Hamstring strains: a state-of-the-art review of the involved pathophysiology to examine current rehabilitation concepts

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Abstract

Background Hamstring strains are a frequent occurrence in sports making up more than ⅓ of all muscular injuries. Despite of this high prevalence, to date only little to mostly inconclusive evidence exists for their rehabilitation. This study will ‘go back to the basics’ and comprehensively review the involved pathophysiology of partial muscle tears with an emphasis on the biceps femoris long head (BFLH). With this basis, current rehabilitation concepts designed to optimize recovery will be assessed towards their effects on physiological recovery and specific recommendations towards a more adequate conceptual rehabilitative framework will be presented.

Methods A comprehensive literature search was carried out within the Medline (PubMed) database including studies published between Dec 1st 2004 and Dec 5th 2010. In total 59 studies met eligibility criteria and were included for the review. Three rehabilitative concepts designed to optimize recovery were examined on a pathophysiological basis: (1) PRICE (Protection, Rest, Ice, Compression, Elevation), (2) strengthening, (3) stretching. An illustrative flowchart was synthesized as a result.

Results The 59 included studies used to review the pathophysiology of BFLH partial tears were able to form a complete picture of current pathophysiological understanding. 1) Strengthening appears to have several enhancing short term and long term effects, however, should only be started after a four to six days period of relative immobilization. Whether connective tissue adaptations from strengthening serve an instrumental or detrimental role is unclear. 2) Stretching yields potential regeneration enhancing effects and can already be started at light intensities after two to three days post-injury by avoiding keeping the limb in shortened positions. Stretching should not be used for increasing flexibility before later stages of the recovery process. PRICE presents with the greatest inconsistencies in pathophysiological reasoning. 3) Protection is essential and rest, for reasons of unclear underlying physiological mechanisms, appears useful when applied for no longer than four to six days. The use of ice, also due to unknown physiological mechanisms, seems effective when applied immediately after injury, but similar to compression and elevation yields unfavourable results when used to decrease inflammation. To avoid rerupture, caution is to be applied throughout the entire recovery phase, due to high intensities, eccentric exercise, and possibly altered pain sensation resulting from cooling and stretching.

Discussion Most insight into pathophysiological events and physiological effects of all studied treatment concepts is derived from either animal studies or studies done on healthy humans, therewith highlighting a considerable lack of existing evidence.

Conclusion Several recommendations towards a more adequate conceptual rehabilitative framework were made based on pathophysiological rationales. The key to optimizing recovery is to match rehabilitation to pathophysiological events. For this, a model for determining and progressing intensity has been proposed for strengthening and stretching, however, with several strong limitations. All of the theoretical constructs of the studied treatment concepts can only to a rather superficial degree be motivated on a pathophysiology basis, with more detailed recommendations towards parameters such as duration, number of repetitions or frequency shown to be beyond the current understanding of the pathophysiology of partial BFLH tears commonly occurring in running.

Keywords: Hamstring strain, muscle tear, pathophysiology, rehabilitation, recovery
1. BACKGROUND

Muscular injuries are very frequent and often occur during sporting activities with more than 90% related to excessive strain (commonly through non-contact injury mechanisms), or contusion (mostly through contact injury) (Järvinen et al. 2005). Especially in football, track and field, and other running sports muscle strains are rather common: recent epidemiological evidence shows that these represented more than 30% of all injuries with a relative incidence of 1.8–2.2 per 1,000 hours of exposure. (Bennel et al. 1996, Junge et al. 2004, Volpi et al. 2004, Waldén et al. 2005, Junge et al. 2006, Servicios Médicos del Futbol Club Barcelona 2009).

Anderson et al. (2001) reports that muscle strains often affect muscles that are biarticular, have primarily fast-twitch type-2 muscle fibres, and undergo eccentric contraction at the moment of injury. In this regard it is not surprising that the hamstring muscle with its unique biomechanical properties in running is a frequent site of injury (Stanton et al. 1989, Garrett 1990, Kujala et al. 1997, Woods et al. 2004, Brockett et al. 2004, Chumanov et al. 2010) with a seasonal incidence in football players and track and field athletes of 12–15% of all injuries (Bennel et al. 1996, Woods et al. 2004, Engebretsen et al. 2010) representing 35% of all muscular injuries (Servicios Médicos del Futbol Club Barcelona 2009). Of the 3 muscles making up the hamstring complex, the biceps femoris is most susceptible to be involved (Thelen et al. 2005a, Carlson 2008) with 53% and 86% of hamstring strains occurring to this muscle (Woods et al. 2004, Servicios Médicos del Futbol Club Barcelona 2009).

Rehabilitation programmes are designed to concomitantly a) optimize recovery and b) prevent recurrence, as previous injury precipitates recurrence at almost twice the risk (Engebretsen et al. 2010). The former is particularly relevant to athletes of all levels where an early return to sports and pre-injury level is of significant financial and strategic importance. Most research has been conducted into the prevention of hamstring strains by identifying and attempting to eliminate risk factors associated with recurrence, whereas less research exists for interventions aimed at optimizing recovery. Consequently, most evidence based rehabilitation protocols are mainly based on the prevention and reduction of risk factors, with wide variations existing concerning the optimization of recovery.

Neither for prevention, nor for optimizing recovery unfortunately has evidence been well established. Two recent Cochrane reviews have analysed the efficacy of interventions for both rehabilitation components, with results being inconclusive (Mason et al. 2007, Goldman et al. 2010) this mostly being due to poor methodological quality and a lack of randomized controlled studies available. Therefore, to date most recommendations for rehabilitation are based on expert opinion with comparably more inconsistencies towards the spectrum of enhancing recovery. (Petersen et al. 2005, Copland et al. 2009, Servicios Médicos del Futbol Club Barcelona 2009, Heiderscheit et al. 2010).

Many interventions designed to optimize recovery are widely employed by therapists and commonly encountered in treatment protocols (Servicios Médicos del Futbol Club Barcelona 2009, Heiderscheit et al. 2010). These include PRICE (Protection, Rest, Ice, Compression, Elevation) (Kerr et al. 1998 (ACPSM guidelines), Malliaropoulos et al. 2004, Sherry et al. 2004), strengthening exercises (Sherry et al. 2004), and different forms of stretching (Malliaropoulos et al. 2004, Sherry et al. 2004), all of which have been studied in two RCT’s (Malliaropoulos et al. 2004, Sherry et al. 2004) and included in one systematic review (Mason et al. 2007). Despite high occurrence rates and all recent scientific interest, evidence of their efficacy is not well established. Given this background, these three interventions (PRICE, strengthening, stretching) will serve as the main focus of this study, as the authors and two clinical experts chose them to be most relevant.

