Prevention and Rehabilitation of Hamstring Injuries

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Foreword by Michael Kjær

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To everyone who wants a more thorough understanding of hamstring injuries—beyond the usual "you'll be back in three weeks" approach.

Foreword

The present book is a monumental effort in order to cover all aspects of hamstring injuries taking it all the way from the basic understanding of its nature and pathogenesis, over the risk factors, diagnosis and treatment, and to the potential preventive measures in order to limit the incidence of this very common sports injury that limits both elite athletes and recreational sports-active individuals in their attempt to carry out their regular exercise.

The strength of the book is not only its comprehensive nature where more than 30 of the most front-line international experts in different aspects of the field have contributed, but also the depth of each chapter where up-to-date knowledge is presented (more than 1100 references in total in the book) and excellent figures and tables are provided and guide the reader through the different aspects of hamstring injuries.

What is worth mentioning is the fact that many previous books have had a very selective approach either towards basic biology or towards the clinical approach, and thus have not always provided a full well-balanced view of the field. This book covers both basic and clinical aspects in a very qualified way. This results in a truly translational textbook, where the different aspects of the injury and its handling are appreciated.

To bring the chapters together, the three editors have made sure that each of them are coauthors on most chapters so a natural "flow" between the different parts of the book is ensured. The approach that this book has to hamstring injuries should be a guideline not only for handling of hamstring injuries but also for other sports medicine approaches to regional injuries and it is the hope that both clinicians and basic researchers will find it attractive and thus stimulate interaction between the different disciplines needed to provide a comprehensive understanding of sports injuries. This does not imply that the book should be read from one end to the other. However, regardless of which research or clinical angle you come from, the book provides up-to-date knowledge in the field with which you are comfortable. It should also inspire you to read adjacent chapters and maybe, for you, "out-of-the-box" aspects

of hamstring injuries to obtain a full understanding of the complexity of the injury and its clinical handling in a scientifically sound way.

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Conflicts of Interest

Dr. David Opar is listed as a co-inventor on a patent filed for a field testing device of eccentric hamstring strength (PCT/AU2012/001041.2012), as well as being a minority shareholder in the company responsible for commercialising the device. Dr. Opar is also the Chair of the company's Research Committee.

Associate Professor Anthony Shield is listed as a co-inventor on a patent filed for a field testing device of eccentric hamstring strength (PCT/AU2012/001041.2012), as well as being a minority shareholder in the company responsible for commercialising the device.

Acknowledgements

All authors have been chosen due to their expert knowledge and important scientific contributions to the field of hamstring injury prevention and rehabilitation. All editors and authors in this book have waivered getting any fee for their contributions to this book. Instead of a fee, all editors and authors have received one copy each of the book for which they have collectively provided all the content.

About the Book

This innovative book presents the latest insights into hamstring injuries, a common problem in elite and recreational sport, with a unique focus on prevention and rehabilitation. The research within this area has evolved rapidly over the past 10 years and this text offers a comprehensive overview of the recent and most relevant advances. It fills a gap in the literature, since other books focus on muscle injuries in general and their surgical treatment. Structured around the current evidence in the field, this book includes sections on functional anatomy and biomechanics; basic muscle physiology in relation to injury and repair; assessment of risk factors; and factors associated with hamstring strains. It also discusses considerations in relation to examination and assessments of acute and long-standing injuries, hamstring injury prevention, including pre-season and in-season interventions, as well as management strategies and rehabilitation protocols. The final chapter is devoted to additional interventions when conservative rehabilitation and injury prevention fail. All 13 chapters build on each other in a logical order, but each chapter can also be read in isolation. Written by renowned experts in the field, this book will be of great interest to sports physiotherapists, sports physicians, physical trainers, coaches and athletes.

Contents

About the Editors

Kristian Thorborg is Professor at Copenhagen University. He is currently employed as a Senior Researcher at the Sports Orthopedic Research Center—Copenhagen (SORC-C) and the Physical Medicine and Rehabilitation Research—Copenhagen (PMR-C) programme. Both centres/programmes are part of the Copenhagen IOC Research Centre in Injury and Illness Prevention. Professor Thorborg has been a Specialist in Sports Physical Therapy since 2004, with more than 20 years of clinical experience within sports and orthopaedic injury prevention, assessment and treatment. Professor Thorborg has published more than 150 peer-reviewed articles and 30 book chapters and is ranked by expertscape.com (online guide to find biomedical experts and institutions) as the leader within the area of both "Hip" and "Groin".

David Opar is a Senior Lecturer in the Discipline of Exercise Science at Australian Catholic University (Melbourne Campus). Having completed a Bachelor degree (with Honours) in Human Movement at RMIT University in 2008, Dr. Opar commenced a PhD on hamstring injuries at Queensland University of Technology under the supervision of Associate Professor Anthony Shield in 2010. Dr. Opar was conferred his PhD in December 2013. Since then, Dr. Opar has published over 40 peer-reviewed articles, most of which are focused on hamstring injury. Dr. Opar's research covers many areas in hamstring injury, from risk factor analysis and intervention strategies through to optimising rehabilitation and return to play strategies. Dr. Opar was the co-inventor of the NordBord device, which is a tool utilised around the world to measure eccentric hamstring strength in the field.

Anthony Shield is an Associate Professor of Exercise Science at the Queensland University of Technology in Brisbane, Australia. He completed his PhD at Southern Cross University in 2003. Associate Professor Shield has written more than 50 peer-reviewed publications, of which more than 30 are on the topic of hamstrings. He and his group have introduced the concept that neuromuscular inhibition may be a contributing factor to hamstring injury recurrence and have investigated the role of eccentric strength and hamstring muscle architecture on injury risk.

Anatomy of the Hamstrings

1

Ryan Timmins, Stephanie Woodley, Anthony Shield, and David Opar

1.1 Introduction

The posterior muscles of the thigh, semimembranosus (SM), semitendinosus (ST), biceps femoris (BF) long head (BF_{LH}) and short head (BF_{SH}) are referred to as the "hamstrings" (Fig. 1.1). The long hamstring muscle group (SM, ST, BF_{LH}) crosses both the hip and knee joints, therefore having a role in hip extension, knee flexion and internal (SM and ST) or external knee rotation (BF), during concentric contraction.

The anatomy of the hamstrings is unique and suggested to be one of the reasons for the high incidence of injuries in this muscle group. The biarticular nature of the long hamstrings [2], the dual innervation of BF [3] and the shortness of its fascicles (a bundle of fibres) [4] are some factors which have been proposed as reasons why hamstring anatomy influences injury risk. In addition, the intramuscular tendon within the BF is an anatomical feature that is suggested to add an extra layer of complexity when considering rehabilitation approaches [5].

This chapter will outline the anatomy of the hamstrings including their proximal insertion sites, musculotendinous junctions (MTJs), muscle architecture, distal MTJs, insertions and neurovascular supply. Whilst describing the key structural features of the hamstrings, anatomical variations will also be highlighted.

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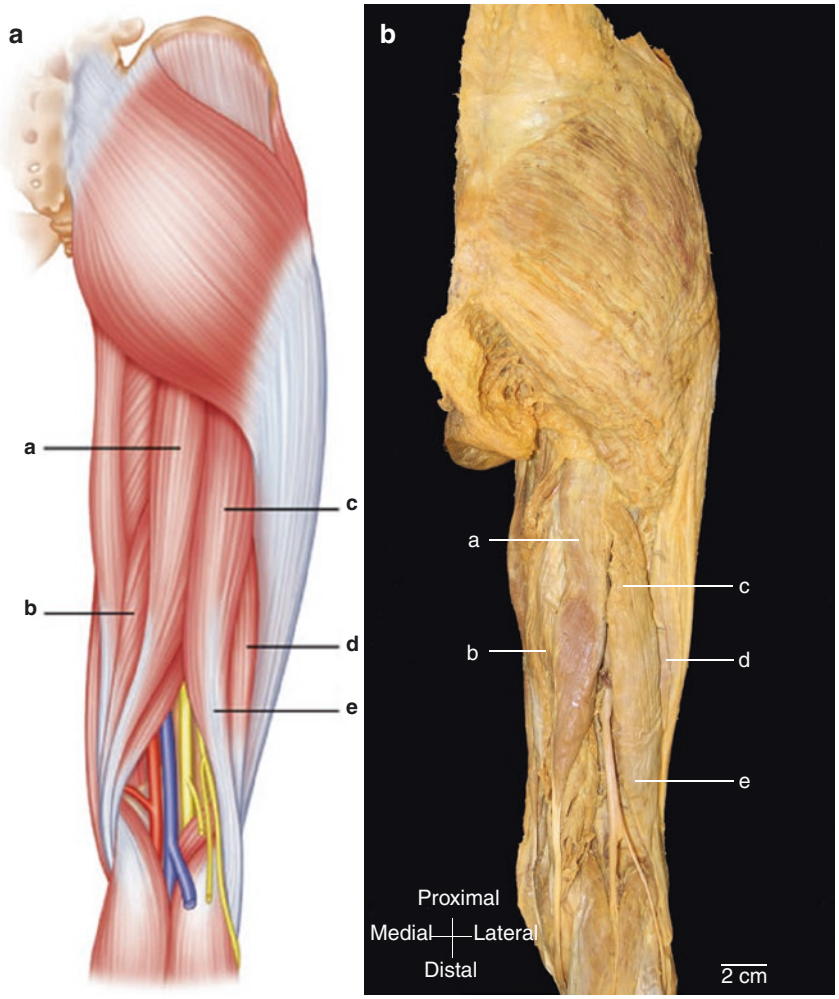

Fig. 1.1 Illustration (**a**) and dissection (**b**) of the right posterior thigh demonstrating the gross anatomy of the hamstring muscle group. The hamstrings consist of ST (**a**) and SM (**b**) on the medial side and the long head (**c**, **e**) and short head (**d**) of BF, laterally. (Figure **a** printed with permission from Kaeding and Borchers (2014) [1])

1.2 Proximal Insertions

1.2.1 Semimembranosus

The proximal insertion of SM is commonly described as the lateral facet or aspect of the ischial tuberosity [6–14], positioned lateral and anterior to the origin of the conjoined tendon of BF_{LH} and ST [10, 13] and posterior (superficial) to the origin of the quadratus femoris muscle [10, 11] (Figs. 1.2 and 1.3). It is generally accepted that the SM origin is separate to that of the conjoined tendon; however, there is some suggestion that the most proximal part of the SM tendon blends with the conjoined tendon

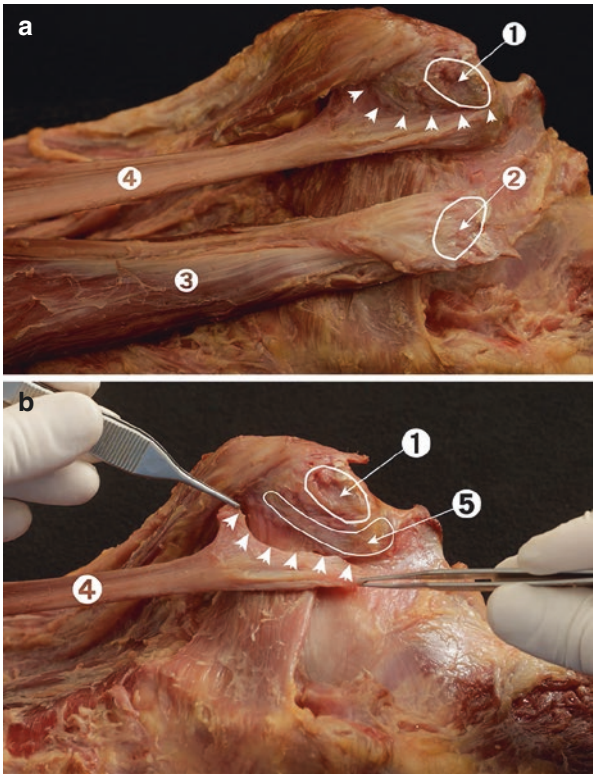

Fig. 1.2 (**a**, **b**) Dissection photograph, posterolateral view of the area of the proximal attachment of the right hamstring muscles. (1) Area of the attachment of the conjoined tendon of the ST and the BF_{LH}; (2) the proximal attachment area of the conjoined tendon; (3) conjoined tendon of the ST and the BF_{LH}, cut and rotated 180°; (4) proximal tendon of the SM muscle; (5) area of the attachment of the SM muscle; arrowheads, shape of the SM attachment. (Printed with permission from Stepien et al. [15])

of BF_{LH} and ST [13, 16, 17] or has connections with the BF_{LH} [6–8], separating approximately 3–5 cm from the ischial tuberosity [13, 18]. A common tendon comprised of all three muscles has also been observed as an anatomical variant [19].

In addition to its main proximal tendon, SM has an additional tendinous component that arises from the inferior surface of the ischium and is intimately associated with adductor magnus (AM) [8, 10, 11, 17]. This "accessory tendon" has a rectangular-shaped footprint with a mean area of 1.2 cm^2 (95% CI 1.0–1.3 cm^2) and forms an angle of approximately 105° with the main proximal tendon [10]. It is hypothesised that this tendinous structure acts to dissipate the force from the main SM tendon, providing a possible reason why SM is not injured as frequently as BF_{LH} and ST [10].

The footprint of SM is crescent shaped [9, 10] or "longitudinal oval" [19] (Fig. 1.2) with a mean surface area of 4.1 cm^2 [10]. With regard to linear footprint dimensions, nomenclature is variable, but the mean proximal-distal length ranges between 3.1 and 4.5 cm compared to anterior-posterior and medial-lateral dimensions of approximately 1 cm [9, 10, 13, 19] (Table 1.1).

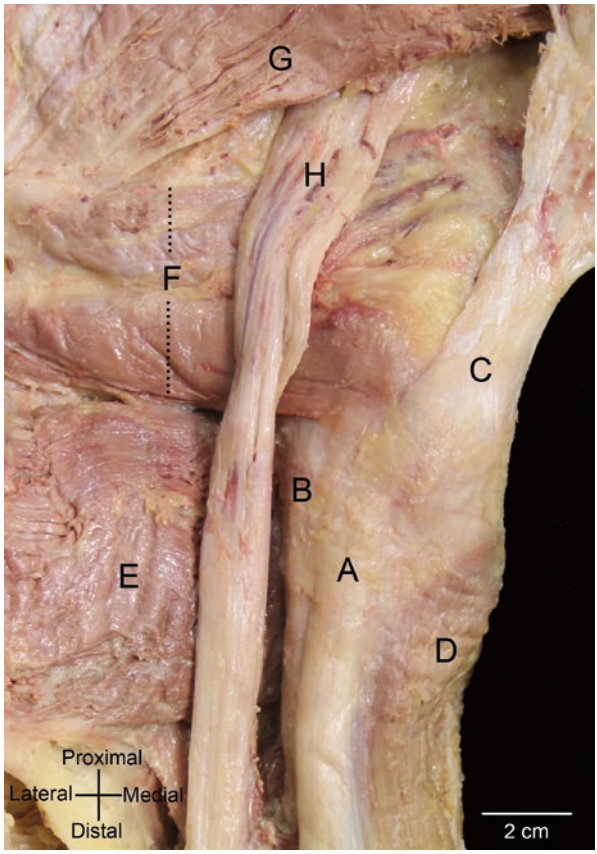

Fig. 1.3 Dissection photograph of the proximal hamstring insertions at the ischial tuberosity (left limb, posterior view). The conjoined tendon (A) arises from the posteromedial aspect of the ischial tuberosity, medial and posterior to the SM tendon (B), and has some connections with the sacrotuberous ligament (C). Muscle fascicles of ST (D) originate directly from the ischial tuberosity, the medial border of the conjoined tendon and an aponeurosis on the anterior aspect of the muscle (not visible). E, quadratus femoris; F, gemelli muscles and tendon of obturator internus; G, piriformis; H, sciatic nerve

Table 1.1 Footprint dimensions of the proximal SM and conjoined tendon of BF_{LH} and ST

Author(s), date	Footprint dimensions (cm) Mean ± SD[a]		
	Proximal to distal	Anterior to posterior	Medial to lateral
SM			
Feucht et al., 2015 [19]	4.5 ± 0.5 (length)	1.2 ± 0.3 (height)	
Miller et al., 2007 [9]	3.1 ± 0.3	1.1 ± 0.5	
Philippon et al., 2015 [10]	3.3 (95% CI 3.1–3.5)		1.5 (95% CI 1.3–1.7) (width)
Van der Made et al., 2015 [13]		1.1 ± 0.5	1.3 ± 0.3
Conjoined tendon of BF_{LH} and ST			
Feucht et al., 2015 [19]	3.9 ± 0.4 (length)	1.4 ± 0.5 (height)	
Miller et al., 2007 [9]	2.7 ± 0.5	1.1 ± 0.5	1.8 ± 0.2
Philippon et al., 2015 [10]	3.6 (95% CI 3.3–3.9)		2.1 (95% CI 1.7–2.4) (width)
Van der Made et al., 2015 [13]		1.8 ± 0.2	2.6 ± 0.4

CI confidence interval
[a]Unless stated otherwise

1.2.2 Semitendinosus and Biceps Femoris Long Head

The proximal tendons of the BF_{LH} and ST form a common "conjoined tendon" which originates from the medial facet or posteromedial aspect of the ischial tuberosity (Figs. 1.3, 1.4 and 1.5) [6, 11, 12, 14]. The thick, round tendon of BF_{LH} occupies the lateral part of the medial facet [6, 10, 14] and has some connections with the sacrotuberous ligament [8, 10, 11, 17, 20–22]. From a phylogenetic perspective, it is suggested that the sacrotuberous ligament represents the upper, degenerated remnant of the BF_{LH} tendon [8], yet the morphological relationship between these two structures is not well defined. In addition to its

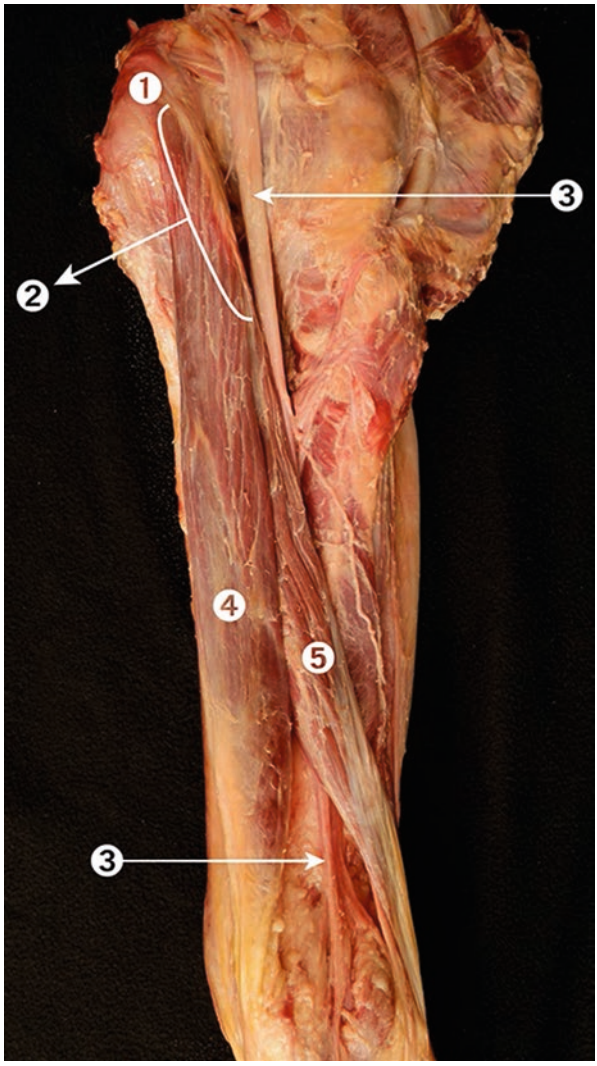

Fig. 1.4 Dissection photograph, posterolateral view of the posterior thigh of a right thigh. (1) Ischial tuberosity, (2) conjoined tendon of the ST and the BF_{LH}, (3) sciatic nerve, (4) ST muscle, (5) BF_{LH} muscle. (Printed with permission from Stepien et al. [15])

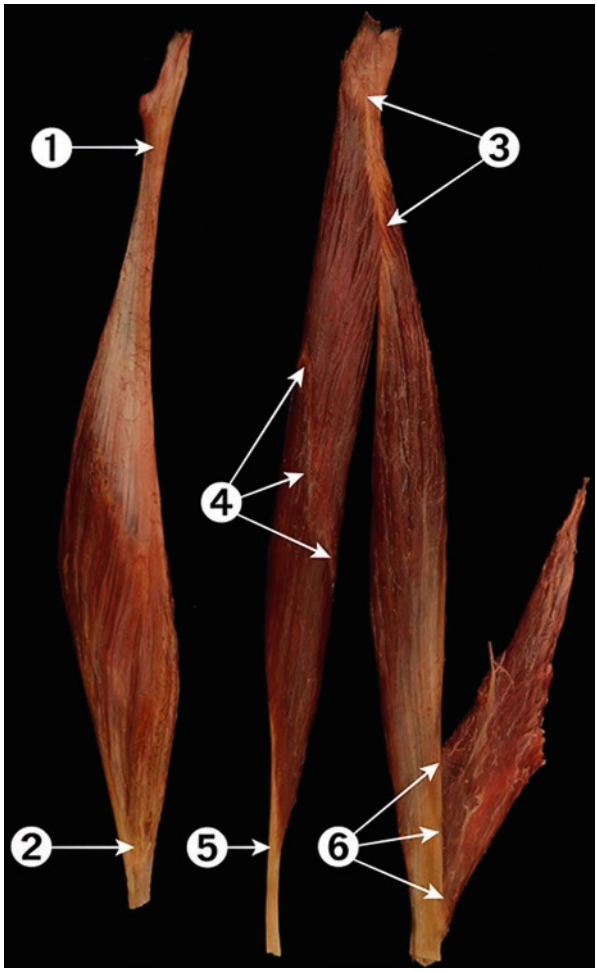

Fig. 1.5 The hamstring complex. (1) Proximal tendon of the SM muscle, (2) distal tendon of the SM muscle, (3) conjoined tendon of the ST and the BF_{LH}, (4) tendinous inscription (raphe) of the ST muscle, (5) distal tendon of the ST muscle, (6) common distal tendon of the long and short head of the BF muscle. (Printed with permission from Stepien et al. [15])

insertion into the ischial tuberosity, the lateral superficial fibres of the sacrotuberous ligament [21] appear to be confluent with the superficial fibres of the BF_{LH} tendon [11, 21] (Fig. 1.3), but not necessarily in all individuals [21, 22]. Functionally, these connections are thought to be critical when considering transfer of forces across the sacroiliac joint [21, 22], with the sacrotuberous ligament also potentially providing an additional soft tissue anchor for the conjoined tendon that may serve to limit tendon retraction following a hamstring rupture [20].

The origin of ST is positioned medial to that of BF_{LH} and is predominantly muscular [6, 10, 14], occupying a mean area of 2.0 cm^2 (95% CI 1.5–2.4 cm^2) on the ischial tuberosity [10] (Figs. 1.2, 1.3 and 1.4). Fascicles (a bundle of muscle fibres) of ST also originate from the medial border of the conjoined tendon (which gives rise to the largest proportion of fascicles) and from a short proximal aponeurosis on

the anterior aspect of the muscle, which appears to be a medial extension of the BF_{LH} tendon [6, 10, 11, 14, 23, 24].

The conjoined tendon accounts for 57.4% (95% CI 54.0–60.8) of the total proximal hamstring footprint [10]. It is oval in shape (Fig. 1.2) with a mean proximal-distal length of between 2.7 ± 0.5 and 3.9 ± 0.4 cm. Measures of its anterior-posterior and medial-lateral footprint dimensions are highly variable (Table 1.1) [9, 10, 13, 19].

A rectangular-shaped retinaculum-like structure, devoid of fibrocartilage (5.6 ± 0.45 cm long, 4.1 ± 0.16 cm wide and 925 ± 13 μm thick), covering the insertion of the sacrotuberous ligament and origins of the proximal hamstring tendons has been recently described [25]. Composed of transversely oriented fibres, this retinaculum is anchored directly to the medial and lateral aspects of the ischial tuberosity, with its deep fibres strongly adhered to the BF_{LH} epitenon, but separated from the epimysium of ST by loose connective tissue. An additional fascial expansion from the anterior epimysium of gluteus maximus (GM) attaches to the superior and superficial aspect of retinaculum. Based on its morphology, it is suggested that functionally this retinaculum anchors the BF_{LH} tendon, rather than enabling longitudinal sliding, and also potentially facilitates the transmission of forces between GM and BF_{LH} during muscle contraction.

1.2.3 Biceps Femoris Short Head

The BF_{SH} originates below the distal insertion site of GM, commencing approximately 15 cm distal to the ischial tuberosity [14] (Fig. 1.1). Fascicles arise from three distinct locations: (1) the length of the linea aspera [7, 14, 17], between AM and vastus lateralis [17]; (2) the upper two-thirds of the lateral supracondylar line [7, 14, 17] to within 5 cm of the lateral femoral condyle [17]; and (3) the lateral intermuscular septum [7, 14, 17], specifically the distal three-quarters of its posterior aspect [26]. Muscle fascicles inserting into these sites span a mean length of 15.7 cm (range 14.5–17.8 cm) [14].

1.3 Proximal Tendons and Musculotendinous Junctions

The tendons of the hamstring muscles can be considered as two distinct components: (1) the “free” tendon which is devoid of any inserting muscle fascicles and (2) the musculotendinous junction (MTJ), which is the portion of the tendon into which muscle fascicles insert (Fig. 1.6).

Most data on proximal hamstring tendon morphometry are derived from dissection-based research, and although there is some consistency between studies, it should be noted that these parameters are often highly variable between individuals. These differences in size and the amount of free or intramuscular tendon have been hypothesised to influence the susceptibility of a muscle to injury [11, 27, 28] (Table 1.2). Little data are available on the three-dimensional morphometry of the MTJs, including their intramuscular portions.

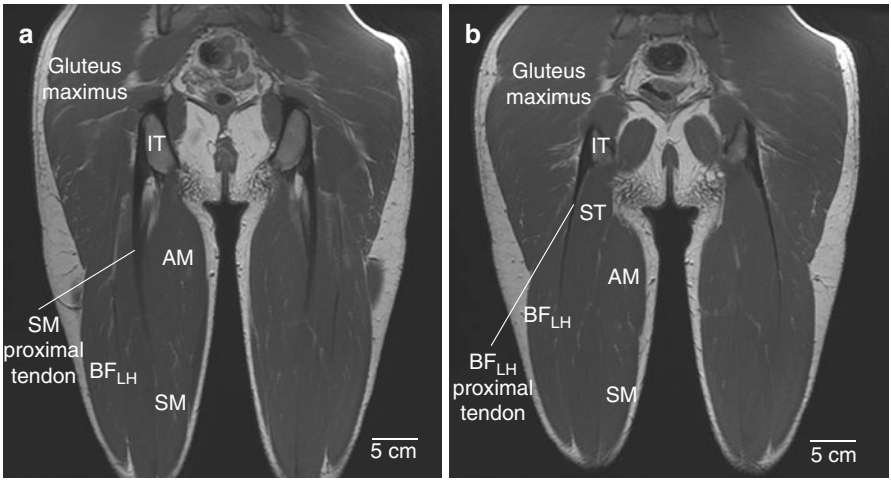

Fig. 1.6 Proton density, coronal magnetic resonance images from a young man demonstrating the long tendons and musculotendinous junctions of (**a**) semimembranosus (SM) and (**b**) biceps femoris long head (BF_{LH}). *AM* adductor magnus, *IT* ischial tuberosity, *ST* semitendinosus

1.3.1 Semimembranosus

From its origin, the tendon of SM passes medially, lying deep to the conjoined tendon of BF_{LH} and ST as it courses distally. Immediately distal to the ischial tuberosity, the tendon rotates approximately 90° [12, 13], to be oriented in the coronal plane [12]. It then widens becoming broad and aponeurotic (Fig. 1.5), with a rounded lateral border flattening into a thin membranous projection medially (resembling a "comma shape" in cross-section) [12, 14].

The proximal tendon of SM is the longest of all of the hamstring muscles, measuring approximately 32 cm and occupying about 75% of the total muscle length [12, 14, 18]. The lateral portion of the tendon extends furthest distally [14] to a point distal to the centre of the muscle belly [7]. The most proximal muscle fascicles of the SM arise from the medial border of the proximal tendon [12] about mid-thigh level [17], distinctly lower than BF_{LH} and ST. As such, the tendon has a substantial intramuscular tendinous component (Fig. 1.6a), with the proximal MTJ accounting for two-thirds of total tendon length (approximately 20 cm, or 48% of total muscle length) [12, 14]. Stretch-induced injury to the SM often involves the proximal free tendon [42, 43], and it could be that the length of this tendon (approximately 11 cm [12, 14]), together with its convoluted course into the muscle belly, predisposes to this type of injury.

1.3.2 Semitendinosus and Biceps Femoris Long Head

Immediately distal to the ischial tuberosity, the conjoined tendon is round or crescentic in shape [6, 8, 12, 14], with a cross-sectional area (CSA) smaller than that of

Table 1.2 Muscle, proximal tendon and musculotendinous junction lengths of the hamstring muscle

Segment	Author(s), date (number of specimens; M, F)	Muscle length cm, mean ± SD[a] (including proximal and distal tendon)				Muscle belly length cm, mean ± SD[a] [as a % of total muscle length]			
Muscle		BF_{LH}	BF_{SH}	ST	SM	BF_{LH}	BF_{SH}	ST	SM
	Wickiewicz et al., 1983 [37] (3; no details)					34.2		31.7	26.2
	Friederich and Brand, 1990 [39] (2; 1 M, 1F)					27.4		28.3	20.8
	Rab et al., 1997 [78] (35; 15 M, 15F)					28.6 ± 3.0		29.4 ± 3.2	
	Woodley and Mercer, 2005 [13] (6; 3 M, 3F)					43.8 (range 41.5–48.5) 28.1 (range 23.6–35.5)	29.1 (range 26.7–32.8) 25.8 (range 24.4–28.6)	43.8 (range 41.5–48.5) 31.6 (range 28.4–37.3)	43.8 (range 41.5–48.5) 26.4 (range 22.0–32.6)
	Makihara et al., 2006 [38] (6; 5 M, 1F)					31.2 ± 5.2		26.8 ± 3.6	28.5 ± 2.6
	Tate et al., 2006[b] (10; 6 M, 4F)					M: 27.0 ± 2.0 F: 19.0 ± 4.0	M: 25.0 ± 2.0 F: 21.0 ± 2.0	M: 34.0 ± 2.0 F: 28.0 ± 2.0	M: 27.0 ± 3.0 F: 26.0 ± 2.0
	Ward et al., 2006 (27 from 21 cadavers; 9 M, 12F)					34.7 ± 3.7[c]	22.4 ± 2.5[c]	29.7 ± 3.9[c]	29.3 ± 3.4[c]
	Kellis et al., 2012 [40] (8; 8 M)					38.9 ± 4.0 29.6 ± 2.5	28.5 ± 1.9 21.2 ± 2.0	47.0 ± 3.0 27.7 ± 1.8	40.4 ± 2.5 25.8 ± 2.3
	Evangelidis et al., 2015 [30] (30; no details)					29.3 ± 2.6			
	Freitas et al., 2018 (20; 10 M, 10F)					US, ll: 25.1 ± 2.2 M: 26.2 ± 2.3 F: 24.0 ± 1.5			
	Kellis et al., 2009 [44] Dissection and US (6; 3 M, 3F)	Dissection 39.2 ± 3.4 US 38.7 ± 4.4		Dissection: 39.0 ± 2.6 US: 38.1 ± 3.4					
	Kellis et al., 2010 [32] (8; 4 M, 4F)	39.8 ± 3.1		46.8 ± 2.0					
	Kumazaki et al., 2012 (13; 8 M, 5F)	39.2 ± 2.9	15.1 ± 1.7	38.2 ± 2.8	38.1 ± 2.8				
	Storey et al., 2015 (Dissection: 10; 10 M)	45.6 ± 2.9		45.8 ± 3.0	45.8 ± 3.0				
	Van der Made et al., 2015 [12] (56; no details)	42.0 ± 3.4		44.3 ± 3.9	38.7 ± 3.5				
	Vadgaonkar et al., 2018 (46; 46 M)			33.2 ± 3.6[b]					

(continued)

Table 1.2 (continued)

Segment	Author(s), date (number of specimens; M, F)	Length cm, mean ± SD[a] [as a % of total muscle length]			
Proximal tendon		BF_{LH}	BF_{SH}	ST	SM
	Garrett et al., 1989 [16] (5; no details)	[60%]		[31%]	[78%]
	Woodley and Mercer, 2005 [13] (6; 3 M, 3F)	27.1 (range 23.4–30.2) [62%]		12.9 (range 8.5–17.7) [30%]	31.9 (range 28.5–37.2) [73%]
	Kellis et al., 2010 [32] (8; 4 M, 4F)	24.0 ± 1.0 [61%]		12.5 ± 1.2 [27%]	
	Storey et al., 2015 Dissection (10; 10 M); MRI (20; 20 M)	Dissection: 25.7 ± 2.9 [56.5 ± 5.5%] MRI: 26.1 ± 2.6		Dissection: 15.0 ± 2.1 [32.7 ± 3.9%] MRI: 11.9 ± 3.8	Dissection: 33.6 ± 2.0 [73.3 ± 1.7%] MRI: 31.7 ± 1.6
	Van der Made et al., 2015 [12] (56; no details)	19.6 ± 4.1 [47%]		12.4 ± 3.6 [28%]	24.3 ± 3.9 [63%]
Proximal free tendon	Woodley and Mercer, 2005 [13] (6; 3 M, 3F)	6.3 [15%]		1.2 [3%]	11.1 [25%]
	Kellis et al., 2009 [44] Dissection (6; 3 M, 3F)	9.9 ± 2.7 [25.7 ± 7.0%]		11.3 ± 2.0 [30.3 ± 2.1%]	
	Kellis et al., 2010 [32] (8; 4 M, 4F)	4.9 ± 0.3 [12%]		1.2 ± 0.2 [3%]	
	Batterman et al., 2010 (101; 39 M, 62F)	5.7 ± 1.5		2.1 ± 0.4	
	Kellis et al., 2012[d] [40] (8; 8 M)	5.0 ± 0.4 [12.9 ± 2.1%]		1.4 ± 0.3 [3.0 ± 0.6%]	8.2 ± 1.4 [20.3 ± 3.2%]
	Storey et al., 2015 Dissection (10; 10 M); MRI (20; 20 M)	Dissection: 7.4 ± 1.1 MRI: 6.4 ± 1.6		Dissection: 2.2 ± 0.9 MRI: 1.1 ± 0.5	Dissection: 11.1 ± 1.6 MRI: 11.2 ± 1.7
	Van der Made et al., 2015 [12] (56; no details)	5.0 ± 3.4 [12%]		0.2 ± 0.7 [0.4%]	9.4 ± 2.6 [24%]

Proximal MTJ	Woodley and Mercer, 2005 [13] (6; 3 M, 3F)	20.6 (range 17.4–26.1) [47%]		11.7 (range 7.5–15.8)]27%]	20.8 (range 17.6–28.6) [47%]
	Kellis et al., 2010 [32] (8; 4 M, 4F)	19.1 ± 0.8 [48%]		11.26 ± 1.0 [24%]	
	Evangelidis et al., 2015 [30] (30; no details)	16.7 ± 2.8 (range 10.5–22) [43–75%]			
	Storey et al., 2015 Dissection (10; 10 M); MRI (20; 20 M)	Dissection: 18.4 ± 2.5 [40.3 ± 4.9%] MRI: 19.7 ± 2.6		Dissection: 12.8 ± 2.0 [27.9 ± 3.6%] MRI: 10.8 ± 3.6	Dissection: 22.5 ± 1.2 [49.4 ± 2.7%] MRI: 20.5 ± 2.2
	Van der Made et al., 2015 [12] (56; no details)	14.6 [35%]		12.2 [28%]	14.9 [39%]

All studies are dissection-based except for Tate et al. (2006) and Evangelidis et al. (2015) [30] which use MRI, Freitas et al. (2018) who use ultrasound; Kellis et al. (2009) [44] which incorporates dissection and ultrasound; and Storey et al. (2015) which incorporates both dissection and MRI

F female, *M* male, *MRI* magnetic resonance imaging, *MTJ* musculotendinous junction, *US* ultrasound, *BFlh* biceps femoris long head, *BFsh* biceps femoris short head, *ST* semitendinosus, *SM* semimembranosus

[a]Unless stated otherwise

[b]Data reported for dominant limb. Differences in data between dominant and nondominant limbs were reported for BFlh (females) and BFsh (males and females)

[c]Number of specimens differs from the total number examined. Data derived from 19 specimens for all hamstring muscles, except for BFlh (18 specimens)

[d]Not clear if these data represent free tendon or MTJ length

SM (0.47 cm^2 compared to 0.86 cm^2) [12]. As it passes distally some muscle fascicles of ST muscle arise from its medial, concave border, and further distally, BF_{LH} fibres originate from its lateral surface (Fig. 1.5) [8, 11, 14, 44]. The BF_{LH} and ST separate approximately 9–10 cm distal to their origin at the ischial tuberosity [9, 10, 19]. The tendon of BF_{LH} then becomes intramuscular [12] (Fig. 1.6b) forming a small, cordlike tendon with a flat aponeurotic expansion visible on the medial surface of the muscle [6, 7, 14]. The proximal tendon of BF_{LH} is expansive, being smaller than that of SM but larger than ST—it measures approximately 25 cm in length, occupying 60% of the muscle length. Its proximal free tendon is reasonably short (5–6 cm) with a long muscle-tendon component of about 20 cm (extending approximately 45% of the total muscle length). The structure of the proximal BF_{LH}, with the majority of it being composed of tendon, has been proposed to contribute to the greater amount of strain in surrounding muscle during sprinting and as such a purported increase in risk of hamstring injury [28]. Furthermore, disparity in the area of the proximal aponeurosis of BF_{LH} (mean 7.5–33.5 cm^2) is attributed to the variation reported in the length of its proximal aponeurosis (MTJ) [36], which is potentially an important morphological finding as it is suggested that a small [36] or relatively narrow [45] aponeurosis may be a factor that increases the risk of injury.

As noted earlier ST has three sites of origin, two from the ischial tuberosity and one common with the proximal tendon of BF_{LH}. This complexity may make the proximal tendon difficult to define, yet measurements are relatively consistent with a mean length of about 12 cm (30% of total muscle length). The free tendinous component is very small (1–2 cm), and ST has the shortest proximal MTJ (formed along the aponeurosis on the anterior aspect of the muscle and the conjoined tendon) of approximately 11–12 cm (occupying 28% of total muscle length) [12, 14, 39].

1.3.3 Biceps Femoris Short Head

Proximally the BF_{SH} originates from the lateral femur and intermuscular septum with a small amount of tendinous tissue attaching the muscle to the bone. However, none of this tissue runs intramuscularly in the proximal region of the muscle. Therefore, as the fascicles of BF_{SH} arise directly from their proximal insertion sites into this small amount of tendinous tissue, the MTJ is minimal.

1.4 Architectural Characteristics of the Hamstrings

Muscle architecture consists of a range of characteristics that influence function. These characteristics affect a muscle's maximal force output [46], shortening velocity [46] and its susceptibility to injury [4]. The architectural characteristics of muscle consist of two main categories: (a) muscle size and (b) fascicles orientation and length.

1.4.1 Muscle Size Measures

The muscle size-related components of architecture consist of CSA which can be further delineated into anatomical CSA (ACSA) or the physiological CSA (PCSA). These two measures of muscle size are typically taken at a point-specific location along the muscle and consider the area of contractile tissue at that site. Whereas the product of a muscle's ACSA across its entire length is referred to as muscle volume [47]. The differences between ACSA and PSCA are highlighted below:

1.4.2 ACSA

The ACSA of a muscle is the area of the tissue which can be measured perpendicular to its longitudinal axis, typically expressed in centimetres squared (cm^2) [47].

1.4.3 PCSA

The PCSA is determined from a slice taken perpendicular to the longitudinal axis of the fascicles (as opposed to the longitudinal axis for ACSA). As there are differing structural arrangements of muscle fascicles (e.g. strap, fusiform, pennate etc.), a measure of PCSA is representative of the fascicles relative to their orientation within the muscle, which is neglected when using an ACSA measure. It is important to understand this distinction as the force a muscle can produce is relative to its PCSA which is influenced by its pennation angle as well as its CSA [48, 49].

1.4.4 Volume

The volume of a muscle is the circumferential, external area of the tissue which can be measured and is typically expressed as centimetres cubed (cm^3).

1.4.5 Fascicle Orientation and Length Measures

Muscle architectural type is defined by the orientation of the fascicles relative to the force-generating axis of the muscle. These different structural arrangements have implications for force-generating capacities (via its PCSA) as well as the shortening velocity of a muscle. The main variable which impacts these structural arrangements is pennation angle. This is the angle at which the fascicles attach to the tendon aponeuroses. With parallel structured muscles, the fascicles run from origin to insertion, therefore resulting in muscle length equalling fascicle length, with small, if any, pennation. Comparably obliquely structured (e.g. unipennate, bipennate) muscles have the fascicles inserting at different angles

along its length. Therefore, fascicle length in these pennate muscles is determined, simplistically, by the fascicle's angle of insertion into the aponeuroses, as well as the thickness of the muscle. Whilst this is a straightforward concept, throughout the hamstrings there are unique structural arrangements of fascicles across the four muscles.

1.4.6 Within Muscle Variability in Architecture

1.4.6.1 Semimembranosus

Based on fascicular orientation, SM is considered to have three distinct regions. Each segment has its own unique fascicular arrangement with the proximal and middle sections being unipennate and the distal portion being bipennate [14]. Despite this difference in structural arrangement, there is a heterogenous fascicular length along the muscle [14]. However, as is the case with the other hamstring muscles, SM displays a variance in fascicle lengths across the literature. Reported fascicle lengths in cadaveric samples range from 5 to 8 cm [14, 29, 30, 32, 35]. Furthermore, the variability in fascicular lengths along the SM leads to comparable differences in pennation angle within the muscle. These range from 15° through to 31° [29, 30, 32, 35, 50].

1.4.6.2 Semitendinosus

Semitendinosus is uniquely structured with a proximal (approximately one-third of the muscle) and distal (approximately two-thirds of the muscle) portion, separated by a tendinous inscription, or raphe (Figs. 1.5 and 1.7). Both segments of ST have fascicles which are parallel in alignment. This structural arrangement allows ST to have some of the longest fascicle lengths reported in the lower limb (along with sartorius and gracilis) [34]. However, the fascicular arrangement within each segment of ST is not consistently reported in the literature, with large variability amongst cadaveric samples. Some studies show no difference in fascicle length between the two segments [14], with others reporting longer fascicles moving from proximal to distal [39] and some showing large variability within each segment [51]. Across the literature, the fascicle lengths of ST range from 9 to 24 cm [29, 30, 32, 34, 35, 39, 50, 51]. These differences highlight the inconsistencies between human cadaveric samples as well as differences resulting from using various methods of assessing living samples (e.g. two-dimensional vs. three-dimensional ultrasound). Therefore, when assessing fascicle length of ST, the standardisation of the site needs to be considered, and consistency is important to enable accurate comparisons.

The pennation angle of the ST fascicles also shows large variability between segments because of the difficulty associated with defining the angle of insertion due to its parallel structure. The most common definition of pennation angle in ST is the fascicular insertion relative to the distal tendon [34]. Using this definition, there is a noticeable variance in pennation angle between the two segments with the distal

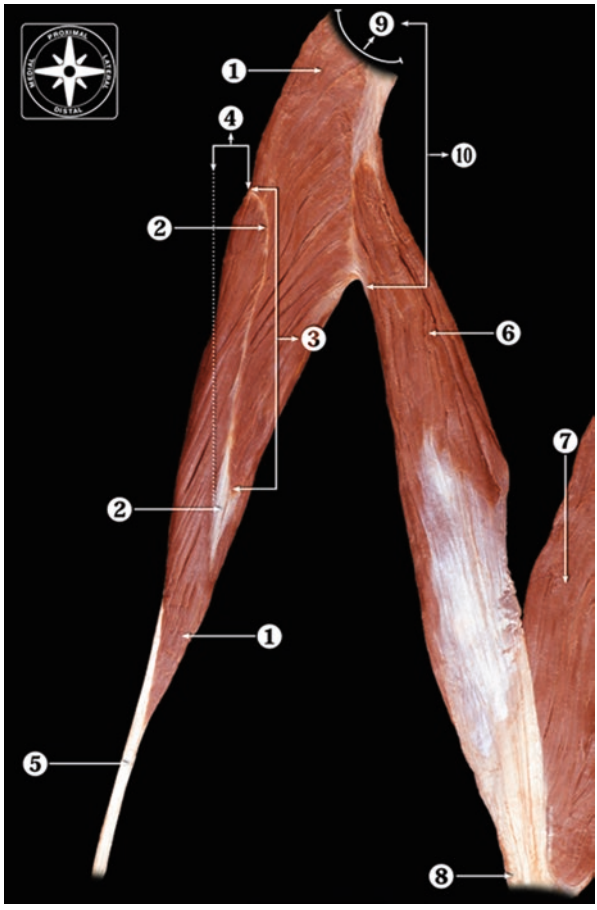

Fig. 1.7 Anatomical dissection showing the muscular characteristics of the ST muscle. (1) Semitendinosus muscle. (2) Raphe. (3) Length of the raphe (range of 5.0–9.0 cm). (4) Width of the raphe (3.0 cm maximum). (5) ST distal tendon. (6) BF_{LH} muscle. (7) BF_{SH} muscle. (8) BF distal tendon. (9) Ischial tuberosity (illustrative representation). (10) Conjoint tendon (BF_{LH} and ST muscles). (Printed with permission from van der Made et al. [13])

portion having a greater angle than the proximal [51]. Across ST, pennation angle ranges from 0° to 18° [14, 32, 34, 35, 38, 39, 51].

1.4.6.3 Biceps Femoris Long Head

Biceps femoris long head is classified as pennate in structure with fascicles running between the proximal and distal tendon (Figs. 1.5 and 1.7), which covers approximately 60% (Table 1.3) of the muscle [14]. Generally, the proximal portion of BF_{LH} possesses longer fascicles than the middle and distal segments of the muscle. However, within the literature there is some variability in BF_{LH} fascicle length with a range of cadaveric tissue or *in vivo* samples used. Some reports have found lengths as small as 5 cm with others reporting fascicles of up to 14 cm long [53, 54].

Like its fascicles, there is some variability in pennation angle along the length of the BF_{LH}, as well as between studies [14, 29, 39]. The proximal region of the BF_{LH}

Table 1.3 Distal tendon and musculotendinous junction lengths of the hamstring muscle

Length (cm)	Author(s), date (number of specimens; M, F)	Muscle cm, mean ± SD[a] [length as a % of total muscle length]			
		BF_{LH}	BF_{SH}	ST	SM
Distal tendon	Garrett et al., 1989 [18] Dissection (5; no details)	[66%]		[56%]	[52%]
	Woodley and Mercer, 2005 [14] Dissection (6; 3 M, 3F)	27.5 (range 24.1–33.9) [62.6%]	11.2 (range 9.2–12.8) [45.6%]	25.0 (range 22.1–33.0) [56.8%]	26.1 (range 23.6–31.8) [59.4%]
	Van der Made et al., 2015 [13] (56; no details)	BF 26.2 ± 2.9 [62%]		24.9 ± 3.7 [56%]	22.0 ± 3.3 [57%]
Distal free tendon	Woodley and Mercer, 2005 [14] Dissection (6; 3 M, 3F)	9.2 [21%]	0.5 [2%]	11.1 (range 9.0–12.1) [25.3%]	6.8 [15%]
	Kellis et al., 2009 [38] Dissection and US (6; 3 M, 3F)	Dissection: 11.8 ± 2.1 [30.2 ± 4.8%] US: 11.2 ± 1.4 [29.3 ± 3.8%]		Dissection: 17.1 ± 2.8 [16.5 ± 3.3%] US: 16.5 ± 3.3 [42.7 ± 6.5%]	
	Kellis et al., 2012[b] [35] (8; 8 M)	5.3 ± 1.8 [13.5 ± 5.3%]	1.4 ± 0.7 [6.7 ± 3.1%]	19.0 ± 2.0 [38.2 ± 4.1%]	7.8 ± 1.4 [19.3 ± 3.4%]
	Van der Made et al., 2015 [13] (56; no details)	BF: 9.1 ± 3.0 [22%]		13.2 ± 2.9 [30%]	5.5 ± 1.9 [14%]
	Vieira et al., 2017 [52] MRI (40, no details)	5. 0 ± 1.7 (range 1.5-10.0)[a] (range 1.5–10.0)[c]			
	Vadgaonkar et al., 2018 [41] (46; 46 M)			15.5 ± 3.2 (range 7.3–22.9)	
Distal MTJ	Woodley and Mercer, 2005 [14] Dissection (6; 3 M, 3F)	18.3 (range 15.4–25.0) [41.4%]	10.7 (range 9.2–12.8) [36.5%]	13.9 (range 10.1–20.0) [31.6%]	19.3 (range 16.6–24.5) [44.0%]
	Van der Made et al., 2015 [13] (56; no details)	BF: 17.1 [14%]		11.7 [26%]	16.5 [43%]

BF biceps femoris, *BF_{LH}* biceps femoris long head, *F* female, *M* male, *MRI* magnetic resonance imaging, *MTJ* musculotendinous junction, *SM* semimembranosus, *ST* semitendinosus, *US* ultrasound

[a]Unless stated otherwise

[b]Not clear if these data represent free tendon or MTJ length

[c]Measurement taken from level of knee joint space, not distal insertion site on fibula

has more pennate fascicles than its middle and distal portions [39]. The variance in pennation angle within the literature shows some samples of 0°, yet some report angles up to 28° [29, 32, 50]. The difference in the site and mode of assessment, the physical activity status (e.g. recreational or elite) and injury history may all influence the level of variability seen in BF_{LH} fascicle length and pennation angle.

1.4.6.4 Biceps Femoris Short Head

Due to the lack of an extensive proximal tendinous insertion, the BF_{SH} muscle has fascicles arising from three different locations: the linea aspera, the lateral supracondylar line of the femur and the intermuscular septum which separates BF_{SH} from vastus lateralis. As a result, its fascicular arrangement is variable and can be split into two regions [14]. Typically, the most posterior region of the BF_{SH} possesses longer fascicles than the anterior portion [14]. Across the literature, BF_{SH} possesses fascicles between 10.4 and 14 cm in length [14, 29, 35]. The pennation angle of the BF_{SH} ranges from 10 to 16° [29, 30, 35].

1.5 Distal Tendons and Musculotendinous Junctions

The lengths of the distal tendons, free tendons and MTJs are presented in Table 1.3.

1.5.1 Semimembranosus

The distal tendon of SM commences proximal to the middle of the muscle [7] and forms a large, broad aponeurosis on the medial aspect of the muscle [8, 14]. Semimembranosus has the longest distal MTJ of all the hamstring muscles (mean length 16–19 cm), but its entire distal tendon is slightly shorter than that of BF_{LH} and ST, measuring approximately 22–25 cm on average and occupying 52–59% of the muscle length [13, 14, 18]. Considering the tendinous morphology of SM, the distal (extending 52–59% the length of the muscle) and proximal (extending 75% the length of the muscle) tendons overlap along the length of the muscle (Figs. 1.7 and 1.8). On the posterior aspect of the lower part of SM, the tendon tapers to become heavy and rounded near its insertion site [8, 17].

1.5.2 Semitendinosus

The distal tendon of ST is long and thin and lies on the superficial surface of SM (Figs. 1.1, 1.7 and 1.8). The tendon commences as a small aponeurosis on the anterior aspect of the muscle at about the mid-level of the thigh [8, 14, 17], forming a MTJ which extends approximately 30% of the muscle length [13, 14]. The free distal tendon is the longest of all of the hamstrings (mean length ranges between 11

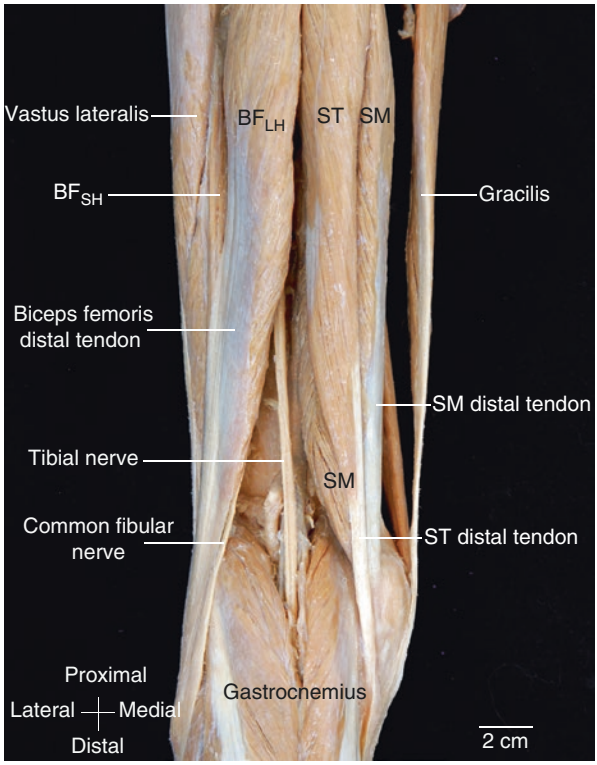

Fig. 1.8 Dissection photograph of the left distal hamstring complex (posterior view). *BF_{LH}* biceps femoris long head, *BF_{SH}* biceps femoris short head, *SM* semimembranosus, *ST* semitendinosus

and 19 cm) [13, 14, 35], and its distal portion is often cradled in a trough formed by the superficial surface of SM [14] before it curves around the medial condyle of the tibia, passing superficial to the medial collateral ligament towards its insertion [17].

1.5.3 Biceps Femoris

The distal tendon of BF_{LH} is the longest of all of the hamstrings, measuring approximately 27 cm, extending 60–65% the length of the muscle [13, 14]. The tendon takes the form of a broad, fan-shaped aponeurosis [14, 17] covering the lateral aspect of the lower portion of its muscle belly and some of BF_{SH} (Figs. 1.1, 1.7 and 1.8), forming a distal MTJ that extends approximately 40% of the muscle length (18 cm) [14]. The most proximal extent of the tendon originates on the lateral, deep aspect of the muscle belly at about the mid-point of the thigh, narrowing to form a broad flat tendon 7–10 cm proximal to the knee joint [55, 56]. The portion of the distal tendon which is devoid of muscle fascicles measures between 5 and 12 cm [13, 14, 35, 52].

The deep surface of the distal BF_{LH} tendon also forms an insertion site for the fascicles of BF_{SH} (Figs. 1.1, 1.7 and 1.8) [7, 14, 17, 55–57], which span a mean

length of 10.7 cm (range 9.2–12.8 cm) occupying 36.5% of the total length of muscle and thereby forming the distal MTJ [14]. The fascicles from each head of the BF are oriented differently and, at their insertion into the BF_{LH} tendon, meet at an angle of approximately 45° [14].

1.6 Distal Insertions

1.6.1 Semimembranosus

The distal SM tendon is an important component of the posteromedial corner of the knee alongside the medial collateral ligament, posterior oblique ligament and posterior horn of the medial meniscus (Fig. 1.9) [58, 59]. At the knee joint, SM likely

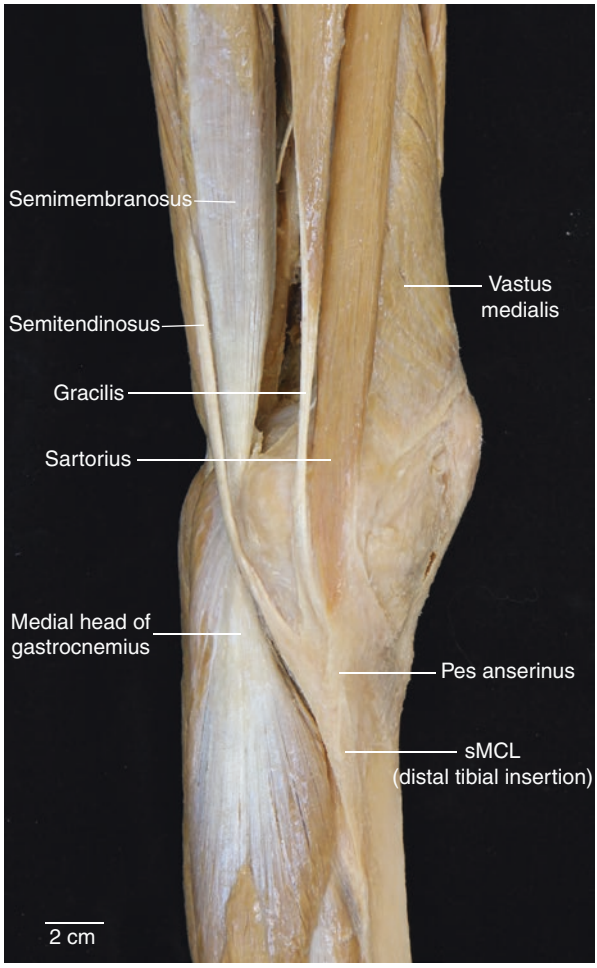

Fig. 1.9 Dissection photograph of the medial aspect of the left knee. Note the contribution of the distal ST tendon to the pes anserinus, alongside the distal tendons of gracilis and sartorius. *sMCL* superficial medial collateral ligament

functions as an active restraint to valgus (when the knee is extended) and external rotation (with knee flexion) [60]. The anatomy of this region is complex, with differences evident in the number and location of arms attributed to the distal SM tendon and their relationship to surrounding tissues. Between three and eight different arms of the distal SM tendon have been described [7, 16, 17, 55, 58, 61–63], with [64] providing the most comprehensive account of its insertional anatomy. Of these eight components, three appear to have been consistently identified and agreed upon in the literature: the direct arm, anterior arm and expansion to the oblique popliteal ligament.

Immediately distal to the joint line, the SM tendon bifurcates into a direct and anterior arm [64, 65], although this separation may not be distinct [58]. The direct arm is derived from the main portion of the SM tendon [64] and courses distally to attach to a tubercle, sometimes referred to as the tuberculum tendinis [16, 17, 64, 66] on the posterior aspect of the medial tibial condyle [7, 16, 17, 55, 61–63]. This arm is described to expand, forming a broad U-shaped convex attachment, which is located approximately 1 cm distal to the joint line [64].

The anterior (reflected or tibial) arm takes the form of a thick tendinous expansion, originating just proximal to the tibial attachment of the direct arm, within the medial edge of the SM [64]. It runs in an antero-inferior direction and attaches to the medial tibial condyle, deep to the proximal tibial insertion of the superficial medial collateral ligament [16, 58, 64, 66, 67]. This insertion site is oval shaped and approximately 1 cm distal to the joint line [58, 60, 64, 66]. The direct and anterior arms of the SM tendon are closely related to the SM bursa, described as an inverted U-shape [68] that forms proximal to the attachment of the direct arm on the tibia [66]. De Maeseneer et al. [58] state that this bursa covers the medial and lateral aspects of the transition area between the direct and anterior arms, while [66] describe the lateral aspect of the bursa lying between the direct arm attachments to the coronary ligament and tibia, with its medial aspect surrounding the anterior arm.

A thin, broad lateral expansion of the SM tendon [16, 17, 58, 64, 69, 70], with possible contribution from the SM tendon sheath [67, 71] or the capsular arm of the posterior oblique ligament [64, 67], forms the medial aspect of the oblique popliteal ligament. La Prade et al. [66] report that a "lateral tendinous expansion" from the main SM tendon, arising just proximal to the bifurcation of the direct and anterior arms, also contributes fibres to the oblique popliteal ligament. The ligament, which has a length of approximately 4.5–4.8 cm, courses posterolaterally towards the lateral femoral condyle. Inconsistencies are apparent regarding its lateral insertions which include the fabella (when present) [64, 71], the posterolateral joint capsule [64, 69, 71] or the lateral femoral condyle [69]. Additional insertions to the popliteus muscle [64, 71] and the lateral aspect of the posterior cruciate ligament facet on the posterior tibia [64] have been reported, with part of the plantaris muscle also gaining insertion into the lateral aspect of the oblique popliteal ligament [64, 71]. Although not well understood, the oblique popliteal ligament is thought to act as a restraint against hyperextension of the knee joint [64, 72] with the tibial attachment having a potential role in providing rotatory stability [64].

Various other components of the distal SM tendon have also been described. A distal tibial or popliteal arm, arising from the inferior aspect of the direct arm [58] or the coronary ligaments adjacent to the direct arm [64], forms a fascial expansion over the popliteus muscle [16, 58, 61, 62, 64]. An extension from the SM tendon or tendon sheath [55, 58] to the posterior oblique ligament [58, 61, 64] and an arm to the posterior horn [58] of the medial meniscus [58, 61, 62, 64] via the coronary ligament [58, 64] are also reasonably consistent findings. With respect to the meniscal arm, it is hypothesised that during knee flexion, contraction of SM displaces the medial meniscus posteriorly, thereby protecting it from impingement between the femoral and tibial condyles [61, 62]. An additional, inconstant expansion to the posterior horn of the lateral meniscus has also been described [73] but not identified in more recent studies [58, 64]. A proximal posterior capsular expansion, described by La Prade et al. [66], located proximal to the oblique popliteal ligament coursing along its superior border to blend laterally with the posterolateral joint capsule [64] has also been reported.

1.6.2 Semitendinosus

Together with the distal tendons of sartorius and gracilis, ST contributes to the pes anserinus on the anteromedial aspect of the proximal tibia (Fig. 1.9). These three tendons insert in a linear fashion along the lateral extent of the anserine bursa (which separates them from the superficial surface of the distal portion of the medial collateral ligament), with sartorius most proximal, gracilis in the middle and ST most distal [17, 66]. The distal tendon of ST fuses with an aponeurotic membrane from the gracilis tendon [17, 74] and has a mean insertional width of 1.1 (range 0.8–1.6) cm, being wider than the tendons of sartorius and gracilis (0.8 cm) [66].

Nomenclature is variable, but a number of accessory bands or tendons or tendinous expansions are associated with the tendons that comprise the pes anserinus. Examples that relate to ST include an accessory tendon that arises from its tendon proximal to where it blends with gracilis, which passes on the deep surface of the ST tendon to fuse with the crural fascia [17, 74]. Thin accessory bands of ST may number between two and three, blending with the medial gastrocnemius fascia [75, 76] and the fascia of popliteus [75]. An understanding of normal and potential variant anatomy is critical for surgical harvest of the ST tendon which can be used for reconstructive repair of the patellar tendon or anterior cruciate ligament [76].

1.6.3 Biceps Femoris

It is generally accepted that the main part of BF tendon inserts into the lateral aspect of the fibular head (Figs. 1.8 and 1.10) [17, 77–79] and is closely related to, and divided by, the fibular collateral ligament [55, 56, 77–79], with an additional extension to the lateral tibial condyle [17, 55, 56]. However, the detailed anatomy of this insertion site at the posterolateral aspect of the knee is complex and has been

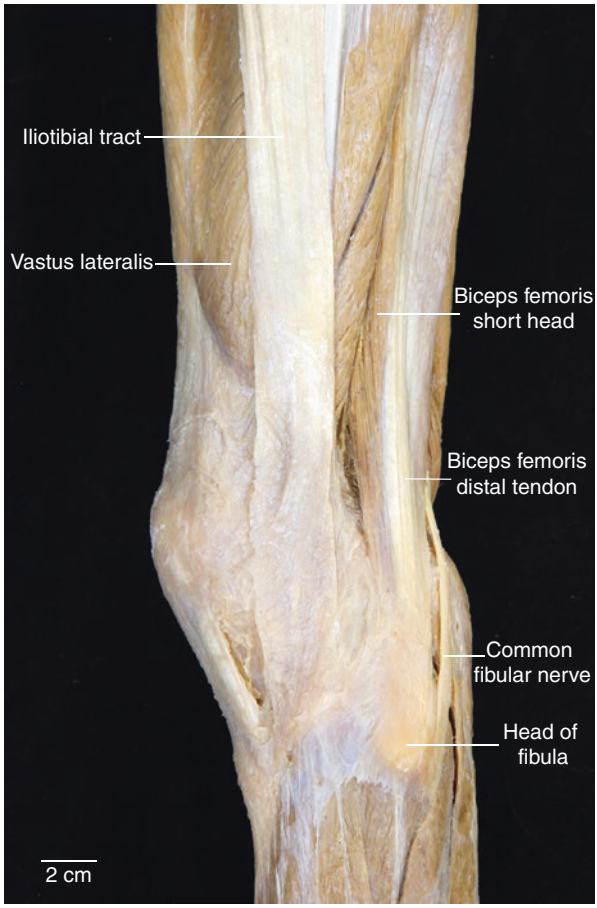

Fig. 1.10 Dissection photograph of the lateral aspect of the left knee. Note the distal tendon and insertion of the BF tendon into the lateral aspect of the head of the fibula

described in a variety of ways, with various names given to different components of the tendon. Slips, extensions or laminae of the BF tendon insert or blend with surrounding tissues including the fibular collateral ligament, crural fascia, iliotibial tract [55, 56, 78, 79], popliteus tendon and the arcuate ligament [79]. An additional fascial attachment to the lateral femoral condyle approximately 3–4 cm proximal to where the BF tendon splits has also been described [79].

A three-layer arrangement of the insertions of BF_{LH} and BF_{SH} is reported by Terry and La Prade [57, 80], which brings together elements from the earlier work of Sneath [56] and Marshall et al. [55]. Five attachments of BF_{LH} are described, consisting of two tendinous components (a direct arm and an anterior arm) and three fascial components (a reflected arm, a lateral and an anterior aponeurosis). The reflected arm is the most proximal component and inserts into the posterior edge of the iliotibial tract just proximal to the fibular head. Insertion of the direct arm is into the posterolateral edge of the fibular head. The anterior arm inserts into the lateral edge of the fibular head, and a portion ascends anteriorly forming the lateral aponeurotic

expansion that covers the fibular collateral ligament. The medial aspect of the anterior arm is separated from the distal quarter of the ligament by a small bursa, with the lateral portion of the anterior arm continuing distally to terminate in an anterior aponeurosis that overlays the anterior compartment of the leg [57, 65, 80].

The remaining insertions are derived from BF_{SH}, and whilst Sneath [56] suggests a three-laminar arrangement, Terry and La Prade [57, 80] describe six components. The first is a muscular insertion into the deep (anterior) and medial surface of the BF_{LH} tendon (as described above). Muscle fascicles of the BF_{SH} also terminate at two other sites: the posterolateral joint capsule (via the capsular arm which passes deep to the fibular collateral ligament) and the capsuloosseous layer of the iliotibial tract. The distal BF_{SH} comprises two tendinous insertions, a direct arm to the superficial surface of the fibular head (positioned medially to the lateral collateral ligament) and an anterior arm, which passes deep to the fibular collateral ligament, partially blends with the anterior tibiofibular ligament and then inserts into tibia, 1 cm posterior to Gerdy's tubercle. Finally, a lateral aponeurotic expansion attaches to the posteromedial aspect of the fibular collateral ligament [57, 80].

At the knee joint, the BF tendon acts a dynamic stabiliser to resist anterolateral-anteromedial rotatory instability [79, 80]. Injuries to structures of the posterolateral corner (fibular collateral ligament, popliteus tendon, popliteofibular ligament) alongside the biceps tendon are associated with severe rotational instability [63].

1.7 Neurovascular Supply

The hamstring muscles are innervated by branches of the tibial division of the sciatic nerve, with the exception of BF_{SH} which is supplied by the common fibular nerve. Arterial supply is predominantly received from branches of the profunda femoris artery (deep artery of the thigh), and venous drainage occurs via tributaries of the profunda femoris vein.

1.7.1 Semimembranosus

Semimembranosus generally receives a single muscle nerve from the tibial division of the sciatic nerve [7, 14, 81, 82] (Figs. 1.11 and 1.12), and this may sometimes arise in common with the nerve supplying the distal compartment of ST [7, 14, 82]. A branch of this muscle nerve also supplies the posteromedial portion of AM, either having a shared common trunk of origin [82] or being derived from a proximal branch of the nerve that supplies SM [7, 14]. The number of primary muscle branches entering SM (motor points) varies from 1 to 5, and this may be due to different interpretations of what constitutes a primary muscle branch [7, 14, 81, 83, 84]. Semimembranosus is usually supplied from all four of the perforating arteries (which arise from the profunda femoris), but predominantly from the first. The inferior gluteal artery may contribute at the proximal attachment of SM, whilst the distal part of the muscle is supplied by a branch of the femoral or popliteal artery [17].

1.7.2 Semitendinosus

Two primary nerve branches serve the ST coming from the tibial nerve with one supplying the proximal portion of the muscle (above the tendinous inscription) and the other the distal portion (Figs. 1.11 and 1.12) [7, 14, 31, 81, 82, 84]. In some instances a single primary nerve branch to ST (which subsequently divides into two) has been identified [14], and one of the nerve branches to ST may share a common trunk with either the nerve to SM [14] or BF_{LH} [82]. The proximal part of ST is supplied by the medial circumflex femoral artery [17, 31], and the first [17] or second [31] perforating arteries supply the distal portion. The inferior gluteal artery contributes at the proximal attachment of ST, and an accessory supply is received from the inferior medial genicular artery at its distal insertion [17].

Fig. 1.11 (**a**, **b**) Entry points of motor branches to the hamstring muscles [15]. (1) Motor branch to the BF_{LH} muscle, (2) two motor branches to the ST muscle, (3) motor branch to the SM muscle. (Printed with permission from Stepien et al. [15])

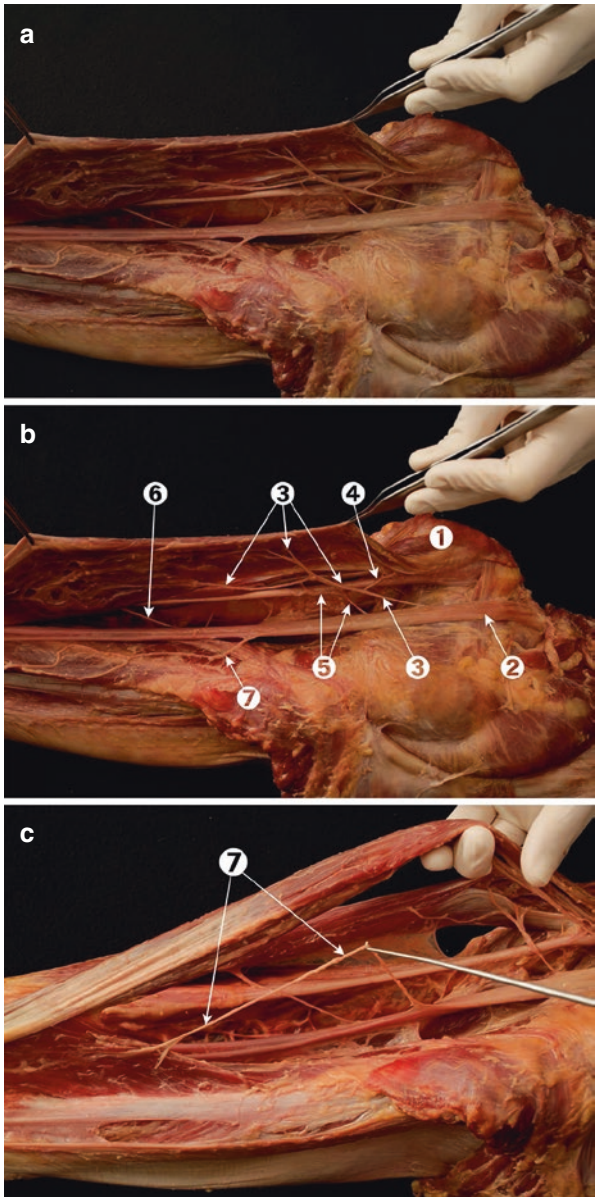

Fig. 1.12 (**a**–**c**) Lateral view of the innervation of the hamstring muscle complex [15]. (1) Ischial tuberosity; (2) sciatic nerve; (3) motor branch to the BF_{LH} muscle; (4) recurrent branch to the proximal attachment of conjoined tendon; (5) motor branch to the ST muscle; (6) motor branch to the SM muscle; (7) motor branch to the BF_{SH} muscle. (Printed with permission from Stepien et al. [20])

1.7.3 Biceps Femoris Long Head

Variation is evident regarding the nerve supply to BF_{LH}. There is consensus that a single primary nerve innervates a proportion (or all) of BF_{LH} muscles (Figs. 1.11 and 1.12) [14, 31, 81, 82, 84, 85], but BF_{LH} may also be innervated by more than one nerve [81, 82, 84, 85]. When one nerve innervates BF_{LH}, it may divide into two branches; this pattern was found in a third of specimens studied by Shanahan et al.

[85] and in all specimens in three other studies [7, 14, 31]. If BF_{LH} is supplied by two nerves, the second branch may arise separately from or share a common point of origin with the first. It may also share a common origin with the nerves which supply AM and SM [82]. The first and second perforating arteries supply BF_{LH} [17, 31] with contributions from the medial circumflex femoral [17, 31] and inferior gluteal [17] proximally; distally the superior lateral genicular artery provides an accessory supply [17].

1.7.4 Biceps Femoris Short Head

The innervation of BF_{SH} differs to the other hamstring muscles, being derived from the common fibular nerve. Once again, variation is evident in the pattern of innervation with reports of one motor primary nerve most common [7, 26, 82, 84], with two motor nerves supplying BF_{SH} in some instances [14, 82]. Arterial supply to the superior BF_{SH} is from the second or third perforating artery, with the superior lateral genicular artery supplying the inferior part [17]. Anastomotic vessels between the two heads of BF are usually present, around the level of where the muscle bellies blend (onto the distal tendon) and mid-way along the length of the BF_{SH} muscle belly [86].

1.8 Conclusion

The structure of each of the hamstrings, like any muscle, determines its function [47]. Therefore, the anatomical variables described in this chapter should assist comprehension across the remaining chapters. As an example, the biomechanical demands of running expose the hamstring muscle group to forceful, repetitive lengthening actions [87, 88]. The ability of the hamstrings to perform these actions, and by extension the likelihood of hamstring injury, will be partially dictated by their structure [87, 89, 90]. Furthermore, architectural characteristics, namely, BF_{LH} fascicle length, have been identified as a variable that can modulate the risk of future hamstring injury [4], and the ability to cause adaptation to this structural characteristic may help to guide preventative efforts [91–93]. In addition, damage to different anatomical structures (i.e. MTJ, muscle fibres, free tendon, intramuscular tendon) is a factor that may require consideration in the rehabilitation and prognostication of hamstring injury as well as the return-to-sport decision-making process [5]. Whilst these present just a few examples of the importance of understanding the anatomy of the hamstrings, it is anticipated that the current chapter provides a foundation to maximise the learnings from the remainder of this book.

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Basic Muscle Physiology in Relation to Hamstring Injury and Repair

2

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2.1 Overview of Structural and Cellular Components Affected by Strain Injuries

2.1.1 Insights into the Basics of Muscle Strain Injuries

Human skeletal muscle can be injured by strain, contusion, or direct laceration. The majority of sports-related skeletal muscle injuries are caused either by strain or contusion [1], as lacerations are almost nonexistent in sports. Hamstring strain injuries (HSIs) are a result of excessive intrinsic tensile forces and inflict substantial damage across myofibres, the myofibres' basement membrane, as well as mysial sheaths and the connected tendon/aponeurosis. Additionally, strain injuries cause blood vessels in the endo- or perimysium to rupture during the trauma [4]. The injury is most commonly located at or adjacent to the myotendinous junction (MTJ) [2–4]. Both the proximal and the distal MTJs of the hamstring muscle group cover an extensive part of the muscles rather than a limited area at either end of the hamstring muscles. As an example, the proximal MTJ of the biceps femoris long head (BF_{LH}) spans approximately a third of the total muscle length [5, 6], whereby the myofibres attach to the aponeurosis to transmit force from the BF_{LH} muscle to the tendon. The proximal and distal aponeurosis is often also called the "central tendon" or "intramuscular tendon," and it is noteworthy that the most severe hamstring

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muscle injuries involve the central intramuscular tendon, emphasising the role of the connective tissue in relation to HSIs [7].

A large number of different experimental animal models have been introduced over the years to enable the study of tissue repair following muscle injuries. In general, the biggest challenge lies in the development of a model which mimics the injury-causing mechanisms. With regard to muscle strains, the injury provoking impulse is excessive tensile strain. Pioneering work by Tidball and colleagues involved application of passive strain to isolated frog muscles and their attached tendons. This model demonstrated that the location of the tear is at the MTJ [3]. Similar observations were made by Garrett and colleagues reporting the injury site to lie within ~0.5 mm of the MTJ [4]. One of the most important findings relates to the failure site, which is located external to the myofibre cell membrane. This means that the basement membrane of the myofibres is torn off and leaves the myofibres separated from the normally attached connective tissue [3, 4].

The experimental model was later applied to whole frog semitendinosus (ST) muscle-tendon preparations while the muscles were stimulated [2]. In this setup, the failure site was also located at the MTJ where the collagen fibres from the associated tendon became torn off. The separated collagen fibrils are clearly seen on electron micrographs taken from the ST muscle-tendon unit (MTU) (Fig. 8 in [2]): The fingerlike processes from the muscular side of the MTJ are clearly visible, but after the strain injury, there is a detachment of the myofibres from the tendon. It is noteworthy that the muscle tissue appears organised with no detectable damage to the Z-lines, suggesting that the skeletal muscle tissue in itself is not greatly damaged.

Other animal models have focused on the ability of skeletal muscle tissue to initiate repair following complete transections of the soleus muscle at the muscle midbelly ("laceration injury model") [8–10], or by subjecting muscles to the forces generated by a spring-loaded hammer [11]. The complete laceration injury models showed how regenerating myofibres can enforce attachment to the extracellular matrix (ECM) on the lateral aspect of the myofibres, thereby increasing stability of the injured tissue during the healing. Findings obtained from the laceration injury models also show how the muscle-specific integrin receptor $\alpha_7\beta_1$ expression as well as its distribution is involved in conferring stability between the myofibres and the ECM during regeneration [12, 13]. In general, integrin receptors enable the linkage of cells to the ECM (Fig. 2.3). More specifically, this cell receptor type couples the intracellular cytoskeleton to the specific binding partners in the matrix on the extracellular side. For the muscle-specific integrin receptor $\alpha_7\beta_1$, the binding partner on the extracellular side is laminin (Fig. 2.1).

An increase, concomitant with a redistribution of the integrin receptor during the repair of muscle tissue, suggests the importance between the coupling of the myofibres and the surrounding matrix. Another important aspect of the laceration models in animal skeletal muscle is time: Studies applying these models reported that scars are not rapidly replaced but may persist over a prolonged time span, potentially permanently [15]. It is important to keep in mind that the muscle midbelly is not a common site of failure in HSIs, and it is also somewhat difficult to

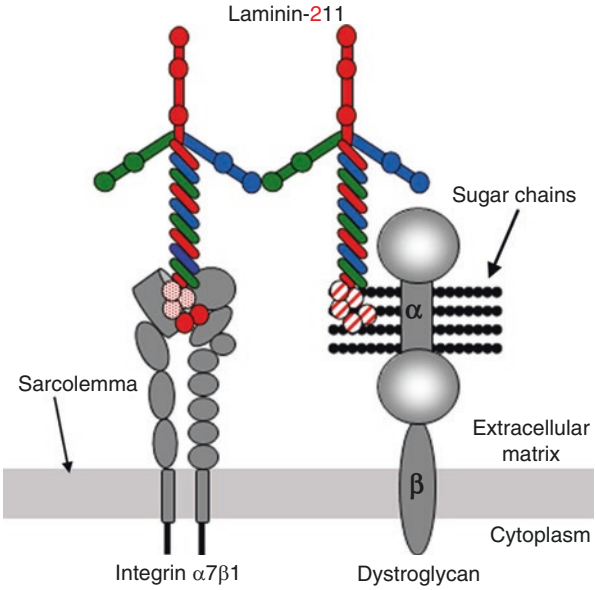

Fig. 2.1 Schematic illustration of the muscle-specific integrin $\alpha_7\beta_1$ and the binding sites on the laminin α_2 chain. The integrin receptor $\alpha_7\beta_1$ has a short intracellular part (in the cytoplasm) and an extended extracellular part, where integrin $\alpha_7\beta_1$ binds to laminin via the laminin globular domains. (Reproduced with adaptation from Fig. 3 of Gawlik and Durbeej [14])

compare results from a contusion injury to the repair processes of a strain injury. Further, even the application of excessive strain is applied under controlled conditions, where the MTU is stretched until failure with a steady strain rate [2]. This controlled and constant strain does not fully replicate the explosive hamstring movements, which precede an acute HSI. Another aspect to consider when dealing with animal models is the aspect of time, as animals grow and heal substantially faster than humans. Additionally, animals do rarely show signs of pain, which complicates the examination of functional deficits related to these injury models. Finally, the role of the central tendon in HSIs cannot be studied in models, as the tendinous inscriptions extending into the muscle belly are not described in animals.

2.1.2 Development of the Myotendinous Junction and Its Adaptation to Loading and Unloading

The most common localisation of strain injuries is the MTJ, which is the interface where the myofibres attach to the collagen fibres of the tendon [2, 16, 17]. Research into the adult human MTJ in general, and the regenerative capacity of the junction in particular, has remained scarce despite the fact that the MTJ is susceptible to strain injuries. In the optimal way, repair of the injured MTJ replicates the developmental processes to re-establish tissue integrity. The MTJ is a highly specialised anatomical region in the locomotor system, where force generated by the muscle is transmitted from the intracellular contractile elements of the muscle cells to the

ECM proteins in the tendon. This linkage enables movement. On a functional level, the MTJ has to overcome a mechanical mismatch as the muscle is highly compliant, but the tendon, on the contrary, is a stiff tissue [18]. The consequences of connecting mechanically different tissues are strain concentrations, which increase the risk of injury. Local strains at the MTJ in the hamstrings can be modified when either muscular (e.g. change in cross section) or tendinous (e.g. change in stiffness) dimensions are altered [19]. Keeping in mind that the connection between different mechanical tissues is a challenge, it is noteworthy that the MTJ can repeatedly withstand high loads [20, 21]. To be able to do so, the MTJ presents with a highly organised structure connecting proteins and matrix from the subsarcolemmal cytoskeleton to the sarcolemma and the basement membrane of the final sarcomeres at the muscular side and then to the collagen fibres on the tendinous part of the MTJ. Both the organisation of the MTJ components and the involved molecules mediate the unique capacities of the MTJ.

