Hamstring muscle strain injuries remain one of the most challenging issues facing sports medicine. By the mid-18th century, the importance of exercise for health was established, and the physical demands of different forms of exercise were recognised, in a manner not dissimilar to descriptions 200 years later. By the start of the 20th century, the Olympics had been re-invented, sport and exercise were recognised as a means of developing character and maintaining health in a world experiencing an explosion in leisure time.

Given the limitations of the training methodology of the time, it is possible that muscle strains were a common finding, but there is limited epidemiological literature available from the time.

*In football we find injuries occur in a poorly conditioned, a tired or dazed man, or in a team that is demoralised and receiving a severe beating. These individuals are slowing up, not coordinating, and are apt to be injured.*

Already in 1902, muscle injuries were recognised as occurring in sedentary individuals and those athletes lacking adequate preparation and warm-up. While these assertions appear to have stood the test of time, with modern, professional, well-trained athletes routinely have hamstring injuries, it is now recognised that this is a dramatic oversimplification of the aetiological processes. Muscle strains were recognised as occurring at either the musculo-tendinous or within the intramuscular components, and there has been a gradual refinement of this understanding over the past 100 years. In 1966, Bass distinguished between intramuscular (poor prognosis) and intermuscular (good prognosis) injuries. Forty years on, this theme has been readdressed, with further evidence that it is the specific location and pathoanatomical nature of the muscle injury that will determine prognosis, rather than simply symptom severity and injury size.

Also articulated in 1902 was the belief that "only a cursory examination is necessary" to diagnose a hamstring muscle strain and despite evidence to the contrary, this remains a common misconception. Clinical evaluation has consistently shown to have a high false positive rate when compared with MRI, even in those injuries clinically diagnosed as grade II muscle strain. This clinical appearance of muscle strain injury, in the absence of confirmed local muscle pathology, remains a paradox today.

In 1906, management of muscle tears was typically conservative involving "holding the limb under a cold water tap as long as you can bear it, and as often as is possible." Plaster immobilisation "in the direction of the fibres of the muscle" (which remained popular until the 1950s), complete rest for 3 to 6 days, followed by "active work," and the use of "embrocations" (massage creams). Of note, even at this early stage in the understanding of the hamstring muscle injury, it was recognised that tearing of musculo-tendinous insertions from the bone requires a longer period of rehabilitation and that surgery may occasionally be indicated for complete ruptures. Over recent decades, the indications for surgical intervention have been further elucidated; surgical repair may provide superior outcomes for a completely ruptured proximal hamstring tendon.

In contrast, intramuscular injuries are typically treated conservatively and rupture of the distal hamstring tendons, in particular the semi-membranosus and semitendinosus appear to be able to be satisfactorily managed conservatively. By the 1930s, there was an increased interest in, and understanding of the histopathology of acute muscle injuries. Remarkable experiments compared physical, chemical, infective and pharmacological injury on rabbit skeletal muscle. These led to clear descriptions of the histological manifestations of acute muscle injury, initially described as "acute molecular degeneration." Additional studies involving confuscd rabbit muscles further characterised the histological transitions associated with muscle injury into haemorrhage, degeneration, white cell infiltration and proliferation. There was infiltration of leucocytes and lymphocytes within 24 h, as well as the progressive damage after the initial insult. While not recognised at the time, these two processes appear related, as it has recently been illustrated that minimising the leucocyte activity at the site of acute muscle injury may limit muscle damage, and enhanced muscle regeneration.

This principle of reducing the acute inflammatory...
phase of muscle injury is the basis for the use of simple anti-inflammatory tools such as ice, and the more controversial pharmaceutical anti-inflammatories. 35 Fishaback 31 also identified that “muscle proliferation is started by the muscle nuclei set free by the breaking up of severely traumatised fibres”, likely reflecting the mobilisation of satellite cells, critical for the repair process and considered an index of muscle regeneration. 56 This is a remarkable insight into the process of muscle regeneration, identified but not fully understood for over half a century.