For creating an adequate rehabilitation program of biceps femoris strains aiming at optimizing recovery, a comprehensive understanding of the involved pathophysiology becomes vital and in fact fundamental (Lieber 2009). Yet, with interest we observed that not all proposed concepts of rehabilitation seem to abide by this rather basic rule (Lowering 2008).

To date, two published reviews (Järvinen et al. 2005, 2008) studied the physiology and different treatment modalities of muscle injuries in general, including contusion injuries and total ruptures. Whereas the pathophysiology was reviewed extensively, the physiotherapeutic treatment was covered rather scarcely and recommendations were mostly made in regards to the management by physicians.

Given the clear lack of evidence for the efficacy of rehabilitation approaches, the aim of this study is to “go back to the basics” and comprehensively review the pathophysiology of acute biceps femoris long head (BFLH) partial tears from running. This will then serve as a basis to examine the rehabilitative concepts of (1) PRICE (Protection, Rest, Ice, Compression, Elevation), (2) strengthening and (3) stretching, used to optimize recovery. An illustrative flowchart will be synthesized as a result. The argumentation of this study consists of (1) the highest available evidence and (2) the subsequent application of this evidence into therapeutic context.

We are not aware of any previous works that has been carried out to examine current concepts of rehabilitation for biceps femoris partial tears by means of reviewing the pathophysiological basis. This review can thus be regarded as a pilot project, seeking to improve the qualitative standard of the management in recovery.

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2. METHODS

To review the pathophysiology of acute biceps femoris partial tears, a comprehensive literature search was carried out within the Medline (PubMed) database combining the keywords muscle OR hamstring with injury OR strain OR laceration OR tear AND pathophysiology OR pathology OR physiology OR regeneration OR repair OR healing OR biology. This search resulted in 131,966 studies being published between Dec 1st 2004 and Dec 5th 2010. To further specify the search a comprehensive list of NOT keywords was added (see appendix I) for a complete listing of keywords, resulting in 7,118 hits. 323 studies were selected based on title relevance, of which 78 studies were selected based on abstract relevance. Further content and quality criteria were applied, excluding studies with irrelevant content, illogical reasoning and expert opinion not referencing experimental, exploratory or observational studies. 37 studies were selected and 4 additional studies were recommended through contact with internationally respected experts within the field. By searching their references 15 additional studies were selected. In total, 59 studies were included in this review consisting of 37 reviews and 22 experimental, exploratory or observational studies (publication date after Dec 1st 2004, n=45).

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3. RESULTS

PATHOPHYSIOLOGY OF BFLH PARTIAL TEARS

The following review consists of six sections: (1) clinical classification, (2) anatomy, (3) locations, (4) aetiology, (5) mechanism of strain, (6) recovery process.

3.1 Clinical classification

Jackson et al. (1973) was the first to classify muscle strains into 3 grades based on clinical impairment with mild (Grade I) strains representing a tear of only a few muscle fibres with minor discomfort accompanied with no or minimal loss of strength and range of motion (ROM). Moderate (Grade II) strains, in turn, affect a greater number of muscle fibres with clear loss of function, whereas a Grade III strains involves a complete rupture of the muscle, representing the most severe form of muscle strain. Whereas surgical management for total ruptures has been well established, the management for partial tears continues to have a lack of evidence base. The following review will therefore solely concentrate on partial tears great enough to entail an inflammatory reaction, comprising both Grade I and II muscle strains.

3.2 Anatomy

Skeletal muscle consists of two main components, (1) myofibres (MF) and (2) connective tissue that binds MF together, providing the constructive network for force transmission (Doessing et al. 2010). A myotendinous junction (MTJ) is defined as the portion of a tendon, termed aponeurosis (Epstein et al. 2006, Azizi et al. 2009), into which muscle fibres insert. It is important to note that these MTJs, which span a relatively large distance are to be distinguished from the actual MTJs at cellular level, measuring only a few microns (Garrett et al. 2000) (For a review
of muscular anatomy see Woodley et al. 2005, Battermann et al. 2010, Linklater et al. 2010).

3.3 Locations
Muscle strains most commonly occur close to the MTJ, a fact that also applies to the hamstring muscle with 90% of strains occurring at this anatomical location (Slavotinek et al. 2002, Woodley et al. 2004, Asking et al. 2007, Koulouris et al. 2007, Hancocka et al. 2009), which has been described as the weakest point of the muscle-tendon unit (Bencardino et al. 2005). Furthermore, the proximal MTJ of the biceps femoris long head (BFLH) has been reported to be mostly affected. Three mechano-morphological reasons explain this to be related to the fact that (1) the proximal aponeurosis is much narrower than the distal one (Rehorn et al. 2010), (2) that MF muscle have a bipennate appearance, of which the pennation angle is lateral and distal (Potier et al. 2009) and (3) that MF stretches are non-uniform with the greatest amount of stretch occurring at this location during eccentric contractions (Rehorn et al. 2010).

3.4 Aetiology
Why most strains in running occur to the BFLH still remains unclear (Woods et al. 2004, Servicios Médicos del Futbol Club Barcelona 2009); however several factors that may all contribute to a higher incidence have been proposed in literature.

The BFLH reaches its peak force and elongation velocity (Thelen et al. 2005b, Yu et al. 2008, Chumanov et al. 2010) at the end of the swing phase and acts eccentrically as a decelerator to the extending knee and flexing hip (Bencardino et al. 2005), a phase it is most susceptible to rupture. Guilhem et al. (2010) reports in his review that eccentric activity, when compared with concentric, shows lower EMG activity which was suggested to be due to a reduced number of activated motor units (MUs), leading to a distribution of mechanical stress on fewer MUs and subsequently to a higher risk of rupture for the recruited MUs (Guilhem et al. 2010). At the end of the swing phase in running the BFLH also reaches a higher peak stretch (9,5% beyond own length) (Thelen 2005a, Thelen et al. 2006, Yu et al. 2008, Schache et al. 2009, Chumanov et al. 2010) than its synergists, semimembranosus (7,4%) and semitendinosus (8,1%), therewith highlighting the effect of different knee moment arms (Koh 2008).

The peak length combined with peak force and elongation velocity is a crucial factor contributing to BFLH’s injury (Foreman et al. 2006), decreasing its capacity to generate force to a minimum (force/length and force/velocity relationship) (Hill 1938).