The architecture of the adult MTJ is characterised by extensive folding, which results in a significant increase in the muscle-tendon contact area. Shown in rat MTJ development, the organisation of the junction undergoes substantial changes in the first days after birth. Right after birth, the MTJ has a smooth and even appearance, but already 2 weeks later, the junction is more complex, and folding becomes obvious. Following another 2 weeks, rat MTJ has deep recesses and the folding is extensive [22]. It should be noted here that these processes take considerably longer time in human MTJ development.

For a long time, these folds have been described as fingerlike processes, but new imaging techniques on the ultrastructural level in human adult MTJ revealed that the structures resemble ridgelike protrusions. Three-dimensional reconstructions of electron micrographs further showed that collagen fibres of the tendon condense and the tendinous collagen fibres expand into myofibrillar indentations [23]. Based on 2D images, it was postulated that the extensive folding increases the contact area approximately 10–20 times compared to an interface with smooth transitions from one tissue to the other, but these numbers are most likely significantly higher when taking the three dimensions into account (Fig. 2.2).

Functionally, the enclosure of tendon tissue (i.e. collagen fibres) around the myofibres might enable the "grab and trap effect" as discussed by Knudsen and colleagues [23]. The underlying mechanism is based on the mechanically stiff collagen fibres encasing the myofibres at the junction. When the muscle contracts, the myofibres shorten and become wider and the surrounding collagen fibres might reinforce the muscle-tendon connection, thereby improving force transmission. The tendon tissue covering the muscle, also termed "aponeurosis," is specialised for force transmission from the muscle to tendon and then bone. Biomechanical analyses demonstrated that the aponeurosis has different mechanical properties compared to the free tendon [25]. Magnusson and colleagues showed that the free tendon had a significantly greater strain compared to the aponeurosis under isometric maximum voluntary contractions. The authors suggest that part of the observation reflects the energy storing and releasing capacity of the free tendon, while the aponeurosis is predominantly designed to effectively transmit contractile force at the MTJ [25].

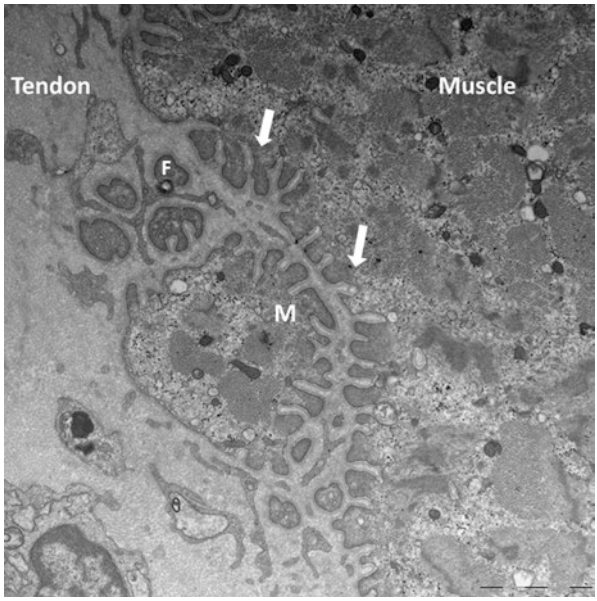

Fig. 2.2 Electron micrograph of the human myotendinous junction (2D). Figure illustrates the marked folding and interdigitations at the contact area between muscle and tendon. (Reproduced with adaptation from Fig. 3 of Bayer et al. [24])

The interaction between the myofibres and the aponeurosis/tendon during isometric contractions is relatively straightforward compared to dynamic situations such as those that occur during stretch-shortening cycle movements. During such movements the muscles are activated prior to the stretch to stiffen the contractile component so that the elongation during the stretch (braking) phase occurs predominantly at the tendon [26]. This provides elastic energy for the shortening (propulsive) phase. In this situation the contractile component (myofibres) is stiffer than the noncontractile component (tendon). However, the level of muscle activation and the length of the muscle fibres can affect the stiffness of the contractile component. At lower activations or longer muscle lengths, the contractile component may be less stiff than the noncontractile component and MTU elongation may occur at the MTJ as opposed to the tendon. With respect to hamstring injury, interrupted motor control and altered biomechanics during stretch-shortening cycle movements, such as sprinting, can place MTUs at lengths and tensions that can predispose them to injury.

The specialised organisation of the MTJ is mediated by a composite of molecules, which act in an orchestrated way during development and homeostasis. On the muscular side, an important protein complex responsible for force transmission from myofibres to the connective tissue is dependent on binding of intramuscular actin to the dystrophin group. Dystrophin at the intracellular part of myofibres is associated with a large oligomeric complex of sarcolemmal proteins and glycoproteins, also known as the dystrophin-glycoprotein complex. Force transmission is allowed through dystrophin binding to the C-terminal of β-dystroglycan. On the extracellular side, α-dystroglycan, which is anchored to β-dystroglycan, serves as a receptor for ligands such as laminin in the basement membrane surrounding

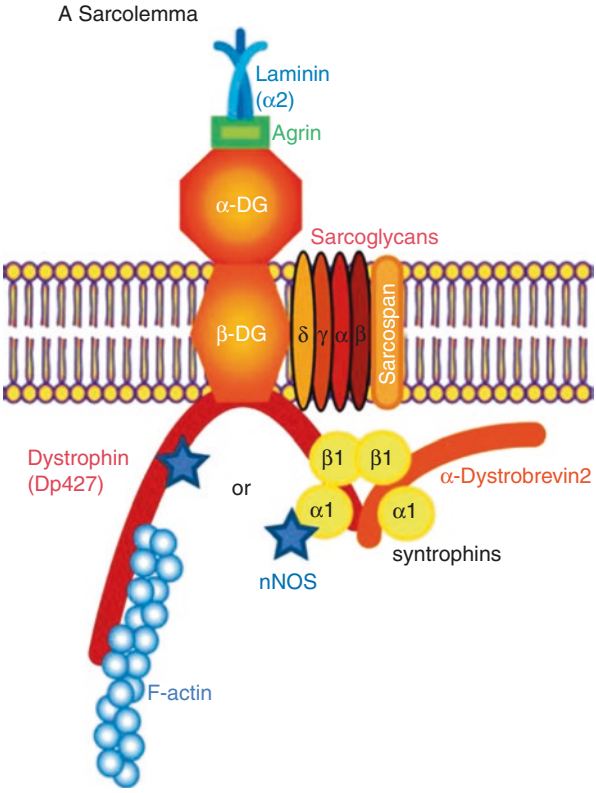

Fig. 2.3 The dystrophin-glycoprotein complex composition in mammalian skeletal muscle. In skeletal muscle, β-dystroglycan is linked to the extracellular α-dystroglycan, which, depending on the tissue, links laminin α_2 along the sarcolemma. In addition, β-dystroglycan associates with δ-sarcoglycan by which the sarcoglycan-sarcospan complex is stabilised at the sarcolemma. (Reproduced with adaptation from Fig. 1 of Pilgram et al. [30])

myofibres [27, 28] (Fig. 2.3). At the MTJ, the dystrophin-glycoprotein complex is found at high concentrations to provide mechanical stability [29].

Another fundamental segment of the force transmission at the MTJ involves the binding of actin filaments within the terminal sarcomeres to associated "attachment" proteins. These proteins include α-actinin, talin, vinculin, paxillin, and tensin, and they bind to the intracellular β_1-subunit of the receptor integrin $\alpha_7\beta_1$ (Fig. 2.1). The transmembrane integrin receptor $\alpha_7\beta_1$ is enriched at the MTJ and binds to laminin in the basement membrane [31]. It is important to note that laminin becomes incorporated in collagen fibres at the MTJ and thereby constitutes an integral part of the junctional ECM. A lack of integrin $\alpha_7\beta_1$ causes abnormal MTJ morphology seen as a clear reduction of the characteristic folding at the MTJ as well as significant weakening of the muscle [32]. In the context of MTJ development, it is noteworthy that the appearance and accumulation of integrin $\alpha_7\beta_1$ coincides with membrane folding and myofibril insertions during developmental stages and might therefore play a role in the very early steps of MTJ organisation [31].

The morphogenesis of the MTJ during embryonic development reflects the coordinated processes of chemical and mechanical signalling as well as the interdependence between myogenic cells and cells differentiating into the tendinous

lineage. During development, tendon cells connect with the developing muscle at the MTJ, and the development is to a significant degree governed by the interaction of integrin receptors and ECM molecules secreted by both the muscle and tendon cells [33, 34]. The collagen type XXII (COLXXII) and its receptor binding serve as a good example of the interaction between muscle and tendon cells at the developing MTU and exemplifies the direct link between muscle and tendon tissue on a cellular and molecular level. COLXXII is a collagen subtype predominantly found at the MTJ; it belongs to the collagen subtype of "fibril-associated collagens with interrupted triple helices" (FACITs) and is expressed during development by undifferentiated muscle cells located next to the basement membrane [35]. At that stage, COLXXII expressing cells are in close contact with tendon cells (also called tendon fibroblasts or tenocytes). On the tendinous side, the cell receptors integrin $\alpha_2\beta_1$ and $\alpha_{11}\beta_1$ are expressed by tendon fibroblasts. Visualisation by the immunofluorescence technique clearly demonstrates co-localisation of COLXXII and $\alpha_2\beta_1$ integrin as well as COLXXII and $\alpha_{11}\beta_1$ [36]. This co-localisation means that COLXXII and the integrin receptors form a functional unity, which is a necessity for the development of the MTJ. The connection between COLXXII and its binding partners on the tendinous side is required for mechanical stability at the MTJ as the lack of COLXXII (tested by creating a genetic deletion of COLXXII in zebrafish) is associated with a significant decrease in muscle force. This force reduction did not seem to be a result of a defect of contractile elements within myofibres, and also, the basement membrane of the mutant animals remained firmly attached to the sarcolemma. The study of these mutant animals showed that the linkage between muscle fibres and the junctional collagen fibrils was disrupted, myofibres were detached from the MTJ, and the characteristic MTJ architecture of interdigitations was lost [37]. Importantly, COLXXII is identified not only as a part of the MTJ development but also expressed and localised to the myofibre edge at the adult human MTJs [38].

As COLXXII appears to be a key element in conferring mechanical stability to the MTJ, at least during development, it is natural to ask whether loading or unloading in the adult MTJ would affect COLXXII synthesis. This was tested in untrained men undergoing a training programme with heavy resistance exercises of the hamstring muscles for 4 weeks. The intervention did not induce any change in the synthesis or localisation of this collagen type [38], suggesting that COLXXII in the human adult MTJ is not, or only to a minor degree, load sensitive. It could, however, also be that 4 weeks of training was a too short time span to cause significant changes at the human MTJ. Whether COLXXII is affected by immobilisation in humans has remained unexplored.

Tenascin-C is another ECM protein at the MTJ expressed by fibroblasts on the tendinous side of the MTJ. It is a large elastic ECM glycoprotein, which can be stretched several times its resting length by mechanical loading [39]. Tenascin-C is involved in tissue morphogenesis, including tendon, and is load sensitive in the free tendon, the MTJ, and myofascial junction as well as in muscle connective tissue but not in skeletal muscle tissue [40]. Probably its best-known function relates to modulation of cell (de-)adhesion and mechanosensitivity [41]. The lack of mechanical

force (generated by muscle contraction), as observed during cast immobilisation, leads to significant changes of tenascin-C expression: In an animal experiment, rat hind limbs were immobilised for 3 weeks and this intervention resulted in a dramatic decrease of tenascin-C at the MTJ and the free tendon. The downregulation of tenascin-C was, however, reversed when the rats were allowed to use their legs normally again. After 8 weeks of remobilisation, tenascin-C was re-expressed at the MTJ, the myofascial junction, and the free tendon [40].

Thrombospondin-4 is a subtype of the thrombospondins, a group of glycoproteins regulating protein-protein and protein-ECM interactions [42]. It serves as a key scaffolding protein mediating the organisation of the MTJ, which probably relates to its role in regulating the structure of collagen fibrils in connective tissues [43]. Thrombospondin-4 is expressed by both myoblasts and tendon fibroblasts and binds integrins through a specific motif (the "KGD motif"), thereby enabling specific cell-cell as well as cell-matrix interactions. Thrombospondin-4 deficiency causes dysfunctional integrin signalling and a disruption of the laminin network at the MTJ. Additionally, the lack of thrombospondin-4 caused muscle detachment from the tendon tissue, suggesting that this protein is a central element in structural and functional integrity, at least in the embryonic stage [33]. The role of thrombospondin-4 is unknown in the human adult MTJ and it is not known whether this molecule is load sensitive and involved in MTJ regeneration after injury.

Understanding of the complexity and the steps involved in the development of MTJ is essential in order to acknowledge the processes required for successful regeneration of the junction after HSIs. Although there is still much unknown about MTJ development, it is obvious that the communication between muscle and tendon cells is fundamental for the development of both tissues and the organisation of the linkage between the different tissues. A good example illustrating the dependency of the several tissues on each other is an avian animal model, in which muscleless wings were produced. In these animals, tendons formed early during development, but did not mature to form individual tendons and were subsequently degraded [44]. Thus, the proximity of muscle and tendon cells is crucial for correct MTJ formation and function. Although speculative, in the context of tissue repair after a strain injury, the separation of muscle and tendon caused by both granulation and scar tissue might hinder the propagation of signals from both tissues and complicate regeneration. MTJ regeneration therefore depends on the cells residing in skeletal muscle as well as cells within the tendon.

Importantly, the MTJ appears to be a very active region, and at least on the muscle side, adaptations to loading have been shown. A recent study on human MTJ samples reports the presence of multiple muscle fibres with central nuclei and positive immunostaining of CD56 [45]. The number of myofibres at the MTJ with central nuclei was as high as 43–50%, compared to 3% in the resting muscle belly of the vastus lateralis [46]. Both central nuclei and CD56 immunoreactivity (marker expressed by newly formed myofibres [47]) are signs of high cellular activity, and these findings indicate that the human MTJ is characterised by continuous remodelling. The authors further report that the number of fibroblasts at the MTJ was not different from the numbers in the muscle belly, but the activity status of these cells

was not investigated. It should be noted that this study did not find any differences between MTJ samples from individuals subjected to 4 weeks of heavy resistance training targeting the hamstring muscles and a control group with no specific training. Thus, the loading per se did not seem to affect any of the characteristics measured in this study [45]. Human hamstring muscles subjected to both an acute bout of heavy resistance training and training for 4 weeks prior to tissue sampling at the MTJ demonstrated a higher expression of tenascin-C and the fibril-associated collagen type XIV in the muscle connective tissue, but no direct changes of these markers or any other factors analysed in the study were detected at the MTJ [38].

The architecture of the MTJ and potential changes to different types of training or immobilisation have not been examined in the aforementioned studies including human subjects. A study on rats showed, however, that training can cause modifications at the structural level of MTJs. These rats were subjected to 6 weeks of uphill running, and the percentages of branched interdigitations afterwards were significantly higher compared to a sedentary control group. The researchers specify that both the number and the length of the interdigitations increased in the trained compared to the non-running rats [48]. On the contrary, limb unloading led to a substantial decrease in the muscle-tendon interface seen as a substantial decline in the membrane folding relative to muscle fibre cross-sectional area at the MTJs of rats subjected to 4 days of space flight [49]. Interestingly, this study found an increase in fibroblast-like cells in the proximity of the MTJ of unloaded animals compared to control rats [49], suggesting there is a rapid cellular response to hypoactivity. The underlying mechanisms and the role of these cells are unexplored.

Besides a reduction in the protrusions, unloading, through either hind limb suspension or spaceflight, had an adverse effect on the organisation of the MTJ. The tendinous side was characterised by a more disorganised collagen fibril structure and the muscular side showed z-band disorganisation in myofibres [50]. Strikingly, the induction of exercise during unloading prevented the loss of membrane folding and even increased the complexity of the interdigitations [51]. Similar findings to rat immobilisation were demonstrated with MTJ samples obtained from patients after prolonged bed rest. The MTJ endings of these samples obtained from the lower leg showed a reduction in the protrusions compared to control samples obtained from healthy individuals [52]. It is important to note that all of these studies used 2D analyses and the response might be even more pronounced when using 3D image analysis.

2.1.3 Regeneration of Skeletal Muscle and the Connective Tissue

The study of regeneration following muscle strain injuries is complicated as these injuries predominantly occur at the MTJ [2–4]. It is therefore crucial to keep in mind that repair after HSIs does not exclusively involve the regeneration following myofibre necrosis (i.e. myofibre death); it requires the fusion across myofibres and, importantly, includes the attachment of myofibres to the tendon/aponeurosis to

enable force transmission. One major factor driving rehabilitation is the timing of loading. A recent study investigating the effect of early compared to delayed loading after muscle strain injuries showed clearly that a delay in loading of damaged musculoskeletal tissue significantly prolongs return to sport (RTS) [53]. These findings underline the importance of mechanical stimulation of healing tissues and cells across different tissues.

Taking into consideration the animal model of strain injuries, one rather surprising finding is that the skeletal muscle tissue in itself does not appear to suffer from damage [2]. The myofibres adjacent to the rupture site have a regular appearance with an organised Z-line alignment, but the basement membrane is torn off and the connection to the connective tissue (tendon/aponeurosis) is lost [2, 3]. However, it is also important to remember that these findings are based on an animal model, and it is very likely that human hamstring muscle strains, as a result of explosive movements, may cause disruption of the MTJ concomitant with myofibre damage. The adverse impact of strain injuries on the muscle has been clearly demonstrated by studies reporting substantial muscle atrophy as a result of strain injuries [54, 55]. This could be due to poor myofibre regeneration, but also reflect the failed repair at the MTJ, meaning that the tight link between the muscle and the connective tissue/tendon/ aponeurosis may not fully reform after a strain injury. A lack of the firm muscle-tendon attachment will influence the mechanical properties of myofibres and tendon collagen fibrils. Further, the neuromuscular innervation might be negatively affected and thereby involved in the adverse long-term outcome of strain injuries on the involved muscle.

Skeletal muscle tissue has powerful regenerative potential, which relies on the activation of muscle stem cells, also known as satellite cells, due to their sublaminar location and association with the plasma membrane [56–58] (Fig. 2.4).

At steady state, the satellite cells are mitotically quiescent (G0 phase, meaning that these cells have reversibly left the cell cycle and do not divide), and they become activated through signals from a diseased or damaged environment. Activated (proliferating) satellite cells are referred to as myogenic precursor cells [59]. Strikingly, satellite cells can become stimulated even though they are located at the other end

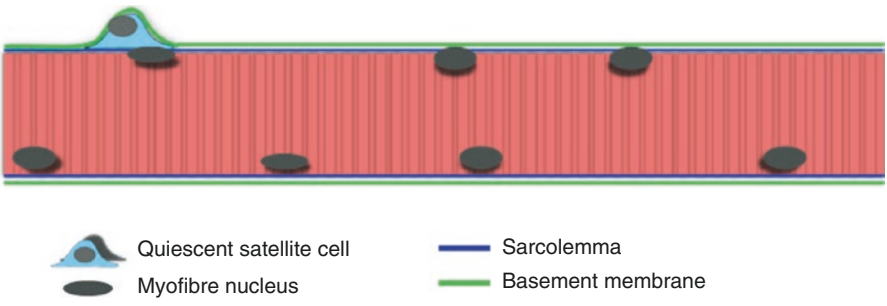

Fig. 2.4 Schematic drawing of a longitudinal myofibre illustrating the sarcolemma (plasma membrane) and the basement membrane along with a satellite cell and several myonuclei. (Reproduced with adaptation from Fig. 15 of Mackey and Kjaer [58])

of a damaged myofibre [60]. Satellite cells are also motile, with an ability to migrate across basement membrane layers [61]. Within hours, several key myogenic factors (such as MyoD, desmin, myogenin) are expressed, and after a few cycles of cell proliferation, the majority of the myogenic precursor cells enter the myogenic differentiation programme. Following exit from the cell cycle, myogenic cells fuse with damaged myofibres or fuse with each other to develop nascent multinucleated myofibres. To avoid depletion of the satellite cell pool, satellite cells have the ability to self-renew by either asymmetric or symmetric division [59].

It is interesting to note that different types of muscles are associated with inherent differences in the activation and differentiation potential of satellite cell subpopulations [62]. The regenerative potential of satellite cells residing in the human hamstring muscles have been investigated far less extensively compared to the quadriceps muscles; therefore, the myogenic repair potential in human hamstring muscles remains somewhat unexplored. A study including both hamstring and calf strain injuries found no differences between the muscle groups in functional or structural recovery after strain injuries [54]. These findings argue against a poor myogenic potential of hamstring muscles. Additionally, when the number of satellite cells was determined in human tissue samples from the hamstring muscles, the number of 0.12 satellite cells/fibre reported is comparable to ratios calculated in other muscles [45]. Therefore, it is more likely that the nature of the strain injury and the different tissues affected prolong or even impede complete tissue healing.

It is noteworthy that the ECM appears to play a key role in the repair of myofibres after injury. This hypothesis is supported by several studies on focal muscle damage, showing that necrotic ("dying") myofibres require an existing basement membrane [58, 63]. This means that the matrix encasing the myofibres serves as a scaffold and orientation/ guidance for satellite cells to restore damaged myofibres [58] (Fig. 2.5). Thus, the preservation of the basement membrane is a central element in successfully regulating the regeneration after injury.

Experimental animal data indicate that the basement membrane after a strain injury is not retained [3, 64], which probably hampers or even prevents complete repair of damaged muscle tissue as a result of strain injuries. In animal models in which the muscle, along with the basement membrane, was severely damaged, the injured skeletal muscle forms a nonfunctional scar tissue between the ruptured skeletal muscle fibres and the regenerating myofibres [15, 65]. The newly formed myofibres were poor at expanding within the granulation tissue and it can be hypothesised that the damage to the basement membrane scaffold hindered myofibre expansion. These animal models report a failed repair process in which the ruptured skeletal muscle fibres remain separated by the scar. As the defect was still evident 12 months after the trauma, the observation strongly indicates that the scar is permanent.

It is interesting that the integrin subtype $\alpha_7\beta_1$ (Fig. 2.1) appears to play an important role also in the regeneration of myofibres following the rat model of complete muscle transection [12]. There was both an increase in the expression of integrin $\alpha_7\beta_1$ within the regenerating muscle fibres and a dynamic redistribution during regeneration [12]. However, the presence of an intact basement membrane is also required to establish firm adhesions between the intracellular cytoskeleton and the

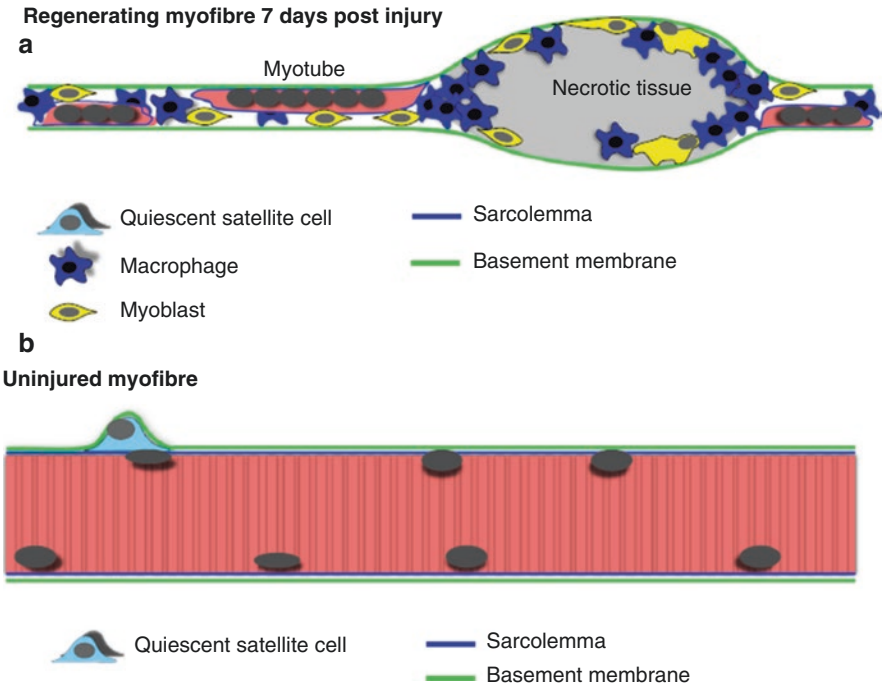

Fig. 2.5 Schematic drawing of a regenerating myofibre after experimentally induced muscle damage. (**a**) shows the regenerating myofibre with the basement membrane as a scaffold/orientation for inflammatory cells and myoblasts during repair. Note that the fibre is devoid of a sarcolemma. (**b**) Illustrates an intact myofibre for comparison. Both sarcolemma and the basement membrane are present. (Reproduced with adaptation from Fig. 15 of Mackey and Kjaer [58])

matrix. Another interesting result was that the redistribution of integrin $\alpha_7\beta_1$ to the lateral sarcolemma during skeletal muscle tissue repair only occurred if the injured muscle was mechanically stimulated [10, 12, 66]. This observation suggests the requirement for injured tissue to be put under loading and could be one part of the explanation why athletes who commence rehabilitation early after injury recover faster compared to those who have a period of rest [53]. Given the crucial role of integrin $\alpha_7\beta_1$ in MTJ development, it seems plausible that this integrin subtype is involved not only in myofibre regeneration but also MTJ repair. It is, however, unknown how integrin $\alpha_7\beta_1$ binding is re-established when the basement membrane is absent as demonstrated in muscle tissue put under excessive strain.

In relation to the regenerative capacities of the human MTJs in hamstring muscles, none of the key factors involved in MTJ development have been investigated following HSIs. This means that following hamstring strains the sequence of expression and the localisation of key molecules, such as integrin $\alpha_7\beta_1$, COLXXII, tenascin-C, and thrombospondin-4, are unknown. Moreover, there is a lack of studies on the multiple cellular components at the MTJs and their interplay after an injury. Finally, it has remained elusive whether the characteristic junctional organisation of

complex ridgelike tendon protrusions into the muscle tissue is re-established after disruption near or at the MTJ.

The reconnection of myofibre-connective tissue junction and the reformation of the specialised architecture at the MTJ to withstand the high stress put on the tissue probably pose the greatest challenge in repair after strain injuries. The basement membrane of myofibres emphasises the role of the matrix in repair, and it can further be hypothesised that the tendon matrix on the tendinous side of the MTJ limits regeneration. Recently, the intramuscular tendon of the BF_{LH} has gained increased attention in the clinic as time until successful RTS was substantially prolonged when the intramuscular tendon was injured [7]. Additionally, the risk of recurrence might be increased when this tendinous structure is affected, although there are conflicting findings about the association between the intramuscular tendon and the re-injury risk [7, 67]. These findings support the idea that the connective tissue and its regenerative capacities play a decisive role in the repair of strain injuries and the severity of HSI is greatly aggravated when a larger part of the connective tissue structures is involved.

Unlike skeletal muscle, human tendons do not have great regenerative potential. Human tendon tissue has a very slow turnover [68, 69] and tendons contain a low number of cells [70]. Acute tendon ruptures show ongoing signs of repair up to 1 year after the trauma [71] and chronic overuse injuries of tendons cause symptoms and inferior tendon function for a long time, in some cases several years [72, 73]. Cells residing in tendons are generally described as fibroblasts, which are non-haematopoietic, non-epithelial, non-endothelial cells. These cells are arranged in between collagen fibrils along the direction of strain and have an elongated morphology. Tendon fibroblasts are also found in the interfascicular space [74].

Even though tendon fibroblasts are presumably terminally differentiated cells, they still can adapt to the environment, e.g. when mechanical stimuli are withdrawn: when placed in an unloaded environment, tendon fibroblasts shift towards an inflammatory phenotype [75]. These findings are supported in similar experiments which showed that unloading causes catabolic (i.e. negative) adaptations [76, 77]. These findings may have clinical implications when it comes to regeneration at the MTJ after HSIs. As the tendon/aponeurosis is supposedly being torn off from the muscle in a strain injury [2, 3], the collagen fibrils as the basic element of tendon/aponeurosis will become unloaded and influence cellular processes. Thus, a potential shift towards an inflammatory, catabolic phenotype has potentially a dramatic negative impact on the capacity of the tendon/aponeurosis to activate a repair process following muscle strain injuries [64]. It should be noted that stem cells have been identified in adult tendons and that these cells would also require the proper mechanical stimuli, which are presumably not present after a traumatic strain injury [33]. In general, whether and how tendon stem cells are activated in response to human musculoskeletal injuries remains unknown.

Besides the low number of cells present in the tendon, another factor contributing to the limited connective tissue repair might be the poor vascularisation of tendon tissue [78, 79]. Revascularisation of the injured area is a vital process in tissue regeneration; it is one of the first signs of regeneration and a prerequisite for

subsequent morphological and functional recovery of the injured skeletal muscle and connective tissue [80, 81]. As the MTJ is a key area through which blood supply to the tendon is provided [78], the restoration of vasculature after a strain injury in this anatomical area is crucial.

Although there are no studies investigating the impact of hypoxia following hamstring strains, intra- or intermuscular hematoma formation [17, 54, 82, 83] is a sign that there is significantly impaired oxygen and nutrient supply. In addition, recent samples of muscle strain hematoma in athletes showed a substantial release of the pro-angiogenic factor vascular endothelial growth factor-A (VEGF-A) over a prolonged period post injury [84]. The growth factor VEGF-A represents a key element in angiogenesis and is one of the target genes of the major transcription factor induced by hypoxia, hypoxia-inducible factor-1α. New formation of capillaries provides the regenerating area with an adequate supply of oxygen and nutrients, which are necessary for energy metabolism for the regenerating myofibres [80] and cells residing at the tendinous part. Whether the vascular supply is completely restored at the injured MTJ remains unknown, as whether the vascularisation of the tendon through the MTJ occurs following this type of injury has not been explored. A recent examination of tissue perfusion following muscle strain injuries reported that there is an increased tissue perfusion for at least up to 6 months post injury [54]. Interestingly, early mobilisation following a crush injury in rats had a positive effect on sprouting of capillaries [80], which supports the recommendation of early loading after injuries [53]. It should be, however, remembered that the response regarding neovascularisation after a HSI might be very different to capillary growth after mid-belly contusion.

Successful regeneration would mean a replication of developmental stages of MTJ formation and data are scarce on this topic, especially in relation to the human MTJ. It is complicated by ethical and anatomical considerations, as it is questionable whether repeated tissue samples should be obtained from patients after a strain injury. Additionally, the MTJ is a very discrete area in the musculoskeletal system, and it would require elaborate equipment and techniques to obtain representative tissue samples.

Finally, another important aspect of regeneration following HSIs is innervation. Even though there is a lack of data on the human neuromuscular junction (NMJ) in general, and the extent to which innervation and NMJs are affected in HSIs, weakness along hamstring muscle length and reduced muscle activation are reported [85, 86]. These observations suggest that neuromuscular adaptations following hamstring strains could be negatively affected. In an animal model applying complete transection of the rat extensor digitorum muscle, the denervated, severed muscle stumps become reinnervated via penetration of new axon sprouting through the connective tissue scar and the formation of the new NMJs [87]. The presence and distribution of NMJs at the interface between the muscle and the tendon/aponeurosis in the human hamstring complex has remained unexplored, and it is therefore difficult to speculate how neural innervation, in general, and the NMJs, in particular, are affected by strain injuries. It is, however, obvious that the presence of a fully restored basement membrane is a prerequisite for functional NMJs [88].

In summary, a hamstring strain is a complex traumatic injury, which does not only affect skeletal muscle but also, and probably even more, the connective tissue in the muscle and the attached tendon/aponeurosis. In the vast majority of strain injuries, the MTJ is damaged, a tissue junction, which is designed to withstand high loads. During development, the coordinated signalling from both muscle and tendon cells ensures MTJ formation. Loading of the MTJ appears vital for maintaining its unique structure and, most likely, its function. The adult human MTJ is a very active region with significant potential for remodelling. The regeneration of the MTJ in athletes suffering from a hamstring strain has remained largely unknown; from a clinical standpoint it is obvious that the greater involvement of connective tissue (i.e. the central tendon) complicates healing. This emphasises the importance of focusing rehabilitation techniques on both muscle and connective tissue stimulation. As immobilisation of the MTJ has detrimental effects, early loading onset after the hamstring injury probably contributes to improving stability of damaged MTJ components post injury.

2.2 Hamstring Injury Sequelae

2.2.1 Structure and Cellular Components of Scar Tissue

Subsequent episodes of pain and re-injury following hamstring strains are frequent and mainly affect the same region as the index injury [89, 90]. Despite the high prevalence, the underlying causes and mechanisms behind the recurrent injuries have not been extensively studied in sports medicine. As re-injuries mostly happen within the first year, and in more than half of these injuries, within the first 25 days after the index injury [90], it can be hypothesised that (1) the repair after the HSI is not completed when the hamstring muscles are fully loaded, or (2) the repaired tissue does not withstand the high loads placed on the injured tissues, or (3) a combination of incomplete healing and mechanically immature repair tissue. In general, there are very few tissues in adult humans that heal by a complete regenerative response, synthesising a tissue identical in structure and function to what it was pre-injury. Mostly, damaged tissue is replaced by structurally and functionally inferior material.

Strain injuries are associated with the formation of scar tissue [55, 91]. Generally, the development of scars refers to the formation of excess fibrous connective tissue in a tissue or organ as a result of prolonged reparative or reactive processes [92]. In the context of HSIs, the formation of scar tissue might hamper the cross-talk between muscle and tendon cells and hypothetically cause the muscle or, more precisely, the myofibres to be permanently disintegrated from the normally attached tendon/aponeurosis. Whether this separation is transient or permanent is currently not known, but there is accumulating evidence that fibrotic tissue is a long-term pathological outcome following muscle strain injuries. Animal models also suggest that the scar tissue is permanent [15].

The initial response to a traumatic injury such as muscle strains is the formation of granulation tissue. Granulation tissue can also be viewed as a provisional matrix

comprised of newly formed loose connective tissue filling any gap caused by tissue disruption. It is an evolutionarily conserved process aimed at the reconstitution of tissue integrity promptly after injury [93]. Granulation tissue is rich in the ECM components, fibronectin, tenascin-C, as well as collagen type III, and is mainly synthesised by (myo-)fibroblasts [94–96]. Tenascin-C is deposited early during the healing phase to provide elasticity to the granulation tissue to withstand the strains placed on the transient scaffold [97]. Tenascin-C is furthermore involved in the formation of an adhesive environment, which is favourable to cells and acts as a chemokinetic agent [98]. During optimal repair progression, fibronectin and collagen type III are sequentially replaced by collagen type I [99, 100]. This transition in collagen types improves mechanical stability, to a large extent due to the formation of multiple intrafibrillar cross-links in collagen type I [101]. Regarding very early recurrent hamstring injuries, it can be speculated that the strength of the provisional granulation matrix after the strain injury is not mechanically stable enough to allow for explosive movements when commenced too soon after the injury.

On the cellular level, myofibroblasts are the main cell type in granulation tissue, along with a myriad of other cell types, including inflammatory cells, fibroblasts, endothelial cells, and pericytes. Myofibroblasts belong to a specialised group of fibroblasts and their activation involves multiple factors. The two predominant stimulants are transforming growth factor-β1 (TGF-β1) and high matrix stress/stiffness [102]. Additionally, the presence of a splice variant form of fibronectin, ED-A fibronectin, seems to be required for myofibroblast differentiation [103]. Myofibroblasts express α-smooth muscle actin, and the organisation of α-smooth muscle actin into stress fibre-like bundles provides cytoskeletal characteristics of contractile smooth muscle cells. Thus, myofibroblasts can actively contract and remodel the granulation tissue, a process by which scar tissue is stabilised at least during wound healing after skin lesions [102–104]. How granulation tissue develops into scar tissue and how the scar is resolved following hamstring strains remains unknown. At this point, it should be remembered that a stable and organised ECM is pivotal to withstanding the high mechanical loads, for example, when the MTJ is subjected to explosive movements. Therefore, the sequence of ECM remodelling appears to be a major player in determining successful or ineffective repair. In the context of HSIs, failure to fully repair might be reflected in increased recurrence.

In successful repair, the myofibroblast-driven contraction of granulation tissue halts and tissue integrity is re-established. Further, concomitant processes such as angiogenesis, which is an integral part of the healing process, cease and myofibroblasts become apoptotic resulting in a largely avascular tissue [105]. However, the observation of fibrotic tissue following HSIs suggests that complete tissue restoration is not accomplished. So far, fibrotic tissue resulting from hamstring strains has only been demonstrated by magnetic resonance imaging (MRI), and therefore, the structure and composition of scars in human athletes remained unexplored. Recently, some insight into the long-term scar tissue has been gained through biopsies obtained from muscle strain injuries, which occurred at least 6 months prior to sampling. These biopsies clearly show high cellularity among disorganised connective tissue, adipocytes interspersed with myofibres, as well as the presence of large blood vessels (Fig. 2.6). The human samples furthermore clearly reveal the absence of any

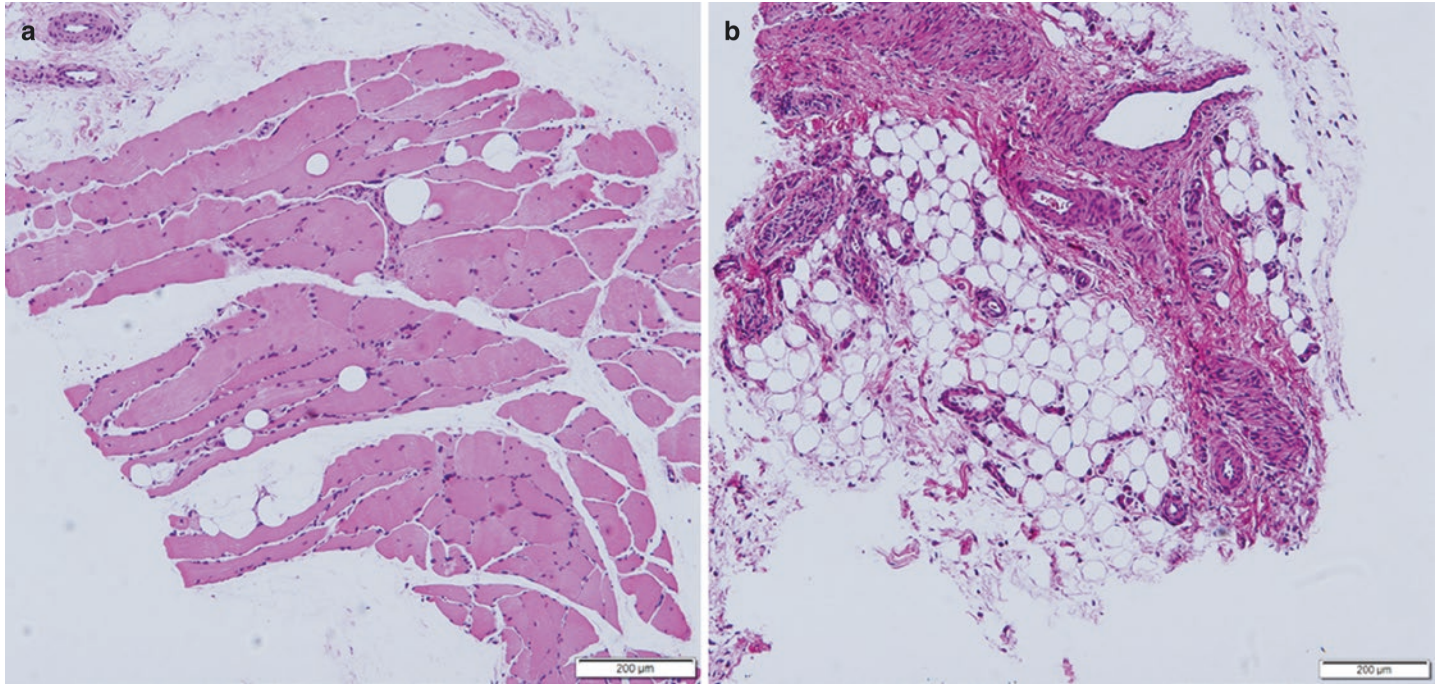

Fig. 2.6 Human biopsy material from strain injuries, haematoxylin-eosin staining. (**a**–**c**) Samples are taken at least 6 months after a strain injury. (**a**) Longitudinal myofibres are interspersed with adipocytes. (**b**) Scar tissue shows disorganised connective tissue, substantial accumulation of adipocytes, and blood vessels [54]

structured interface between myofibres and tendon/aponeurosis. These findings emphasise that scars are in fact highly active regions instead of inert, avascular structures, at least in musculoskeletal tissue, indicating that scar formation and development are tissue dependent. A similar picture was recently gained from the myocardium after a myocardial infarct where the scar is highly populated with cells a long time after the injury with signs of continuous reorganisation of the injured tissue [106].

The question that remains is whether the presence of scar tissue is associated with an increased risk of recurrence. Despite the scarcity in observations related to HSIs, it is fair to say that scar tissue always replaces functional tissue with potential dramatic consequences on function. From other tissues and organs such as the heart or lung, it is known that scars significantly alter cardiac muscle extensibility and impair lung expansion as well as gas diffusion [106–108].

No association between MRI diagnosed fibrosis and recurrent hamstring injuries was reported during a 1-year follow-up period [91]. Interestingly, a study by Silder and colleagues [109] reported higher tissue strains in previously injured hamstrings during lengthening contractions. Their data suggest that the repaired tissue is more compliant compared to healthy uninjured hamstrings and might infer that force transmission at the MTJ in injured hamstring injuries is substantially altered. The presence of scar tissue following a HSI changes the mechanical properties, but whether this has other functional consequences would depend on the size, the maturation, and the organisation of the scar. Additionally, the connection between the myofibres and the scar is a critical factor to the function of the reformed transition area and ultimately the risk of recurrence.

An additional clinical concern is that scar formation at the injury site may result in adverse neural tension. While there is limited research on the topic, a high prevalence of neural tension was reported in a small sample of athletes with prior hamstring strains [110]. Adverse neural tension may contribute to weakness with the hamstrings in a lengthened position [111], and since athletes with prior hamstring injuries are prone to weakness at longer muscle lengths, it may be important to assess for adverse neural tension to optimise treatments [112].

2.2.2 The Inflammation-Fibrosis Link and Its Potential Role in Scar Formation After Strain Injuries

The development of fibrotic tissue is associated with prolonged inflammation [113–117]. It can therefore be anticipated that there is a relationship between inflammatory processes after a HSI and the pathobiological fibrotic changes described in athletes suffering from a hamstring strain [91, 118]. It is, however, important to note that there are no studies investigating this direct relationship. A recent investigation of human muscle strains included a surrogate marker of inflammation and reported long-term increases in perfusion of the injured tissue indicating that inflammation persists for at least 6 months post injury [54].

After a traumatic injury, the inflammatory response often starts with activated platelets during coagulation following the rupture of blood vessels. Activated platelets change shape and secrete the contents of their granules, which involve, among various other factors, cytokines and chemokines to promote activation of inflammatory processes [119, 120]. Further, a major factor driving the sterile (nonpathogenic) inflammatory response is the presence of necrotic ("dying") cells and cell debris belonging to the endogenous damage-associated molecular patterns (DAMPs). DAMPs, which can also be damaged ECM proteins; proteoglycans, which were released from the ECM; and stress-induced proteins are recognised by the innate immune system through cell receptors such as the toll-like receptors, RIG-I-like receptors, NOD-like receptors, and C-type lectin receptors [121, 122]. When danger signals are sensed, intracellular signalling cascades are activated, of which the nuclear factor-κB (NFκB) pathway is considered one of the key activators of pro-inflammatory responses in macrophages, neutrophils, and mast cells [123]. The immediate response to NFκB activation involves the induction of pro-inflammatory cytokines such as IL-6, IL-8, TNF-α, and adhesion molecules [124, 125].

Inflammatory cells multitask at the wound site by facilitating wound debridement and producing chemokines/cytokines, metabolites, and growth factors needed for tissue repair. At the same time, inflammatory cells also release matrix metalloproteinases (MMPs), which are enzymes involved in the degradation of matrix proteins and could therefore contribute to further damage of the ECM [126]. While research on mechanisms associated with acute pro-inflammatory processes has been extensive, there is still much unknown about the resolution of inflammation and concomitant tissue remodelling, at least for musculoskeletal injuries. It is, however, obvious that failed resolution of inflammation is linked to tissue hyperplasia and scar formation [127, 128]. Resolution of inflammation is tightly associated with the function of anti-inflammatory macrophages and the factors that these cells synthesise and release. Additionally, the termination of the acute inflammatory response is regulated by active processes synthesising endogenous lipid factors that are both anti-inflammatory and pro-resolving [129, 130]. Resolution is therefore an active rather than a passive transition which affects inflammatory cells but also non-myeloid cells.

Monocytes can adopt very different phenotypes, crudely divided into "pro-inflammatory" and "anti-inflammatory" macrophages. Readers should, however, keep in mind that the separation into "pro"- and "anti"-inflammatory macrophages represent only two phenotypes in a wide and still evolving spectrum of macrophage polarisation. Pro-inflammatory macrophages are induced by cytokines interferon-γ and IL-1β and release high amounts of the pro-inflammatory cytokines TNF-α, IL-1β, IL-6, IL-12, and IL-23, as well as with reactive oxygen species (ROS) and nitric oxide (NO) [125, 131, 132]. The pro-inflammatory macrophages are mainly involved in phagocytosis of necrotic cells, whereas anti-inflammatory macrophages are prominent regulators of tissue repair and regeneration and thereby linked to inflammation resolution, tissue remodelling, and angiogenesis. This set of macrophages is stimulated by IL-4 and IL-13 and produce IL-10 as well as the TGF-β1 [125, 132–134].

Inferior tissue healing is associated with the presence of anti-inflammatory macrophages, which continue to synthesise and secrete growth factors [125]. A key growth factor in this context is TGF-β1 which is pivotal in the activation of myofibroblasts, a prominent cell type modulating the formation of fibrotic tissue [102, 135] (see also Sect. 2.2.1, page 45). Further, TGF-β1 stimulates the synthesis of ECM proteins [136, 137] and might thereby contribute to the accumulation of excessive connective tissue. Additionally, macrophages are sources of transglutaminases, which are enzymes involved in collagen cross-linking [116], and macrophages themselves play a major role in activating the fibroblast-to-myofibroblast transition [138].

It might therefore appear beneficial to dampen the inflammatory processes after injuries such as hamstring strains, but it is important to remember that interfering with inflammatory processes at any stage will not necessarily lead to improved tissue healing. The role of inflammation on healing of the MTJ after HSIs is unknown, but in skeletal muscle repair, pro-inflammatory macrophages stimulate the proliferation of myogenic cells besides their role in phagocytosis of damaged myofibres [139]. The next sequence in muscle repair is the skewing of pro-inflammatory to anti-inflammatory macrophages which stimulate the myogenic cells to fuse to become new myofibres [140]. Interfering with either the pro-inflammatory or the anti-inflammatory cascade leads to impaired tissue regeneration, emphasising the importance of an orchestrated inflammatory process [141, 142]. Also, in relation to connective tissue, the interference with the anti-inflammatory pathway has adverse effects. Whereas the deletion of a major anti-inflammatory factor (IL-10) speeded up skin wound healing, the long-term response was the development of a disorganised matrix with excessive collagen deposition [128]. In another study, the deletion of IL-4 receptor α, a major factor involved in anti-inflammatory macrophage activation, led to impaired wound healing as a result of a failure of macrophages to initiate successful repair [143]. Another adverse outcome to the inhibition of inflammation was also described in tendon ruptures, where treatment in the very early phase after the trauma resulted in impaired mechanical properties. Noteworthy, dampening of inflammation at a later time point improved material properties [144].

However, the suppression of inflammation following hamstring injuries might be adjuvant to the recovery and the reduction of scar formation, in particular since tissue resident cells can become activated by persistent inflammation [145] and change their cellular behaviour [146, 147]. But given the fact that inflammation and the resolution thereof are considerably complex, it is difficult to determine a certain time frame, during which it would be beneficial to blunt inflammatory agents.

In conclusion, scar formation following traumatic injuries is a pathobiological consequence in many tissues. Following hamstring strains, scars have been visualised and tissue samples suggest that the fibrotic regions are characterised by high a cell number and disorganised connective tissue. Additionally, adipocytes accumulate at the injured site, a process which might contribute significantly to changes in mechanical properties post injury. Observations reporting altered tissue strain after hamstring injuries in human subjects have been made, but there is little data in this field. Scar formation involves a myriad of cellular processes, and it is tightly

coupled to persistent inflammation. Whether this is the case in HSIs remains speculative. An important yet under-researched area in the musculoskeletal field is the effect of mechanical loading on the structure and function of scar tissue. One perspective of early onset of loading followed by appropriate load progression might be to stimulate the granulation tissue and later scar tissue optimally to improve strength and stability of the repair tissue.

2.3 Proximal Hamstring Tendinopathy

2.3.1 Pathological Changes in Tendinopathy

Overuse of hamstring tendons is, in most cases, confined to the origin of the hamstring tendons, i.e. the proximal site. Therefore, this paragraph will only discuss the proximal hamstring tendinopathy (PHT). The reader should note that the literature is very limited, and thus, studies of tendons other than PHT are discussed. Tendinopathy is an umbrella term for non-rupture tendon overuse injuries, which cause symptoms such as soreness, pain, swelling, and dysfunction. In contrast to acute, traumatic injuries such as the hamstring muscle strain, tendinopathy is a chronic condition with a gradual onset. Tendinopathy is very common, but the injury aetiology has remained somewhat elusive. The development of tendinopathies is associated with repetitive exposure to both a magnitude and volume of loading that exceed the physiological capacity of the tendon and can be viewed as a repeated disturbance of tendon homeostasis [73]. In other words, overuse injuries emerge as a result of the inability of tendons to keep up with the synthesis of key components constituting the tendon matrix. A fundamental question that has remained unanswered is what defines "healthy" loading leading to tendon adaptations and "excessive" loading resulting in degeneration, pain, and functional impairment. Similar to tendinopathies in other anatomical sites, the PHT appears to be caused by overuse as hardly any non-athletes suffer from chronic injuries of hamstring tendons [148]. Benazzo and colleagues reported that approximately 50% of injured hamstring tendons affect the biceps femoris tendon, 30% the semimembranosus, while the ST appears to be the least affected [149].

Knowledge of structural features of human tendinopathic tendons at the tissue level is predominantly based on samples obtained from chronically injured patella or Achilles tendons [150–153]. Characteristics of chronic histopathology include disorganised collagen fibres, an increased amount of blood vessels, and ingrowth of sensory nerves [154–157]. Other histopathological signs of tendinopathic samples show an increase in collagen type III, areas devoid of cells [158] indicating cell death and/or cell migration towards other parts of the tendon, as well as areas of hypercellularity [159]. Fibroblasts in healthy tendon have long extensions projecting into the matrix and through which these cells can communicate with each other. These cellular extensions are absent in tendinopathic tendons, and further, cells lose their tight contact with the ECM. This means that the tight link between the cell and the matrix is disturbed. Lastly, instead of longitudinally extended nuclei which are

aligned along the axis of tension, tendon cells in chronically injured tendons have misshaped, more rounded cell nuclei [151].

Histological analysis of samples obtained during surgery for PHT revealed very similar characteristics compared with the aforementioned findings in the Achilles or patella tendons. Samples were collected from human patients with a gradual onset of symptoms at the proximal hamstring origin [160]. These tendons revealed a disorganised collagen matrix, a rounded shape of cell nuclei, and an increase in blood vessels. Further, an increase in mucin (heavily glycosylated proteins) ground substance was seen along with some adipocytes within the tendon matrix. None of these pathological signs were observed in the healthy hamstring tendon control sample [160].

In an early model presented by Gross [161], repeated cycles of injury, inflammation, and repair are suggested to result in the development of poor-quality tissue with inferior mechanical properties. Whether inflammatory events are involved in the development of tendinopathy is a matter of ongoing discussions, but there is accumulating evidence that inflammation plays a role in the early stages of tendinopathy [162]. It is interesting to note that there seems to be an association between the increased number of inflammatory cells and enhanced fibroblast cellularity in early tendinopathic tendon [162]. This suggests an interplay between several cell types in the early events of tendon overuse injury. It is also important to keep in mind that the tendon resident fibroblasts can adopt an inflammatory phenotype depending on the biomechanical environment [75].

Catabolic events that might be involved in the development of tendinopathy include MMPs which digest and degrade connective tissues. These enzymes are separated into four clusters based on their substrate affinity. One important group of MMPs in tendon disorders is the collagenases, i.e. MMP1, MMP8, and MMP13, which degrade fibrillar collagen and are responsible for the balance of collagen synthesis and degradation. Important to keep in mind is that MMP activity is the complex product of synthesis, activation, inhibition, and degradation; an upregulation does not necessarily translate into higher activity. In tendinopathic tendons, MMP1 and MMP13 were found to be upregulated [163], suggesting that there is an increase in remodelling of the tissue. This is supported by a recent study reporting a higher collagen turnover in tendinopathic tissue compared to healthy control tissue [164]. Whether this shift towards more rapid collagen remodelling is also evident in PHT is unknown, yet very likely.

In the more chronic state, tendinopathy has been described as a degenerative process devoid of inflammation. This long-held theory has recently been challenged as inflammatory cells were found in samples from chronic tendon disorders. These cells revealed a complex inflammation signature, which involves the pro-inflammatory interferon, NF-κB, STAT-6, and the glucocorticoid receptor pathways. At the same time, cells in tendinopathic tendons express markers such as CD206 and CD163, which are linked to the alternative inflammatory pathway, suggesting chronic inflammation and ongoing repair processes [146]. Inflammatory cells were also found in chronic Achilles tendinopathies, but it should be noted that healthy control samples also revealed the presence of inflammatory cells such as

CD3-positive T-lymphocytes, CD56-positive natural killer cells, and mast cells [152]. In healthy tendons, the number of the inflammatory cells was, however, lower compared to the chronically injured samples. Another study on chronic tendinopathy showed an increase in members of the IL-6 family, indicating ongoing inflammatory processes, but strikingly, the increase in IL-6 was only detectable in the chronically injured Achilles tendon and not the posterior tibialis tendon [165]. This suggests that there might be differences in cellular responses depending on the tendon. Whether or not inflammatory cells play a role in PHT is unknown due to the lack of data on inflammation in PHT.

2.3.2 Cellular Adaptations to Loading and Unloading in Tendon: How Is Mechanical Loading of Tendons Associated with Healing Processes?

Rehabilitation of chronic tendon injuries involves loading-based interventions, in most cases slow and heavy resistance training. These rehabilitative measures have been shown to alleviate pain and promote tissue healing [150, 166]. In particular, regular eccentric training with high loads has been associated with improvements of tendinopathic tendons [167]. Also for PHT, recommendations include eccentric hamstring strengthening [148]. There is, however, very little research on the effectiveness of loading regimes and PHT. Although the eccentric strength training has been viewed as the treatment of choice, other rehabilitation regimes such as heavy slow resistance training with both the concentric and the eccentric phase result in pain reduction and improved function [166, 168], although not specifically for PHT. Further, static (isometric) training has been suggested to improve chronically injured tendons; with greater acute pain reduction after isometric exercises than that after isotonic exercises [169, 170].

Strength training not only improves symptoms of tendinopathic tendons, but also on a structural level, slow, heavy resistance training led to a normalisation of collagen fibril distribution in human samples obtained from tendinopathic patellar tendons [150]. The cellular mechanisms underlying the positive adaptations to heavy loading have remained elusive, but it is important to note that mechanical loading in general is essential for tendon development, homeostasis, and repair.

Short-term strength training of healthy tendon tissue upregulates mRNA for collagen types I and III as well as tendon regulatory factors such as insulin-like growth factor I, TGF-β, and connective tissue growth factor, as well as cross-link forming enzymes. Interestingly, the response was similar regardless of whether the loading regime was based on isometric, concentric, or eccentric muscle contractions [171, 172]. These findings indicate that tendons respond to loading but do not distinguish between different contraction types, which is also supported by another study reporting nearly identical anabolic responses to any of the contraction modes [173]. Whether the picture is similar in human tendinopathic tendon is somewhat unexplored.

The abovementioned studies were performed on rats, and although human tendons show an upregulation of collagen following loading regimes, the response is far more moderate compared to animals [174]. Further, the anabolic response might differ dramatically in tendinopathic tendons, but despite these considerations, it is important to remember that mechanical loading has clear beneficial effects on chronically injured tendons. In tissues other than tendon, mechanical loading of scars can profoundly modify the structure of scar tissue [106, 107]. It is therefore likely that mechanical stimuli can have an impact on collagen fibril structure in tendinopathic tendons including the proximal hamstring tendons.

In the clinic, the beneficial effect of different loading regimes has been repeatedly demonstrated, which raises the question of how tendon fibroblasts in chronically injured tendons sense and translate the mechanical signals to promote tissue healing. One key factor might be the alignment of collagen fibres, as tendon fibroblasts are tightly bound by specific receptors to the collagen matrix. Any change in the organisation of the collagen fibrils inside the tendon will simultaneously affect the tendon cells. This was clearly demonstrated by a dramatic and rapid shift in receptor binding as a result of unloading of a collagen-rich matrices [75, 175]. This means that the cells within the collagen-rich matrix rapidly react to the change in the mechanical environment (i.e. the unloading) and modify the way by which they bind through cell receptors to the matrix.

The strict parallel alignment and the elongated shape of tendon fibroblasts have clearly been shown to promote the expression of tendon cell markers and the expression of collagen type I [176]. Misaligned collagen fibres, random orientation, and the adaptation of a rounded cell shape are features of tendinopathy and have detrimental effects as tendon fibroblasts switch on matrix degradation pathways [177]. Thus, the application of tensile load to the injured tendon through slow muscle contractions might stretch the collagen fibrils and cells and thereby initiate anabolic responses, such as collagen expression, upregulation of integrin receptors, and induction of growth factor signalling [73, 178]. It is important to note that during heavy and slow muscle contraction, the speed of the movement and the magnitude of loading (strain) that the matrix and the cells are subjected to are fundamentally different compared to injury provoking conditions such as running and jumping.

The positive effects of mechanical strain on the degenerative tendon matrix and the tendon fibroblast might reflect the concept put forward by Arnoczky and colleagues [76, 77]. In contrast to the belief that tendinopathy is an overloading of tendon cells, they suggest that pathological changes in tendinopathy are a result of under-stimulation of tendon fibroblasts [77, 179]. An in vitro study on tendons revealed how tendon cells react to stress deprivation; 48 h of unloading caused tendon cells to upregulate MMP13, an enzyme which degrades fibrillar collagen leading to a weakening of the tendon matrix. Unloading for 48 h caused further detachment of the tendon cells from the collagen matrix, which suggests the loss of cell-matrix adhesions [76]. The underlying mechanisms for this hypo-stimulation could be a focal overloading of the tendon matrix, subsequently leading to micro-damage of collagen fibrils and/or a reduction in their stiffness. This change in mechanical properties of the collagen fibrils translates into a reduction of the mechanical load on the tendon cells. Thereby, the

complete linkage from the matrix, through integrin receptors and the cytoskeleton, to the nucleus becomes modified.

It is noteworthy that tendon fibroblasts form a network with neighbouring cells through gap junctions [180, 181], which enable signal propagation from one cell to another. While cell communication is an essential tool for nutrient exchange and signal transduction in homeostatic conditions, the tendon cellular network might enforce local pathobiological cellular changes and thereby affect larger parts of the tendon matrix. During healing, however, this cellular network might be advantageous to propagate positive cellular adaptations in one to other tendon areas.

Another positive effect of load on injured tendons is suggested by the finding that strain protects collagen fibrils from collagen degradation. In an elegant study, a group of researchers showed that mechanical strain preserves collagen fibrils in the presence of MMP8; while unloaded fibrils were readily degraded, the strained fibrils were resistant to degradation for a prolonged time [182]. In tendinopathic tendons, increased amounts of several MMPs were measured and a disorganisation of collagen fibrils was described in several studies, suggesting collagen fibrils that are subjected to reduced strain. This combination might be a vicious circle contributing to enhanced tendon matrix catabolism as the disorganised collagen fibrils are more prone for enzymatic digestion. In the context of tissue repair, the regular application of high tensile load in slow motion might gradually straighten the collagen fibrils and thereby protect the matrix from degradation. Although this idea appears as an attractive mechanism, it should be remembered that it is speculative as there are no data on the strain that collagen fibrils in healthy and injured tendons are subjected to during slow loading.

In the clinical practice, one of the most effective treatment options for tendinopathy is heavy slow loading of the injured tendons, but exactly how the tendon cells and the tendon matrix are stimulated and activate a healing response is unclear. It is, however, obvious that unloading leads to catabolic changes in the tendon, indicating that “rest” periods should be avoided. Although the proximal hamstring tendons have not received much attention in tendon research, it is fair to speculate that loading regimes to treat tendinopathic hamstring tendons are suitable to reduce pain and improve structure of chronically injured hamstring tendons.

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Hamstrings Biomechanics Related to Running

3

Nirav Maniar, Anthony Schache, Bryan Heiderscheit, and David Opar

3.1 Introduction

Hamstring strain injuries (HSIs) occur frequently in sports characterised by high-speed running [1–4]. Subsequently, a thorough understanding of hamstring function during high-speed running may provide clinicians with a better understanding of HSI mechanisms and directly inform injury preventative and rehabilitative interventions. In sports that require high-speed running, this is by far the most frequently reported mechanism of HSI [2, 5–7]. Although there are other commonly reported mechanisms of HSI (e.g. kicking [2] and slow stretching [8, 9]), these mechanisms will not be the focus of this chapter, primarily due to a lack of biomechanical data providing insight into hamstring function during these mechanisms.

The following chapter aims to provide an overview of hamstring function during running, with a particular emphasis on high-speed running. As HSI typically occurs in the biarticular hamstrings (as opposed to the biceps femoris short head (BF_{SH}), a particular focus will be placed on these muscles. After providing a general overview

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of methods to quantify hamstring function, this chapter will describe hamstring function across the running stride cycle. Hamstring function will be described in reference to hamstring muscle activation, kinematics and kinetics. Additionally, key considerations for clinicians will be covered. These considerations include an overview of the effect of prior HSI on hamstring function during running, a brief discussion on the critical point of the running stride cycle where HSI is most likely to occur and an overview of key factors that influence strain of the most vulnerable hamstring muscle (biceps femoris long head [BF_{LH}]) during swing.

3.2 Quantification of Hamstring Function

Hamstring function during running can be quantified in multiple ways. The following section provides a brief overview of some of these methods, with a specific focus on outcome measures that reflect the loads experienced by the hamstrings during running.

3.2.1 Hamstring Activation

Muscle activation involves the measurement of the electrical activity associated with muscle contraction, which usually involves the application of surface electrodes to the skin directly over the target muscle of interest. This process is known as electromyography (EMG). The muscle EMG signal is best used to describe the onset and offset of muscle activation, e.g. with respect to other muscles or with respect to key events in the running stride cycle such as foot strike and toe-off. Whilst greater muscle activation can reflect an increase in muscle force production, the relationship between EMG signal intensity and force is difficult to determine and will be influenced by many factors, especially muscle length and muscle shortening velocity. It is also worth noting that recording EMG signals via surface electrodes can be susceptible to measurement error such as crosstalk, which is the measurement of the electrical activity of any muscle other than the targeted muscle. Due to the proximity of the hamstrings relative to each other, surface EMG can only separate the activation of the medial (semitendinosus (ST) and semimembranosus (SM)) from the lateral (BF_{LH} and BF_{SH}) hamstring group with reasonable confidence.

3.2.2 Hamstring Kinematics

Motion capture experiments have provided much of the current knowledge of hamstring function during running. These laboratory-based experiments typically involved the use of skin surface markers, placed on various anatomical locations of participants. Using multiple specialised cameras, the three-dimensional positions of these markers are tracked whilst the participant performs the required movements.

These data can then be used to calculate motion of the body, including joint angles, velocities and accelerations.

Motion capture data can be input into musculoskeletal models, which contain a detailed representation of the entire skeleton including various muscle-tendon unit (MTU) actuators that are attached to the skeleton at their anatomically correct origin and insertion sites. Such a model allows for direct estimation of the length of the hamstring MTUs during running. MTU length data are typically presented as absolute lengths (in units of metres, centimetres or millimetres) or relative lengths (usually computed as % of the MTU length assumed in upright standing). These data can also be differentiated to compute shortening and lengthening velocities of each MTU, which can be used in conjunction with muscle activation data to determine the contraction modes of each MTU. Outputs from musculoskeletal modelling can also be input into a finite element model that allows for more complex representations of muscle fibre and tendon dynamics, yielding detailed information such as region-specific strain patterns within a given MTU [10, 11].

3.2.3 Hamstring Kinetics

Joint motion data obtained from motion capture experiments can be combined with ground reaction force data (if synchronously collected) and estimates of body segment inertial properties to solve for the generalised forces and moments necessary to cause the observed motion, via a process called inverse dynamics. Since the net joint moments obtained from these calculations are considered to represent the net moment produced 'internally', primarily by muscles, inverse dynamics can provide some indirect insight into hamstring function during running by considering the specific joint moments to which the hamstrings can be expected to provide a dominant contribution (i.e. 'internal' hip extension and knee flexion moments). Nevertheless, one must be cautious about inferring muscle function via this approach, as inverse dynamics yields only the net joint moments, which could theoretically be contributed by many muscles other than the hamstrings. Whilst direct (in vivo) measurement of hamstring muscle kinetics during running cannot be achieved non-invasively, it is possible to provide estimates.

These estimates can be computed via musculoskeletal modelling, provided that each MTU actuator in the model contains representations of properties needed to provide physiologically reasonable estimates of muscle force. Whilst the level of complexity of these models varies, generic properties may include representations of activation-contraction dynamics, whilst specific properties may include representations of force-generating capacity and architectural properties, typically derived from cadaver experiments. Using these muscle models, as well as input experimental data (typically joint angles, ground reaction forces and sometimes EMG), estimates of muscle forces can be predicted using numerical optimisation algorithms. Whilst the detail of this modelling approach is beyond the scope of this chapter, the interested reader is referred to published works to obtain a more comprehensive understanding [12, 13].

Recently, innovative methods are emerging in an attempt to quantify in vivo muscle forces non-invasively [14]. In this work, researchers attached a low-profile tapper device over the distal biceps femoris tendon of two participants performing treadmill running at multiple speeds. The device is capable of measuring shear wave speed, which can be used as an indicator of tendon tensile loading. Whilst this is limited and does not yield direct muscle force estimates (i.e. in Newtons of force), the researchers demonstrated that shear wave speed is related to tendon tensile loading within physiological loads and thus could provide a useful general indicator of muscle force patterns.

3.3 Hamstring Function During Running

For the purposes of this chapter, temporal aspects of running will be described over the 'stride cycle'. The stride cycle refers to the entire sequence of events that occurs between foot strike (i.e. the first point in time the foot contacts the ground, denoted as 0% of the stride cycle) and the subsequent foot strike on the same leg (i.e. 100% of the stride cycle). This method exploits the cyclical nature of running and is commonly employed in running-based studies to compare data across conditions involving contrasting running speeds and stride durations. In the following section, hamstring function during running will be described separately for each of the two primary phases of the stride cycle: stance and swing. The decision to describe the two key phases of the stride cycle separately in this chapter is based on prior convention adopted in the literature and it permits ease of interpretation for the reader. Nevertheless, we do not want this decision to distract the reader. There is only one continuous phase of hamstring activity per stride cycle, as the hamstrings begin activating during the final third of the swing phase and continue activating throughout the stance phase until just after toe-off [15, 16]. Given that the hamstrings begin activating during the swing phase, we have decided to describe hamstring function during swing followed by that during stance.

3.3.1 Swing Phase of the Stride Cycle

The swing phase is defined as the period in which the foot is not in contact with the ground and typically accounts for ~75% of the stride cycle during maximal sprinting [16]. The swing phase is often subdivided into three sub-phases. Early swing occurs between toe-off and maximum knee flexion, mid-swing between maximum knee flexion and maximum hip flexion and late swing between maximal hip flexion and foot strike [17].

3.3.1.1 Hamstring Activation

Both the medial and lateral hamstrings are heavily recruited during the swing phase of running starting from mid-swing onwards (Fig. 3.1) [16, 17]. For both muscle

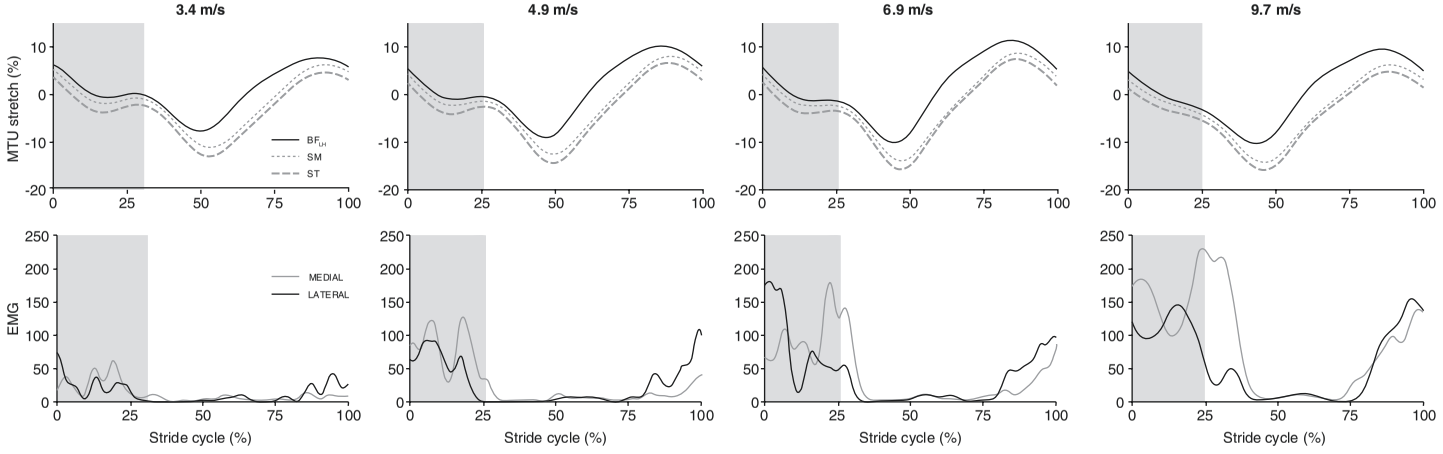

Fig. 3.1 Hamstring musculotendon unit stretch and EMG at various overground running speeds derived from Schache et al. [16]. Musculotendon unit stretch is defined as the percentage change in length from standing upright posture. Electromyography data was normalised to the mean signal obtained across the entire stride cycle for the maximum running speed (for each muscle group). Shaded region represents the stance phase. *MTU* musculotendon unit, *EMG* electromyography, BF_{LH} biceps femoris long head, *SM* semimembranosus, *ST* semitendinosus

groups, the average magnitude of muscle activity appears to increase with running velocity [16, 17]. For example, Higashihara and colleagues [17] showed that average medial and lateral hamstring activity increased 2.5- and 2.9-fold, respectively, during late swing as running velocity progressed from 50% to 95% of maximum. Similarly, Schache and colleagues [16] showed that the average medial and lateral hamstring activity during terminal swing increased 3.5- and 4.4-fold, respectively, as running velocity increases from ~30% to 100% of maximum running velocity. There is also evidence of differences in activation of the medial and lateral hamstrings, and these differences appear to be affected at least to some extent by the sprinting condition, i.e. maximal acceleration sprinting vs. maximal constant-velocity sprinting [18]. The medial hamstrings exhibit greater activation than the lateral hamstrings in both the early swing and the first half of the mid-swing phases in both sprinting conditions [18]. This difference is also evident in the second half of the mid-swing phase for maximal constant-velocity sprinting, but not maximal acceleration sprinting [18].

3.3.1.2 Hamstring Kinematics

During the swing phase, the biarticular hamstring MTUs shorten from toe-off until ~50% of the stride cycle (~33% of swing phase, Fig. 3.1) [15, 16, 19]. After this point, each MTU lengthens until reaching its peak at ~85% of the stride cycle (~60% of swing) and shortens thereafter until foot strike [16, 19, 20]. Given the hamstrings are activating during the mid- and late swing sub-phases, each hamstring MTU is therefore undergoing an active stretch-shortening cycle during this period. The magnitude of this peak MTU stretch increases when running velocity increases from low to high (~30–80%) [16], but is invariant as running speed approaches maximal sprinting (80–100%) [16, 19, 21]. Additionally, the magnitude of the peak MTU stretch during maximal sprinting (Table 3.1) is greatest for the BF_{LH}, followed by the medial hamstrings [15, 16, 19, 22]. Most studies show that peak MTU stretch is greater for SM than ST [15, 16, 19], although the reverse has been reported [22] which is most likely attributable to variability in modelling properties.

3.3.1.3 Hamstring Kinetics

Model-based studies have predicted that peak muscle forces for all of the biarticular hamstrings occurs during the late swing phase of running (~60% of swing or ~85% of stride cycle), regardless of running velocity (Fig. 3.2) [15, 19, 21]. The magnitude, however, is sensitive to running velocity as well as the specific hamstring muscle. As running velocity increases from 80% to 100% of maximal sprinting velocity, hamstring muscle force increases ~1.3-fold [19, 21]. Regardless of running velocity, the SM produces the most force, followed by the BF_{LH} and the ST (Table 3.1) [15, 19, 21, 23]. As each hamstring MTU is also actively lengthening for a certain portion of the late swing sub-phase, the hamstrings perform negative work at this stage of the stride cycle (Fig. 3.2). The magnitude of negative work is also

Table 3.1 Hamstring kinematics and kinetics during the swing phase of maximal sprinting

	Running velocity (m/s)	BF_{LH}	SM	ST
Peak MTU strain (%)[a]				
Schache et al. [16]	9 ± 0.7	11.5 ± 2.5	9.4 ± 14	8.3 ± 1.5
Chumanov et al. [19]	8.0 and 7.1[b]	13 ± 2	11 ± 3	10 ± 3
Thelen et al. 2005 [22]	9.4 and 8.1[b]	9.8 ± 2.8	7.5 ± 1.6	8.3 ± 1.8
Schache et al. [15]	9.0 ± 0.7	12.0 ± 2.6	9.8 ± 1.2	8.7 ± 1.3
Peak force (N/kg)				
Schache et al. [15]	9.0 ± 0.7	26.4 ± 5.2	46.8 ± 6.3	5.5 ± 0.8
Thelen et al. 2005 [23]	9.3	17.6	NR	NR
Chumanov et al. [21]	9.1 ± 6 and 8.2 ± 0.8	21.4 ± 5.4	27.9 ± 7.6	7.9 ± 1.8
Chumanov et al. [19]	8.0 and 7.1[b]	13.2 ± 1.5	23.9 ± 3.5	5.9 ± 1.9
Negative work (J/kg)				
Schache et al. [15]	9.0 ± 0.7	0.3 ± 0.1	1.1 ± 0.3	0.1 ± 0.1
Chumanov et al. [21]	9.1 ± 6 and 8.2 ± 0.8[b]	0.8 ± 0.3	1 ± 0.4	0.4 ± 0.2

BF_{LH} biceps femoris long head, *SM* semimembranosus, *ST* semitendinosus, *MTU* musculotendinous unit, *NR* not reported
[a]Expressed as % of length in upright static standing
[b]Reported as velocities for males and females

related to both running velocity and muscle. The SM produces the greatest amount of negative work, followed by the BF_{LH} and ST (Table 3.1) [15, 21]. As running velocity increases from 80% to 100% of maximal sprinting velocity, the negative work during swing increases 2-fold for the SM, 1.7-fold for the ST and 1.6-fold for the BF_{LH} [21].

3.3.2 Stance Phase of the Stride Cycle

The stance phase is defined as the period in which the foot is in contact with the ground (i.e. from foot strike to toe-off) and typically accounts for ~25% of the full stride cycle during sprinting [16]. Although it is widely believed that HSIs occur during the swing phase, some have suggested that the high ground reaction forces that occur during stance can also cause HSI [24]. Additionally, previous research has shown that hamstring function during stance plays an important role in running performance [25, 26], which can be a key component of HSI rehabilitation progression and return to play (RTP) decisions [27, 28]. Subsequently, an understanding of hamstring function during stance is important for practitioners.

3.3.2.1 Hamstring Activation

Across the stance phase of running, both the medial and lateral hamstring groups continue to activate (Fig. 3.1) [16, 20]. As the hamstrings are considered to be important contributors to forward propulsion of the centre of mass during the stance phase of running [25], it is unsurprising that the magnitude of hamstring activation during stance appears to increase as running velocities progress from low to high

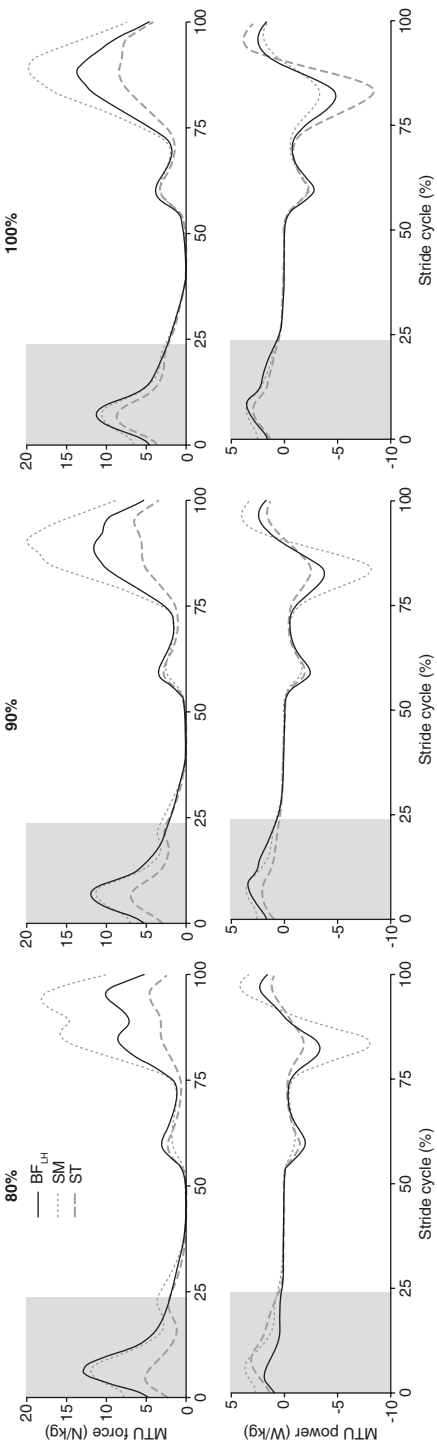

Fig. 3.2 Hamstring musculotendon unit force and power at various treadmill running speeds (expressed as % of maximum running speed) derived from Chumanov et al. [19]. Shaded region represents the stance phase. *MTU* musculotendon unit, *EMG* electromyography, BF_{LH} biceps femoris long head, *SM* semimembranosus, *ST* semitendinosus

[17]. For example, the average lateral and medial hamstring activity during stance increases 2.8- and 4.1-fold, respectively, as running velocity increases from 50% to 95% of maximum speed [17]. Within higher running velocities (≥85% of maximum velocity), mean muscle activity for both hamstring groups remains relatively unchanged during stance [17]. Differences between muscle groups appear to vary across sprinting conditions, i.e. maximal acceleration sprinting vs. constant-velocity sprinting [18]. Lateral hamstring activation is greater than medial hamstring activation in the early stance phase of maximal acceleration sprinting, whereas no differences between muscle groups appear to exist in this phase for maximal constant-velocity sprinting [18]. In contrast, medial hamstring activation exceeds lateral hamstring activation in the late stance phase of maximal constant-velocity sprinting, whereas no differences appear to exist in this phase for maximal acceleration sprinting [18].

3.3.2.2 Hamstring Kinematics

The length of each hamstring MTU during stance is less than that experienced during swing (Fig. 3.1) [15, 16, 19]. Studies have shown that hamstrings' MTU length at initial contact is approximately 5% greater than its length in upright stance [15, 16, 19]. Throughout stance, the MTU length of the biarticular hamstrings progressively shortens such that by toe-off the hamstrings' MTU length is approximately 5% shorter than its length in upright stance [15, 16, 19]. This trend appears to be consistent regardless of running velocity, and similar patterns exist for each of the different biarticular hamstring muscles [16, 19].

3.3.2.3 Hamstring Kinetics

Whilst the hamstrings generate force across the stance phase (Fig. 3.2), the peak force production appears invariant to running speed at higher running velocities (80–100% of max sprinting speed) [19]. Regardless of running velocity, peak MTU forces are greatest for the SM, followed by the BF_{LH} and ST (Table 3.2) [15, 19], similar to what has been found during the late swing phase. As the hamstring MTUs are shortening during this same period, the hamstrings primarily perform positive work [15, 19].

