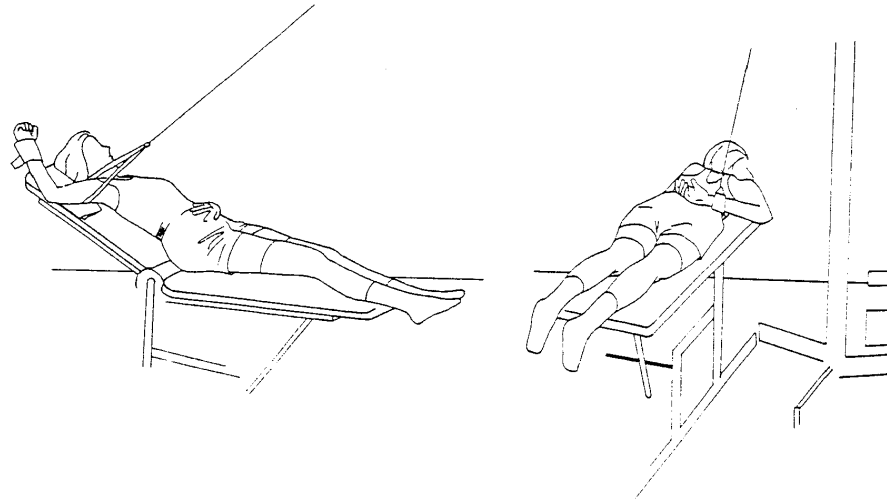
Stretching with the intent to permanently elongate the tissue must be followed up within 24 hours by another session.

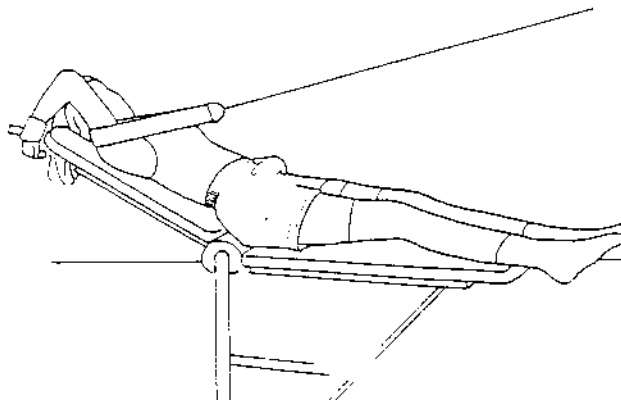
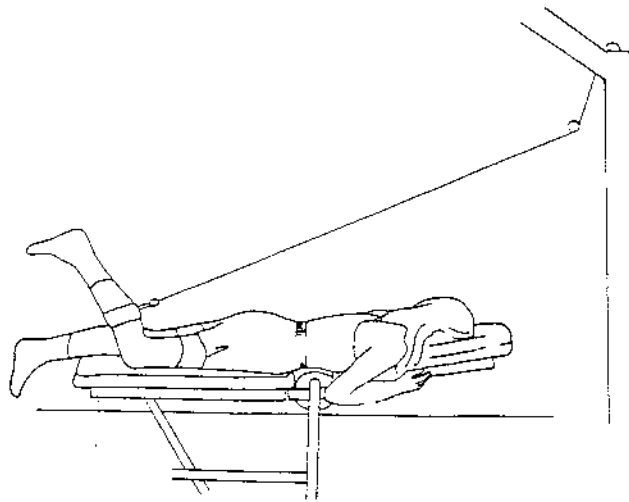
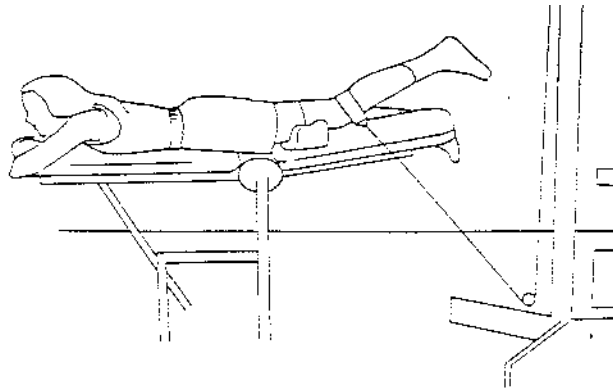
Short-term stimulus

- Typically used in manual therapy
- 4-6% strain is common.
- Inflammation typically results (a strain of 6-8% has been shown to tear tissue, with inflammation following)

Long-term stimulus

- No more than 2% strain, for at least 20 minutes
- Excessive force is identified by inflammation
- Average rate of gain is 3 degrees per week, with a range of 1-9 degrees.
- After the first session, reduce the applied stress, as the tissue is probably weaker due to the disruption of bonds. If the same level of stress is applied, inflammation will result.