The year 1936 saw the clear articulation of the principle of approximating the torn ends of the muscle injury to enhance the healing process, and also the notion that premature disruption of this approximation may increase scarring. 37 Intramuscular scarring is now considered to be a poor prognostic indicator. 38

“It is true that coach and trainer still attempt to exert their influence by calling men yellow at times who claim to have been injured, or try to bring undue influence on the athletic physician to permit an excellent player to return to competition before he should. 39

Despite being recognised at the time as both lacking consistent efficacy 42 and potentially having a negative impact on muscle regeneration, 43 corticosteroid injections for acute muscle injury were popular through the 1950s and 1960s. 20–42 44 In vitro studies have confirmed the negative long-term consequences of cortisone on muscle injury repair, 45 and as such, despite case series appearing to support its clinical efficacy, this technique remains controversial. 36–47

“treatment must be designed to minimise the haemorrhage and inflammatory reaction so that there will be as little granulation (scar) tissue formed as possible...”26

The imperative of controlling haemorrhage and inflammatory reaction to minimise scar tissue remains unchanged 60 years on. The theme of scar tissue minimisation has been resurrected in recent years, with the identification of numerous medications that may potentially assist in this task. 48–53 While their clinical utility remains to be delineated, 57 years on, this remains an exciting area of research.

In contrast to these underlying principles which have generally withstood the test of time, Delarue 26 also recommended the use of combined intralesional hyaluronidase/local anaesthetic injections, in the management of acute muscle injuries. This modality appears well recognised at the time 26 54 but by the 1970s had lost popularity. 55 Hyaluronidase is an enzyme involved in the breakdown and inactivation of hyaluronic acid and was believed to be of benefit when used in the initial phase of muscle injury. 54 Intriguingly, the in vivo function of hyaluronic acid has recently been elucidated, with its synthesis up-regulated in areas of acute soft tissue injury and functioning to promote and modulate inflammation, as well as potentially minimising scar tissue development. 56 Thus, at least theoretically, minimising any hyaluronic acid excesses in the early phase of an acute soft tissue injury (thereby reducing inflammation) may be of some benefit, although paradoxically hyaluronic acid itself has been advocated for use in soft tissue injuries, with apparently good results. 57–59

While core stability is now recognised as a critical component of hamstring rehabilitation and athletic performance, 60–62 the concept was first recognised as early as 1958. Writing after the VI British Empire and Commonwealth Games held in Cardiff the same year, Lloyd (1958) 52 recognised that hamstrings strains were one of the most common injuries observed, but also that the risk for all sports injuries may be minimised by an ‘alerted posture’.

“Alerted posture’ means alerting the prime fixer muscles so that quick movements can be carried out by activators and synergists acting on a frame already made firm by the prime fixers.”42

By the late 1960s, the use of ‘enzymatic preparations’ including trypsin, chymotrypsin, streptokinase and streptodornase had replaced hyaluronidase injections. 44 55 The use of oral enzyme preparations in the management of muscle injuries remains popular with some practitioners, 63 64 despite limited scientific evidence of any benefit. 8 16 Other medications such as Hirudoid (a heparinoid) 65 and muscle relaxants 20 were also popular at the time.

Non-steroidal anti-inflammatory drugs (NSAIDs) were first mentioned in the routine management of muscle strain injuries in the late 1960s 55 66 and their use has persisted in sports medicine. 57 While they may have a role in limited situations (eg, reducing the incidence of myositis ossificans following muscle contusion injuries), increasing in vitro evidence suggests that NSAIDs may impede regeneration and increase

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**Table 1** Management of acute muscle injuries: 1954

<table>
<thead>
<tr>
<th>Stage one: haemorrhage control</th>
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<tr>
<td>Rest/protection</td>
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<tr>
<td>Compression (24 h)</td>
</tr>
<tr>
<td>Water immersion or ice bags 30 to 60 min</td>
</tr>
<tr>
<td>Elevation</td>
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<tr>
<td>Re-evaluate at 24 h</td>
</tr>
</tbody>
</table>

| Stage two: absorption of blood and exudate (following haemorrhage control) |
| Local heat (whirlpool bath, shortwave diathermy) |
| Massage (avoid stimulation of further bleeding) |
| Avoiding damaged area in the first 48 h |
| Never vigorous or painful, as this will prolong injury |

Graduated exercises
Reduce swelling
Maintain tone and strength of the muscles
Maintenance of general fitness important

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fibrosis during muscle healing, and hence should be considered relatively contraindicated in muscle strain injuries. 35 68–70