Table 3.2 Hamstring kinematics and kinetics during the stance phase of maximal sprinting

	Running velocity (m/s)	BF_{LH}	SM	ST
Peak force (N/kg)				
Schache et al. [15]	9.0 ± 0.7	4.6 ± 1.0	6.5 ± 1.6	3.6 ± 0.7
Chumanov et al. [19]	8.0 and 7.1[a]	11.6 ± 1.9	12.1 ± 2.4	6.2 ± 2.2
Positive work (J/kg)				
Schache et al. [15]	9.0 ± 0.7	0.05 ± 0.02	0.06 ± 0.02	0.04 ± 0.02

BF_{LH} biceps femoris long head, *SM* Semimembranosus, *ST* Semitendinosus
[a]Reported as velocities for males and females, respectively

3.4 Effect of Prior Injury on Hamstring Function During Running

Although this chapter has described 'typical' hamstring function during running, it is important to recognise that some of these observations appear to be different in individuals with a history of HSI. It is well known that residual deficits in hamstring strength and flexibility persist well beyond apparent 'successful' RTP following HSI [29]. As running ability is an important component of rehabilitation progression [28] and RTP decisions [27], understanding residual deficits in hamstring function during running is also warranted. Although available data on this topic are limited and often heterogeneous, a brief overview is provided below. To explore this issue, some studies have specifically targeted participants with a history of unilateral hamstring injury and thus compared the previously injured side to the contralateral injury-free side. Other studies have adopted a between-subjects design, comparing people with a past history of hamstring injury to a matched group who have never previously sustained a hamstring injury.

3.4.1 Muscle Activation

It is unclear whether the hamstrings of previously injured legs exhibit altered muscle activation patterns during running. One investigation involving participants with prior unilateral HSI found no differences in the magnitude, onset time, offset time or duration of medial or lateral hamstring EMG activity at running velocities of 60%, 80%, 90% or 100% of maximum compared to the contralateral uninjured leg [30]. However, the lack of observed differences may be nullified to some extent by normalising the EMG data to the maximum value obtained by the same (injured) muscle. Another study instead normalised hamstring EMG to values obtained from other uninjured muscles during treadmill running at 20 km/hr [31]. This study found a lower magnitude of lateral hamstring EMG ratios (along with the ipsilateral gluteus maximus, erector spinae, external oblique and contralateral rectus femoris) during the late swing phase in the injured leg compared to the uninjured control group.

3.4.2 Kinematics

Several studies have compared joint or hamstring MTU kinematics during running in unilaterally injured participants to their contralateral uninjured leg [30–32]. In an investigation of treadmill running at 80% of maximal velocity, Lee and colleagues [32] observed a lower peak hip flexion angle in previously injured legs during TU late swing. This decreased hip flexion was thought to be a strategy to reduce MTU stretch in the injured muscle group. However, in contrast, Silder et al. (2010) did not observe any between-leg differences in BF_{LH} stretch when

investigating previously injured participants running at velocities of 60–100% of maximum [30]. Finally, Daly et al. (2016) collected joint kinematics during treadmill running at a steady-state speed of 20 km/hr from a previously injured group of athletes and a group who had never suffered a hamstring injury. These authors reported greater asymmetries in previously injured participants compared to uninjured participants favouring increased peak hip flexion angles, as well as increased anterior pelvic tilt and internal tibial rotation during late swing in previously injured legs [31]. These results implied that the previously injured athletes put their hamstrings in a more lengthened position during late swing, thus opposite to the findings from Lee and colleagues [32]. When results from all studies are considered together, no systematic findings regarding the effect of prior HSI on hamstring kinematics during running are evident.

3.4.3 Kinetics

Although no studies have estimated hamstring muscle forces in participants with a history of HSI, one study [32] provided some insight into hamstring muscle force production through the evaluation of the net hip extension and knee flexion joint moments during running. This study found no differences in lower limb joint moments between the injured and contralateral uninjured legs when running at 80% of maximum sprinting velocity.

Another way to grossly infer biomechanical load on the hamstrings is through the evaluation of horizontal ground reaction force production, as the hamstrings are considered to be a key contributor to the forward propulsion of the body's centre of mass during stance [25, 26]. During non-motorised treadmill sprinting at 80% of maximum sprinting velocity, previously injured legs have been shown to display substantial deficits in maximal horizontal ground reaction force production compared to the uninjured contralateral leg and an uninjured control group [33]. However, a similar study failed to replicate these findings in maximal effort non-motorised treadmill sprinting [34]. Results from a third study [35] suggest that deficits in horizontal ground reaction force production exist during maximal velocity overground sprinting at the time of RTP, but tend to resolve within 10 weeks post RTP. Further to this, when performing ten maximal effort sprints (6 seconds each) on a non-motorised treadmill, the decrement in horizontal ground reaction force production between the first and tenth sprint has been shown to be significantly greater in previously injured legs compared to the contralateral uninjured leg and an uninjured control group [36].

Whilst some emerging evidence is available that horizontal ground reaction force production may be reduced following hamstring injury, further research is required to fully elucidate the exact function of hamstrings during the stance phase of running and whether or not a reduction in horizontal ground reaction force for the recently injured limb is a valid indicator of a persisting deficit in hamstring performance and thus a potential warning sign of likelihood for re-injury.

3.5 When Is the Critical Point in the Running Stride Cycle Where the Hamstrings Are Most Vulnerable to Injury?

Muscle strain injury is most likely limited to periods of stride cycle when hamstrings are highly activated and thus the muscle-tendon junction is subjected to high tensile loads, which based on EMG recordings is during late swing and stance. As previously documented, each hamstring MTU undergoes an active stretch-shortening cycle during late swing; hence this time of the stride cycle has been identified as a potential critical time point for injury. Circumstantial evidence is available from two case studies [37, 38], both of which suggest that the onset of injury occurred during the late swing phase.

Alternatively, early stance has also been proposed as a potential critical time point for injury, based on the proposed role of the hamstrings as a key contributor to forward propulsion of the body's centre of mass at this time [25, 26, 39]. Evidence of potentially high loads being imparted onto the hamstrings during early stance has been provided by some inverse dynamics-based studies [40, 41]. Specifically, for a brief period immediately following foot contact, the ground reaction force may pass in front of the knee joint thereby creating an 'external' extension moment at the knee which will be directly opposed by the hamstring muscles. Nevertheless, the presence of this specific joint moment in sprinting remains somewhat controversial, because it could simply be a by-product of a mismatch in cut-off frequencies when digitally filtering the kinematic and ground reaction force data [42].

Ongoing debate on this issue persists in the literature [43–46]. Whilst further research on this topic is warranted, ultimately it may simply be an academic argument. The critical point in the stride cycle might well vary from person to person, dependent upon contextual factors such as the presence of compromised tissue thresholds (e.g. from recent heavy training) and/or the exact nature of the functional activity being performed at the time of injury. It is noted that the majority of the literature covered in this chapter is derived from analysis of constant-speed running, and additional work in acceleration and deceleration efforts is warranted, as well as efforts requiring change of direction.

3.6 Factors That Influence Biceps Femoris Long Head Strain During Sprinting

Given that (a) HSI most commonly involves BF_{LH} [47], (b) HSI commonly occurs during high-speed running [48] and (c) peak MTU stretch during the terminal swing phase of high-speed running has been shown to be greatest for BF_{LH}, researchers have understandably been tempted to link these observations [15, 16, 19, 21, 22]. Understanding factors that may modulate peak MTU stretch may have important implications for interventions aiming to alter risk of HSI.

3.6.1 Muscle Coordination

In an effort to identify the influence of muscle force on peak BF_{LH} stretch during swing, one study [21] conducted a perturbation analysis of musculoskeletal simulations of the double float phase (i.e. when both legs are simultaneously in swing) during maximal sprinting. These authors found that greater stretch in the BF_{LH} was induced by muscle force from the ipsilateral rectus femoris and iliopsoas, as well as the contralateral iliopsoas, erector spinae and rectus femoris. Muscles with the greatest potential to decrease BF_{LH} stretch were the ipsilateral adductor magnus and hamstrings, as well as the contralateral internal oblique. It is currently unclear to what extent these simulation results reflect reality and therefore whether they can be used to directly inform rehabilitative and preventative interventions.

3.6.2 Series Elastic Component Stiffness

This chapter has provided evidence from multiple studies describing MTU stretch of the hamstrings during running. Although MTU stretch during running may well be a relevant variable for understanding the biomechanics of HSI, it is important to recognise that this term describes length changes of the entire MTU. Due to elastic properties of the series elastic component (i.e. tendon, aponeurosis), length changes of the entire musculotendinous unit are not necessarily accurate representations of length changes within the muscle fibres. The decoupling of muscle fibre and series elastic component length changes during dynamic activities is well established in vivo for other human lower limb muscle groups such as the ankle plantar flexor muscles (e.g. [49–51]). Equivalent in vivo data for the human hamstrings during running are not presently available; however, musculoskeletal modelling studies have shown that, across a range of physiologically reasonable tendon stiffness values, the relative strain experienced by the BF_{LH} muscle fibres during swing is directly related to the stiffness of the series elastic component [23]. This may suggest that tendon stiffness is an important regulator of muscle fibre strains experienced during swing and might therefore be important for injury risk. It is currently unknown, however, whether alteration of tendon stiffness will provide meaningful change in the risk of HSI.

3.6.3 Non-Uniform Strain Distribution

Musculoskeletal modelling studies describing MTU stretch during sprinting use simplified representations of muscle-tendon architecture and therefore dynamics, assuming uniformity in fibre strain distribution across the entire MTU. Whilst human in vivo data for the hamstrings is currently lacking, non-uniform muscle tissue strain distributions have been observed in the human biceps brachii muscle during loaded elbow

flexion [52]. As these non-uniformities are due to the complex architecture of skeletal muscle, it is plausible that the human hamstrings may exhibit similar non-uniformity during running. To examine this, prior studies [10, 11] have utilised advanced imaging techniques to develop finite element models of the BF_{LH}, which contain more physiologically accurate complex representations of muscle fibre and tendon architecture and dynamics than what is typically accounted for in musculoskeletal modelling studies. Using these complex models and input experimental data from sprinting (i.e. MTU kinematics and muscle activation data), these studies have been able to provide insight into region-specific BF_{LH} muscle fibre strain patterns during the swing phase of sprinting. These data suggest that local muscle fibre strains exhibit non-uniformity across the MTU, with the greatest strains observed at the proximal musculotendinous junction [11]. This observation may provide an explanation as to why the proximal musculotendinous junction is the most frequently reported site of BF_{LH} strain injury [53]. Additionally, both the magnitude and non-uniformity of local fibre strain appear to increase as running velocity is increased [11].

3.7 Conclusion

In summary, the current evidence base suggests that the hamstrings are recruited for the entire stance phase, as well as during a portion of the swing phase (from mid-swing onwards). The late swing phase has been identified as the most likely period of injury, as the hamstrings undergo active lengthening and experience peak lengths. The forces produced by each hamstring muscle during this period increase with increasing running velocity, whilst the peak length experienced during this same period is largely invariant amongst high running velocities (>80% max). Whilst hamstring function is likely compromised following HSI, the findings from investigating studies are often conflicting; thus, more research is needed to identify which specific parameters need the most consideration during rehabilitation. Overall, the information in this chapter may inform clinicians aiming to develop HSI preventative and rehabilitative interventions.

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Extrinsic and Intrinsic Risk Factors Associated with Hamstring Injury

4

Tania Pizzari, Brady Green, and Nicol van Dyk

4.1 Introduction

Across all sports exists a trade-off between risk and reward; the risk of injury versus the reward of performance. There has been considerable research and many risk factors proposed for hamstring injury. Risk factors for injury can be classified as extrinsic or intrinsic. Extrinsic factors are external to the individual and can include variables such as the type of sport, exposure to the sport, training, and playing environment [1]. Intrinsic factors are internal personal factors that can be further dichotomised into modifiable and non-modifiable risk factors. Non-modifiable risk factors cannot be altered, such as previous injury, age, gender, and ethnicity. Modifiable intrinsic risk factors represent those factors that can be influenced, for example, physical fitness, strength, and flexibility.

Several reviews have combined and synthesised the literature on hamstring injury risk factors [2–5], with the most comprehensive meta-analysis identifying age, previous history, and greater quadriceps strength as potential risk factors for hamstring injury [3]. In this chapter we synthesise these results, together with recently published work, from a clinical perspective. Conflicting results are commonplace and the tendency to assess variables in isolation likely confounds the understanding of this multifactorial problem [6, 7]. If we interpret and approach risk factor findings correctly, we may be able to assist our athletes in better risk management and, subsequently, greater participation and performance.

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4.2 Interpreting Risk Factors

Effective identification of modifiable, intrinsic risk factors is a vital component of injury prevention [1, 8]. The injury aetiology model proposed by van Mechelen in 1992 identified principles for understanding injury risk and included three steps: (1) identify the magnitude of the problem (incidence or severity); (2) ascertain the aetiology, risk factors, or injury mechanisms responsible; and based on these findings, (3) introduce a preventative measure to address the injury occurrence. Finally, the effect of the intervention is evaluated by repeating the first step. Over the past decades, several other injury prevention models have been proposed that expand on the van Mechelen model. Meeuwisse et al. [9] developed the understanding of injury risk by accounting for the interaction of multiple risk factors, both intrinsic and extrinsic. In 2005, Bahr and Krosshaug [10] expanded on the characteristics of the injury mechanism during the inciting event as a component of the causal pathway. Later Meeuwisse et al. [11] acknowledged the non-linearity of sports injury and attempted to account for the interaction of multiple risk factors and the potential of an inciting event to change an athlete's intrinsic risk and predisposition to injury. Finch [8] advanced the original injury prevention model further by addressing the issues related to implementation and integration of such interventions and their effectiveness, through the Translating Research into Injury Prevention Practice (TRIPP) framework. In this framework, two important steps were added before repeating step one: (1) determining the ideal conditions to perform the preventative measure and (2) evaluating the effectiveness of the prevention programme in an implementation context. For team sports, the model has been adjusted to reflect the nature of working in a professional team into the new Team-sport Injury Prevention (TIP) cycle [12].

An athlete's risk of injury fluctuates over time as individual intrinsic and extrinsic risk factors change. For example, exposure to load/sport varies and intrinsic features such as strength and power vary across a season and can even change from 1 day to the next. As a result, traditional systems of screening the risk profile of athletes at a single time point, often in preseason, may fail to identify important risk factors for hamstring injury. The risk profile of individuals in the same cohort may also vary according to the interdependence of more than one factor. When considering athletes in different sports, it is possible that risk factors are not the same due to differences in the demands of competition (i.e. running demands during match play), training history (i.e. exposure to high-intensity sprinting), or overall management practices (i.e. eccentric strengthening) [11, 13–15].

The presence or absence of a risk factor in an individual athlete does not predict with any certainty that the athlete will sustain an injury [16, 17]. Current risk factor research identifies variables in groups of injured athletes that are statistically different to groups of uninjured athletes. These studies of large cohorts do not allow direct translation of risk to the individual athlete [16], since despite the statistical differences between groups, there is substantial overlap when comparing scores between injured and uninjured athletes. In practice, an athlete may be deemed high risk based on one or more factors, but never go on to sustain a hamstring injury. The reverse is also true, and athletes who exhibit a low-risk profile can go on to sustain an injury. Importantly, even in situations where an athlete is deemed to have an

increased risk, if the baseline risk of injury in the first instance is trivial, then the risk remains trivial even if it is increased. For example, the prevalence of hamstring injuries in football, Australian football, and cricket is reportedly between 11% and 16% over a season [18]. Even if an athlete is twice as likely to sustain an injury if they have a past history of a hamstring injury (i.e. relative risk) [19], then the athlete still only has a 22–32% chance of sustaining a hamstring injury (absolute risk) and a greater than 65% chance of not sustaining an injury within a season, based on past history as a risk factor.

In addition to the limitations for injury prediction, the traditional method of risk factor identification, whereby the relationship between individual factors and injury occurrence is evaluated, has been criticised as reductionist and simplistic [6, 7, 11]. This method fails to account for the complex and fluctuating interactions between risk factors and the context for each individual athlete. A greater awareness of the complexity involved in sports injuries is required and a new approach has now been suggested to incorporate how these factors mediate, moderate, and interact with each other [6]. The current literature does not yet adequately consider the interactions and dynamic nature of risk factors and as such the details presented in this chapter are constrained somewhat by these limitations. Nevertheless, the information will assist clinicians when interpreting risk profiles in each athlete within the context for that particular athlete and, where appropriate, within the team they are part of. The detection of musculoskeletal deficits, pathology, or disease may allow for appropriate and early intervention which in turn may prevent the occurrence of injury or minimise the severity of injury [16].

In practice, most clinicians will perform a battery of tests that includes the athlete's injury history and implement interventions customised to the athlete's impairments and activity limitations [20]. Based on these findings, interventions can be designed for perceived at-risk athletes over and above any universal prevention efforts already in place for the entire team (such as warm-ups and eccentric strengthening).

4.3 Intrinsic Risk Factors

A plethora of non-modifiable and potentially modifiable intrinsic risk factors have been examined and discussed in the literature. Figures 4.1 and 4.2 highlight the variables examined and depict the confidence in the available evidence based on the number of studies evaluating a particular factor, the quality of the studies, and the consistency of findings.

4.3.1 Non-modifiable

4.3.1.1 Age

Advancing age has been implicated as a risk factor for hamstring strain in many studies [3]. This finding is not consistent across all studies [19, 21–30], although meta-analysis of available data suggests that age is significantly associated with

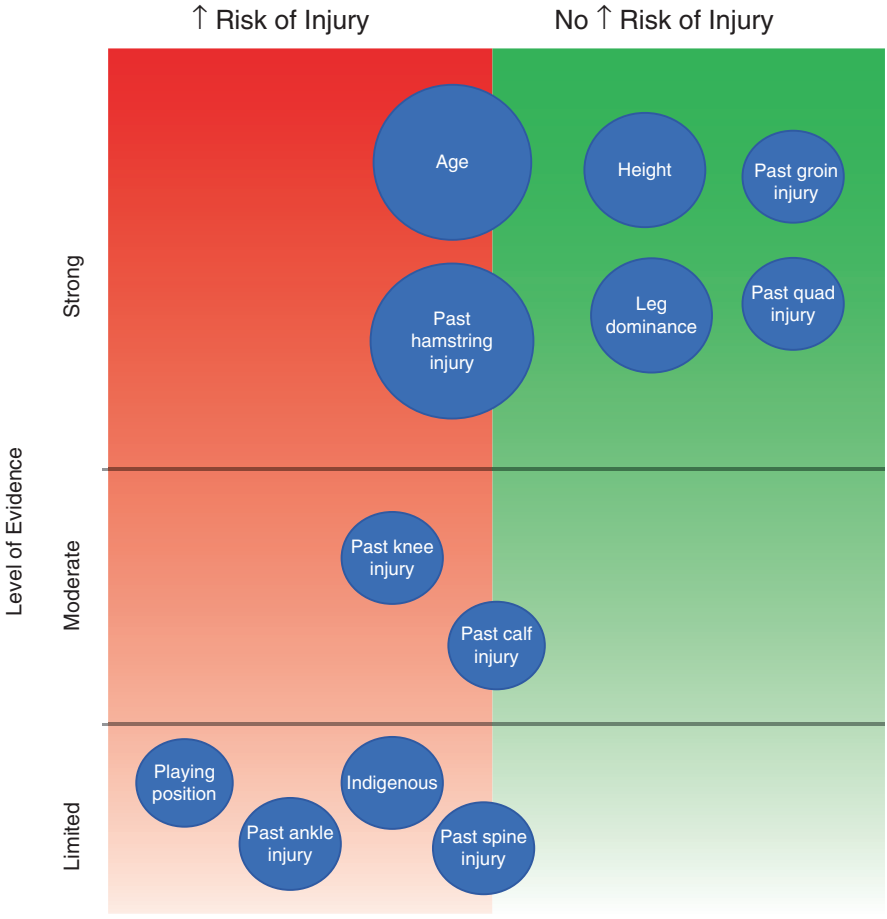

Fig. 4.1 Intrinsic, non-modifiable risk factors for hamstring injury, levels of evidence, and relationship to injury risk. The size of the circle is a pictorial reflection of the amount of literature around each risk factor, without being an exact indication. Where circles cross the midline, there is conflicting evidence of their association with injury

hamstring injury [3]. The mechanism behind this relationship is not entirely clear. Age-related degenerative changes in the lumbar spine, as well as the ensuing impact on the L5 nerve root and lumbar spine flexibility, have also been theorised as an explanation for the association between age and injury risk [31]. However a direct link between lumbar flexibility and subsequent injury is lacking [27, 32–34]. In addition, older athletes have generally been participating in sport for a longer period, resulting in a greater likelihood of a previous hamstring injury as a consequence of greater exposure. Arnason et al. [35] did identify age as an independent risk factor for hamstring injury, not mediated by previous history of injury. However, the age range of the athletes in this study (16–38 years) was greater than most studies that have examined this non-modifiable risk factor [3].

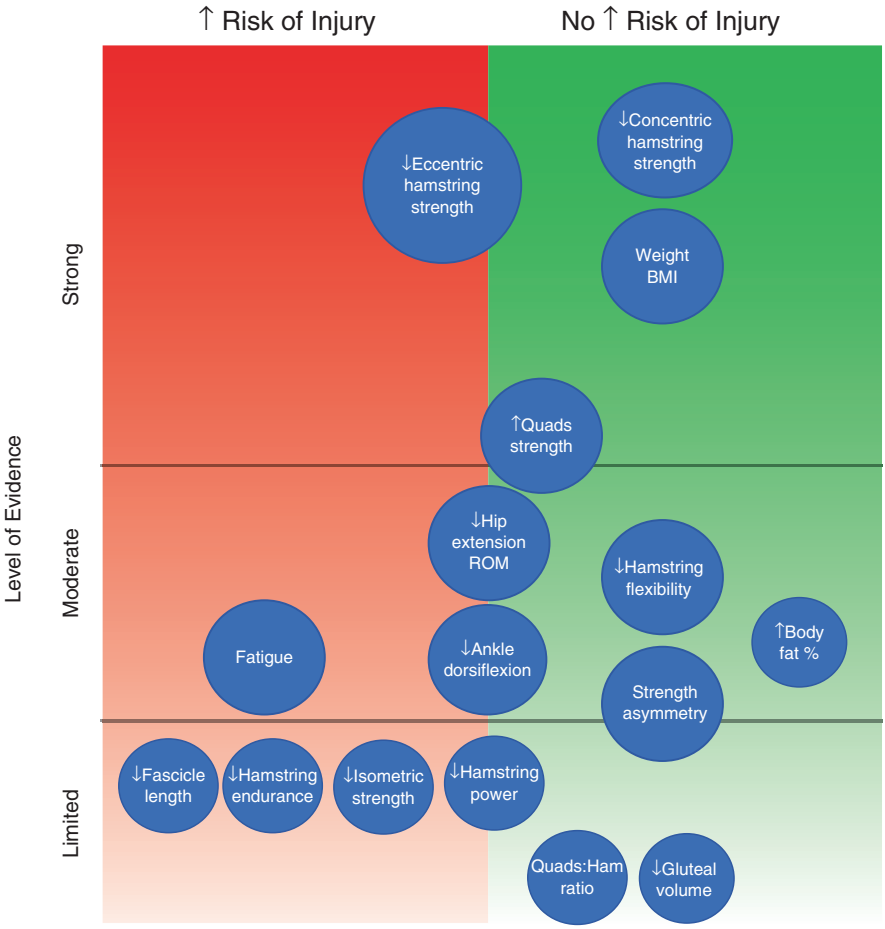

Fig. 4.2 Intrinsic, modifiable risk factors for hamstring injury, levels of evidence, and relationship to injury risk. The size of the circle is a pictorial reflection on the amount of literature around each risk factor, without being an exact indication. Where circles cross the midline, there is conflicting evidence of their association with injury

Athletic qualities evidently decline with ageing, including metrics of strength [36, 37], power [36, 38], and running [39, 40]. Older athletes could therefore have a greater injury risk if they are less equipped to cope with sporting demands, but direct evaluations of these changes and how they interact with hamstring injury are lacking. Changes in the structural characteristics of the ageing muscle-tendon unit (MTU) are possibly the basis for declining function, such as muscle mass [36, 38], stiffness [41], fibre population [42], and tissue quality [43, 44], but these changes typically occur to a greater extent after a professional career has ended. Similarly, age-related changes in the nervous system are another possible explanation, in particular a progressive loss in high-threshold motor units [45–47], which could make

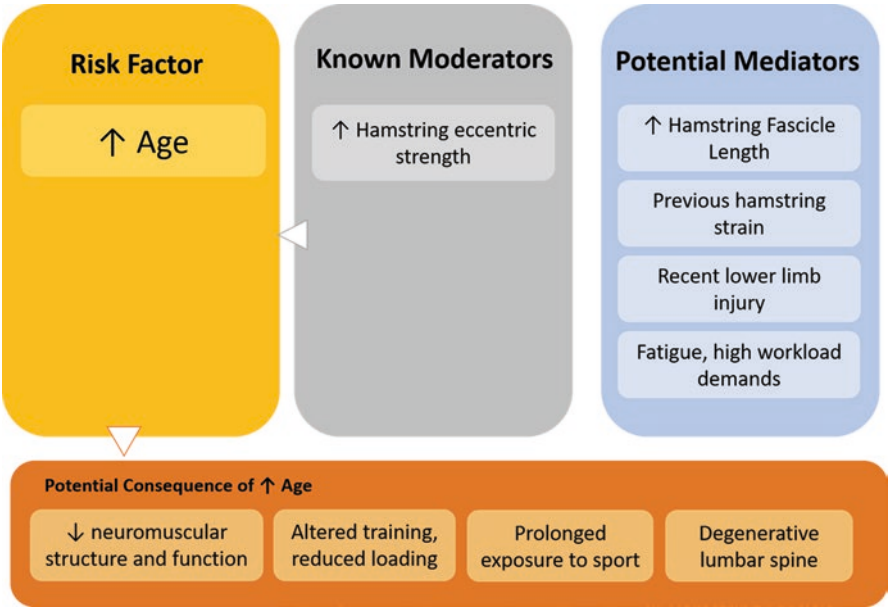

Fig. 4.3 The potential consequence of advancing age, the known moderators, and potential mediators related to age as a risk factor for hamstring injury

older athletes susceptible to injury from fast and forceful muscle actions during activities such as running [48].

Altered training in older athletes is a frequently employed strategy to minimise injury risk [49], but it might also be implicated in loss of muscle strength [50] as athletes may train less, at a reduced intensity, or with a greater focus on sports-specific training in place of strength training. The training history and habits of individual athletes may therefore moderate injury risk with ageing [49, 51]. In support of this concept, Opar et al. [25] found the risk of hamstring injury in older Australian football players to be reduced with improvements in eccentric strength (Fig. 4.3).

4.3.1.2 Previous Hamstring Injury

Once an injury has occurred, the athlete is at an increased risk of a subsequent injury [3], particularly in the first 4 weeks following return to sport [52]. The precise duration of heightened risk following injury remains unknown and is likely to be unique to each athlete. In Australian football, the risk of recurrence is elevated for the entire season [53, 54] and the subsequent season [54]. Despite increased research efforts into risk factor identification, prevention, and management, recurrence rates remain high [55]. It should be noted that not all studies identify previous history as a risk factor for subsequent injury [3, 23, 25, 26, 56–59], although when the majority of data are combined in a meta-analysis, athletes with a past history are at a three times greater risk of sustaining a hamstring injury (relative risk = 3.19; 95% CI, 2.5–4.5; $p < 0.001$).

Following index injury, there is evidence of ongoing deficits in the hamstring muscle [60]. These could be associated with inadequate rehabilitation and a failure to address the potential persistent muscle changes, which is one explanation for previous injury being a risk for re-injury. Sustaining an index injury may also indicate the presence of other individual factors that place the athlete at greatest risk for injury [61]. Contextual factors such as behaviours, genetics, biomechanics, activity exposure, and psychology may elevate an individuals' re-injury risk.

4.3.1.3 Previous History of Other Lower Leg Injuries

There is moderate evidence that a previous knee joint injury [50, 62] is associated with an increased risk of hamstring injury and limited evidence that an ankle joint injury also increases the risk of hamstring injury [63]. Conflicting evidence exists for the relationship between hamstring injury and previous anterior cruciate ligament (ACL) injury [19, 21, 25, 64–67] and previous calf strain [21, 24, 25, 65–67] and no evidence for a relationship with previous groin injury [21, 25, 62, 67], quadriceps injury [21, 24, 25, 67], or adductor strain [24].

4.3.1.4 Ethnicity

The role of ethnicity in hamstring injury is not entirely clear and there are limited studies evaluating this variable. Indigenous Australian [62] and Black African or Caribbean [68, 69] athletes are purported to be at greater risk of hamstring injury, with muscle fibre type and excessive anterior pelvic tilt suggested as contributing factors in these populations [62, 69]. Indigenous Australian football players have also been identified as at greater risk of calf, ACL, and quadriceps injuries [70] and overall injuries combined [71]. However, when certain characteristics of this subgroup are controlled for in multivariate analyses, the influence of this variable is reversed. Specifically, being lighter in body mass, having lower aerobic capacity, and/or playing in a forward position contribute to injury risk [71]. When these factors are controlled for, the influence of indigeneity is no longer important. This further highlights the multifactorial nature of injury and the need to evaluate variables in combination to elucidate the most important factors, as well as those that may be interrelated or a surrogate for another measure. A large study (n = 592) of risk factors for hamstring injury in the Arabic Peninsula found no influence of ethnicity when comparing Arab participants (60%) with the rest of the multi-ethnic cohort [59].

4.3.1.5 Playing Position

Predictably, goal keepers in football (soccer) are at a reduced risk of hamstring injury [24], likely due to the reduced volumes and velocities of match-related kicking [72], reduced running loads, and high-speed running [73, 74]. In American football, defensive backs, running backs, and wide receivers are over-represented in hamstring injury groups [75, 76] and this could be related to greater exposure to sprinting in these positions. In rugby union, blind-side flankers sustain more hamstring injuries than forwards, with the majority of injuries occurring during sprinting or high-speed running [77]. In Australian football, midfielders (a position

requiring high running volumes and high-speed running) were over-represented in the injured group [19, 25]. Based on these findings, it is reasonable to assume that playing position interacts closely with or may be a proxy for running load exposure, another proposed risk factor for hamstring injury [19, 78].

Clinical Implications: Intrinsic Risk Factors (Non-modifiable)

- Previous injury is a strong risk factor for subsequent hamstring injury and the potential for persistent muscle deficits should be addressed to minimise risk of recurrence.
- Age holds inherent risk of injury but may be mitigated by strength and inducing morphological changes.
- Ethnicity has limited evidence and could be a surrogate measure of differences in muscle morphology, playing position, body size, and running demands.
- Playing position also has sparse evidence and could reflect difference in loading demands and body type, so it should be considered in a multifactorial approach.

4.3.2 Modifiable Risk Factors

4.3.2.1 Strength

Poor muscle strength has long been considered a risk factor for muscle injury [79]. Intuitively, if an activity requires loading beyond the capacity of the MTU, it could result in structural damage. Research investigating strength as a risk factor for hamstring injury is extensive and includes the evaluation of multiple lower limb muscles, using multiple methods and contraction modes, at variable speeds, in varying athlete populations, and in varying positions [3, 80]. As a result, synthesis of the evidence can be onerous and findings can vary depending on the study results combined. The evidence for the influence of strength on injury risk is ever evolving, requiring clinical reasoning to make meaningful conclusions.

The potential for improvements in strength, particularly eccentric hamstring strength, to ameliorate the risk presented by the presence of non-modifiable factors [25] is vital information for the athlete, coach, and clinician (Fig. 4.4b). The interaction between strength and the intrinsic, non-modifiable risk factors of age and past history of hamstring injury is demonstrated in Fig. 4.4a.

Eccentric Hamstring Strength

In recent times there has been a focus on reduced eccentric hamstring strength and injury risk, which is logical given the known persistent deficits in eccentric strength post-injury [81–84] and the common mechanism of injury in the terminal swing phase during high-speed running [85, 86]. There is also evidence that hamstring injuries can be reduced in cohorts of athletes undertaking adequately intense eccentric strengthening [87].

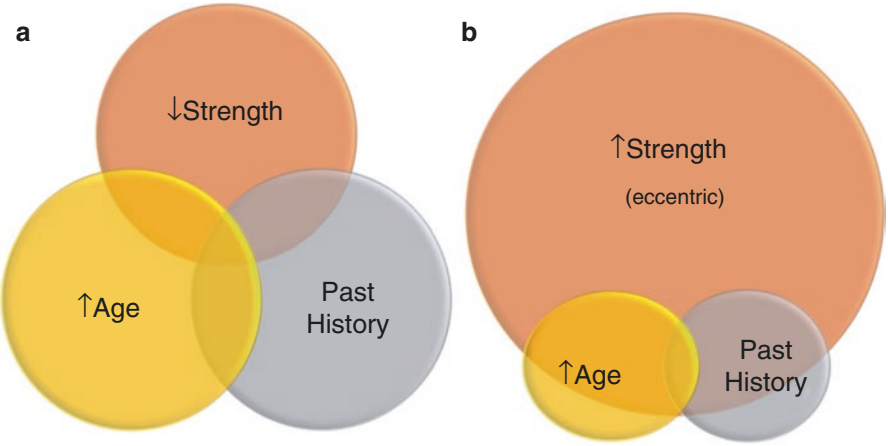

Fig. 4.4 (**a**) Interaction between intrinsic risk factors, namely strength, age, and past history. (**b**) Potential moderation of increasing strength on non-modifiable intrinsic risk factors. The size of the circle represents the significance of the risk factor

Measuring eccentric hamstring strength using the Nordic hamstring exercise (NHE) as a test has gained recent attention following two studies showing football and Australian football players with lower levels of eccentric strength in the preseason to be at a greater risk of sustaining a hamstring injury in the season that followed [25, 67]. The NHE activates the hamstrings at high levels of muscle activity and at angles similar to the joint angles at which peak hamstring activation occurs during sprinting [88]. Other studies using the NHE as a screening test in football [58] and one in rugby union [21] did not identify a relationship between peak Nordic forces and future hamstring injury. Furthermore, when the raw data from all four studies [21, 25, 58, 67] are combined in a meta-analysis, absolute eccentric strength (standardised mean difference [SMD] −0.31, 95%CI −0.97 to 0.4, $p = 0.13$) and eccentric strength relative to body mass (SMD −0.34, 95%CI −1.1 to 0.4, $p = 0.14$) are not significantly associated with risk of future hamstring injury. The lack of association could suggest the role eccentric strength plays in moderating injury risk is not uniform between athletes in the same sport, or athletes in different sports [11, 13]. For example, when considering differences between athletes in the same sport, there is evidence that Nordic eccentric strength interacts with other risk factors, such as previous injury history and age [21, 67].

In a recent systematic review and meta-analysis, isokinetic strength testing was found to have a limited role in predicting future hamstring injury [80], except for two variables of eccentric strength. Absolute and relative eccentric knee flexor weakness at $60° s^{-1}$ have a small predictive effect, although these results were derived from combining only two studies. Using a handheld dynamometer to measure eccentric strength, Goossens et al. [89] also identified that deficits in this strength parameter were associated with hamstring injury. Despite some inconsistencies between

studies, the weight of the evidence would suggest that eccentric hamstring strength is an important consideration when screening injury risk in athletes [80, 89, 90].

Concentric and Isometric Hamstring Strength

When compared with eccentric hamstring strength, measures of concentric and isometric strength generally show limited or no association with hamstring injury [80, 91]. Combining all available data for concentric hamstring strength measured using isokinetic dynamometry showed no relationship between this contraction mode and future hamstring risk [80]. Isometric hamstring strength has been examined in one small study using a strain gauge and provides limited evidence of an association with hamstring injury [91].

Importantly, regular strength monitoring of athletes has been advocated as a method of hamstring injury prevention [92], since reductions in isometric strength may precede hamstring injury [93]. Regardless of the mode of contraction, fluctuations in strength occur within a match cycle [94–96] and over the course of a competitive season [97]. Regular testing of strength over time may help to identify when strength deviations extend beyond these normal fluctuations and change the athlete's risk profile. Substantial deviations could indicate failed recovery of the hamstring muscle after sport/loading and allow for early interventions to facilitate recovery prior to participating in training or competition [16, 92].

Asymmetry and Strength Ratios

Between-limb strength asymmetries have traditionally been considered important to identify because, theoretically, the weaker side could be at an elevated risk of injury [98] or re-injury [99]. While it is plausible that a degree of asymmetry can exist between limbs without predisposing to injury, clinical wisdom holds that large differences may indicate that one limb is less proficient than the other at meeting the work demands or moderating the stresses athletes are subject to [100]. Large discrepancies could also result in less effective movement patterns or biomechanics that predispose to injury [48, 100]. To this end, almost all methods of strength assessment have evaluated whether between-limb asymmetries are predictive of future hamstring injury. In particular, isokinetic testing [80], the single-leg hamstring bridge [50] and the NHE [21, 25, 58, 67] have all included quantification of between-limb asymmetries.

Meta-analyses of isokinetic studies show that asymmetries in concentric hamstring strength ($60° s^{-1}$, $180° s^{-1}$) are not predictive of future hamstring injury [80]. Individual studies have investigated concentric strength imbalances at other angular velocities ($240° s^{-1}$, $300° s^{-1}$) [26, 30], as well as eccentric strength imbalances ($30° s^{-1}$, $60° s^{-1}$, $180° s^{-1}$) [23, 101], with all variables showing evidence for no association with increased risk of future hamstring injury [80]. Similarly, in three of the four studies measuring asymmetry in Nordic eccentric strength, there was not a significant imbalance in the players that sustained a hamstring injury compared to those that did not [25, 58, 67]. One study found imbalances of ≥15% and ≥20% to be risk factors for future hamstring injury in rugby union players [21]; however collectively,

imbalances in eccentric strength measured using the NHE lack validity for determining hamstring injury risk. Asymmetry between hamstring endurance during the single-leg hamstring bridge was also not predictive of injury [50].

A large study of 462 professional soccer players identified that low hamstring strength relative to quadriceps strength was associated with hamstring injury [102]. Even before this notable study, an imbalance between the strength of the hamstrings to the strength of the quadriceps had been presumed to be an important risk factor for hamstring injuries. A weak hamstring is theorised to be inadequate to control and brake the lower limb in terminal swing, following a forceful contraction of the strong quadriceps in the early swing phase [103]. Over the past 15 years, hamstring-to-quadriceps strength ratios and ratios quantifying between-side asymmetries have been well researched; however, when all data and evidence are combined, these ratios demonstrate no association with hamstring injury [80].

Strength-Endurance Measures

Reduced strength-endurance in the single-leg bridge has been shown in one study to be a risk factor for future right-sided hamstring injury [50]. Like the NHE, the test position replicates joint angles at the hip and knee akin to what is important for running function, a common mechanism of hamstring injury [85, 100]. Australian football players with a preseason deficit in the strength-endurance in the right leg were more likely to sustain a right-sided hamstring injury in the season that followed [50].

In a study evaluating the strength-endurance of hamstring muscles in football players, a fatiguing bout of lightly loaded (~5 kg) hamstring curls was used [104]. Football players were reportedly at an increased risk of re-injury if they had a deficit in the strength-endurance of the knee flexors, measured as the total time to repetition failure [104]. Since fatigue has been implicated in the risk of hamstring muscle injury, it is feasible that reduced strength-endurance of the muscle is a risk factor; however, further studies are required to support this. Despite the potential of hamstring strength-endurance for identifying athletes at risk, the assessment of this variable can be difficult, particularly in the elite setting. The time taken to complete the tests and the potential for resultant muscle soreness and fatigue can be unacceptable to the athlete, coach, and other key stakeholders.

Quadriceps Strength

An earlier meta-analysis combining results from four studies identified greater quadriceps strength as a risk factor for hamstring strain [3]. The proposed mechanism for this is identical to that described for the hamstring-to-quadriceps ratio, with the potential for excessive eccentric hamstring activity required to brake the force created by the hip flexors and strong quadriceps as they flex the hip and extend the knee during the swing phase of running. The addition of further research in this area and the separation of meta-analyses into different angular velocities of isokinetic testing have since failed to identify quadriceps strength as a risk factor for injury [80].

Other Lower Limb and Trunk Strength Measures

In a study of 64 track and field athletes, isometric hip extension and hip flexion strength measured using a strain gauge were not related to hamstring strain [91]. In a smaller study of 30 elite male sprinters [105], there was limited evidence for a relationship between isokinetic concentric hip extensors and hamstring injury, but only at slower isokinetic speeds of $60° s^{-1}$ [80]. Other strength measures have been described as being related to athletic function, such as back squat strength (1-repetition maximum) [106] or measures of abdominal strength [26]; however, these tests do not show evidence for an association with future hamstring injury.

4.3.2.2 Architectural Factors

A small retrospective study by an Australian research group published in 2014 identified the biceps femoris long head (BF_{LH}) fascicle length in the previously injured hamstring of participants was significantly less than the contralateral BF_{LH} [107]. Following this study, the research group performed a large prospective study with similar results. Players with shorter BF_{LH} (<10.56 cm) were four times more likely to sustain a hamstring strain injury (HSI) than those with longer fascicle lengths [67]. The mechanism by which shorter fascicles are more prone to injury remains ambiguous. Theoretically, shorter fascicles, with presumably fewer sarcomeres in series, will be more susceptible to damage as a consequence of sarcomere 'popping' while lengthening [108]. Longer fibre lengths permit greater excursion of a muscle and the length-tension and force-velocity curves are both influenced by fibre length in skeletal muscle [109]. More sarcomeres in series widen the length-tension curve and increase maximum velocity of the force-velocity curve [109]. In the terminal swing phase of running, greater force generation at long lengths could protect the hamstring muscles from injury.

In another example of the interaction between risk factors, the risk profile of athletes worsens considerably if they display a combination of shortened fascicles and reduced eccentric strength [67]. These results suggest, albeit indirectly, increasing biceps femoris (BF) fascicle length in parallel with improvements in eccentric hamstring strength may be an effective strategy for reducing HSI risk. Fortunately, clinical strategies are available to alter fascicle length in skeletal muscle. Exposure to an eccentric loading stimulus appears to be one of the most important methods for inducing adaptations in fascicle length. Using the NHE has been advocated as one method to achieve this in the hamstring muscles [110].

4.3.2.3 Power and Ballistic Function

The hamstrings contribute significantly to explosive athletic tasks, which is particularly evident in fast running [111–113]. From a mechanical standpoint, to reach greater running speeds, the hamstrings must have the capacity to operate at faster contractile velocities and to generate greater positive work in a shorter space of time (i.e. greater power output) [112–115]. It is plausible that deficits in power generating capacity or overall ballistic function could increase susceptibility to hamstring injury, irrespective of how well athletes perform during lower velocity tasks. Numerous methods have therefore been used to test this hypothesis, including the countermovement jump (CMJ) [116], running vertical jump [26], squat jumps

(without countermovements immediately before the concentric phase) [29], single-leg CMJ [35], and single-leg hop for distance [89].

The CMJ has been a popular test to determine if jump performance is a risk factor for hamstring injury [26, 29, 35, 106, 117], with the majority of studies finding no association with risk of hamstring injury [26, 29, 35, 117]. Iguchi et al. [106] reported a lower CMJ height to be associated with an increased risk of hamstring injury, but the power output during the CMJ was not associated with increased risk of injury. This result may reflect the fact that impulse (the force-time integral) rather than power determines vertical jump height. Two studies have provided evidence for no association between the squat jumps and increased risk of hamstring injury [29, 35], while another found football players to be at greater odds of sustaining a future hamstring injury with better performance in the squat jumps [116]. Furthermore, a higher squat jump to CMJ ratio was found to be a risk factor for hamstring injury in football players [29].

A study by Goossens et al. [89] also identified poor performance in the maximum single-leg hop for distance as a risk factor for future hamstring injury. The single-leg hop for distance, or other tests of single-leg plyometric ability, may have a role in forming hamstring injury risk due to the contractile mechanics of the task [89]. For example, for the propulsive phase to be effective the hamstrings must act synergistically with the other hip extensors, knee extensors, and ankle plantar flexors to generate positive work [118–120]. Following propulsion, for effective landing, the hamstrings must act eccentrically to decelerate and control multiplanar motion at the hip and knee [120–123]. Deficits in the hamstrings during these activities that require high-velocity work to be carried out (positive and negative) could reveal impairments that reduce the efficiency of the MTU and subsequently increase the risk of injury [100]. While power-based metrics are correlated strongly with markers of athletic performance, such as maximum sprinting velocity and horizontal force output during acceleration [112, 124, 125], the role they play as risk factors for hamstring injury is unclear and warrants further investigation.

4.3.2.4 Flexibility, Mobility, and Range of Motion

Reduced flexibility, mobility, or range of motion (ROM) has traditionally been considered to put athletes at greater risk of hamstring injury. Movements such as sprinting and kicking involve large stresses [85, 100] at reasonably long MTU lengths [100]. Although it is apparent that most athletes do not require exorbitant tissue lengths or ROM, it is generally accepted that there may be a minimum requirement to function effectively in sport. The majority of tests related to flexibility, mobility, and ROM, however, do not show a relationship with increased risk of future hamstring injury. None of the four traditional clinical tests evaluating hamstring or neural mobility are associated with an increased risk of future hamstring injury: (1) active or passive knee extension [27, 33–35, 117, 126–128], (2) straight leg raise (active, passive) [30, 33, 116, 128], (3) slump [33, 126, 127], and (4) lumbar flexion (standing, seated) [26, 27, 29, 32, 33, 127–129]. Isolated findings do show some tests are associated with risk of injury to the hamstrings, although the association, albeit significant, is best described as weak [59, 130].

There is mixed evidence that reduced range of hip extension motion in the modified Thomas test [27, 33, 126, 127] is a risk factor for future hamstring injury. Reduced hip extension may increase the risk profile due to alterations in the mechanical loading conditions of the hamstrings during running, which are influenced by the lengthening and activation characteristics of the iliopsoas [100]. Other tests of hip ROM do not provide clinical value in determining risk of future hamstring injury: flexion, internal rotation, and external rotation [27, 32, 33, 126, 127].

Reduced ankle dorsiflexion ROM may also indicate risk of future hamstring injury, although research findings are conflicting [33, 50, 59, 126, 127]. Adequate ankle dorsiflexion mobility is a necessary component for running [131] and decreased ankle mobility changes the touchdown position of the foot during sprinting, reducing the horizontal force production [132]. As hamstring muscle activity is highly correlated with increased horizontal force production [112], limited ankle dorsiflexion mobility might lead to increased work required from the hamstring muscle, predisposing it to injury. However, hamstring injuries are believed to occur either during the terminal swing phase, when the foot is not in contact with the ground, or during the early stance phase, when the foot is in a relatively plantar-flexed position [115, 133]. Therefore, the reasons for the observed relationship between low ankle dorsiflexion ROM and risk of hamstring injury remain unclear.

Deficits in ROM are apparent after a hamstring injury has occurred, and this could be one reason tests of this kind have been traditionally considered to be related to injury risk [134, 135]. Based on the body of evidence to date, it is plausible that, similar to strength measures, assessment of flexibility, mobility, and ROM is more suitably placed in: (1) periodic health evaluations and (2) staging rehabilitation when an injury has occurred [92, 94–96, 135].

4.3.2.5 Muscle Activity and Motor Control

The term neuromuscular function is wide-ranging and is used to describe different aspects needed for optimal motor output. A skilled motor performance requires a feedforward mechanism where information is continually fed into sensory-motor loops from peripheral to central neural networks [136]. A primary role of the hamstring muscle group during high-speed running is active deceleration of the forward moving thigh during the terminal swing phase [137]. This is considered as the phase in the gait cycle where most of the hamstring injuries occur, with high force eccentric contractions decelerating the limb in a lengthened position [85, 100]. Therefore, the rate of torque development, together with the timing of muscle activity in relation to the action of the limb, are necessary to produce optimal eccentric contraction of the hamstring muscle during high-speed running.

In a prospective investigation of ‘intrinsic’ neuromuscular function, neither rate of torque development nor the onset of muscle activity for any of the concentric or eccentric quadriceps and hamstring isokinetic modes of testing was associated with risk of hamstring injury [57]. It is important to acknowledge that these results only represent one aspect of neuromuscular function.

Recent prospective investigations into the lumbopelvic-hip complex suggest that the neuromuscular coordination in the posterior kinetic chain and lumbopelvic complex influences the risk of hamstring injury in male football [27] and elite Australian football players [138]. The results of these studies suggest a potential protective effect if the neuromuscular function of global musculature is addressed. Poor motor control, as measured by movement discrimination during a leg swing, was found in a small study of elite Australian football players to be associated with hamstring injury [56]. Motor imagery, measured by the reaction time of athletes to identify limb orientation in pictures (mental rotation), has also been found to be associated with hamstring injury [139] and might reflect some deficits in central nervous system (CNS) processing. Prospective studies into the neuromuscular function are small and varied and require replication before firm conclusions can be formed as to the impact of these variables on risk of hamstring injury.

4.3.2.6 Muscle Fatigue

Fatigue has long been implicated as a risk factor for hamstring injury. The absorption of energy before structural failure is reduced in fatigued muscles [140]. As noted above, the reduced strength-endurance of the hamstring muscle might be a causative factor for hamstring injury, suggesting that an athlete's ability to withstand muscle fatigue could be vital. There are a number of proxy indicators that highlight the importance of fatigue. For example, a disproportionately high number of hamstring strains commonly occur in the late stages of football [69, 141] and rugby matches [68] and football training sessions [141]. There is also an increase in hamstring injury rates during periods of match congestion [142].

Match play and training induce muscle damage [143] and neuromuscular fatigue [144], resulting in altered biomechanics [145, 146] and reduced hamstring muscle strength [96]. Reductions in hamstring eccentric strength, rate of force development (RFD), and muscle activation have been identified in players subjected to soccer-specific exercise protocols designed to simulate match-day fatigue levels [147–149]. Using different match simulation protocols, several studies have also demonstrated reduced post-fatigue strength of the hamstring complex with isometric [145, 150, 151], concentric [145, 150, 151], and eccentric contractions [107, 149, 150, 152]. The magnitude of change is greater in isometric and eccentric hamstring strength than concentric contractions and greater for the hamstring muscles compared to quadriceps post-match [153].

In addition, the running kinematics of elite soccer players are altered when fatigued, with a significant reduction in hip flexion and knee extension ROM, increased anterior pelvic tilt, and a subsequent reduction in hamstring muscle excursion [146, 154]. Single and repeated sprint performances are also impaired during and after match play [155], and players experience reduced range of hip and knee motion [146], reduced BF activation [107], reduced passing and shooting accuracy [156, 157], and inferior jump performances after games [145]. These changes in running kinematics and performance could lead to overloading of the hamstring muscle complex and an increased risk of hamstring injury. The reductions in strength, RFD, and muscle activation may also put the hamstring at risk late in

games or during periods of match congestion or high workloads where the muscle does not have a chance to return to its pre-fatigued functional state [92].

Hamstring muscle deficits caused by fatigue can last several days post-match in football players [144, 158–161]. A recent report indicated that peripheral fatigue contributes more than central processes to post-match fatigue, likely due to the match-induced muscle damage [144]. Central fatigue has been described as a reduced capacity of the CNS to stimulate skeletal muscle. Central fatigue appears most prominent immediately post-match with clear improvements at 24 hours and complete restoration at 48 hours post-match. Peripheral fatigue is also present acutely post-match without complete restoration noted at 72 hours [144].

The influence of fatigue on hamstring risk is probable and intimately related to the intrinsic variable of hamstring strength-endurance and the extrinsic variable of running workload. The athlete must possess adequate hamstring capacity to withstand the demands of training and match play and the training and match play loads must remain within the athlete's envelope of function [162].

Clinical Implications: Intrinsic Risk Factors (Modifiable)

- Strength is associated with increased risk of hamstring injury: clinicians should focus on eccentric strengthening as both primary and secondary prevention.
- Flexibility of the ankle may be important to consider.
- The need for flexibility of the hamstring muscle as a whole is not supported; however, the length of individual muscle fascicles of the hamstring may be critical.
- Fatigue and impaired recovery of muscle function after loading are essential considerations for examining risk.
- Neuromuscular inhibition may be present post-injury, and rehabilitation should include efforts to improve components that contribute to neuromuscular function.
- Power and ballistic measures, such as the CMJ, may add valuable information for understanding an athlete's risk profile.

4.4 Extrinsic Risk Factors

4.4.1 Environmental Factors

Studies have investigated whether the environmental conditions predispose athletes to a hamstring injury [163, 164]. To date, there is no evidence that the temperature on match day, wind speed, rainfall (on match day or in the previous 7 days), evaporation in the previous 7 days, playing altitude, playing surface, time of the match, or a time zone change prior to the match are risk factors for future hamstring injury

[163, 164]. Shoe-surface interaction is associated with an increased risk of overall injury, especially rotational traction, although the specific impact on hamstring injury has not been examined [165].

4.4.2 Match Play, Sports Performance, Workload

The physical demands of elite athletes are high [15, 51, 73, 166]. Team sports, such as football, Australian football, and rugby, all have large running demands, and these demands rise with the playing standard [15, 73, 166]. The rate and risk of hamstring injury can therefore be higher with a greater level of competition [69].

Between sports there are however large differences in running and playing requirements, which may influence the patterns of injury and risk profiles seen [15, 167]. In professional football there are higher rates of hamstring injury once the competition season has commenced, and the rate of match injury is significantly higher than training [24]. The associated risks of match and training injuries may therefore be different. For example, higher rates of hamstring injury during the competition season have been hypothesised to be associated with two key factors: (1) a greater exposure to high-speed running in matches [168] and (2) the congested playing schedule of professional football [142, 167–169]. These associated risks are related to both the acute and chronic loads to which football players are exposed.

In a 10-year study of American football players, over half (53.1%) of all hamstring injuries occurred in the preseason, despite it being only 7 weeks' duration [75]. Rates of hamstring injury were particularly high in the first full month of participation and in playing positions requiring the greatest running velocities. Authors hypothesised this could be related to detraining during the off-season, which can cause maladaptive changes to strength, endurance, neuromuscular control, and running conditioning [75]. Similarly, in a 1-year prospective study of sprinters, 58% of hamstring injuries occurred within the first 100 h, or 10 weeks, of training and competition exposure [30]. The running demands of match play may also increase the risk of a hamstring injury in Australian football [65, 66]. Australian football players who have been interchanged more frequently in the preceding weeks (21–24 days) may be less likely to sustain a hamstring injury in the current match [65, 66]. Clearly, quantifying acute and chronic load exposure is worthwhile in ongoing athletic monitoring and may contribute to the assessment of an athlete's risk profile as it changes over the course of a season.

Advances in sports and performance science have recently increased the general understanding of how factors such as the acute workload, chronic training history, and the characteristics of match play could interact with injury risk [19, 78, 170–172]. To allow an examination of association between training load and injury risk in elite soccer players, the acute/chronic workload ratio (ACWR) is evaluated, as well as prior 1-, 2-, 3-, and 4-week loads. It is logical that if an athlete suddenly performs a volume of high-speed running that they have not been adequately prepared for [170], they will be at an increased risk of injury in the muscles important to fast running, such as the hamstrings [19, 78, 172].

The ACWR is commonly used in applied sport science settings as an indicator of potential injury risk. Although various 'versions' of the ACWR have been used [171, 173, 174], the most common method articulated in the literature has been through rolling averages that compare the most recent week to longer-term training [51]. Specifically, this method is calculated as the sum of workloads for the last week, divided by the average weekly workload for the previous 4 weeks [175, 176]. A ratio >1 indicates that the workload in the most recent week is greater than the workloads during the previous month of training. High ACWR values have been associated with increased injury likelihood in the periods following workload spikes. A major criticism of the commonly used ACWR are the biases inherent in discretisation of continuous data (i.e. workload data), which can result in a much higher false discovery rate. Therefore, the results of individual studies should be examined carefully if being used to direct decision-making on athlete management [177, 178].

Two studies of Australian football players evaluated the relationship between metrics of high-speed running exposure and hamstring injury risk [19, 78]. Both studies found increased exposure to be predictive of a future hamstring strain, and the largest effects of high-speed running exposure on injury risk were observed in the 2-week interval preceding the exposure. Specifically, Duhig et al. [78] found the greatest association for the total high-speed running distance in the week prior to injury, and Ruddy et al. [19] found weekly high-speed running (>24 km/h) distance (absolute) and the week-to-week change in total high-speed running distance (absolute, relative) to have the strongest association with risk of injury.

Other previous investigations into associations of training load with injury risk use a composite measure of internal load, combining exposure (i.e. duration) with intensity, as measured by ratings of perceived exertion (RPE) [179–183]. The resultant metric is RPE-load [184]. While this approach is useful for quantifying weekly and training phase load, a specific breakdown is difficult as the score neglects quantification of intensity and duration in isolation, both of which are important for effective training planning [185]. Despite the fact that composite scores have the advantage of simplicity and are reliable and valid, they may well come at the expense of detail [186]. Sensitivity may be gained from using exposure and intensity as discrete variables within matches/competitions, as well as training, to capture exposure to: (1) high-speed running, (2) periods of acceleration, and (3) deceleration.

4.4.3 Managing Injury Risk in the High-Performance Environment

Injury risk management should be a shared decision-making process. In team sports the role of other members of the multidisciplinary team, including the coach, is vital in protecting the athlete from unnecessary risk. It is easy to appreciate how contextual pressures, such as the coaches' expectations, can impact player outcomes for the most common soft tissue injury in elite sport (i.e. hamstring injuries). Following a hamstring injury, a panel of staff (medical, physiotherapy, coaches, sport science/

strength and conditioning) should be involved in planning rehabilitation and estimating return to play (RTP). There are two obvious consequences when this approach is not followed: (1) index injuries occurring in athletes who are underrecovered or physically compromised attempting to train or play and (2) early recurrences in athletes attempting to return from injury prematurely. In the high-performance environment, it is essential for coaching staff to be informed regarding the results of certain risk factor findings as it pertains to the athlete and, when applicable, the team. There is some preliminary evidence that the leadership style and communication quality of the head coach may be associated with the overall injury rates and player availability of their teams [187, 188].

Clinical Implications: Extrinsic Factors

- Training load influences risk of hamstring injury and performance. As yet, no specific metric demonstrates overall effectiveness in identifying which players will be injured.
- Higher chronic training loads could potentially lower the risk of hamstring injury.
- Avoiding acute spikes in training load, especially during preseason training camps, should be considered when planning for the season.
- Recent exposure to high-speed running can influence the subsequent susceptibility to hamstring injury. High-speed running exposure should be monitored where possible.
- Other stakeholders, such as the player and coaching staff, should be involved in decision-making as one potential strategy to avoid injury.

4.5 Hamstring Injury Sequelae

Following index injury there is evidence of ongoing deficits in the hamstring muscle [60]. These sequelae might impact athlete performance and increase the risk of recurrent hamstring injury (Fig. 4.5). Numerous studies have identified hamstring deficits in previously injured athletes [60], although in many cases it is not clear if the deficits were present prior to injury or as a consequence of the injury.

Pain-induced neuromuscular inhibition is one of the major sequelae that has been proposed to occur after a hamstring injury [60]. The pain following hamstring injury is variable and often resolves quickly, although in more severe injuries pain can be present for a longer period and the athlete may require the use of crutches. Pain at the time of injury might be an indicator of injury severity and prognosis [189]. In the initial stages of rehabilitation, pain while walking and during resisted hamstring tests might predict time frames for RTP [190, 191]. The pain adaptation theory proposes that muscle activity decreases with pain, reducing the force-producing capacity of the muscles [192, 193]. In support of this theory in hamstring injury, lower levels of normalised electromyographic activity in maximal voluntary eccentric

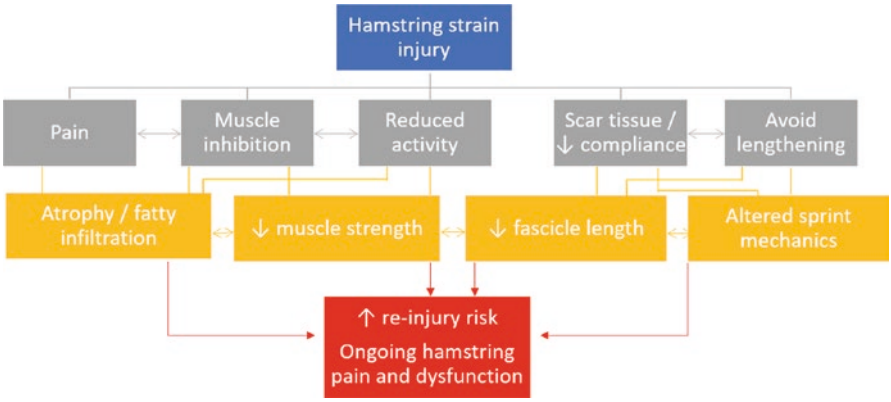

Fig. 4.5 Potential sequelae following hamstring injury and association with risk factors for hamstring injury

actions at longer muscle lengths [84, 194, 195] have been identified in previously injured individuals. In addition, the rate of torque development and early contractile impulse during eccentric contractions is lower in the injured limb as compared to the uninjured limb [195]. These adaptations as a response to pain can be beneficial in the short term to protect the area from pain or further injury, but have the potential to be detrimental in the long term [192]. Ongoing muscle inhibition might result in changes in muscle structure (i.e. atrophy, fascicle length) and function (i.e. strength, sprint mechanics).

Persistent strength deficits are a recognised post-injury sequelae. Reductions in concentric and eccentric hamstring strength have been shown to persist from months to years following injury [81, 82], with greater deficits often seen in eccentric muscle actions [81, 84, 194–197]. Atrophy and fatty infiltration of the previously injured BF_{LH} has also been identified, with corresponding hypertrophy of the biceps femoris short head, suggestive of some compensation by the short head [198, 199]. Changes in morphology could negatively affect the capacity of the hamstrings. Peak torque of the hamstrings is also generated at shorter muscle lengths following injury and this change can persist for years after injury [84, 197, 200]. Considering that the work of the hamstring muscle is greatest at terminal swing [201] when the hamstring is in a moderately lengthened position, a shift in peak torque to shorter lengths may predispose the muscle to trauma as it attempts to generate high levels of tension in this position.

Sequelae following hamstring injury have also been proposed to result from conservative rehabilitation strategies that avoid long muscle lengths in the early stages and base progression of rehabilitation on the absence of pain during testing and functional tasks [60, 202]. These conventional management strategies [203] could negatively influence muscle activity, compliance, and fibre length and potentially increase the risk of re-injury. Maladaptive architectural changes might also result from the physiological healing process following a muscle tear. Following injury, persistent oedema in the injured hamstring muscle [198, 199, 204] and an increase

in hamstring tendon volume and tendon scarring [199, 205] have been identified and shown to alter force transmission and tissue compliance [83, 205]. During post-injury remodelling, contractile tissue is being replaced with non-contractile connective tissue ('scar' tissue), which may be excessive in some individuals [206], resulting in an increase in stiffness of the musculotendinous junction and reduced compliance of the hamstring muscle [83, 205]. These post-injury sequelae may be clinically important since changes in the width, length, and thickness dimensions of tendon/aponeuroses of the hamstrings significantly alter the magnitude of peak strain and where the peak strain occurs in the MTU [207]. Changes have been identified in sprinting [208] and during active lengthening [209] activities and are proposed to be implicated in risk of re-injury following hamstring strain. Despite these hypotheses, there is moderate evidence for no association between the presence of fibrosis, measured at the time of RTP, and risk of recurrence [210], although this finding is based on results from one high-quality study [211]. The influence of changes in muscle structure following hamstring injury deserves further evaluation.

It is likely that these sequelae moderate the risk of future hamstring injury [60, 83, 205], but little is known about precisely how long they persist. Inadequate rehabilitation and a failure to address the potential persistent muscle changes described above is one explanation for previous injury being a risk for re-injury. It might be considered that current rehabilitation practices fail to recognise and address persistent changes and may not be adequate to alter potential neuromuscular influences. A better understanding of which post-injury maladaptations contribute most to the increased risk of subsequent injury could inform targeted rehabilitation strategies and reduce the risk of recurrence.

4.6 Risk Factors for Proximal Hamstring Tendinopathy

Specific risk factors for the development of proximal hamstring tendinopathy (PHT) have not been investigated. Proposed risk factors have typically been derived from clinical wisdom, biomechanical modelling of hamstring tendon strain, and the wider tendon literature, in particular patellar and Achilles tendon research.

Unlike hamstring muscle injuries, which typically occur acutely following an incident, PHT is considered to be a chronic, degenerative condition that develops in response to repetitive mechanical overload [212]. There are several conceptual models proposed to explain the aetiology of tendon pathology. These can be divided into three main groups: (1) collagen disruption/tearing [213], (2) inflammatory [214], and (3) tendon cell (tenocyte) response [215]. It is likely that all models explain aspects of the pathogenesis of tendinopathy [216], although the tendon cell response model more logically explains the tendon adaptation observed in different loading strategies [217]. The tendon cell response model places the tenocyte at the centre of a cascade of cellular and extracellular changes. The role of the tenocyte is to maintain the extracellular matrix in response to changes in its environment. Changes in tendon load are sensed by the tendon cell and give rise to a cascade of

events including cell activation, proteoglycan expression, and changes in collagen type [215, 218]. Repetitive load beyond the capacity of the tendon is thought to be the driver of tendon pathology and pain [216]. For the proximal hamstring tendon, repetitive loads during activities that require energy storage of the tendon or cause compression of the tendon, or both, are considered provocative [219].

Energy storage of the proximal hamstring tendon is greatest in the late swing to early stance phase of running [219]. Repetitive loading in this position could explain why sprinters and middle- and long-distance runners are particularly susceptible to this condition [220]. Energy storage loads and compression loads on the tendon are greater when the hip or trunk is flexed [221], which explains why training errors such as increasing volume and intensity of running, or the sudden addition of sprinting, lunging, hill running, or hurdles, are also thought to be implicated in the development of hamstring tendinopathy [219].

Altered biomechanics in running are intrinsic risk factors proposed to contribute to the development of PHT [222]. Specifically, an increase in anterior pelvic tilt is thought to increase the tensile stress and energy storage loads of the hamstring tendon as the muscle is lengthened over the hip and the tendon compressed against the ischial tuberosity. Compression of the tendon at the enthesis is implicated in tendinopathy throughout the body and might also explain the occurrence of this condition with sitting [220].

Other proposed intrinsic risk factors include reduced hamstring strength [212], poor lumbopelvic stability [212], overactive hip flexors/reduced hip extension movement [222], and inhibition of the gluteus maximus [222]. Risk factors considered universal to tendon pathology should also be considered when assessing athletes for risk of proximal hamstring tendon pathology. Advancing age, male sex, excess adiposity, menopause, genetics, inflammatory and autoimmune conditions, diabetes, hyperlipidaemia, and medications are intrinsic risk factors for tendinopathy [216, 219].

4.7 Conclusion

Understanding risk factors for hamstring injuries is an essential step in the prevention of these injuries in athletes. Despite the large volume of research undertaken to elucidate these factors, evidence for many is weak or conflicting. Limitations in the methods of studies combined with the likely variation in risk factors between sports and within sports across a season might explain these inconsistencies.

Increased chronological age, a history of previous hamstring strain, and reduced eccentric strength are most commonly identified as variables that place an athlete at a greater risk of hamstring strain although there is not absolute agreement in findings even across these commonly evaluated factors. The importance of eccentric hamstring strength for reducing risk of injury has been identified in several studies and has been shown to minimise risk in older athletes and those with a past history of injury. Extrinsic variables including match demands, training workloads, and volume of high-speed running appear to be important considerations for managing

hamstring injury risk in athletes. Loading the athlete within their functional capacity or gradually improving functional capacity within appropriate workload demands and achieving adequate recovery following activity is vital for avoiding injury.

The risk profile of any athlete needs to be considered relative to the individual's sporting demands, past history, the presence or absence of risk factors, the dynamic nature of risk factors and workload demands, the psychology and well-being of the athlete, and the limitations of risk factor evidence. Clinicians and coaches should be mindful that screening and risk factor identification cannot predict with certainty that an athlete will be injured but does allow for the identification of athletes with high-risk profiles and for the implementation of early preventative strategies.

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5 Neuromuscular Factors Related to Hamstring Muscle Function, Performance and Injury

Matthew Bourne, Joke Schuermans, Erik Witvrouw, Per Aagaard, and Anthony Shield

5.1 Neural Factors Relating to Hamstring Muscle Function, Performance and Injury

5.1.1 Leg Muscle Strength, Rate of Force Development (RFD) and Sprint Performance

In human athletic activities, maximal acceleration and sprint capacity are strongly determined by maximal strength and the rate of force development (RFD) of the lower limb muscles. In terms of lower limb muscle strength, strong inverse relationships ($r = -0.94$ to -0.61) have been observed between 1-RM squat strength and the time to cover 5, 10 and 20 m sprint distances performed from a standing start [1–3]. These observations indicate that a large proportion ($r^2 = 37–88\%$) of the inter-individual variance in acceleration/sprint capacity is governed by differences in leg extensor strength.

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Independently of maximal leg muscle strength, the ability to generate force rapidly also exerts a governing influence on human sprint performance. For example, static squat RFD normalised to maximal force (maximum voluntary contraction [MVC]) correlated strongly to 5 m sprint time ($r = -0.62$) when examined in British elite rugby players [4]. Further, athletes with a superior acceleration capacity (5 m sprint time <1 s) were characterised by a 40–60% higher leg extensor RFD in the very early phase of rising muscle force (0–100 ms) compared to players with less explosive acceleration capacity (5 m sprint time ≥1 s). Notably, maximal isometric squat force was not associated with sprint performance ($-0.04 < r < 0.25$), suggesting that lower limb RFD serves an independent role for human acceleration/sprint ability.

Recent reports have examined the effect of hamstring RFD on sprint and acceleration capacity. For example, Ishøi and colleagues [5] demonstrated that in elite youth football players (16–17 years), isometric hamstring RFD during the early phase of rising muscle force (0–100 ms) was inversely related with 5 m ($r = -0.45$), 15 m ($r = -0.47$) and 30 m ($r = -0.41$) sprint times. Similar relationships have been observed on isolated RFD assessment for the quadriceps and hamstring muscles. Examining Danish elite team handball players ($n = 12$, DHF Premier League) using isokinetic dynamometry, isometric quadriceps RFD (normalised to body mass) measured in the early contraction phase (0–100 ms) was found to be closely associated ($r = -0.71$ to -0.78) with the time to cover 5 m and 10 m sprints using a standing start (Aagaard et al., unpublished data). Notably, an equally strong association was noted between isometric hamstring RFD and 10 m sprint times ($r = -0.82$) (Fig. 5.1). Collectively, these data suggest that hamstring RFD represents an important factor in human sprint and acceleration performance.

5.1.2 Eccentric Hamstring Strength and Sprint Performance

Sprint speed is governed by two distinct factors: stride length and stride frequency. Theoretically, higher levels of eccentric hamstring strength may improve the rate at which the forward swinging shank can be decelerated during the terminal swing phase of sprinting, which would likely contribute to better sprint performance via greater stride frequency. In addition, high eccentric hamstring muscle force production during the transition from eccentric to concentric contraction, just prior to initial foot contact (start of stance phase) (Fig. 5.2), would be expected to transfer into an enlarged hip extensor moment during the late propulsive stance phase. In support of this notion, maximal eccentric hamstring strength measured in the Nordic hamstring exercise (NHE) was positively related ($r = 0.52$) to short-distance (20 m) sprint acceleration performance in highly trained youth soccer players ($n = 119$) [6]. Further, 10 weeks of NHE training in amateur football players (age 17–26 years) stimulated a significant improvement in maximal eccentric hamstring strength which paralleled gains in sprint acceleration performance [7]. Additional reports exist of improved sprint capacity in response to 7–10 weeks of training with the eccentric NHE [8], an eccentrically biased flywheel leg curl [9], or a combination of free weight hamstring exercises [10]. However, sprint acceleration performance

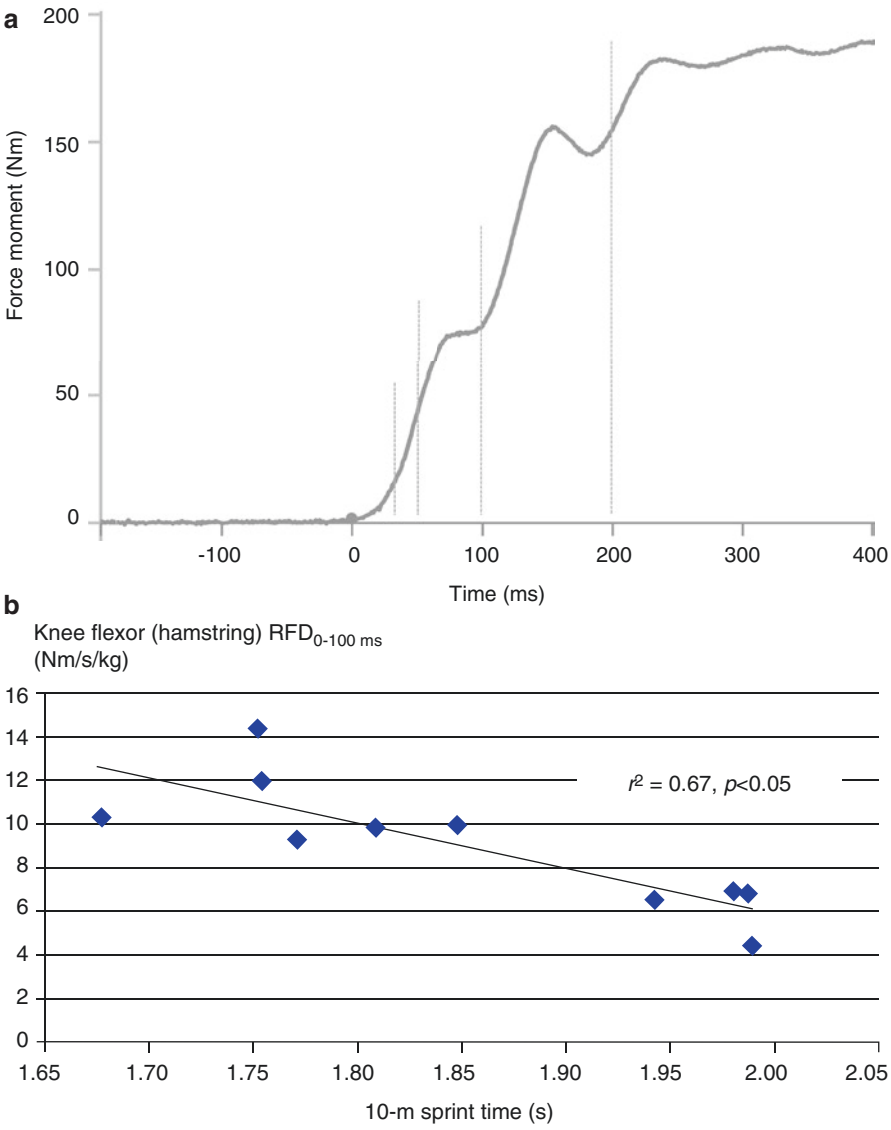

Fig. 5.1 Unpublished observations demonstrating the relationship between isometric knee flexor rate of force development (RFD), as assessed on an isokinetic dynamometer (**a**), and 10 m sprint times in n = 10 Danish elite team handball players (**b**) (Aagaard and colleagues, unpublished data)

appears to be most consistently improved as reflected by faster short-distance sprint times (5, 10, 15 m) [7, 8, 10], whereas gains in longer-distance sprint capacity (30 m) are less frequently observed [9]. In conclusion, training-induced improvements in maximal eccentric hamstring muscle strength can result in enhanced acceleration capacity and maximal sprint speed, and these effects appear to occur independent of training status.

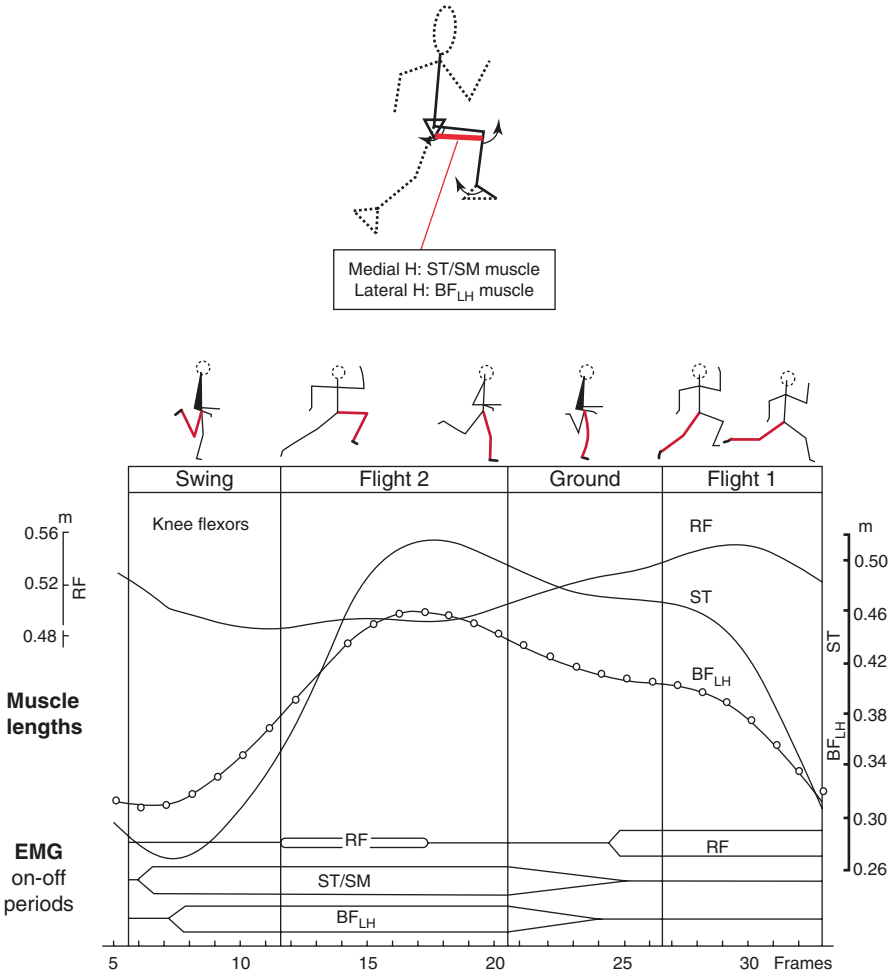

Fig. 5.2 Surface electromyographical (sEMG) activity and muscle lengths of the medial (semimembranosus and semitendinosus (SM and ST) and lateral (biceps femoris long head BF_{LH}) hamstrings throughout various phases of the gait cycle. *RF* rectus femoris, *m* metres

5.1.3 Neuromuscular Hamstring Activity, Sprint Performance and Risk of Muscle Strain Injury

The hamstring muscles are highly active during human sprinting, characterised by periods of peak electromyographical (EMG) activity during terminal swing, followed by a second bout of peak EMG activity in the midstance phase [11, 12] (Fig. 5.2). The specific pattern of hamstring activation bears significant importance for overground sprint performance. Recently, Morin and co-workers [13] used multivariate regression analysis to combine measurements of maximal isolated

eccentric hamstring strength obtained by isokinetic dynamometry and lateral hamstring (BF) EMG activity recorded during the terminal-swing phase of maximal sprinting, respectively. Together, these measures explained a substantial portion ($r^2 = 0.49$) of the horizontal ground reaction force (F_H) produced during the sprints, which represents the main governing factor for maximal sprint acceleration performance. Interestingly, maximal eccentric hamstring strength alone was not associated with F_H, underlining that the magnitude and timing of neural drive to active hamstring muscle fibres during the sprint stride plays an important role for a successful sprint performance.

Maximal eccentric muscle force production is influenced by spinally modulated sources of neural inhibition that limit efferent neural drive to the contracting motor units, despite maximal volitional effort (for review, see [14]). This inhibition in voluntary activation capacity can be downregulated by means of physical training, most effectively in the form of heavy-resistance strength training [14–16]. Experimental evidence of suppressed motor neuron activity during eccentric muscle actions has been reported for the knee extensors, ankle plantar flexors [14] and the hamstrings [17]. As a consequence, we might expect that increases in eccentric hamstring strength are likely to be highly beneficial not only for sprint acceleration performance (as discussed above), but also for the prevention of muscle strain injury.

In addition to the aforementioned performance benefits, high levels of eccentric hamstring strength may have implications for mitigating the risk of strain injury. Inspection of the in situ force-length relationship for skeletal muscle reveals a pronounced mismatch at elongated muscle lengths between the magnitude of active force production from muscle fibres and the passive stretch forces arising from elongation of the parallel-elastic muscle structures, in steep favour of the latter (Fig. 5.3). This observation predicts that muscle strain injury (1) will predominantly occur during active lengthening at elongated muscle lengths (as contractile force production ≪ passive force production) and (2) will mainly manifest as cellular signs of myotendinous/aponeurosis junction failure. Interestingly, these theoretically derived conditions are well matched by real-life observations [18]. Consequently, reduced eccentric strength expression due to the presence of neural inhibition may well

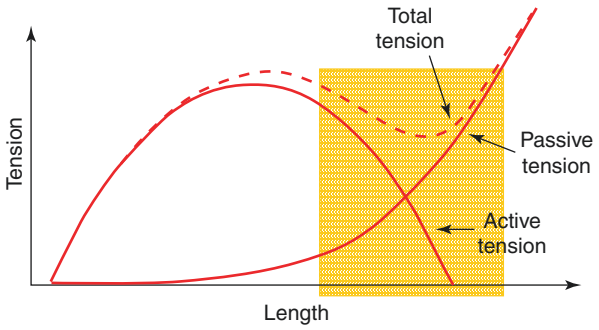

Fig. 5.3 The length-tension relationship for skeletal muscle, illustrating the contribution of active (i.e. contractile) and passive (i.e. series and parallel elastic) components to total tension

elevate the risk of hamstring strain injury (HSI) [19] due to an increased mismatch between active and passive force-generating structures. Conversely, increasing maximal eccentric hamstring muscle strength as a result of training-induced gains in neural drive (resulting from reduced spinal motor neuron inhibition, as discussed above) reduces the mismatch between active and passive tissue force generation, which would be expected to contribute to a reduced risk of HSI. Evidence for the role of hamstring strength and strength-endurance as a risk factor for subsequent hamstring muscle strain injury is discussed below.