Unloading

In order to restore normal movement patterns and/or prolong the number of repetitions performed in an exercise session, one may exercise with less than full body weight as resistance. In many cases this will make it possible to eliminate pain and improve neuromuscular recruitment. This allows re-establishment of normal movement patterns and an increase in AROM. A patient can then perform a sufficient number of repetitions for a given activity so that circulation can be challenged, O₂ depleted and the super compensation during the rest period can aid in tissue healing and strengthening.

Most of us are familiar with water therapy, where the buoyancy gives the chance for longer duration exercise as there is less pain connected with exercise in water. Even more acute patients can go through high repetition exercises. However, pool therapy requires very expensive equipment and takes up a lot of space. Unloading equipment does not take up much space and is fairly inexpensive in comparison.

Principles

Patients will sometimes describe that their joint pain increases upon weightbearing, or as time goes by. It is possible to imagine that the stresses caused by gravity may surpass the margin of tolerance in the tissues to such a degree that it causes pain and/or a possible inflammatory reaction. The reasons for such a response can be several:

- Disruption of tissue leading to compromised integrity
- Degenerative changes in the tissue, with resulting low level inflammation being present.
- Tissue is traumatized, either an acute major trauma, or multiple micro trauma, such as weekend warrior sports in the middle aged individual with sporadic bursts of intense physical exertion.
- Tissue may suffer from decreased energy supply locally, especially secondary to long-term immobilization. Mobilization has then started too soon, too fast and not enough time has been permitted for adaptive changes in the energy production mechanisms.

Pain during weightbearing in most cases will lead to compensatory reactions in the neuromuscular system. These include abnormal firing patterns, abnormal gait, and abnormal postures. If pain can be eliminated by partial weightbearing during gait, such compensatory reactions will disappear. Tissue healing will improve and adverse tissue reactions will disappear.

Another reason to use unloading may be related to muscle weakness. In this case, weightbearing and locomotion lead to early onset of fatigue. ADL, activities related to work, and/or leisure pursuits will be limited and painful. There is a linear relationship between load and O₂ consumption in the tissues. In the case of early onset of muscle/tendon fatigue, it is possible to postpone the fatigue reaction and

have the patient exercise longer and thus present a larger stimulus to the muscle/tendon structures in question for increasing internal energy supply.

Cautionary note

All back exercises using unloading on the treadmill must be painfree or at least not increase pain. In the case of a patient who has constant pain, we generally contract with the patient not to have increased tightness or discomfort during the exercise. Increased tightness during the exercise usually translates into increased pain afterwards. Therefore you should repeatedly remind the patient to inform about increasing tightness and give them the responsibility to stop the exercise when that occurs. We have found we need to remind our patients repeatedly, and we still have patients who exercise with increased discomfort and thus create more pain after treatment.

Exercise with unloading equipment must always be attended by an aide or a therapist.

Indications for use of unloading

- When weight bearing hurts
- When there is a limp
- When there is more pain at the end of the day
- When weight bearing causes swelling
- When weight bearing AROM is much less than non weight bearing AROM
- When motion segments have abnormal movement and firing patterns
- When ligaments, tendons and muscles are injured and weak

Unloading for low back HNP and radiculopathy

- No functional tests recommended
- Assist up to 85-95% of trunk weight (TW) which is 65% of body weight (BW)
- Use slow TM .1 - .5 mph
- Take frequent breaks if tightness, soreness or pain presents
- If possible ambulate 40 - 50 minutes
- If pain relief is short, change speed, change incline or try a more forward bent posture
- Use UBE with or without unloading

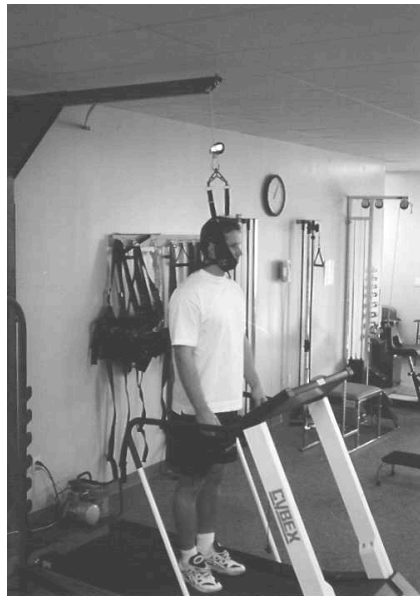


Unloading for patients with spinal stenosis

- No functional test needed
- Assist 20 - 40 % of TW
- Ambulate .8 to 2 mph
- Use slouched or forward bent position if necessary
- Use UBE sitting, with or without unloading
- Progress to 50 minutes of ambulation
- Include squatting with hip flexion
- Progress pulley exercises from sitting to standing
- Progress UBE from sitting to standing