Another consideration that has been postulated to play a role in the BFLH’s susceptibility to injury is that the muscle is mainly composed of type II fibres (Bencardino et al. 2005, Koh 2008); however no physiological reasoning was provided.

3.5 Mechanisms of strain
With partial muscle tears, mechanical force usually extends across the complete cross-section of the affected MF and consequently disrupts the integrity of sarcomeres, sarcolemma, membrane, cytoskeletal, z-line along with blood vessels, intramuscular nerves and the extracellular matrix (ECM) (Prisk et al. 2003, Toumi et al. 2003, Järvinen et al. 2005, Koh 2008).

When ruptured, the affected MFs retract and a haematoma forms in the remaining gap between the fibre stumps (Ciciliot et al. 2010). The extravasated blood increases intramuscular pressure, which compresses and eventually stops the bleeding of wounded blood vessels (intramuscular haematoma); a process that is supported by vasocostriction, platelet aggregation, degranulation, and coagulation to form a blood clot, collectively called hemostasis (Järvinen et al. 2005). In more severe cases the epimysium of the injured muscle may also rupture, leading to an intermuscular haematoma (Järvinen et al. 2008).

Initially after injury there is an influx of extracellular calcium, resulting from membrane, sarcolemma and sarcoplasmic reticulum rupture (Lovering 2008). This loss of calcium homeostasis leads to a cascade of calcium-activated neutral proteases processes that result in even more focal necrosis of the MF (Sayers et al. 2008, Koh 2008, Ambrosio et al 2008) and is also responsible for an impaired E-C coupling process, accounting for the majority of loss of force production (Koh 2008).

3.6 Recovery process
Three sequential but overlapping recovery phases have been identified (Huard et al. 2002, Järvinen et al. 2005, Greffe et al. 2007): (1) destruction, (2) repair and (3) remodelling.

1) Destruction phase
With the development of the haematoma, the intramuscular pressure rises significantly, leading to (1) secondary hypoxia (aggravated by an increased oxygen cell demand) (Bleakley et al. 2004, 2007, Block 2010), (2) a vicious circle of reduced blood circulation and increased swelling (Knobloch et al. 2011), and (3) pain caused by sensitization and compression of nerves; with the former two creating a ischaemic environment leading to secondary muscle damage (Merrick 2002).

Within the first hour after injury, there is a significant influx of leukocytes, a process commonly referred to as extravasation or diapedesis, which is additionally enhanced by satellite cells (SC) and necrotic tissue that release various chemoattractants (i.e. growth factors, cytokines, chemokines, etc.) (Warren et al. 2004, Järvinen et al. 2005). With the ensuing inflammation, inflammatory cells (mainly neutrophils and macrophages, which are a class of leukocytes) not only phagocyte injured tissue, but also adjacent healthy tissue. Peak neutrophil levels occur within

Two types of macrophages exist, with M1 macrophages as the phagocytosing, destructive and therefore pro-inflammatory ones, and M2 macrophages having a reparative and hence anti-inflammatory role; the former chronologically preceding the latter, whose concentration level in turn stays elevated longer (Chazaud et al. 2009, Tidball 2010, Lu et al. 2011). It has been hypothesized that M1 macrophages also play a role in repair, however to date there is insufficient evidence available to substantiate this (Lu et al. 2011).

While these destructive and reparative functions of macrophages have been well documented in literature, recent findings unfold the same dual function for neutrophils. The negative role of neutrophils includes the phagocytosis of nearby located healthy tissue (Tidball 2010) and the release of oxygen free radicals and toxic enzymes (lysosomal proteases + elastases), which causes secondary necrosis (Bleakley 2009, Toumi et al. 2003). Their reparative function, on the other hand, comprises the removal of tissue debris, activation of satellite cells (see MF regeneration), as well as initiating the reparative process of M2 macrophages (Toumi et al. 2006, Butterfield et al. 2006).

Recent studies indicate that the inflammatory and neutrophilic response to muscle strain is a highly regulated and graded process, which may act to limit inflammation-related secondary damage while still optimizing repair (Brunelli et al. 2008, Paulsen et al. 2009, Tidball et al. 2010).

Another mechanism to avoid unnecessary damage of healthy tissue is the formation of a cross-sectional contraction band within the injured MF, which serves as a ‘fire door’ that hinders the spreading of necrosis along the length of the MF (Ambrosio et al. 2008). This occurs at approximately twelve hours after injury and is eventually accompanied by a new sarcolemma sealing the ruptured MF (Järvinen et al. 2008).

2) Repair phase

The repair phase is characterized by two simultaneous supportive and competitive processes, (1) phagocytosis, regeneration of MFs, blood vessels and nerves, and (2) scar formation (Järvinen et al. 2005).

Phagocytosis:
The repair phase begins with the previously outlined phagocytosis of necrotized tissue. Owing to their dual role in inflammation, macrophages and neutrophils are thus involved in both the destruction as well as the repair phase of recovery. Macrophages eventually secrete anti-inflammatory cytokines that contribute to end the inflammation and after this release factors that stimulate myogenic proliferation, growth and differentiation (Cantini et al. 2002, Sonnet et al. 2006, Tidball et al. 2008, Chazaud et al. 2009).

Scar formation:
The blood clot in the gap of the injured muscle stumps is being disposed by inflammatory cells and becomes replaced by early granulation tissue, composed of cross-links of fibrin and fibronectin, which provide a framework for fibroblasts to synthesize ECM proteins, resulting in the formation of a connective tissue scar. This has been reported to start as early as 48 hours post-injury (Järvinen et al. 2005), however does not occur without immobilization, and provides the early strength to withstand early contraction-induced forces (Ciciliot et al. 2010). With time, adhesive properties and density of the scar enhance progressively, starting with the formation of superfibronectin, which provides more tensile strength and early elasticity (Järvinen et al. 2005).

MF regeneration:
Although mostly regarded as irreversibly postmitotic, MFs have a remarkable capacity to regenerate, owing to a pool of quiescent undifferentiated muscle stem cells, named the satellite cells (SC). A vast majority of these are stored between the basal lamina and sarcolemma of each individual MF (see Fig 1) and become activated at approximately 24 hours (Järvinen et al. 2005, Ten Broek et al. 2010, Ciciliot et al. 2010). They undergo rapid proliferation at two days post-injury, migrate to the site of trauma and differentiate into myoblasts at three to five days post-injury (Ciciliot et al. 2010). Myoblasts then join with each other to form multinucleated myotubes (at approximately five days post-injury), and lastly either fuse to injured MFs or fuse with each other to generate entirely new MFs (Järvinen et al. 2008, Shortreed et al. 2008). Through this myogenic process the necrotized parts of the ruptured MFs inside the remaining old basal lamina are being replaced by the regenerating MFs at approximately five to six days post-injury (Järvinen et al. 2008). The maintenance of the basal lamina is thus a crucial factor for successful muscle regeneration (Ciciliot et al. 2010).