5.1.4 Hamstring Strength, Endurance and Injury Risk

Eccentric knee flexor weakness is arguably the most commonly cited risk factor for HSI [20–22]. However, as discussed in Chap. 4, the results from prospective investigations are mixed and a recent meta-analysis of isokinetic studies concluded that eccentric knee flexor strength is only weakly associated with hamstring injury [21]. In the largest of these studies, involving 190 hamstring strains in 614 elite Qatari footballers, lower levels of eccentric knee flexor strength significantly increased the risk of future hamstring injury (odds ratio = 1.37; 95% CI = 1.01–1.85) [23]. However, the strength difference between subsequently injured and uninjured players was extremely small (9.1 Nm; effect size <0.2), which indicates that it would be impossible to distinguish between these individuals clinically. Elite Australian rules football [24] and professional soccer players [25] with lower levels of eccentric knee flexor strength (<279 N and <337 N, respectively) during the NHE have been shown to be significantly more likely to suffer hamstring injuries in the following season than stronger players, although contradictory results from studies of similar design [26] and with larger samples [27] have been reported. In the studies to have found an association between eccentric strength and injury rates, interactions were observed between eccentric strength, age and previous hamstring injury, whereby higher levels of strength appeared to counter the risk of injury associated with being older or having a history of hamstring injury [24, 25]. These findings suggest the possibility that eccentric strength may be a more important consideration in athletes who simultaneously present with other predisposing risk factors. However, low to moderate specificities and sensitivities for conventional isokinetic or Nordic knee flexor strength tests suggest that there is very limited value in trying to predict who will sustain hamstring injuries [21, 23, 24, 26]. It is possible that tests of knee flexor strength are simply not specific enough to running and that more specific strength tests may better reflect injury risk.

The effects of fatigue may also limit the value of strength screening because tests are typically performed in a fresh and relatively rested state and this does not reflect the potential impact of repeated sprinting [28–31] or kicking [32]. Eccentric knee flexor strength falls significantly more than concentric strength after a range of running protocols that include repeated high-speed efforts [28–30], and there is some evidence that this is correlated with a decline in surface EMG (sEMG) from the BF

long head (BF_{LH}) but not medial hamstrings [31]. Repeated drop-punt kicking also causes preferentially eccentric weakness, and while this occurs alongside reductions in both BF_{LH} and medial hamstring surface EMG, the decline in eccentric strength is correlated only with sEMG changes in the more frequently injured BF_{LH} [32]. While not conclusive evidence, the contraction-mode-specific decline in strength and surface EMG is consistent with the possibility of a deficit in muscle activation which, during the stretching of isolated animal muscles, decreases the amount of energy absorbed prior to specimen failure [33]. Testing eccentric strength, or perhaps some aspects of sprinting performance such as horizontal ground reaction force [34], before, during and after repeated sprint sessions may therefore prove to be of greater value than assessments performed in a rested state.

Hip extensor endurance may be a risk factor for HSI. For example, Freckleton and colleagues [35] reported that amateur and semi-elite Australian rules footballers who sustained a right limb hamstring injury during follow-up performed significantly fewer single leg bridges on that side than players who did not sustain an injury. However, the same was not true for left leg injuries [35]. Schuermans and colleagues [36] have also provided prospective evidence, suggesting that poor knee flexor endurance is associated with higher risk of HSI.

As discussed in detail below (cf. *Intra and intermuscular coordination*), an altered timing profile in peak hamstring muscle activity and lower amounts of gluteal and trunk muscle activity during the airborne phases of sprinting suggest that imbalances in muscle synergist activation and 'load sharing' may also play a role in subsequent strain injury. These observations underline that neuromuscular factors related to muscle activation and coordination may contribute to the aetiology of muscle strain disorders, independently of muscle strength, endurance and RFD. Importantly, such neural factors most likely will be modifiable with training, which suggests a need to develop and implement specific training exercises that will result in a motor reprogramming into less hazardous muscle activation patterns (discussed in the following subsection). Only few reports exist on the effect of hamstring muscle exercise on the specific activation pattern for this muscle group. One study demonstrated that 6 weeks of training with the NHE stimulated increased surface EMG activity of both the semitendinosus (ST) and BF during the performance of the exercise [37]. However, more work is required to validate the transfer effect of specific exercise training on the pattern of hamstring muscle activity during sports activities that involve a high risk of HSI (e.g. sprinting).

5.2 Intra- and Intermuscular Coordination

While the BF_{LH} is the most commonly injured hamstring during high-speed running, it has been proposed that injury risk is not only related to neuromuscular characteristics of this muscle but also to the coordination and the relative contribution of the other hamstrings (*intra*muscular coordination) [36, 38] and lumbopelvic muscles (*inter*muscular coordination) [39–41].

5.2.1 Intramuscular Coordination of the Hamstrings

Intramuscular hamstring coordination refers to the spatial and temporal patterns of hamstring muscle activation during planned movement. Schuermans and colleagues [38] were the first to propose that altered intramuscular coordination (i.e. between the lateral and medial hamstrings) might contribute to hamstring muscle injury by changing the distribution of load within these muscles. In this study [38], amateur male soccer players completed leg curl exercise until task failure (when they could no longer maintain the required cadence) with 5-kg weights attached to their ankles. Functional magnetic resonance imaging (MRI) assessments of hamstring metabolic activity (from T2 relaxation times) were performed immediately before and after exercise. Participants with a previous history of hamstring injury displayed lower metabolic activity from the ST which was partially compensated for by higher activity from the BF (both heads combined). A prospective follow-up study [36] of 44 male amateur soccer players demonstrated that this greater reliance upon the BF was associated with an increased susceptibility to primary HSI in the following 1.5 seasons. Subsequently injured players also reached task failure in the leg curl test significantly earlier than those who remained injury-free. Schuermans and colleagues [36] suggested that a relatively high reliance on BF was associated with reduced endurance, and this is at least partly supported by more recent sEMG findings, which showed that a disproportionate reliance upon any of the hamstring muscles was related to poor endurance when 20% of maximal knee flexor force was held until task failure [42]. These findings suggest that intramuscular coordination makes a significant contribution to hamstring fatigue [36, 38, 42] and injury risk [36], presumably via its influence on 'load sharing' between the hamstring muscles. The prospective observation [36] also suggests that the imbalanced load sharing observed in prolonged isolated knee flexion exercise may also be evident in high-speed running, although this has yet to be observed.

While we currently do not know how to alter the relative reliance upon different hamstring muscles, these findings suggest that to adequately protect athletes from running-related hamstring injury, training should not focus solely on stimulating the BF, but that just as much attention should be given to conditioning its agonists and possibly specifically targeting the ST [36, 38]. Interestingly, the NHE preferentially activates the ST [43], and this observation might partly explain why this simple eccentric hamstring exercise has proven successful in primary and secondary HSI prevention [44, 45].

5.2.2 Intermuscular Coordination: More Than Just a 'One-Muscle Job'

To effectively contribute to the development of horizontal ground reaction forces in running, the hamstrings need to be coordinated with both synergists at the hip and knee and stabilisers at these and adjacent joints. Recently, Schuermans and colleagues investigated lower limb and lumbopelvic kinematics [40] and muscle

activity patterns [39] by performing three-dimensional (3D) motion capture and sEMG measurements between the 15th and 25th metre of 40-m straight line sprints (Fig. 5.4). Sprint techniques were examined in a cohort of 29 male football players using statistical parametric mapping, which allowed investigation of kinematics across the entire gait cycle rather than looking at more discrete time intervals just before or at touch down. Four players who went on to sustain hamstring strains exhibited greater anterior tilt of the trunk and pelvis and higher levels of lateral

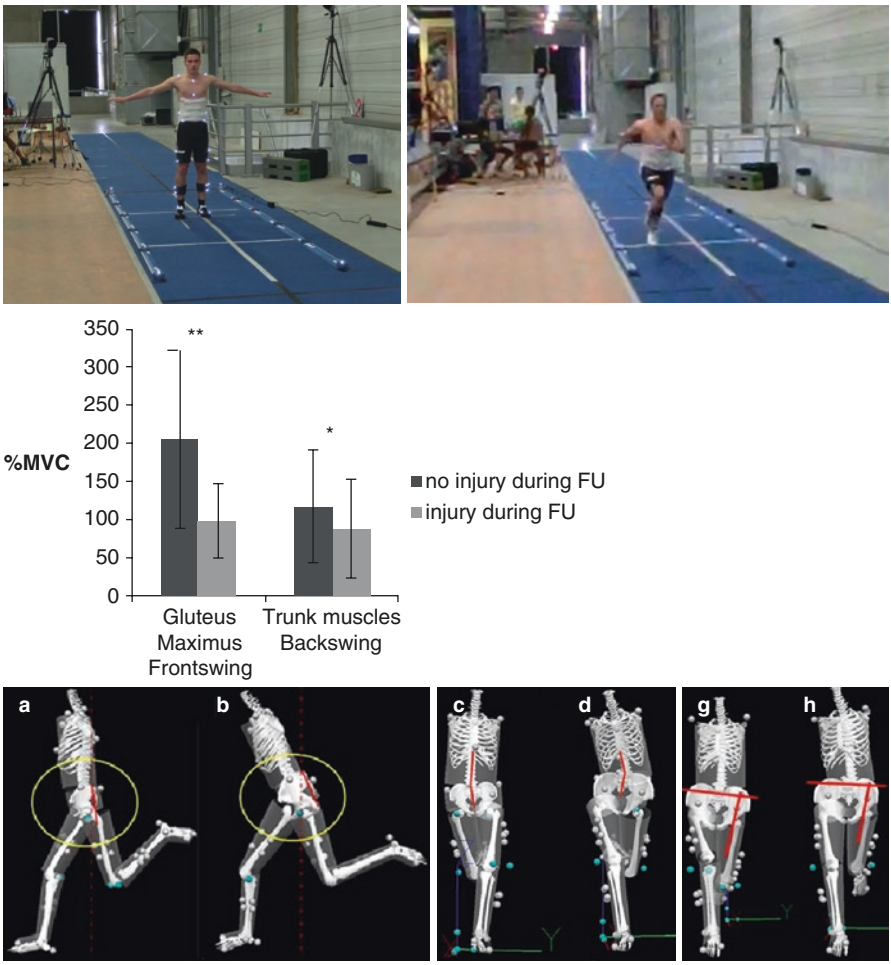

Fig. 5.4 Top: 3D kinematic and sEMG analysis of maximal overground sprint. Middle: Players who subsequently sustained hamstring injury displayed lower gluteus maximus (GM) EMG in forward swing and lower trunk muscle EMG during back swing (airborne) phases of high-speed running than those without injury. Bottom: Soccer players who subsequently sustained hamstring injury during follow-up (FU) demonstrated more anterior pelvic tilt (**b**) and thoraco-pelvic lateral flexion (**d**) than players who did not sustain injury (**a** and **c**). (Reproduced from Schuermans et al. [39, 40], with permission)

trunk flexion than 25 players without injury [40] (Fig. 5.4). Of 51 players who performed preseason sprinting with sEMG analysis, 15 went on to sustain a hamstring injury in the subsequent 18 months (1.5 seasons) [39]. Those who suffered hamstring injury exhibited lower levels of normalised gluteus maximus (GM) activity in forward swing and lower levels of trunk muscle activity (obliques and erector spinae muscles combined) in back swing than players who remained injury-free (Fig. 5.4). These between-group differences reached their maximums near the end of the front swing for GM activation and at the end of the backswing for the trunk muscles. The results are supportive of prior biomechanical modelling [46] which suggested that the coordination of lumbopelvic muscles plays an important role in protecting the hamstrings during high-speed running. Chumanov and colleagues [46] suggested that small deviations in the level of lumbopelvic muscle activation would alter the strains experienced by the BF_{LH} in the late forward swing phase of gait, with excessive hip flexor activity and insufficient hip extensor activity both having potential to increase BF_{LH} strain and the risk of injury.

Schuermans and colleagues [41] also conducted a prospective study to examine whether the timing of sEMG onset of the lateral and medial hamstring, GM and erector spinae muscles during prone hip extension had any association with hamstring injury susceptibility (Fig. 5.5). Fifty-one amateur male soccer players performed three prone hip extensions on verbal command and were then followed for injury across 1.5 seasons, during which 15 players sustained hamstring injuries. No significant differences were observed in erector spinae or GM sEMG onsets between subsequently injured and uninjured players. However, subsequently injured players did display a significantly greater delay in hamstring sEMG onset than those with no subsequent injury. No differences were found in sEMG amplitude or contraction intensity of any of the investigated muscles. These findings suggest that the temporal coordination of the hamstrings along with the GM and erector spinae muscles may play a role in safe hamstring functioning and primary injury prevention. Previous hamstring injury history had no influence on the timing of sEMG onset [41]. If a similar delayed onset of hamstring activity were to occur during sprinting, it may contribute to altered loading, potentially increasing the strain that these muscles experience during the terminal-swing phase of gait.

5.3 Structural Factors Relating to Hamstring Muscle Function, Performance and Injury

At the simplest level, muscles are collections of sarcomeres which are laid in series and in parallel with each other so as to span the distance between the aponeuroses and tendons from which they originate and insert. The number of in-parallel sarcomeres determines the physiological cross-sectional area (PCSA) and, to a significant extent, the muscle's force-generating capacity [47]. The

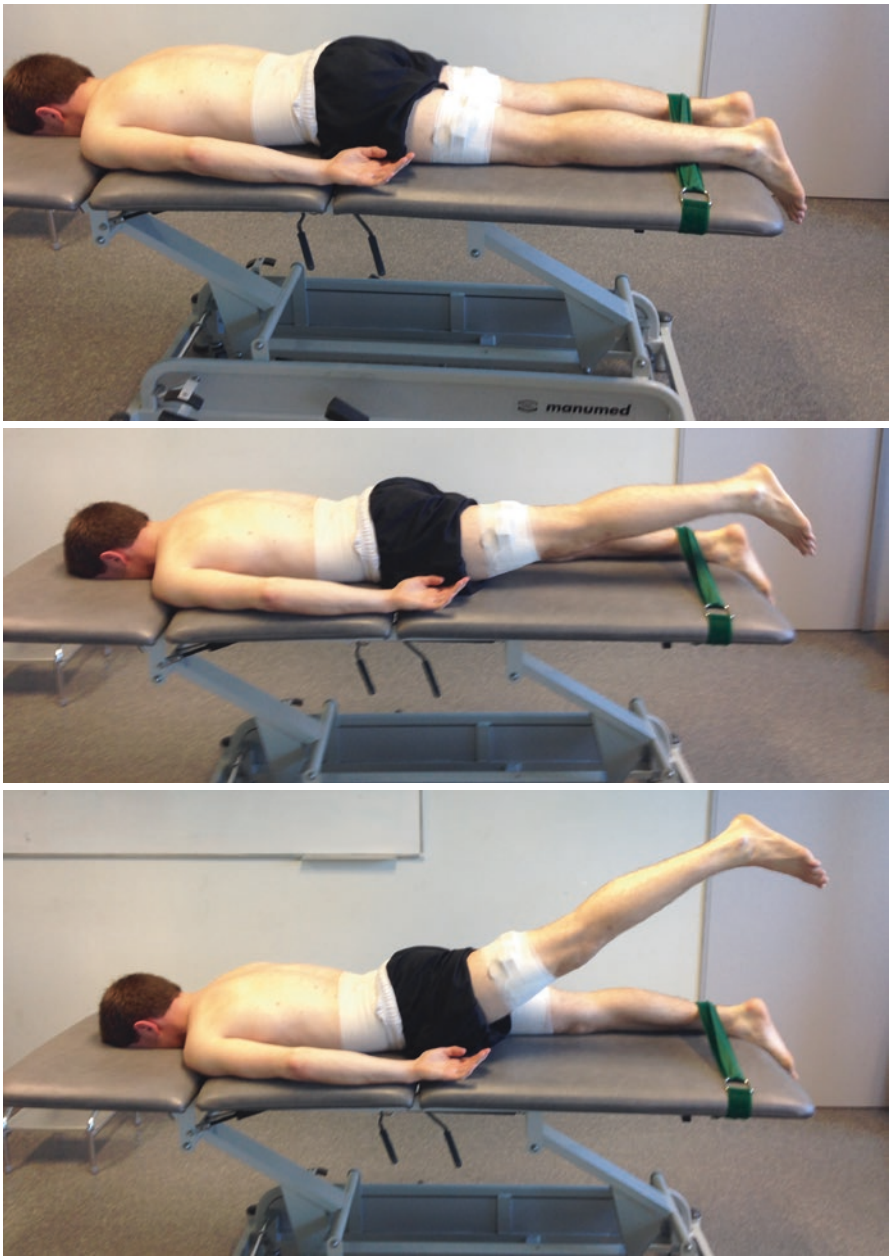

Fig. 5.5 Prone hip extension test with sEMG measurement of posterior chain muscle recruitment

number of in-series sarcomeres influences contractility (the extent to which a muscle can shorten), the theoretical maximum rate at which it can shorten (if completely unloaded) and, to some extent, its force generation while shortening [47]. This latter effect comes about because in muscle shortening at a given rate, sarcomere shortening rates are lower (and force accordingly higher), when there are more contractile elements in series.

The orientation of muscle fascicles and their constituent fibres relative to the long axes of the muscle and the aponeuroses (the pennation angle) also has a significant impact on muscle performance because this dictates the relative distribution of sarcomeres in series and in parallel. As a consequence, a strap-like muscle with a close to zero pennation angle will generate less isometric force but exhibit a greater contractility and a faster maximum rate of shortening than a pennate muscle of equal volume. These two muscles will also have equal peak power outputs, because this measure is proportional to muscle volume, but the strap-like muscle will generate its peak power at higher rates of shortening [47]. The 'gearing' of pennate muscles also has an impact because fascicle angle and length changes both contribute to tendon excursion, and this allows fibres to contract more slowly and undergo smaller length changes than the whole muscle-tendon unit (MTU) [48]. Slower fibre shortening enhances force generation in accordance with the force-velocity curve and shortening across a smaller range potentially allows for the muscle to remain close to its optimal length.

As discussed in Chap. 1, the morphological and architectural characteristics of the human hamstrings have received significant research attention. Early studies relied predominantly upon cadavers for assessments of muscle volume, fascicle lengths, pennation angles and estimates of PCSAs [49–51], and these parameters are now also being determined via scanning technologies such as MRI and ultrasound (US) [52–55]. Studies of healthy 'active' humans [56] and sprint-trained track and field athletes [53] have revealed significantly larger hamstring muscle volumes and PCSAs than those reported for cadaveric samples which almost invariably come from the sedentary elderly [49]. Table 5.1 shows results from a selection of studies that examined hamstring muscle volumes in young adults [42, 52, 53, 56]. Avrillon and colleagues [42] also assessed hamstring fascicle lengths and pennation angles (at multiple positions along each muscle) via panoramic B-mode US and then estimated PCSA for BF_{LH} and biceps femoris short head (BF_{SH}) and semimembranosus (SM) muscles from the equation:

$$\text{PCSA} = \left(\text{muscle volume} / \text{fascicle length}\right) \times \text{cosine}\left(\text{pennation angle}\right).$$

Here the ST muscle was deemed to be fusiform (pennation angle was taken as zero) and its PCSA was determined by its volume divided by its length [42]. Handsfield and colleagues [56] employed MRI measures of muscle volumes and combined these with the architectural features from cadaver studies. These PCSA measurements (Table 5.1) suggest that the maximum force-generating capacity is ranked SM > BF_{LH} > BF_{SH} > ST [42, 56].

Table 5.1 Hamstring muscle size and architecture measurements or estimates from four studies [42, 52, 53, 56]

	Muscle	Avrillon et al.	Handsfield et al.	Handsfield et al.	Bourne et al.[1]
Participants		Healthy adults 16 female 19 male Dominant limb	Healthy adults 8 female 16 male	NCAA Div I Sprint and jump athletes 7 male 8 female	Recreational level athletes in field and court sports 30 male Right limbs
Age (years)		24 (3)	25.5 (11.1)	18 (0.6)	22.0 (3.6)
Height (cm)		173 (9)	171 (10)	176.8 (8.1)	180.4(7.0)
Body mass (kg)		66 (11)	71.8 (14.6)	68.9 (8.5)	80.8(11.1)
Volume (cm^3)	BF_{LH}	184.6 (41.6)	206.5 (48.4)	262 (43)	235.7 (38.6)
	BF_{SH}	86.3 (37.6)	100.1 (32)	127 (32)	127.3 (27.8)
	ST	173.4 (67.3)	186.0 (47.0)	289 (72)	247.1 (54.3)
	SM	207.5 (56.7)	245.5 (54.2)	297 (61)	269.9 (42.6)
PCSA (cm^2)	BF_{LH}	15.2 (3.6)	25.9 (4.9)		
	BF_{SH}	7.9 (3.3)	7.8 (1.8)		
	ST	10.2 (3.9)	9.3 (2.3)		
	SM	23.4 (7.6)	37.8 (9.1)		
Fascicle length (cm)	BF_{LH}	12.1 (1.7)			10.6 (1.0)
	BF_{SH}	10.7 (1.4)			
	ST	17.2 (4.4)			
	SM	8.9 (1.4)			
Pennation angle (degrees)	BF_{LH}	9.0 (1.6)			13.6 (1.2)
	BF_{SH}	12.4 (2.4)			
	ST	0 (assumed value)			
	SM	10.7 (2.0)			

BF_{LH} biceps femoris long head, *BF_{SH}* biceps femoris short head, *ST* semitendinosus, *SM* semimembranosus, *PCSA* physiological cross sectional area

[1]Pre-training data from the right limbs of 30 participants, not previously reported, from Bourne et al. [52]

Direct comparisons between the results of different studies must be made with caution because of methodological differences and the use of mixed sex cohorts with varying proportions of male and female participants. Nevertheless, Table 5.1 shows a general trend for hamstring muscles to be larger in athletes. It is also worth noting that the relative sizes of the hamstring muscles vary significantly between cadavers [49] and athletes [52, 53]. For example, Ward and colleagues [49] reported that the ST mass was ~74% of that of the SM in cadavers and this is consistent with the relative volumes of these two muscles in healthy adults (~76%) [56]. In sprint and jump athletes, however, the ST and SM have almost identical volumes (ST is 97% of SM volume) [53].

Handsfield and colleagues [53] have compared lower limb muscle volumes in sprinters and jumpers (hereafter referred to as 'sprint-trained' athletes) to those of sedentary young adults [56]. Volumes were determined via MRI and corrected for body size by being expressed relative to the product of height and mass. The greatest degree of relative hypertrophy of all lower limb muscles was observed for the

ST, which was 54% larger (relative to body size) in sprint-trained athletes. By contrast, the BF_{LH} and BF_{SH} were both 26% larger, SM 20% larger, GM 31% larger and adductor magnus (AM) 26% larger in the athletes. Hip flexor muscles, the tensor fascia latae, rectus femoris and sartorius were also particularly hypertrophied in athletes, with relative volumes 42%, 40% and 37% larger than non-athletes, respectively. At the other end of the spectrum, some muscles like the gluteus medius (+8%), lateral (+7%) and medial gastrocnemius (+4%) displayed little 'relative' hypertrophy according to volume differences [53]. However, some of these muscle bellies may have been shorter in sprint-trained athletes and could potentially have been relatively large in terms of PCSA. For example, sprint-trained athletes have been reported to have thicker lateral gastrocnemius muscles than sedentary adults [57, 58]. While a degree of muscle size difference may have pre-dated sport involvement and potentially helped to 'select' certain individuals as speed athletes, these data also suggest the possibility that high-speed running and the associated strength and conditioning programmes place significantly greater 'overload' on some muscles than others.

Hamstring muscle morphology also varies considerably between individuals with similar training status and this is often overlooked because we typically see means and standard deviations presented in the literature. So, while SM is fairly consistently reported as the largest of the hamstrings by volume (Table 5.1), Bourne and colleagues observed that it was the largest in only 16 of 30 young adult men who were engaged in recreational sport, while ST and BF_{LH} were the largest by volume in 10 and 4 men, respectively [52]. Furthermore, the BF_{LH} was small as 23% and as large as 35% of the total hamstring volume in the two athletes with the smallest and largest BF_{LH} proportions. Similarly, ST volume ranged from 24% to 39% of the total hamstring volume and similar variability was observed for anatomical cross-sectional areas (ACSAs). These findings suggest considerable inter-individual variability in the hamstring muscles with the greatest potential power outputs and force generation capacities. As yet it is unknown whether this variability has any impact on running performance or the risk of HSI.

The joint torque created by a given skeletal muscle is the product of its force and the length of its moment arm, and sagittal plane hamstring moment arms at the knee vary between hamstrings and with knee angle. For example, the ST moment arm (5.7 ± 0.7 cm) is larger than that of the SM (4.8 ± 0.5 cm) and BF muscles (4.6 ± 0.4 cm) when the knee is flexed by 45° [42]. This partially compensates for the ST's relatively small PCSA, but in untrained adults, this muscle is still estimated to have 53% and 56% as much torque-generating potential in isometric contractions as the SM and BF muscles, respectively [42]. ST and BF moment arms shorten appreciably as the knee extends between angles of 90° and 0° of flexion, while the SM moment arm remains relatively constant in this range [59]. Consideration of muscle PCSAs [56] and moment arms suggests that the hamstrings are the major providers of knee flexor torque, although a considerable contribution is potentially made by the gastrocnemius which has a large PCSA but relatively small moment arms [60].

Hamstring sagittal plane moment arms at the hip are larger for the ST (5.6 cm) and BF (5.4 cm) than the SM (4.6 cm) [61], and these change across the hip's range

of motion, reaching their peaks between ~30° and 50° of flexion [62]. By contrast, the moment arm of the GM declines, while that of the AM increases considerably between the hip angles of 0° and 90° of flexion. These changes suggest a highly variable contribution to total hip extension torque from the GM and AM across the range of motion (ROM) while the hamstrings' contribution is likely more constant. Furthermore, the estimated PCSAs of the combined hamstrings (~81 cm^2) is substantially larger than that of the GM (~47 cm^2) and AM (~46 cm^2) [56] suggesting that the hamstrings are particularly strong hip extensors. Nevertheless, given the significant potential for non-hamstring muscles to generate torque, neither knee flexor nor hip extensor strength tests can be considered as assessments of hamstring strength alone.

5.3.1 Muscle Architecture and Function

Vastus lateralis and gastrocnemius fascicles, expressed in absolute terms or relative to limb lengths, are longer in track and field sprinters than in sedentary adults and generally longer in sedentary adults than in endurance-trained runners [57]. Furthermore, fascicle lengths in these muscles are significantly correlated with 100-m sprint performance in male athletes whose best times ranged from 10.0 to ~11.7 s [58] and in female athletes with best times between ~11.0 and 13.4 s [63]. In these studies, sprint-trained athletes exhibited smaller vastus lateralis pennation angles than distance runners, although little difference existed between these groups' gastrocnemius pennation angles [58, 63]. As the data is retrospective, it is unwise to attribute fascicle length differences to training programme design. Nevertheless, it is likely that longer fascicles are well suited to high-velocity and high-power activities. Furthermore, shorter fascicles are well suited to endurance activities because of the efficiency of having fewer energy-consuming sarcomeres in series. At the time of writing, we are not aware of studies that have compared hamstring fascicle lengths or pennation angles in different athletic groups or attempted to correlate these lengths with sprint performance.

5.3.2 Hamstring Fascicle Lengths, Pennation Angles and Injury Risk

Fascicle lengths and pennation angles vary considerably between the hamstring muscles [42] and, as discussed in Chap. 1, even along the lengths of individual muscles. Fascicles are longest in the ST and shortest in the SM, while pennation angles are highest in the SM and lowest in the ST, although some differences exist between studies (see Table 5.1). These observations suggest that the ST should be able to generate significant forces across a large ROM and when they are shortening rapidly. By contrast, the SM is thought to be best suited to force and power generation at shorter lengths and at slower speeds.

A single prospective study of 152 elite Australian soccer players has demonstrated that those with shorter BF_{LH} fascicles (<10.56 cm) were ~four-fold more likely to sustain a future HSI than those with longer fascicles [25]. Furthermore, the probability of injury was reduced by 21% for every 1-cm increase in fascicle length, while pennation angle and muscle thickness had no association with injury rates. In this study, interactions were also observed between fascicle length, age and prior hamstring injury, whereby longer BF fascicles countered the risk of injury associated with being older or having a history of this injury (Fig. 5.6) [25]. Other studies have reported shorter BF fascicles in limbs with a history of injury to this muscle [64]. Furthermore, the fascicles of previously injured BF muscles have been reported to lengthen less in response to the demands of preseason Australian rules football training than those in uninjured muscles [65]. It has been proposed that short fascicles, with fewer in-series sarcomeres, are more susceptible to damage as a consequence of sarcomere overextension during active lengthening [66].

There is a pressing need to more conclusively establish fascicle length as a risk factor for HSI, and the inconsistency of risk factor studies [21, 67] should be considered when designing a training programme with increasing fascicle lengths in mind. It should also be acknowledged that there are limitations to the methods that have most often been employed to estimate skeletal muscle fascicle lengths in vivo [68]. Firstly, two-dimensional US cannot determine the lengths of all fascicles in a complex three-dimensional structure. Secondly, estimates of fascicle length (generally in the region of 9–13 cm) have typically been made on the basis of extrapolating fascicle and aponeurosis structures visualised within the ~4- to 4.6-cm wide fields of view (FOV) with straight lines outside the FOV. In reality, fascicles and aponeuroses are often curved and this may lead to considerable errors in the estimates of fascicle lengths and muscle fibre pennation angles [68].

5.3.3 Altering Muscle Architecture: The Roles of Contraction Mode and Muscle Excursion

There is mounting evidence that eccentric knee flexor training results in lengthening of the BF_{LH} fascicles [52, 55, 69–76], although this adaptation has not always been observed [54, 73]. For example, Lovell and colleagues [73] reported fascicle lengthening when the NHE was performed before but not after soccer training. Increases in fascicle lengths have also been found after both high- and low-volume hamstring training [74] (see Table 5.2). There is also emerging evidence for fascicle lengthening in the ST after NHE training [70] and in the SM after combined NHE and eccentric stiff-leg deadlift training [77].

The findings that knee flexion tasks do not selectively activate the BF_{LH} but nevertheless evoke significant (~10–24%) increases in estimated fascicle lengths [52] might suggest that high levels of activation are not necessary for stimulating architectural changes in the hamstrings. However, absolute levels of BF EMG activity

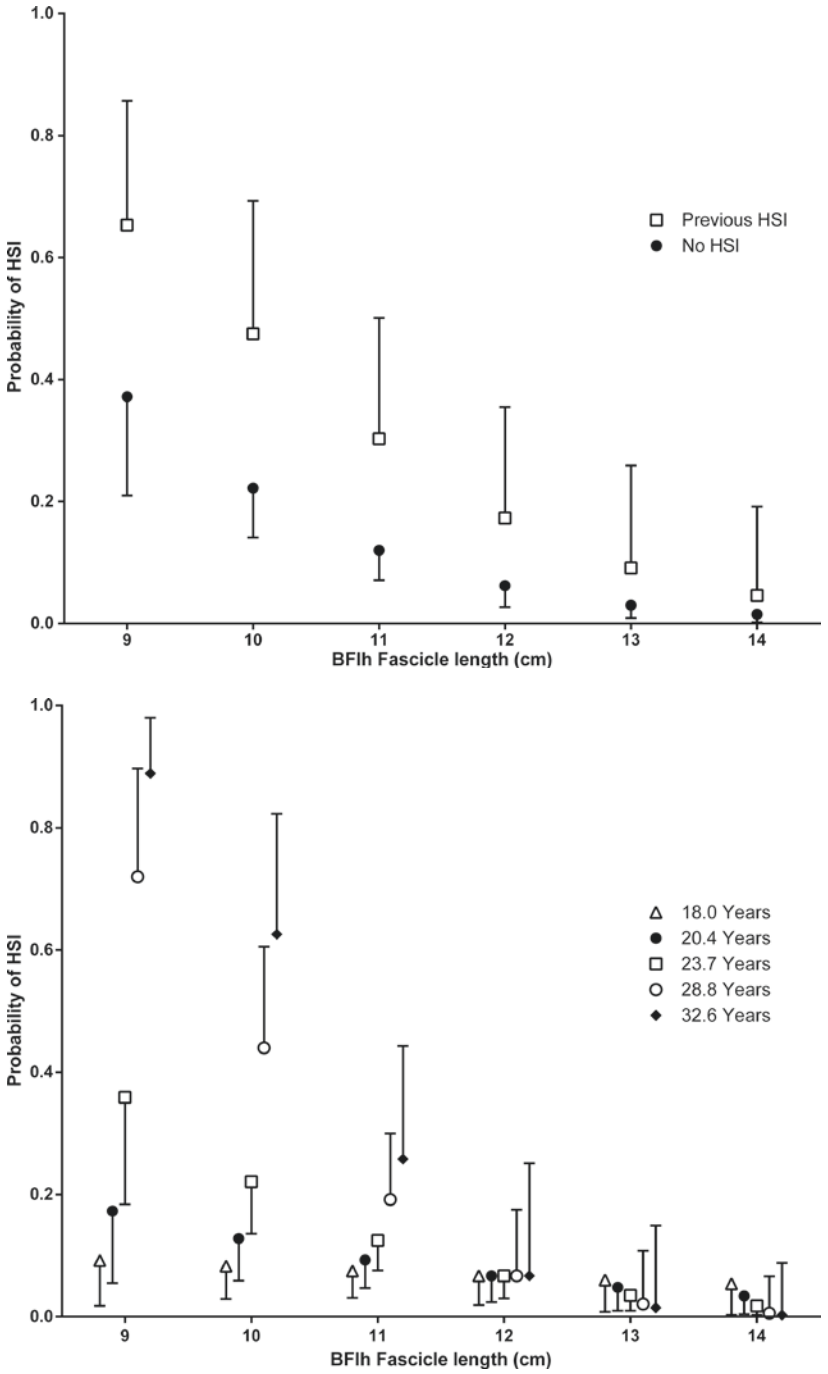

Fig. 5.6 Top: The interaction between BF_{LH} fascicle length and history of HSI and the probability of future HSI. Bottom: The interaction between BF_{LH} fascicle length and age and the probability of future HSI. Error bars represent 95% confidence intervals. (Replicated from Timmins et al. [25], with permission)

Table 5.2 Strength training interventions that have assessed architectural adaptations to the BF_{LH}

Study	Exercise	Contraction mode(s)	Peak MTU length	Intensity	Maximum volume (sets × reps/ session)	Maximum frequency (sessions/ week)	BF_{LH} fascicle length % change
Presland et al. [74]	Nordic	Ecc	Mod	Extra loads	5 × 10	2	+23
	Nordic	Ecc	Mod	Extra loads	4 × 6	2	+24
Duhig et al. [76]	Nordic	Ecc	Mod	Extra loads	5 × 6	2	+3
	Leg curl	Conc	Mod	6–8RM	5 × 6	2	−6
Ribeiro-Alvares et al. [69]	Nordic	Ecc	Mod	Body mass	3 × 10	2	+22
Alonso-Fernandez et al. [70]	Nordic	Ecc	Mod	Body mass	3 × 10	3	+23.9
Seymore et al. [54]	Nordic	Ecc	Mod	Body mass	3 × 8–12	3	+0.0
Bourne et al. [52]	Nordic	Ecc	Mod	Extra loads	5 × 10	2	+21
	Hip extension	Conc + Ecc	Long	6–10RM	5 × 10	2	+13.2
Pollard et al. [75]	Nordic (with extra load)	Ecc	Mod	Extra loads	4 × 6	2	+15.9
	Nordic (with body mass)	Ecc	Mod	Body mass	4 × 6	2	+6.0
	Razor curl	Pseudo-isometric	Mod	Extra loads	4 × 6	2	−0.5
Timmins et al. [55]	Seated isokinetic knee flexion	Ecc	Long	Max effort	6 × 8	3	+16
		Conc	Long	Max effort	6 × 8	3	−11.8
Guex et al. [71]	Seated isokinetic knee flexion	Ecc	Long	Max effort	5 × 8	3	+9.3
	Lying isokinetic knee flexion	Ecc	Short	Max effort	5 × 8	3	+4.9
Potier et al. [72]	Leg curl	Ecc	Mod	1RM	3 × 8	3	+34

Ecc eccentric, *Conc* concentric, *MTU* muscle-tendon unit, *Extra loads* performed with body mass plus extra loads, *Body mass* performed with body mass only, *Mod* moderate, *RM* repetition-maximum, *Max effort* maximal voluntary effort

are high in the Nordic exercise [43], and there is also evidence that when low-volume NHE training (eight repetitions per week) is conducted, higher intensities (obtained by holding 5- to 30-kg weights on the chest) are more effective at inducing fascicle length change [75].

Hip extension training using a 45° Roman chair and conventional loading (concentric and eccentric loads were identical) has been shown to lengthen BF_{LH} fascicles in recreationally active men [52]. This is the only training study of which we are aware to have examined the effect of a purely hip extension intervention on BF_{LH} muscle architecture. The effects of the commonly employed Romanian or stiff-leg deadlifts are as yet undetermined.

It is worth noting that despite theories to the contrary [78], the moderate hamstring muscle lengths experienced during the eccentric NHE are not a barrier to fascicle lengthening. In fact, when directly compared, the NHE and the 45° hip extension were shown to stimulate statistically indistinguishable increases in BF_{LH} fascicle lengths, although the mean changes favoured the NHE (21% vs. 13%) [52]. Furthermore, concentric training at long [55] and moderate [76] muscle lengths has been reported to reduce BF_{LH} fascicle lengths so it appears that contraction mode exerts a powerful effect on architectural adaptations. Nevertheless, muscle excursion is also likely to influence fascicle length changes and the one study to have examined long- versus short-length eccentric training reported a statistically insignificant trend for greater fascicle lengthening after long-length training [71].

At the time of writing, we are not aware of any published studies that have examined the impact of sprint training on hamstring fascicle lengths. Nevertheless, increases in vastus lateralis and rectus femoris fascicle lengths have been reported after 5 weeks of sprinting and bounding training in recreationally active individuals [79], so there is nothing infeasible about fascicle length changes in response to high-speed running programmes.

The BF_{LH} fascicle length increases induced by eccentric hamstring training occur very rapidly (within 2 weeks of training with an isokinetic dynamometer) and are lost within 1–4 weeks once training is stopped [55, 74, 75]. The shortening induced by concentric training is also noted within 2–4 weeks but the changes appear to be smaller and more persistent than those seen after eccentric training. For example, Timmins and colleagues [55] observed that the fascicle shortening during 6 weeks of a concentric isokinetic intervention persisted at least for 4 weeks after the cessation of training. The rapidity of some of these changes has prompted doubt as to whether or not the estimates of fascicle length are valid, primarily because some believe that 2 weeks is too short a time for meaningful architectural adaptations to occur within humans. We should also consider the possibility that fascicle lengthening may not be the mechanism that mediates the protective effect of eccentric interventions. Changes in the composition of connective tissue, as discussed below, or other adaptations are possible. However, a muscle's resistance to the damage caused by eccentric exercise is significantly improved by a single exposure to a small number (6–30) of strong eccentric actions [80]. So, muscle resistance to microtrauma can change drastically with a small number of exercise sessions and this is central

to the rationale for eccentric exercise in hamstring injury prevention [81–84]. However, the repeated bout effect is also known to last for weeks and months [80] while fascicle length changes are reversed more rapidly than this.

Regardless of whether fascicle lengths account for the protective effects of eccentric training, we should consider the contrasting effects of purely eccentric and purely concentric exercise on the susceptibility of muscles to exercise-induced damage. Concentric training has been shown to increase the susceptibility of human [85] and animal muscles [83, 84] to eccentrically induced damage and this may translate to a greater risk of strain injury but will almost certainly influence muscle soreness. As a consequence, the balance between eccentric and concentric stimuli should be carefully considered when designing a training programme.

Two studies have reported that BF_{LH} pennation angles declined with eccentric and increased with concentric training, one involving training on an isokinetic dynamometer [55] and the other involving either the NHE or concentric leg curl [76]. Studies employing the NHE have generally reported small to moderate reductions in BF_{LH} [70, 73–75] and ST pennation angles [70]. However, no significant pennation angle changes were observed by Seymore and colleagues [54], who also reported no change in fascicle lengths after NHE. Lovell and colleagues [73] observed a decrease in pennation angle when the NHE was employed before but not after soccer training and the reduction in pennation angles occurred along with an increase in fascicle length. In fact, across most hamstring architecture studies, there is a trend for pennation angles to decrease as fascicle lengths increase [70, 73–75]. One eccentric leg curl study reported no changes in pennation angle despite very large increases in fascicle length increases [72]; however, this study differed from all others mentioned here in that the US assessments of BF_{LH} were taken at the distal end of the muscle.

5.3.4 Muscle-Tendon Junction Morphology

The muscle-tendon junction (MTJ) represents the interface between muscle and tendon and is mechanically the weakest part of the MTU [86]. The majority of running-induced hamstring strains affect the proximal MTJ of the BF_{LH} [87], which is also the site of greatest localised tissue strains during active lengthening [88]. Recent work has suggested that the morphology and composition of the proximal MTJ may be associated with its increased propensity for damage.

5.3.5 Aponeurosis Geometry

A narrow proximal BF aponeurosis and a large muscle to aponeurosis width ratio have been proposed as potential risk factors for future HSI [89]. Biomechanical modelling [88, 90] has demonstrated that the geometry of this structure strongly influences the location and magnitude of strain within the BF. For example, an

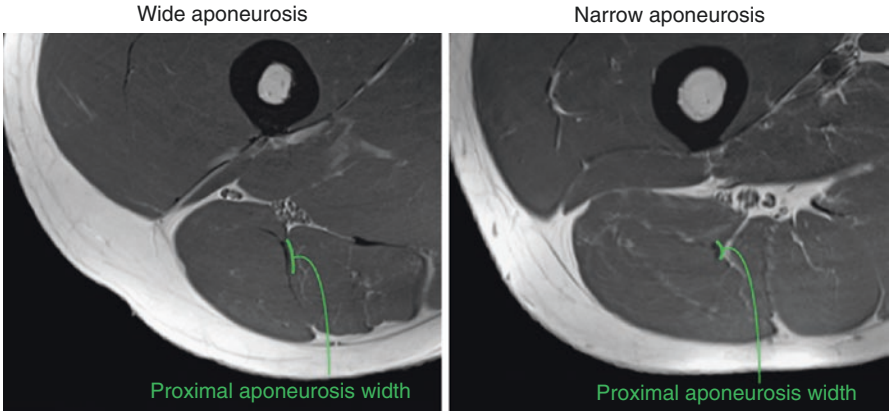

Fig. 5.7 BF aponeurosis width measurements showing a wide and narrow aponeurosis. (Reproduced from Fiorentino et al. [90])

80% reduction in the width of the proximal BF aponeurosis increases strain within the commonly injured proximal MTJ by 60% [88]. Recent work has also identified substantial interindividual variability in the size of the proximal BF aponeurosis and the muscle to aponeurosis width ratio [89, 90] (Fig. 5.7), although no prospective study has explored whether these factors are associated with hamstring injury risk.

If the aponeurosis to muscle width ratio is established as a risk factor in the future, interventions which increase the size of the proximal aponeurosis while having minimal effects on BF size may be valuable for mitigating the risk of running-induced HSI. However, no study to date has assessed training-induced adaptations to BF aponeurosis. Observations from other muscle groups suggest that aponeurosis surface area may increase as a consequence of skeletal muscle hypertrophy, although in the short term at least, muscle dimensions increase significantly more and this may increase muscle fibre strains rather than reduce them. For example, 12 weeks of unilateral knee extensions evoked a 1.9 ± 3.8% increase in the width of the vastus lateralis distal aponeurosis and a 10.7 ± 7.6% increase in its ACSA [91]. However, weightlifters have been reported to display 32% larger vastus lateralis aponeuroses than untrained individuals [92] and this raises the possibility that long-term training may have relatively larger and potentially positive effects.

If aponeurosis to muscle width ratio is established as a risk factor for hamstring injury, subsequent work might then examine the impacts of altering this ratio to differing extents with exercises that target the BF_{LH} to different extents. In this context, it may be worth considering that training with the NHE has relatively small effects on BF muscle volume compared to the hip extension exercise [20].

The collagen composition of the MTJ and its adjoining fibres may be an important factor influencing its susceptibility to damage. Although prospective studies are lacking, a recent training intervention involving individuals

scheduled for anterior cruciate ligament surgery demonstrated that 4 weeks of hamstring strength training involving the NHE, leg curls and hip extensions altered collagen expression in the endomysium of muscle fibres at the distal MTJ of the ST and gracilis [93]. Specifically, the intervention appeared to increase the amount of collagen XIV, a protein that may be important in strengthening the extracellular matrix of the MTJ [93]. These results may provide at least one additional mechanism by which strength training interventions protect against HSI.

5.4 Conclusion

Hamstring function is determined by the interaction of a number of neuromuscular characteristics. The ability to generate force rapidly, particularly during active lengthening, is important for optimal hamstring performance and this is influenced to a significant extent by the capacity to fully voluntarily activate these muscles. Structural features such as muscle volume, PCSA, fascicle lengths and pennation angles are also important determinants of hamstring function, and these vary considerably between the heads of this muscle group and between individuals. While more proof is required, short BF_{LH} fascicles may also increase the risk of strain injuries and there is evidence that fascicle length can be altered relatively rapidly with strength training. The complex coordination of hamstring muscles and their synergists also has a role in determining performance, including the endurance capacity of the knee flexors. There is also emerging evidence that dysfunction in intra- and intermuscular coordination plays a role in hamstring injury causation.

At present, there is ample evidence that muscle morphology and architecture can be altered, at least in uninjured individuals, with well-planned exercise programmes. However, there is currently little understanding of how best to improve the various aspects of lumbopelvic coordination so as to best protect the hamstrings from injury.

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Hamstring Injury Prevention and Implementation

6

Nick van der Horst, Kristian Thorborg, and David Opar

6.1 Introduction

'An ounce of prevention is worth a pound of cure', Benjamin Franklin (1706–1790) once stated. Nowadays, the definitive answer to the prevention of hamstring injuries would probably be worth gold, considering the consequences and cost for the individual and their associated team/organisation due to this most common injury type. Compounding the importance of hamstring injury prevention is the well-established knowledge that prior injury is the strongest predictor of future hamstring injury. As a result, the prevention of an initial hamstring injury can allow an individual to avoid the potential injury-reinjury cycle.

In the last few decades, worldwide initiatives have been undertaken to develop strategies for sports injury prevention. There has been an exponential increase in knowledge, research, technological developments, implementation efforts and even international conferences focusing on sports injury prevention. Consequently, an increasing amount of evidence is available for clinicians and practitioners to inform a hamstring injury prevention strategy for any individual athlete or team.

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Injury prevention can be divided into primary, secondary and tertiary approaches [1]. Primary prevention relates to the prevention of the initial event. This is done by preventing exposures to hazards that cause injury, altering unhealthy behaviour and increasing resistance to injury when exposure occurs. Secondary prevention aims to reduce the impact of the injury that has already occurred. This is done through counselling about reinjury prevention and proper rehabilitation strategies. Examples of secondary hamstring injury prevention are prevention and treatment protocols that aim for optimal hamstring health and recovery with minimal risk of injury/reinjury. Tertiary prevention aims to soften the impact of an (ongoing) injury that has lasting effects. When discussing hamstring injury tertiary prevention, this relates to hamstring injury rehabilitation (Chap. 10) and if conservative rehabilitation and primary prevention fails (Chap. 13).

The purpose of this chapter is to inform the reader about strategies for primary prevention of hamstring injury, noting that secondary prevention and tertiary prevention will also be covered later in this book (Chaps. 9, 10, 11, 12 and 13, respectively). Given the increase in available knowledge, hamstring injury prevention strategies should be informed by the best available evidence [2]. For the sake of this chapter, the strongest available evidence, level 1a evidence, is deemed to come from systematic reviews with meta-analysis. Level 1b evidence, which is also considered high-level evidence, comes from randomised control trials (RCTs) that employ interventions aimed at reducing the rate/incidence/frequency of hamstring injury. Although of lower quality, findings from lower levels of evidence such as level 2 (cohort studies and low-quality RCTs), level 3 (case-control series), level 4 (case series and poor-quality cohort studies) and level 5 (expert opinion) will also be discussed in this chapter (see Fig. 6.1).

Whilst many applications (i.e. massage, foam rolling, dry needling, acupuncture, taping techniques) have been popularised to varying extent in practice, there is little to no evidence of sufficient quality to support their efficacy, and these will not be discussed in this chapter.

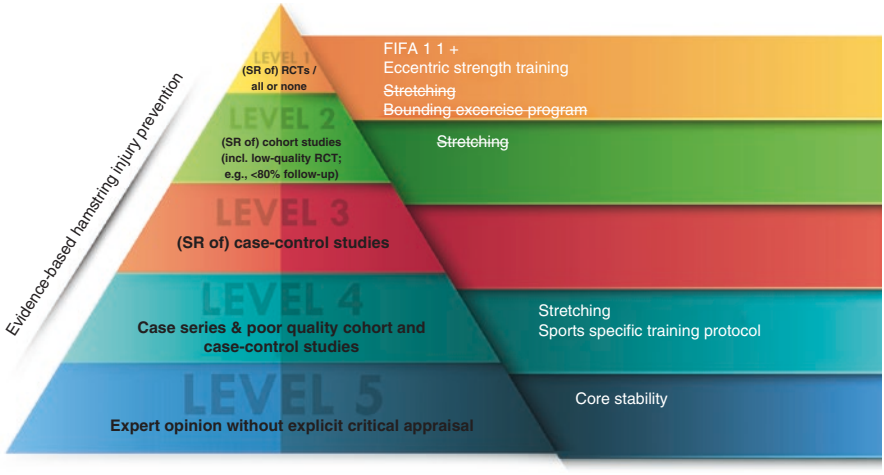

Fig. 6.1 Levels of evidence based on [2]. Variables with a strike-through the text indicate that this variable has shown no preventative effect

6.2 Hamstring Injury Prevention

6.2.1 General Warm-Up

For general injury prevention, a proper warm-up is considered essential, as insufficient warm-up strategies could increase the risk of future (hamstring) injury [3]. Warm-up regimes can be applied in different formats, from running drills to targeted exercises, with or without additional weight and equipment. Ultimately, the aim of a good warm-up is to be prepared physically and mentally for an upcoming bout of activity. In a landmark study by Ekstrand et al. [4], warm-up was part of a multimodal approach, and this study was one of the first indications that a general warm-up may play an important part in reducing injuries in football. Despite this, no evidence exists on general warm-up approaches and their effects on (the prevention of) hamstring injuries. Other warm-up programmes have focused on additional strength and conditioning elements for the purpose of general injury prevention. These types of warm-up strategies have been investigated in different sports, such as balance training using a wobble board in basketball [5], and specific warm-up exercises in volleyball [6], handball [7] and basketball [8], each of which have been effective in reducing overall injury rates.

6.2.1.1 FIFA 11 and FIFA 11+ Programmes

One of the best known and widely adopted prevention programmes is the FIFA 11, a pretraining warm-up specifically designed to prevent lower limb injuries in football. In addition to fair play, the FIFA 11 programme is entirely exercise based. Initial research on the FIFA 11 showed no preventive effect for overall and hamstring injuries [9]. Subsequently, the FIFA 11 was amended to the FIFA 11+, which includes running, strength, plyometric and balance exercises, each with three levels of difficulty to allow for progression (see Fig. 6.2) [10]. The programme has traditionally been used as a warm-up in football, but recent data have shown superior efficacy when it is employed before and after training as opposed to being used as warm-up alone [11]. This is very interesting as it questions whether the effect of the FIFA 11+ is purely driven by warm-up and preactivation of certain muscle groups that lead to improvements in movement competencies or instead is driven by a dosage-specific adaptation response to strength and conditioning, resulting in reduced risk of injury.

The impact of four large-scale (sample sizes ranging from 383 to 2540 participants) RCTs [12–15] across male and female youth football and male senior and veteran football was summarised in a systematic review and meta-analysis [10]. This meta-analysis (level 1a evidence) showed that the FIFA 11+ is an effective injury prevention tool when compared to a control group, and this effect extended to a 60% reduction in hamstring injuries across two of these cohorts (incidence rate ratio, 0.40; 95%CI, 0.19–0.84) [10]. Given the breadth of exercises included in the FIFA 11+, it is impossible to determine if the preventative effect should be attributed to a single hamstring-specific exercise such as the Nordic hamstring exercise (NHE), which is part of the FIFA 11+ (and discussed later on in this chapter), or to the programme as a whole.

FIFA 11
10-15 minutes duration

Exercises	Repetitions (reps) Seconds (s)
Core stability	
The bench	4 x 15s
Sideways bench	2 x 15s each side
Balance	
Cross-country skiing	2 x 15s each leg
Chest passing in single-leg stance	3 x 15s each leg
Forward bend in single-leg stance	3 x 15s each leg
Figures-of-eight in single-leg stance	3 x 15s each leg
Plyometrics	
Jumps over a line	15 jumps of each type
Zigzag shuffle (20 metres)	2 reps in each direction
Bounding (20 metres)	3 x 10-15 jumps
Strength	
Nordics	5 reps

FIFA 11+
20 minutes in duration

Exercises	Repetitions (reps) Seconds (s)
Part 1: running exercises	
Running, straight ahead	2 reps
Running, hip out	2 reps
Running, hip in	2 reps
Running, circling partner	2 reps
Running, shoulder contact	2 reps
Running, quick for- and backwards	2 reps
Part 2: Strength, plyometrics, balance	
The bench	
Level 1: static	3 x 20-30s
Level 2: alternate legs	3 x 20-30s
Level 3: one leg lift and hold	3 x 20-30s
Sideways bench	
Level 1: static	3 x 20-30s each side
Level 2: raise & lower hip	3 x 20-30s each side
Level 3: with leg lift	3 x 20-30s each side
(Nordic) hamstrings	
Level 1: beginner	3-5 reps
Level 2: intermediate	7-10 reps
Level 3: advanced	12-15 reps
Single-leg stance	
Level 1: hold the ball	2 x 30s each leg
Level 2: throw ball with partner	2 x 30s each leg
Level 3: testing partner	2 x 30s each leg
Squats	
Level 1: with toe raise	2 x 30s
Level 2: walking lunges	2 x 30s
Level 3: one-leg squats	2 x 10s each leg
Jumping	
Level 1: vertical jumps	2 x 30s
Level 2: lateral jumps	2 x 30s
Level 3: box jumps	2 x 30s
Part 3: Running exercises	
Running, across the pitch	2 reps
Running, bounding	2 reps
Running, plant & cut	2 reps

Fig. 6.2 Exercises included in the FIFA 11 and FIFA 11+ prevention programmes [10]

In summary, level 1 evidence suggests that the FIFA 11+ warm-up programme prevents hamstring injuries in football, although more studies across different athletic cohorts are needed to support broader generalisations of these findings.

6.2.2 Eccentric Strength Training

Numerous studies, primarily conducted in different football codes and baseball, have reported that hamstring strength training [1, 16–21], especially when training with an element of eccentric overload, reduces hamstring injury risk, as long as compliance with the intervention is high [22].

6.2.2.1 Flywheel Training

The first study to indicate a preventative effect from eccentric training for hamstring injury was from Askling and colleagues and included flywheel training [17].

Such devices, introduced in the literature by Berg and Tesch in 1994 [23], involve concentric contraction to accelerate the flywheel. Eccentric actions are then required for flywheel deceleration. Actively decelerating throughout a lesser range of motion (ROM) (compared to the concentric phase), allows for a period of eccentric overload.

The Askling study was performed in two Premier League teams in Sweden and was the first, and currently the only RCT to evaluate the preventive effect of preseason hamstring flywheel strength training [17]. Players in the intervention group performed both concentric and eccentric knee flexor actions in a prone position on a Yo-Yo flywheel ergometer, with the eccentric contraction performed over approximately the final two-thirds of the ROM compared to the concentric contraction [17]. This intervention (n = 15) was performed for a total of 16 sessions across a 10-week preseason period in addition to normal team training, whilst the control group (n = 15) completed normal team training only. During a 10-month follow-up, the intervention group had significantly fewer hamstring injuries (three injuries in 15 players) than the control group (ten injuries in 15 players) [17]. Whilst the sample size in the investigation is small (compared to other RCTs in this area) and the rate of hamstring injury in the control group is exceptionally high, this study was the first to indicate that eccentric training may impact hamstring injury incidence.

6.2.2.2 Nordic, Russian or Hamstring Lowers

The earliest published mention of the NHE (Fig. 6.3) can be traced back to 1880, when George Herbert Taylor described the movement in his book *Health by Exercise* [24] referring to it as 'wing-kneeling'. Despite some contention around its name, this chapter will refer to the exercise as the NHE, as this currently seems to be the most commonly adopted term globally. The exercise has alternatively been called Nordic curls, Russians, Russian leans and hamstring lowers.

Fig. 6.3 The Nordic hamstring exercise

The NHE is typically performed in pairs as a body weight exercise [1, 25]. Individuals are instructed to start in a kneeling position, with the torso from the knees upwards held rigid and straight. The training partner ensures that the exercising individual's feet are in contact with the ground by applying pressure to the heels/lower legs. The exercising individual then lowers their upper body to the ground, as slowly as possible, to maximise loading during the descent, requiring an increasingly forceful eccentric contraction of the knee flexors. The hands and arms are used to catch the forward fall and to push the individual back up to the start position after the chest has touched the ground, to minimise loading in the concentric phase.

The first study to examine the protective effects of the NHE was conducted in community-level Australian football ($n = 220$) [19]. This RCT exposed the intervention group to five sessions of the NHE across a 12-week period. The NHE protocol, which was delivered identically across all five sessions (see Table 6.1 for a summary of all NHE intervention prescriptions), was devised from laboratory-based studies that had shown a shift in the knee flexor torque-joint angle relationship towards longer muscle lengths after the performance of 72 repetitions of the exercise (12 sets of six reps). The intervention group ($n = 114$) was encouraged to complete the

Table 6.1 Training volumes of NHE protocols derived from prevention RCTs [1, 18–20]

Study	Intervention period	Week	Sessions, p/wk	Sets	Reps	Rest period
Gabbe 2006 [19]	12 weeks; 3 sessions in preseason and 2 sessions during first 6 weeks of competition	–	–	12	6	10 s between reps; 2–3 min between sets
Engebretsen 2008 [18]	10 weeks	1	1	2	5	Not reported
		2	2	2	6	Not reported
		3	3	3	6–8	Not reported
		4	3	3	8–10	Not reported
		5–10	3	3	12, 10, 8	Not reported
Petersen et al. 2011 [1]	10 weeks (plus ongoing maintenance throughout the season)	1	1	2	5	Not reported
		2	2	2	6	Not reported
		3	3	3	6–8	Not reported
		4	3	3	8–10	Not reported
		5–10	3	3	12, 10, 8	Not reported
		11+	1	3	12, 10, 8	Not reported
van der Horst 2015 [20]	13 weeks	1	1	2	5	Not reported
		2	2	2	6	Not reported
		3	2	3	6	Not reported
		4	2	3	6, 7, 8	Not reported
		5	2	3	8, 9, 10	Not reported
		6–13	2	3	10, 9, 8	Not reported

NHE protocol at the completion of the main team training session but prior to the cool-down, whilst the control group ($n = 106$) completed a number of flexibility and mobility exercises without exposure to the NHE. The intervention group did not show a reduction in hamstring injury risk compared to the control group (relative risk (RR), 1.2; 95% CI, 0.5–2.8) [19]; however, the intervention group suffered from very poor compliance (30% failed to complete a single NHE session, 53% failed to complete at least two sessions), and the primary reason reported by players was delayed-onset muscle soreness which often limited their involvement in subsequent training sessions. When comparing the rates of hamstring injury in the intervention group who completed at least two sessions to the control group, still, no effect was found (RR, 0.3; 95% CI, 0.1–1.4).

A subsequent RCT by Engebretsen and colleagues employed a more graduated 10-week NHE protocol (Table 6.1) in Norwegian soccer players who were considered at high risk of hamstring injury (based on injury history and a subjective questionnaire) [18]. Those identified as being at high risk of hamstring injury ($n = 161$) were randomised either into the intervention group ($n = 82$) or control group ($n = 76$), with the control group completing normal team training, but no additional intervention. The incidence of hamstring injury did not differ between these two groups (intervention group, 1.5 hamstrings injuries per 1000 h of exposure; control group 0.9 hamstring injuries per 1000 h of exposure); however, this study again suffered from low compliance, with only 21% ($n = 12$) of players completing more than 20 sessions of the 24 total NHE sessions. Whilst a per-protocol analysis also found no difference in hamstring injury incidence between those in the intervention group who were compliant and the control group, this analysis was limited by the small sample size.

The issues encountered by both Gabbe and colleagues [19] and Engebretsen and colleagues [18], that of poor compliance and relatively low sample sizes, were overcome by two separate studies completed in Danish and Dutch professional and amateur football cohorts [1, 20]. More than 1500 football players were included in these two RCTs (Petersen et al., [1], $n = 942$; van der Horst et al. [20], $n = 579$). Control group players performed regular team training, whereas intervention group players performed comparable NHE protocols (Table 6.1). The Danish [1] study involved a 10-week protocol followed by a weekly maintenance session across the remainder of the season, whereas the Dutch [20] study concluded NHE exposures after the 13-week intervention period. In the Danish study, coaches decided when the exercises were performed, being advised not to perform the NHE without a prior warm-up, and in the Dutch study, players were advised to perform the exercises after regular training but before the cool-down.

The Danish RCT showed that the NHE reduced the rate of new hamstring injuries by 70% (rate ratio, 0.29; 95% CI, 0.14–0.63) [1] and that the programme was even more effective for decreasing the rate of recurrent hamstring injuries, which were reduced by ~85% (rate ratio, 0.156; 95% CI, 0.05–0.53). The numbers needed to treat, defined as the number of players that need to complete the programme to prevent one injury, were 25 and 3 for new and recurrent injuries, respectively. Similarly, the Dutch RCT [20] reported a threefold reduction in the risk of

hamstring injury for players who performed the NHE (rate ratio, 0.30; 95% CI, 0.12–0.73). Neither the Dutch nor Danish study reported any effect of the NHE on injury severity as the time lost to each injury was similar in experimental and control participants.

A level 2b study, which was the first to assess the effect of the NHE on hamstring injury rates specifically in soccer players, was performed in Premier League and First Division footballers from Iceland and Norway [16]. These footballers participated in a study designed as a non-RCT that investigated the preventive effect of flexibility training, with or without the addition of the NHE, on the incidence of hamstring injuries. The warm-up protocol that combined the NHE with flexibility training reduced the rate of hamstring injury risk by half (RR, 0.43; 95% CI, 0.19–0.98) compared to stretching and flexibility training of the hamstrings alone.

Some level 2b evidence is also available from other sports than football. This exemplifies the potential crossover effect to other sports. In a non-randomised cohort study in a single Major League Baseball organisation [21], there was no standardised prescription of the NHE. Instead, the strength and conditioning coaches for seven (out of a total of eight) teams ($n = 243$) were instructed to incorporate the NHE into daily workouts, and players were deemed compliant if they completed on average more than 3.5 repetitions of the NHE per week across the season. The control group ($n = 40$) was the Major League team who were not explicitly instructed to perform the NHE. Not a single hamstring injury was sustained by a compliant individual in the intervention group (31% of the intervention group were deemed compliant), whereas the control group had an injury rate of 8.8%. Across the organisation, the year-on-year days missed due to hamstring injury were reduced by 50% during the intervention season (273 days missed in the year prior to the intervention compared to 136 days missed during the intervention year) [21]. A descriptive study (level 4 evidence) in sprinters also highlighted how the injury incidence seemed to decline during consecutive seasons as agility, flexibility and NHE were added to a more traditional and concentrically based strength training programme [26].

One common criticism of the NHE is that it has not been compared to alternative exercise programmes. For example, it has been argued that the NHE may not protect athletes who already engage in conventional strength training. However, a cross-sectional cohort study (level 3 evidence) by Brooks and colleagues [27] has compared hamstring injury rates in English rugby clubs that did and did not use the NHE in addition to their strength training programmes. Teams that employed regular strength training with exercises including both concentric and eccentric phases and regular flexibility training (144 players) experienced injury rates of 7.5 (95% CI, 4.4–10.6) per 1000 h of training, while teams that employed the same methods with the addition of the NHE (200 players) experienced injury rates of 4.2 (95% CI, 2.3–6.0) per 1000 h [27]. Teams that employed the NHE did so, on average (±SD), in 65% of training weeks, with 1.3 ± 0.5 training sessions per week, 2.8 ± 0.7 sets per training session and 6.7 ± 1.5 repetitions per set. So, while there are limitations

in the strength of this evidence due to study design, it does suggest the possibility that hamstring injury prevention requires a high-intensity eccentric component for it to be optimally effective.

6.2.3 Stretching

The first, and only, RCT (level 1b evidence) known to the authors focusing on the effect of stretching for hamstring injury prevention dates back to 1993, when the effectiveness of stretching exercises alongside warm-up/cool-down protocols was studied [28]. Following a 22% dropout after a 16-week intervention period, data of 326 athletes were analysed. There were no differences between intervention and control groups with regard to lower limb injuries (RR, 1.05; 95% CI, 0.22–5.13) or hamstring injuries (defined as 'injuries on the posterior side of the upper leg') with three hamstring injuries in both intervention and control group. However, a low compliance with the stretching exercises (47%) may have influenced the results.

Other studies into the effects of flexibility training on hamstring injury rates have been conducted, although these have a higher risk of bias due to methodological issues. For example, the preventive effect of stretching exercises was also investigated in the aforementioned study by Arnason and colleagues [16], who included one intervention arm with stretching exercises alone (without the addition of eccentric strength exercise). In addition to their standard warm-up stretching, these teams were required to use a partner-assisted contract-relax stretch for the hamstrings before sprinting or shooting exercises prior to training and matches. The teams were asked to perform this exercise three times per week during preseason and one or two times per week during the competitive season. No effect was detected from stretching alone, although a preventive effect was found in the other intervention arm that included the NHE alongside the warm-up and stretching. Hence, the results of this level 2b study suggested that the stretching component showed no preventive effect, and as such, the preventive effect was derived from the NHE alone.

A level 4 study that adopted stretching as an element of a prevention programme was performed in 2005 in Australian rules football [29]. Passive isometric hamstring stretches were performed during breaks in playing and training, particularly when players were considered to have muscle fatigue. Other elements of the intervention included more high-speed running, the removal of heavy strength training for the lower limbs and the use of 'stooped' running drills in which players ran while paddling a ball along the ground. Stretching was performed with the knee in varying degrees of flexion (0°, 10° and 90°) and subsequent flexion of the trunk to stretch the hamstrings. Athletes were encouraged to hold each stretch for at least 15 s. The team sustained 27 hamstring injuries in 2 years before the intervention and eight hamstring injuries in 2 years after its implementation. In addition, this led to significantly fewer matches being missed due to injury (69 in year 1–2 compared to 21 in year 3–4). Although this study provided promising results, the multifaceted

nature of the intervention makes it impossible to assess the preventive effects of each of its components. Consequently, this study does not substantiate the effectiveness of stretching as a prevention strategy for hamstring injuries.

Lastly, the relationship between stretching protocols and hamstring injury incidence was investigated in a level 4 study that included the top four English professional divisions [30]. After collecting data through self-administered questionnaires, a relationship between a standard stretching protocol and hamstring injury risk was identified. It was stated that the more the stretching protocol was used, the lower the hamstring injury risk was. However, a high risk of bias due to the design of this study needs to be taken into consideration when interpreting these results.

In conclusion, the currently available scientific literature does not support the use of stretching as a means of preventing hamstring strain injury.

6.2.4 Core Stability

Core stability is specifically addressed in this chapter as it is often emphasised as important for preventing injuries in the lower limb generally and the hamstrings specifically. However, the term core stability is poorly defined in both scientific studies and clinical use. The 'core' can mean different things to different people, such as the 'lumbopelvic region', 'lumbar spine' and even the 'trunk'. Furthermore, while the term 'stability' enjoys widespread use in the sports medicine literature, it has never been quantified in any hamstring studies [31]. Instead, researchers tend to measure strength, endurance or nothing at all and then too often assume that 'stability' has then been changed in the desired direction. There is, however, some preliminary level 3 evidence that certain aspects of lumbopelvic kinematics, such as exaggerated degrees of forward tilt of the pelvis and lateral trunk flexion, may be associated with an elevated hamstring injury risk [32].

6.2.5 Plyometric and Running Drill Interventions

High-speed running, accelerations and decelerations are critical for performance in many sports, but these variables are also closely linked to hamstring injury aetiology and mechanism [33]. Running drills are often included in training programmes with the belief that there is both a performance and injury prevention benefit [34].

As mentioned above, the effectiveness of the FIFA 11+ for hamstring injury prevention is possibly attributed to the NHE, but the FIFA 11+ also incorporates plyometrics and running drills. Unfortunately, there has been very little research specifically on plyometrics and running drills as an isolated means to prevent hamstring injury. To date, there is only a single RCT (level 1b evidence) in this area, conducted in 32 competitive amateur Dutch football teams (sixth division) ($n = 400$) [35]. The intervention consisted of 12 weeks of walking lunges, 'tripplings', drop lunges and bounding, after which a maintenance programme was conducted for the

rest of the season. The intervention group performed these exercises after regular training, whereas the control group performed regular training without these additional exercises. Hamstring injury incidence, number and severity in the intervention group (1.12/1000 h; $n = 31$; lay off time, 33.0 ± 42.7 days) did not differ from the control group (1.39/1000 h; $n = 26$ injuries; lay-off time, 21.35 ± 12.7). Compliance with the bounding programme (metres performed/metres prescribed ×100) was 71%. These findings suggest that including walking lunges, 'tripplings', drop lunges and bounding to regular team training does not reduce the incidence of hamstring injury in amateur soccer, albeit with a modest level of programme compliance. It should be noted that there are a multitude of different plyometric and running drills that are utilised in practice, so further work is needed to explore the possible impact of these derivatives on hamstring injury incidence.

6.2.6 Sports-Specific Interventions

It can be argued that high levels of sports-specific fitness should enable athletes to better withstand the demands of their sport and thereby be less likely to sustain injury. The aforementioned study by Verrall and colleagues [29] is one example. Given the addition of knowledge since the publication of this study, it would seem reasonable that the intervention elements that focused on running exposures and drills that better replicated the demands of Australian football are the 'active ingredients' of this intervention. However, due to the lack of a control group, it is not possible to infer whether the benefits arose because of the intervention or just represented normal seasonal variations. In addition, the level 4 study design makes it impossible to determine which part or parts of the injury prevention programme were important for hamstring injury prevention and what the additional value of each element was with regard to the reduced number of hamstring injuries.

6.3 Hamstring Injury Prevention Conclusion

At present, there is level 1a evidence showing that eccentric hamstring strength training delivered via the NHE is an effective measure for hamstring injury prevention [36], so long as the exercise is implemented gradually, with appropriate volumes, and compliance is high [22]. It is important to acknowledge that most of these studies on the NHE were performed in semiprofessional and amateur football environments, so how these findings translate to other populations (e.g. elite level, woman or other sports) remains a topic for further research. However, there are some indications that including eccentric strength training in the hamstring injury prevention approach may reduce the risk of sustaining hamstring injuries in other sports such as baseball, track and field and rugby union. There is also level 1 evidence of a preventive effect from the FIFA 11+ warm-up programme, but it is

unclear if the FIFA 11+ provides additional benefit to hamstring injury prevention from exercises other than the NHE. So far, the evidence from stretching studies indicates that these interventions do not seem to be effective at reducing hamstring injuries. The evidence on core stability, plyometrics, running drills and sports-specific training currently remains too limited to fully understand their effectiveness on hamstring injury prevention.

6.4 Hamstring Injury Implementation

Hamstring injury prevention programmes need to be implemented and adhered to by the targeted end users to show effectiveness [22]. Implementation of (hamstring) injury prevention in a sports environment is often a difficult task, even for athletes at increased risk of hamstring injury [18]. Multiple stakeholders can be involved such as sports associations (for rules and legislations), club boards, coaches, medical staff members, agents and so forth. In a sport setting the coach and medical staff are often the most important administrators of the prevention programme. Ultimately, however, the athlete is the end user, and his/her views with regard to the drivers and barriers for adoption of evidence-based hamstring prevention programmes need to be considered. Clearly, there are many reasons to excuse athletes from injury prevention: these include heavy game schedules, competing training priorities, poor staff communication, player and staff motivation and limited knowledge of preventive strategies [37, 38]. However, such barriers to hamstring injury prevention participation may play an important part in the lack of risk reduction seen in some sports over the last decade [39].

Research on hamstring injury prevention adherence in both professional and amateur football has shown that despite its effectiveness, the full evidence-based NHE programme (as employed by Petersen et al. [1]) is almost never adhered to by Champions League, Norwegian Premier League and Dutch amateur teams [37, 40]. Although the majority of the coaches of Champions League and Norwegian Premier League football were positive about the NHE, some reported unsatisfactory outcomes including muscle soreness and difficulty getting the players to comply with the programme. In addition, only 4% of physiotherapists, sports scientists and strength and conditioning coaches from the academies of elite soccer clubs in the United Kingdom reported using the FIFA 11+, with 9% stating the use of a modified version of the FIFA 11+ [41].

Different factors can stimulate injury prevention behaviour at the level of the athlete. Studies have shown that adoption of preventive measures can be stimulated by player motivation [37, 42], staff support [43, 44] and knowledge about injuries and injury prevention [37, 45–47]. For example, football players that had personally experienced an evidence-based hamstring injury prevention programme reported higher compliance at 2-year follow-up than those who had not [37]. While community football players acknowledged their effectiveness, they also believed that the injury prevention programmes need to be short in length and

that some variety in drills/exercises is preferable [48]. In addition to this, athletes stated that their personal knowledge of the effectiveness of the injury prevention programme and their personal motivation are key factors for future adoption. Coaches and medical staff members reported these factors as important as well but also stated that consensus among the team staff was important for successful implementation [37].

6.4.1 Practicalities of Implementation

6.4.1.1 How Can the Attitude Towards Hamstring Injury Prevention Be Positively Stimulated?

Stimulating injury prevention adherence should be a mutual effort from all stakeholders involved. The individual athlete, the coaching staff and the medical team are often responsible for planning of sports-specific technical and medical routines for each training and match activity. From a practical perspective for the *athlete*, it is suggested that the NHE programme should be supervised to improve adherence [37]. Athletes need to be made aware of, or educated about, the importance of hamstring injury prevention considering the increased risk for hamstring injuries as well as the high recurrence rates after the initial injury [46, 47].

With regard to the *coaches*, it should be recognised that injuries have a significant influence on team performance [49]. Lower injury burden and thus higher training and match availability are associated with more points in league matches, as well as more success in Champions League and Europa League football. Further awareness of the performance benefits of injury prevention is also an important part of implementing preventive strategies and involving coaches in hamstring injury prevention. Both the FIFA 11+ programme and the NHE protocol have been shown to improve jumping and balance performance and short sprinting ability [3, 50, 51] suggesting that there is also a physical capacity benefit, which might resonate more readily with coaching staff.

For the *medical staff*, there is an important role to play in facilitating knowledge transfer from evidence-based medicine to end users (e.g. the athlete) and the administrators (e.g. the coaches or other members of team staff) because knowledge of the effectiveness of an intervention plays a major role in hamstring injury prevention adherence for all of these stakeholders.

6.4.1.2 When Should Prevention Exercises Be Scheduled in Footballers?

Careful planning of prevention exercises is important to optimise effectiveness, facilitate performance and stimulate adherence [52–55]. However, the timing of prevention exercises entails many considerations such as when to perform prevention exercises in a competitive season (pre-, in- or off-season), when in a weekly schedule and before, during or after training.

6.4.1.3 Scheduling Prevention During a Competitive Season

All studies derived from level 1 evidence that showed a preventive effect for hamstring injuries in football through eccentric strength training implemented their intervention protocol during the preseason [1, 17, 20]. The Danish and Dutch RCTs both involved a progressive increase in volume of the exercise during preseason and a maintenance phase in-season. In contrast, the FIFA 11+ programme has been investigated as a weekly intervention programme, with 2–3 scheduled pretraining/warm-up sessions a week, during football training.

6.4.1.4 Scheduling Prevention in a Weekly Schedule

Strong scientific evidence is lacking in regards to the optimal scheduling of eccentric exercise within the training week. In addition, a 'one size fits all' approach is not feasible as different sports, leagues and competitions have varying fixture structures that heavily influence such a decision. As mentioned previously, there is evidence for the effectiveness of the FIFA 11+ warm-up (which includes the NHE) to reduce hamstring injury risk in youth and amateur players. Therefore, one strategy at these levels could be to simply introduce the 11+ (or components of it) into the pre-football training warm-up, particularly in these populations. However, specific injury prevention sessions that are separate from the main football training session are popular methods in practice [54], particularly for higher-level players (e.g. professional and semiprofessional).

Level 5 findings from a recent Delphi study found that, in general, when players play only one match per week (i.e. ≥5 days recovery between matches), the main eccentric exercise session is recommended to be performed at 72 h (otherwise referred to as match day plus 3 days or MD + 3) following the match ([56], in review) and 2–3 days before the next match. However, caution should be taken that there are no residual fatigue or soreness effects prior to the next match. A study in semiprofessional players [52] showed that when an eccentric exercise session was performed on MD + 3, some residual fatigue and muscle damage markers were still present on the day before the match (otherwise referred to as match day minus 1 day or MD - 1). In particular, creatine kinase (a blood marker of muscle damage) and perceived muscle soreness remained elevated in players. However, isometric strength was unaffected by scheduling the eccentric exercise on MD + 3 [52, 57]. In the Delphi survey of professional teams, familiarising players with eccentric exercise was reported to be important as a means of minimising the damage response ([56], in review). The experts agreed that during periods with ≤4 days recovery between matches (which may be more applicable to professional senior and youth football teams), low-intensity eccentric exercises can be used (i.e. low load, low volume) ([56], in review). The experts' view was that players should be accustomed to performing eccentric exercise to allow low intensity eccentric training during short recovery periods between matches. Interestingly, the study by Lovell and colleagues [52] showed that performing eccentric exercises on the day after a match (MD + 1) was not only tolerated by players but meant that there was also no residual fatigue or muscle damage markers evident on the MD - 1. This could be particularly important during these periods where there are ≤4 days recovery.

6.4.1.5 Scheduling Prevention Before, During or After Training

Another important question about prevention exercise scheduling is whether or not to perform an eccentric exercise session before or after the main training session. Unfortunately, again, there is no strong scientific evidence to recommend one over the other, and each approach has advantages and disadvantages. While the Delphi survey of practitioners from professional football ([56], in review) agreed that the eccentric session can be performed either before or after, it appears that the timing of the session may have different effects on the muscle. For example, Lovell and colleagues [53] found that estimated BF_{LH} fascicle lengths were increased and pennation angles decreased when NHE training was performed before football training sessions. The performance of NHE training after football training was, by contrast, associated with increases in pennation angle and muscle thickness without change in estimated fascicle lengths. Given the preliminary evidence that BF_{LH} fascicle length may influence hamstring injury risk, these findings suggest the possibility that the scheduling of eccentric training may impact on its injury protective effects. However, it seems unlikely that any single adaptation would mediate the benefits of an injury prevention programme. Regardless of the timing of the delivery of eccentric exercise, both approaches have been found to lead to similar chronic increases in hamstring strength [53]. Performing eccentric exercise after a training session has also been shown to enhance the ability of players to maintain eccentric knee flexor strength at half-time and at the end of simulated matches [55].

While it appears appropriate to perform eccentric exercise before or after the main football training session, there are some considerations to take into account as either approach could increase the risk of injury [53]. The acute effect of performing eccentric exercise may result in muscle fatigue and could increase the risk of injury [58] (although such risk is yet to be substantiated), particularly if the planned session is to be performed at high intensity and/or with high amounts of high-speed running and sprinting. Therefore, the subsequent exposure should also be considered when deciding whether or not to perform eccentric exercise before or after a training session.

It is worth raising the point that the larger hamstring injury prevention RCTs [1, 20] have employed the NHE either before or after training, and both approaches have resulted in reductions in injury rates. The Al Attar study provides an interesting perspective on this topic as well, since performing the FIFA 11+ pre- and post-training was more effective in reducing hamstring injury rates compared to performing the FIFA 11+ programme only before training [11]. From this, it is hypothesised that there could be a dosage-specific adaptation response to injury prevention exercises, resulting in a reduced risk of injury. Translating this to practice, the decision around the timing of delivery of an eccentric strength training stimulus may be dictated by other contextual factors such as coach, practitioner and athlete preference and consideration of the nature of the upcoming training session.

6.4.1.6 Should Hamstring Injury Prevention Strategies Be Tailored to the Individual?

Practitioners are often encouraged to provide tailored injury prevention strategies. Programmes may be tailored based on sport- or position-specific requirements or on the injury risk profile of the individual (noting that this risk profile will have some

degree of subjectivity). This approach is predicated on the notion that screening for risk factors can assist risk profiling, hence providing guidance for tailored injury prevention programmes. Yet, the evidence for providing interventions based on a risk assessment [18, 59] is limited, and results have been mixed. With this in mind, it is critical that all individuals are provided with interventions that have been proven to reduce the incidence of hamstring injury, regardless of the perceived risk profile [60].

Beyond the blanket application of proven primary prevention strategies, an individualised approach to support the overarching hamstring injury prevention strategy may very well include the individualised interpretation of serial monitoring data, via secondary and tertiary prevention strategies, and this will be discussed in more detail in Chap. 11.

6.5 Hamstring Injury Implementation Conclusion

This chapter has presented an evidence-based framework to guide the development and implementation of hamstring injury prevention strategies (Fig. 6.1). Level 1 evidence strongly indicates that primary hamstring injury prevention should utilise exercise programmes for hamstring strength with eccentric overload. Ultimately, stimulating adherence to preventive measures is the final step to make evidence-based hamstring injury prevention work in a real-world setting.

It is imperative to consider injury prevention from a performance perspective as well, especially when faced with a congested schedule that can make planning of injury prevention exercises difficult. Therefore, it seems important to carefully plan and gradually increase eccentric training load for each athlete.