Unloading for neck patients

- Assist up to 70 % of head weight
- Head weight is 8 % of body weight
- Walking speed from .8 mph to 3 mph
- With HNP and interscapular flatness use forward incline rope
- Progress to UBE with unloading
- Do not use for TMJ patients
- Functional test with treadmill or UBE with and without unloading



Unloading exercises are not just applied to the trunk but to the extremities as well. In order to restore normal movement patterns and/or prolong the number of repetitions performed in an exercise session, one may exercise with less than full body weight as resistance. In many cases this will make it possible to eliminate pain and improve neuromuscular recruitment. This allows re-establishment of normal movement patterns and an increase in AROM. A patient can then perform a sufficient number of repetitions for a given activity so that circulation can be challenged, O₂ depleted and the super compensation during the rest period can aid in tissue healing and strengthening.

If a patient has pain or exhibits weakness during arm elevation, a number of factors could be the cause. These range from cervical spine or interscapular stability to tendinopathy or ligament weakness in the shoulder joint. A detailed evaluation will help to pinpoint the weak link in the chain. There is nothing that will promote the ability to use the arm better than to exercise and train with those certain patterns of movement that are deficient.

When we want arm elevation to improve, we have to engage arm flexors. In order to minimize soreness after exercise we also want to balance the assist in such a way that we eliminate the eccentric phase of the exercise. Therefore when working with the concept of max assist, the assist is monitored in such way that every movement is concentric/concentric. So concentric flexion is followed by concentric extension.

The following examples of arm elevation are used to illustrate the concept of max assist at the end of available range.

Max assist at 90 degrees

With a pulley, a weight will give the greatest assist when the rope from the pulley is perpendicular to the body part that is assisted. Thus, when max flexion is 20 degrees, the pulley is set up in such way that the rope and the arm exhibit a 90-degree angle at 20 degrees of flexion. When maximum range is 60 degrees the person performing arm elevation, the pulley is set up in such way that the rope and the arm exhibit a 90 degree angle at 60 degrees of flexion.

Assisted exercises max assist

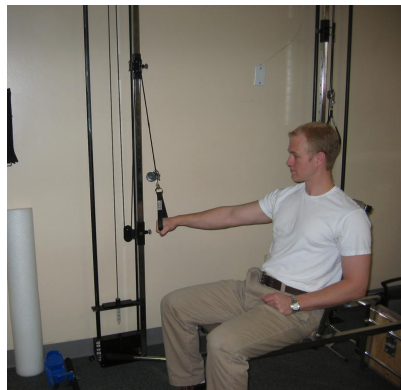
Exercise 1

Patient sitting and we are applying max assist at 20 degrees of flexion.



Exercise 2

The patient is performing flexion and receiving max assist at 90 degrees elevation.



Exercise 3

The patient is sitting and receiving max assist at 120 degrees of elevation.



Examples of exercise programs for selected conditions

Patients with pain in the cervical spine following whiplash who present in the sub acute phase, need repeated low level, low range contractions for the cervical muscles.

The purpose of these exercises is to provide a stimulus for bloodflow and therefore, tissue healing and regeneration.

Exercise 1

Supine assisted bilateral arm elevation. The assist is usually between 2-3 pounds. Ensure that the exercises are performed as concentric/concentric flexion/extension movements.



Exercise 2

Patient sitting and assisting arm elevation with adduction. This is a concentric/concentric exercise. The head is supported. The further you take the arm in flexion/adduction, the more strenuous the exercise. The exercise is initially performed with 10-15 reps per set with 2-3 minutes rest in between, and working as many sets as possible without reproducing symptoms, up to 10 sets per session, as well as gradually working up to 30 reps per set.