The activated SC do not just give rise to new MFs, but are also able to self-renew and replenish their quiescent pool for future myogenic needs (for a review see Collins et al. (2005), Kang et al. (2010), Ten Broek et al. (2010)). Furthermore, a great diversity of stimulatory and inhibitory growth factors (such as IGF-1 and TGF-β1) regulates SC activity (Ten Broek et al. 2010). Interestingly, the amount of SC has been reported to be higher in oxidative type I fibres than in fast glycolytic type II fibres (Shortreed et al. 2008); a fact pointing towards a slower recovery in terms of BFLH partial tears. Besides SC, a range of other multipotent muscle- and non-muscle-derived stem cells are involved in skeletal muscle regeneration (Järvinen et al. 2005, Järvinen et al. 2008, Ten Broek et al. 2010).
At approximately five to six days post-injury, the regenerating MFs begin to penetrate partly into the scar (Järvinen et al. 2008) and start to adhere to the scar at seven days post-injury, forming new mini MTJs identical in composition to the proximal or distal MTJs (Järvinen et al. 2005). It is important to note that the MFs never pierce through the scar entirely and fuse into one fibre (Kääriäinen 2001, Prisk et al. 2003, Ciciliot et al. 2010). While the developing scar has not yet gained sufficient strength, a remarkable mechanism exists to enable contractions without retracting of the MF and widening of the gap. The MFs start to form adhesions with the ECM along the sarcolemma surrounding each MF after as short as three days post-injury (Kääriäinen 2001, Järvinen et al. 2005, Ciciliot et al. 2010). When researching this mechanism, Kääriäinen (2001) interestingly discovered in her dissertation that this would occur to a lesser degree in the absence of mechanical stress. As the scar and new mini MTJ increase in tensile strength, the responsible adhesion molecules move along the sarcolemma towards the scar between 7-14 days post-injury, thereby strengthening the new mini MTJs. The lateral adhesions are thus only temporary, but serve to prevent rerupture and enable earlier contractions in the short term (Kääriäinen 2001).

Vascular regeneration:
Adequate vascular supply is self-evidently essential to MF regeneration. Given the fact that there is capillary damage, it seems convenient in this regard that young myotubes have less mitochondria and thus an increased anaerobic metabolism (Järvinen et al. 2005). During the later stages of regeneration, however, aerobic metabolism becomes the main energy pathway, placing the maturing MFs at risk for hypoxia unless adequate capillary ingrowth has been established to cover the demand of oxygen (Järvinen et al. 2005). Regrowing capillaries were observed to sprout towards the non-vascularised injured tissue at five days post-injury (Järvinen et al. 2005).

Neural regeneration:
Sufficient innervation marks another vital criterion for adequate MF regeneration. Fortunately, also here, early myogenesis does not stagnate without innervation. If the period of denervation extends three days, marked atrophy will set in (Ciciliot et al. 2010). However, the distance that neural axonal regrowth has to cover is relatively small, as axons commonly rupture within or in close proximity to MFs (Järvinen et al. 2005).

3) Remodelling phase
This phase is the period of maturation of all involved structures. The scar further enhances in tensile strength by progressing from superfibronectin to type III collagen followed by type I collagen production (Järvinen et al. 2005). After this point is thus mainly composed of type I collagen fibres, identical in composition to that of normal tendon tissue. Compared to the initially large granulation tissue (formed from the haematoma), the resulting scar is reduced in size, but despite prevailing beliefs does not resolve completely, but rather condenses into a thin layer of connective tissue, permanently separating the regenerating MF stumps (Ciciliot et al. 2010). The previous tendon-muscle-tendon continuum is thus changed into a structure of tendon-muscle-mini tendon-muscle-tendon (Äärimaa et al. 2004). As an end result, contractile muscle tissue is replaced by non-contractile tendonous tissue; a fact possibly accounting for some minimal loss of force after recovery. Similar to normal tendon, also this newly formed mini tendon functions as force transmission (Järvinen et al. 2008).

The dense scar tissue not only hinders the MF stumps from rejoining but may also prevent some new nerve axons from reaching denervated MFs (Kääriäinen 2001), which may subsequently undergo atrophy following denervation (Prisk et al. 2003, Ciciliot et al. 2010). Conversely, axons have also been reported to be able to pierce through the fibrotic scar tissue (Järvinen et al. 2008). Furthermore, it has been suggested that proliferation of fibrotic tissue can sometimes be excessive and can possibly restrict MF regrowth (Li et al. 2002, Järvinen et al. 2008).

In the remodeling phase, also regenerating MFs will further mature into a contractile apparatus and strengthen their attachment to the scar (which retracts and pulls the muscle stumps closer together), allowing greater contractile forces (Ciciliot et al. 2010).

Kääriäinen et al. (1998) studied the tensile strength of regenerating MFs using an experimentally induced MF rupture of rat muscle. At different time intervals, force required to rupture the muscle through stretch was meas-
Fig 2. The regeneration of muscle strain:

(A) Ruptured MF and BL, hemostatic formation of haematoma

(B) Contraction band and membrane seals MF stumps. Inflammation is initiated and activated SC begin to proliferate.

(C) SCs differentiate into myoblasts. Haematoma is replaced by scar tissue by proliferating fibroblasts.

(D) Myoblasts fuse into myotubes. Scar consist of superfibronectin

(E) Myotubes fuse with surviving parts of the ruptured MF and start to form new MTJs. Scar progression to type III collagen followed by type I collagen.

(F) Fully matured and regenerated MF separated by organized scar tissue and MTJs attached to it.

Adapted from Järvinen et al. 2008

ured. The authors conclude that the tensile strength progresses with time, reaching almost the equivalent strength (96%) of healthy control muscles after 56 days post-injury. Interestingly, the scar was shown to be the location where rupture occurred until 10-13 days, therewith representing the weakest link of the entire muscle at that point. After 14 days post-injury, the rupture consistently occurred in muscle tissue adjacent to the new mini MTJs, indicating a new weakest location.

It is noteworthy, that also with MF regeneration abnormalities exist. Occasionally, regenerating MFs within the same basal lamina may not fuse, leading to the formation of MF with a variety of patterns (e.g. forked fibres or clusters of smaller fibres), that limit muscular function (Järvinen et al. 2008, Ciciliot et al. 2010).