In conclusion, the evidence suggests building up a hamstring injury prevention programme during preseason and maintaining it in the in-season. When playing one match per week, the recommended day to perform the main eccentric exercise session seems to be on MD + 3. Players should, however, be accustomed to the eccentric stimulus by maintaining at least weekly sessions to minimise the damage response prior to the next match. Based on expert opinion, it may also be appropriate to perform low-load/low-volume eccentric exercise on the MD + 1, but again, players must be accustomed to this. Finally, eccentric conditioning sessions have proven effective whether they were conducted before or after the main football session, so the context of the planned football session and other factors that may improve coach or athlete buy-in are important considerations before planning an implementation strategy.

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Diagnosis and Prognosis of Hamstring Injury

7

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7.1 Introduction

A clear diagnosis is the starting point in managing your injured athletes and essential for a proper prognosis and therapeutic plan. This is definitely the case for hamstring injuries, as "one" hamstring injury does not exist; it is a heterogeneous group of injuries that have a complex multifactorial aetiology.

In practice, hamstring injuries in athletes can generally be divided into three clinical scenarios:

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1. Acute hamstring injury.
   Acute onset muscle strains, complete ruptures and avulsion injuries. This also includes acute reinjury.
2. Hamstring injury sequela.
   Persistent or repeated complaints (strains and pain) related to initial or successive acute hamstring injury.
3. Hamstring tendinopathy.
   Localised hamstring tendon pain as result of tendon pathology.

Although the clinical diagnosis of acute hamstring injury is often straightforward, this is not always the case. From a diagnostic and therapeutic perspective, hamstring injury sequela and hamstring tendinopathy are often more challenging to manage. Scientific knowledge is limited, and definitions/terminologies for these challenging clinical scenarios are issues of ongoing debate. Importantly, the diagnostic workup is aimed at obtaining relevant information that has prognostic value and can guide the clinician or therapist to differentiate between therapeutic options. Estimating prognosis is one of the major challenges in managing hamstring injuries, due to the large variations in injury duration from 1 day [1] all the way up to >100 weeks [2].

The basis of the diagnostic workup is accurate history taking and physical examination of the injured athlete. Subsequent imaging investigation may provide additional information to support diagnosis and prognosis.

7.2 Diagnosis of Acute Hamstring Injury

It is vital for effective management of hamstring injuries to establish a correct diagnosis. History taking and physical examination are essential when evaluating an athlete with suspected hamstring injury. An acute hamstring injury usually involves a moment that athletes can recall as a result of a specific movement of the lower leg or pelvis. The athlete often feels a sharp twinge in the posterior thigh and typically reaches for this area [3]. The movement of the lower leg is classically associated with contraction and/or stretch of the hamstring muscle group, such as high-speed running, forceful stretching, splits and extreme kicking [4, 5]. Table 7.1 notes specific clinical signs and diagnostic tests that can be used for diagnosing acute hamstring injuries. It should be mentioned that these tests are useful for ruling out the condition and that a comprehensive clinical examination is advised to establish the diagnoses, rather than to rely on one specific test [6]. The typical triad that is observed on physical examination after an acute hamstring injury is pain on hamstring muscle stretch, pain on resistance testing of the hamstrings and localised pain on palpation [7]. Acute tendon pathology is another entity, which can be divided into acute bony avulsion and avulsion of the hamstring origin or insertion tendons. This presents with an acute onset, and the classic mechanism is a combination of passive hip flexion and knee extension.

Table 7.1 Clinical signs and symptoms that are important for diagnosing acute hamstring injuries [6]

Criteria for establishing the diagnosis of acute hamstring injury
Acute onset of posterior thigh pain in relation to movement or a specific motion
Pain during stretch of the hamstring muscle groups (Fig. 7.1) – Passive straight leg raise test (Fig. 7.1a) – Passive knee extension test (Fig. 7.1b) – Active knee extension test (Fig. 7.1c)
Pain during contraction of the hamstring muscle groups (Fig. 7.2) – Isometric contraction at the inner range (90° of knee and hip flexion) (Fig. 7.2a) – Isometric contraction at the mid range (15° of knee flexion and neutral position of the hip) (Fig. 7.2b) – Isometric contraction at the outer range (maximum degrees of knee extension and 90° of hip flexion) (Fig. 7.2c) – Taking-off-the-shoe test (Fig. 7.2d)
Localised pain on manual hamstring muscle palpation

7.2.1 Prognosis of Acute Hamstring Injury

Once the diagnosis of acute hamstring injury has been confirmed based on the clinical findings mentioned above, the collection of potential prognostic factors for recovery is helpful for adequate management. These prognostic factors, derived from history taking and physical examination, can then be used to estimate time to return to play (RTP) and/or to assess the risk for a reinjury.

The section below describes the prognostic factors that can be taken into account when estimating time to RTP. It not only describes factors that are associated with recovery time but also factors that are frequently considered in the clinical setting.

7.2.1.1 Patient's History

Several elements from the patient's history aid in providing a prognosis of the time to RTP. The most important elements to consider are (1) patient demographics, (2) previous hamstring injury, (3) sports activity level, (4) injury mechanism, (5) symptoms during or after the injury and (6) self-estimated or physician-estimated time to RTP [8]. In the section below, we will display the prognostic value of these elements.

Patient Demographics

Higher age is frequently mentioned as prognostic risk factor for prolonged recovery. However, three prospective studies [8, 9, 16], including one with a low risk of bias [9], have reported no association between age and time to RTP [9]. Similarly, other demographic variables, such as sex, height and weight, have been shown to have no association with the speed of recovery [10, 11].

Previous Hamstring Injury

Previous hamstring injury is a clear risk factor for a subsequent hamstring injury [12], but there is no convincing evidence that recurrent hamstring injuries do involve

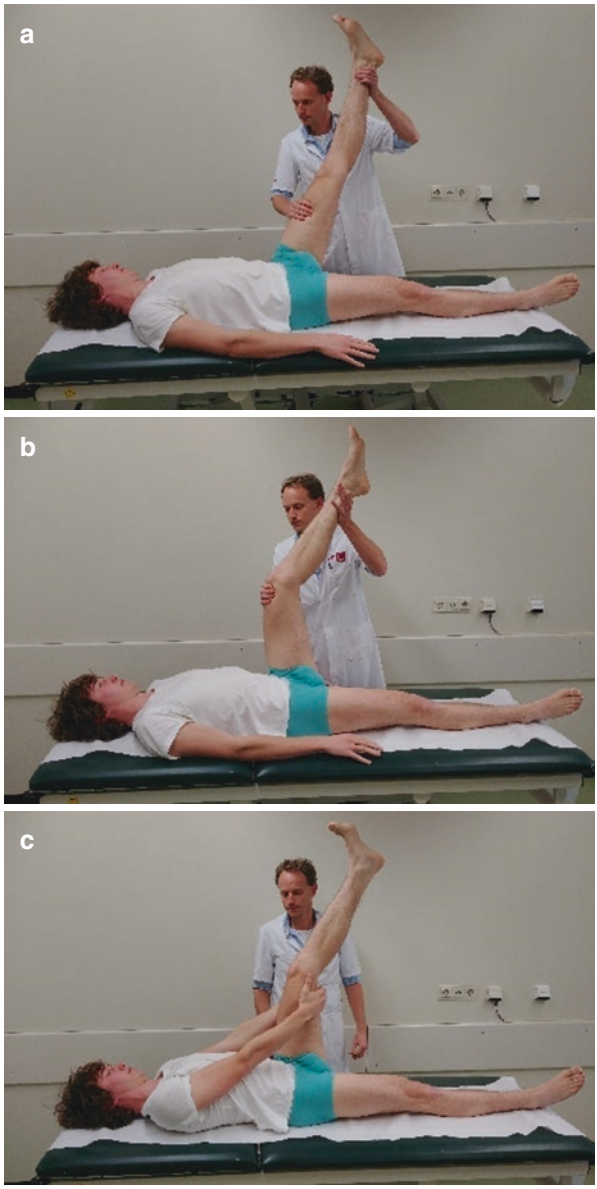

Fig. 7.1 (**a**–**c**) Stretch test of the hamstring muscle group. All tests are considered positive if the patients experience localised pain on the stretch test. (**a**) Passive straight leg raise: the tester flexes the hip while keeping the knee in full extension until maximum (tolerable) stretch. (**b**) Passive knee extension test: the hip of the tested leg is positioned in 90° of flexion. The tester extends the knee until maximum (tolerable) stretch. (**c**) Active knee extension test: the patient holds the hip of the tested leg in 90° of flexion and extends the knee actively until maximum (tolerable) stretch

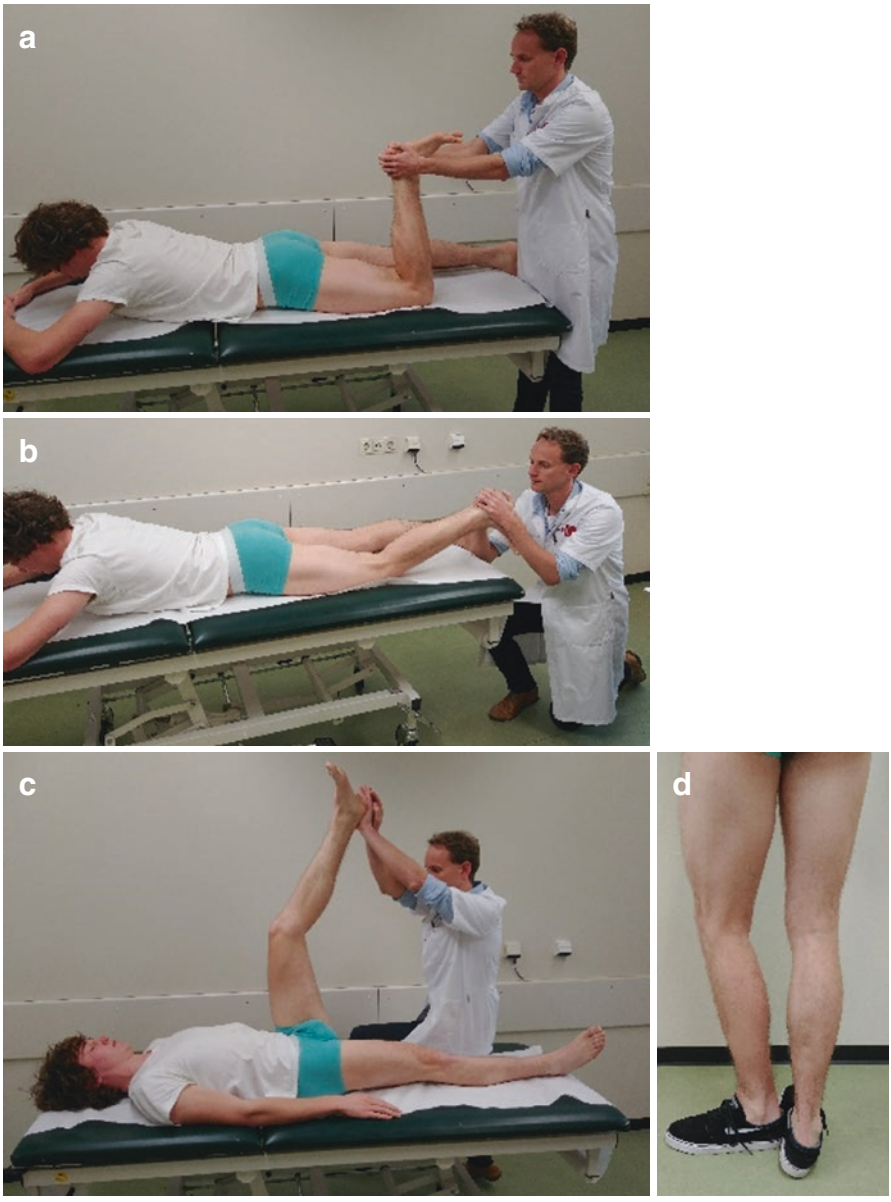

Fig. 7.2 (**a**–**d**) Isometric contraction tests of the hamstring muscle group. All tests are considered positive if the patients experience localised pain on contraction. (**a**) Isometric contraction at inner range: 90° of knee and hip flexion. (**b**) Isometric contraction at inner range: 15° of knee flexion and neutral position of the hip. (**c**) Isometric contraction at outer range: maximum degrees of knee extension and 90° of hip flexion. (**d**) Taking-off-the-shoe test: in standing position, the patient is asked to take off the shoe of the affected side with the help of his other shoe. During this manoeuvre, the injured leg's hindfoot presses the longitudinal arch of the uninjured foot. The hip of the affected leg is in approximately 90° external rotation and the knee 20–25° knee flexion

longer recovery times [9, 10, 13]. A previous hamstring injury can also be iatrogenic of origin, for example, a hamstring autograft can be used for anterior cruciate ligament (ACL) reconstruction. However, athletes with a history of ACL reconstruction involving hamstring autograft do not necessarily have a prolonged recovery time after hamstring strain injury [9].

Sport Participation

Neither the level nor the frequency of sport participation contributes to a prolonged recovery in recreational athletes according to one study [9]. There are, however, no studies comparing professional athletes with recreational athletes. When comparing the mean RTP times in professional (approximately 20 days) [14] and amateur football players (approximately 40 days) [15], it is possible that this difference exists. It is questionable whether the type of sports influences recovery time. One study with high risk of bias showed that dancers have longer recovery times than sprinting athletes [16]. This might be a result of the injury mechanism and different tissue involvement (myotendinous junction versus free tendon) (see paragraph below). Two other studies showed no differences in recovery times between football players and other athletes [8] or between football, track and field, lacrosse and rugby athletes [17].

Injury Mechanism

The injury mechanism has been reported to be an important factor in hamstring injury prognosis, and the distinction between "sprinting" and "stretching" injuries has received particular prominence in the literature [17]. Askling et al. found that "slow stretching type" injuries in dancers [18] take a lot longer time to recover than "sprinting type" injuries in sprinters [19]. Also, in a cohort of football players, Askling et al. reported a prolonged recovery time for "stretching" injuries compared to "sprinting" injuries [20]. One study in another cohort, however, showed no association between these types of injury and recovery time in a population of mainly football players [9]. Nevertheless, the typical stretch mechanism in football probably differs from the predominantly slow stretching mechanism experienced by dancers [18]. Based on the data mentioned above, it seems that dancers with slow stretch injuries have longer recovery than athletes who are injured during sprinting. However, there is conflicting evidence whether the stretching and sprinting types of injury mechanisms play a significant role in recovery time within sports such as football [10, 13].

Symptoms During or After Injury

When athletes hear a popping sound at the time of injury, there might be a prolonged recovery time [21], although there is only limited evidence for this association. The same applies to the cessation of the sport activity just after the injury. When players have to stop within 5 min after the injury, they are probably more likely to have a longer recovery time than those who manage to continue to play more than 5 min despite the acute onset of pain [10]. The level of pain at the moment

of injury is associated with recovery time in multiple studies [10, 21, 22]. Ascending stairs pain-free within 1 day is not associated with time to RTP, and it was previously thought that this association was present for walking pain-free within 1 day [13]. A recent study did not find this association, and therefore, it is unclear whether this is a true prognostic factor [9]. The progress of symptoms over time may still be relevant, as muscle pain during everyday activities for more than 3 days is likely to result in prolonged recovery time [21].

Self-Estimated or Physician-Estimated Time to Return to Play

A simple question for the athlete could be to estimate his or her time needed to RTP. This is a valuable question, as it has been shown to be related to the actual recovery time [9]. The same was found for the predicted estimation by the physician [22, 23]. It should, however, be noted that both the injured athlete and physician are influenced by the results of history taking, physical examination and imaging. This kind of information should therefore not be the only guide for estimating RTP but may aid the clinician in providing some guidance on overall recovery time.

7.2.1.2 Physical Examination

For prognostic considerations, clinical examination can be divided into (1) inspection, (2) range of motion (ROM) tests, (3) resistance tests, (4) special tests and (5) palpation.

Inspection

On inspection, swelling and a haematoma might be observed. The presence of a haematoma has been associated with prolonged recovery time [21]. The physician should therefore actively inspect for bruising, especially when examination is performed within the first week after injury.

Range of Motion

There has been considerable research interest in the possible relationship between ROM changes and recovery time. The presence of pain on trunk flexion and active knee extension (AKE) during the initial examination are both associated with prolonged time to RTP [10]. For the other ROM variables, there is conflicting evidence for their association with recovery time. There is conflicting evidence for an association between recovery time and deficit in PSLR test (in degrees compared to the uninjured side) [9, 21], pain on PSLR test [21], deficit in AKE testing [24] and pain on AKE tests [10]. Therefore, these measures should be used with caution as prognostic risk factors.

Resistance Tests

Knee flexion strength as assessed with hand-held dynamometry (HHD) in an extended knee position was not associated with time to RTP in a group of dancers and sprinters with acute hamstring injury [16]. More recently, isometric knee flexion force measured in 15° of knee flexion using HHD was associated with recovery

time in recreational athletes [9]. There is also conflicting evidence for this association with pain during resisted knee flexion [9, 10, 13, 21].

Slump Test

There is scarcity of special test results in relation to recovery time. A positive slump test (neuromeningeal stretch testing) has not been associated with prolonged recovery in the literature and should therefore not be used for prognostic considerations [10, 13].

Palpation

Palpation pain is an important feature for diagnostic considerations but less important as a prognostic indicator. Neither the location nor the length of palpation pain is consistently related to recovery time [9, 10, 18, 19]. Furthermore, there is no consistent evidence that the distance from the ischial tuberosity to the maximum palpation pain is related to recovery time. There is only limited evidence that a wider area of palpation pain increases the chance of prolonged recovery time [10].

7.2.1.3 Can Return to Play Be Predicted at the Initial Assessment?

There is currently little data that can aid the clinician and athlete in providing a prognosis for time to RTP. Figure 7.3 summarises these data in relation to recovery time. There are three factors with moderate levels of evidence. The level of pain, measured on a visual analogue scale (VAS), was associated with recovery time. The mean maximum pain score in the specific studies was 5.5 on a scale from 0 to 10 (range 4.5–6.2). Athletes who recall a higher level of maximum pain during the injury moment are more likely to have a prolonged recovery time [10, 21, 22]. Self-estimated recovery time is another factor of influence. In the particular study, athletes estimated a mean of 32 days in recovery time (with a standard deviation of 12 days). The majority of athletes had a previous hamstring injury which might have influenced this outcome [9]. The physician-estimated recovery time is also associated with the true recovery time [22, 23]. No absolute values have been reported in these studies, but a significant correlation was reported. Potential bias could be introduced by the fact that the physician manages the injury and therefore influences recovery time. For the other mentioned factors, there is limited evidence for their association with recovery time. There is only limited evidence for association between recovery time and measures of physical examination. Factors for which there are no evidence or conflicting evidence are not displayed.

It should be emphasised that there is a lot of heterogeneity between the studies that assessed these prognostic risk factors. The type of athlete, timing of history taking and physical examination, definition of RTP and study design all influence the results. Another limitation is that these factors only explain a small amount of the variation in time to RTP. There are still many unknown variables that potentially play a larger role in estimating recovery time. All the variables that are described above could only explain 20–50% of the variance in the total recovery time. Therefore, the current available research does not allow us to provide an accurate

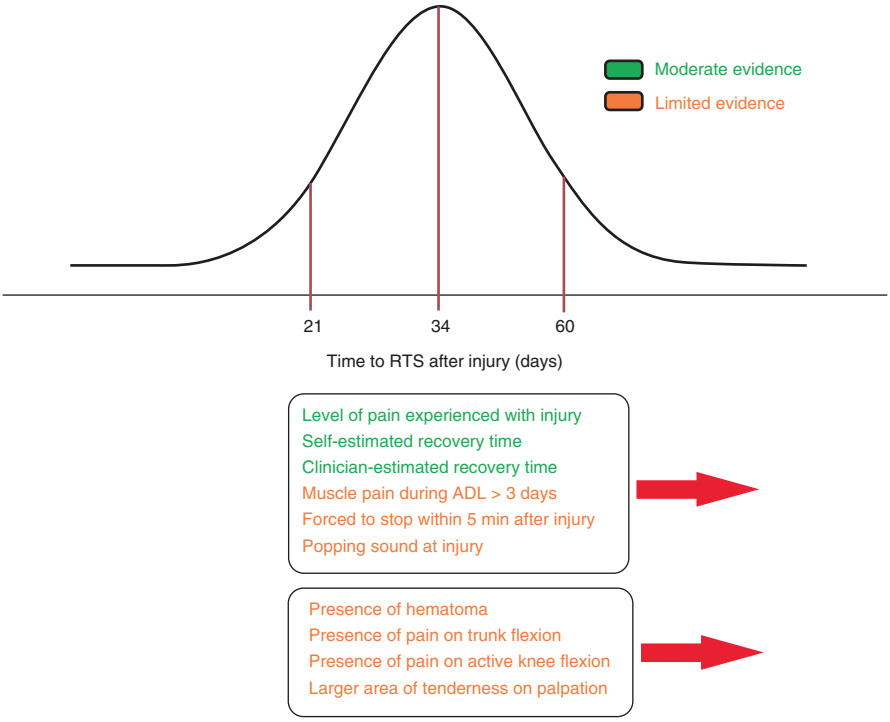

Fig. 7.3 Prognostic factors that are associated with time to RTS after acute hamstring injury. The median (34 days), 25th percentile (21 days) and 75th percentile (60 days) are based on publications included in a recent systematic review [8]. *ADL* activities in daily living

prediction of the injury duration for the individual athlete just after injury. We will illustrate this using the data of a recent prospective study [25]. A total of 90 athletes were examined, and multiple variables were collected at the time of presentation after injury. All these factors explained only 50% of the variance. For example, with the prognostic factors assessed at baseline, the predicted mean injury duration is 24 days; however, the 95% confidence interval with this prediction is from 8 to 47 days. This indicates that we can tell the athlete and the coach that there is a 95% chance that RTP will be possible within 8–47 days. The athlete and coaching staff will probably argue that this estimation of the injury duration is a long way from being satisfactory.

7.2.1.4 Follow-Up Clinical Examination Can Provide More Accurate Prognosis of Recovery Time

A valid criticism of these prognostic risk factor studies is that variables obtained at only one point in time just after injury do not reflect the way of working in the sports medicine setting. Especially in elite sports, the injured athlete is monitored on a regular basis. Intuitively, the speed of progression on variables such as pain, strength, flexibility and other functional tests may well provide more accurate prediction.

One research group analysed the prognostic value of a follow-up assessment after 7 days [25]. They found that a prediction model with a combination of ten clinical variables of the follow-up assessment explained 97% of the variance and was able to predict the injury duration with an accuracy of ±5 days. This seems a reasonable predictive ability that is clinically helpful. Considering all the factors measured at baseline and at follow-up examination in this study, the key variables to monitor are the following: the change in strength from initial examination to day 7, isokinetic knee extension strength of the uninjured leg on day 1, the maximum pain reported by the athlete at the time of injury, playing the sport of football, the presence of pain on performing a single-leg bridge at day 7 and the delay in starting physical therapy treatment. The authors provided an online tool that allows calculating the predicted injury duration based on their data: http://bjsm.bmj.com/content/50/7/431/suppl/DC1. See Fig. 7.4 for an example of this calculator [25]. It is important to note that this is currently the only study that has performed such a comprehensive analysis of the predictive value of follow-up examination. As these findings are not validated yet in other cohorts of athletes, applying the outcome of this calculation to clinical practice should be done with reservation.

7.2.1.5 Evaluating Reinjury Risk

High reinjury rates remain a major problem following acute hamstring injuries, despite the increasing use of sophisticated imaging techniques, prevention and treatment options. This may be due to a combination of insufficient rehabilitation and a premature RTP. Reinjuries may occur many weeks or months after RTP [26], but a recent study showed that 50% of the reinjuries occur within the first 50 days of RTP in footballers [27]. This emphasises that RTP and reinjury are interrelated factors.

	CASE 1	CASE 2	CASE 3
Change in strength through first week mid range:	6	5	3
Peak torque of Hamstrings at 60 degrees per second	55	50	50
Maximum pain at the time of injury	4	7	9
Number of days to walk pain free	3	5	6
Does the athlete play football?	Yes	Yes	No
Inner range strength at day 1	12	13	13
Can the athlete do a single leg bridge painlessly at day 7?	Yes	Yes	No
Is the outer range strength test painless?	Yes	No	No
How many days until they started physio?	0	0	0
What is their outer range strength as a percent of the uninjured side?	95%	70%	80%
Predicted Days to RTP:	6	24	47

Fig. 7.4 Example of an online calculator for estimating recovery time that is based on 90 athletes with an acute hamstring injury [25]. Three different cases are displayed with the estimated recovery times. Note that change in strength and inner range strength is measured in kilograms, and peak torque of hamstrings at 60 degrees is measured in Newton metres and level of pain on a 0–10 scale

A systematic review showed only limited evidence for ipsilateral ACL reconstruction and severity of the initial injury on magnetic resonance imaging (MRI) as risk factors for hamstring injury recurrence [28]. In the ideal situation, the results of history taking and clinical findings would enable the clinician to predict a safe RTP without a high risk of reinjury. In one study, standardised clinical tests were performed within 7 days after RTP, and the athletes were followed for 12 months to establish the number of reinjuries [29]. A higher number of previous hamstring injuries, more degrees of AKE deficit, isometric knee flexion force deficit at 15° and presence of localised discomfort on hamstring palpation just after RTP were significant independent predictors of reinjury. Athletes with localised discomfort on hamstring palpation just after RTP were consequently almost four times more likely to sustain a reinjury.

The above-mentioned findings emphasise that it is of major importance to monitor the athlete within the first week after RTP. Although it seems reasonable to postpone the timing of RTP and focus on recovery of the clinical abnormalities in athletes who are at increased risk for reinjury, future studies should be performed to evaluate the effect of these interventions.

7.2.2 Imaging as Diagnostic and Prognostic Tool

Imaging investigation of an acute hamstring injury can provide additional information to support the initial clinical diagnosis and prognosis and inform aspects of the rehabilitation plan.

7.2.2.1 X-Ray

While MRI and ultrasonography are the ideal modalities to assess hamstring injury, X-ray may have a specific role as a supplementary investigation if there is clinical suspicion of a bony avulsion of the proximal tendon attachment to the ischial tuberosity. This may be of particular relevance in younger athletes in whom the growth plate has not fused (<25 years) as a cortical fragment on MRI scan is often low signal, within a similarly low signal retracted tendon, thus making MRI diagnosis of a bony avulsion more challenging. X-ray is also suitable for the detection of calcification in myositis ossificans. X-ray is not helpful in the diagnosis of acute muscle injury.

7.2.2.2 Ultrasonography

Ultrasonography of acute muscle injury is quick and cheap and enables dynamic assessment of hamstring muscle injury. Therefore, it provides some advantages over MRI and can be used as an alternative or an adjunct to diagnosis. Sonography may also be used in follow-up to assess haematoma resorption or assist aspiration and in the early detection of calcification, although this is less common than myositis following muscle injury in the quadriceps. Ultrasonography also informs a limited, but widely used, grading system from grade 1–3 to describe muscle injury [30]. Ultrasonographic findings have not been associated with time to RTP [31].

There are some limitations with ultrasonography as an alternative to MRI. Ultrasonography is operator dependent, and it may be more challenging to detect muscle oedema than on MRI. A study in calf injury demonstrated that ultrasonography was much less sensitive than MRI in detection and diagnosis of muscle injury [32]. The assessment of the intramuscular tendon injury, including a retracted tendon within haematoma, may also be challenging via ultrasonography. As such, both small injuries and injuries to the intramuscular tendon may be overlooked if ultrasonography is the only imaging modality performed. With some evidence that imaging-negative injuries are associated with a better prognosis [33–35] and intramuscular tendon injuries with a more negative prognosis [19, 36–39], MRI may be important for certain athlete groups.

7.2.3 Magnetic Resonance Imaging

Magnetic resonance imaging is ideally suited to complement the clinical assessment of hamstring muscle injury due to its high sensitivity in detecting muscle oedema and intra-tendon injury. It can provide an objective assessment of the intramuscular and extra-muscular tendon of the muscle and provide information regarding the complete muscle-tendon-bone unit and differential diagnosis. In recent years, there has been increased focus on the role of MRI in hamstring muscle injury diagnosis, classification and informing management. Recent evidence has also noted that MRI appearance does not change in the first 7 days following a muscle injury, indicating that imaging can be performed on any day within the first week of the injury [40].

7.2.3.1 MRI in Diagnosis and Informing Rehabilitation

In acute hamstring injury, an MRI will detect the injured hamstring muscle(s). The hamstring muscle group is comprised of four hamstring muscles that have specialised functional roles in sporting activity [41, 42]. An understanding of these functional roles and knowledge of which muscle is injured should assist the clinician in exercise prescription. Muscle inhibition and aberrant biomechanics have been noted following previous hamstring injury [43–46]. This may be correctable by more targeted, focussed rehabilitation, although studies that have directed rehabilitation to the specifically injured muscle have not yet been performed.

The new muscle injury classification systems that place importance on the detection of injury to the hamstring tendon, either free tendon or intramuscular, are based on the understanding that the healing physiology and response to tissue loading for tendon injury is different to that of muscle tissue injury [47, 48]. The principle of accurate structural diagnosis enabling optimal loading in rehabilitation by the process of mechanotransduction is well-established in musculoskeletal injury [49]. Tendon injury is also difficult to detect clinically [50], and therefore, MRI scanning is of particular value in determining tendon involvement.

While there have been a number of MRI-based classification proposals, there are currently no studies that have targeted specific rehabilitation to the defined classes.

Further work in this area will help to determine if there is benefit in classifying these injuries to inform a more effective rehabilitation approach.

7.2.3.2 MRI and Prognostication with Respect to Return to Play and Reinjury

MRI-Negative Hamstring Injury

In both clinical practice and in the muscle injury literature, the grade 0 injury has been determined as a clinical syndrome of muscle abnormality without imaging evidence of pathology. The grade 0 or MRI-negative hamstring injury has been consistently associated with a quicker RTP [1, 33, 38].

Intramuscular Tendon Injury

A number of studies have noted that intramuscular tendon involvement is associated with an increased time to RTP or reinjury rate in athlete cohorts [36–39, 51]. These studies were completed on an elite-level cohort, and there is consistent evidence in competitive athletes that intramuscular tendon injuries appear to take longer to RTP. However, these studies were somewhat limited in design and have associated bias [4, 5], most often because the treating clinicians were not blinded as to whether there was intramuscular tendon injury [52, 53]. In a study group of mainly Qatari footballers, when clinicians were blinded to tendon involvement, there was also a moderate increase in RTP time [39]. This same cohort demonstrated no increase in reinjury within 1-year follow-up [54]. The reinjury rate at 3 months in the intramuscular tendon group seemed to be double that of the non-tendon injury group, although a post hoc analysis showed that this tendency was not significant.

The relevance of the hamstring intramuscular tendon injury may depend on the demands of the athlete's sport, particularly the athlete's high-speed running demand as the work done by the hamstring muscle increases in a non-linear manner with increasing speed [55]. Further work is required, particularly in high muscle-tendon unit demand cohorts, to determine the significance of the intra-tendon hamstring injury with respect to RTP and reinjury. This will help to define the requirement for MRI in the diagnosis of hamstring injury.

7.2.3.3 Value of Imaging in Addition to the Clinical Examination

Clinicians should not rely upon MRI alone to provide an effective RTP prediction. Indeed, it has been suggested that MRI findings explain only 2.8% of RTP variance, while the clinical findings explained 29% of total variance [10]. A number of studies have determined that initial clinical assessments can only explain between 20% and 50% of RTP variance [9, 10, 25]. Follow-up clinical evaluation at 1 week to reassess clinical variables has been demonstrated to improve on this prognostication [25]. There is no evidence that MRI has a role in informing the RTP decision-making process. A majority of hamstring injuries may still have abnormal MRI imaging features at the point of a successful RTP [56]. Fibrosis on MRI at RTP does not influence reinjury risk [57].

In summary, MRI cannot accurately determine prognosis, management or RTP in isolation. It may inform rehabilitation strategies which are continually adapted and progressed based on regular clinical reasoning and assessment and understanding of the demands to which the athlete must return. The prognosis and management should therefore be determined by a clinical reasoning process, to which the MRI findings may contribute, depending on the athlete's injury and sporting demands.

7.3 Diagnosis and Prognosis of Hamstring Injury Sequela

We use hamstring injury sequela as a collective term for those athletes that present with persistent or repeated complaints (recurrent acute injuries and pain) related to initial or successive acute hamstring injury. From a diagnostic perspective, this clinical scenario is often challenging to manage, and scientific knowledge is limited.

The diagnostic workup when athletes present with hamstring injury sequela should be aimed at the following:

1. Differential diagnosis of other causes of persistent or repeated episodes of posterior thigh pain
2. Evaluating (risk) factors that affect persistent or repeated episodes of hamstring complaints

7.3.1 Differential Diagnosis of Posterior Thigh Pain

In case of hamstring injury sequela, the clinician should (re)consider whether the origin of the symptoms is hamstring related or from other sources causing posterior thigh pain. For a comprehensive overview of diagnosis causing posterior thigh complaints, we refer to Chap. 13. As there is a wide range of possible causes for posterior thigh pain, including both hamstring-related causes and causes from other anatomical structures (Table 13.1), there is no standardised diagnostic workup. This largely depends on the clinical suspicion in individual cases.

7.3.2 Evaluating (Risk) Factors that Affect Hamstring Complaints

In the diagnostic workup, the proposed modifiable (risk) factors related to hamstring complaints need to be (re)assessed. These modifiable risk factors may include hamstring strength deficits [29, 58], imbalances in hamstring-quadricep strength [59], reduced flexibility [29], deficits in recruitment patterns of hamstrings, gluteal and trunk muscles [60–62] and external loading [63, 64]. For a complete overview of risk factors associated with hamstring injury, we refer to Chap. 4 (External and Intrinsic Risk Factors) and Chap. 5 (Neuromuscular Factors). There is no evidence

that these risk factors are related to hamstring sequelae, nor there is evidence that correcting these factors is beneficial as a management strategy. The best available evidence however suggests that hamstring eccentric strength deficits and markedly decreased H/Q ratios exist in athletes with hamstring injury sequela, and thus, rehabilitation focusing on restoring eccentric hamstring muscle function seems to be of importance [65]. This advice is based on the currently available best practice.

7.3.3 Imaging in Hamstring Injury Sequela

Additional imaging in hamstring injury sequela can be used to (1) assess morphological/structural changes of the hamstring muscle complex and (2) explore the differential diagnosis of posterior thigh pain in case of clinical suspicion such as neural-related (e.g. radiculopathy), bone-related (e.g. stress fractures) or joint-related pain (for a comprehensive overview of differential diagnosis of posterior thigh pain, see Table 13.1). Both sonography and MRI can be used to assess altered morphological characteristics, such as a missed avulsion injury, (excessive) fibrosis formation, muscle atrophy, intra- or intermuscular seroma or myositis ossificans.

7.3.4 Prognosis of Hamstring Injury Sequela

There is currently no evidence available on the prognosis of hamstring injury sequela regarding either the time needed for recovery or reinjury rates.

7.4 Diagnosis and Prognosis of Hamstring Tendinopathy

7.4.1 Clinical Diagnosis

Hamstring tendinopathy differs from the acute injuries in both presentation and management. This paragraph will focus primarily on the more common proximal hamstring tendinopathy (PHT). Distal hamstring tendinopathies are rare. Proximal hamstring tendinopathy is characterised by classic histological tendinopathic changes in the affected region. Lempainen et al. suggest that pathology is always limited to the semimembranosus (SM) portion of the tendon of origin [66], whereas Benazzo et al. report a distribution across the common tendon (23%), biceps femoris (41%) and SM (29%), with only rare involvement of the semitendinosus involvement (6%) [67].

Proximal hamstring tendinopathy diagnosis is frequently complicated due to the presence of many related structures that closely mimic its symptoms and signs, the most common of which is sciatic nerve irritation [68]. Puranen and Orava first described peripheral sciatic nerve entrapment, termed “hamstring syndrome”, and methods to differentiate it from hamstring tendinopathy [69]. More recently, this has become known as deep gluteal syndrome [70], although these terms seem to

embrace the same clinical entity, that is, sciatic neuritis. Accurate history, pain localisation and behaviour as well as physical examination assist in clarifying the issue. Not uncommonly, multiple pathologies may coexist, further complicating the management.

Hamstring tendinopathy typically affects cyclists, racewalkers and middle-aged female runners, athletes from various football codes and sedentary older females. Symptoms, which are generally provoked by prolonged sitting, cycling, lunging, squatting or change of direction activity, are well-localised to the ischial tuberosity with no radiation or associated neural signs. Examination should seek to reproduce the patient's pain with combined passive stretch and compression tests such as those described by Cacchio et al., specifically the modified bent-knee stretch test which has a sensitivity of 0.89 and specificity of 0.91 [71]. Alternatively or in addition, active tension and compression such as a hamstring bridge in greater than 45° of hip flexion or the arabesque manoeuvre may assist confirmation [72]. For all tests, the clinician is seeking to reproduce the pain type and consistent location at the ischium. Notably, higher tendon loads via single-leg tests with greater speed or resistance should provoke more pain. Use of repeated applications of isometric loading of the hamstring tendon in a long leg bridge may lead to pain reduction of the tests above, assisting in confirmation of the involvement of the tendon [72, 73].

Associated weakness and atrophy of the hamstrings with long-standing symptoms is a common feature [72]. Lumbar spine or ipsilateral hip dysfunction are common contributing elements also necessitating thorough orthopaedic and functional assessment.

Prognosis of hamstring tendinopathy is extremely variable in terms of time frames, due to the variability in presentation in terms of pain behaviour, associated weakness, involvement of other structures and the sporting and functional demands of the patient. Resolution may range from 6 weeks to 12 months or longer.

7.4.1.1 Differential Diagnoses

A history of recent overstretch injury should be closely scrutinised to exclude SM tendon partial or complete tear [2]. Not uncommonly, these lesions take considerable time to resolve and may differ in rehabilitation from the more proximal tendon of origin.

Younger male or female patients who on closer questioning have concomitant significant lumbar spine or hip joint stiffness may require exclusion of ankylosing spondylitis.

Deep gluteal syndrome embraces the spectrum of sciatic nerve entrapments that can occur deep to gluteus maximus (GM) from the interval spanning the sciatic notch through to the lateral hamstring interface described by Puranen and Orava [69, 74]. Provocative interfaces can include the obturator internus [75], the gemelli, highly variable fibrous adhesions [76, 77], piriformis, ischiofemoral impingement or hamstring tendinopathy itself. Pain on prolonged sitting is also a feature with most presentations as in pain on stretch, generally worsened with combined hip adduction. Pain site is somewhat more general with some radiation following the nerve path distally. Variability in pain site is more a feature of nerve involvement. Martin et al. [70]

suggest that a positive finding on active piriformis test in combination with a positive seated piriformis stretch test provides a specificity of 0.91 and sensitivity of 0.80.

The adductor magnus is situated more medial to the hamstring enthesis but on occasion can mimic proximal hamstring tendon symptoms. It does have a variable tendon of origin which can itself become tendinopathic [78]. In these authors experience, this is best identified through isometric adduction against resistance combined with bilateral hip extension, with the patient in lying prone.

Martin et al. [74] also describe ischiofemoral impingement, which in its classic form leads to compromise of the quadratus femoris muscle in the space between the ischial ramus and lesser trochanter of the femur. On occasions, this can also compromise the sciatic nerve or hypertrophied hamstring tendon. Reproduction of a deep ischial pain provoked by hip extension with long walking strides (Fig. 7.5) has a specificity of 0.94 and sensitivity of 0.85 [79]. Further, differentiation between extension-neutral and extension-adduction of the hip with the patient in side lying [80] (Fig. 7.6) is reported to have a specificity of 0.82 and sensitivity of 0.85. Martin et al. [74] recommend both these test procedures are included to determine ischiofemoral impingement.

Further differential diagnoses include the lumbar spine, hip joint, ischiogluteal bursa, sacrotuberous ligament, trigger points or muscle strains of the GM, obturator internus, gemelli or quadratus femoris. These are all identified with a combination

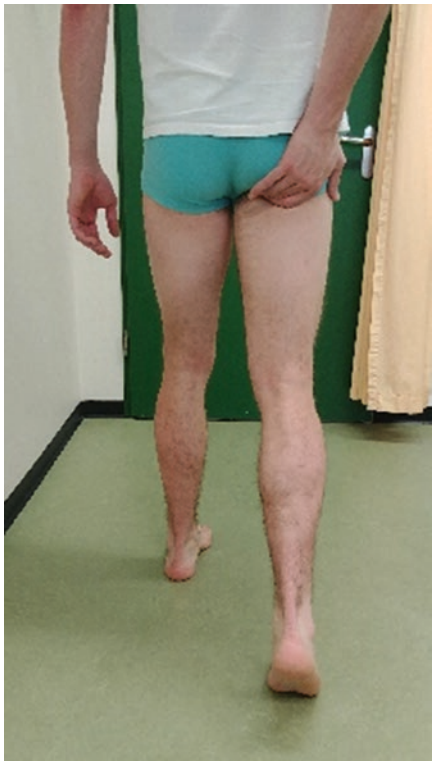

Fig. 7.5 Reproduction of a deep ischial pain provoked by hip extension with long walking strides

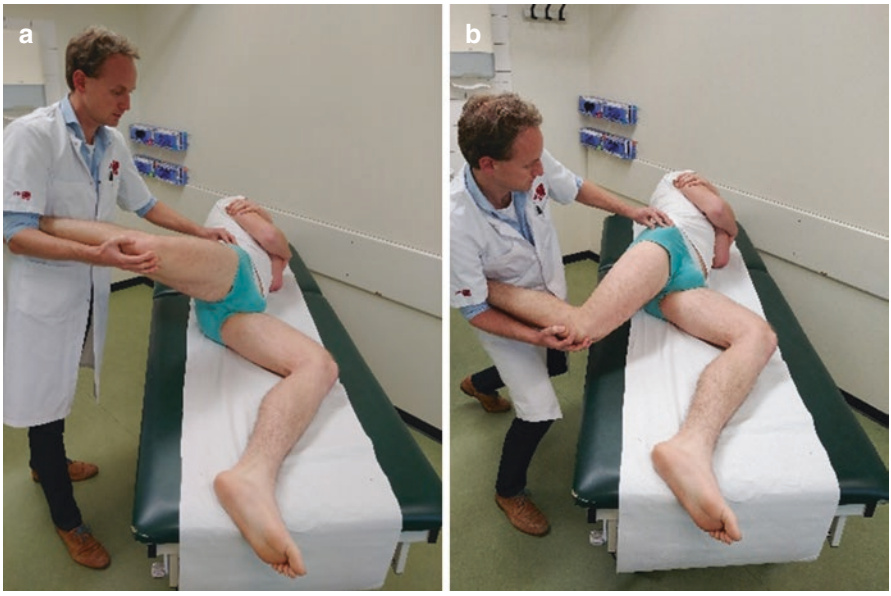

Fig. 7.6 (**a**–**b**) The ischiofemoral impingement test is performed with the patient in side lying position; the affected hip is taken into passive extension. The findings of this test are considered positive when the symptoms are reproduced in adduction or the neutral position (**a**), whereas extension with abduction does not reproduce the symptoms (**b**)

of clinical examination and imaging. Bony lesions include avulsion fractures of the ischium in the young or stress fractures of the ischial ramus or sacrum in the distance runners or gymnasts.

7.4.2 Imaging in Diagnosis and Prognosis

Zissen et al. [81] describe various MRI associations including peritendinous oedema, tendinopathy, partial tears and ischial bone marrow oedema in an interventional series [81].

De Smet et al. [74] reviewed a series of 118 pelvis MRIs (mean age 41 years, range 4–87 years) and related radiological findings to clinical history [82]. Of the 236 hemipelves, 21 had symptoms of hamstring tendinopathy and 215 were symptom-free. Notably, 90% of all hamstring tendons imaged had increased intratendinous T1 or T2 signal that was *not* associated with clinical features of hamstring tendinopathy. Symptomatic hamstring tendinopathy had greater width than nonsymptomatic tendons [75]. Peritendinous T2 signal at distal levels of the tendon of origin was more likely to be associated with clinical tendinopathy, as was associated ischial tuberosity oedema; however, these findings can also be seen in asymptomatic individuals. Importantly, in the opinion of the authors, imaging findings must

Table 7.2 Common PHT differential diagnoses

Common PHT differential diagnoses
PHT
SM partial tear of proximal tendon
Adductor magnus tendinopathy
Sciatic neuritis
Deep external rotator muscles (quadratus femoris, obturator internus, gemelli)
Avulsion fracture at the ischium
Stress fracture of the ischial ramus
Stress fracture of the sacrum
Ischiogluteal bursitis
Ischiofemoral impingement
Referred pain from lumbar spine, sacroiliac joint, semitendinosus or sacrotuberous ligament

align with physical examination to have validity in clinical decision-making. This opinion is also shared by Lempainen et al. [66].

As described above, the complexity of clinical presentations provides many differential diagnosis options (Table 7.2). Whilst detailed history, pain behaviour and physical examination provide the major elements required for diagnosis, individual cases may require further investigation. Contemporary approaches would include imaging as a means of assisting where the diagnosis is complex or unclear or where management course is not following expectations. Clinical reasoning utilising the findings of patient history, symptom behaviour, pain site and physical examination with MRI, ultrasound, plain X-ray or bone scan as appropriate provides the most complete means of inclusion or exclusion of these alternative diagnoses. Imaging has little to offer in terms of clinical progress or prognosis of hamstring tendinopathy.

7.5 Conclusion

Hamstring injury can be divided into three clinical scenarios: (1) acute hamstring injury, (2) hamstring injury sequela (repeated episodes of injury and/or pain) and (3) hamstring tendinopathy.

An acute hamstring injury is usually not hard to recognise in the clinical setting: an acute onset of posterior thigh pain and the triad of localised pain on hamstring stretch, resistance testing and palpation. With a relatively simple clinical examination including history taking, stretch, strength, palpation and specific hamstring tests, prognostic information can be obtained, although prediction of the injury duration for the individual athlete just after injury remains inaccurate. A delayed clinical evaluation about 1 week after injury seems to be more appropriate for prediction of injury duration. Additional imaging with MRI or sonography may complement the clinical assessment of hamstring muscle injury, although the value for injury prognosis in addition to clinical examination is limited. There is no evidence that imaging has a role in informing the RTP decision-making process.

Managing hamstring injury sequela is often challenging as scientific knowledge is limited. The diagnostic workup should be aimed at differential diagnosis of other causes of posterior thigh pain and evaluating (risk) factors that affect persistent or repeated hamstring complaints.

Presentation of hamstring tendinopathy differs from acute injury and is characterised by well-localised pain to the ischial tuberosity with no radiation or associated neural signs, generally provoked with prolonged sitting, cycling or lunging, squatting or change of direction activity. Clinical examination should seek to reproduce the patient's pain with combined passive stretch and compression tests. Additional (MRI) imaging only has value in clinical decision-making if aligned with physical examination, as imaging abnormalities are frequently observed in asymptomatic individuals.

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Muscle Injury Classification and Grading Systems

8

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8.1 Introduction

The "classification" of muscle injury refers to the process of describing or categorising a muscle injury according to type. Approaches to the classification of muscle injuries have utilised the site of injury (proximal versus distal), mechanism of injury (contusion versus non-contact), predominant tissue involved (tendon versus muscle), nature of onset (overuse versus acute) and imaging findings (magnetic resonance

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imaging (MRI) positive/negative). The effective classification of muscle injury is of relevance when determining the most appropriate management, informing patients of the injury nature, and when trying to evaluate the efficacy of different diagnostic or treatment approaches. By contrast, to "grade" a muscle injury is to provide an indication of the injury severity [1]. Severity may be determined by the injury history or mechanism, significance of the described symptoms and clinical signs and extent of imaging findings. However, from the perspective of athletes and coaches, the most important measure of injury severity is the length of time taken to return to full sports participation following injury.

Despite (or perhaps because of) numerous muscle injury classification and grading systems existing, international consensus on the most appropriate approach remains divided. The following chapter aims to provide an overview of existing (historical and current) classification and grading approaches to acute muscle injury.

8.2 History of Muscle Injury Classification and Grading

In the first half of the twentieth century, increasing participation in sporting activities led to clinical interest in the recognition and management of musculoskeletal injuries. Muscle injuries were recognised as a frequent occurrence and from an early stage were identified and described in terms of their various symptoms, signs and recovery. Reflecting this, in 1966, the American Medical Association (AMA) published a categorical system for clinically classifying and grading muscle injuries [2]. Despite lacking an evidence base, the approach of the AMA formed the basis of subsequent approaches to grading muscle injuries (see Table 8.1).

In the 1990s, the availability of MRI and ultrasound (US) allowed for the visualisation of underlying muscle structure and injury. Radiologists correlated clinical observations in injured patients with observed imaging characteristics and

Table 8.1 AMA system for muscle injury grading

First-degree strain (mild strain; slightly pulled muscle)	Trauma to musculotendon unit (MTU) due to excessive force or stretch. Localised pain aggravated by movement. Minor disability, mild swelling, ecchymosis, local tenderness. Tendency to recur. Minimal haemorrhage and inflammation with some disruption of musculotendon tissue
Second-degree strain (moderate strain; moderately pulled muscle)	Trauma to MTU due to violent contraction or excessive forced stretch. Localised pain, aggravated by movement. Moderate disability and swelling, with ecchymosis and local tenderness. Stretching and tearing of fibres but without complete disruption. Tendency to recur
Third-degree strain (severe strain; severely pulled muscle)	Trauma to MTU due to violent contraction or excessive forced stretch. Severe pain and disability. Severe swelling, ecchymosis, haematoma, with a palpable defect and loss of muscle function. Muscle or tendon rupture, including the musculotendon junction or avulsion with bone

Modified from Rachun, *Standard Nomenclature of Athletic Injuries.* First edition ed. Chicago, IL: American Medical Association; 1966 [2]

established early radiological grading approaches [3–6]. However, the initial radiology literature was limited by small sample sizes and as a result lacked any substantive evidence of a relationship between imaging appearance and prognosis [5–7].

In the early twenty-first century, using larger cohorts, researchers attempted to correlate MRI findings with clinical outcome [3, 8, 9]. Consistently, it was found that injures that were MRI negative for any observable abnormality had a significantly better prognosis than all other grades of injury [10–15]. Using a large sample of elite footballers with hamstring injuries, researchers also observed a statistically significant difference in clinical outcome between MRI-determined grades 1 and 2 muscle injury [8]. However, the wide variance observed in reported return to play (RTP) durations appears to limit the clinical utility of this finding [16]. Concurrently, researchers assessed MRI severity as a continuous variable determined by length, cross-sectional area and estimated volume of imaging abnormality and correlated these findings with RTP duration [11–13, 17, 18]. Methodological constraints and limitations in the data have meant that this approach has had limited success in predicting RTP duration [19]. Finally, disruption to the intramuscular tendon has been proposed as a key predictor of RTP duration, but further evidence is required to determine its true significance [20–23].

In recent years, there has been increased attention directed at developing standardised and practical approaches to muscle injury classification and grading [1, 21, 24–28]. The following section will briefly consider the strengths and weaknesses of the proposed systems.

8.3 Modern Approaches to Muscle Injury Classification and Grading

8.3.1 MRI-Based Muscle Injury Scoring Scale for Return to Play—Cohen et al. [25]

Nature:	Grading
Study sample:	National Football League (American football)
Muscle:	Hamstring

Cohen et al. proposed a novel scoring system based on age and a range of MRI variables including the number of muscles involved (1–3), location (proximal, middle, distal), insertional involvement (yes/no), cross-sectional percentage of muscle involvement, amount of muscle retraction (cm) and long axis muscle involvement. Each variable was allocated a score and the total score considered for severity assessment [25].

Analysing 43 National Football League injuries over a 10-year period for a relationship between "total MRI score" and the number of games lost to injury, the authors concluded that a rapid RTP was more likely in those injuries with an MRI score of less than 10, compared to a score of greater than 10. Indicators of a poor prognosis included multiple muscle involvement, a higher percentage (>75%) of transverse muscle involvement, more than 10 cm of craniocaudal involvement and

muscle retraction [25]. However, its 10-year retrospective nature, limited subject numbers, lack of detail regarding the RTP process and failure to be reproduced have limited this study's impact [29].

8.3.2 MRI- and US-Based Acute Muscle Strain Classification System—Chan et al. [24]

Nature:	Classification
Study sample:	N/A
Muscle:	General

Chan et al. described a three-layered image-based classification system for non-contact muscle injury, with the novel aspects pertaining to the detailed MRI-based description of the injuries' anatomical locations. Initially, radiologically classified as proximal musculotendinous junction (MTJ), muscle or distal MTJ, the injury is then further subclassified as proximal, middle or distal, before being defined by the principle tissue involved, specifically, intramuscular, myofascial, myofascial/perifascial, myotendinous or combined [24].

As a radiological classification, there is no published reliability or validity. This system has a narrow focus, having no inclusion for a primary tendon injury and no approach to more than one muscle being involved, and is unclear in terminology and taxonomy [24].

8.3.3 The Munich Consensus Statement—Mueller-Wohlfahrt et al. [26]

Nature:	Classification and grading
Study sample:	Professional football
Muscle:	General

The Munich consensus approach addresses both the classification of muscle injury and the grading of severity for non-contact muscle injury using both clinical and radiological information. As such, it is unique with respect to the modern era approaches by including a combination of clinical and radiological findings to define the nature of muscle injury.

Taxonomically, the classification distinguishes direct (contusion and laceration) from indirect muscle injury. Indirect muscle injuries are then further classified as either functional or structural injuries, sub-classified further into a type of injury, and finally divided into either a diagnostic group (e.g. fatigue-induced muscle disorder, DOMS, muscle- or spine-related neuromuscular disorder) or severity grade (minor partial, moderate, subtotal, complete or avulsion). Each classification/grade is defined and described with expected symptoms, signs and imaging findings. Research on UEFA footballers supported the observation that structural injuries (largely determined by those that are MRI positive for muscle

damage) have a greater time loss than functional injuries and that moderate and subtotal/total injuries have a worse prognosis than minor partial muscle tears [30].

While comprehensive in approach, elements of the construction of the Munich classification and grading system are based on principles that are not universally accepted. For example, the term "functional" in this classification has a specific meaning, distinct to its use in other areas of medicine [31]. While the use of the term "functional injuries" may be clinically appealing, there remains only limited basis upon which to base functional diagnoses such as "spine-related neuromuscular muscle disorder" and "muscle-related neuromuscular muscle disorder" [32]. It could be argued that functional injuries actually reflect either microscopic (structural) damage below the current resolution of imaging or a combination of factors.

With regard to non-contact muscle injuries, the Munich consensus utilises a mixed approach of three classifications [26]. Minor and moderate partial tears (type 3A and 3B) are differentiated taxonomically from (sub)total muscle tears/tendinous avulsions (type 4). There is no distinction based on the specific tissue involvement (such as the intramuscular tendon); rather, the separation is based on the extent of the injury as determined by imaging and clinical appearance, with many similarities to the nomenclature of the 1960s [2].

Efforts to validate the Munich classification system [30] revealed a wide range in RTP durations for minor partial (3a), moderate (3b) and (sub)total tears (3–132, 8–111 and 52–61 days, respectively). This observation suggests the system may have limited utility in predicting the RTP duration for these distinctly classified muscle injuries.

8.3.4 British Athletics Muscle Injury Classification—Pollock et al. [27]

Nature:	Classification and grading
Study sample:	Track and field
Muscle:	Hamstring

Based on hamstring injuries in elite track and field athletes, this approach specifically addresses non-contact injuries [27]. The approach grades injury severity from 0 to 4 based on a combination of clinical and MRI features, before refining grades 1–3 to reflect the predominant structure involved (myofascial, muscle tendon junction or intra-tendinous). Grade 0 injuries are those that are MRI negative, and an additional differentiator of "N" may be applied to any grade when there is a clinical "suspicion of a neural component" to the injury. The authors have illustrated substantial levels of intra- and inter-rater reliability [33] and, in a subsequent retrospective study of 65 hamstring injuries, assessed the time to return to full training (TRFT) and recurrence rate versus the grade and classification of injury [21]. MRI-negative (grade 0) injuries were associated with a shorter TRFT than all other injury grades, but there was no difference in prognosis between

grades 1 (small tear) and 2 (moderate tear) or between myofascial and MTJ injuries. There was also a significant difference in both RTP and re-injury rate for injuries that involved the intramuscular tendon [21]. While an independent prospective study applying this approach in a football cohort found an overall effect for severity grading and anatomical sites [33], they accounted for only 7.6–11.9%, respectively, of the total variance in time to RTP. Subsequently, while the length of time to RTP on average was greater for higher-grade injuries, we urge clinicians to look beyond the average values and to consider the implications of the overlap (variance) between the injury categories when considering its prognostic utility.

The degree of anatomical detail provided by this approach is enticing and may be relevant for determining best practice treatment modalities. The approach has been shown to be reliable and is based upon the available evidence of prognostic elements involved in muscle injury. Contusions are ignored in this approach to muscle injury due to their limited relevance in track and field.

8.3.5 The MLG-R Muscle Injury Classification System—Valle et al. [28]

Nature:	Classification and grading
Study sample:	N/A
Muscle:	General

Driven by the experiences of the Barcelona Football Club and in collaboration with international colleagues, a muscle injury classification and grading approach based on four taxonomic layers was proposed [28]. This classification system for muscle injuries is based on a four-letter initial system: MLG-R, respectively, referring to the mechanism of injury (M), location of injury (L), grading of severity (G) and number of muscle re-injuries (R). Based on clinical history, the first identifier distinguishes the mechanism of injury as either direct (D) or indirect (I), with indirect injuries additionally identified as sprinting or stretch related. The second and third major identifiers are MRI variables describing the anatomical location and grade of the injury, respectively. The grade of the injury is determined by specific features of oedema and haemorrhage and the cross-sectional area of signal hyper-intensity. The final identifier (R) relates to the re-injury status.

Unique to this system is the incorporation of re-injury status into the grading. The presence of re-injury may influence rehabilitation progression and RTP decisions and may therefore be relevant in a classification paradigm. There is currently no reliability or validity study on the potential of this system to provide distinguishing prognoses.

While the detailed approach to the injury description supports the effective understanding of the injury nature, it includes a complex nomenclature which may limit the appeal of the system to the broader clinical and sporting community.

8.3.6 Grading Based on Connective Tissue Injury—Prakash et al. [34]

Nature:	Grading
Study sample:	Various sports
Muscle:	Calf

Prakash et al. grade injury severity 0–3 based on connective tissue involvement on MRI in calf injuries. They define grade 0 injury as oedema or fluid adjacent to an intact tendon/aponeurosis/epimysium without myofibril detachment; grade 1 injury as myofibril detachment without tendon/aponeurosis/epimysium change; grade 2 injury as myofibril detachment with adjacent tendon/aponeurosis/epimysium increased signal, delamination or defect, but no retraction; and grade 3 injury as myofibril detachment with adjacent tendon/aponeurosis/epimysium retraction indicating failure.

In a retrospective analysis in 100 patients with calf injuries, they found a correlation between higher grade and longer time to RTP. Although the time to RTP on average was greater for higher-grade injuries, there is an overlap between the grades which limits prognostic effectiveness in any individual athlete. Furthermore, to what extent these results can be generalised to hamstring injuries remains unknown.

8.4 Conclusion

In the last decade, traditional means of categorically describing and grading muscle injury have been challenged. This has been largely driven by the high rate of muscle injury and the desire of athletes and coaches to have an accurate prognosis for RTP. An ongoing challenge to any consensus on muscle injury nomenclature is a consistent approach to language. While there are broad similarities in the description of the classic non-contact sprinting injury to muscle, there remains variability in the language utilised (e.g. tear, strain, injury). The Munich consensus argued against the use of strain to describe non-contact muscle injury due to its confusing history and implied aetiology and suggests preferentially utilising the term ‘tear’. The use of “tear” is also the preferred approach of British Athletics, while Chan et al. continue to utilise strain, and both Valle et al. and Cohen et al. utilise the neutral term “muscle injury”. These differences may be superficial, but there remain other disagreements in language that may not be so easily reconciled. This includes the use of non-standardised terminology such as functional, muscle-related neuromuscular disorder and other diagnoses, which potentially reflect the range of experience of clinicians involved in the classification development and other factors which may challenge consensus.

While increased availability of imaging and larger research cohorts has led to increased understanding of relevant features in determining RTP, ultimately, injury rehabilitation is influenced by a myriad of pathophysiological, social and

psychological factors. As a result, the clinical and radiological appearance of any given injury at a single time point in an injury process is unlikely to provide more than a small part of the prognostic picture [35, 36]. This may also partly explain the limited scientific evidence for the prognostic efficacy of such approaches.

Ultimately, a clear diagnosis, based on the accumulated evidence from a careful history, examination and imaging, should assist in the prescription of an appropriate rehabilitation programme. This should be the goal of any clinician. When classifying and grading muscle injury using any of the current approaches for the purposes of providing a prognosis, this should be done while recognising the limitations incumbent in that system. In essence, more evidence on the clinical utility of these systems is needed prior to clinical practice being able to rely solely on a particular classification and grading system.

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Clinical Assessment of Hamstring Injury and Function

9

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9.1 Introduction

The starting point of an effective clinical assessment and a subsequent rehabilitation programme, is a thorough subjective history. This initial injury history will allow the clinician to create a differential and individual clinical diagnosis and give direction to further examination and evaluation processes. Information on the mechanism of injury is important. With acute hamstring injuries, the mechanism is generally obvious with a sudden, sharp acute posterior thigh pain, occasionally accompanied by a popping or pulling sensation, causing an immediate cessation of activity [1]. Acute hamstring injuries can occur during a variety of athletic manoeuvres and situations, resulting in several distinct types of injuries, each with a unique mechanism. The most common type of acute hamstring strain occurs during high-speed running [2, 3]. Irrespective of mechanism (i.e. which phase of sprinting), these injuries appear to preferentially involve the biceps femoris (BF) in comparison to the medial hamstrings at a rate of approximately 4:1 [4, 5]. Another type of acute hamstring injury is a stretch-type injury, where the hamstring muscle is stretched into an end-range

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position with the hip flexed while the knee remains extended, or the athlete is pulled forward rapidly, such as water skiing or moving into split position [6]. These acute stretching-type injuries often damage the proximal free hamstring tendon, particularly the semimembranosus (SM) portion, and it has been reported that these stretching injuries take significantly longer to return to premorbid level of activity compared to acute sprinting-type injuries, meaning that athletes should anticipate a longer rehabilitation period if the proximal free tendon is involved [7–9].

Hamstring injury sequelae are persistent and/or repeated complaints of posterior thigh pain and dysfunction related to a previous injury or injuries, in which symptoms fail to fully resolve or continually reoccur without a distinct reinjury. Establishing a differential diagnosis is particularly important to rule out the possibility that pain is not referred from adjacent muscles, joints or neural tissues in the lower back, hip and pelvis region.

Gradual-onset hamstring muscle injuries often present as a diagnostic challenge for the clinician due to the lack of a discrete injury or aetiology. Tendinopathy generally develops as result of excessive load to the tendon tissue and results in tendon thickening, frequently causing chronic, localised pain. For this reason, determining a loading profile may shed light on the causative factors for the current pathological condition. It may also serve as a guide for the clinician in determining the magnitude and timing of load intervention to be applied during rehabilitation and return to sport (RTS). Baseline loading assessment will assist the clinician with important information concerning relevant exercise and running prescription and progression. Proximal hamstring tendinopathy (PHT) is often a result of training errors, such as increasing frequency and intensity too rapidly [10] which may cause excessive tensile loading beyond the capacity of the tendon. There is also biomechanical evidence demonstrating a shearing force between the ischial tuberosity and the proximal hamstring tendon attachment when replicating loading in in-vivo models [11]. For this reason, it behoves the clinician to investigate any common movement or loading patterns that the athlete is performing which may be causing and/or exacerbating symptoms, so that these can be modified and addressed in rehabilitation.

9.2 Subjective Assessment

As with any pathology, a logical place to start an assessment is with subjective history taking. During the patient interview, the clinician will start with broad history taking about mechanism of injury, onset of symptoms, and the progression of pathology and then funnel down the interview to more specific aspects of the history. With this top-down questioning, the clinician may deductively establish diagnosis and aetiology. The clinician may also use hamstring injury-specific patient-reported outcome measures to measure the severity of symptoms and dysfunction as well as to quantify and monitor changes throughout the rehabilitation process.

9.2.1 Open Questions for History Taking

A good place to start the subjective assessment is to use open-ended questions. This will enable the clinician to start out from general questions and then filter down into more symptom and history-specific questions as more information is garnered. Examples of simple open-ended questions include the following:

- Tell me about your symptoms: Can you describe your symptoms qualitatively? What is the exact location (e.g. near gluteal fold or mid-substance of hamstring muscle belly, medial, or lateral) and nature of your posterior thigh pain (e.g. sharp, diffuse, burning)? When did you first start noticing your symptoms? How have your hamstring symptoms evolved over time? Was there any change in activity level or intensity preceding symptoms? What aggravates or alleviates your current hamstring symptoms? What movements/activities are limited/prevented as a result of your symptoms? Can you recreate your symptoms?
- Tell me about the mechanism of injury: Did your symptoms occur suddenly or was the onset gradual?
- Tell me about your injury history: Have you previously experienced a similar hamstring injury, or do you have a history of similar posterior thigh symptoms, and if so, how often/when was the last occurrence? If you've had similar symptoms previously, what interventions have you tried, and what has and hasn't helped? Do you have any other symptoms or pain in parts of your body other than your posterior thigh region?
- Tell me about your tolerance to specific loads: Can you run? How far/long can you run before onset of symptoms? How long does the pain last? Does the pain go away with activity? How intense is the pain at its worst, and on average (rated from 0 to 10)?
- Tell me about how this injury affects you: How has your activity level changed since the injury? Have you had to cease/modify all or parts of your training or daily activities?

9.2.2 Hamstring-Specific Questions

With this background information, the subjective interview can then shift to more hamstring-specific information that will need to be incorporated into the assessment and treatment programme. Hamstring muscle injuries range from acute, sudden onset to long-standing sequela and insidious gradual onset with the athlete unable to recall a discrete injury episode, with severity ranging from benign delayed onset muscle soreness to total tendon ruptures [12]. For an acute hamstring injury, the hamstring-specific line of questioning and reasoning include the following:

- *Tell me, how did this happen? Were you in the middle of training or game?* If the injury occurred during a specific aspect of training (e.g. plyometric drills, a particular stretching exercise, sprint technique drill), it's important to understand the nature of

this demand the athlete is required to be able to tolerate. Perhaps, this aspect of training can be replaced by something achieving the same goals with less risk, or at least temporarily replaced in the training programme after consultation with the coach.

- *Tell me, were you able to walk off by yourself, or did you need help? Can you walk without pain now? How long did it take until you could walk without pain?* Early research suggested walking pain free within 2 days of an acute hamstring injury was associated with a shorter rehabilitation duration [13], although this is controversial [14]. Athletes reporting extreme pain at the time of injury, perhaps with a "pop" sensation or close to 10 on a visual analogue scale (VAS) of 0–10, and who required assistance from the field need to be considered as possibly having complete ruptures/avulsions for which imaging and a surgical opinion may be indicated. Both the initial level of pain and its change over time appear to be related to the ultimate duration of rehabilitation [14, 15].
- *Tell me, were you running, kicking, stretching, or bending to reach for a ball or something else?* The specific mechanism of injury will guide treatment because it is important to restore the athlete's ability to perform these movements proficiently and with confidence. Differentiating high-speed running injuries as stance- or swing-related may help to optimise injury prevention and performance [15], so it may be of interest to ask athletes when they felt their injury occur. This may provide direction in the design of the rehabilitation programme.
- *Tell me, did this injury follow a bout of intense or unaccustomed exercise?* For an acute hamstring injury, it is important to distinguish between muscle strain injury and exercise-induced muscle damage as they are very different entities, with the latter rarely requiring treatment [16].
- *Tell me, how much pain did you experience at the time of the injury? What about later that night and the next day? How is your pain now just on everyday things like walking and getting up and down the stairs and in and out of chairs?* Occasionally, most of the pain appears to arise from fluid accumulating in the tissues adjacent to the injury. In this case, pain will worsen in the day or so after the injury and can be problematic in rehabilitation as this collection of fluid sitting in the thigh is uncomfortable and can be worrying as it "moves around" while the athlete exercises. It is important to reassure the athlete that this sensation is not "damaging" and not a cause to delay progression of their rehabilitation. This fluid is often palpable as a thickened, turgid area, and when imaging is not available, this may be your only indication of its presence.

If the athlete presents with more gradual-onset hamstring symptoms, another line of questioning and reasoning may be required as follows:

- *Tell me, are your symptoms insidious in nature? Has there been a recent change in the intensity, duration, or frequency of your training?* When there is no discrete, acute mechanism of injury that causes posterior thigh pain, the clinician must consider tendinopathy, particularly when the pain is near the proximal hamstring tendon. As with other tendinopathic conditions, PHT most often correlates with repetitive stress to the hamstring muscle complex [17] or with tasks involving repetitive hip flexion [18].

- *Tell me, where is the pain located?* Location of the pain will give the clinician indication as to what structures are injured. Proximal hamstring tendinopathy generally manifests in the lower gluteal region, near the ischial tuberosity, and may occasionally radiate distally into the posterior thigh [17]. Pain at the distal SM tendon may be indicative of SM tendinopathy or irritation to the surrounding U-shaped bursa, although these conditions are rarely reported in the literature [19]. Acute hamstring injuries will present in the muscle belly and are more frequently lateral than medial as the BF muscle is implicated more often than the semitendinosus muscle [20].
- *Tell me, does the pain occur or increase with long periods of sitting, sitting on hard surfaces, or deep squatting?* The proximal hamstring tendon attaches to the ischial tuberosity making it an insertional tendinopathy and vulnerable to compression with hip flexion and adduction. Compression of pathological tendons is thought to be a key component in eliciting or exacerbating tendon pain [21].
- *Tell me, do you have any metabolic issues? Have you experienced hormonal changes recently? Are you taking fluoroquinolone antibiotics?* Metabolic issues such as glucose intolerance or insulin resistance [22], changes in hormone levels [23], and fluoroquinolone antibiotics [24] have each been shown to be a risk factor for developing tendinopathies.

9.2.3 Patient-Reported Outcome Measures

Another more standardised but also less individual way to quantify self-reported aspects of hamstring injury is through the use of patient-reported outcome measures. In addition to covering specific standardised questions that may give the clinician a quick overview of situations in which the patient has pain or functional limitations, the patient-reported outcome measures can also be an efficient way to objectively assist the examiner in determining the severity of the injury. Objectifying the severity of injury can help the clinician to monitor the progress of the patient's symptoms, which may play a role in the clinical decision-making during the rehabilitation process [25]. The following specific patient-reported outcome measures have been developed for specific hamstring pathologies.

9.2.3.1 Functional Assessment Scale for Acute Hamstring Injuries

For athletes presenting with acute hamstring muscle injury, there is the Functional Assessment Scale for Acute Hamstring Injuries (FASH) [26]. The FASH was constructed by a committee of experts on hamstring muscle injury based on the literature, a focus group, and athlete interviews. The FASH consists of ten questions used to determine an athlete's pain level with various movements, such as jogging and static stretching, along with the athlete's ability to perform relevant movements, such as the Nordic hamstring exercise (NHE) and one-legged hops. The FASH quantifies the responses using VAS of 0–10 with a possible total of 100 points, with 100 indicating highest level of physical function. The FASH has been shown to have excellent reliability (intraclass correlation coefficient (ICC), 0.9; $p < 0.001$), good concurrent

validity compared to Victorian Institute of Sport Assessment-Proximal Hamstring Tendons (VISA-H) Questionnaire (r, 0.856; $p < 0.01$), good internal consistency (α, 0.98), and large effect size, indicating good responsiveness [26]. Due to the responsiveness of the FASH it may be utilised to monitor changes in symptoms and clinical progression. The minimal detectable change (MDC) (based on a 95% confidence interval) for those with a hamstring injury was 3.05 [26]. This questionnaire is also recommended for athletes with hamstring injury sequela.

9.2.3.2 Perth Hamstring Assessment Tool

Another patient-reported outcome measure designed for proximal hamstring muscle injury is the Perth Hamstring Assessment Tool (PHAT) [27]. Like the VISA-H, the PHAT quantifies how long an athlete can perform various activities such as sitting, driving, and running along with how much pain they experience while doing these. The PHAT has been shown to have high internal consistency (α, 0.80), high reproducibility (ICC, 0.84), and high sensitivity to change on patients with surgically repaired proximal hamstring tendons and is therefore recommended to objectively monitor operative cases of the proximal hamstring tendon repair [27].

9.2.3.3 Victorian Institute of Sport Assessment-Proximal Hamstring Tendons (VISA-H) Questionnaire

For PHT, there is the VISA-H Questionnaire [28]. The VISA-H was developed following the successful introduction of the VISA-A (Achilles tendon) and VISA-P (patella tendon) and is designed specifically for the proximal hamstring tendon. Like the FASH, the VISA-H was developed based on a literature review and interviews with experts and athletes presenting with PHT. Questions are designed to quantify pain with various activities such as sitting, stretching, running, and participating in sport. The VISA-H is simple to administer, consists of eight questions, with a possible score ranging from 0 to 100, where a score of 100 indicates no dysfunction. The VISA-H has been shown to be valid (r, −0.89 with p, 0.001; r, 0.88 with p, 0.001) in nonsurgical subjects compared to the Nirschl phase rating scale and the generic tendon grading system proposed by Curwin and Stanish. The VISA-H has also been shown to be reliable (ICC, 0.92; 95% CI, 0.80–0.97 for nonsurgical subjects) for measure of pain, function, and sporting activity in individuals with PHT [28]. The VISA-H has been shown to have a large effect size (2.2) and standard response mean (1.6) indicating good responsiveness. The minimum clinically important difference is 22 points [28].

9.3 Physical Assessment

The thorough and accurate subjective history provides a focus for the physical assessment. Physical assessment of hamstring injury has mainly been described in relation to acute hamstring injury, and the following section describes some of the most common and practical ways to physically assess hamstring function and to do so in an

objective way. We suggest that strength and flexibility testing can also be used in clinical situations in which patients present with hamstring injury sequela and hamstring tendinopathy. As these measures are predominately developed and tested on acute hamstring muscle injury, the application of these measures for hamstring injury sequela and hamstring tendinopathy needs to be based upon individual clinical reasoning. Assessing pain on palpation of the muscle and tendon and deficits in both strength and flexibility are appropriate for these pathologies as they are commonly seen clinically in patients with hamstring injury sequela and tendinopathy [29].

9.3.1 Inspection and Palpation

A key component of the physical assessment of a hamstring muscle injury is visual inspection and palpation of the local tissue. Ecchymosis may be assessed visually following acute hamstring muscle injury; however, hematomas that are small and located deep in the muscle belly may not manifest on the skin [30]. Broad ecchymosis in the posterior thigh may indicate a proximal avulsion injury [31], although in the absence of an avulsion it is of little clinical predictive utility [14, 15, 32].

The hamstring muscle belly and its proximal insertion to the ischium should be explored for pain or tenderness to palpation. Focal tenderness can tell the examiner several key things about clinical diagnosis and prognosis. Our clinical experience is that it is important to palpate both injured and healthy legs simultaneously while questioning the athlete as to whether one side is painfully different to the other rather than simply asking if the injured side hurts. Patients with an acute painful hamstring injury can be quite fearful of the examination, and incautious "poking" and asking about pain is almost always met with a positive response. There is value in palpating a healthy region (e.g. the proximal calf) and explaining to the patient that this area should feel uncomfortable on firm palpation in both limbs. This approach allows the patient to differentiate between palpation of healthy and injured tissue.

Tenderness to palpation of the proximal hamstring tendon has been shown to be consistent in locating the most painful point and is in agreement with findings on magnetic resonance imaging [8]. Research has shown that an acute-onset injury associated with focal tenderness close to the ischium is typically associated with a longer time to RTS, at least pertaining to the stretch type of proximal hamstring injuries [8]. The proximal hamstring tendon can be palpated by locating the ischial tuberosity in prone with legs extended with the gluteal fold serving as a useful landmark to find the ischial tuberosity. A study by Askling et al. [9] found the average distance from maximal tenderness to the ischial tuberosity in athletes with stretch-type injuries to be two centimetres; however, there was no correlation between length of tenderness and return to play (RTP) time.

Proximal-to-distal length of tenderness to palpation (as a percentage of total posterior thigh length) in acute mid-substance hamstring injury (i.e. non-proximal hamstring injury), deemed "hamstring mapping," has been shown to be related to time to RTP from day of initial evaluation (R^2, 0.72) [33]. Furthermore, the length of tenderness during rehabilitation is also related to RTP from the day of that

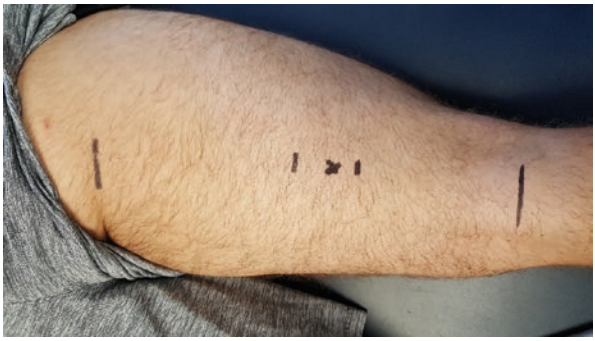

Fig. 9.1 Hamstring map displaying longitudinal tenderness to palpation with borders delineating posterior thigh. In order to use this measurement clinically, the clinician measures the length of tenderness and divides it by the distance of the posterior thigh (i.e. gluteal fold to popliteal fold) which will express the tenderness as a percentage of the posterior thigh length. This number can then be plugged into the regression model, *predicted days until return to sport (RTS)* = 2.70 × (% of tenderness) − 12.25. For example, if the tenderness was 22% of the thigh, the equation would read *predicted days until RTS* = 2.70 × (22) − 12.25 = 47 days predicted RTP

respective measurement (R^2, 0.68) [33]. By garnering this palpation information in the assessment, the clinician may then use this to assist in establishing a prognosis. The hamstring mapping is done with the athlete in prone with the thigh exposed and consists first of marking proximal and distal borders of the posterior thigh using the gluteal fold and popliteal fold, respectively. The area of peak tenderness is then found and marked by the examiner. From this site of peak tenderness, the examiner moves one thumb breadth proximally and while applying firm, consistent pressure asks the athlete whether they sense "pain" or "pressure." If palpation results in pain, the examiner moves one more thumb breadth proximal and again applies pressure. Where the athlete reports "pressure," a line is marked indicating the proximal extension of tenderness. This same process is then repeated to ascertain the distal extension of tenderness. This proximal-to-distal length of tenderness can then be measured and taken as a percentage of the total posterior thigh (gluteal fold to popliteal fold) (Fig. 9.1) which can be inserted into the linear equation to determine predicted days until RTP. Furthermore, the change in the length of pain documented on daily examination has shown a clinically useful association with RTP such that, once the length of pain is approximately half its initial length, the athlete is approximately halfway through their rehabilitation [15].

9.3.2 Hamstring Flexibility

Clinical examination of knee extension and hip flexion range of motion (ROM) is a common practice to evaluate hamstring flexibility. This can be measured both actively and passively as well as at multiple hip flexion angles. In addition to measuring the flexibility, pain and apprehension should also be noted.

9.3.2.1 Passive and Active Knee Extension Tests

Several tests are available to practitioners, and recently, two tests, passive and active knee extension (AKE) tests [34], have been recommended as RTS criteria. The passive straight leg raise (PSLR) test is associated with marked reductions of approximately 21% in ROM during the initial 9 days post-injury [35]. Impaired ROM may remain up to 40 days post-injury, but the magnitude can be expected to decline to about 13% on days 10–20 with a smaller and probably irrelevant deficit of 6% 20–30 days post-injury [35]. Practitioners can therefore expect increases in ROM weekly during early stages of rehabilitation but should not necessarily expect daily gains or a complete resolution of deficits prior to 30 days post-injury when making decisions around returning athletes to sport. By contrast, if there is a lack of, or a reduction in, ROM gains, it may indicate severe injury or an unwanted response to rehabilitation loads.

A recent meta-analysis of hamstring flexibility after hamstring strain injury did not identify significant differences in AKE between injured or non-injured limbs [35]. However, one study found the AKE test to be an independent predictor of hamstring reinjury when mean deficits of 2° are present a week after returning to sport [36]. An absolute deficit in AKE, defined as the difference in total ROM between the injured and uninjured limb, increased the risk of reinjury by 13% for every degree of AKE deficit [36].

- For the passive knee extension test, the athlete is put in supine with legs extended. The examiner bends the involved knee to 90° and lifts the involved leg until the hip is in 90° flexion. The examiner then passively increases knee extension until the maximal tolerable stretch was experienced. The contralateral leg remains flat on the examination table. At the end point of maximal tolerable stretch, the knee flexion angle is measured and recorded. This process is repeated on the contralateral side for comparison (Fig. 9.2). The passive knee extension test has good intertester reliability (ICC, 0.77) [37] and fair correlation to the straight leg raise test (r, 0.63) [38].
- The AKE test is performed in a similar manner as the passive knee extension test, however, with the athlete actively performing the knee extension component. To administer the AKE test, the athlete is positioned with the ipsilateral hip in 90° flexion. The athlete will then extend the knee until experiencing maximal tolerable stretch, with the contralateral leg fixed flat on the examination table. The absolute knee angle is then measured and recorded [39] (Fig. 9.3). Again, this process is repeated on the contralateral side for comparison. The intertester reliability of the AKE test (ICC, 0.89) is superior to the passive test (0.77) [37].

In both the passive and the AKE tests, the flexibility measured can be compared to the contralateral leg to establish the absolute flexibility deficit. The minimal detectable difference in an injured limb is 21° with the passive test and 15° with the active test [37].