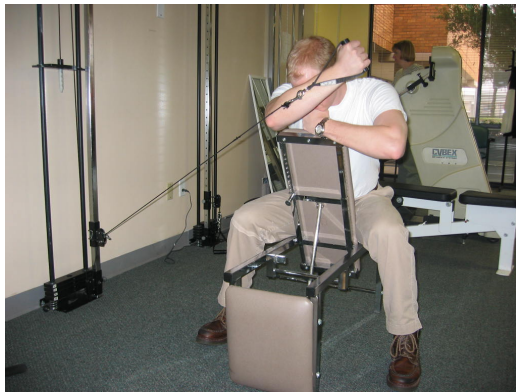
Exercise 3

Resisted arm elevation in reclined position. During transition from assisted to resisted arm elevation it might be practical to continue the assisted arm elevation and then let the last set turn into resisted exercise.



Exercise 4

Patient sitting side ways and resisting arm elevation with adduction.



Exercise 5

Supine assisted arm elevation with elbow extension.



Exercise 6
Sidelying push.



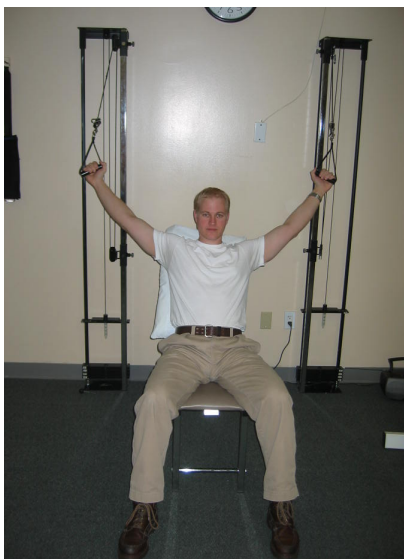
Exercise 7
Sidelying pull. A lower progression exercise would be just to extend the humerus to neutral. A higher progression for the trunk musculature would then be to combine posterior trunk rotation towards extension with the pull.



CT mobilization

Exercise 1.

Supine assisted bilateral arm elevation.



Exercise 2

Sitting with trunk and neck supported - CT extension



Thoracic mobilization

Exercise 1

Extension



Exercise 2

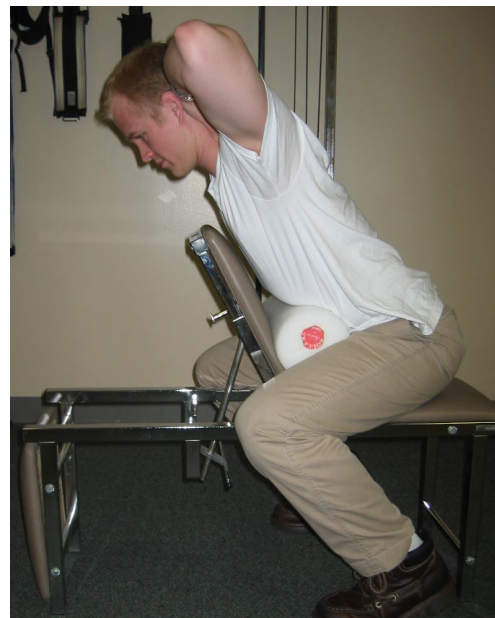
Sidebending



Shouldergirdle - upper trunk strengthening

Exercise 1

Sitting with trunk support. Trunk extension with arm abduction/external rotation.



Exercise 2

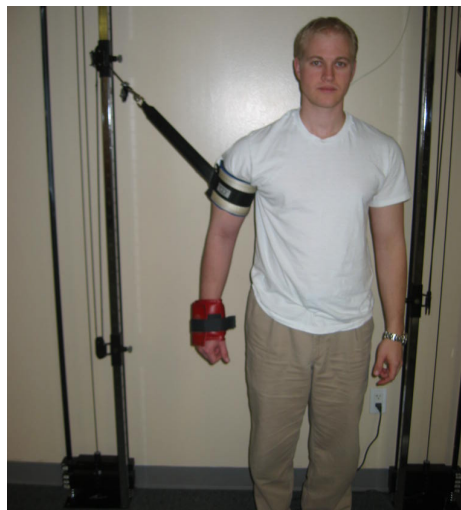
Sitting without support. Trunk extension with arm abduction/external rotation.



Exercise 3
Kneeling trunk extension from prone position.



Exercise 4
Standing resisted abduction with traction.



Exercise 5
Y's and T's on slant board



Conditioning and stabilization exercises low back

Intermediate

Extension of spine on angle bench to neutral only. No hyperextension.
 Extension of spine over dome pillow.
 Seated forward bend with neutral spine.
 Seated knee lifts.
 Prone leg lifts.
 Prone active extensions.
 Slant board sit ups.
 Supine pulley rotations.
 Seated lat pull down. Use underhand grip with hands close together and pull to chest level only.