4. REHABILITATIVE CONCEPTS

4.1 PRICE

Protection:
Protective behaviour and so-called muscular guarding is generally a naturally occurring physiological process to prevent worsening of the injury and manifests itself by increased tone, pain and voluntary functional adaptation (Domzal et al. 1980, Domzal 1999, Scholz et al. 2002, Shrier 2005).

Rest:
A reasonable rationale for the use of immediate rest can be found in the fact that rest can avoid further contraction induced increase of the gap between muscle stumps, which if disregarded would result in a greater haematoma and consequently larger scar. Also an enhanced vascularization of traumatized tissue has been reported (Järvinen et al. 2008).

However, also several adverse effects of rest can be identified: Atrophy of uninjured MFs starts as early as four hours of rest after injury (Kasper et al. 2002, Ambrosio et al. 2008). If the length of rest is prolonged, rat muscle studies show that, regenerating MFs will remain atrophic and their orientation increasingly disordered (Ciciliot et al. 2010). Along with decreased activity levels also comes a decrease in MF volume, oxidative capacity, capillary density, increased intermuscular adipose tissue and proliferation of connective tissue (Gomes et al. 2004, Clark et al. 2009). Lastly, the increased proliferation of connective tissue and reduced number of serial sarcomere may both contribute to a marked loss of flexibility (Ambrosio et al. 2008). Within the first two weeks of disuse, strength decreases to a larger extent than MF volume. This is mainly due to a decrease in neuromuscular efficiency (Deschenes et al. 2002, Clark et al. 2009).

As the injured MF starts to establish lateral adhesions with the ECM at three days post-injury and findings indicate mechanical stress to be required for this (Kääriäinen 2001), it would seem logical to start mobilization of the recovering muscle already at this point of time.

Berg et al. (1996) reports that 10 days of unloading decreased torque by 13%, submaximum EMG by 25%, and no change in maximum EMG activity. The authors additionally also note that after four days of resumed weight-bearing
all values recovered. This necessitates the consideration that immobilization may not be as detrimental as expected.

Similarly, other findings postulate that a substantial period of immobilization is pivotal for early formation of scar tissue (Järvinen et al. 2005), which has shown to be the weakest link in the first 10-13 days post-injury (Kääriäinen 2001).

Given both beneficial and adverse effects, the advised length of rest will depend on which of these outweigh. Kieb et al. (2010) conclude in their review that the key to an optimised therapy rests in the appropriate timing between immobilization and remobilization. Most evidence for rest is derived from animal studies on immobilization. Järvinen et al. (2008) have studied the effects of immobilization after muscle trauma on rats and come to the conclusion that four to six days of immobilization yields the optimal ratio for incorporating beneficial aspects whilst limiting adverse effects to a minimum. However, it has to be considered that this is only applicable for totally immobilized muscles. Rest, in turn, does not inherently imply total immobilization, and rather refers a kind of relative immobilization. Based on limited amount of experimental studies more detailed recommendation towards the optimal amount of rest cannot be made on a pathophysiological basis.

**Ice:**
The exact effect of cryotherapy on muscle strains has not been fully elucidated (Hubbard et al. 2004) and experimental studies are lacking (Thorsson 2001, Hubbard et al. 2004).

Despite the prevailing lack of evidence, icing is widely used in the stage of bleeding and inflammation. Its application is generally believed to minimize secondary damage to healthy tissue elicited by enzymatic and ischaemic mechanisms (see earlier section ‘Destruction phase’). The main rationale for the use of cryotherapy is to reduce the metabolic rate of cells, and thus their oxygen demand, in order to allow longer survival in the ischaemic environment typically associated with the injury site (Merrick 2002, Hubbard et al. 2004, Bleakley et al. 2007, Knobloch et al. 2011, Block et al. 2010). However, the activity of important enzymes, such as those necessary to form a blood clot (coagulation) is simultaneously down-regulated (Kobbe et al. 2009). And indeed several studies report a reduction in tissue temperature to negatively affect hemostasis (= process of terminating bleeding), with both reduced platelet function and enzyme activity as likely contributors to a longer (3 times longer) and impaire (lower end-quality) coagulation process (Rohrer et al. 1992, Valeri et al. 1995, Wolberg et al. 2004, Kobbe et al. 2009). A larger haematoma and subsequently larger scar would in theory be the result.

Conversely, ice was also reported to support hemostasis by inducing vasoconstriction and decreasing the microcirculation (Block et al. 2010), leading to a sequence of decreased hematoma (Deal et al. 2002), decreased swelling, decreased intra-muscular pressure, and therefore less secondary hypoxia (Knobloch et al. 2011). Per contra, a decreased blood circulation makes the environment also more ischaemic (Merrick 2002).

Two recent randomized controlled animal studies conclude that cryotherapy immediately after trauma yields beneficial outcomes. When started immediately after trauma (3 sessions of 30 min with 90 min interval), injury dimensions measured at 10 ½ hours post-injury were significantly reduced (Oliviera et al. 2006). Matheus et al. (2008) found that even a single session of 30 min resulted in significant improvement of mechanical properties (maximal elongation, failure load, stiffness) at six days after injury. Nonetheless, the extrapolation of these results to human models is limited.

Cryotherapy is also frequently used to moderate overall inflammation, originating from the idea that this would limit inflammation mediated muscle damage secondary to the destructive function macrophages and neutrophils (Schaser 2007). However, it is unclear to what degree attempts at minimizing this neutrophilic and macrophage mediated damage, may also interfere with the reparative function of macrophages and neutrophils (Butterfield et al. 2006, Tidball 2010) and, as shown in an experimental study on rats, not only lead to a delayed differentiation and proliferation of SC in the repair phase, but also result in impaired MF regeneration in the remodelling phase as well as thicker collagen deposition around the regenerating MFs (Takagi et al. 2010). Judging from these findings, it may seem better to avoid cryotherapy during the inflammation phase.

In addition, hypothermia was reported to have an analgesic effect on the injury (Hubbard et al. 2004) resulting from reduced swelling as well as sensory nerve conduction, both of which desensitize the injured area (Bleakley et al. 2007, Algaflly et al. 2007, Block et al. 2010). Unsurprisingly, also here another side to the coin exists; pain serves as a protective signal that when inhibited may cause unguarded contractions and worsening of the initial injury.

Correct adjusting of parameters, such as duration, tissue temperature, application pressure, etc., seems rather essential; unfortunately, to our knowledge, no direct evidence of these parameters is available in literature (Merrick et al. 2002).