Fig. 9.2 The examiner passively extends the knee as far as possible with the athlete's hip flexed to 90°. A goniometer or inclinometer is used to measure the knee flexion angle or tibial position, respectively

Fig. 9.3 The athlete actively extends the knee as far as possible with the athlete's hip flexed to 90°. A goniometer or inclinometer is used to measure the knee flexion angle or tibial position, respectively

9.3.2.2 Maximal Hip Flexion and Active Knee Extension

Recently, a clinical hamstring flexibility test that combines maximal hip flexion and active knee extension (MHFAKE) has demonstrated potential to assist in decision-making around RTP [40]. Whiteley et al. [40] compared the traditional straight leg raise test to the MHFAKE test (expressed as a percentage of the uninjured leg at initial examination). The research shows that relative straight leg raise flexibility is essentially normalised early in rehabilitation with very few subjects showing <90% of the uninjured leg within a few days of commencing rehabilitation [40], thus limiting its utility in assessment during much of the rehabilitation process. The MHFAKE, alternatively, is a promising clinical indicator varying from approximately 70% of the uninjured leg early up to 100% by the end of rehabilitation. The test is documented to have an inter-rater $ICC_{(2,1)}$ of 0.96 (0.92–0.98) and is associated with an MDC of approximately 11%. These features suggest it's a more useful clinical measure of flexibility than the straight leg raise [40].

- The MHFAKE test is an adapted AKE test, where the athlete keeps the hip in maximal flexion by clutching the thigh to the chest, with the hands holding opposite elbows, and then actively extending the knee until reaching the point of maximal tolerable stretch of the hamstring muscle or the point where pain is reported, and this angle is then recorded with an inclinometer. The examiner may use his leg to stabilise the athlete's contralateral leg (Fig. 9.4).

Fig. 9.4 To perform the MHFAKE test, the athlete embraces the thigh to the chest and actively extends the knee as far as possible. The examiner should be positioned with one leg on the contralateral leg to stabilise the athlete to minimise pelvic excursion and use a digital level or inclinometer to measure the angle of the tibia

9.3.3 Hamstring Strength Assessment

Hamstring strength is important for sporting activity as increased hamstring strength has been demonstrated to have an effect on improved running performance, load tolerance, and injury risk profiles [20, 41, 42]. The importance of hamstring strength is greater post-injury [43] and is therefore indicated in the RTS criteria-based decision-making process. Previously injured hamstrings have even demonstrated significant strength deficits as long as 2–3 years post-injury [43–45]. Therefore, it is critical that strength deficits are identified in the initial assessment, when appropriate, and as a part of ongoing assessment throughout the course of rehabilitation.

When choosing a strength assessment technique, a myriad of factors need to be considered. The rehabilitation professional must consider several issues including whether or not the muscle is healthy (as in preseason baseline testing) and whether the muscle is acutely injured or a result of hamstring injury sequela. Also, one must take into consideration the performance demands of the muscle based on the specific demands of the subject's sport. It should be apparent that the expression of hamstring strength is, to some extent, specific to the test employed. Contraction type and hip angle are also considerations for the practitioner when selecting a hamstring strength test [46] for RTS criteria-based decision-making. Hamstring peak torque, irrespective of contraction type, is not achieved at 0° of hip flexion [46], and as demonstrated in the following section, both hip and knee flexion angles play a critical role when performing the strength assessment.

9.3.3.1 Isometric Strength Measurement

Isometric hamstring strength testing should be performed at both mid- and outer ranges and is indicated throughout the RTS continuum as it can inform the criteria-based decision-making process. A standardised hip flexion angle of 45° tested in prone or a multi-angle lengthened state measurement (both described below) should therefore be considered. Isometric hamstring strength RTS criteria should demonstrate restoration, within the test–retest minimal detectable change 95% confidence interval (MDC95% CI), to pre-injury levels of the same limb, or compared to the uninjured leg in the absence of pre-injury data. Clinical interpretations of between-limb strength should, however, recognise that the dominant leg has been found to produce significantly greater force [47].

To determine an accurate measurement of strength, force must be quantified which is most frequently done using a dynamometer, either in the handheld form or with an isokinetic device. Often, practicality plays a critical role in hamstring strength assessment. For example, when assessment is performed in an on-site setting where transportation of an isokinetic device is impractical or cost is an issue, a handheld dynamometer may be used, given the portability and affordability of a handheld dynamometer, and therefore, it may be more practical relative to an isokinetic device. Hamstring muscle strength recorded isometrically with a handheld dynamometer is reproducible and significantly correlated with isokinetic testing [48], indicating that

this method may in some cases be a useful alternative to an isokinetic dynamometer. One caveat is that the handheld dynamometer should not be used when measuring knee flexion-to-extension strength ratios as research has shown it to not be a valid measurement when compared against the isokinetic standard [49].

In fact, researchers have established excellent inter-rater reliability for isometric hamstring strength assessment at midrange, both in the seated position ($ICC_{(2,1)}$, 0.83 (0.68–0.90); MDC%, 15.5) [50, 51] and in prone [52].

Wollin et al. [47] developed and investigated the reliability of an externally fixed dynamometry isometric hamstring strength test designed to mimic hip and knee angles associated with the terminal swing phase of the running gait cycle. Standardised isometric hamstring strength testing included 45° of hip flexion and 30° of knee flexion from terminal extension. The test demonstrates good absolute and relative reliability, ICC (95% CI) 0.86 (0.74–0.93), standard error of measure 5.0%, and MDC95 14.0% [47].

To perform this test, the athlete is positioned in prone on the 45° wedge with anterior superior iliac spine of the pelvis placed at the peak of the inclined wedge (Fig. 9.5). A metal clip seat belt is placed five centimetres proximal to the distal point of the lateral malleolus. The examiner ensures that the knee is placed on the wedge during the testing. Two warm-up repetitions (5 s duration) are performed. The first warm-up is followed by 10 s rest, and 20 s of rest is allowed between the final warm-up and maximal test effort. The athlete is instructed to bend their knee to take up the slack in the seat belt. The examiner uses standardised instructions of "go ahead, pull, pull, pull, pull, and relax" for each repetition. Verbal encouragement is applied for the maximal test. In lieu of a specialised table as used in the studies, a foam wedge may be used to obtain this hip flexed, prone position, making the procedure simpler in a standard clinic.

This method has demonstrated ability, at preseason screening, to identify previously injured hamstrings 3 years post-injury [44]. Additionally, standardised unilateral isometric hamstring strength [47] is impaired after competition [44, 53, 54]. A 12.5% reduction in peak isometric knee flexor torque differentiated previously injured to uninjured hamstrings in sub-elite Australian rules football players [44].

Fig. 9.5 Standardised isometric hamstring strength testing included 45° of hip flexion and 30°of knee flexion from terminal extension

Lengthened State Hamstring Strength Measurement

In addition to midrange strength testing, the outer range or lengthened state position should be tested as well. Strength testing, particularly at long lengths (outer range), is clinically possible and relevant since injury-related strength deficits can be persistent [35, 40]. It is also a risk factor for hamstring reinjury [36]. A relative force deficit, defined by dividing the maximal force of the injured leg and non-injured leg, indicates a 4% increased reinjury risk for every Newton force deficit on testing 7 days after returning to sport [36].

Researchers have hypothesised that hamstring weakness in an elongated position reflects a chronic shortening of the hamstring muscle fibres and a subsequent leftward shift in the muscle length–tension relationship. Subsequently, Tyler et al. [55] used multi-angle isometric strength assessed bilaterally at 80°, 60°, 40°, and 20° knee flexion while the hip was placed in 120° flexion (Fig. 9.6) to provide a measure of the length–tension relationship for the descending limb of the torque curve in order to identify hamstring lengthened state weakness compared to the contralateral limb (Fig. 9.7). In this test setup, the knee flexion angle was 40° when the dynamometer arm was horizontal (parallel to the floor). The limb mass and torque due to passive hamstring tension should be subtracted from torque values at each angle to

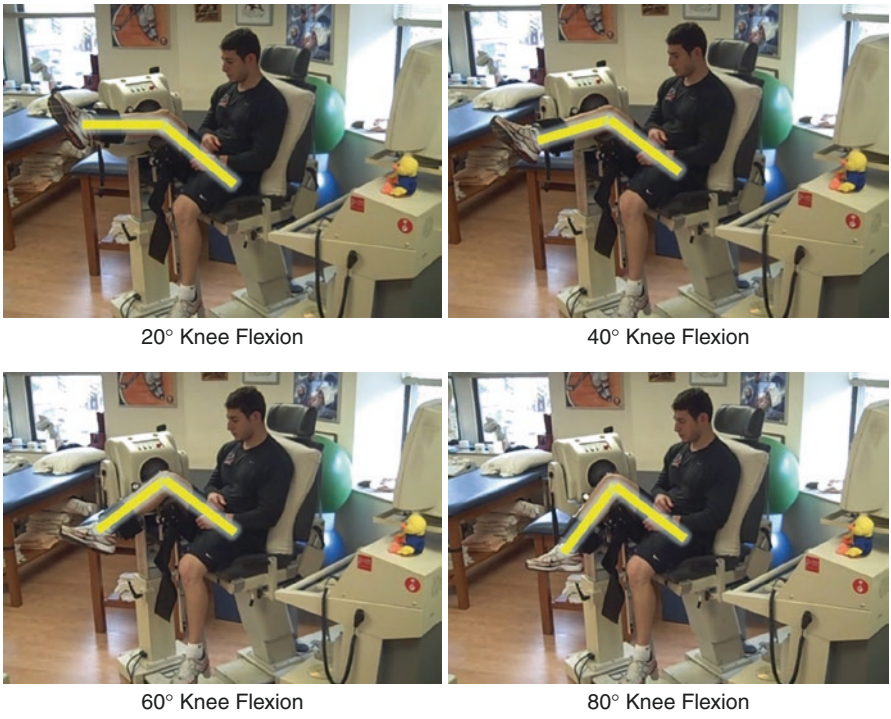

Fig. 9.6 Multi-angle isometric testing position to create length–tension curve. The recorded strength at each angle may be used to create a length tension curve

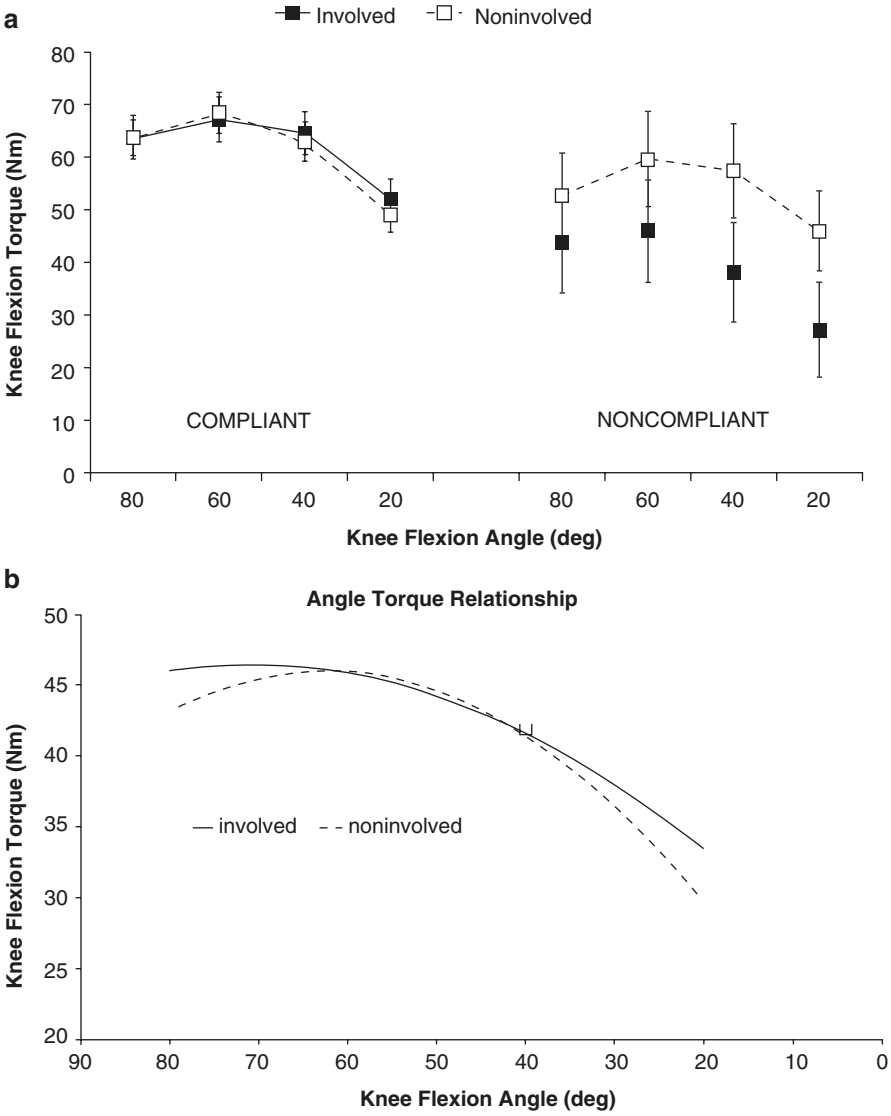

Fig. 9.7 (**a**) Example of muscle length to torque generated across multiple knee flexion angles. This graph compares athletes who underwent lengthened state training vs those athletes who did not complete rehabilitation. (**b**) Example of muscle length to torque generated across multiple knee flexion angles. This is an example of athlete post lengthened state rehabilitation, wherein he successfully gained strength in the lengthened muscle position

provide a measure of hamstring contractile torque production only. Two maximal contractions are performed at each angle (averaging the two), progressing from short to long muscle lengths. The multi-angle lengthened isometric strength-testing protocol was shown to be reliable (the standard error of measurement for repeated

Fig. 9.8 Outer range strength test

strength measures at 20° of knee flexion in the control group was 7 Nm (11.4% of mean absolute torque value) and 7.6% for the test–retest difference in relative torque at 20°; at other angles, standard error of measurement was comparable to, or lower than, the standard error of measurement at 20°) [55]. This test has also been shown to be effective at demonstrating changes in the length–tension relationship due to passive stretching [56, 57]. An alternate outer range strength assessment of eccentric strength has been documented using handheld dynamometry and is reported to have inter-rater reliability ($ICC_{(2,1)}$ of 0.79 (0.62–0.88)), with an MDC% of 20.2 [40]. This test is performed with the player positioned in supine with fixating belt over the pelvis in line with the anterior superior iliac spine. The clinician passively flexes the player's knee on the testing leg to 90° while the contralateral leg remains flat. Standing at the side of the examination table, holding a handheld dynamometer with both arms and vertically positioned against the player's posterior heel, the clinician resists an isometric maximum voluntary contraction against the handheld dynamometer for 3 s, before a break is performed (Fig. 9.8).

9.3.3.2 Concentric and Eccentric Strength Measurement

Isokinetic testing is another common form of strength assessment used to evaluate the hamstrings. Concentric isokinetic testing has been utilised to quantify knee flexion torque in different lower extremity pathologies for the last 40 years [58, 59]. Isokinetic strength testing may be more appropriate to include when evaluating severe, ongoing, or long-standing hamstring injury problems, recurring strains, unresolved signs and symptoms of hamstring injury sequela, and tendon-related pain, since they are typically associated with longer recovery time frames where dynamic hamstring strength restoration is possible.

If isokinetic strength testing is conducted, there are several considerations. Testing may be performed eccentrically or concentrically and at various speeds. Lower concentric test speeds ($60° s^{-1}$) appear most sensitive to identifying strength deficits compared to higher isokinetic test speeds [35]. A small strength reduction of 5.2%, based on the pooled means displayed in the systematic review and meta-analysis by Maniar et al. [35], distinguished injured hamstrings to non-injured on concentric $60° s^{-1}$ isokinetic testing.

When testing eccentrically, research has shown that -30° s^{-1} and -120° s^{-1} speeds have moderate supporting evidence [29, 60–62], as opposed to -230° s^{-1} [61] and -300° s^{-1} [63], and therefore, slower speeds are recommended. Li et al. [64] tested knee flexion strength reliability isokinetically at -60° s^{-1} and -120° s^{-1} which revealed test–retest reliability ranging from 0.82 to 0.91 for peak torque, total work, and average power. When measuring peak torque, the ICC for knee flexion, specifically, eccentric (-60° s^{-1}, 0.83; -120° s^{-1}, 0.83) and concentric (60° s^{-1}, 0.84; 120° s^{-1}, 0.88), were not shown to be significantly different [64].

Eccentric-hamstring-to-concentric-quadriceps torque ratio ($_{Ecc30}H{:}Q_{Con240}$) has demonstrated the largest reductions in the injured compared to the non-injured limb among reported hamstring/quad ratios [35]. A pooled average of such hamstring/quad ratios indicated an 18.7% reduction in the injured limb compared to the non-injured side [35].

9.3.3.3 Nordic Hamstring Strength Measurement

Opar et al. [65] have recently investigated the utility of a strength-testing device based on the NHE. This test has demonstrated moderate to high reliability (ICC, 0.83–0.90; typical error, 21.7–27.5 N; typical error as a coefficient of variation, 5.8–8.5%) with an MDC at a 95% confidence level of 60.1–76.2 N [65]. They have shown that athletes with a previously injured hamstring have a residual strength deficit of 15% compared to the contralateral limb [66]. Low levels of hamstring strength measured with the Nordic hamstring test at the start (below 256 N) and end (below 279 N) of the preseason in elite Australian football players were prospectively shown to increase the risk of hamstring injury by 3.3-fold (p, 0.002) and 2.8-fold (p, 0.027), respectively. Bourne et al. [67] also revealed that a between-limb imbalance of greater than 15%, when tested eccentrically, was associated with more than double the risk of hamstring injury, and asymmetries of greater than 20% more than tripled the risk in Australian rugby union players.

The portability of this device, like the handheld dynamometer, makes it a more practical option to the large and expensive isokinetic testing devices [68, 69] and makes it a good tool for baseline strength testing.

9.3.4 High-Speed Hamstring Flexibility and Apprehension Test (the *H*-Test)

The H-test is a reliable high-speed active hamstring flexibility and apprehension test that has been proposed as a RTS criteria after hamstring injury [70]. It can measure and compare ROM, speed of limb movement, and the athlete's subjective rating of apprehension during completion of the ballistic task between injured and non-injured limbs. Small deficits in ROM (8%) and reduced limb speed (26° s^{-1}) between injured and non-injured legs have been shown [70]. A subjective rating of insecurity or apprehension during the task is also recorded at testing. Ratings around 50 out of a maximum 100 have been demonstrated in the injured compared to zero ratings in non-injured legs [70].

To perform the *H*-test as described in the study protocol, the athlete lays supine with both legs extended, and the injured leg is braced to ensure knee extension throughout. The athlete is instructed to "perform a straight leg raise as fast as possible to the highest point without taking any risk of injury" [70]. The athlete performs three trials per leg without a warm-up. The uninjured leg is measured first. The ROM and degree of insecurity felt are then recorded (Fig. 9.9). In place of an electrogoniometer (if unavailable) as used in the initial study, the authors suggest using a simple tape measure if the examiner desires to quantify the measurement. The authors generally recommend waiting until there are no signs or symptoms remaining to perform this test.

The subjective rating of apprehension or feeling of insecurity during the task is most relevant to practitioners since it does not require specialised equipment (the researchers used an electric goniometer in the study). The inclusion of the *H*-test has been proposed once certain results are achieved: pain-free palpation of the hamstrings; PSLR test, and manual isometric hamstring strength tests in 0°, 45°, and 90° of knee flexion shows no bilateral asymmetry [70]. Despite meeting these criteria, one in two (49%) athletes will report insecurity during the *H*-test after rehabilitation

Fig. 9.9 (**a**, **b**): To perform the *H*-test, the athlete's involved leg is braced, and tape measure is affixed just proximal to the malleoli. The examiner braces the contralateral leg and holds the tape measure against the medial malleolus (**a**). The starting number on the tape measure is noted. The athlete then flexes the hip as fast and as high as possible (**b**). The number on the tape measure at maximal height is recorded. The initial measure on the tape is subtracted from the maximal height to give the examiner a total leg raise height

[71, 72] extending their rehabilitation and RTS (full training and competition) time frames on average by 1–2 weeks until the *H*-test clears (i.e. no ROM deficits or apprehension).

9.3.5 Running and Sprinting Assessments

Running is both a key sport performance component and a factor in injury, reinjury, and subsequent injury [73–77]. Balancing the benefits of early running against the pathobiology of muscle injury [30, 78] and the risk of recurring and subsequent injury [76, 77, 79] is essential. Biomechanical modelling highlights this importance showing an increase in outer range lateral hamstring peak force and eccentric load with increased running speeds [80]. Lateral hamstring peak force increased by 44% when running at maximal speed compared to 80% of maximal speed [80]. At the time point where many athletes commence running in rehabilitation, clinical tests will still demonstrate impairments, and muscle healing is ongoing, indicating that judicious selection of return to running programming and criteria is critical. For example, a gradual accumulation of moderate to high total sprint running distances in lower limb muscle injury rehabilitation has been found to mitigate the risk of subsequent injury in team sport athletes [76, 77]. Interestingly, the risk of subsequent injury was significantly increased if running commenced within 4 days of injury, but suspending running until at least day 5 post-injury did not increase time to RTS [76].

9.3.5.1 Running and Sprinting Loads

In the absence of available load data, recent daily clinical findings associated with acute hamstring injury showed that athletes' self-rated perceived efforts (0–100) during running are useful feedback on which to base running progressions [40]. A 5–10% increase in effort per lap over a 30 m "run" on an indoor track appears to be acceptable when the athlete is confident and has no discomfort running [40]. A strong correlation is seen between perceived running speed (0–100%) and outer range strength expressed as a percentage of the uninjured leg (Fig. 9.10). In situations where the therapist is unable to supervise the staged return to running, this measurement can be used as an approximation of the athlete's likely upper limit for any given day, although it should be noted that there is a reasonably large amount of "noise" in this relationship, so the athlete is best advised to carefully progress/regress depending on their symptoms (e.g. perceived tightness or any exacerbation) rather than slavishly attempting the predicted maximal effort based on their strength testing.

Load monitoring and particularly detailed information of previous and current running exposure is rarely considered in the clinical rehabilitation literature. As mentioned previously, as a part of the subjective assessment, it is recommended that practitioners consider incorporating load and running monitoring principles in the decision-making process and programming of running rehabilitation [76]. External load monitoring (e.g. distance, speed, time, and type) is recommended and can also be complemented by internal load data (e.g. sessional rate of perceived exertion and heart rate monitoring). In cases where external load monitoring is unavailable,

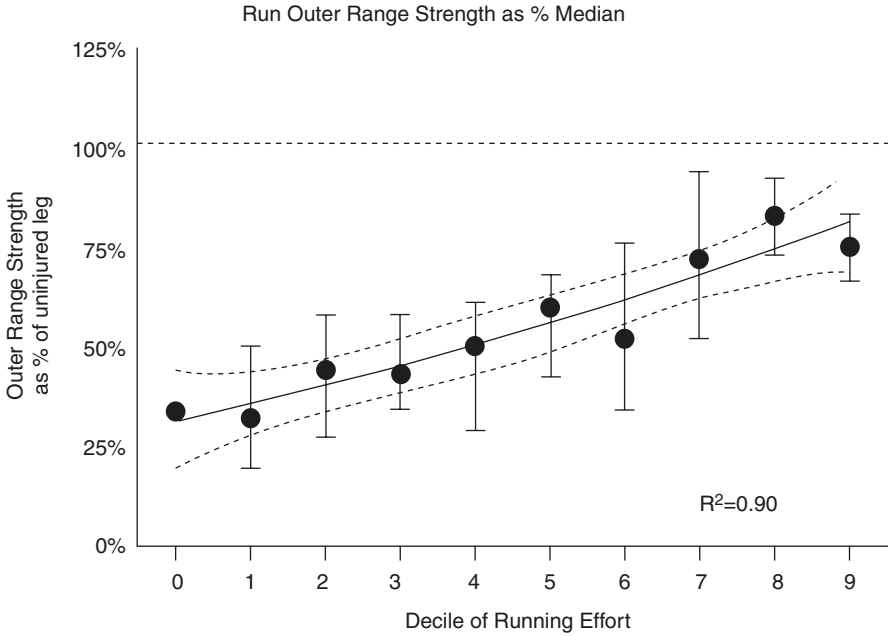

Fig. 9.10 Correlation of outer range strength to running effort which may be used as an approximation of the athlete's likely upper limit for any given day [40]

internal load is an alternative primary option, and the acute-to-chronic workload ratio can be calculated from sessional ratings of perceived exertion to guide rehabilitation requirements [81]. Sport- and position-specific running criteria should be defined, gradually pursued, and completed in rehabilitation. Running criteria can be modelled on pre-injury data from pre- or in-season sport-specific running tests of various sprint distances and intermittent endurance fitness tests in addition to training and competition demands.

9.3.5.2 Speed and Force Production

Speed and force production should be considered during sprinting assessment. Mendiguchia et al. [45] measured athletes' speeds via radar gun at time of RTP and 2 months later and found horizontal force and power output were decreased at time of RTP compared to non-injured controls, but differences between groups were deemed "trivial" by 2 months post RTP test following 2 months of regular training. These results suggest a relationship between previously injured hamstring muscle and strength deficit in hip extension and knee flexion, despite returning to sport. Furthermore, horizontal forces and acceleration in sprinting are related to hamstring rate of force development (RFD) [41]. Given these findings, RFD, horizontal force, and power production should be considered as a part of the assessment, particularly in later phases of rehabilitation after hamstring injury. Recently, a smartphone app called *My Sprint* was demonstrated to have excellent interobserver

reliability (ICC, 0.998; CI, 0.997–0.998) and have near-perfect agreement with a photocell (ICC, 1.00; CI, 1.00–1.00) and radar gun (ICC, 0.987–1.00) setup with regard to measuring horizontal force, max power, and theoretical velocity, making it a practical way for clinicians to assess these values [82]. This app is a practical tool for those clinicians who do not have access to more sophisticated measurement equipment.

9.4 Conclusion

The assessment of hamstring injuries may be done most effectively by following a structured, evidence-based procedure. The clinician develops an initial assessment and differential diagnosis based on the patient's subjective report, which will then guide the clinician in the physical assessment. The information garnered during inspection, palpation, flexibility testing, strength testing, and running assessment allows the clinician to establish a functional diagnosis and develop a plan of rehabilitation. The findings during the assessment may also serve as a point of reference for comparison to allow the clinician to monitor progress throughout the course of rehabilitation and assist the clinician in the decision-making process with regard to prognosis and RTP.

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Rehabilitation of Hamstring Injuries

10

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10.1 Introduction

Following any type of athletic hamstring injury, sports medicine clinicians are constantly under pressure to facilitate a quick and safe return of the athlete to training and competition. To meet these challenging requirements, it is essential to combine a range of training parameters to ensure that the athlete is able to work near the limit of his or her capacity while concurrently ensuring that sufficient time is allowed for the injured tissue to heal. If the athlete is progressed too aggressively during rehabilitation, there may be an increased risk of pain and injury exacerbation, consequently delaying the time to return to sport (RTS). Yet, a too cautious rehabilitation approach may keep the athlete out of training and competition longer than necessary.

Hamstring injuries may vary significantly in type and severity [1]. However, the specific injury characteristics and the presentation of symptoms should guide the clinician to choose the most appropriate rehabilitation approach. Overall, the most common hamstring injury types are acute muscle injuries (including acute strain injuries and recurrences), complete tendon avulsion ruptures, apophyseal avulsion fractures, and proximal tendinopathies. Each type of injury requires specific and targeted rehabilitation interventions which are further discussed in this chapter.

10.2 General Principles

The main goal of a rehabilitation programme after hamstring injury should be to facilitate that the athlete is returning to sport at the highest possible performance level as fast as possible but with a minimal risk of reinjury [2–4]. Clinicians must therefore aim to address acute deficits in hamstring muscle structure and function, as well as mitigate modifiable risk factors that may have contributed to the original injury. Furthermore, the rehabilitation process should be viewed as a window of opportunity to not only reduce reinjury risk but also optimise performance. Thus, not only the specific tissue damage following injury but also the performance consequences of the injury should be considered. A safe and effective rehabilitation plan should always strive for low risk but prepare the athlete for high demand [4], and an important element of the rehabilitation plan is effective goal setting [5, 6]. The goals of rehabilitation should be established from the outset through shared decision-making, involving the patient, the coach (if applicable), and the practitioner.

10.2.1 Rehabilitation Through Phases

The content and structure of hamstring injury rehabilitation programmes are typically divided into phases including specific rehabilitation goals, as shown in Fig. 10.1. Within each phase, restoring specific key elements (such as muscle strength components and flexibility/range of motion (ROM)) is emphasised, with progression through the phases based on either linear predetermined time frames [7–12] or clinical and functional criteria-based progression [2–4, 13–17]. Although the use of defined phases may be a good way of structuring the rehabilitation, it should be kept in mind that there are no distinct borders between these phases. Thus, rehabilitation can be considered as a process where the phases will often be overlapping.

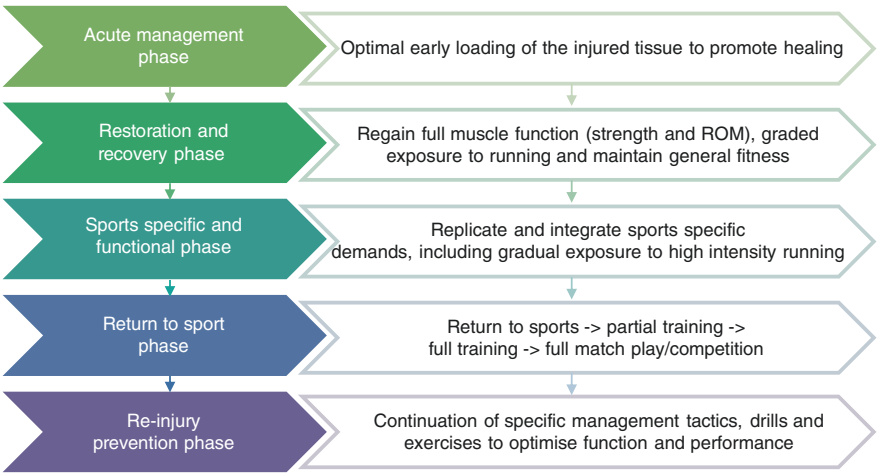


Fig. 10.1 General rehabilitation phases and rehabilitation goals after hamstring injury

10.2.2 Rehabilitation is Part of the Return to Sport Process

The RTS process is considered as a continuum paralleled with recovery and rehabilitation—not simply a decision taken in isolation at the end of the recovery and rehabilitation process [18]. A multifactorial approach including a comprehensive evaluation of health status, participation risk, and factors involved in the decision modification is suggested to provide clinicians with an evidence-based rationale for RTS decision-making [19, 20]. Importantly, these factors should be considered along the course of the rehabilitation RTS continuum [18] after hamstring injuries.

In the following sections, the evidence for the efficacy of hamstring rehabilitation programmes is investigated. How to optimise monitoring of hamstring muscle structure and function during the rehabilitation phases is further discussed and elaborated.

10.3 Rehabilitation of Acute Hamstring Muscle Injuries

The high incidence of hamstring reinjuries remains enigmatic, and previous injury is reported as the most common risk factor for a subsequent injury [21, 22]. It is currently debated whether this high recurrence rate may be due to intrinsic factors that were present prior to the index injury and left unaddressed by rehabilitation [2, 4, 14, 23, 24]. At the time of RTS, residual magnetic resonance imaging (MRI) findings are commonly present [25–27], indicating that, although athletes have met clinical clearance and have returned to their sports activity, the healing of the injured muscle may still be ongoing, incomplete, and/or inadequate. This may leave the athlete in a vulnerable state for reinjury after RTS. Reinjuries commonly occur early (within the first 2 months) after RTS [28–30], but an increased susceptibility seems to be present for several months after the index injury [23, 31].

Therefore, an effective rehabilitation process promoting muscle tissue repair and recovery of function after a hamstring injury is important not only for a quick RTS but also for minimising the risk of reinjuries.

10.3.1 Acute Management and Early Loading

There is little evidence for the early management of acute muscle (strain) injuries [32]. However, the protection, rest, ice, compression, elevation (PRICE) principle is traditionally considered the cornerstone for treating acute soft tissue injuries [8, 33]. Protection, optimal loading, ice, compression, elevation (POLICE) is suggested as an alternative acronym, where optimal loading means replacing rest with balanced and incremental loading which encourages early recovery [34].

During the early rehabilitation phases, resolving the clinical signs and symptoms and restoring function should be emphasised. Key goals throughout the acute management phase are to trying to limit the size of the hematoma and scar tissue formation and facilitating a rapid and intensive re-capillarisation (vascular ingrowth) and neuronal resprouting. In this context, early and adequate loading could therefore be viewed as a way of enhancing stimulus for regeneration and minimising muscle atrophy [8, 10]. There is, however, no consensus as to how fast or aggressive the initial mobilisation and loading should be. It has recently been shown that starting rehabilitation early (2 days) after muscle injury rather than delaying rehabilitation (waiting for 9 days) significantly shortens the interval from injury to pain-free recovery and RTS by 3 weeks without any significant increase in the risk of reinjury [7], thus supporting the importance of early loading of injured musculotendinous tissue. We therefore suggest that progressive loading is commenced according to the athlete's tolerance, and that simple daily activities, and regular active movements through functional ranges of motion be commenced as early as possible [33].

10.3.2 Optimal Load Progression

An optimal load progression through the rehabilitation process is desirable to allow for adequate healing of the musculotendinous tissues and to prevent injury recurrences. Rehabilitation should therefore include fundamental therapeutic exercises, commonly referred to as mechanotherapy [37]. Optimal loading may be defined as "the load applied to structures that maximises physiological adaptation and restores function" via various cellular and neural mechanisms [33]. Manipulation of loading variables can have profound effects on the nature, structure, and function of the wider neuromusculoskeletal system. Theoretically, it is suggested that, as tissues adapt to changes in their mechanical properties, the sensory information provided during movement will also change prompting the central nervous system (CNS) to adapt to these changes [33].

10.3.3 Summary of Evidence

There is yet limited consensus regarding the effectiveness of various rehabilitation protocols (RPs) for acute hamstring injuries in different sports [14, 35, 38, 39]. However, the evidence is emerging. Pas et al. [39] reported the efficacy of conservative treatments for hamstring injury in an updated systematic review published in 2015 (Level 1a). A further updated summary of randomised controlled trials (RCTs) evaluating specifically rehabilitation and exercise interventions is presented in Table 10.1, based on previous search strategies [38, 39], and is discussed further in the following section.

10.3.3.1 Early Time-Based Rehabilitation

Recently, Bayer et al. [7] reported that an early commencement of rehabilitation (2 days after injury) resulted in a significantly hastened recovery compared to a delayed commencement (9 days after injury) (Level 1b).These athletes followed a standardised four-stage therapy regimen with time-based progression, including daily repeated static stretching (week 1), daily isometric loading with increasing load (weeks 2–4), dynamic loading with increasing resistance three times per week (weeks 5–8), and implementation of functional drills, such as sprints and jump exercises, combined with heavy strength training three times per week (weeks 9–12). This study underpins the importance of early mobilisation and loading and that a time-based progression may also be considered during hamstring rehabilitation. However, the time to RTS was generally long in both groups (62.5 days vs. 83.0 days), which may be related to the injury type (the study also included calf injuries and one Quadriceps injury), age, activity, and performance levels of the included participants (see Table 10.1).

10.3.3.2 Progressive Agility and Trunk Stability (PATS) Exercises

One of the first RCTs on acute hamstring injuries was published in 2004 by Sherry and Best [42], comparing a programme including progressive agility and trunk stabilisation (PATS) exercises and icing to a protocol consisting of static stretching (STST), isolated progressive hamstring resistance exercise, and icing. The authors reported fewer recurrences after rehabilitation in the group implementing the PATS protocol compared to STST exercises, and therefore, neuromuscular control exercises gained popularity in hamstring injury rehabilitation following this study. However, a follow-up study by Silder et al. [26] did not reveal any differences between the PATS programme and a progressive running and eccentric strengthening rehabilitation programme, with both programmes yielding similar results with respect to hamstring muscle recovery and function at the time of RTS (25.2 days vs. 28.8 days) (Level 1b). This study is further discussed in Sect. 10.3.3.10.

10.3.3.3 Hamstring Lengthening Exercises

In the last decade, lengthening exercises introduced by Askling et al. have gained increased attention. In two RCTs among Swedish elite football players [35] (Level 1b) and Swedish elite sprinters and jumpers [36] (Level 1b), a protocol (the

Table 10.1 Overview of RCTs (Level 1b) investigating the effect of exercise and/or rehabilitation interventions following acute hamstring injuries

Reference	*N*	Population	Intervention A/intervention B	Follow-up	Primary outcome	Results	Effect
Cibulka et al. [40]	20	Patients with a clinical diagnosis of hamstring injury and sacroiliac joint dysfunction	A: SI manipulation + moist heat and passive stretching B: Moist heat and passive stretching	None reported	Hamstring peak torque (ft lbs)	A: 46.4 ft lbs (SD 17.47) B: 45.7 ft lbs (SD 22.70)	=
					Passive knee extension (ROM)	A: 155.0° (SD 14.2) B: 144.6° (SD 16.7)	
Malliaropoulos et al. [41]	80	Athletes with an ultrasonographic grade 2 hamstring injury	A: "Intensive" stretching B: "Normal" stretching	Until RTS	Time to RTS	A: 13.27 days (SD 0.71) B: 15.05 days (SD 0.81)	+
					Time needed for equalisation of active knee extension	A: 5.57 days (SD 3.3) B: 7.23 days (SD 0.53)	
Sherry and Best [42]	24	Athletes with acute hamstring injury, grades 1 and 2 based on PE	A: Core stability + agility + icing (PATS) B: Static stretching + progressive resistance exercises + icing (STST)	1 year	Time to RTS	A: 22.2 days (SD 8.3) B: 37.4 days (SD 27.6)	+
					Reinjury	A: 0/13 B: 7/10	
Silder et al. [26]	29	Athletes with suspected hamstring injury (≤past 10 days) confirmed by PE and MRI	A: Core stability + agility (PATS) B: Running + eccentric (PRES)	1 year	Time to RTS	A: 25.2 days (SD 6.3) B: 28.8 days (SD 11.4)	=
					Craniocaudal length of injury	A: 7.9 cm (95% CI 2.7–13.1) B: 15.9 cm (95% CI 8.4–23.4)	

Askling et al. [35]	75	Elite Swedish football players with MRI (<5 days after injury) confirmed hamstring injury	A: L-protocol[a] B: C-protocol[b]	1 year	Time to RTS	A: 28 days (SD 15) B: 51 days (SD 21)	+
					Reinjury	A: 0/37 B: 1/38	
Askling et al. [36]	56	Swedish elite sprinters and jumpers with MRI (<5 days after injury) confirmed hamstring injury	A: L-protocol[a] B: C-protocol[b]	1 year	Time to RTS	A: 49 days (SD 26) B: 86 days (SD 34)	+
					Reinjury	A: 0/28 B: 2/28	
Mendiguchia et al. [14]	48	Male football players with grade I structural hamstring injury (<4 days after injury) confirmed with US and PE	A: Individualised and multifactorial criteria-based algorithm (RA) B: General rehabilitation protocol (RP)	6 months	Time to RTS	A: 25.5 days (SD 7.8) B: 23.3 days (SD 11.7)	=
					Reinjury	A: 1 (4%) B: 6 (25%)	
Bayer et al. [7]	23/42[c]	Athletes from various sports (males and females) (<48 h) confirmed with US and CE	A: Early time-based rehabilitation (2 days after injury) (n = 11 hamstring injury) B: Late time-based rehabilitation (9 days after injury) (n = 12 hamstring injury)	12 months	Time to RTS	A: 62.5 days (IQR 48.8–77.8) B: 83.0 days (IQR 64.5–97.3)	+
					Reinjury	A: 1/20 B: 0/22	

(continued)

Table 10.1 (continued)

Reference	*N*	Population	Intervention A/intervention B	Follow-up	Primary outcome	Results	Effect
Hickey et al. [43]	43	Recreational to sub-elite male athletes from various running-based sports with hamstring injury confirmed by PE ≤ 7 days of injury	A: Hamstring strength exercises and progressive running performed within limits of pain rated ≤4/10 (pain threshold) B: Hamstring strength exercises and progressive running performed within limits of pain rated <1/10 (pain-free)	6 months	Time to meet RTS clearance criteria	A: 17 days (95% CI 11–24) B: 15 days (95% CI 13–17)	=

C control, *CE* clinical examination, *HSI* hamstring strain injury, *I* intervention, *IQR* interquartile range, *MRI* magnetic resonance imaging, *PATS* progressive agility and trunk stabilisation, *PRES* progressive running and eccentric strengthening, *PE* physical examination, *ROM* range of motion, *RTS* return to sport, *SI* sacroiliac, *STST* stretching and strengthening, *US* ultrasonography, *Ft lbs*, foot-pound (torque)

[a]L-protocol—aimed at loading the hamstrings during extensive lengthening exercises

[b]C-protocol—conventional hamstring exercises with less emphasis on lengthening

[c]Number of hamstring injuries included out of the total number of muscle injuries

L-protocol) aimed at loading the hamstring muscles during lengthening exercises was reported to be more effective in promoting time to RTS (i.e. time from injury to full participation in the team training) after acute hamstring injury compared with a conventional protocol (C-protocol), which included exercises with less emphasis on lengthening. The time from the date of injury (total ruptures were excluded) to the date of RP initiation in these two studies was 5 days for both protocols, and no pain provocation and/or analgesic treatments were allowed at any time during the rehabilitation process. Each RP consisted of three different exercises, all performed in the sagittal plane. In both protocols, exercise 1 was aimed mainly at increasing flexibility, exercise 2 was a combined exercise for strength and lumbopelvic control, and exercise 3 was a specific strength training exercise (see images and descriptions of the L-protocol and the C-protocol in Figs. 10.2 and 10.3). Intensity and volume of training were matched as closely as possible between the two protocols. The speed and load of exercises in both protocols were increased over time as tolerated by the athletes, within pain-free limits.

In addition to their specific protocol, all athletes completed a general rehabilitation programme with an athlete-specific progression. The general programme was performed three times a week and started with stationary cycling 10 min, 10 × 20 s fast foot stepping in place, 10× jogging 40 m with short strides, and 10 × 10 m forward/backward accelerations. When the above part of the general programme could be performed without pain and/or discomfort, a progressive running programme was initiated, including high-speed running drills performed three times a week (see Box 10.2). In addition, all athletes were asked to conduct as much as possible their standard training programme without experiencing any pain and/or discomfort. For football players, time to RTS was significantly shorter in the L-protocol compared with the C-protocol (see Table 10.1), regardless of whether the injuries were of the sprinting or stretching type. The average time to RTS for the sprinting type of injuries within the L-protocol was 23 days (SD ± 11; range, 8–44 days). Similar findings of the effect of the L-protocol were found among the elite sprinters and jumpers, using the same method as in the study of football players (Table 10.1) [36]. It should be noted that the inclusion of an extra criterion test, the Askling *H*-test [44] during ballistic flexibility movement (see description in Chap. 9), increased the days to RTS in both studies. On average, the rehabilitation period was prolonged by 7 days (1SD ± 2.7; range, 3–14 days) for the football players and by 10 days (1SD ± 3.4; range, 3–20 days) for the sprinters and jumpers due to the execution of the Askling *H*-test. Based on these studies, it seems reasonable to emphasise lengthening exercises as described in the L-protocol (Fig. 10.2) over shorter-range concentric-eccentric exercise for hip extensors and knee flexors early in the rehabilitation (initiated already in the restoration and recovery phase). The optimal volume and intensity of eccentric training-based exercises through the entire rehabilitation period after acute hamstring injuries and reinjuries are, however, not yet clear based on Askling's work.

Fig. 10.2 The L-protocol described by Askling et al. [35, 36]. The "extender," aimed at increasing flexibility, is performed with the athlete holding and stabilising the thigh of the injured leg with the hip flexed approximately 90° and then performing a slow knee extension to a point just before pain is felt (2× per day, 3 sets × 12 repetitions). The "diver" is performed as a simulated dive (i.e. as a hip flexion from an upright trunk position) of the injured, standing leg and simultaneous stretching of the arms forward and attempting maximal hip extension of the lifted leg while keeping the pelvis horizontal; angles at the knee should be maintained at 10–20° in the standing leg and at 90° in the lifted leg. Owing to its complexity, this exercise should be performed very slowly in the beginning (once every other day, 3 sets × 6 repetitions). The "glider" is started from a position with an upright trunk, one hand holding on to a support and legs slightly split. All the body weight should be on the heel of the injured (here right) leg with approximately 10–20° flexion in the knee. The motion is started by gliding backward on the other leg (using a catslide or a low friction sock) and stopped before pain is reached. The movement back to the starting position should be performed by the help of both arms, *not* using the injured leg. Progression is achieved by increasing the gliding distance and performing the exercise faster (1× every third day, 3 sets × 4 repetitions)

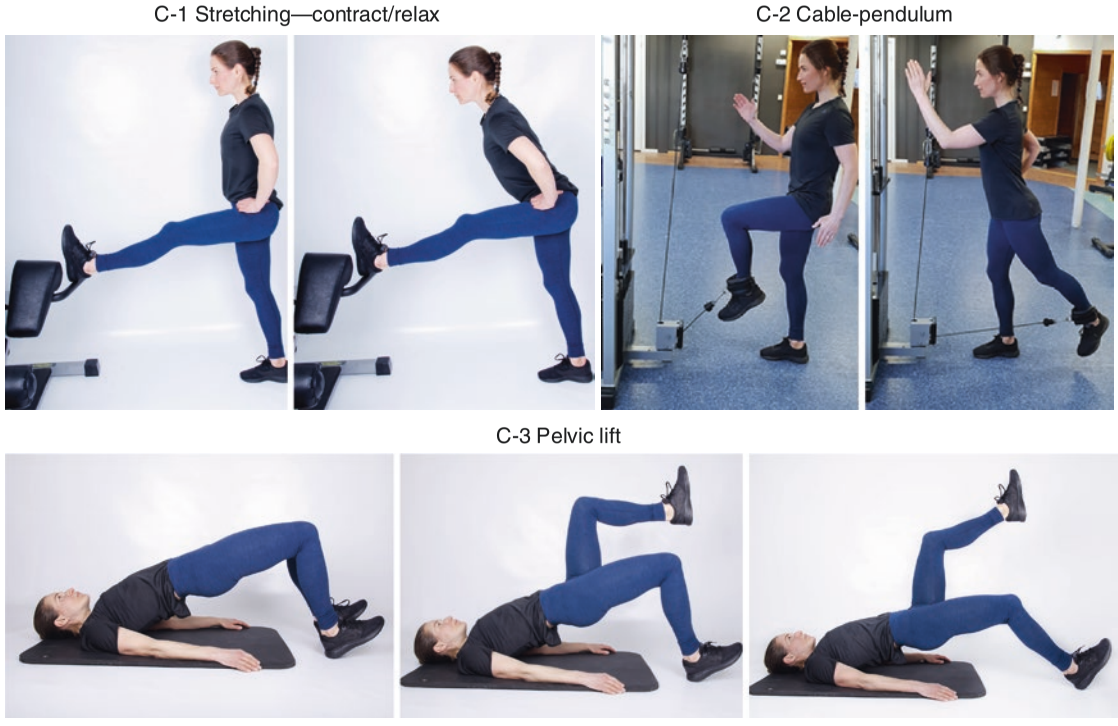


Fig. 10.3 The C-protocol described by Askling et al. [35, 36]. C-1 Contract/relax stretching is performed with the heel of the injured leg placed on a stable support surface in a high position (close to maximum) with the knee in approximately 10° flexion. The heel is pressed down for 10 seconds (s), and then, after relaxation for 10 s, a new position is assumed by flexing the upper body slowly forward for 20 s (2× every day, 3 sets × 4 repetitions). C-2 Cable pendulum is performed with a stationary cable machine or expander. With the uninjured leg as standing leg, forward-backward hip motions are performed with the injured leg with the knee in approximately 20–30° flexion. This exercise involves the whole body and should be performed slowly in the beginning of the rehabilitation period (1× every second day, 3 sets × 6 repetitions). C-3 Pelvic lift is started in a supine position with the body weight on both heels, and then, the pelvis is lifted up and down slowly. Start with the knee in 90° of flexion. The load is increased by putting more of the body weight on the injured leg and by having a greater extension in the knee. Ultimately, only the slightly bent injured leg is carrying the load (every third day, 3 sets × 8 repetitions)

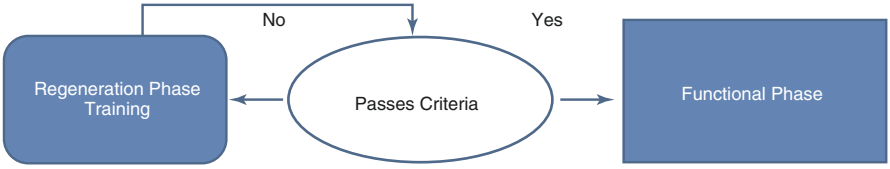
10.3.3.4 Multifactorial Approach

Multifactorial approaches to acute hamstring injury rehabilitation have been recently emphasised [4, 13, 14, 17]. Mendiguchia et al. [14] showed in a RCT (Level 1b) that male football players, who underwent an individualised, multifactorial, criteria-based algorithm (multifactorial programme) with a performance- and primary risk factor-oriented training programme from the early stages of the process, markedly decreased the risk of reinjury compared to a general protocol where the lengthening exercises as described by Askling et al. [35] were prioritised (one reinjury vs. six reinjuries). The time to RTS was longer for the multifactorial programme compared to the general protocol (lengthening exercises) (25.5 days vs. 23.3 days) (Table 10.1). The multifactorial programme (RA) included a restoration and recovery phase (starting from day 5 after injury) and a sport-specific and functional phase with specific criteria for progression into the functional phase (see Fig. 10.4), and the individualised exercise programme included a range of training variables and rehabilitation modalities (manual therapy, flexibility, strengthening of the gluteus muscles, hamstring strength, plyometrics, ankle stabilisers, lumbopelvic control, and running technique) (Box 10.3A). During the restoration and regeneration phase, daily sessions were performed, whereas during the functional phase, a 3-day block training periodisation was implemented to optimise training adaptations and minimise potential negative training interferences. A minimum of three sessions of the 3 days block training was required to allow the player to RTS. Additionally, basic aerobic conditioning commenced when players were able to perform at least three sessions of running technique without any discomfort or pain in the regeneration phase. One running session was performed every third day and included four sets of 5 min at a low to moderate intensity (player rated). In the functional phase, the running session consisted of two sets of 10 min performed at moderate to high intensity (player rated) (see Box 10.3B).

10.3.3.5 Criteria-Based Rehabilitation and Running Progression

Criteria-based rehabilitation has also been included in a single study centre RCT investigating the effect of platelet-rich plasma (PRP) injections [45] (Level 1b) and in a follow-up study including 131 athletes [17] (Level 2b). In these studies, the athletes had to complete an intensive three-stage physiotherapy rehabilitation programme, including a progressive running programme (see Box 10.4), to continue to three consecutive stages of sport-specific training sessions of increasing difficulty before being declared fit to RTS. The athletes were monitored through daily clinical examinations and progressed into the next stages based on predetermined criteria based on clinical and functional tests. The RCT investigating the effect of the PRP showed no benefit of a single PRP injection over the intensive rehabilitation programme in professional athletes who sustained acute, MRI-positive hamstring injuries [45].

a	Regeneration Phase	
Variable	Test	Criteria for Progression
Pain after injury	Prone with knee flexed to 15°	No pain
Isolated strength at long muscle lengths	Prone with knee flexed to 15°	<10% asymmetry
Neural deficiencies	Slump test	No pain
Hamstring flexibility	Active knee extension (AKE) test	<10% asymmetry
Hip flexor flexibility	Modified Thomas test (MTT)	+5 symmetry below horizontal



b	Functional phase	
Variable	Test	Criteria for Progression
Pain	Palpation	No pain
Peak torque (H/H) and conventional ratio (H/Q)	Isokinetic knee flexion/extension at $60°{\cdot}s^{-1}$	<10% H/H and H/Q>0.45 (Biodex) or >0.47 (Cybex)
Hip extension strength	Prone hip extension	< 10% asymmetry between legs
Distance	Triple hop test	<10% asymmetry between legs
Endurance (Repetition number)	Single leg bridge test	> 25 and < 10% asymmetry between legs
Torsion capabilities	ASLR test	No compensations
Insecurity and Pain	Askling H-test	No pain and insecurity

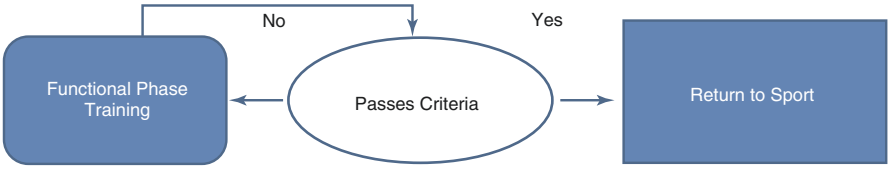


Fig. 10.4 Criteria used to progress a football player through each phase of a multifactorial criteria-based rehabilitation programme Functional (RA) by Mendiguchia et al. [14]. (**a**) Regeneration phase criteria. (**b**) Functional phase criteria. Reproduced with permission

10.3.3.6 Passive Treatment Modalities and Static Stretching

Passive treatments, such as mobilisation of the sacroiliac joint and/or static stretching, are commonly integrated by practitioners as part of the rehabilitation programme [2, 3]. Two RCTs have investigated these passive treatment modalities separately [40, 41]. Cibulka et al. [40] did not reveal any significant effect of sacroiliac manipulation and static stretching, compared with static stretching alone in patients with a clinical diagnosis of hamstring injury and sacroiliac joint dysfunction [40] (Level 1b). Malliaropoulos et al. [41] showed that "intensive" static stretching (30 s × 4, 4 times daily) was more effective than "normal stretching" (30 s × 4, 1 time daily) (Level 1b), although the actual reduction in rehabilitation time is largely clinically irrelevant (1.7 days difference in time required for full rehabilitation) (see Table 10.1).

10.3.3.7 Progression of Hamstring Strength Exercises During Rehabilitation

Due to the multi-joint nature of the long hamstring muscles, it is suggested that that both hip- and knee-based strength exercises should be incorporated throughout rehabilitation, with an emphasis on eccentric contraction modes. Exercises performed at a long hamstring muscle length emphasising eccentric contraction modes, such as Askling's "diver" and "glider," should be introduced as early as tolerated during rehabilitation [35, 36] (see Fig. 10.2). These exercises can then be progressed so that external load can be added such as the Romanian dead lift (RDL) or 45° hip extension [46] (see Fig. 10.5). Knee-dominant strength exercises should be eccentrically biased to target increases in eccentric knee flexor strength, commencing at submaximal intensities as early as tolerated. For example, the supine eccentric sliding leg curl can be performed bilaterally at a submaximal intensity and then with increased intensity via a unilateral variation and the addition of the Nordic hamstring exercise (NHE) (Fig. 10.6).

The introduction of eccentric strength training during hamstring strain injury (HSI) rehabilitation can be a difficult decision for clinicians, as current guidelines lack a clear evidence base. The benefits of eccentric strength training in eliciting adaptations and reducing HSI risk are well-established; however, introduction during rehabilitation is often delayed due to fear of reinjury [10]. Earlier conventional guidelines recommend that rehabilitation should commence with isometric exercise, progressing to isotonic exercises at short to moderate muscle lengths, with long-length and eccentrically biased exercises typically introduced during the final stages of rehabilitation [2, 3, 10, 16]. Furthermore, progression to eccentric exercises has typically not been recommended until isometric knee flexor strength assessments are performed without pain [3, 4, 14, 16, 47]; however, there is no evidence supporting the need for this criteria.

In contrast to these conventional guidelines, Askling's L-protocol safely implemented eccentric exercises at long muscle lengths from the very start of rehabilitation, which was 5 days following HSI. Following on from Askling's work, Hickey et al. [43] described a RP where eccentric exercises were also introduced from the start of rehabilitation, which was on average 3 days following HSI. In Hickey et al.'s protocol, eccentric

Fig. 10.5 Examples of higher intensity hamstring exercises, the Romanian deadlift and the 45° hip extension. Both exercises can be progressed from bilateral to unilateral and can be performed with heavier loads through progressively greater ranges of motion. More information on exercise selection is available in Chap. 12

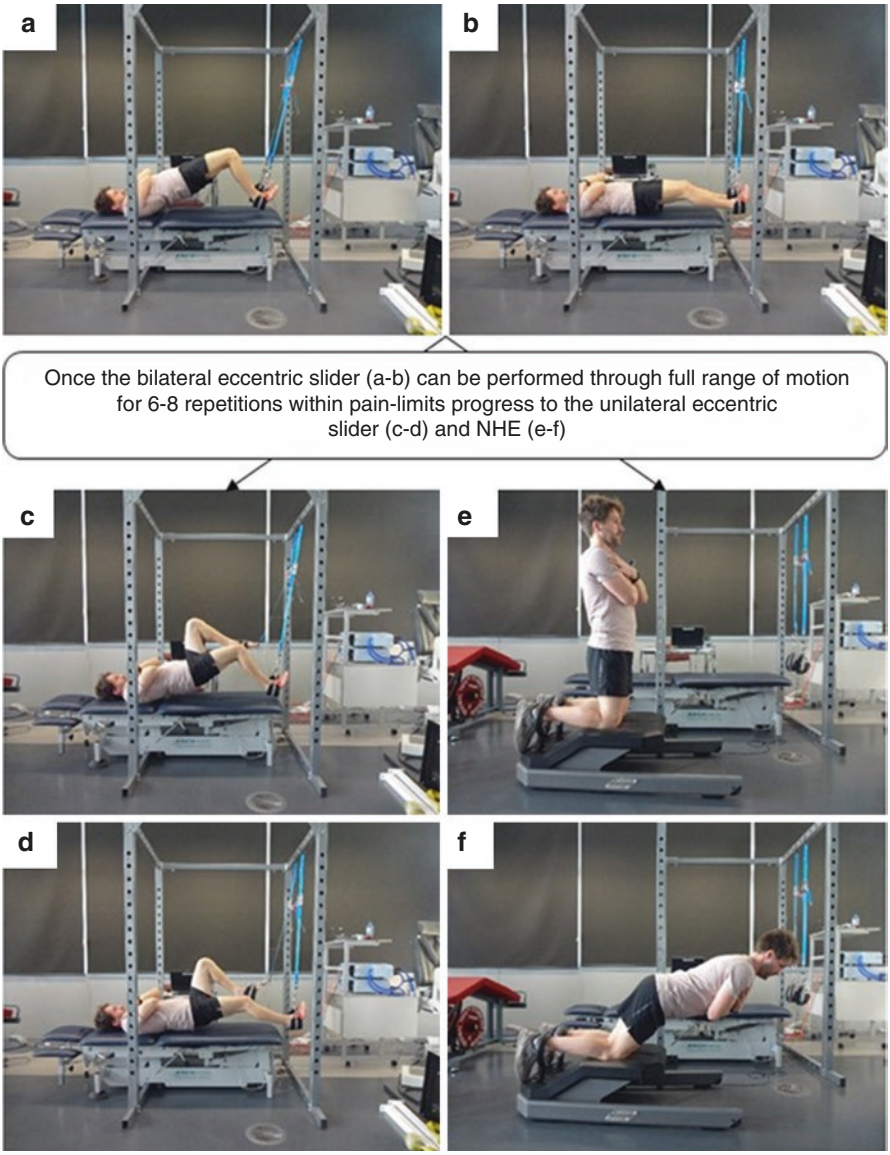


Fig. 10.6 Examples of knee-dominant eccentric-biased hamstring strengthening exercises

loading commenced with a bilateral eccentric sliding leg curl, and once 6–8 repetitions could be performed through full range of motion within acceptable pain limits (≤4 out of 10), it was progressed to the NHE. Eccentric exercise was progressed in this way as the bilateral eccentric slider replicates the joint action and contraction mode of the NHE.

Both Askling et al. and Hickey et al.'s protocols introduced and progressed eccentric loading based on an exercise-specific approach rather than waiting for isometric knee flexor strength assessments to be pain-free. Further to this, Hickey et al. found that eccentric loading such as the NHE could be safely tolerated in the early stages of HSI rehabilitation

despite concurrent pain and between-leg deficits in isometric knee flexor strength [43]. Therefore, it appears that the exercise-specific approach allows for a more individualised progression of rehabilitation and that the conventional guideline of delaying eccentric loading based on isometric knee flexor strength assessments must be questioned.

From a practical perspective, it is important to explain the benefits of eccentric exercise to the injured individual and encourage a gradual buildup of intensity to reduce fear of reinjury. Cueing the injured individual to focus on the eccentric phase of these exercises and encouraging slow and controlled repetitions are recommended. When progressing to movements such as the NHE, encourage the injured athlete to attempt some submaximal repetitions to ensure they feel comfortable with exercise technique and then build up to maximal intensity. Prescription of lower repetition ranges of 4–6 is recommended for eccentrically biased exercises as this encourages maximal effort and has been shown to elicit beneficial adaptation [48].

10.3.3.8 Monitoring ROM/Flexibility and Stretching Interventions During Rehabilitation

In addition to strength deficits, the acute stages following hamstring injury often involve a loss of ROM or flexibility [17, 49–51]. Deficits in ROM are commonly monitored via the passive straight leg raise (PSLR) [50, 52] and/or active knee extension (AKE) [53, 54] tests, which tend to recover quicker than strength following HSI [17, 49, 50]. However, if deficits in AKE ROM persist around the time of RTS, risk of reinjury may increase [31]. It has recently been recommended to measure AKE ROM from a position of maximal hip flexion (MHFAKE), as between-leg deficits on this test provide a better prognostic indication of recovery time than the PSLR following HSI [17]. Furthermore, it has been shown that, even when no signs of injury are present during the PSLR test, between-leg deficits and pain are often still present during performance of a ballistic hip flexion ROM assessment, Askling's *H*-test [44]. Studies implementing the *H*-test have been associated with low rates of reinjury but also relatively lengthy RTS times [35, 36, 47]. We recommend monitoring recovery of ROM/flexibility via the AKE or MHFAKE throughout rehabilitation and implementation of the H-test as part of the RTS criteria, especially if reducing risk of reinjury is a major priority.

Restoration of flexibility may not necessarily require direct intervention following hamstring injury, and the need to include interventions such as stretching as part of rehabilitation is not clear based on current evidence. One Level 1b study has reported that performing static hamstring stretching in a position of hip flexion and knee extension more frequently throughout rehabilitation may slightly accelerate both recovery of AKE range of motion and rehabilitation duration by approximately 2 days [41] (see Table 10.1). However, this study only compared frequencies of the same stretching exercise, rather than comparing the effectiveness of stretching to another intervention. Long-length eccentric and active range of motion exercises have been shown to expedite alleviation of pain and apprehension during the *H*-test and in turn accelerate RTS time when compared to static stretching and short-length exercises [35, 36]. It is therefore recommended to emphasise active range of motion exercises performed at a long hamstring muscle length to enhance recovery of dynamic flexibility following hamstring injury, especially in sports and situations where the athlete experiences restrictions and apprehension related to such movements and activities.

10.3.3.9 Running Progressions

Progressive exposure to running is an imperative part of the rehabilitation process as the majority of hamstring injuries occur in sports involving high-speed running such as soccer, Australian rules football, track and field, and rugby [28, 55–59]. Large and/or rapid deviations from normal exposures to running have also been associated with hamstring injury risk [60, 61]. Running progressions are integrated in many of the recent RPs investigated in Level 1 studies (Table 10.1) and are likely important for superior results. Throughout hamstring injury rehabilitation, progressive running should aim to restore the injured individual's ability to accelerate, maintain constant speed, and decelerate. Progression of running intensity can be objectively measured using timing gates or stopwatch and/or subjectively monitored via a self-rated percentage of perceived maximal effort sprinting. Global positioning system data can also be used to monitor intensity as well as quantify exposure to high-speed running volume and distances appropriate for the injured individual's chosen sport prior to RTS. Additional techniques have recently been described to quantify individual power-force-velocity profiles during acceleration using measurements of speed and distance via radar gun [62, 63]. Such monitoring techniques have been proposed to allow clinicians to individualise progressive running and exercises throughout hamstring injury rehabilitation to enhance sprinting performance [62, 64, 65]. For the purposes of the following section, we will focus on the basic principles of running progression, as not all clinicians will have access to equipment or expertise required for the analysis of some of the aforementioned monitoring methodologies.

It is recommended to commence progressive running as early as possible during rehabilitation, ideally as soon as the injured athlete can walk normally with minimal pain [26]. In the early stages of rehabilitation, caution should be taken to avoid sudden changes in speed, with more gradual increases and decreases of running intensity over acceleration and deceleration distances of greater length [26]. Intensity of progressive running should commence by transitioning from a walking acceleration phase to a slow jog and then decelerating back to a walk, with the distance of the acceleration and deceleration phases decreasing as tolerated [26]. The next stage of progression should involve accelerating from a jog to a run at an intensity of approximately 70% of the injured individual's maximum speed, with a gradual reduction in acceleration and deceleration distances [13, 17, 26]. Progression through the early stages of running rehabilitation is often quicker than expected but then slows when attempting to reach speeds above 70% of maximum. As a result, the final stages of progressive running should involve buildup from 70% to 90% speed in approximately 10% increments and a more gradual increase in intensity from 90% to 100% speed in 5% increments [13]. The ability to sprint at 100% effort without any pain or apprehension is typically recommended prior to allowing RTS [47]. However, it is also recommended to ensure that exposure to high-speed running and sprinting is of a similar total volume and/or distance to the chosen sport/activity of the injured individual to not only ensure readiness for RTS but also to enhance performance. Examples of running progressions suggested in the literature are shown in Boxes 10.1–10.4.

Box 10.1 Progressive Running Schedule by Silder et al. [26]

Progressive running schedule included in Level 1b study (both intervention groups; see Table 10.1 for details) among individuals with suspected hamstring injury (≤10 days) involved in sports that require high-speed running (e.g. football) minimum 3 days per week [26]. Table is reproduced with permission.

Progressive running schedule [26]

Exercises

- Five minutes of gentle stretching before and after each session, 3 × 20 s each
  - Standing calf stretch
  - Standing quadriceps stretch
  - Half-kneeling hip flexor stretch
  - Groin or adductor stretch
  - Standing hamstring stretch
- Repeat each level three times, progressing to the next level when pain-free
- Maximum of three levels per session
- On the following session, start at the second-highest level completed
- Ice after each session, 20 min

	Acceleration distance, m	**Constant speed (maximum, *75%* speed) distance, m**	**Deceleration distance, m**
Level 1	40	20	40
Level 2	35	20	35
Level 3	25	20	25
Level 4	20	20	20
Level 5	15	20	15
Level 6	10	20	10
	Acceleration distance, m	**Constant speed (maximum, *90%* speed) distance, m**	**Deceleration distance, m**
Level 7	40	20	40
Level 8	35	20	35
Level 9	25	20	25
Level 10	20	20	20
Level 11	15	20	15
Level 12	10	20	10

Box 10.2 General Programme Including Progressive Running Programme by Askling et al. [35, 36]

"General programme including progressive running programme" included in two Level 1b studies by Askling et al. [35, 36] (both intervention groups; see Table 10.1 for details) among Swedish elite football players [35] and elite sprinters and jumpers [36] with acute hamstring injury

General programme including progressive running programme [35, 36]	
First part	• Performed three times a week, starting with the following: – Stationary cycling 10 min – 10 × 20 s fast foot stepping in place – 10 × jogging 40 m with short strides – 10 × 10 forward/backward accelerations
Progressive running programme	• Performed three times a week, initiated when the "first part" is performed without pain and/or discomfort – High-speed running drills 6 × 20 m – High-speed running drills 4 × 40 m – High-speed running drills 2 × 60 m

Box 10.3A Rehabilitation and RTS Algorithm Programme for Hamstring Injury by Mendiguchia et al. [14]

"Rehabilitation and RTS algorithm programme for hamstring injury" [14] included in one Level 1b study (only in the RA intervention group; see Table 10.1 for details) among semi-professional and professional football player. Table is reproduced with permission. The programme includes running technique (grey row) and basic aerobic conditioning (Box 10.3B)

'REHABILITATION AND RTS ALGORITHM PROGRAMME FOR HAMSTRING INJURY' (Mendiguchia et al., 2017)

	'Regeneration phase'	'Functional phase'
Manual Therapy	Manual therapy: - Plantar fascia, gastrocnemius and hamstring (avoiding injury site) massage - Lumbar Z-joint mobilisation - Sliding Neural Mobilisation (3 x 12 reps) NMES	Manual therapy: - Plantar fascia, gastrocnemius and hamstring (avoiding injury site) massage - Lumbar Z-joint mobilisation
Flexibility	Psoas static flexibility with pelvic retroversion (4 x 15sec) Quadriceps dynamic mobility (2 x 8 reps) Hamstring dynamic mobility with fitball (2 x 8 reps) Hamstring dynamic mobility supine (2 x 8 reps)	Hamstring dynamic mobility + contralateral psoas flexibility (2 x 5 reps) Hamstring wall flexibility (Push/pull) (3 x 3 reps)
Gluteus	**Gluteus Maximus** (Choose an option daily as pain tolerated): **Option A** Prone hip extension (2 x 10 reps x 3 sec) Single leg bridge + contralateral kick (as tolerated) (2 x 5 reps x 3 sec) Double leg bridge (50% BW; 3 x 6 reps x 3 sec) **Option B** Hip thrust (40% BW; 3 x 6 reps x 3 sec) Single leg bridge + contralateral kick (as tolerated) (10% BW; 2 x 4 reps x 3 sec) Single leg hip thrust + contralateral kick (as tolerated) (3 x 6 reps x 3 sec) **Gluteus medius** Clamshell with band (3 x 6 reps x 3 sec) Side lying hip abduction with band (3 x 6 reps x 3 sec)	**Gluteus Maximus** (Choose an option daily as pain tolerated): **Option A** Single leg hip thrust (10% BW; 3 x 4 reps x 3 sec) Double leg hip thrust (60% BW; 3 x 8 reps x 3 sec) Walking sled push (75% BW; 15 m x 2 reps) **Option B** Single-leg foot and shoulder elevated hip hip thrust + contralateral kick (2 x 4 reps x 3 sec) Single leg back extension + perturbations (2 x 4 reps) Single leg hip extension + contralateral hip flexion (2 x 3 changes) **Gluteus medius** Side step running with band (5 m x 5 go and back) Monster running with band (5 m x 5 go and back)
Hamstring strength	Prone isometrics (mid and long length) (2 x 5 reps x 5 sec) Standing long length isometrics (2 x 5 reps x 5 sec) Supine isometrics (tolerated degrees) (2 x 5 reps x 3 sec) Submaximal eccentric manual resistance in prone (intensity as tolerated) (2 x 8 reps)	(4 hamstring strength exercises per session selecting 2 hip dominated and 2 knee dominated) **HIP dominant** Double leg deadlift with 4 kg medicine ball (2 x 8 reps) Lunge (15% BW; 2 x 6 reps) Single leg deadlift with 15 kg + step up (2 x 6 reps) **KNEE dominant** Double leg slide curl (2 x 6 reps) Nordic hamstring (2 x 4 reps) Sprinter eccentric leg curl (2 x 6 reps) Double leg hurdle hop with trunk extension (2 x 4 reps) Double broad jump with 5 kg (2 x 4 reps) 2 consecutive explosive scissor jumps (3 times)

Plyometrics		Single leg horizontal jump (2 x 3 reps) Double leg hurdle hop with trunk flexion (2 x 4 reps) Double broad jump with 5 kg (2 x 4 reps) 2 consecutive explosive scissor jumps (3 times) Single leg horizontal jump (2 x 3 reps)
Ankle stabilisers	Double leg hamstring/gastrocnemius dissociation drill (3 x 6 reps) Single leg hamstring/gastrocnemius dissociation drill (2 x 6 reps) Step bounding side to side (25% BW; 2 x 10 reps)	Ankle drill 1 (20% BW; 10 m x 4 reps) Ankle drill 2 (20% BW; 10 m x 4 reps)
Lumbopelvic control	Side bridge feet in bench + perturbation (2 x 5 reps x 5 sec) Birdog (2 x 5 reps x 5 sec) Long lever posterior pelvic plank (2 x 4 reps x 5 sec) Leg scissors arms on the floor (2 x 5 reps x 5 sec)	Stir the pot with fitball (3 x 2 reps) Leg scissors arms on the chest (2 x 5 reps x 5 sec) Single-leg stand rotating reaches 4 kg (2 x 6 reps) TRX helicopter (2 x 4 reps) Sprinter push/pull with pulleys (2 x 6 reps)
Running technique	**Frontal plane** running drills (10 m x 5 reps) - Low-to-moderate-intensity sidestepping - Low-to-moderate-intensity grapevine stepping - Low-to-moderate-intensity steps forward and backward over a tape line while moving sideways **Sagittal plane** running drills (vertical emphasised execution specially first days of painful subjects) - Running 5 m + 5 m deceleration (4 reps) - Running 10 m + 5 m deceleration (3 reps) - Running 15 m + 5 m deceleration (3 reps)	**Warm up** Hamstring ballistic stretching (2 x 6 reps) Static "B" drill with resisted bands (2 x 5 reps) Hurdle drills (4 variations) (1 set walking lower intensity,1 set bounding higher intensity) - Hurdle drill 1 (2 reps) - Hurdle drill 2 (2 reps) - Hurdle drill 3 (2 reps) - Hurdle drill 4 (2 reps) Running exercise drills (statics in place, dynamics over 8 m) - Military march (15 m x 2 reps) - Lunge + deadlift (4 reps for each leg) - Lunge + "B" drill (4 reps for each leg) - From skipping to running (20 m x 4 reps) - Sprint bounding (15 m x 3 reps) - Running with hurdle jumps (15 m x 1 rep) - Sprinting 5 m (3 reps), 10 m (3 reps), 15 m (4 reps), 20 m (3 reps), 30 m (2 reps) and 40 m (1 reps) -> (15 sec of rest per each 1 sec sprinting) - Sled push resisted accelerations (30% BW), 5 m (3 reps) and 10 m (2 reps)

1, contents corresponding to the training day 1; 2, contents corresponding to the training day 2; 3, contents corresponding to the training day 3. Minimum of three blocks 1–2–3 in the functional phase before RTS. *Reps* repetitions, *BW* body weight, *NMES* neuromuscular stimulation. Mild discomfort allowed during exercise execution

Box 10.3B Basic Aerobic Conditioning

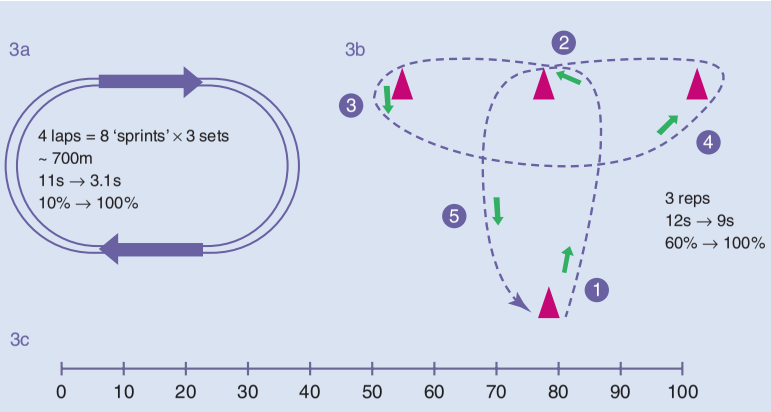
Basic aerobic conditioning included in the "Rehabilitation and RTS algorithm programme for hamstring injury" [14]

Basic aerobic conditioning (Mendiguchia et al. [14])		
	"Regeneration phase"	"Functional phase"
Performed every third day	Four sets × 5 min at low to moderate intensity (player rated)	Two sets × 10 min at moderate To high intensity (player rated)
• Commenced when the player is able to perform at least three sessions of running technique without any discomfort or pain in the regeneration phase		

Box 10.4 Running progression Whiteley et al. [17]

- Commenced when the athlete can perform 3× pain-free single leg squats and stationary bike for at least 5 min (2× BW effort in watts):
  - Performed on an indoor oval track with approximately 30 m straights, approximately 100 m around.
  - Performed with no pain and/or discomfort.
  - Including running drills, straight line running progression and direction changes (modified *t*-test).
- Running drills: In between each of the running sets, the athletes complete 2 laps:
  - Triple extension walks (1 lap).
  - 'B' drills (walking with late swing knee extension, see Fig. 10.11) (1 lap).
- Straight line running progression (3a):
  - One session consists of 3 sets (× 4 laps of 8 'runs').
  - Running effort is estimated by a visual analogue scale (0–100%), explaining the athlete that a 100% run would equal a maximum effort sprint, while 0% would be the slowest possible speed that the athlete could run at (3c).
  - The athletes start the straight portion of the 'run' from a walking start, and decelerate on the corners. Each time the athletes complete a set of the running (four laps, eight 'runs'), they are asked to rate how fast they thought they ran at their maximum effort during that set. The time of the run across the central 27 m (marked with cones) of the 30 m straights is recorded.
  - Progression: The first session is commenced at approximately 10–20% effort. During each session, if the athlete is able to complete a set confidently and without any discomfort, it is suggested to increase the running effort by 5–10% for each set. If any discomfort is experienced, if the athlete does not feel confident or displays lack of adequate mechanics or control (visual evidence of 'limping' or 'favouring' a leg), the athlete is instructed to return to the previous set's perceived running speed.
- Direction change running (modified *t*-test, including 90° and 180° turns) 3 × 1 (3b):
  - Commenced once the athlete is at or beyond 70% of maximal effort.
  - Athletes are initially instructed to run 10% slower than they achieved on the straight line running. The *t*-test is performed 3× per session and progress with 5–10% increases for each set.
- Running programme is successfully completed when full sessions of straight line running with *t*-test at 100% maximal effort are performed.

Running drills	– Triple extension walks (3 sets × 1 lap) – 'B' drills (3 sets × 1 lap)
Straight line running (3a)	– 3 sets × 4 laps (8 'runs') – Gradual progression from 10–20% up to 100%
Direction change running (*t*-test) (3b)	– 3 sets × 1 lap – Gradual progression from 60% to 100%
Running effort scale (0–100%) (3c)	



10.3.3.10 Lumbopelvic Control

Terminology such as "core strength" or "lumbopelvic stability" is often used interchangeably with "lumbopelvic control," which we will use in the following section. Lumbopelvic control is used to describe the desired adaptation from exercises involving muscles of the lumbopelvic and hip region, which appear to emphasise technique over absolute force output and tend to focus on balance, stability, and maintenance of posture during dynamic movement. Lumbopelvic control exercises gained popularity in hamstring injury rehabilitation following the mentioned study by Sherry and Best [42]. Fewer recurrences were found after rehabilitation in the group implementing the PATS protocol compared to STST exercises [42], and the findings led many clinicians to conclude that the PATS protocol enhanced lumbopelvic control and that this was the reason for fewer recurrences. However, it is unknown whether the PATS protocol actually caused any changes to lumbopelvic control, as no outcome measure of this variable was included in the study. Further to this, some exercises in the PATS protocol involved long-length and eccentric hamstring actions, compared to the relatively low-intensity STST exercises, which were predominantly isometric and performed at short muscle length. It has therefore been argued that the lower recurrence rate following the PATS protocol could be due to greater exposure to long-length and eccentric exercises and that the STST protocol was of a poor quality possibly inflating reinjury risk [67]. Typical lumbopelvic control exercises used in hamstring injury rehabilitation are variations of bridging exercises (Fig. 10.7), which to a certain degree also load the hamstring muscles specifically [68–70].

A training intervention focused on running technique drills and coordination has been shown to improve performance of a lower limb motor discrimination task compared to usual warm-up in elite Australian rules footballers [71]. While there may be a link between elements of lumbopelvic control and hamstring injury risk, current methods of objective assessment require laboratory-based testing, limiting most clinician's ability to monitor such variables throughout rehabilitation. Nevertheless, the potential value of lumbopelvic control exercises and perhaps, more importantly, the strength of the surrounding muscles of the lumbopelvic and hip region during hamstring injury should not be discounted. The argument that strength of these muscles will help support the actions of the biarticular hamstrings is plausible and warrants further investigation. In addition, many of these muscles are critical for a return to performance and should be targeted throughout the rehabilitation process regardless of their direct role in reducing risk of hamstring injury. Muscles, such as the gluteus maximus (GM) and adductor magnus (AM), may be strengthened during hip extension exercises already included in rehabilitation; however, there may be additional benefits of strengthening these muscles in movements such as hip thrusts and squats, which do not preferentially recruit the hamstrings. Including these exercises during rehabilitation allows maintenance and/or improvement of strength in movements related to sporting performance and can generally be performed at a relatively high intensity regardless of rehabilitation progress.

Fig. 10.7 Examples of double- and single-leg bridging exercises with a variety of progressions. The two columns to the left show double-leg bridging exercises, and the two columns to the right shows similar exercises progressed as single-leg bridging exercises. In the first row, a simple low-loaded bridging exercise is performed with the feet on the floor and with approximately 90° of knee flexion. The hip/pelvis is lifted towards full hip extension. Similar movement is performed, but with progression towards longer lengths (less knee flexion) and with the heels on the floor (second row) or with the heels on a step (third row). The movement can be performed with higher speed (more explosive), and for the single-leg bridging exercises, the uninvolved leg can be extended towards longer lever to increase the load and intensity. In the fourth row, a combination of the bridging exercises and an eccentric leg curl using catslides is shown, as a variation of the bilateral and unilateral eccentric slider shown in Fig. 10.6

10.3.3.11 Summary

A variety of rehabilitation approaches have been employed with reasonable results from high-level evidence. Common elements that seem to play a valuable role for rehabilitation after acute hamstring injuries include early loading and initiation of rehabilitation (i.e. within a few days after injury) and lengthening- and eccentric-biased exercises at progressively longer muscle lengths. Furthermore, gradual progressions in running loads (see Boxes 10.1–10.4) and sport-specific emphasis at the late phases of rehabilitation provide superior results to programmes where these elements are not included.