Advanced

Seated pulley rotation.
 Standing lat pull downs.
 Slant board sit ups, progress to a more horizontal position.
 Seated 1-2-3-4
 Standing forward bending (kinesthetic training)
 Corner pulleys, in diagonal patterns while keeping spine in neutral.
 Diagonal pulley rotation in standing

Low back

Assisted back extension, standing.

Resistance between 4-16 pounds. Patient bends forward by curling the shoulder girdle and upper back. Useful to get an extension movement and contraction of bilateral extensors in lumbar spine without creating much tension.

Good exercise for patients who have repeated low back pain and exhibit strong palpation tenderness all the way down to the L-S junction insertion of the multifidi and erector spinae. These patients have what may be considered a tendinitis of the low back muscles.

In order to strengthen the low back muscles you must first address the irritability of the tendons and its insertions, for which this exercise is good.



Assisted back extension, sitting



Seated pulley rotation



Supine pulley rotation



Diagonal pulley rotation



Conditioning and stabilization exercises cervical spine and cervicothoracic junction

Intermediate)

Neck sidebends on slant board, progress to a more horizontal position. Sidebend to neutral only.

On slant board, perform Y's and T's.

On slant board, prone chin tucks.

Shoulder shrugs with dumbbells.

Supine cervical pulley rotations.

Lat pulls. Underhand grip, pull to chest only and keep chin tucked.

Seated rotation facing pulley with strap around shoulder. Localize movement to interscapular and cervical area.

Chin tucks with neck flexion on slant board.

Advanced

Cervical pulley rotation with increased resistance.

Scapular retractions with lat bar. Use angle bench reclined to 45 degrees, head supported, pull bar to chest.

Seated, with strap around head, sidebend with pulley resistance.

Cervical spine

Cervical pulley rotation



Chin tuck and neck flexion on slant board



Chin tuck and neck flexion on slant board with superimposed arm movement

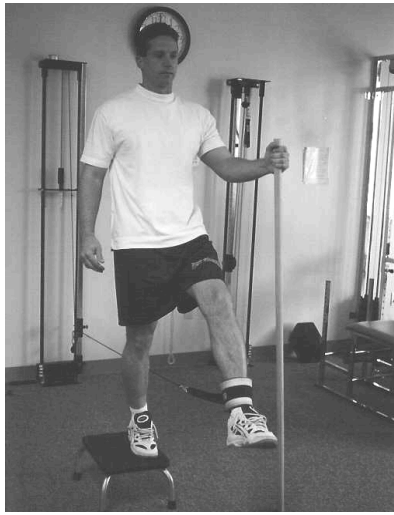


Knee exercises

Endrange flexion/ extension



Resisted flexion/ extension of the uninvolved leg. This requires significant stability of the involved extremity.



Assisted knee extension



Ankle foot exercises

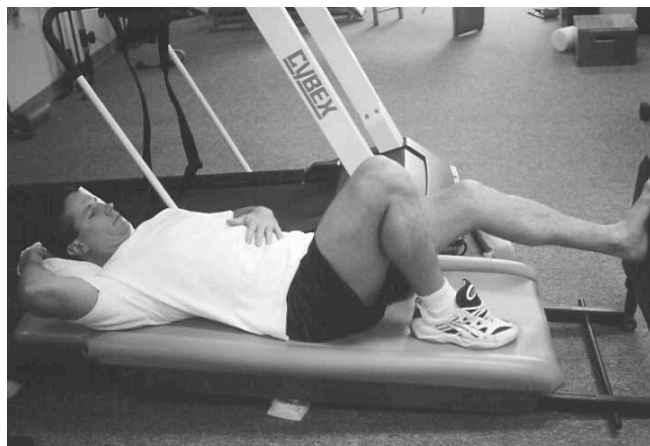
Total Gym half squats in sidelying

The foot is placed forward on the foot plate, as far forward as necessary to squat without pain.



Total Gym half squats with patient supine.

As you progress you can place more of the ball of the foot on the plate, thus permitting more dorsiflexion. Start with bilateral squats, progress to unilateral.



Unloaded walking.

Start with speeds between .1 and .5 mph and instruct the patient to walk with short steps.

Unloaded diagonal walking.

When the patient's weightbearing tolerance improves, it is important to start with diagonal walking. Straight walking will mobilize/activate mostly the talocrural joint. With diagonal walking you will also activate the other joints in the foot.