In conclusion, several positive and negative effects can be identified for the use of cryotherapy during the early stages of recovery. Most evidence, despite being mostly derived from studies with rats, indicates that the use of cryotherapy seems beneficial when applied immediately after injury, as opposed to seemingly adverse results when being applied during the inflammation phase.
Compression/Elevation:  
The use of compression originates from the concept that a compressed soft tissue injury may suppress blood invasion, and thereby reduce the size of the hematoma and secondary damage (Block et al. 2010). It may also translocate the haematoma away from the injury site towards proximal non-compressed tissues where it is more efficiently resolved by the lymphatic system (Block et al. 2010). Similarly, elevation is mostly recommended because it reduces blood flow, hydrostatic pressure and subsequently accumulation of interstitial fluid, and facilitates return to the heart (Järvinen et al. 2008).

As stated before, a reduced blood flow may, on the other hand, also be detrimental to healing by (1) impairing oxygen delivery, thus creating a more ischaemic environment resulting in more secondary damage (Merrick 2002) and (2) compromising the amount of neutrophil and macrophage infiltration and hence their instrumental reparative function (Tidball 2010).

Also for compression and elevation, very limited evidence has been brought forward. Thorsson et al. (1997) report that maximal compression was able to reduce blood flow by 50%, but did not enhance recovery of athletes. Similarly, Oliviera et al. (2006) concludes that compression when compared with no treatment does not yield any significant benefits.

Interestingly, the addition of compression to ice was reported to produce significantly intramuscular cooler temperatures than ice alone (Merrick et al. 1993). Therefore compression appears to increase the effectiveness of ice to reduce metabolic needs of injured tissue. However, to our knowledge, no experimental studies have been carried out to test this hypothetically added benefit compared to ice alone.

Conclusion PRICE:  
While protection is essential and rest has proven useful when applied for no longer than four to six days, ice may be beneficial when applied immediately after injury, but similar to compression and elevation yield unfavourable results when used to decrease inflammation. There is continued confusion as to whether inflammation is excessive and detrimental to recovery, or, whether it is in fact fundamental for optimal repair (Bleakley 2009).

Further high qualitative human studies are needed to deepen the insight into exact physiological effects of the components of PRICE at early stages of recovery.

4.2 Strengthening  
Strengthening has traditionally been a main component of rehabilitation programmes of hamstring strains. To our surprise, also here well designed trials verifying its efficacy are lacking.

Several beneficial short term aspects can be identified, including enhanced vascular and neural ingrowth (Kjaer et al. 2006), neural recruitment, SC activation, prevention of early atrophy, and increased secretion of IGF-1, an insulin-like growth factor which has the capacity to stimulate regeneration through both promoting SC proliferation and MF fusion (Ambrosio et al. 2008). At the same time, mechanical loading has shown to decrease myostatin (a TGF-β superfamily member), which was reported as a negative regulator of skeletal muscle regeneration by decreasing inflammation, causing atrophy, and block activation and proliferation of SC (Ambrosio et al. 2008). As previously mentioned the formation of lateral adhesions along the sarcolemma of the ruptured MF is enhanced by mechanical stress (Kääriäinen 2001). Loading would thus be highly favourable to prevent further increase of the gap, rupture and provide early strength. However, the optimal amount of mechanical stress needed to achieve this effect, is unclear.

Also in the long term strengthening has shown to yield positive effects. These include a better MF alignment and orientation (Ciciliot et al. 2010), and the fact that strengthening may clearly compensate for the loss of strength, which is a sequelae of relative immobilization, loss of contractile tissue, denervation by ruptured nerve axons, wrong orientation, and impaired EC-process.

Besides these short and long term effects, several studies have also reported certain connective tissue adaptations. Mechanical loading of muscle will stimulate secretion of collagen-inducing growth factors (Kjaer et al. 2009, Doessing et al. 2010) with significant changes apparent already after 3 days (Heinemeier et al. 2007). These growth factors induce ECM protein production resulting in a stronger matrix (Sarasa-Renedo et al. 2005, Doessing et al. 2010) and include (1) IGF-1, (2) connective tissue growth factor (CTGF) and (3) transforming growth factor-β-1 (TGF-β-1). Levels of the latter TGF-β-1 are already increased following muscle injury, and by means of loading will be even more increased. TGF-β-1 induces muscle derived stem cells to differentiate into myofibroblasts (Li et al. 2002, Arsic et al. 2008, Mackey et al. 2008, Cencetti et al. 2010), which are known to be primarily responsible for producing ECM proteins (Mackey et al. 2008).

Recent thought-provoking findings suggest that the ensuing type 1 collagen is capable of preventing differentiation of myogenic cells, which become fibrogenic and produce more collagen (Cencetti et al. 2010). In the short term this mechanically induced collagen synthesis could theoretically provide earlier strength for the ruptured MFs, prevent rerupture and further increase of the gap between stumps, and (in an animal experiment) has shown to provide an earlier matrix network for ingrowth of capillaries and nerve axons (Kjaer et al. 2006). Nonetheless, in the long term, this increased collagen synthesis could possibly also lead to excessive fibrosis (Li et al. 2002), delayed recovery and contractile muscle tissue...
that is replaced by non-contractile connective tissue (Nozaki et al. 2008). Indirect evidence for this has been de-

rived from experimental injection of anti-fibrotic agents and inactivation of TGF-ß-1 showing improved healing in

t rat muscle (Li et al. 2002). In addition, the basement mem-

brane (consisting of Type IV collagen) surrounding MFs appears to down regulate SC activity (Mackey et al. 2008).

The exact time point to start with strengthening exercises links to the previously elaborated recommendation for rest to be limited to maximally four to six days (see section on ‘Rest’). Thus, strengthening is contra-indicated before this time, as adverse effects would outweigh positive ones (Järvinen et al. 2005). It goes without saying that mobilization should be started gradually and one should always be cautious about applying high intensities to avoid rerupture which was reported to cause the longest lost time from sport activity (Brooks et al. 2006). Unfortunately, information on appropriate intensity of strengthening exercises, which achieve maximum effect while not risking rerupture and delaying recovery, is very limited. To date, one study has measured tensile strength of rat muscle at different time intervals after injury (Kääriäinen et al. 1998) by noting the force needed to rupture the muscle through stretch. The results, as displayed in Fig 3, show that the tensile strength progresses with time in a near-logarithmic fashion, with almost the equivalent value (85%) of healthy control muscles being reached within 3 weeks post-injury, and levelling off thereafter.