10.4 Rehabilitation After Proximal Hamstring Tendon Avulsions

10.4.1 Summary of Evidence

The vast majority of published literature on proximal hamstring tendon avulsions focuses on outcomes after surgical treatment [72–75]. It should therefore come as a surprise that very little information is available with regard to postoperative rehabilitation specifics. Rehabilitation protocols also demonstrate a marked variability in both composition and timing of rehabilitation components [76] (Level 5). Generally, postoperative rehabilitation includes an initial period of several weeks with restrictions in weight-bearing and range of motion [77–87] (Level 4). In this phase, the goal is to avoid excessive stresses on the repair and simultaneously load the tissue to minimise muscle atrophy. Several authors in Level 4 studies [78–83, 85, 88–91] and in one Level 2b study [92] have advocated the use of postoperative immobilisation (i.e. with a brace or cast) (e.g. in cases where intraoperative assessment of the repair reveals excessive tension). However, no consensus exists. In the early phase, it is important to regain good control (i.e. activation) of the hamstrings, so control and gait training are started early, followed by a progressive strengthening programme and sport-specific exercises. Return to sport is generally allowed from 6 months on [77, 80–82, 88].

10.4.2 General Postoperative Recommendations and Rehabilitation Guidelines

In general, following surgery of a proximal hamstring tendon avulsion injury, patients are recommended to follow the specific restrictions and advice provided by the surgeon, and individual considerations should always be taken into

account. A recent narrative review with more details on the rehabilitation programme was recently published [93] (Level 5) and is the basis for the following paragraphs.

Two of the main goals of rehabilitation are to minimise atrophy and the loss of flexibility, while providing adequate rest and optimal conditions for the healing of musculotendinous tissue. In addition, it is very important to do exercises to regain control and muscle strength as well as good mobility of the posterior thigh and the entire lower limb. All exercises should be commenced and progressed with minimal pain or discomfort (see Box 10.5 and Box 10.6).

Box 10.5 General Postoperative Recommendations After Total Proximal Tendon Avulsions

General postoperative recommendations after total proximal tendon avulsions (Askling et al. 2013) [93]	
Crutches	Crutches are generally recommended for approximately 6 weeks (outdoors), but there are some individual differences. Walking without crutches indoors can usually be performed earlier, but this may depend on the specific restrictions from the surgeon
Walking	In the first 3 weeks after surgery, only short steps are recommended to avoid excessive stretching of the hamstring muscles
Hip and knee ROM	Avoid excessive hip flexion bending the first 3 months, especially in combination with knee extension, to avoid excessive pull on operated hamstring muscles
Isolated hamstring exercises	Isolated exercises of hamstrings against resistance are not recommended before week 5
Passive stretching	Aggressive passive stretching to improve muscle flexibility is not recommended during the first 3 months after surgery. Active ROM exercises are preferred
Hamstring muscle activation and muscle atrophy	Voluntary activation of the repaired hamstring muscle group may be challenging initially in postoperative rehabilitation. Muscle atrophy is common, particularly involving the biceps femoris long head with compensatory hypertrophy of the short head [94]. The feedback technique by instructing the patient to put her/his hand on the operated muscle when trying to isometrically contract it may be useful to address this problem
Muscle strengthening	Muscle strength is difficult to regain, especially eccentric strength. Most patients need to perform muscle strengthening exercises during an extended period of time (often ≥1 year) to reach equal strength in both legs. A prolonged period of hamstring muscle strengthening is therefore recommended

Box 10.6 Week by Week Rehabilitation Progression Guidelines

Week by week rehabilitation progression guidelines (Askling et al. 2013) [93]	
Week after surgery	Recommended rehabilitation guidelines
Week 1	• The operated hamstring muscles should be kept in a shortened and relaxed position to avoid traction on the reattached tendon • Sitting on the affected ischial tuberosity should be avoided, except when using the elevated toilet seat • The patient is allowed to place body weight on the operated leg in a neutral standing upright position with crutches, but only toe touch weight-bearing is permitted • "Safe" and simple exercises can be performed, including isometric contractions of the quadriceps and gluteal muscles, ankle pumps to avoid deep vein thrombosis, and carefully performed muscle flexibility exercises by allowing approximately 30–45° of knee flexion in supine position. These exercises are recommended to be performed four times daily with 3 sets x 10 repetitions within pain-free limit
Week 2	• Therapeutic exercises for the next 5 weeks are recommended. Since three out of four hamstring muscles span two joints, both hip and knee joint positions need attention when prescribing exercises • When 30° of hip flexion is reached in a straight leg raise, the patient is allowed to walk using crutches with full weight-bearing and short strides on the operated leg and also permitted to stand on the operated leg, single-leg stance, and perform minor knee flexion/extension exercises • "Safe" exercises such as supine isometric contractions of the hamstring muscle of the operated leg should be carried out as tolerated • Sitting for short periods of time in an elevated chair is allowed • In a prone position, passive knee flexion/extension exercises can be performed. These exercises should also be done with assistance at home twice daily, 3 sets x 10 reps • The main objectives during the second week are to be able to activate the hamstrings on the operated leg and to walk with short (foot long) weight-bearing strides with crutch assistance
Week 3	• If the exercises during week 2 are performed cautiously and without pain, the rehabilitation should progress with two complementary exercises: – First, standing on the nonoperated leg with full weight-bearing and careful knee flexion exercises of the operated leg with the ankle joint in plantar flexion are added – Secondly, stationary slow walking on a thick pad with increasing knee lifts is encouraged • Calf strengthening is permitted in a standing position with a straight leg and full weight-bearing
Week 4	• If there is adequate balance/postural control and motor control when standing/walking, there is no further need for crutches indoors • Pool training with a belt is allowed if the wound is healed • Stationary biking with the saddle in a high position is permitted when the patient can reach 70° of hip flexion combined with 90° of knee flexion • Isolated resistance exercises involving the operated hamstring muscles should still be avoided

Week by week rehabilitation progression guidelines (Askling et al. 2013) [93]	
Week after surgery	Recommended rehabilitation guidelines
Week 5	• All exercises from the first weeks may now be stopped (isometric contractions, passive flexion and extension exercises, and standing knee flexion) • Specific hamstring strengthening exercises with increased intensity but performed slowly are now included, such as static leg curls in a sitting position (Fig. 10.8) and single-leg catches with a cable • In addition, lumbopelvic exercises are also introduced (Fig. 10.7)
Week 6	• By this point, the patient's gait should be "normal," that is, ambulation without limping • During the sixth week, exercises for improving muscle flexibility, single-leg balance, and neuromuscular control including lumbopelvic control training are introduced, such as lunge walking, and specific isometric hamstring contractions in the prone position with resistance to the heel (in leg curl machine) – By performing this exercise with the leg in internal rotation, the medial hamstring muscles may be preferentially isolated. With the leg in external rotation, the biceps femoris (BF) muscle may be preferentially loaded
Week 7–	• Eccentric training of the operated hamstring is typically initiated together with at least 2 days of rest a week for this muscle group • A manual strength evaluation should be performed initially in the prone position with knee flexion and hip extension on a weekly basis. Later in the rehabilitation process, multiple test positions are utilised to assess strength and provoke pain • Evaluation of ROM of both the hip and knee joints should be included. A side-to-side comparison for strength and flexibility is recommended. It is advised that patients perform 2–4 hamstring exercises at each training session with a 100% focus on quality rather than quantity • A common problem is that the patient often uses the agonist muscles such as the GM, AM (both being strong extensors of the hip joint), and gastrocnemius, as well as the short head of BF, rather than the operated hamstring muscles • Cautious jogging, both forward and backward, with short strides, including accelerations/decelerations is now permitted. Stationary jogging with high knee lifts at increasing intensity over time can be performed. The single-leg bridge is a good example of an isolated hamstring exercise in the supine position with combined hip extension and knee flexion. In each training session, specific hamstring strengthening exercises should be combined with more complex exercises such as lunges, squats, and different types of jumps. Dynamic leg curls in both the prone and sitting positions should be a part of the strengthening phase of the programme
Sport-specific and functional phase	• More aggressive, sport-specific activities are integrated, allowing full unrestricted ROM in an effort to prepare the patient for return to prior level of sports activity • Outdoor training, slope training, video filming of the running technique, or other sport-specific movements are encouraged • The duration of this sport-specific and functional phase may vary depending on the individual athlete and the specific sports requirements

Week by week rehabilitation progression guidelines (Askling et al. 2013) [93]	
Week after surgery	Recommended rehabilitation guidelines
Return to sport (±6 months after surgery)	• It is recommended that patients can return to sports when sport-specific activities and functional abilities such as jumping, running, and cutting can be performed without pain, stiffness, or a feeling of insecurity • The time to RTS may vary depending on the progression of the rehabilitation and sport-specific and functional phase for the individual athlete. In many settings, specific predetermined criteria are required to be completed

Fig. 10.8 Examples of isometric hamstring exercises for postoperative total proximal tendon avulsions. The first two rows show simple, low-loaded isometric exercises. The exercises are mainly knee dominated (lying in supine position with heels on the floor or on a step/chair with various degrees of knee flexion, or sitting on a chair when tolerated). Increased knee extension and/or hip flexion can be performed to increase the load towards longer lengths. Higher-loaded knee-dominated isometric contractions can be performed in a leg curl machine with the hips in a relative neutral position and the knees flexed at approximately 90° and towards longer lengths (shown in the third row). Be aware that specific hamstring strengthening exercises against resistance should be avoided in the first 5 weeks after surgery [93]

10.5 The Young Athlete: Nonoperative Management of Avulsion Fracture Injuries

Adolescents are no exception to the high injury incidence rates that are reported among athletes [96, 97]. However, in contrast with adults, these young athletes have not yet reached full skeletal maturity, which leads to a predisposition to avulsion fractures. In adolescents, incomplete apophyseal ossification and fusion to the pelvis coincide with a hormonally induced increase in muscle strength [98–101]. Consequently, rather than the musculotendinous junction, the physis is the weakest link in the bone-tendon-muscle complex, potentially resulting in avulsion fractures following forceful contraction [94, 98–102]. Similar to hamstring tendon avulsions, the extent of retraction (or fragment displacement) (Fig. 10.9) has been mentioned by various authors as a factor that guides the choice for either surgical or nonoperative treatment. Despite differences in cutoff values for fragment displacement (e.g. 10, 15, or 20 mm), there seems to be a higher risk of nonunion and inferior outcomes of conservative management with higher degrees of displacement [101, 103, 104]. However, high-level evidence is lacking. Based on a recent systematic review of available literature (including only Level 4 studies), fragment displacement of >15 mm warrants surgical consultation [101]. For avulsion fractures with <15 mm displacement, conservative treatment is recommended as primary treatment.

Few detailed rehabilitation programmes have been published [99, 103–106] (Level 4), as is clear from the literature overview provided in Table 10.2. Generally, these time-based protocols include an initial acute management phase of rest with weight-bearing and sitting restrictions for several weeks. This phase may be prolonged in case of more displacement, delayed presentation, or persisting

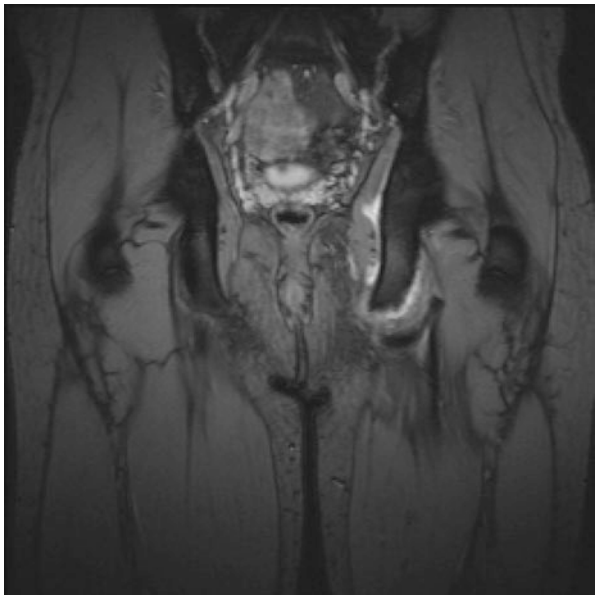


Fig. 10.9 Coronal T2-weighted MR image of a 15-year-old male demonstrating a left-sided avulsion fracture involving the ischial tuberosity with fragment displacement <15 mm

Table 10.2 Literature overview of employed rehabilitation programmes after avulsion fractures (Level 4 studies)

Reference	Phase I	Phase II	Phase III	Phase IV	Phase V	Phase VI	Comments
Ferlic et al. [103]	*Weeks 0–6*: • PWB (crutches)	• Isokinetic and isometric strengthening of hamstrings and adductors	• Concentric and eccentric strengthening of hamstrings and adductors	–	–	–	
Kujala et al. [104]	*Weeks 0–4*: • Rest from all sports • FWB allowed	*Weeks 4–8*: • Modified training	–	–	–	–	Phases I and II may be prolonged for a displaced fracture (>5 mm), delayed presentation (>1 month), or persisting symptoms
Metzmaker and Pappas [105]	*Days 0–7*: • No activity • PWB (crutches) after 3 days	*Days 7 to 14–20*: • Guided exercise with progression in ROM • PWB (crutches)	*Days 14–20 to 30*: • Progressive strengthening (resistance training)	*Days 30–60*: • Progression of strengthening exercises • Limited sport-specific training • FWB/cycling	*Days 60 to RTP*: • Sport-specific training • Aim at full ROM and strength	–	Progression is based on subjective pain, pain and findings on palpation, range of motion, muscle strength, level of activity, and radiographic appearance

Schoensee and Nilsson [106]	*Weeks 0–2*: • Neuromobilisation (ankle motion) • NWB (crutches) and protected sitting	*Weeks 2–4*: • Soft tissue mobilisation • Limited hamstring ROM • Isometric lower extremity strengthening • Core and upper body strengthening • PWB and protected sitting	*Weeks 4–6*: • Progression of previous exercises • Progression of PWB to WBAT	*Weeks 6–8*: • Eccentric strengthening • WBAT	*Weeks 8–12*: • Progression of previous exercises • FUNCTIONAL training • Running • FWB	*Weeks 16+*: • RTP phase	Rehabilitation programme is designed for symptomatic delayed union and is preceded by ultrasound-guided percutaneous needle fenestration RTP may be guided by criteria such as no pain and a functional hop test score of 90% (compared to the contralateral leg)
Schuett et al. [99]	*Weeks 0 to 4–6*: • PWB (crutches)	• Physical therapy with focus on flexibility					For subacute avulsion fractures, the initial phase consists of activity modification

NWB no weight-bearing, *PWB* partial weight-bearing, *WBAT* weight-bearing as tolerated, *FWB* full weight-bearing, *ROM* range of motion, *RTP* return to play

symptoms. In the restoration and recovery phase, weight-bearing is progressed, and isometric exercises are introduced, followed by concentric and eccentric strengthening. Finally, in the sport-specific phase, the athlete is prepared for RTS with sport-specific exercises. There is no consensus on RTS criteria, but factors such as full range of motion, strength, hop tests, and radiographic evaluation have been used [105, 106].

10.6 Rehabilitation of Proximal Hamstring Tendinopathy

This section is focusing primarily on management of isolated proximal hamstring tendinopathy (PHT), although, not infrequently, clinicians may be required to also manage comorbidities, most commonly sciatic neuritis. The evidence for treatment and rehabilitation following PHTs is limited, and hence, clinical recommendations and expert opinions [95, 107] (Level 5), as well as work in other tendinopathies, form the mainstay. Clinical assessments will very much direct the rehabilitation. Most presentations have a combination of pain, muscle weakness, and loss of function. Further, the patient may also experience related problems along the kinetic chain which may perpetuate the issue. Fundamentally, rehabilitation comprises a graduated approach to loading of the affected region while minimising exacerbation of symptoms. This generally necessitates a stepwise approach to controlling pain, increasing muscle strength and function followed by introduction of faster loading and RTS.

10.6.1 Restoration and Recovery Phase

Control of pain in an exercise sense is suggested to be mediated initially through isometric exercise [108, 109]. The important element in dealing with hamstring tendinopathy is applying this in a neutral hip position in an attempt to minimise compression of the complex hamstring tendon of origin [110]. Although no evidence exists for PHT, the parameters for isometric loading provided by Rio et al. for patellar tendinopathy may be quite applicable, that is, four repetitions of 45 s contractions, three or four times daily [95, 107]. Exercise approaches that are commonly utilised include isometric trunk extension or a bilateral long leg bridge, which can be progressed to the unilateral version as symptoms permit. Others include isometric straight leg pull down with hip approaching extension or isometric hamstring leg curl (see Fig. 10.10). Importantly, the resistance should be progressed to meet improvements in strength to stay within the recommended resistance around at least 70% of maximal voluntary contraction (MVC) [109]. Sitting can be painful, particularly in older females, so another means of assisting pain management may be through the use of pressure-relieving padding/cushions. Monitoring pain while adjusting the load stimulus appears

Fig. 10.10 Isometric hamstring exercises for pain control in the early rehabilitation phase for PHT. The exercises are performed in a hip in neutral position, progressing from two legs (left) to one leg (right). The first row shows isometric double- and single leg long leg bridges (also see Fig. 10.7, row 2 and 3). The second row shows isometric trunk extensions with progressions (note that the position of the hips on the support pillow has to be adjusted according to the athlete's ability to tolerate the load). The third row shows isometric long leg curls in a prone position

to be a key element of successful rehabilitation. Selection of an appropriate provocative exercise test is somewhat dependent on stage of rehabilitation and sporting aspirations of the patient. For example, early challenges may be a single-leg bridge at 60° of hip flexion, with later challenges being arabesques or lunging manoeuvres, quickly flexing the torso over the plant leg [95]. These tests are best applied 24 h or so following the exercise session to determine the patient's tendon response to the load applied. In general, this 24-h response is ideally managed to pain of less than 2/10 on the provocative test.

Application of strength or heavy slow resistance exercises [111, 112] has been demonstrated to have good utility in the management of patellar and Achilles tendinopathy and may similarly be applied in the hamstring paradigm. This is generally commenced once pain on provocative tests has been controlled as above. The major challenge in symptomatic tendinopathy is applying this principle while minimising exacerbation through proximal compression in the earlier stages. These isotonic exercises generally consist of 3–4 sets of 8–15 repetitions at a resistance calibrated to the moving 8 or 15 repetition maximum for the last set. Each repetition is generally performed slowly, i.e. lifting for 3 s and lowering for 3 s. The use of a metronome may also have a positive effect on strength gain [113]. To optimise stimulus and strength gains, single-leg exercises are preferred wherever possible. Higher-level athletes may combine two or three exercises in the one session for a total of six to eight sets to maximise and vary the stimulus. Typical exercises include prone hamstring curls, hamstring bridges (with knee extended to approximately 45°), NHE, Bosch high-load isometric holds (single leg), or 45° hip extensions, limiting the hip flexion in early phase to avoid tendon compression [114] and progressed later according to the pain response the following day. Walking lunges, step-ups, sled push, and RDLs all with resistance and performed slowly may also be utilised later in this phase, progressing hip flexion over weeks from 45° to deeper angles of 80–90° [95] or further depending on the demands of the sport (see Fig. 10.11). Careful monitoring of next-day response in this phase is often required as the increase in range in some athletes may provoke exacerbation. In cases where this limits progress, continuing on to higher-speed work then revisiting the range challenge later may be a more pragmatic approach. Other approaches to train muscle strength and progress range such as single or multiple sets to exhaustion at a moderate weight, or the utilisation of occlusive training may also achieve similar aims but these are yet to be assessed in this condition. Strength training is generally performed every second day to allow recovery from such a stimulus, with three sessions per week considered to provide a modest benefit over two sessions [115]. Continuation of isometrics on the intervening days is recommended. They may also be of some value pre-strength training given their utility in reducing the motor system inhibition which appears to be associated with tendinopathy [109, 116].

Many sedentary people or recreational athletes may not require further progression at this phase. However, if the athlete is returning to a sport involving accelerations, deep hip flexion, and/or lunging or change of direction, further rehabilitation is required to prepare for these demands by incremental addition of challenges involving higher rates of force absorption, deeper eccentric work, and multidirectional challenges.

10.6.2 Sport-Specific Phase

Progression to higher-speed challenges of the recovering hamstring tendon requires incremental increases which may also be monitored in terms of next-day pain

Fig. 10.11 Examples of exercises applied in the later restoration and recovery phase following PHT. The main aim is to improve strength and gradually introduce speed/energy storage loads towards the sports specific phase. In the first row, progressions of one leg hip thrusts with a barbell are shown. The second row shows walking lunges at various degrees (45 degrees and deep lunges) and step up exercises; on the right is a step up a step up performed with higher speed, combined with a forward drive of the opposite leg towards the wall. In the third and fourth row, box jumps and sled push exercises are demonstrated, respectively

response to provocative activities such as the arabesque or forward lunge. The tendinopathy should be well-controlled in terms of pain at slow loading and provocative testing prior to commencing this stage [114]. Loads should be carefully quantified and progressed using load management principles [117] to avoid exacerbation. Recommendations of a 3-day loading cycle to allow the tendon to accommodate and adapt to the higher-level stimulus [118] reflect the challenge of this component. Continuation of strength and isometric loading on the intervening days also falls within these recommendations [95, 107]. Common exercises include stair or step-up drills, faster sled push, scooter pushing up an incline, and running drills such as A-skips and B-skips (see Fig. 10.12). Most of these are primarily propulsive drills. These can be complimented with more eccentric/absorption bias such as split squat jumps, kettlebell, and change of direction drills. In planning these challenges, commencing with the elements primarily in the propulsive group and then progressively replacing these with drills from the latter group would appear to provide

Fig. 10.12 Examples of running drills. A-skips are demonstrated in the first row, where the aim is to rapidly drive the knees high (above the hips) and bring them down fast while maintaining an upright posture and actively using the arms. In the second row, B-skips are demonstrated; the first movement is similar to A-skips (rapidly driving the knee high above the hips), but instead of rapidly snapping them back down, the knee is extended and the leg is brought back down pawing the ground with the forefoot and kicking back until it is aligned with the hip. The B-skip is supposed to be performed with the feeling of moving the leg in a circular motion.The A- and B-skips can be progressed by increasing the speed and/or going for more height

effective graduation. In returning to training and ultimately competition, the athlete should be prepared for a similar number of higher-speed and elastic challenges as the training and games present, through gradual progression of the drills, gradually replacing them with on-field activities as training involvement increases. It appears important not to add further tendon load over and above that encountered within the training and playing of a sport [119].

It is worth noting a small subgroup of middle aged, more commonly female, runners with hamstring tendinopathy who uncommonly present with very good hamstring but very poor GM function. Better outcomes appear to be associated with less focus on rehabilitation of the tendinopathy and more predominantly on

regaining function in the GM through activation, strength, and synergy [120], for example, hip thrusts and hip extension exercises.

10.7 Individualisation Throughout Hamstring Injury Rehabilitation

There is no consensus as to an optimal approach to guide progression through hamstring injury rehabilitation nor has it been directly studied. For acute hamstring injuries, time-based progressions come mostly from experimental studies of the pathophysiological healing of induced damage to animal muscle tissue [121–127]. These studies provide valuable information on the muscle regeneration and healing process after injury but may not necessarily be the most appropriate way to determine whether an injured individual is ready to progress exercise throughout rehabilitation. Partly in conflict with time-based progressions, the rehabilitation and RTS process following a hamstring injury is recognised as a dynamic continuum during which the nature and difficulty of exercises are progressed in response to tissue healing [18] as well as the functional abilities of the athlete. Thus, the content and progression of a rehabilitation programme may vary between athletes and will often be individualised. Criteria-based RPs have recently gained popularity, as they allow for a more individualised approach to progression than pathophysiological time frames for muscle healing [4, 13, 14]. These protocols include an increased emphasis on training loads and performance-related factors that might be necessary to prepare the athlete for unique sporting demands [4]. Progressing rehabilitation based on the injured individual's tolerance to different exercises provides an alternative approach to using independent clinical assessments or predetermined time frames. This exercise-specific approach considers rehabilitation along a continuum of progression/regression based on exercise difficulty and intensity, and progression of each type of exercise is specific to its joint action, muscle involvement, and contraction mode, rather than separating exercises into discrete phases of rehabilitation. The disadvantage of current criteria-based RPs is that criteria are most often set arbitrarily, meaning that passing certain criteria is no guarantee for optimal progression. Thus, more research into these criteria and their prognostic value and validity is needed in the future.

10.8 Conclusion

For acute hamstring muscle strain injuries, there is growing evidence that early loading and initiation of rehabilitation as well as lengthening and eccentric-biased exercises at progressively longer muscle lengths seems to be of importance. Furthermore, gradual progressions in running loads and sport-specific emphasis seem to provide superior results to programmes where these elements are not included. Following surgery of proximal hamstring tendon avulsion

injury and avulsion fractures in the adolescent athletes, there is a lack of high-level evidence. However, specific restrictions should be followed, and individual considerations should always be taken into account. The rehabilitation process is mainly time based with exercises commenced and progressed with minimal pain or discomfort. Following surgery, the main rehabilitation goals are to avoid muscle atrophy and reduced flexibility more than necessary while providing adequate rest and optimal conditions for muscle tissue healing. The evidence for rehabilitation following proximal tendinopathy is lacking. However, based on the general tendinopathy literature, rehabilitation is suggested to comprise a graduated approach to loading of the affected proximal hamstring tendon while minimising exacerbation of symptoms.

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Return to Sport After Hamstring Injuries

11

Martin Wollin, Noel Pollock, and Kristian Thorborg

11.1 Introduction

Returning athletes to training and competition after hamstring injury can be complex. This is evidenced by substantial and unchanged re-injury rates associated with hamstring injury in sport over the last 30 years [1]. Most athletes return to sport (RTS) 3 weeks after a hamstring injury [2, 3]. However, about one in three athletes may re-injure in the first few weeks after returning to sport [2–5]. There is also risk to the athlete of sustaining a subsequent injury to another area of the body [6, 7]. Discussions and different opinions regarding accelerating RTS following hamstring injury have been ongoing for many years [8, 9]. Much of this debate focuses on the potential advantage of increasing the number of available players, which may increase the chance of team success. This point is balanced against the increased risk of re-injury and reduced performance of individual athletes associated with a lack of full hamstring and sprinting function. While athletes in some team sports may be able to perform and be selected to compete despite reduced hamstring function, individual athletes such as sprinters will be more directly affected. Their inability to produce maximal acceleration and velocity and thus achieve optimal running speed and performance makes an early return to competition irrelevant from a performance perspective. Recommendations and reasoning concerning RTS decisions are therefore always specific to the individual sports context and risk-taking assessment.

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This chapter aims to introduce a criteria-based approach designed to monitor athlete progress, which stakeholders can collectively consider when navigating athletes through the rehabilitation and RTS phases.

11.2 Return to Sport Principles After Hamstring Injury

An evidence-based consensus on how to best return athletes to sport after hamstring injury is currently not available to practitioners. A clear RTS definition is also absent in the literature. Attempts have been made to develop consensus statements around the RTS definition, criteria and decision-making in sport generally [10] and football specifically [11, 12]. However, differences remain between these expert-based opinion pieces, evidenced by reports of different RTS criteria within the same sport [11, 12]. A group of 58 international medical experts omitted hamstring strength and training load from their RTS criteria [11], whereas a different study that involved medical practitioners from professional football clubs demonstrated a complete agreement to include hamstring strength and training load parameters in the RTS criteria-based decision-making process [12]. A recent international Sports Physical Therapy consensus statement recommended that RTS processes are aligned with the athlete's sport and their level of participation for the planned sporting return [10]. That statement outlined three steps as part of a RTS continuum: return to participation (modified training), sport (full training) and performance (back to the same level of competition standards), which highlights a gradual progression in function while simultaneously underlining that workload (sport-specific preparation) is an important element in the criteria-based RTS process. To seamlessly map and implement ongoing strategies designed to reduce recurring and subsequent injury, we recommend that tertiary prevention is added as the 'plus one' to the three step RTS continuum outlined by Ardern et al. [10]. The 'three plus one' RTS phases following hamstring injury is outlined in Fig. 11.1. Additionally, Fig. 11.1 illustrates where each criterion is applicable in the RTS continuum according to current evidence.

This chapter also considers the steps in the Strategic Assessment of Risk and Risk Tolerance (StARRT) decision-making model [13] and current available and emerging evidence relevant to returning athletes to sport after a hamstring injury, understanding that the RTS decision is multifactorial and unique to each case and that the StARRT model might be applied at different points during the continuum.

Throughout the rehabilitation and RTS process, athlete progress can be evaluated using clinical and functional tests. Such tests can be considered not only for RTS criteria but also for tertiary prevention. This approach involves performing a range of intrinsic objective and subjective tests on the athlete in the clinical setting, evaluated by medical staff. Functional testing reflects the physical demands of the sport, athlete position and level of competition. Sport-specific readiness involves monitoring and managing workload criteria to provide data on the extent to which the athlete has trained and how well they have performed during their hamstring injury rehabilitation. Sport-specific readiness is considered a critical component in the RTS

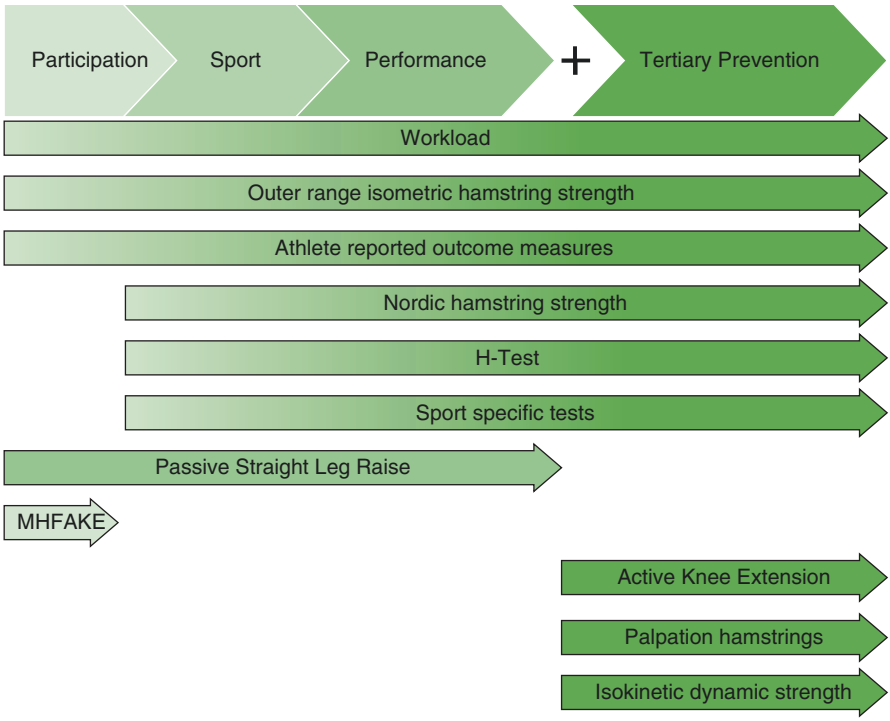

Fig. 11.1 The return to sport continuum [10] complemented by tertiary prevention in a 'three plus one' model that outlines where test criteria can be considered for application on the continuum according to current evidence. *MHFAKE* Maximal Hip Flexion and Active Knee Extension

decision-making process due to its association with increased or decreased risk of injury in sports where hamstring injury is prevalent [14–20]. Such a RTS systems approach, through four domains, presents a progressive scale of standards reflective of a graduated rehabilitation and prevention process. Data collected in each domain can be interpreted in context and assist in providing information to the stakeholders when making a shared RTS decision. The approach aims to facilitate a RTS process that evaluates athlete performance across multiple domains and criteria when transitioning towards a successful return to performance. It reinforces that the RTS process is not an isolated procedure that follows completion of the rehabilitation, but is instead a process that starts concurrently with the initiation of hamstring injury rehabilitation.

11.3 Return to Sport Decision

Elite athletes undertake a host of clinical tests during the year, e.g. baseline screening, in-season monitoring, injury diagnosis and evaluation of rehabilitation progress. Tests might be applied throughout the year to monitor athlete health states or injury susceptibility, whereas some tests are utilised at defined periods during the

RTS continuum. Such an approach reflects the steps in StARRT: assessment of health, activity risk and overall context-specific risk tolerance. Specific information on relevant impairment and performance-based tests and their execution can be found in Chap. 9.

In the absence of a consensus on the best RTS criteria-based process, an overall decision-based model has been introduced to assist practitioners [21]. It provides a three-step process to consider when returning an athlete to sport after injury. The initial step involves examining medical factors to ascertain the current health status of the athlete. The second step reviews sporting risk specifically in relation to modifiable variables such as type of sport, playing position or level of competition. Finally, externally influencing factors such as time of season and pressure from athlete or third parties are considered in the process. A strategic assessment of risk and risk tolerance framework (StARRT) in relation to RTS decision-making has been proposed by Shrier [13]. This framework includes tissue health and stress level assessments of health and activity risks in relation to contextual risk tolerance [13] that may be valuable for RTS decision after hamstring injury (Fig. 11.2).

11.3.1 Multidisciplinary Review of Standards

It is clear that most athletes RTS with hamstring impairments, which may increase the risk of re-injury. A multidisciplinary and shared decision-making process is therefore recommended [10] when evaluating an athlete's capacity and the risk involved in returning to sport. Practitioners are advised to communicate a proposed set of standards for key stakeholders' consideration, including seeking a consensus on the decision-making process and level of risk tolerance at the outset, to optimise rehabilitation and RTS outcomes. It reflects that, in elite and professional sport, shared-decision-making is ideally collaborative and collective; no single entity holds a veto on RTS criteria post hamstring injury. Once a multidisciplinary, shared criteria-based RTS decision has been made, the athlete should remain in tertiary prevention irrespective of whether they have returned to training, competition or top performance. Based on available data, this should be in place for at least 3 years post-injury. Planning (including roles and responsibilities) and producing the tertiary prevention programme should be part of finalising RTS processes. This is warranted due to the high rates of recurrence and subsequent injury and will involve an array of interventions including exercise programmes, load and athlete monitoring. It is acknowledged that contextual circumstances such as timing of season, athlete age, importance of event, chance of winning versus risk of losing and other 'risk tolerance modifiers' might influence how the four domains are utilised in individual cases within the continuum. A truly shared decision-making model collects broad perspectives that include nonphysical measures to gain understanding of the athlete's psychological and physical readiness to RTS.

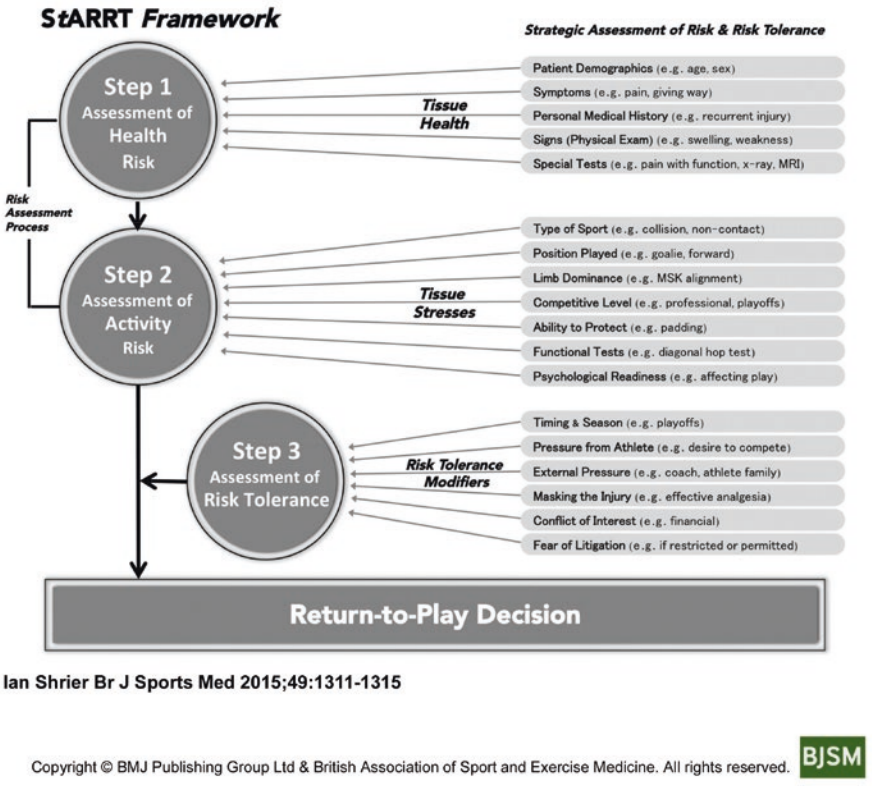

Fig. 11.2 The Strategic Assessment of Risk and Risk Tolerance (StARRT) framework for return-to-play (RTP) decisions. This framework illustrates that patients should be allowed to RTP when the risk assessment (steps 1 and 2) is below the acceptable risk tolerance threshold (step 3), and not allowed to RTP if the risk assessment is above the risk tolerance threshold. The StARRT framework groups factors according to their causal relationships with the two components of risk assessment (Tissue Health, stresses applied to tissue) and risk tolerance, as opposed to the three-step framework that groups factors according to the sociological source of the information. In some cases, apparently a single factor can have more than one causal connection and would be repeated. For example, play-offs will increase the competitive level of play and therefore increase Tissue Stresses and increase risk. However, it is also expected to affect a patient's desire to compete (i.e. mood, risk of depression) and could affect financial benefit as well. These causal effects would lead to increased risk tolerance. In this framework, each outcome is evaluated for RTP, and the overall decision is based on the most restricted activity across all outcomes (see text for details)

11.4 Psychological Factors in Return to Sport

At the time of RTS, athletes may develop negative psychological responses including anxiety, low self-esteem and fear [22]. These emotions can impact both on the time taken to, and level of, RTS athletes achieve post-injury [22]. The psychological

responses might be heightened in athletes returning from severe injuries such as recurring, injury sequela and hamstring tendon pain. Athlete anxiety is also a potential predictor for recurring and subsequent injury at the time of RTS [23, 24]. A premature RTS can lead to fear, anxiety, recurring and subsequent injury, depression and poorer performance [25]. Psychological readiness to RTS is multifaceted, complex and reliant on several factors [26]. Validated outcome measures to monitor 'psychological readiness' exist, and the information might be considered in a shared multidisciplinary decision process to evaluate this parameter [22, 26] during the RTS continuum.

11.5 Sport-Specific Readiness

During the RTS process, athlete sport-specific readiness is ascertained to establish if sufficient training, workload and performance have occurred to successfully RTS at the desired level of the continuum. This process involves a gradual increase in training and workload that is monitored and managed towards performance criteria. Most sports, particularly at the elite level, require complex coordinated movements to sprint, kick or change direction at high speed. The restoration of normal sport-specific kinematics at speed should therefore be considered within progressive rehabilitation and assessed prior to RTS, as this may influence hamstring re-injury risk and optimise sporting performance [27–29].

11.5.1 Workload

Monitoring and management of workload has become routine in elite and professional sport. A recent consensus statement suggests that load monitoring is an essential assessment tool for determining the effectiveness of training adaptations, athlete response to training, fatigue and recovery and minimising risk of injury and illness [30]. Load is generally classified as internal or external. Internal loads refer to physiological and psychological athlete responses to external loads. The actual workload performed by the athlete in training and competition is reported as external load. Monitoring both categories of load has been recommended where possible, since they can produce diverse risk profiles [6, 15]. Load monitoring and associated athlete management is an ongoing process including periods of rehabilitation; return to participation, sport, performance; and tertiary prevention phases. Monitoring of running load is of particular interest since this is the main hamstring injury mechanism [6, 20, 31]. Commencing running during rehabilitation within 4 days of lower limb muscle injury (41% hamstring) resulted in significantly increased risks of recurring and subsequent injuries compared to when running started 5–9 days post-injury [6]. Importantly, delaying running to at least 5 days post-injury did not delay RTS [6]. Workload appears to have a greater influence on the risk of recurring and subsequent injuries than the results of clinical tests such as active knee extension and outer range isometric hamstring strength [6, 32]. Additionally, the number of training sessions

completed from the time of medical clearance after injury to match play have been shown to influence muscle re-injury rates [33]. Completion of fewer than four training sessions was associated with a reinjury rate that was three-fold higher than the average muscle injury rate in professional football [33]. This risk was reduced by 13% for each additional training session completed before the first match after injury [33], and this highlights the importance of sport specific preparation and readiness as part of the RTS and tertiary prevention processes after hamstring injury.

11.5.1.1 External Load Monitoring

Monitoring workload with individual global positioning system (GPS) units produces data that might be of interest in returning athletes to sport after hamstring injury. Variables of particular interest include acceleration, deceleration and the type, speeds, volume and distances of running. It has been established that higher sampling rates of GPS units are associated with improved validity and reliability [30]. The precision of GPS running speed data is decreased in the presence of large speed variability [30]. A recent consensus statement on monitoring athlete workload with GPS recommends caution when interpreting acceleration, deceleration, change of direction and within-subject test-retest data [30]. In game scenarios, where precise athlete test-retest results from explosive actions and high-speed running are required to establish sport-specific readiness, GPS data might best be presented with indications of the minimal detectable change with 95% confidence intervals (MDC 95%CI).

11.5.1.2 Internal Load Monitoring

External load monitoring and exposure to high-speed running in particular appear to be important in the management of rehabilitation, RTS and risk with respect to hamstring injury. Internal load monitoring is also commonplace and typically includes rate of perceived exertion (RPE) [30]. RPE provides a subjective report on the athlete's physiological and psychological response to loading. The relationship between recent and historical internal load data appears to be associated with fatigue, injury and re-injury. It may therefore be useful to monitor internal workload to monitor sport-specific readiness. However, internal load monitoring provides no correlate of high-speed running exposure.

Acute-to-Chronic Workload Ratio

The acute-to-chronic workload ratio (ACWR) is an index of an athlete's workload in the most recent 1-week period (acute load) usually compared to their cumulative average workload over the last 3 or 4 weeks (chronic load) [18, 34]. The index is based on internal and/or external load data [15, 18, 35] to provide information on sport-specific readiness [36]. Inclusion of ACWR as a RTS criteria has been recommended [37] since rapid increases in acute workloads are associated with increased injury risk in a host of sports [15, 17, 18, 38] as are low chronic workloads. A high chronic load combined with a balanced acute load appears protective against injury [18]. This is an important recognition that should be reflected in rehabilitation plans

by commencing modified training (return to participation) early while still considering the pathobiology of a muscle injury, to retain or regain sufficient chronic sport-specific loading. An early return to participation should be balanced against the possibility of increased recurrence and subsequent injury rates if running is commenced prematurely out of sync with muscle pathobiology [6]. Risk management of re-injury associated with ACWR as an injury risk factor is reflected in the sport-specific and decision modifiers of the StARRT framework. The actual ACWR index linked to injury or re-injury differs between sports, cohorts and individual athletes [15, 18, 34]. A universal ACWR 'sweet spot' does not appear applicable and the ACWR RTS criteria should reflect context-specific data. Additionally, recent discussions about how best to calculate ACWR data are ongoing and involve using rolling averages and exponentially weighted moving averages [36, 39]. Recent data show that large spikes in ACWR in either model are associated with significantly increased injury risk [36].

11.6 Ongoing Monitoring and Prevention

Passing and progressing through agreed RTS standards including all or some of the clinical, functional, sport-specific readiness and RTS criteria does not mean that an athlete has arrived at a designated end point of injury management. Once an athlete has sustained a hamstring injury, they host a potent non-modifiable injury risk factor: previous injury. A symptom or consequence of previous hamstring injury is impaired function demonstrated by deficits in: running performance [28], isometric and eccentric strength [27, 40], high-repetition concentric hamstring strength and reduced resilience to withstand fatiguing sporting demands [41] and difficulty improving Nordic exercise strength [42] for up to 3 years after the injury. Subsequently, the risk of recurring or subsequent injury is elevated [7, 43, 44]. Management should commence early, within 7 days, upon RTS [32]. This represents the stage of tertiary prevention. Tertiary prevention describes 'clinical activities' aimed at preventing deterioration or reducing complications of a diagnosed condition [45]. Components of tertiary prevention in relation to hamstring injury in sport include regular exposure to eccentric hamstring stimuli, high-speed running and sprinting, load monitoring and management and in-season athlete monitoring of hamstring function.

11.6.1 In-Season Athlete Monitoring of Hamstring Function

Ongoing athlete monitoring post-RTS is indicated. Hamstring function is influenced by sport-specific demands and previous injury, which suggests that hamstring injury risk is dynamic during in-season periods [41, 42, 46–49]. This is further supported by recent findings of substantially reduced resilience by previously injured hamstrings to cope with the physical demands of sport up to 2 years post-injury [41]. Single preseason or one-off RTS criterion testing of hamstring strength is unable to evaluate possible in-season fluctuations in hamstring function and increased injury

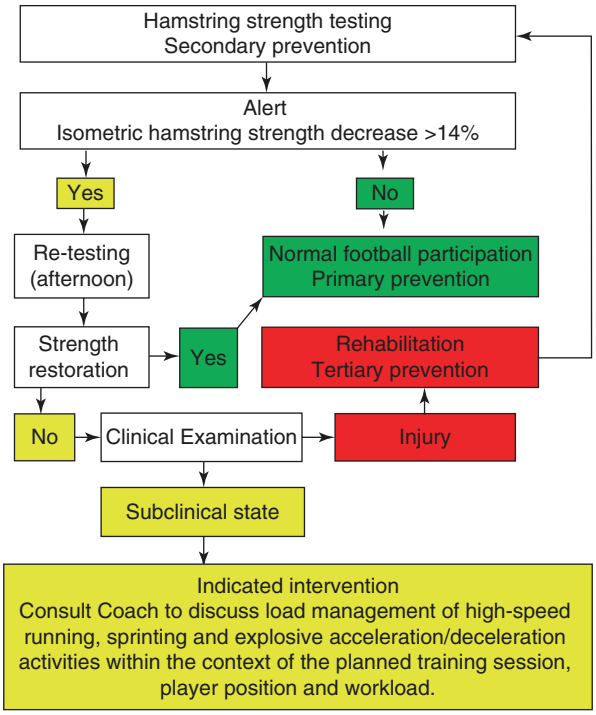

Fig. 11.3 Athlete monitoring process of isometric hamstring strength reductions as part of secondary and tertiary prevention strategies [51]

susceptibility. RTS and preseason testing are therefore best served for baseline measures and preseason identification of athletes required to commence secondary or tertiary prevention. A recent systematic review recommended testing hamstring strength post competition (match play) to identify functional impairments to assist in individual athlete management [50]. Since no difference in isometric or eccentric magnitude of change post competition was found, isometric hamstring testing has been put forward as the safer option [50]. Potential test options and their respective MDC 95%CI have been outlined in the clinical assessment chapter (Chap. 9) for practitioners' consideration. In-season monitoring of hamstring strength in athletes that never had a hamstring injury is a secondary prevention strategy. It involves a two-step clinical screening process that occurs in the subclinical stage of injury (Fig. 11.3). It is implemented in-season to facilitate early detection and management of hamstring injury susceptibility in elite athletes [48, 49, 51]. The same in-season monitoring process specifically for previously injured athletes occurs in tertiary prevention. Considering the elevated susceptibility of hamstring injury recurrence, associated with RTS in the short term and previous injury history in the long term, continuous athlete monitoring of hamstring strength is indicated as part of a tertiary prevention strategy during and beyond all three phases of this RTS process.

11.7 Conclusion

RTS after hamstring injuries involves multidisciplinary expertise collaborating to reach a shared decision about the case-specific requirements to facilitate a successful athlete outcome. The shared decision-making process is supported by the StARRT to reflect the individual context of each case. This chapter reflects that rehabilitation is gradual and progressive which involves a graded return to modified training (participation), full training (sport) and eventually the same level of competition standards (performance). A 'three plus one' approach is introduced by the addition of tertiary prevention to seamlessly map and implement ongoing management aimed at reducing the susceptibility of re-injury after returning to sport. This RTS approach is supported by four domains that monitor athlete progressions against clinical, functional, sport-specific readiness and RTS standards. Ongoing monitoring after hamstring injury is recommended to track functional and performance impairments which typically persist and possibly contribute to elevated susceptibility to reinjury after RTS.

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12 Optimising Hamstring Strength and Function for Performance After Hamstring Injury

Anthony Shield and Matthew Bourne

12.1 Introduction

One major premise of the current chapter is that high levels of sport-specific fitness and strength will likely be associated with a reduced risk of non-contact injury. This argument is supported by observational studies which link higher volumes of training with a lower incidence of sports injuries (e.g. [1–3]). Furthermore, there is some evidence that stronger, faster [4] and fitter [5] athletes are more resistant to the injuries associated with high workloads and load 'spikes'; the latter of which are often experienced with hurried returns to competition. Obviously, effective strategies for enhancing athlete fitness do not focus specifically on the hamstrings. However, another premise of this chapter is that there are some persistent deficits in neuromuscular function after moderate to severe hamstring strain injuries (HSIs) [6], and these deserve some attention during rehabilitation and even after the return to sport (RTS). It has been proposed that neuromuscular inhibition of previously injured hamstring muscles may account for the persistence of deficits in sprint performance, eccentric weakness, muscle atrophy and short fascicles despite adherence to conventional rehabilitation programmes.

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12.2 Deficits in Neuromuscular Function After Hamstring Strain Injury

A number of the neuromuscular deficits associated with prior hamstring injury persist through conventional rehabilitation and remain evident well after the return to full training and competition schedules [6]. For example, deficits in the horizontal ground reaction forces of sprinting have been revealed in athletes well after their return to play [7, 8], sometimes as much as 1 year post-injury [8]. Furthermore, a history of hamstring injury is also associated with a greater loss of horizontal ground reaction forces during repeated 6-s sprints [8]. There are also reports of eccentric weakness [9–11] and reductions in rate of torque development [12] in athletes ~1–36 months after injury despite a full RTS. Lee and colleagues [10], for example, reported ~10% and ~13% deficits in peak eccentric knee flexor work and torque, respectively, in athletes who had incurred grade 2 or 3 injuries 19 ± 12.5 months prior to isokinetic testing. There has also been a report of deficits in biceps femoris long head (BF_{LH}) muscle volume 5–23 months post-injury [13]. Finally, previously injured biceps femoris (BF) muscles also have shorter fascicles than uninjured muscles after the RTS [14, 15], and these deficits persist from one season to the next and are not normalised by preseason training in elite Australian footballers [14]. It is important to acknowledge that these deficits are revealed by comparisons between previously injured and uninjured limbs, and the retrospectivity of these observations prevents the firm conclusion that these are the result of injury. As a result, it might be argued that these between-limb differences predated original injuries.

The persistence of inelastic scar tissue, described more fully in Chap. 2, is another long-term detrimental consequence of muscle strain injury. This fibrous tissue may persist for months to years [13, 16] and increase strain in the adjacent portions of the muscle [17], thereby increasing the risk of injury recurrence. It is also plausible that unrecognised risk factors or a complex interaction of risk factors may persist through rehabilitation and thereby contribute to injury recurrence [18].

12.2.1 Do Neuromuscular Deficits Contribute to Injury Recurrence?

While many of the commonly cited neuromuscular risk factors for HSI and recurrence are not well-supported [19, 20] (see also Chap. 4), it is possible that deficits in strength or fascicle length may still contribute to a heightened risk of injury recurrence via interactions with other factors such as age and previous injury [21, 22] as discussed in Chap. 5.

12.2.2 Why Do These Deficits Persist?

Regardless of whether or not neuromuscular deficits are caused by HSI, their persistence might be interpreted as evidence of absent or inadequate rehabilitation.

Parameters such as ground reaction forces in acceleration, strength and muscle fascicle length are all trainable. However, despite rehabilitation (the details of which are often not reported in retrospective studies), many of these deficits persist in elite and sub-elite athletes who have returned to full training and competition, sometimes for more than a year. Presumably, this level of competition requires adherence to reasonably effective training programmes, and there has been no convincing explanation as to why fascicles stay short, muscles remain atrophied and ground reaction forces diminished many months and sometimes years after HSI.

We have proposed that neuromuscular inhibition, initially induced by muscle pain and isolated to previously injured muscles, may sabotage hamstring rehabilitation and contribute to the relative permanence of maladaptations after moderate to severe strain injuries [23, 24]. Fig. 12.1 shows a theoretical model (adapted from Fyfe et al. [23]) that has been modified to include the possible effects of fatigue created by repeated sprint running. This inhibition reduces muscle activation during eccentric contractions, which would otherwise provide a powerful stimulus for positive adaptations such as strength gain and fascicle lengthening. For example, Bourne and colleagues [25] employed functional magnetic resonance imaging (fMRI) to show that the previously injured BF_{LH} was ~30% less active than the uninjured homologous muscle during the Nordic hamstring exercise (NHE), after the full RTS and 2–24 months after injury (mean = 9.8 ± 8.7 months). There is also evidence that the BF surface electromyogram (sEMG) in maximal eccentric actions is lower, when normalised to the maximal concentric sEMG, in limbs with a history of hamstring strains than in uninjured contralateral muscles 2–18 months after injury (mean = 5.3 months) [11]. Furthermore, a pilot study employing twitch interpolation suggests that hamstring voluntary activation after injury is reduced during maximal eccentric, but not maximal concentric knee flexor contractions [26]. This reduction in activation may also be evident during running [27] and might explain the persistence of short BF_{LH} fascicles in previously injured athletes [14, 15] despite the use of otherwise effective training methods. The relative permanence of these maladaptations is consistent with the chronic nature of central nervous system responses to muscle pain that have previously been reported (e.g. [28, 29]).

While some aspects of the neuromuscular inhibition model have been supported, there is currently no direct evidence that it explains high hamstring injury recurrence rates. Further work is required to show that reversing neuromuscular inhibition also results in restoration of normal fascicle lengths and a marked reduction in injury recurrence rates.

Not all studies have reported neuromuscular inhibition after HSI. Blandford and colleagues [30] assessed hamstring sEMG during the eccentric NHE and normalised it to that obtained from maximal isometric contractions in elite youth soccer players with and without a history of HSI. This study showed higher normalised BF sEMG during the NHE in injured than uninjured limbs, although a number of methodological issues prevent valid comparisons with previous findings. Firstly, normalising eccentric to isometric sEMG may well give different results to those observed when

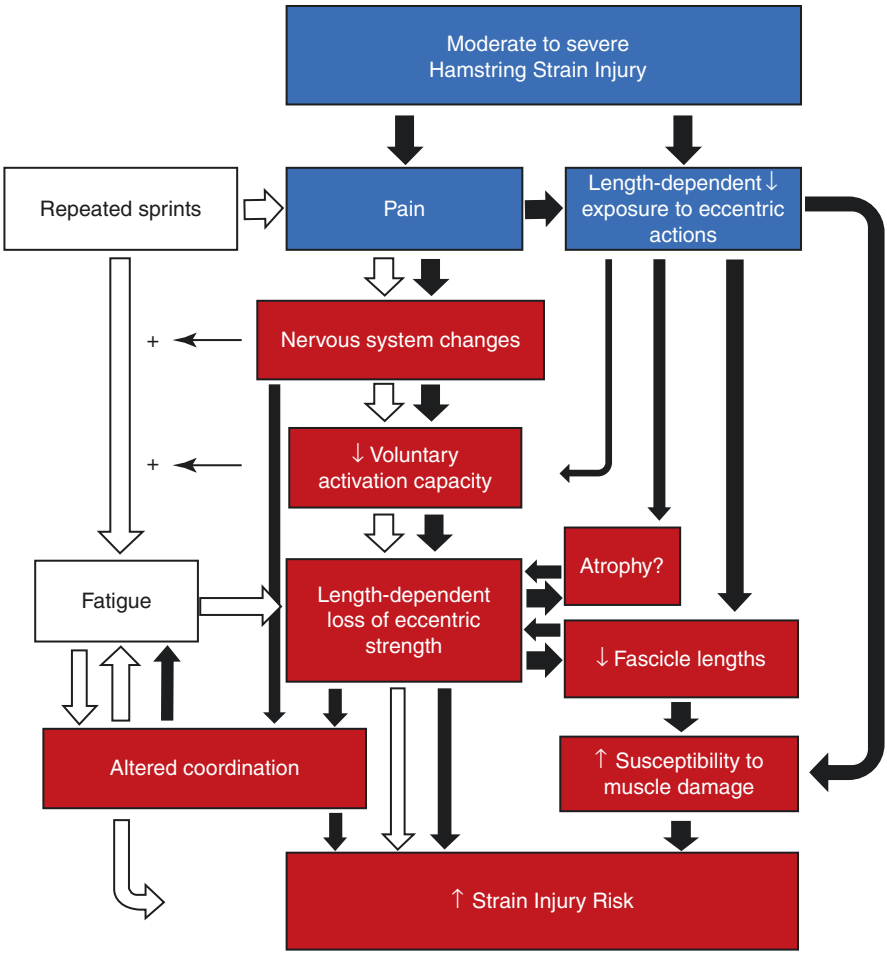

Fig. 12.1 A theoretical model that attempts to explain the role of neuromuscular inhibition in creating deficits in eccentric strength and muscle damage resistance, thereby increasing the risk of injury recurrence. The possible effects of fatigue during repeated sprinting and coordination changes have been added since original publication [23]. Unfilled boxes and arrows show the acute effects of repeat sprinting. The amplification of sprint-induced fatigue in previously injured athletes is shown with '+' symbols. Blue boxes show the transient effects of injury (lasting weeks to months). Red boxes show the chronic effects which, when caused by moderate to severe strain injury, often persist through rehabilitation and may still be present months to years after the return to sport. Altered coordination may occur at intramuscular (between hamstrings) and intermuscular (between hamstrings and their synergists) levels as discussed in Chap. 5

normalising eccentric to concentric sEMG [11]. If both eccentric and isometric contractions are inhibited, the former method would not differentiate between injured and uninjured muscles, and normalisation to concentric sEMG is based on the relative preservation of concentric strength that has been observed in a number of studies [9–11, 18]. A recent observation of persistent isometric weakness one to three seasons after HSI [31] suggests the possibility that this contraction mode is not the most

appropriate one to which others are normalised. Secondly, Blandford and colleagues [30] did not report the muscles affected or the severity of the injuries in their cohort. However, inhibition has been reported to be muscle specific [25], and it has been proposed that only moderate to severe hamstring injuries will result in lasting deficits in voluntary activation capacity [23]. In fact, recent work from one of the authors' laboratories showed no between-limb differences in eccentric or isometric strength in participants with a unilateral history of grade 1 hamstring injuries, although previously injured BF muscles did have shorter long head fascicles than the uninjured BF muscles [32].

An addition to the original neuromuscular inhibition model for hamstring injury recurrence [23] is the hypothesised interaction between the fatiguing effects of repeated sprinting and hamstring muscle activation (Fig. 12.1). As discussed in more detail later in this chapter, certain performance-related aspects of repeated sprinting decline with fatigue, and this decline seems to be greater in previously injured athletes than in those without a history of hamstring strain [33]. The new elements of the model hypothesise that repeated sprinting results in acute reductions in hamstring voluntary activation (central fatigue/neuromuscular inhibition), regardless of hamstring injury history. Reductions in maximal voluntary quadriceps activation have been reported for the quadriceps muscles after repeated 30 m sprints [34], although, as far as we are aware, no similar studies of hamstring muscle activation exist. However, Timmins and colleagues [35] have reported that repeated sprinting resulted in reductions in eccentric knee flexor strength that were associated with a decline in the BF, but not medial hamstring sEMG. Furthermore, the fatiguing effects of sprinting are not proposed to be limited to the hamstrings. A decline in the coordination of a number of lumbopelvic muscles could also potentially increase the risk of hamstring strains as discussed more fully in Chap. 5.

12.3 Addressing Deficits in Sprint Performance

12.3.1 Avoiding Spikes in Sprinting Workloads

Rapid increases in high-speed running loads are associated with an elevated risk of HSI in elite Australian rules footballers [36, 37]. These observations add to the broader literature which suggests that a gradual progression in training load assists in minimising injury risk. So, while the pressure for an early return to play after any form of injury may encourage some risk taking, consideration should be given to the benefits of a slightly delayed return as this enables the more gradual accumulation of sprint running volumes [3].

12.3.2 Ground Reaction Forces

It has been suggested that the hamstrings provide a particularly significant proportion of the horizontally oriented ground reaction force in sprinting [38], and a compelling argument has been made for monitoring these forces and emphasising this

aspect of performance during hamstring rehabilitation and after the RTS [7, 39, 40]. Furthermore, relatively inexpensive technologies (iPhone, timing gates or Rader gun) have been found to be valid ways of estimating these forces and other aspects of the force-velocity profile of sprinting [41, 42].

The optimal methods for improving horizontal ground reaction forces after HSI are not yet known, although an argument can be made for resisted sprinting such as sled pulling. A systematic review by Alcaraz and colleagues [43] suggests that while both sled pulling and unresisted sprinting are effective at improving acceleration phase sprint performance, sled training is not superior to conventional (unloaded) running. However, a majority of sled training studies have employed light loads (<20% body mass) and assessed sprint performance rather than ground reaction forces [43]. Morin and colleagues [44] have recently made a case for the benefits of unconventionally heavy (80% of body mass) sled training in improving force application and acceleration capacity in uninjured soccer players. These authors observed slight benefits in favour of sled training over conventional unloaded sprint training in terms of maximal force application, maximal power and the direction of force application (it became more horizontal) after 8 weeks of training. Sled training sessions in this study involved five to eight 20 m sprints twice each week, in training segments that took approximately 21 min to complete, so this form of training appears to be particularly time efficient [44]. Further research on the optimal means of improving force application to the ground, particularly in previously injured athletes, is warranted.

12.3.3 Repeated Sprint Ability

As mentioned previously, Australian rules footballers with a history of hamstring injuries show greater losses in horizontal ground reaction forces, on the side of injury, during repeated treadmill sprints (ten repetitions of 6-s sprints interspersed with 24-s of jogging) [8]. Previously injured limbs exhibited ~13% reductions in horizontal ground reaction forces, while the contralateral limbs and both limbs of control players exhibited ~3% drop-offs [8]. Roksund and colleagues [45] showed that the decline in repeated 20 m sprint performance across eight repetitions (with 30 s recovery) was greater in soccer players with a history of hamstring injury in the previous 2 years than in players without injury in that time. However, athletes with a history of hamstring injury in this study were faster during the initial 20 m sprints than control participants and despite exhibiting greater rates of fatigue they ran their final sprints in a virtually identical time to that of the control players [45].

Until relatively recently, there had been little research regarding the optimal training methods to improve repeated sprint ability [46]. The fatigue experienced during repeated sprints is likely mediated by depletion of energy substrates, deficits in aerobic and anaerobic metabolism and the build-up of waste-products such as inorganic phosphate and the hydrogen ion [47, 48]. However, recent observations suggest that an inability to fully activate the working muscles, presumably as a consequence of central fatigue, may be another important factor limiting performance during this type of activity [34, 49]. Because repeated sprint ability depends on both sprint performance and the ability to recover between sprints, it is sensible to prioritise the

development of both of these factors [46]. Maximal running velocity can be developed via a combination of specific sprint training (short sprints separated by recovery periods of three or more minutes) [50] and strength and power training. Given the major role of the hamstrings in generating horizontal velocity [38], interventions aimed at improving strength, power and activation of these muscles and their synergists, particularly after injury, may be important in improving sprint performance. It has been argued that fatigue resistance during repeat sprint efforts is best improved via high-intensity (80–90% VO2 max) interval training [46]. This type of training, with work to rest ratios >1, has been shown to simultaneously improve aerobic fitness [51], phosphocreatine resynthesis [52] and H^+ buffering capacity [53], all of which potentially limit performance during repeated sprints.

Running protocols designed to simulate the demands of soccer matches result in significant acute reductions in knee flexor strength, particularly in eccentric actions [54–56]. It has been proposed that these declines may contribute to the increasing likelihood of HSIs across each 40–45 min 'half' in rugby [57] and soccer [58]. There are a small number of training studies that have been shown to reduce this running-induced decline in eccentric strength. Small and colleagues [56] reported that eccentric knee flexor strength loss (after 90 min of the SAFT running protocol) was significantly reduced after an 8-week period of eccentric strength training with the NHE. However, this effect was noted when the NHE exercises were performed after, but not before, on-field soccer training sessions [56]. More recently, Matthews and colleagues [59] demonstrated that 4 weeks of eccentric NHE training with strength (5 sets of 4 repetitions) and strength-endurance approaches (5 sets of 12 rubber band-assisted repetitions) had similar protective effects against the strength loss induced by a 45 min intermittent running protocol. Delextrat and colleagues [60] further investigated the effects of the two different approaches on strength loss induced by a 90 min running protocol in female soccer players. Ten players were randomly allocated to a strength training programme (3–5 sets of 6 repetitions with 3 min between sets), and 11 were assigned to a strength-endurance programme (3 sets of 12–20 repetitions with 45–90 s between sets), with all performing the seated leg curl and stiff leg dead lift over 7 weeks. In this study, only the strength-endurance approach resulted in reduced strength loss after running [60].

While the repeated sprinting demands of field and court sports are most specifically improved by running programmes, heavy resistance training may also contribute positively to the maintenance of eccentric strength during repeated sprinting. It is not currently clear whether strength-oriented (high intensity, low repetition) or strength-endurance-oriented (moderate intensity, medium to high repetition) training is optimal for this purpose; however, both approaches may work when purely eccentric or eccentrically biased exercises are employed.

12.3.4 Sprint Running Technique

As discussed in Chap. 5, there is now a small amount of evidence that aberrant lumbopelvic kinematics, in the form of elevated anterior pelvic tilt and lateral trunk flexion, are associated with a heightened risk of HSI [61]. However, it is important

to recognise that this evidence comes from a prospective trial with 29 soccer players and just 4 prospective injuries [61]. So, while these results are promising, more work is required to show that these findings are robust.

Schuermans and colleagues [61] also compared 30 soccer players with previous HSIs with 30 control participants and reported that there were no discernible differences in the sprint kinematics observed between 15 and 25 m of maximal sprinting. These findings are seemingly at odds with another study in which nine Gaelic footballers with a history of hamstring injury exhibited greater anterior pelvic tilt, hip flexion and medial knee rotation during treadmill running (at 20 km. h^{-1}) than eight control athletes [27]. Unfortunately, neither study reported the severity of the previous injuries or the muscles in which they occurred, and it is possible that some were quite minor given the 7-day [61] and 2-day [27] minimum times for RTS which were employed to classify injuries. Nevertheless, it remains possible that prior hamstring injury may not always lead to lasting changes in the kinematic variables that have thus far been examined. Furthermore, coaches and clinicians, who might most often use the 'naked eye' to assess running technique, may not be able to reliably 'see' small changes of the sort reported by Daly and colleagues [27].

If lumbopelvic kinematics do contribute to hamstring injuries, the next great challenge for clinicians, coaches and researchers lies in determining the best methods for improving them. Optimising running technique may also be particularly challenging for athletes outside of track and field who typically have limited time to devote to such endeavours. The prospective study by Schuermans and colleagues [61] suggests that for athletes with excessive anterior pelvic tilt, a more upright trunk and pelvis position may help reduce hamstring injuries. However, it is not known whether excessive anterior tilt is indicative of poor strength, inadequate mobility or poor coordination and for now, training programmes may need to address all of these factors. Finally, it should be acknowledged that the evidence base for the role of kinematics in hamstring injury is scant, and there is considerable room for further research [62].

12.3.5 Sport-Specific Running Requirements

Athletes in ball sports frequently run at near-maximal speeds while twisting their trunks and turning their heads to observe the path of a ball or an opponent or to pass and receive a ball. Furthermore, hamstring injuries are sometimes observed when footballers flex at the hip and trunk to catch an imperfectly delivered pass [63]. Clearly, an appropriate focus on sport-specific conditioning (small-sided games or well-designed drills) will expose athletes to some high-speed running while twisting and/or stooping, although the total volume of these movements may not represent an adequate training stimulus. Running with a significant forward stoop (while paddling a ball along the ground) was one 'drill' in an apparently successful non-randomised intervention study by Verrall and colleagues [63] designed specifically

for Australian Rules players. The combination of an increase in the volume of high-speed running (and a reduction in slower longer distance runs), hamstring stretching in a fatigued state and the ball paddling drill (used twice per week for 5 min each time) was shown to reduce the hamstring injury rate in one club from 27 in the two seasons prior to the intervention to 8 in the two subsequent seasons [63]. The design of this study, with its multiple interventions, prevents conclusions as to the effectiveness of each element of the programme. As a consequence, more research is required to establish that stooped running, as employed while paddling a ball, can reduce hamstring injury rates.

12.4 Addressing Deficits in Strength and Muscle Architecture

While post-rehabilitation conditioning for athletes need not focus unduly on previously injured muscles or on the aforementioned deficits (e.g. [64]), it is possibly advantageous to include some exercises and drills that effectively target them [21, 22]. Deficits in strength and fascicle lengths have been discussed in this and previous chapters, and there is an understandable inclination to address these specifically after the RTS. It should be noted, however, that the highest level of evidence comes from randomised controlled trials (RCTs), not the studies that have, inconsistently, shown associations between strength and hamstring injury rates. Furthermore, an association between BF fascicle lengths and injury rates have, at the time of publication, only been observed in a single study [22], which needs replication. As a consequence, the remainder of this chapter deals more generally with exercise selection for the hamstrings and other lumbopelvic muscles, addressing inhibition and the argument for including exercises with an eccentric bias.

12.4.1 Exercise Selection

A growing body of research has highlighted the heterogeneity of hamstring activation patterns in different tasks [25, 65–70] and the nonuniformity of muscle adaptations to different exercises [67]. In theory, this evidence should provide a framework for selecting exercises to induce specific adaptations in target muscles (or portions of those muscles) to reduce the risk of injury and enhance performance. However, this work appears to have had little influence on clinical exercise guidelines for hamstring injury prevention [71, 72] or rehabilitation [73, 74]. Understanding muscle activation and adaptation patterns in response to common hamstring exercises allows for specific targeting of individual hamstring muscles and their synergists with resistance training exercises. However, it must be recognised that none of the findings regarding muscle activation patterns constitute evidence for the efficacy of

any particular exercise as a means of preventing injury or improving performance. Randomised controlled trials are needed before we can confidently state that any particular exercise or combination of exercises is effective.

12.4.1.1 Methodological Issues in Assessing Muscle Activation

Skeletal muscle activation is an important determinant of the structural adaptations caused by strength training [75–77]. Studies of hamstring muscle activation patterns have employed either sEMG or fMRI to map the acute electrical or metabolic activity of the hamstrings in different tasks. Surface EMG measures the electrical activity generated by active motor units via electrodes that are placed on the skin overlying the target muscles. This technique provides an indirect assessment of activation with high temporal resolution. However, a major limitation of sEMG is its susceptibility to crosstalk from neighbouring muscles [78], and this makes it impossible to completely discriminate between muscles that lie close to each other such as the long and short heads of the BF or the semimembranosus (SM) and semitendinosus (ST) [66]. Surface EMG amplitude is also influenced by the amount of subcutaneous tissue [78], motor unit conduction velocities [79] and the degree to which motor unit firing is synchronous [80]. Another rarely appreciated limitation of sEMG studies is the normalisation process. Because sEMG signals in millivolts have no real significance, these amplitudes are normalised to the sEMG signal obtained during maximal voluntary contractions (MVCs). However, the MVCs are very often performed isometrically [66], and this typically dictates an arbitrary choice of joint angles (and muscle lengths) which may not be replicated during the exercises that are examined. For example, hamstring sEMG in a range of exercises might be normalised to that observed during an isometric leg curl at a fixed knee angle [66]. This is almost certainly not a valid means of normalising the sEMG observed during a hip extension exercise or even during a dynamic leg curl across a range of motion (ROM) because the volume of muscle immediately under the electrodes will change with muscle length. The choice of different normalisation 'tasks' very likely leads to different interpretations of sEMG results, and these limitations may explain the commonly observed discrepancies between sEMG studies. The limitations of normalisation are perhaps no better demonstrated than by observations that sEMG amplitudes in dynamic or isometric exercises are frequently higher than those observed in the task to which they are normalised [66]. This suggests that some muscles are not optimally activated during the chosen MVCs. For the abovementioned reasons, we recommend that exercise prescription guidelines should not be made on the basis of sEMG studies alone.

Functional MRI is based on the observation that muscle activation is associated with a transient increase in the transverse (T2) relaxation time of tissue water, which can be measured from signal intensity changes in fMRI images. These T2 shifts, which increase in proportion to exercise intensity [81, 82], can be mapped in cross-sectional images of muscle with excellent spatial resolution [83, 84]. However, fMRI involves scanning before and after exercise, so it does not provide

insight into the timing or sequencing of muscle activation. T2 relaxation time changes are the consequence of osmotically driven fluid shifts between different muscle compartments caused by the buildup of metabolites of glycolysis. T2 changes will therefore be higher after concentric than eccentric exercise, because the former has a greater metabolic cost, even if muscle force and work duration are identical. The T2 response also varies with the duration of muscle activity and is dependent on the total work performed. As a consequence, it is problematic to compare T2 changes between exercises with different contraction modes or work durations. T2 responses to exercise are also influenced by muscle fibre type and the vascular dynamics of the active tissue [84, 85], and previous HSI is associated with diminished exercise-induced T2 changes during eccentric exercise [25]. Perhaps for these reasons, large differences in T2 changes can be observed between individuals despite them performing the same exercise with the same number of repetitions and relative intensities. It is therefore inadvisable to compare T2 changes between exercises that are performed by different participants (e.g. [86]). Despite these limitations, both sEMG and fMRI can yield valuable information on the extent and timing of muscle activation during exercise. Ideally, these observations should be verified by measurement of chronic adaptations caused by training, and this process has started in the case of hamstring exercises [67].

12.4.1.2 Nordic Hamstring Exercise

A number of studies [66, 87, 88] have established that the NHE evokes very high levels of normalised EMG (nEMG) from both the BF (72–91% of that recorded in MVC) and medial hamstring (82–102% MVC). Early work suggested that the exercise may have preferentially recruited the BF over the medial hamstrings [88]; however, more recent studies have reported higher levels of medial hamstring than BF nEMG [66, 87]. Despite preferential medial hamstring activation, it should be noted that the nEMG of BF is considerably higher in the NHE than almost any other hamstring exercise studied to date [66, 87]; however, the intensity of this eccentric exercise (>the 1Repetition Maximum (RM) for most people) is also markedly higher than the concentric-eccentric exercises (typically with 6–12RM loads) to which it is compared. Nevertheless, the level of nEMG in the Nordic exercise is particularly remarkable when compared to the 10–60% values reported for the eccentric phases of eight common hamstring exercises [66].

Functional MRI studies [25, 66, 68, 86, 89] show that the NHE involves selective activation of the ST rather than the medial hamstrings as a whole and that the short head of BF is more heavily activated than the long head. T2 changes for individual muscles and each head of the BF are shown in Fig. 12.2. It should be noted that these T2 changes closely match the increases in muscle volumes when the NHE is employed in a training programme. Bourne and colleagues [67] have reported that 10 weeks of training with the Nordic exercise resulted in relatively selective volume increases of the ST, with moderate hypertrophy of the BF_{SH} and small changes within the BF_{LH} and SM (Table 12.1). These observations suggest

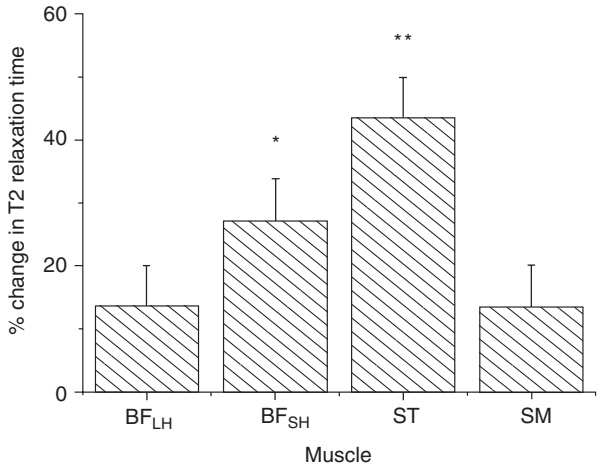

Fig. 12.2 Muscle activation in the NHE as indicated by percentage changes in T2 relaxation times after 50 repetitions of the exercise performed by healthy recreational athletes. (From Bourne et al. [25] with permission.) ∗∗ST activation was higher than that of BF_{LH} and SM. ∗BF_{SH} activation was higher than BF_{LH} and SM. Error bars depict standard errors. *BF_{LH}* biceps femoris long head, *BF_{SH}* biceps femoris short head, *ST* semitendinosus, *SM* semimembranosus

Table 12.1 Effects of 10 weeks of progressively overloaded strength training on changes in hamstring muscle volumes and the proportional contribution to whole hamstring muscle volume change made by individual muscles or muscle segments

Training exercise	Measure of hypertrophy	BF_{LH}	BF_{SH}	ST	SM
NHE	% Change in volume	5.6 ± 5.9	15.2 ± 9.8	20.9 ± 11.3	4.9 ± 6.3
	% Contribution to hamstring volume change	14	21	52	13
45° hip extension	% Change in volume	12.3 ± 7.0	8.4 ± 7.3	14.0 ± 8.4	10.4 ± 7.5
	% Contribution to hamstring volume change	29	10	33	28

From Bourne et al. [67]

that chronic training effects are also indicative of muscle activation patterns observed via fMRI.

12.4.1.3 Seated and Prone Leg Curl

Seated and prone leg curls elicit very high levels of BF and medial hamstring nEMG (>80% MVIC) [66, 87, 88]. As for the NHE, fMRI shows that the leg curl preferentially recruits the ST and, to a lesser extent, the short head of BF with lower levels of BF_{LH} and SM activation [69, 86, 90]. Ono and colleagues [69] observed selective activation of the ST during an eccentric-only prone leg curl (120% 1RM) and during a conventional prone leg curl performed at 50% 1RM. Similarly, Mendiguchia and colleagues [90] reported preferential recruitment of the ST following eccentric

prone leg curls performed at 120% 1RM. In this study, the T2 values in the ST, but not BF_{LH} or SM, remained elevated 72 h after exercise, which suggests that only ST experienced significant damage [90]. More recently, Fernandez-Gonzalo and colleagues [86] reported greater T2 shifts in the ST (65%) and BF_{SH} (51%) than the BF_{LH} (14%) and SM (~4%, but not significant) during an inertial flywheel leg curl exercise.

12.4.1.4 Supine Sliding Leg Curls

Two studies [88, 91], involving female athletes, reported very high levels of BF and medial hamstring nEMG (>100% MVIC) during the supine leg curl in which high-intensity loading is limited to the eccentric portion of the movement. In the first of these studies, Zebis and colleagues [88] observed significantly higher nEMG of BF than the medial hamstrings. More recently, Tsaklis and colleagues [91] observed no significant difference between the BF and medial hamstring nEMG in the same task. As far as we are aware, there have been no fMRI studies of this exercise, and it is not possible to state, definitively, which muscles are preferentially targeted in this movement (Fig. 12.3).

12.4.1.5 Glute-Ham Raise

There are a number of variants of this exercise, and its intensity is altered by moving the footplate closer to or further from the semicircular knee/thigh pad. Placing the knees, rather than the thighs, on the padding makes the external moment arm longer and increases the exercise intensity. Bourne and colleagues [66] examined medial and lateral hamstring activation during the eccentric portion of the glute-ham raise exercise which was performed with a long external moment arm that prevented participants from completing the concentric portion of the movement. Like the NHE, the glute-ham raise involved relatively high levels of medial (~75–80% MVC) and lateral nEMG (~60% MVC) and therefore relatively selective medial hamstring activation (Fig. 12.4) [66].

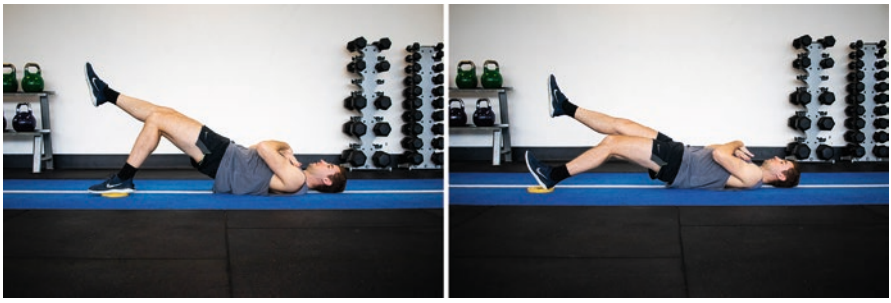

Fig. 12.3 The sliding leg curl. The sliding (eccentric phase) can be done with one or two limbs, and extra mass can be held on the hips

Fig. 12.4 The glute-ham raise exercise

12.4.1.6 Razor Curl

The razor curl, a relatively popular alternative to the NHE, involves simultaneous hip and knee extension. One variant of this exercise, performed from a glute-ham machine with the mid-thighs positioned over the padding at full knee and hip extension, has been examined in a sEMG study [92]. These authors observed greater activation of the medial (nEMG = 85%) than the lateral hamstrings (nEMG = 65%). van den Tillaar and colleagues [93] also observed higher medial than lateral hamstring involvement in the more conventional form of the exercise that was performed kneeling on a flat surface. These authors normalised the sEMG to that observed in sprint running, and this precludes a comparison of nEMG between these two razor curl studies (Fig. 12.5) [92, 93].