The 1970s and 1980s saw increasing attention being paid to physiotherapeutic means of rehabilitation. There remained a focus on minimising bleeding and inflammation while maximising regeneration, by the increasingly novel use of cooling, electrotherapy (which was clinically already being used in the latter 19th century), 71 manipulative techniques 72 and specific strengthening programmes. 73 74 Sports medicine had started to identify the role that muscular trigger points may play in muscle-related pain (recognised in muscles since the 1930s) 75 perhaps contributing to the false positive (MRI negative) 24 25 muscle injuries. 76 The use of invasive injection techniques such as Traumeel and Actovegin in the management of acute muscle injuries has been promoted in Europe for at least three decades, 63 but their use remains controversial and not established in mainstream sports medicine literature 76 – predominantly due to a lack of scientific evidence. 9 Their clinical use has, however, persisted and despite a lack of substantial evidence, has in recent years expanded beyond Western Europe.

The last two decades have seen an explosion in literature regarding the hamstring muscle strain, with increased understanding of the epidemiology, aetiology and pathophysiology. 2–6 Subtle refinement of techniques for minimising inflammation and maximising regeneration has evolved, 77 78 but many of the underlying principles of treatment have remained unchanged from the mid-20th century. Novel approaches to prevention and treatment continue to appear, but often lack an evidence base. 79 Blood injections have been recognised as potentially therapeutic in sports medicine for over 60 years, 80 and refinement of this in the form of autologous plasma preparations (platelet-rich plasma) has recently been popularised for muscle strain injuries. Currently, the evidence to support this remains limited from the basic science and clinical perspective. 86 81–87 – in fact to date the published evidence is only slightly greater than that for the use of hyalurondase injections, and possibly slightly less than the evidence for the use of corticosteroid injections in acute muscle injuries.

CONCLUSION

Hamstring muscle strains have been recognised as a significant injury for over 100 years. Remarkably, by the mid-20th century, the foundations for our current management strategies were well established. Furthermore, despite the remarkable advances of the past 30 years, we have made only incremental progress in our understanding and management of this complex injury. 1 The history of hamstring injury management is characterised by interventions which over time have failed to become established, typically without evidence ever being produced either for, or against their application. Most novel treatment interventions currently used continue to lack scientific validity; if we are to avoid repeating past mistakes, we must take up the challenge of obtaining high-quality data. To achieve this, we must challenge dogma that underpins ‘usual care’ and subject all elements of our management strategies to scientific scrutiny.

There is now evidence to use MRI to distinguish MRI-positive and MRI-negative injuries as these have a distinct prognosis and treatments. However, whether MRI or US aids in predicting the prognosis beyond this distinction remains to be determined. Minimising acute inflammation and optimising regeneration remain key goals in hamstring injury management; current techniques having changed little in 40 years and the medical manipulation of these pathophysiological processes warrants further clinical and laboratory research (eg, role of PRP). Finally, the development of scar tissue appears related to recurrent injuries and poor outcomes. Recently identified techniques for inhibition of growth factors involved in the development of scar tissue present an exciting development, but require further high-quality clinical trials in order to establish themselves in the standard management of muscle injuries.

To progress, we must encourage communication among (I) the proponents of published evidence-based, clinical guidelines, (II) those practitioners overtly working outside the recognised scientific evidence base and applying novel therapies based on their personal experience and (III) those researchers able to test novel hypotheses. Typically, these groups have not interacted, likely to the detriment of the profession and patients. We are at the start of the specialist era in Sports and Exercise Medicine, and maintaining status quo is unacceptable. For the credibility of all clinicians in the field of sports injury management, it is vital to accumulate appropriate evidence to progress from the scientific baseline established by our forbears in the mid-20th century.

Acknowledgements The author would like to acknowledge the support of Aspetar, Qatar Orthopaedic and Sports Medicine Hospital, in the preparation of this document, in particular the ongoing support of Dr Hakim Chalabi.

Competing interests None.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

Corrections

Hamilton B. Hamstring muscle strain injuries: what can we learn from history? (Br J Sports Med 2012;46:900–3). The sentence ‘It is true that coach and trainer still attempt to exert their influence by calling men yellow at times who claim to have been injured, or try to bring undue influence on the athletic physician to permit an excellent player to return to competition before he should.18’ was a direct quote from reference 18, and should have been in italic and in quote marks.