Evidently, strengthening exercises should always be carried out within pain limits. However, also this common recommendation has certain limitations, as pain as a determinant of exercise intensity is highly subjective, not evidence-based, and inaccurate when cryotherapy was applied (Arendt-Nielsen et al. 2002). Moreover, it is not known at what pain level tissue damage occurs. Other optimal parameters, such as duration, frequency and repetitions remain unclear and cannot be laid out with current pathophysiological understanding.

In terms of different contractions types, differences in physiological features of the different muscular contrac-
tions remain unknown (Middleton et al. 2004) and to our knowledge, no evidence exists for their efficacy at different time points of the recovery process. Nevertheless, the choice of contraction type can be viewed as another variable for adjusting the intensity. Therefore, a possibility to design a strengthening programme can be to start with isometric contractions, the least intense amongst the contraction types, before progressing to concentric and lastly eccentric exercises being the most provocative form of contraction for partial tears.

Over the last decade, eccentric training has played an increasingly important role in rehabilitation of hamstring strains. The reason for this is mostly preventative, as eccentric exercise was shown to be able significantly increase the mean optimum peak torque angle (Kilgallon et al. 2007, Aquino et al. 2010), which was identified as a possible risk factor for injury recurrence (Brughelli et al. 2010). Whether a reduced mean optimum peak torque angle reflects the muscle’s pre-injury state or is consequential to injury yet remains to be established (Brockett et al. 2004).

A rather popular eccentric exercise in the rehabilitation of hamstring strains is Nordic hamstring exercises (see Fig 4). This represents an exercise of maximal intensity, which based on the physiological healing process we can only recommend to use in final stages of recovery, as the regenerating MFs have gained sufficient strength to withstand such intense contraction forces.

Besides inducing greater strength gains than concentric or isometric (Guilhem et al. 2010), eccentric training of the BFLH has several physiological adaptations. These include hypertrophy, a longer fascicle length (through a process of sarcomerogenesis) and an increased neuromuscular activation (Butterfield et al. 2006, Roig Pull et al. 2007, Potier et al. 2009). Compared with concentric training,

These values provide information about (1) the progression of exercise intensity, and (2) maximally tolerated intensity in terms of load and this can be made measurable with force measurement (e.g. dynamometer) in relation to the uninjured side, which serves as a 100% mark measured by maximally achieved force. When calculating training intensity, it is important to bear strength differences between dominant and non-dominant side in mind. The dominant leg was reported to be on average 8.6% weaker in the knee flexors, as compared with the non-dominant leg (Lanshammara et al. 2010).

Despite of being the best available evidence on intensity, it has to be noted that the practical use of this data has several strong limitations. These include the fact that (1) rat and not human models were used in the experiment, (2) injury mechanisms were different (laceration, not partial tear), (3) stretching load may present with different properties than strengthening load, (4) tensile strength was measured in a non-contracting state, (5) studied muscle did not include the hamstring muscle, and (6) rats did not undergo any form of treatment, but were allowed to move freely.
eccentric training has shown to enhance the expression collagen-inducing growth factors more (Heinemeier et al. 2007), which is probably due to greater force production, or greater local shear stress. Whereas these adaptations would also likely occur in recovering BFLH partial tears, they are derived from studies on healthy subjects; a fact that limits its extrapolation.

While eccentric exercise seems to have many promising effects, it has to be applied with caution. The risk of rupture is higher than with any other contraction type (see previously explained ‘mechanism of injury’).

Conclusion Strengthening:
The evidence level for the efficacy of strengthening is limited, mostly for being based on animal studies or healthy subjects. Literature indicates that mobilization involves an increased collagen production with both positive and negative potential effects on healing. Whether one of the two outweighs the others remains unclear. Strengthening has shown to have several enhancing short and long term properties based on pathophysiological rationales. It is not recommended to start remobilization before four to six days, after which strengthening exercises should be gradually increased. A model for determining and progressing intensity has been proposed, however, with several strong limitations. Vastly based on pathophysiological understanding, caution is to be applied when using high intensities and eccentric exercise.

4.3 Stretching

Also stretching has conventionally been used in rehabilitation of hamstring strains. In general, this is motivated by an attempt to compensate for the marked loss of flexibility that is clinically observed with hamstring strains. This loss of flexibility, as previously mentioned, may be sequelae of immobilization and protection; i.e. increased proliferation of connective tissue and reduced number of serial sarcomeres (Ambrosio et al. 2008).

Whereas stretching was reported to be able to reduce the loss of sarcomeres in series (Gajdosik 2003, Soares et al. 2007), it appears not capable of altering visco-elasticity of structures even over a series six weeks intense daily stretching (Weppler et al. 2010). Instead, very recent evidence indicates that ROM gains are to a vast majority a product of an increased tolerance to stretch (Folpp et al. 2006, Aquino et al. 2010, Ben et al. 2010) with a modified sensation accounting for this increased tolerance (Guissard et al. 2006, Weppler et al. 2010). Flexibility has been defined as the total achievable excursion, within the limits of pain, of a body part through its range of motion (Saal 1998). The increased stretch tolerance is partly due to an analgesic effect, which results in a dampened pain-perception (Shrier 2005, Weppler et al. 2010). Pain, on the contrary, serves a protective function and when inhibited, may place the regenerating MFs at considerable risk of rerupture (Carter et al. 2000).

Other rationales for the use of stretching in aiding recovery include a strongly evidenced associated rise in SC activation by releasing HGF (hepatocyte growth factor) (Tatsumi 2010), a reduction in immobilization-mediated atrophy (Gomes et al. 2004), and a better MF alignment/orientation in later phases of recovery.

Järvinen et al. (2008) promote stretching in early phases of recovery, with the main purpose to distend the scar in a phase that it is still plastic, subsequently increasing its tensile strength. Their statement, however, is not supported with any references.

Another potential effect of stretching may be enhanced lateral adhesion of the MF, as mechanical stress was reported to be supportive of that process (Kääriäinen 2001). While this was only observed with strengthening exercises, it may after all not apply to stretching per se, albeit also constituting mechanical stress.

Most of this evidence is derived from experimental studies on either animals or healthy humans. A systematic on the effects of hamstring stretches on healthy subjects (Decoster et al. 2005) found most studies to be of poor methodological quality, thereby confirming the prevailing lack of evidence. Nonetheless, the authors conclude that stretch was beneficial with a gained ROM of 5 – 33 degrees.

Also in terms of different types of stretching, clear evidence is scarce. Nonetheless, several points favour an active stretching type compared to static or passive ones. Meroni et al. (2010) reports that active stretching in the healthy person may produce greater ROM gains that are maintained longer, and need lower compliance to produce effects. Aquino et al. (2010) furthermore conclude in their RCT that static stretching is not able to induce a shift in mean optimum torque angle, indicating that it would not induce a build up of serial sarcomeres. What effects the different stretching types have other than on flexibility has, to our knowledge, not been studied yet.

The time point at which to start stretching is theoretically already possible as soon as the scar forms (2 days post-injury). At this point the MF is nevertheless still highly vulnerable and only a very low intensity would not rerupture the MF. Harris et al. (2006) showed that if the immobilized limb is placed in a neutral or outer range position,
less atrophy and more maintained flexibility is the result. This could represent a slight stretch that can be carried out already in the rest phase after three days of injury, by avoiding a shortened position (as long as within pain-limits).

To determine the progression and intensity of the stretch from day 5 to 56, the same model as for strengthening can be applied (Kääriäinen 2001) with the same limitations (see section on strengthening). The maximally tolerated intensity in terms of load and can also here be made measurable by means of force measurement (e.g. dynamometer) in relation to the uninjured side, serving as a 100% mark measured by force needed to reach maximum pain tolerance during stretch.

Analogous to strengthening, stretching exercises should always be carried out within pain limits. This is very important, as any stretch crossing pain limits will result in increased tolerance to stretch, lowering pain perception and thereby increasing rerupture risk (Carter et al. 2000, Shrier 2005, Weppler et al. 2010).

Any further recommendations towards optimal parameters, such as duration, frequency and repetitions of stretch remain unclear and cannot be specified with current pathophysiological understanding.

Conclusion stretching:
Stretching has a potentially instrumental effect on regeneration and is capable of counteracting flexibility loss through increasing stretch tolerance and serial sarcomerogenesis. The increased stretch tolerance, on the contrary, could simultaneously desensitize the muscle to some extend to detect damaging loads, presenting with great inconveniences and a higher risk of rerupture. Therefore, stretching should not be used to modulate flexibility before later stages of the remodeling phase, but rather with the aim to enhance MF regeneration, scar properties, lateral adhesions, and to reduce atrophy. Optimal parameters to achieve this are not known and yet remain to be explored. In terms of stretching intensity and progression, the same model as explained for strengthening can be applied, however, also here with several strong limitations.

5. DISCUSSION

Pathophysiology review:
The 59 included studies used to review the pathophysiology of BFLH partial tears were able to form a complete picture of current pathophysiological understanding.

It is important to consider that a majority of published studies concerning the pathophysiology of muscle tears comes from animal research; a fact mostly explained by ethical limitations, highlighting the limited extrapolations of our pathophysiology review. To what extent this limits the results is unknown (Pound et al. 2004, Matthews 2008, Wall et al. 2008).

This previous point also explains the fact that little pathophysiological information was hamstring strain specific, but rather mostly based on general muscle injury models. However, several authors state that the pathophysiological events occurring after trauma due not differ substantially from contusion to strain, or laceration, and are applicable to all skeletal muscle (Järvinen et al. 2005, 2008, Koh et al. 2008, Ciciliot et al. 2010, Tidball et al. 2010). Many of the events described and their time course naturally take place without intervention. In reality, however, most injuries do undergo some form of treatment, which would certainly alter their entire recovery phase. To date, unfortunately, no information on this is available though.

To deepen pathophysiological understanding, further studies exploring the recovery process of BFLH partial tears should address (1) detailed time points of recovery stages, (2) time-dependent injury locations during rerupture, (3) the development of tensile properties and loadability of force and stress at different time points (4) the role of inflammation, (5) presence of excessive inflammation, (6) M1 macrophages reparative role, and (7) long term mechanical changes.

Rehabilitative concepts:
In regards to the proposed method to determine intensity and intensity progression for strengthening and stretching exercises, Kääriäinen (2001) provides useful information. Even though this has many limitations, it represents the best available evidence to date.

Despite attempts to make specific recommendations towards BFLH partial tears, very few studies were based on this type of injury, if not healthy subjects or animals; a fact making the extrapolation certainly difficult.

Due to time constraints, no systematic literature search was carried out to find physiological effects for each concept. Therefore, more updated physiological effects may have been missed in this work.

Valuable information in regards to the rehabilitative concepts that is to be addressed in future studies, include (1) underlying physiological adaptations of cryotherapy, compression, elevation, and different amounts of relative immobilization (2) physiological effects of the combination of PRICE, (3) the role and amount of strengthening contributing to lateral adhesion formation, and excessive fibrosis, (4) the different types of strengthening and stretching, and (5) effects of using different parameters of PRICE, strengthening and stretching.
6. CONCLUSION

In conclusion all of the theoretical constructs of the studied treatment concepts can only to a rather superficial degree be motivated on a pathophysiology basis, with more detailed recommendations towards parameters such as duration, number of repetitions or frequency shown to be beyond the current understanding of the pathophysiology of partial BFLH tears commonly occurring in running. Most insight into the occurring pathophysiological events as well as the physiological effects of the studied treatment concepts is derived from either animal studies or studies done on healthy humans, thereby highlighting the alarming lack of existing evidence.

Of the studied concepts, strengthening appears to have several enhancing short term and long term effects, however, should only be started after a four to six days period of relative immobilization. To what extend connective tissue adaptations from strengthening serve an instrumental or detrimental role yet remains to be established.

Pathophysiologically rationales explain that stretching should not be used for increasing flexibility before later stages of the recovery process. Stretching yields potential regeneration enhancing effects and can already be started at light intensities after two to three days by avoiding keeping the limb in shortened positions.

For both strengthening and stretching, a model for determining and progressing intensity has been proposed, however, with several strong limitations.

The concept of PRICE, albeit meeting most international consent, presents with the greatest inconsistencies in pathophysiologically reasoning. Protection is essential and rest, for reasons of incompletely understood underlying physiological mechanisms, appears useful when applied for no longer than four to six days.

The use of ice, again due to unclear physiological mechanisms, seems effective when applied immediately after injury, but similar to compression and elevation yields unfavourable results when used to decrease inflammation.

For an increased risk of rerupture, caution is to be applied throughout the entire recovery phase, due to high intensities, eccentric exercise, and a possibly altered pain sensation resulting from cooling and stretching.

A deeper pathophysiologically insight as well as more evidence-based rehabilitative constructs is needed to enable specific recommendations towards changes that have to be made for a more adequate conceptual rehabilitative framework to enhancing recovery of BFLH partial tears.

7. REFERENCES


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8. Appendices

Appendix I — List of keywords used for literature search

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Appendix II – FLOWCHART: Recovery Process of partial muscle tear + Treatment recommendations