12.4.1.7 Forty-Five Degree Hip Extension from Roman Chair

In a recent sEMG investigation of nine common hamstring exercises, Hegyi and colleagues [94] reported that the 45° hip extension exercise (with a 12RM load) was the only task to elicit greater nEMG activity of the BF_{LH} than the ST. This is consistent with earlier work by Bourne and colleagues [66] who demonstrated that the 45° hip extension exercise involved the highest BF to medial hamstring sEMG ratio of ten common exercises. In both studies, participants performed the exercise with 12RM loads, and high absolute levels of BF (up to 75% MVIC) and

Fig. 12.5 The razor curl

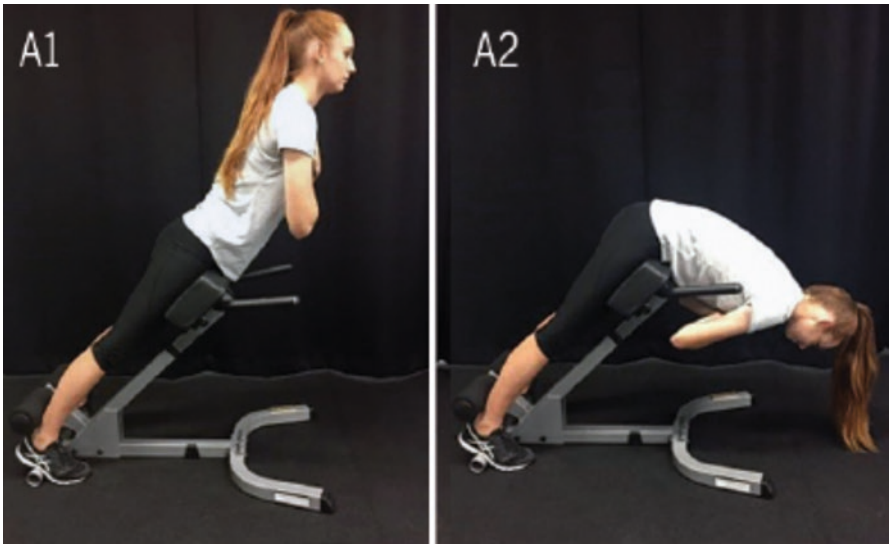

Fig. 12.6 The 45° hip extension exercise. (From Messer et al. [89] with permission)

medial hamstring (up to 61% MVIC) nEMG were observed [66, 94]. Bourne and colleagues [66] also employed fMRI to map the spatial patterns of hamstring activity during this exercise. The results of this analysis revealed that the 45° hip extension exercise involved relatively uniform activation of the biarticular hamstrings and, as expected, modest recruitment of BF_{SH}. More recently, these fMRI observations have been corroborated in a cohort of recreationally active female athletes [89]. Both of these fMRI studies [66, 89] reported that the 45° hip extension exercise elicits a significantly higher BF_{LH} to ST ratio than the NHE (Fig. 12.6).

Ten weeks of training with the 45° hip extension exercise elicits hamstring muscle volume changes [67] that closely match the acute T2 changes observed

immediately after the exercise is performed [66] (Table 12.1). The changes in BF_{LH} volume were significantly larger than those observed in another experimental group that performed the NHE over the same period [67].

12.4.1.8 Stiff Leg Dead Lift and Romanian Dead Lift

Ono and colleagues [70] reported selective nEMG of the BF_{LH} and SM relative to the ST during the eccentric and concentric phases of a stiff leg dead lift, while Zebis and colleagues [88] observed more selective sEMG activity of the medial than lateral hamstrings during a Romanian dead lift (RDL). McAllister and colleagues [95] have reported significantly higher BF_{LH} nEMG in the eccentric RDL than the eccentric prone leg curl.

As far as we are aware, there are no published fMRI studies of the RDL. However, Ono and colleagues [70] have employed fMRI to map the T2 shifts immediately after and in the days following the performance of a stiff leg dead lift. Their analysis revealed a significant increase in T2 values of the SM, which exceeded the changes observed within BF_{LH} and ST immediately after the exercise. This is, to our knowledge, the only observation of relatively selective activation of the SM over other hamstring muscles (Fig. 12.7).

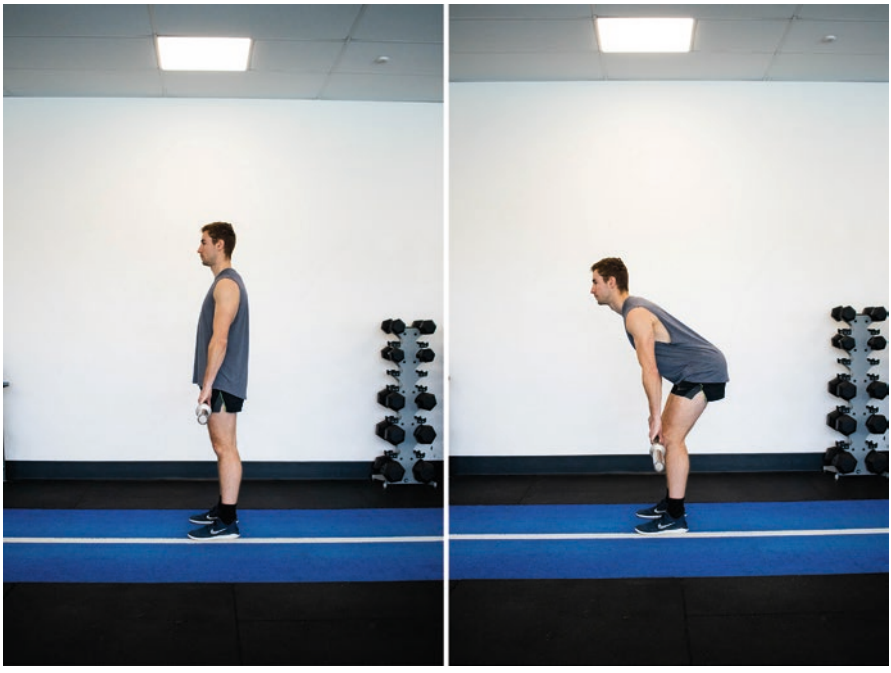

Fig. 12.7 The Romanian dead lift

12.4.1.9 Supine Bridges

The supine bridge exercise can be performed with varying degrees of knee flexion. The highest levels of hamstring nEMG have been observed when the exercise is performed with an extended knee, and this position typically results in relatively even EMG of the BF and medial hamstrings [66, 94]. These sEMG observations are in line with a recent fMRI study [65], which reported no significant difference in BF_{LH} and ST activation during the straight-knee supine bridge. This study showed that BF_{LH} was preferentially recruited over its short head and that the ST was significantly more active than the SM and BF_{SH} [65]. When performed with the knee flexed (i.e. bent-knee bridge) rather than fully extended, the magnitude of hamstring nEMG is significantly reduced, and the exercise appears to more selectively recruit the medial hamstrings (Fig. 12.8) [66, 94].

12.4.1.10 Good Morning Exercise

Recently, Hegyi and colleagues [94] demonstrated that the good morning exercise elicited the lowest levels of BF_{LH} and ST nEMG of nine common hamstring exercises performed with a 12RM load. In this study, the medial hamstrings were more active than the BF in the eccentric, but not concentric, phase of the movement. These observations are in line with earlier work by McAllister and colleagues [95]

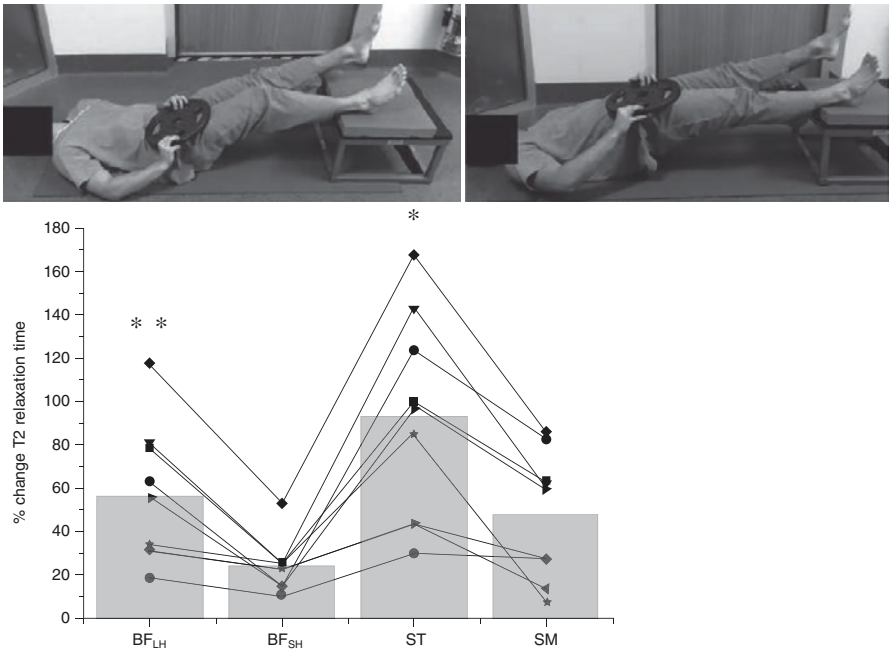

Fig. 12.8 Top: the long-levered or straight-knee bridge exercise. Bottom: the T2 changes are shown to demonstrate the significant variation between individuals in the absolute size of this response. (From Bourne et al. [65] reproduced with permission)

Fig. 12.9 The good morning exercise

who reported higher levels of medial than BF nEMG during the good morning exercise. The low levels of hamstring activation suggest that this exercise may rely relatively heavily upon other hip extensors, such as the gluteals and adductors (Fig. 12.9).

12.4.1.11 Kettlebell Swing

There are several variations of kettlebell swings; however, most are performed explosively and with relatively light loads. A recent study by Del Monte and colleagues [96] reported significantly higher medial hamstring than BF nEMG during hip hinge, squat and double-knee extension kettlebell swings. In this study, the hip hinge exercise produced the greatest magnitude of hamstring sEMG of the three variants [96]. These observations are in line with earlier work from Zebis and colleagues [88], who reported that kettlebell swings resulted in the most selective activation of the medial hamstrings out of the 14 exercises examined in that study. We are unaware of any fMRI investigations of this exercise (Fig. 12.10).

12.4.1.12 Hip Thrusts

The hip thrust is typically performed to target the synergists of the hamstrings at the hip, including the gluteus maximus (GM) and adductor magnus (AM). The exercise involves higher levels of GM than BF nEMG, and both these muscles

Fig. 12.10 The kettlebell swing exercise

Fig. 12.11 The hip thrust exercise

appear to be more active in the hip thrust than in a squat with similar relative loads (10RM) [97]. The hip thrust has also been reported to involve higher levels of GM activity and lower levels of BF activity than the conventional barbell dead lift [98]. As far as we are aware, fMRI techniques have not been employed to assess the muscle activation patterns of the hip thrust (Fig. 12.11).

12.4.1.13 Squats, Leg Press and Lunges

Squats, leg press and lunges all involve simultaneous hip and knee extension with similar ranges of movement at the hip and knee joints. As a consequence, they do

not involve significant hamstring (or rectus femoris) length changes. For example, Jonhagen and colleagues [99] have reported that there is no significant active hamstring lengthening (eccentric action) in either the walking or jumping lunge variants.

Surface EMG studies of the hamstrings during squats have reported widely discrepant levels of muscle activity (30–80% MVIC) [100, 101] possibly due to differences in electrode placement and crosstalk from other muscles. Functional MRI suggests that the hamstrings contribute very little during this exercise. In 1995, Ploutz-Snyder and colleagues [102] reported no significant T2 changes within the hamstrings after a conventional bilateral squat protocol involving six sets of ten repetitions with ~10RM loads performed by strength-trained men. These results were corroborated by observations of acute muscle swelling (increases in anatomical cross-sectional areas driven by fluid shifts into active muscles), which was limited to the vastii muscles and the adductors [102]. These fMRI results have now been replicated at least three times, most recently by Illera-Domínguez and colleagues [103], who observed no significant T2 increase in any of the hamstrings immediately after a flywheel-resisted squat training session. Together, these data suggest that the conventional squatting exercises are poor activators of the hamstrings regardless of whether barbells or flywheels act as the external resistances.

It is worth considering that many strength and conditioning coaches believe the hamstrings to be important contributors in the squat. Indeed, there are a number of influential leaders in the powerlifting community who advocate certain squatting techniques on the basis of their presumed ability to make better use of the hamstrings. Some athletes also mistake adductor muscle soreness in the days after squatting as evidence for hamstring involvement. Advocates for exercises with a proven ability to significantly activate the hamstrings may need to employ a significant education component to counter the view that the needs of the hamstrings are well addressed with squats.

The leg press, like the squat, involves simultaneous hip and knee extension, and these two exercises involve similar thigh muscle activation patterns. Enocson and colleagues [104] have reported no changes in the hamstring fMRI signal intensity after submaximal and maximal leg press (50%, 75% and 100% of the maximum load that can be lifted in five sets of ten repetitions) efforts performed by strength-trained men. In fact, the hamstrings fMRI signal intensity changes after leg press were almost identical to those observed after the leg extension exercise in which these muscles are antagonists [104]. Similar results have been observed with a leg press against a flywheel resistance [105].

Very low levels of BF and medial hamstring nEMG (~<20% MVIC) have been observed during lunges, even when relatively heavy loads are employed, although the exercise may selectively activate the BF [66]. An fMRI study of professional soccer players [90] reported an elevated T2 value in a single proximal slice of the BF_{LH} immediately following a session of body weight lunges;

however, in the same study, there were no statistically significant T2 changes in the remaining seven slices of the same muscle. While these data might be interpreted as evidence that lunges are effective in targeting the BF_{LH}, particularly at its proximal end, the very low nEMG amplitudes suggest that the exercise likely provides a suboptimal stimulus for improving strength or evoking adaptations in this muscle. As noted previously, there appears to be little or no active hamstring lengthening in at least two variants of the forward lunge [99], and this brings into question claims that these exercises are good alternatives to those with a proven capacity to change hamstring muscle size, architecture and strength.

The limited hamstring activity in the squat, leg press and lunge does not imply that these exercises will have no value in athlete preparation or in hamstring injury prevention. These movements involve significant activation of other hip extensors, including the GM and the adductors (particularly AM), and these muscles may 'protect' the hamstrings from excessive strain during high-speed running [106]. Furthermore, numerous studies have reported correlations between squat strength and 5–40 m sprint performance [107–109], while others have shown that squat training results in improvements in short sprint performance (e.g. [110]).

12.4.2 Functional or Effective?

It is often argued that exercises performed in training should, whenever possible, closely resemble the movement patterns performed in competition because this should maximise the 'transfer' of benefits. Many use the term 'functional' to describe such exercises, despite the fact that it is not well-defined. Some devotees of functional exercise also argue, despite level 1 evidence to the contrary, that the NHE will be relatively ineffective at reducing injury rates because the exercise is not sufficiently specific to high-speed running. This argument completely ignores the role of structural factors (muscle and tendon adaptations) that also have the potential to influence injury susceptibility. Exercises that isolate the hamstrings have a proven capacity to alter muscle architecture [67, 111–117], change the expression of collagen at the muscle-tendon junction [118] and stimulate substantial and selective hypertrophy [67], and these and other adaptations may reduce injury risk. It might therefore be said that these hamstring exercises are structural and, we argue, that structure also matters! The idea that exercises must be specific to running (in terms of posture, movement velocity, laterality and ROM) to be effective in preventing injury is clearly not supported by the evidence at hand [119–121]. Furthermore, very few appear to fully understand the significant limitations of the research that has examined the concepts of specificity. Typically, these studies have explored the impact of training method X on the performance of another task (task Y) such as a vertical jump or 30 m sprint over a period of 6–12 weeks in previously untrained people or recreational-level

athletes. The brevity of these interventions limits the contribution of structural adaptations and exaggerates the role of neural factors, particularly improved coordination and technique, which are extremely task specific. Furthermore, these studies almost never combine training methods (e.g. method X plus sprint training), and the impact of the combination is not observed. In contrast, athletes always combine multiple training methods and train for many months of the year.

It must be acknowledged, however, that as yet untested exercise interventions involving different exercises or high-speed sprinting may one day prove to be equally or more effective than those previously examined. Furthermore, even if alternative interventions are less effective in RCTs, they may end up having a more positive effect on injury rates in sport if they are more widely adopted [122]. At the time of writing, however, the level 1 evidence for injury prevention is limited to isolated knee flexor exercises [119–121, 123]. Future work, examining the impact of alternative exercises (or combinations of exercises) and additional high-speed running, seems warranted.

12.4.3 Exercise Selection for Hamstring Rehabilitation

So how might current findings be used after the RTS? As the previously injured BF_{LH} may be atrophied many months after the RTS [13], it might be advantageous to employ a 45° Roman chair hip extension exercise (or similar) to counter this. Stretch-related hamstring tears seem to selectively impact the SM [124], and these typically take a long time to recover [124, 125]. The limited evidence at hand suggests that the stiff leg dead lift may be a particularly appropriate exercise to target this muscle [70]. Indeed, the study of the stiff leg dead lift by Ono and colleagues [70] is, to our knowledge, the only one in which the SM is reported to be more active than the other hamstrings.

We should also consider the possibility that targeting one or more of the hamstring muscles might reduce the injury risk to others. A case has been made that a high relative reliance upon or ‘use’ of the ST protects against hamstring strains [126], which predominantly occur within the BF muscle. Unfortunately, we do not yet know how to alter the relative reliance upon different heads of the hamstrings, although we do know that the ST is selectively targeted with knee flexion exercises [66, 67, 86, 89], and these have already been shown to significantly reduce hamstring injury rates [119–121, 123].

As discussed in Chap. 5, the potential role of the GM and AM muscles in hamstring injury prevention has been recognised [61, 106]. Modelling of sprint running suggests that if these hamstring synergists are poorly activated in the late swing phase of gait, the BF_{LH} will experience higher than typical strains [106]. These findings support the argument that training should have a broader focus than hamstring exercises. For example, movements such as the barbell hip thrust and short- and long-lever bridges have been employed by Mendiguchia and colleagues [127] in

their rehabilitation RCT, and these seem to be logical inclusions in an ongoing strength programme. Further work is required to determine the effectiveness of specific hip extensor exercise interventions on hamstring injury rates.

12.4.4 Strength Deficits

The optimal methods for reversing deficits in voluntary hamstring activation and strength after HSI are not known. However, it has been proposed that high-intensity resistance training, particularly with an eccentric emphasis, is likely appropriate [23] because of its powerful positive effects on voluntary muscle activation, hamstring fascicle length [67, 112–114] and injury recurrence rates [120, 121]. It is also worth noting that many of the successful published rehabilitation programmes in recent years have a significant component of eccentric hamstring strength training [125, 127–130] and an emphasis, at some stage in their progressions, on exercises performed at relatively long hamstring muscle lengths [127, 128, 130]. As a consequence, a continued emphasis on eccentric hamstring strength, as a part of a multifaceted sport-specific fitness programme [131] appears sensible.

12.4.5 Contraction Mode Emphasis

The injury prevention benefits of eccentric hamstring training are well-evidenced, although the RCTs in this arena have been largely limited to the NHE [120, 121] (see Chap. 6). The clinical utility of the Nordic exercise is significant because no equipment is required for its implementation; however, there is a deficit of evidence regarding alternative exercises and different approaches to injury prevention and RTS after injury. It has recently been argued that hamstring exercises need not be eccentric for them to be of benefit in injury prevention programmes [132, 133]. Van Hooren and Bosch [132, 133] suggest that high-intensity isometric strength training may be of equal or even greater benefit, although there are currently no isometric intervention studies to support this claim. Given the increasing use of isometric methods in sport, there is a pressing need to establish their impact on injury risk and athletic performance. It should also be acknowledged that conventional resistance training, involving mostly concentric and eccentric actions, will form the mainstay of resistance training programmes for most athletes. Furthermore, when significant excursions (long hamstring lengths) are involved, conventional hip extension strength exercise does stimulate increases in eccentric strength and BF_{LH} fascicle lengths [67]. These findings suggest the possibility that purely eccentric or eccentrically biased strength training [120, 121, 123] may not be the only beneficial options available. However, at the time of writing, these are the only approaches with a strong evidence base.

12.5 Conclusion

Persistent deficits in horizontal ground reaction forces and repeated sprint performance suggest that there may be value in monitoring these parameters and addressing them in a sport-specific manner after hamstring injury. Neuromuscular deficits such as reduced voluntary activation and eccentric strength and short hamstring muscle fascicles are, arguably, well-addressed by sport-specific fitness programmes which include heavily loaded hip extensor and knee flexor exercises. There is now significant evidence showing how different exercises can target individual hamstring muscles and their synergists at the hip. Eccentrically biased (the NHE and flywheel leg curl) and conventional strengthening exercises (Roman chair hip extension and RDL) that involve the hamstrings being loaded at long lengths are likely beneficial.

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When Hamstring Injury Rehabilitation Fails

13

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13.1 Introduction

Treatment failure can be defined as a measure of the quality of health care by assessment of unsuccessful results of management. Incidence rates of acute hamstring injuries are well documented in literature, but similar data are lacking for proximal hamstring tendinopathy (PHT). Patients with PHT experience a more gradual onset with fluctuating symptoms over time [1]. This makes it harder to

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apply the definition of treatment failure, as there is no acute onset of a reinjury as we recognise in acute hamstring injuries. For assessment of hamstring injuries, this quality of health care can be assessed with reinjury rates, patient-reported outcomes, strength and flexibility measures, or imaging outcomes. Some of the outcome measures, especially reinjury and patient-reported outcomes, are very useful, while other measures are not associated with the clinical signs of treatment failure.

The most common type of treatment failure in acute hamstring injuries is the onset of a reinjury. Other less frequent types are an inadequate rehabilitation with acute onset of pain or a prolonged rehabilitation without full recovery. Treatment failure in long-standing hamstring injuries is most frequently a result of relapse or persistence of symptoms after full rehabilitation.

13.1.1 Epidemiology of Treatment Failure

The different types of treatment failure have diverse incident rates. High recurrence rates following acute hamstring injuries have been reported in the literature. Hamstring reinjury rate is 14–63% within 2 years after the initial injury [2, 3]. It is known that 50% of these reinjuries occur within the first 50 days of return to play (RTP) [4]. This emphasises the close relationship between time to RTP and occurrence of reinjuries. Inadequate rehabilitation and insufficient criteria for return to sport (RTS) participation might be reasons for the large variation in hamstring reinjury incidence.

The other types of treatment failure are less well-described in terms of epidemiology. The onset of acute hamstring pain during conservative treatment (in most cases, this implies rehabilitation) is sometimes reported as reinjury in literature. This should, however, not be interpreted as reinjury but as inadequate rehabilitation. Consequently, part of the definition of a reinjury is that it occurs after a RTP decision has been made as a result of a rehabilitation programme. It is also less common that athletes experience prolonged symptom duration after an acute hamstring injury. This is known as a distinct clinical entity and has also been found in a recent high-quality trial in this field [5]. Epidemiological data of persisting or relapsing symptoms due to PHT are lacking. It is known that tendinopathy at other locations is treatment resistant in 60% of the cases at 5-year follow-up [6].

13.1.2 Impact of Treatment Failure

Hamstring injuries impair athlete performance, as they result in absence from sports for several weeks or months. The financial burden as a result of hamstring injuries is considerable in elite team sports. For example, in the English Premier League, the salary burden as a result of hamstring injuries reaches over € 20 million per season [7].

The impact is also evident at the individual level. Acute hamstring reinjuries and hamstring injury sequelae frequently lead to insecurity of the athletes. This might lead to kinesiophobia or decreased psychological readiness to RTP. This is even more pronounced in case of proximal tendon avulsion injuries which can be career threatening [8]. The impact of PHT is also evident but results more specifically in decreased participation in sports activities or a decreased performance with fluctuating episodes [9].

13.1.3 Causes of Treatment Failure

Treatment failure is a result of either an incorrect diagnosis or an inadequate rehabilitation. In the sections below, we will outline the potential pitfalls in diagnosing and treating hamstring injuries. This can aid the healthcare provider in managing patients with treatment failure of acute hamstring injuries, hamstring injury sequelae, and hamstring tendinopathy.

13.2 Causes of Treatment Failure in Patients with Acute Hamstring Injuries

13.2.1 Incorrect Diagnosis

In case of treatment failure after an initially diagnosed acute hamstring muscle injury, other possible causes of acute posterior thigh pain should be considered. Table 13.1 provides an overview of these. These causes are divided into hamstring muscle-tendon-bone complex injuries and causes from other anatomical structures. In patients with the clinical picture of acute hamstring injuries that fail to respond to rehabilitation, other hamstring muscle-tendon-bone complex injuries should be considered as cause. These specific diagnostic considerations are described more in detail in the section below.

13.2.1.1 Tendon Avulsion Injury

A tendon avulsion is a severe type of acute hamstring injury, characterised by complete de-attachment of one or more hamstring tendons from the bone. It usually involves avulsion of the proximal tendon(s) from the ischial tuberosity, but distal tendon avulsion may also occur. Although older people are more prone to avulsions, these injuries are also observed in younger athletes.

Hamstring tendon avulsions are relatively rare, associated with a prolonged recovery, and may lead to persistent functional impairments [8, 10]. Due to the rarity of this injury, it is often missed at initial diagnosis, resulting in a diagnostic delay and insufficient initial management.

The injury mechanism typically includes a sudden forceful hip flexion and knee extension, such as gliding over a slippery surface or water ski accidents. Recently,

Table 13.1 Differential diagnosis of posterior thigh pain

Causes of posterior thigh pain	
Hamstring muscle-tendon-bone complex-related causes	
Acute onset	Indirect muscle injury/muscle strain Direct muscle injury/muscle contusion Tendon avulsion injury Ischial tuberosity apophysis avulsion fracture Reactive tendinopathy – PHT – Distal biceps femoris (BF) tendinopathy – Distal semimembranosus/semitendinosus (SM/ST) tendinopathy
Gradual or insidious onset	Tendinopathy – PHT – Distal BF tendinopathy – Distal SM/ST tendinopathy Traction apophysitis of the ischial tuberosity Myositis ossificans
Causes from other anatomical structures	
Neural	Radiculopathy Peripheral nerve entrapment – Posterior cutaneous nerve of the thigh – Sciatic nerve
Vascular	Iliac artery endofibrosis Thrombophlebitis Deep venous thrombosis Post-thrombosis syndrome
Bone	Bone tumours Femoral stress reaction/fracture
Other muscle injury	Adductor magnus (AM) Gastrocnemius medial/lateral head
Joints	Referred pain from the following: – Sacroiliac joint – Hip joint – Knee joint
Bursitis	SM Ischiogluteal
Other	Chronic compartment syndrome of the posterior thigh Ischiofemoral impingement syndrome

an alternative injury mechanism that also involves a considerable hip abduction component (hip flexion-abduction injury mechanism) has been described [11].

In case of a missed avulsion injury, patients often report specific symptoms, even after a period of rehabilitation. These symptoms include persistent hamstring muscle weakness, difficulties in coordinating hip and knee movements, and/or sensory perception in the distribution field of the sciatic nerve. In some patients, the main symptom is persistent pain at the ischial tuberosity during sitting, which is a disabling symptom for patients in their daily living. At physical examination, there is often marked hamstring muscle atrophy and loss of hamstring strength. Some patients are even unable to contract their hamstring muscle. On palpation, the hamstring tendons may not be felt by the examiner due to muscle retraction. Sensory

symptoms may occur in the sensory distribution area of the sciatic nerve as a result of the hematoma formation or due to adhesions. Imaging can confirm this diagnosis, and magnetic resonance imaging (MRI) is most frequently used in the clinical setting as this modality is able to depict which of the three hamstring origin sites has been ruptured (Fig. 13.1).

In both short-living and long-standing cases, there may be an indication for surgical fixation of the avulsed hamstring tendon at the original insertion (see Sect. 13.5.2).

13.2.1.2 Ischial Tuberosity Apophysis Avulsion Fracture

Adolescents are prone to avulsion of the ischial apophysis instead of tendinous proximal hamstring avulsion, especially between the ages of 14–18 years. In any adolescent or young adult with severe (proximal) hamstring pain or difficulties in activating the hamstring muscle after acute injury, an ischial apophysis avulsion should be considered. Plain X-ray can identify a displaced avulsion fracture. In case of a high clinical suspicion and a negative X-ray, a computed tomography (CT) scan or MRI should be considered, as in our experience non- or minimally-displaced apophysis avulsions can be missed.

The literature on treatment of these injuries is limited, and there are no controlled studies comparing conservative with surgical treatment. Based on case series, it is known that with increasing displacement, consolidation of the bony fragment is less likely to occur with conservative treatment [12]. This has led to the expert opinion that larger displaced fragments may require surgical fixation. However, there is no consensus in the literature on the exact cutoff point: >1 cm [13], >1.5 cm [12], and >2 cm [14] are suggested. Other factors that are not related to the fragment displacement, such as sport-specific demands of the hamstring muscles, should be

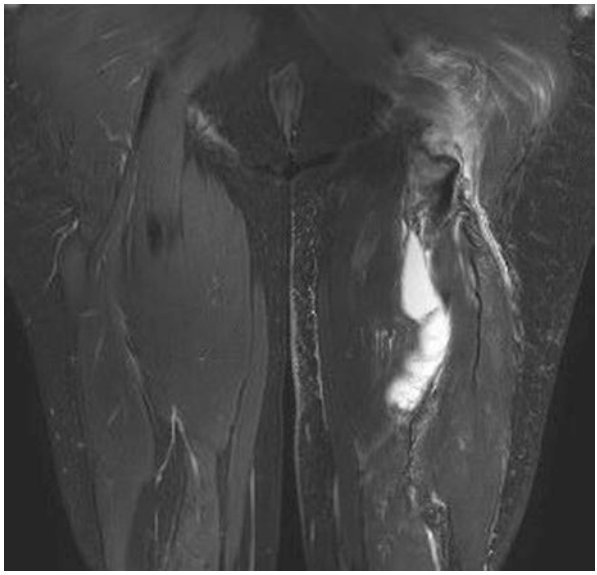

Fig. 13.1 Magnetic resonance imaging (MRI) of a tendon avulsion injury from the ischial tuberosity. Coronal STIR images demonstrating left-sided increased signal intensity distal to the ischial tuberosity. There is an avulsion of all three hamstring tendon insertions from the ischial tuberosity. Note the waviness (or "buckling") of the proximal tendon, which is indicative of a complete tendon rupture

considered in this decision-making. Conservative management consists of 8–12 weeks relative rest post-injury, followed by a progressive exercise programme. Full rehabilitation takes up to 1 year, and hamstring strength deficit may persist in the longer term (see Chap. 10).

13.2.1.3 Adductor Magnus Muscle Injury

The adductor magnus (AM) can be subdivided into two parts: (1) the pubofemoral part that originates from the ischiopubic ramus and inserts on the lower gluteal line and linea aspera and (2) the ischiocondylar part that originates from the inferior ischial tuberosity and takes an almost vertical course to its insertion on the femoral adductor tubercle [15]. The latter ischiocondylar part shares a common innervation and action (hip extension) with the long hamstrings. Adductor magnus injury can mimic a proximal acute hamstring injury, but the prognosis tends to be better. Identifying the precise location of the injury by careful palpation can help to differentiate between these conditions. The AM ischiocondylar origin can be palpated inferior and medial to the proximal hamstring tendons. Additional diagnostics with ultrasound (US) or MRI may help to confirm the diagnosis. When an AM injury has been established, rehabilitation should focus more on actions of this specific muscle group by using hip extension and adduction strengthening exercises.

13.2.1.4 Gastrocnemius Muscle Injury

Acute injury to the proximal gastrocnemius can mimic a distal acute hamstring injury. Differentiating these injuries is relatively easy by clinical examination. Strength testing of the calf muscle by resisted ankle plantar flexion and stretching the gastrocnemius with ankle dorsiflexion with the knee fully extended will provoke symptoms in case of a gastrocnemius injury. On the other hand, strength testing of the hamstrings by resisted hip extension and stretch testing using the (active or passive) knee extension test will provoke symptoms in hamstring injury and not in gastrocnemius injury. Additional diagnostics with US or MRI may help to confirm the diagnosis, especially for more difficult presentations in the popliteal region. When a gastrocnemius injury has been established, rehabilitation should focus more on actions of this specific muscle group by using knee flexion and ankle plantar flexion strengthening exercises.

13.2.2 Inadequate Rehabilitation

A number of considerations arise within the optimal planning of rehabilitation of acute hamstring injury (see Chaps. 10 and 11). Certainly, the need to individualise the approach according to past history of hamstring injury, severity of current injury, type of sport, and the athlete's aspirations remains paramount.

13.2.2.1 Planning of Rehabilitation

One of the first decisions is around balancing when to begin early mobilisation. Starting too early may compromise healing and lead to greater scar formation [16],

whereas too-late mobilisation may lead to a compromised muscle function. Järvinen et al. [16] recommend mobilising (defined as treadmill running) at or around day 5, although obviously this depends on the grade of injury and complexity of the lesion. It has to be acknowledged that broad opinion continues with respect to how aggressive to be in early management, yet it is probable that too-early mobilisation of higher-grade lesions may well compromise longer-term outcomes. Return to running within 4 days conferred significantly greater risk of recurrence than commencing running at 5 days or longer. Interestingly, this delay did not prolong RTP in the latter subjects [17].

In concert with early mobilisation, the choice and timing of supplementary exercises is paramount. Recent research suggests that an overemphasis of concentric exercises will lead to fascicle shortening [18], which will increase risk of hamstring reinjury at higher eccentric loads. Eccentric loading should be introduced early to maintain/improve fascicle lengths and ultimately functional length [2, 19]. The application of knee flexor-dominant or hip extensor-dominant loading is an important consideration which may well be influenced by the site and severity of the lesion. Eccentric exercise of hamstrings at longer muscle lengths appeared to provide a greater fascicle length change, although modest increases were also seen at shorter lengths [20]. Perhaps most importantly, progression of this stimulus is necessary to meet higher loads and functional demands of the sport.

A well-structured running programme to meet demands of the sport including distance, intensity, and acceleration is a key component. This is integral to athlete load management, modelled to RTS in a robust manner. Recent work by Stares et al. [17] presented compelling data demonstrating a more robust RTP was associated with increased volumes of high-speed running prior to resumption of competition. However, this did necessitate a lengthened RTP. This data better informs the risk-reward debate raised previously by Orchard et al. [21] around the RTP decision. These authors described a lack of agreement around robust criteria and the need to consider an increased risk of reinjury associated with earlier return.

13.2.2.2 Managing Risk Factors

As has been stated by many practitioners, history of hamstring injury now confers non-modifiable risk. Conversely, increasing hamstring strength levels and side-to-side balance [2] in the hamstring group offers perhaps one of the best opportunities (in combination with addressing fascicle length) to reduce the inherited risk of the athlete for recurrence or future injury. Considerable work has been contributed in the provision of sport-specific thresholds [18, 22].

As the first weeks of RTS carry the highest risk [4], this requires the management team to closely monitor the athlete with a preparedness to be flexible in sessional demands of the various challenges of the sport [2]. There is currently no scientific evidence that supports adjusting the length of rehabilitation in patients with risk factors. However, in our experience, modification of rehabilitation duration according to risk factors such as past medical history is recommended [21].

13.3 Causes of Treatment Failure in Patients with Ongoing Posterior Thigh Pain, Including Hamstring Injury Sequelae

13.3.1 Incorrect Diagnosis

Ongoing posterior thigh pain is often hard to manage, and other diagnoses should certainly be considered in these cases. Table 13.1 provides an overview of possible causes of posterior thigh pain. Symptoms of hamstring injury sequelae can have their origin in the hamstring muscle-tendon-bone complex or in structures that are not related to the hamstrings. These diagnostic considerations are described below.

13.3.1.1 Hamstring Muscle-Tendon-Bone Complex-Related Causes

Traction Apophysitis of the Ischial Tuberosity

Traction apophysitis occurs in teenagers prior to complete fusion of the ischial apophysis and results from repeated traction injuries on the apophysis without discrete displacement. These teenagers present with exercise-related localised pain at the ischial tuberosity which can be provoked on palpation. Scientific evidence for the effect of different treatment options is scarce; only a few randomised clinical trials have been performed in patients with an equivalent injury of the tibial tuberosity (M. Osgood Schlatter). Treatment of these traction apophysitis injuries is mainly symptomatic using load management advice. The long-term prognosis for a traction apophysitis is good, as complete recovery can be expected with closure of the ischial growth plate.

Myositis Ossificans

Myositis ossificans is a heterotopic non-neoplastic bone or cartilage formation in or adjacent to a muscle [23]. There are three different types: myositis ossificans progressiva (hereditary and severe generalised form), myositis ossificans without history of trauma (associated with burns, haemophilia, and neurological disorders), and myositis ossificans traumatica (either related to a contusion or repeated minor trauma) [24]. While traumatic myositis ossificans is more common in the anterior thigh muscles [25], it can also occur in the hamstring muscles [26]. Subjects may present with pain, swelling, and a palpable mass. The diagnosis can be confirmed with a plain radiograph (Fig. 13.2) or on US examination. Management of myositis ossificans is mainly conservative, including relative rest to control pain and inflammation, followed by gradual progressive exercise with symptom-based progression.

13.3.1.2 Non-Hamstring-Related Causes

Neural Causes

The hamstrings are innervated by the tibial branch (biceps femoris long head (BF_{LH}), semitendinosus (ST), and semimembranosus (SM)) and peroneal branch (biceps femoris short head (BF_{SH})) of the sciatic nerve. The sciatic nerve is formed from the

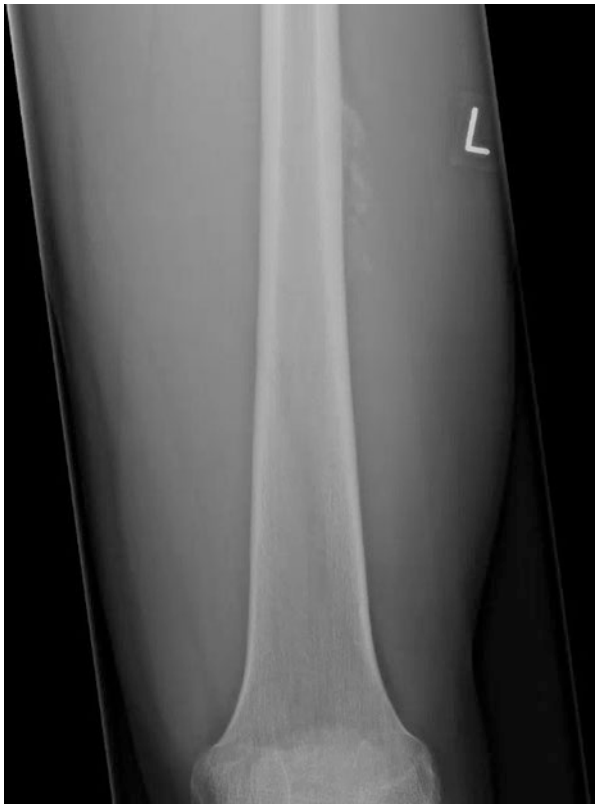

Fig. 13.2 X-ray depicting myositis ossificans. X-ray of the left femur in anteroposterior direction showing calcifications at the lateral side of the femur in an adolescent basketball player who sustained a direct trauma to the posterolateral side of the left upper leg. When these calcifications are observed in relation to a previous trauma on that location, they are specific for myositis ossificans

L4-S3 segments of the sacral plexus. Nerve damage, compression, or irritation can occur at various sites along the way, resulting in posterior thigh pain.

Compression or chemical irritation of the nerve roots in the lower back can occur. Several causes of posterior thigh pain due to nerve root compression are spinal disc herniation, degenerative disc disease, lumbar spinal stenosis, or spondylolisthesis. These causes are usually associated with other neurological symptoms, such as pain and numbness radiating distally, loss of Achilles tendon reflex, or muscle weakness (hamstring muscles, ankle evertors).

Along its pathway, the sciatic nerve can de damaged or compressed by a direct trauma or pelvic trauma [27]. Compression of the sciatic nerve by the hip external rotators has been described (often referred as "piriformis syndrome"), but this clinical entity remains controversial [28]. Other sites of sciatic compression have also been implicated [29–31].

Finally, damage or entrapment of peripheral nerves may cause pain. The skin of the posterior thigh is innervated by the posterior femoral cutaneous nerve (PFCN) via its numerous collateral branches. If symptoms of pain and altered sensation are limited to the specific distribution area of the nerve (from the posterior thigh to the popliteal fossa) and pain is exacerbated with sitting or leaning against the buttock,

then the PFCN should be considered as the source of the pain [32]. A diagnostic US-guided infiltration with an anaesthetic may help in establishing the diagnosis.

Vascular Causes

Iliac artery endofibrosis is a rare condition that may result in a reduced blood flow to the lower extremity in otherwise healthy individuals. It is most common in cyclists but has also been reported in other endurance athletes [33]. The hallmark symptomatology of this condition is leg weakness, thigh pain, and resolution of symptoms within 5 min of exercise cessation. Although the pain is usually in the anterior and lateral thigh, it may also be experienced in the posterior thigh. Exercise testing with ankle blood pressure measurements is the most appropriate way to confirm or exclude the diagnosis. Additionally, imaging (US, angiography) may be used for diagnostic purposes [33].

Other rare vascular causes of posterior thigh pain are thrombophlebitis, deep venous thrombosis (DVT) [23, 34], and post-thrombosis syndrome [35]. These venous conditions usually cause symptoms of the lower leg but sometimes may present with posterior thigh pain. Post-thrombosis syndrome is a long-term complication of DVT as a result of valvular incompetence due to damage to the venous valves. Symptoms may include pain, cramping, heaviness, itching, swelling, skin discoloration, and presence of varicose veins [35].

Bone Pathology

Bone tumours are rare but should not be missed, as delayed diagnosis can be catastrophic, especially in the case of malignant tumours. There are no specific signs that are associated with bone tumours, but night pain and increasing pain that is not associated with activities are signs that increase suspicion of this disease. A plain radiograph is the first step to detect a bone tumour. Subsequently, CT and/or MRI scanning with additional intravenous contrast media may be required.

Stress fractures of the upper thigh (femur, femoral neck) are uncommon but may present as posterior thigh pain. Athletes with high training loads are at increased risk for development of stress fractures of the upper thigh (femur or femoral neck) [36]. Training errors are the most frequent cause of stress fractures, especially a sudden increase in training load. Other risk factors include age, female sex, low bone mass, menstrual cycle disturbance, and bone metabolic disorders. Imaging is often required for confirming this diagnosis. Commonly used imaging modalities to detect stress fractures are plain radiographs, bone scans, MRI, or CT [36].

Joint Pathology

Referred pain from the sacroiliac, hip, or knee joint may present as posterior thigh pain. A careful history-taking and physical examination of these joints should be part of the differential diagnosis workup of posterior thigh pain in treatment failure after (suspected) hamstring injury sequela.

Bursitis

Near the hamstring muscle complex, there are two bursae that may provoke posterior thigh pain: the ischiogluteal bursa and the SM bursa. An ischial bursitis presents with inflammatory pain at the hamstring origin, typically when sitting on a hard surface. It can be difficult to distinguish from a PHT. The SM bursa is located posteromedial of the knee at the medial aspect of the SM tendon. Inflammation of this bursa will likewise result in inflammatory symptoms near this posteromedial region of the knee.

Ultrasound or MR imaging can visualise a bursa filled with fluid [37, 38]. The benefit of anti-inflammatory medication is limited. Corticosteroid injection into the enlarged bursa can be performed, but scientific evidence for its efficacy is absent, and these injections can lead to unfavourable complications, such as tendon ruptures and skin atrophy.

13.3.2 Inadequate Rehabilitation and Restoration of Structure, Strength, and Function

Principal considerations in this section centre around whether persisting structural, architectural, strength, control, or fatigue resistance deficits have been identified and addressed within the limited specific evidence relating to this cohort, largely due to the breadth of clinical presentations, which in many cases are multifactorial [39]. This requires a comprehensive and highly individualised approach to each case.

13.3.2.1 Altered Structural Integrity

These may include persisting deficits in the intramuscular aponeuroses and epimyseal or delaminating lesions of the tendon or aponeurosis of origin or insertion. There is conflicting opinion within the literature which variously apportions an increased rehabilitation interval, an increased likelihood of recurrence, or little significance to this aspect [40–42]. This may be due in part to the differing demands of the sport cohorts utilised or the distribution of sites. These issues may require specific rehabilitation restrictions or approaches, although this area requires further research to improve our current understanding.

13.3.2.2 Muscle Architecture

Muscle morphology, specifically shorter fascicle lengths in BF_{LH} has been described as a risk factor for hamstring injury in the literature [22]. These authors recommend remedial loading through high-intensity, supramaximal eccentric-dominant exercises such as the Nordic hamstring or loaded eccentric hip extension exercise. Timmins and colleagues provide preliminary data suggesting protective thresholds for fascicle lengths in soccer players [43]. By extension, this approach may also be worthy of further exploration in better ensuring successful hamstring rehabilitation [44, 45], at least in at-risk groups.

13.3.2.3 Muscle Strength

Normalisation of hamstring muscle strength does not occur in the majority of athletes who are clinically recovered after a rehabilitation programme [46]. This isokinetic strength deficit at RTP was also not associated with a higher risk for reinjury. On the other hand, increasing hamstring strength levels and side-to-side strength balance in the hamstring group offers an opportunity to reduce the inherited risk of the athlete for recurrence in the first period after RTP [2]. This implies that hamstring strength deficit may be a more important risk factor to determine in the monitoring phase after RTP.

13.3.2.4 Muscle Fatigue

Fatigue of muscles has long been cited as a risk factor for hamstring injury, yet evidence until recently has been limited to early animal studies [47]. More recent work suggests one of the legacies of a previous hamstring injury within 2 years is the comparative reduction in ability to sustain repeated sprint performance [48]. This was despite a lack of differences between past history and control groups in factors such as maximal speed, leg strength, power, and flexibility. Another study demonstrated that repeated sprint efforts in a previously hamstring-injured group led to specific knee flexor and H:Q ratio peak torque deficits when compared to non-injured controls [49]. This fatigue-induced deficit correctly identified the injured side in all subjects. Additionally, increased investment in higher-speed running prior to return conferred increased protection against hamstring injury recurrence [17].

13.3.2.5 Kinetic Chain Considerations

There appears to be some value in the consideration of contributing factors within the kinetic chain as well as diminishing the risks inherited through previous hamstring injury and other injuries such as knee, anterior cruciate ligament, lumbar spine, and to a lesser extent quadriceps and calf [50]. While intrinsic hamstring issues are an important mainstay of rehabilitation, it is important to also address co-contributors across the kinetic chain including trunk, hip, gluteal, and calf function. Sherry and Best suggest the importance of trunk strength and stability [51]. Others found that older players had ipsilateral hip internal rotation deficits as risk factor [52]. Additionally, contralateral hip flexor tightness [53] and hip extensor strength deficits [54] have been associated with acute hamstring injury. This should be contrasted with a recent publication, describing knee flexor rather than hip extensor deficits persisted following a history of hamstring strain injury (HSI) in a male Australian rules football population [55]. Hip extensor strength however is not routinely tested clinically or in a research setting. It would appear, when faced with management of recurrent hamstring failure, cases should be assessed holistically and managed on an individual basis with a perspective that extends where necessary beyond the hamstring muscle group.

13.3.2.6 Hamstring Muscle Activation

Changes within the hamstring muscle group in terms of activation and coordination after injury have also been postulated but are poorly understood to date.

Differential activity of individual hamstrings is described by several researchers [56–58], yet the evidence for changes from the "normal" in the hamstring-injured athlete in functional activities such as running and cutting is incomplete. Deeper understanding of activation or coordination changes within the medial or lateral hamstring groups or in synergists in normal, fatigued, and previously injured states are lacking. Bourne et al. [59] found reduced functional MRI changes specific to the BF_{LH} muscle during performance of the Nordic hamstring exercise in athletes with a unilateral history of previous HSI. Silder et al. [60] also demonstrated significant loss of BF_{LH} cross-sectional area assessed on MRI post-injury which appeared to be in part compensated for by hypertrophy of the BF_{SH}. Further insights into differences between previously hamstring-injured and control athletes performing repeated contractions are available [61]. Schuermans et al. report a propensity for the hamstring-injured group towards earlier fatigue in ST, proposing possible overloading of the BF. However, both MRI and electromyogram studies have recognised shortcomings that challenge the validity of inferring individual muscle properties across the hamstring group. Notwithstanding these open findings, it would appear that in individual cases of recurrent BF_{LH} failure, selective hypertrophy, fascicle length, activation, and synergy aspects may need to be specifically addressed.

13.4 Causes of Treatment Failure in Patients with Hamstring Tendinopathy

13.4.1 Incorrect Diagnosis

Proximal hamstring tendinopathy is a potential cause of pain in the buttock region. This condition is characterised by localised pain in the deep ischial tuberosity area, which is often worse during or after activities with hip flexion movements (such as running, lunging, and squatting). Sitting, especially on harder surfaces, often aggravates symptoms. The pain may radiate along the hamstrings to the posterior thigh [62].

Buttock pain may be caused by multiple other conditions, of which most are displayed in Table 13.2 [1]. Comprehensive examination of the lumbar spine, sacroiliac joint, and hip joint is needed to exclude other potential diagnoses. Sciatic nerve entrapment as a result of adhesions between the nerve and proximal hamstring tendon origin or in the buttock interfaces should be considered [63]. Slump testing may aid in identifying sciatic nerve involvement or referred pain, but this test lacks specificity as it might be painful in other conditions as well. Detailed palpation of the ischial and buttock areas can help in the differentiation between tendinopathy and sciatic nerve involvement. Localised pain at the ischial tuberosity is specific for PHT. More diffuse pain running from the buttock to the posterior thigh that cannot be reproduced on palpation is suggestive of neural pathology. While palpation should not be used as the sole diagnostic indicator, it will guide the clinician in the differential diagnosis.

Table 13.2 Differential diagnosis of buttock pain

Diagnosis	Key features
PHT	– Pain during or after activities with hip flexion movements – Pain during prolonged sitting – Localised tendon pain on resistance test and on palpation of the ischial tuberosity
Sciatic nerve entrapment	– Diffuse pain radiating in posterior thigh – Pain during passive hip adduction – Abnormal slump test
Piriformis syndrome	– Pain in the gluteal area with or without radiation in the posterior thigh – Pain on resisted external rotation or passive internal rotation – Pain on piriformis muscle palpation
Ischiogluteal bursitis	– Mainly pain during sitting – Pain on localised palpation of the ischial tuberosity – Ultrasound or MRI confirming diagnosis
Referred pain from the lumbar spine	– Diffuse pain in the posterior thigh and/or lower leg – Absence of injury pain during hamstring resistance tests and/or localised palpation
Ischiofemoral impingement	– Pain on palpation of the quadriceps femoris muscle – Pain on passive external rotation with the hip in neutral position – MRI confirming diagnosis
Apophysitis or avulsion	– Adolescent athlete – Injury related to overuse (apophysitis) or an acute trauma (bony avulsion injury) – X-ray confirming diagnosis (bony avulsion injury)
Posterior pubic or ischial ramus stress fracture	– History of overuse – Female athletes at higher risk – Pain on palpation over the posterior pubic or ischial ramus
Metabolic disorder, rheumatic disease, or tendon abnormalities induced by medications	– No response to usual care – Family history of hypercholesterolemia, diabetes, gout, or other rheumatic diseases – Use of specific medications (quinolones, statins)

A confirmed PHT that does not respond to therapy could also be caused by metabolic disorders [64]. Internal abnormalities, such as hypercholesterolemia, diabetes, or gout, should be considered as underlying causes. A rheumatic disorder, such as spondyloarthropathy, may have enthesitis as first presentation (Fig. 13.3). Medication is another cause of tendon pain. Use of specific antibiotics (quinolones) and statins is associated with tendon ruptures and tendinopathy.

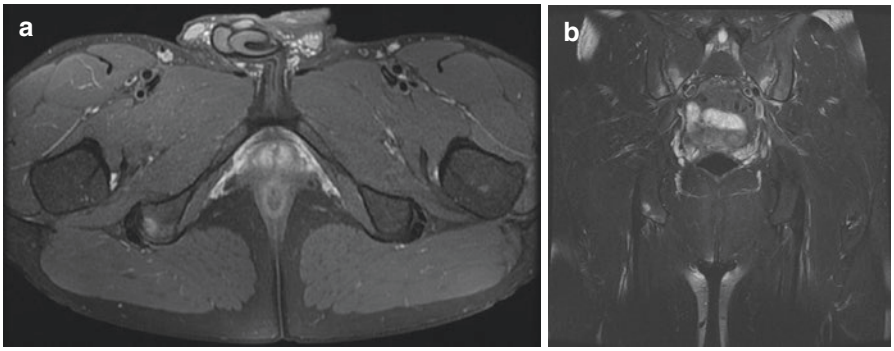

Fig. 13.3 Magnetic resonance imaging (MRI) of a hamstring tendinopathy and sacroiliitis caused by spondyloarthropathy. Panel (**a**) is an axial T2-weighted MR image that demonstrates right-sided increased signal intensity of the ischial tuberosity which is indicative of a bony cyst. There is also an increased thickness of the hamstring tendon origin. Panel (**b**) is a coronal T2-weighted MR image of the pelvis revealing increased signal intensity along the sacroiliac joints in the same patient. Based on the patient's history and abnormalities on this MRI, this abnormality was interpreted as enthesitis and sacroiliitis caused by a spondyloarthropathy

The abovementioned diagnoses should be considered and corrected or treated in case of treatment failure in a patient with buttock pain. As most PHTs will take weeks to months to recover, there is no specific time point for tendinopathies to consider a treatment as "failing." When a structured rehabilitation plan combined with load management does not improve symptoms within 8–12 weeks, a reevaluation and consideration of other potential diagnoses is recommended.

13.4.2 Inadequate Rehabilitation

It must be recognised that the evidence base surrounding hamstring tendinopathy rehabilitation is primarily that of case reports, pilot studies, clinical opinion, and narrative reviews. Failure of hamstring tendinopathy rehabilitation may be attributed to a number of factors relating to the overall structure and implementation of an individualised programme.

In the early stage of the rehabilitation, gaining control of symptoms through graduated progression of localised tensile loading and reduction of compression at the lateral ischium in both activities of daily living and in all exercises appears to be important. In patellar tendinopathy, isometric exercises with repeated 45 s holds at 70% of the maximum voluntary contraction have been shown to be promising in pain management and to address motor inhibition [65, 66]. However, these findings were not reproduced in patients with Achilles tendon pain [67]. This research has not been specifically reproduced for hamstring tendinopathy, yet this approach is considered to have some utility in early management, as it aids in early loading

within tolerance [1, 9, 68]. Alternatively, eccentric exercise over the knee is proposed by others [69]. Almost all approaches describe commencement of loading with the hip in a neutral position to avoid compression of the common tendon against the inferolateral border of the ischium [70]. Isolation of the symptomatic tendon with application of an effective stimulus and monitoring of the response are the basic principles of this stage. These may require revisiting if the response is below expectation.

In the progression of rehabilitation, utilisation of the principles of heavy slow resistance training or similar strength-based approaches to address associated muscle atrophy appears to be fairly common [71]. Again, no evidence is available specifically for PHT. Initial avoidance of compression at the enthesis is progressed into a graduated reintroduction of the hip flexion component, which is vital to meet functional requirements. Most authors above describe increasing range of hip flexion along with this heavy slow resistance approach. Here, exercise selection is determined through these principles and in accordance with individual responses. As an alternative approach, a case report utilising a specific training programme based on loading the proximal hamstring tendon with slow eccentric exercises on a treadmill is also described [72].

Muscle wasting and strength loss are frequent presentations in this patient group, requiring effective exercise prescription to stimulate muscle hypertrophy. Typically, these sessions are performed three times weekly with an intervening day of lower muscle demand to allow recovery. It is generally been suggested these exercise sets consist of slow repetitions (typically 3 s concentric, 3 s eccentric) with sets of at least 60–70 s duration and maximal weight tolerated. Kongsgaard et al. progressed from 15 repetition maximum sets to six repetition maximum sets [71]. Shortcomings typically encountered are of insufficient isolation of the injured side, not enough resistance, exercises performed or progressed too quickly, or with poor focus on technique. Recent work suggests performing these exercises closely regimented by a metronome may enhance motor pathways [73].

Depending on the sporting requirements of the athlete, progression of rehabilitation to full functional range and elastic load demands on the hamstrings requires further progression of rate of loading of the tendon through graduated higher-speed challenges to the proximal hamstring tendon through activities such as bounding, stairs, and fast pushing/dragging activities, ultimately into RTS. Management errors across these stages include a lack of load quantification and careful progression of these higher-speed demands. While evidence is lacking for ideal programming in progressing later-stage tendinopathy, it does appear that the sensitised tendon is unable to tolerate this form of high loading on consecutive days. Evidence around the duration required for tendon adaptation to higher loading is very limited and relies heavily on early work [74]. Progress from 3-day intervals to 2-day intervals as the athlete returns to sport appears to be a more prudent approach, although monitoring of the response of the affected tendon to this loading in terms of latent symptoms is recommended.

Finally, in returning to sport, the gradual reintroduction of appropriate volumes of sport-specific challenges such as change of direction or volume of running to match the typical demands of training and competition is vital. Avoidance of training load peaks and troughs and gradual building of a protective moderate to high-load foundation are key aspects of a successful RTS [75]. Beyond RTS, there appears to be a requirement with most tendinopathies to continue a routine of strength maintenance and tendon load monitoring over a period of a year or two in order to prevent recurrence [76].

13.5 Management of Treatment Failure in Patients with Acute Hamstring Injuries

13.5.1 Conservative Management

The first step in the management of treatment failure is to reconsider the initial diagnosis and repeat a diagnostic workup to confirm the initial diagnosis. Part of this management includes an exploration of the differential diagnosis (see Table 13.1 for the differential diagnosis of posterior thigh pain). When the initially established diagnosis is correct—but the treatment response is not as expected—a change in conservative management may be considered.

There is a continuous search for treatments to improve and accelerate muscle healing, and a number of medical interventions additional to rehabilitation have been proposed. Especially when initial treatment fails, there is a high demand for additional medical interventions, which may put medical practitioners under pressure. We will discuss the most frequently applied treatment methods.

13.5.1.1 Anti-Inflammatory Medications

Treatment with anti-inflammatory medications such as nonsteroidal anti-inflammatory drugs (NSAIDs) and corticosteroids is aimed at reducing the inflammatory response after muscle injury: especially, the use of oral NSAIDs has been widespread. Historically, inflammation was believed to be detrimental for muscle injury healing. However, multiple recent studies have shown that the various phases of the inflammatory process play a critical role in orchestrating muscle regeneration following injury, and there is accumulating evidence that pharmacological inhibition of the inflammatory process actually impairs acute muscle healing [77].

In the field of acute muscle injuries, there is only one clinical randomised controlled trial (RCT) on the efficacy of NSAIDs. This RCT showed that NSAIDs do not exhibit an effect on hamstring pain and muscle strength compared to a placebo intervention [78].

Despite their widespread use, anti-inflammatory medications should not be used following an acute noncontact hamstring injury, as there is growing evidence that it is actually detrimental for muscle healing [79–81].

13.5.1.2 Injection Therapies

Platelet-Rich Plasma (PRP)

Platelet-rich plasma (PRP) is probably the most popular injection therapy for muscle injuries. Since the World Anti-Doping Agency permitted the intramuscular injection of PRP in 2011, this experimental treatment has been increasingly used to treat acute muscle injuries in athletes [82]. PRP is derived from autologous whole blood using centrifuge separation systems to separate the platelets from other blood components. When injected in the injured muscle, platelets release various growth factors like platelet-derived growth factor (PDGF), insulin-like growth factor (IGF-1), basic fibroblast growth factor (bFGF-2), and nerve growth factor (NGF). These growth factors are assumed to provide regenerative benefits to the injured muscle tissue by stimulating myoblast proliferation and accelerating muscle fibre regeneration. There are a multitude of autologous platelet-rich blood products commercially available that differ in their preparation procedure and cellular components. Superiority is often claimed for one PRP product over others, but it remains unproven whether the composition of the PRP is relevant for the efficacy of PRP treatments, and this is subject of an ongoing debate in the literature.

Basic science studies have shown that growth factors can stimulate myoblast proliferation, and in deliberately injured animal muscles, these growth factors increase regeneration [83]. Despite these promising results and apparent widespread clinical use, the positive effects of PRP have not been confirmed in scientific studies on human subjects. A meta-analysis with pooled data of six RCTs showed no superiority of PRP in treating muscle injuries on the time to RTP and the reinjury rate nor were any substantial differences found for pain, muscle strength, flexibility, muscle function, and imaging [84]. There is even evidence that a PRP injection in addition to rehabilitation may be detrimental for muscle healing. A laboratory study in rats demonstrated that rehabilitation alone was more effective for muscle healing than rehabilitation combined with PRP injections [85].

In conclusion, considering the lack of efficacy in high-level RCTs and evidence for a possible adverse effect on rehabilitation, we currently discourage PRP treatment in muscle injuries.

Actovegin®

Actovegin® is a deproteinised hemodialysate of ultrafiltered calf serum which is suggested to have antioxidant and antiapoptotic properties [86]. To date, there is only one non-randomised clinical pilot study that examined Actovegin® in muscle injury [87]. In this study, athletes with grade I injuries that were treated with Actovegin® injections returned to play significantly earlier (12 days on average, $n = 4$) than those that only received physiotherapy (20 days on average, $n = 4$). However, this pilot study is at high risk of bias due to the lack of blinding and randomisation. Future larger randomised studies, including placebo groups and assessment of potential side effects, are necessary to determine whether Actovegin®

injections are safe and effective. We do not currently recommend it as a treatment for hamstring injuries.

Traumeel®

Traumeel® is a homeopathic combination of diluted plant and mineral extracts which is proposed to have an anti-inflammatory effect [88]. This injection therapy is used alone or in combination with Actovegin® in muscle injuries [89], but there is currently no evidence on the effect of intramuscular injection of Traumeel® in muscle injuries.

Stem Cells

There is increasing interest for the use of stem cell therapy in muscle injuries. Stem cells are undifferentiated cells that can renew themselves or differentiate into cells that are programmed for a certain tissue lineage. These cells may have the ability to contribute to muscle regeneration after injury. Therefore, the concept of transplanting stem cells has been explored for some time; however, the available literature focuses mostly on degenerative muscle disorders, such as muscular dystrophies.

Studies on stem cells in acute injury are currently limited to two murine contusion model studies [90, 91]. These studies found that intramuscular transplantation of muscle-derived stem cells promoted angiogenesis and increased the number and diameter of regenerative muscle fibres. Although these findings are promising, it is not known whether the same results can be found in human muscle tissue. Furthermore, concerns have been raised regarding the potential tumourigenic risk of stem cells.

Despite promising results, we currently do not advocate the use of stem cells in hamstring injuries, as their safety and efficacy in human use are yet to be determined.

In conclusion, the current available evidence does not support any of the available interventions in addition to active rehabilitation in acute muscle injury. For some, there is even (indirect) evidence that they may adversely affect outcome of muscle injury.

13.5.2 Surgical Management

With the exception of complete discontinuity of the bone-tendon-muscle unit (i.e. tendon avulsion injury), surgery is very rarely considered as the primary treatment for hamstring injury. One might even say that, in the setting of managing non-acute hamstring injuries, it can be regarded as a last resort or sometimes even a salvage procedure.

However, part of the challenge of managing muscle injuries that predominantly occur in athletes is to prevent a scenario of treatment failure. Surgical intervention in the acute setting might be warranted in order to avoid such a scenario.

In this paragraph, we will briefly go over the indications for surgery in acute hamstring injuries, as well as surgical treatment for acute hamstring injuries in which conservative management has yielded insufficient improvement.

13.5.2.1 Tendon Avulsion Injury

To date, evidence-based indications for surgical intervention are lacking for hamstring tendon avulsion injury. This may be attributed to a scarcity of controlled studies and the striking underrepresentation of conservatively treated cases in published literature that impede a proper comparison of treatment outcomes [8, 92, 93]. Cohen and Bradley [94, 95] have suggested that surgical repair of the ruptured tendons is indicated in two-tendon avulsions with more than 2 cm of retraction and three-tendon avulsions regardless of the extent of retraction. Without a scientific basis for these criteria and the very limited knowledge of the natural course of this injury, this should be regarded as expert opinion.

Surgical repair of proximal hamstring tendon avulsions comprises protection of the sciatic nerve, mobilisation of ruptured tendons, and fixation of the mobilised tendons to the ischial tuberosity with suture anchors [8].

Based on the most recent and comprehensive systematic review [92], surgical repair resulted in significantly higher patient satisfaction, better hamstring strength recovery, and higher scores on single-leg hop tests and functional testing scales compared to conservative treatment. Strikingly, surgical repair did not significantly improve the chance of returning to sports or pre-injury activity level.

Early (i.e. within 8 weeks after injury) surgical intervention leads to significantly higher patient satisfaction, less residual pain, and higher scores on functional scales compared to delayed intervention [92]. Conversely, there is no difference in rate of RTS or pre-injury activity level, hamstring strength, hamstring endurance, and Tegner scores. Moreover, it is often mentioned that delayed intervention is technically more demanding due to development of adhesions requiring a more extensive neurolysis of the sciatic nerve [8], as well as increased retraction of the ruptured tendons [96]. In the latter case, re-approximation can be more difficult, and sometimes, an allograft or autograft reconstruction is needed to bridge a remaining gap or augment the repair [8]. Interestingly, no significant difference in complications between acute and delayed intervention was found [92].

Distal tendon avulsions make up about 2% of all hamstring injuries and are therefore less common than proximal tendon avulsions [97]. As one would expect, the literature is also more limited.

Lempainen et al. [98] retrospectively analysed 18 operatively treated patients with distal hamstring tears, five of whom had full-thickness tears involving either the distal tendon or musculotendinous junction. In case of a free tendon avulsion, refixation was achieved by means of suture anchors. In case of a tear through the musculotendinous junction, sutures were used following excision of any scar tissue. All five patients were able to RTS at pre-injury level without residual complaints after 2–6 months.

The current literature does not allow for a comparison between conservative and surgical treatment. Moreover, it may not be appropriate to pool distal tears of the three different hamstring muscles as they have different functions with respect to dynamic stabilisation of the knee joint.

13.5.2.2 Intramuscular Tendon Injury

In recent years, hamstring muscle injury with intramuscular (or "central") tendon involvement (Fig. 13.4) has become notorious because of initial observations that it might lead to disastrous outcome [99]. To be more specific, these injuries were noted to take three to four times as long to recover [41, 100] and were found to have significantly higher recurrence rates [100]. However, when athletes were treated by a physiotherapist blinded to imaging findings, using a criteria-based rehabilitation programme, differences were notably smaller [101]. The difference in time to RTP between injuries without tendon involvement (mean 22 days) and those with full-thickness intramuscular tendon disruption (mean 32 days) was approximately a week and a half. Moreover, reinjury rates within 12 months after RTP for injuries with and without tendon disruption (both 20%) were not significantly different [40].

Based on this relatively small difference in time to RTP between "regular" hamstring injury and "severe" intramuscular tendon injury, we argue that these intramuscular tendon injuries should not primarily be treated surgically. However, as is the case for persistent or recurrent musculotendinous injuries, there might be a role for surgery in cases that are refractory to conservative strategies.

Lempainen et al. [102] published a case series of eight athletes with intramuscular tendon injuries that were surgically treated. The indication for surgery in acute cases was a full-thickness disruption with a clear gap between tendon ends. For chronic cases, surgical indications included recurrent disabling injury and inability to participate in competitive sports at pre-injury level. The surgical technique depended on the location of the injury and whether it was acute or

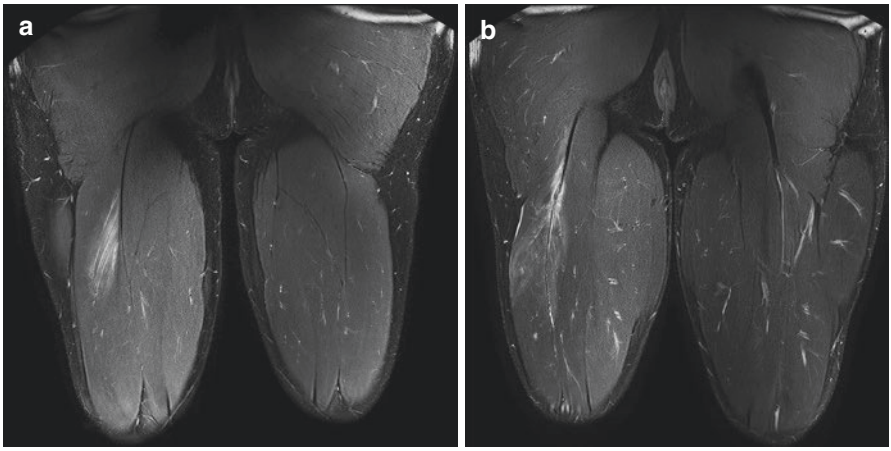

Fig. 13.4 Magnetic resonance imaging (MRI) of an intramuscular tendon injury. Coronal STIR images demonstrating right-sided increased signal intensity in a feather-shaped pattern located in the proximal BF (long head). Panel (**a**) depicts a musculotendinous injury without intramuscular tendon injury. Panel (**b**) depicts an injury with partial-thickness intramuscular tendon injury, as evidenced by increased intratendinous signal intensity, intramuscular tendon disruption, and tendon waviness

recurrent. In acute injuries, tendon ends were approximated and sutured. In recurrent injuries, the tendon was repaired using a gliding Z-plasty (i.e. a surgical technique to increase tendon length). When the injury was in proximity to the ischial tuberosity, a suture anchor was placed for additional support of the repair.

All athletes returned to sports at pre-injury level between 2.5 and 4.5 months postoperatively. It should be emphasised that controlled clinical studies on the efficacy of surgical treatment for this condition are lacking.

13.5.2.3 Musculotendinous Injury

The bulk of hamstring injuries do not demonstrate signs of proximal or distal tendon involvement [97, 103], and they are predominantly located at or near the proximal musculotendinous junction. While it is a common injury in sports that usually heals well with conservative treatment [16], it is an injury that should be taken seriously. One of the major problems is a high tendency to recur [3]. For whatever reason, every clinician will have at least one case of an athlete who has sustained injury after injury, usually in the same location [104].

On rare occasions, these recurrent (or persistent) injuries have been managed surgically. One study described a series of 18 distal hamstring tears [98], of which 12 were partial-thickness tears of the musculotendinous junction. Surgical treatment was carried out when athletes were unable to participate in sports at the pre-injury level after at least 6 weeks post-injury. The intervention comprised excision of scar tissue and mobilisation of the injured muscle to ensure there was no restriction due to adhesions, followed by suturing. Eight athletes (67%) returned to sports at pre-injury level after 2–5 months postoperatively, seven of which without any residual symptoms.

Surgery for hamstring injuries is rarely indicated in the acute setting. With the exception of tendinous or bony avulsion, surgical consultation should be postponed until the point at which conservative treatment strategies have insufficiently improved function or symptoms. Based on the limited evidence that is currently at our disposal, surgery appears to be beneficial in these cases and leads to a good chance of returning to sports at pre-injury level. Yet, due to lack of controlled studies, it is unknown whether this approach should be preferred over continuing conservative approaches.

13.6 Management of Treatment Failure in Patients with Ongoing Posterior Thigh Pain, Including Hamstring Injury Sequelae

13.6.1 Conservative Management

Hamstring injury sequelae are challenging to manage and scientific knowledge is limited. We advise to optimise deficits in hamstring strength and flexibility, and to perform a progressive rehabilitation. Although this is the current mainstay of

treatment for these patients, there is a subgroup of patients that will remain symptomatic, regardless of treatment.

13.6.2 Surgical Management

Surgery for hamstring injuries is rarely indicated in the acute setting, as described above (Sect. 13.5.1). This also accounts for hamstring injury sequelae. One underlying cause of hamstring injury sequela might be a myositis ossificans.

Myositis ossificans that results in persisting complaints of pain and impaired function with restricted sports activities despite conventionally accepted treatment may benefit from surgery. While this approach is often employed in clinical practice, it is unclear at what point exactly one should regard the conservative treatment as failed and when surgery might be indicated. There are no evidence-based guidelines or controlled studies that can serve as the basis for recommendations with regard to if and when surgery should be performed.

Considering that symptoms and dysfunction tend to regress as the lesion matures over the course of months, sufficient time should be allowed for conservative strategies to elicit an effect. In addition, surgical excision before the lesion has fully matured is traditionally believed to result in local recurrence. Therefore, surgical intervention is generally discouraged before at least 6–12 months after the injury [25].

In a recent study, clinical outcome was reported for high-level athletes undergoing isolated excision of a heterotopic ossification [25]. In most cases (84%), the lesion was located in one of the muscle groups in the thigh region. Following the intervention, indomethacin was administered for 3 weeks, and RTS was allowed 4–6 weeks postoperatively. Overall, surgery resulted in clinical improvement, and the vast majority (97%) was able to return to their pre-injury activity level. Most athletes (81%) were able to return to this level with no or mild residual complaints of pain during activity. With the exception of hypoesthesia at the periphery of the skin incision, there were no complications.

In summary, traumatic myositis ossificans is a self-limiting condition that rarely requires surgery. There are currently no evidence-based surgical indications. In athletes with persisting complaints of pain and dysfunction despite adequate and prolonged conservative treatment, surgical excision of a heterotopic bone appears to result in clinical improvement with a good chance of returning to pre-injury activity.

13.7 Management of Treatment Failure in Patients with Hamstring Tendinopathy

13.7.1 Conservative Management

Management of treatment-resistant PHT starts with reconsidering the initial diagnosis (Table 13.2), reevaluating the treatment strategy, repeating diagnostic workup for buttock pain, and performing additional diagnostics if needed.

It is worth mentioning that exercise-based rehabilitation normally takes weeks to months before a treatment effect can be expected [1]. It is therefore important to set realistic time frames before the start of conservative treatment. This will prevent unnecessary requests for additional diagnostics resulting in increased healthcare costs.

When the initial diagnosis is confirmed, medical therapies may be considered for long-standing PHT that is resistant to exercise-based rehabilitation.

13.7.1.1 Medical Treatment Modalities

Proximal hamstring tendinopathy appears to be something of an "ugly duckling" in the literature on tendinopathy. While the current body of evidence on treatment of Achilles and patellar tendinopathy is rapidly expanding, evidence for treatment of PHT is lagging behind. For distal hamstring tendinopathy, this is even more striking.

13.7.1.2 Nonsteroidal Anti-Inflammatories (NSAIDs)

There is limited evidence that NSAIDs can provide a reduction in symptoms in patients with reactive tendinopathy [76]. The mechanism behind NSAID treatment in this phase may be decreased tendon cell proliferation and simultaneous decreased proteoglycan production. Rest and NSAIDs are less favourable in cases of chronic tendinopathy. Rest can have an initial positive effect on symptoms, but it has also been shown to induce a reduction in the amount of collagen. NSAIDs have fallen out of favour for long-standing tendinopathy, as there is no evidence for their efficacy [105].

13.7.1.3 Extracorporeal Shock Wave Therapy (ESWT)

Another frequently applied treatment in tendinopathy is extracorporeal shock wave therapy (ESWT), which delivers an energy flux through the tendon collagen tissue. ESWT is thought to initiate biological responses and tissue regeneration, but this effect is mainly based on laboratory studies. There is one randomised study on the efficacy of ESWT in athletes with PHT [106]. This study showed that ESWT is safe and more effective than exercise alone, although there were some study limitations (small sample size, a lack of disease-specific measurements, and absence of placebo ESWT and blinding of participants). A recent systematic review demonstrated conflicting evidence for the efficacy of ESWT in lower limb tendinopathies [107, 108]. More evidence is needed to define the efficacy of ESWT treatment in PHT. There might be a subgroup of patients that responds well to this treatment, but to date, it is unknown which patients are good responders.

13.7.1.4 Injection Therapies

Effectiveness of local corticosteroid injections for tendinopathy has mainly been described in case reports or case series [109], but no large randomised studies with long-term follow-up have been performed in patients with lower extremity tendinopathy. Effects of corticosteroid injections are not known for PHT. A systematic review showed that corticosteroids in tendinopathy are

effective in the short term but detrimental in the longer term [110]. There is also an association between these injections and occurrence of a total tendon rupture [111]. While the efficacy and safety of corticosteroids is described for tendinopathies in general in these studies [110, 111], it is unknown whether these results can be extrapolated to patients with PHT. A total tendon rupture of the proximal hamstring tendons is a severe complication with dramatic consequences for an athlete. Therefore, clinicians should be cautious with applying intratendinous hamstring injections.

There are numerous other injection treatments that are proposed for tendinopathies. Injection agents that have been used include polidocanol (sclerosing therapy), dextrose (prolotherapy), and autologous blood and PRP. In PHT, no studies have been performed on the effect of sclerosing therapy or prolotherapy. The fact that the sciatic nerve is running next to the hamstring tendon makes it less attractive to inject a sclerosing agent. Prolotherapy results in temporary irritation of this nerve, which can be annoying for patients. There is also no strong evidence for these injection therapies in other tendinopathy locations [112]. Autologous blood injections and PRP injections are used with the aim to deliver growth factors with regenerative effects on the tendon tissue. The use of autologous whole blood and PRP treatment has been evaluated in one randomised study in patients with PHT [113]. Both treatments resulted in an improvement over time, but it is unknown whether these injections are better than other conservative treatments or a "wait-and-see" approach.

In conclusion, there is currently no strong evidence for the effectiveness of second-line treatment options when exercise therapy and load management advice failed for patients with long-standing PHT. Some of the abovementioned options may be considered if the potential benefits and harms are discussed on beforehand with the patient.

13.7.2 Surgical Management

While the histopathological characteristics seen in hamstring tendinopathy correspond with findings in other tendinopathies [1], distinct anatomical features may play a role in the decision-making progress. Therefore, in this section, we will focus specifically on surgical treatment of PHT.

As is the case with almost all musculoskeletal injuries in the athletic population, the primary treatment is conservative [63]. Owing to its heterogeneous presentation and response to treatment, the challenge lies in determining the optimal treatment and the point at which conservative treatment has "failed" and when a surgical approach may be beneficial.

According to a recent review, about one in every five patients experiences persisting symptoms after 6 months of conservative treatment [63]. This point in time has been suggested as the moment at which surgery should at least be considered.

Moreover, the decision for surgical intervention should not only include the duration of symptoms and response to conservative treatments but also the suspected cause of the persisting symptoms. Tendinopathic pain is thought to be related

to a combination of neurovascular ingrowth and production of biomechanical substances (e.g. catecholamines, acetylcholine, glutamate) [62]. Yet, it has been postulated that complaints of pain may also arise from compression of the sciatic nerve [114], which runs in close proximity to the proximal hamstring tendons [115]. Compression resulting from adhesions between the nerve and proximal tendons or direct compression caused by thickening of the proximal tendons, previously also referred to as "hamstring syndrome" [1], may need to be addressed as well in order to improve or resolve pain.

Outcome following surgical intervention has only been investigated using retrospective study designs [1, 63, 116]. In the study by Lempainen et al. [63], 103 cases of PHT in 90 athletes were reported. Surgery was indicated when patients experienced chronic and disturbing symptoms despite conservative treatment. Conservative treatments included modification or suspension of sports activities, hamstring stretching, NSAIDs, corticosteroid injections, and physiotherapy. In almost all cases (97%), surgery was performed after symptoms persisted for at least 6 months.

The authors noted that the proximal SM tendon was commonly thickened. Hence, their surgical technique involved a transverse tenotomy of the (thickened) lateral proximal SM tendon several centimetres distal to the ischial tuberosity. Additionally, any adhesions around the sciatic nerve were carefully removed. The distal part was then sutured to the proximal tendon of the long head of the BF in order to prevent retraction of the muscle. Postoperatively, weight-bearing was gradually progressed in the first 2 weeks, and in the first 3–4 weeks, care was taken to avoid excessive stretching of the hamstrings. Isometric exercises and cycling were started at 4 weeks, and weight training and running were started at 8 weeks. Return to full sporting activities was allowed at 2–4 months postoperatively [63].

Surgery resulted in a high rate of RTS at the pre-injury level (89%) after a mean 5 months with no (60%) or minor symptoms (29%) during activity. The complication rate was 10%. Minor complications included DVT (1%), wound fistula (1%), and transient hyperesthesia of the incisional area (2%). Six cases required a reoperation, four due to early symptoms resulting from scar tissue around the hamstring origin and sciatic nerve and two due to late recurring complaints resulting from a regenerated SM tendon.

Benazzo et al. [115] reported the outcomes of 17 athletes who underwent surgery for persisting complaints of PHT that caused limitations or interfered with sport participation. Conservative treatments prior to surgery included physiotherapy, NSAIDs, and corticosteroid injection. All patients had persisting complaints despite a course of conservative treatment of at least 3 months.

The surgical technique involved identification of the involved (i.e. hypertrophic and fibrotic) tendon, followed by a partial transverse tenotomy or repeated puncturing of the tendon and a release of the sciatic nerve. Directly postoperatively, continuous passive motion of the hip and knee was started. Active motion was encouraged from the first day after surgery, and weight-bearing was progressed during the first 10 days. Progressive strengthening was started in the second week and progressed from open to closed kinetic chain after 4 weeks. Running was allowed after 8 weeks.

All patients were able to RTS at pre-injury level after a mean 4 months, either with no residual symptoms (88%) or pain during intense efforts (12%). The complication rate was 12%, including postoperative hematoma and transient hyperesthesia of the incisional area.

Both surgical techniques are similar in the sense that they involve lysis of adhesions in addition to a (partial) tenotomy. These perineural or peritendinous adhesions, which are also observed during surgery for chronic Achilles tendinopathy [116], might play an interesting role with respect to failure of conservative treatment and outcome of surgical treatment.

It should be noted that no prospective controlled studies have been conducted. It is therefore not known whether surgery is superior to conservative treatments in patients with chronic symptoms. Based on the current available evidence, the sole conclusion that can be drawn is that surgical treatment for refractory PHT appears a viable secondary option.

13.8 Conclusion

Treatment failure, defined as an unsuccessful result of management, is observed on a frequent basis in both acute and long-standing hamstring injuries, and it is due to either an incorrect diagnosis or inadequate response to treatment. There is a large range of differential diagnoses that can be considered in patients with treatment failure after acute and long-standing hamstring injuries. Reevaluation of the patient and expanding diagnostic workup are potential options to explain treatment failure. Numerous alternative treatment options for patients with acute hamstring injuries, hamstring injury sequelae, and hamstring tendinopathy are available. In general, there is no strong evidence for the efficacy of these alternative treatment options. Almost all second-line treatment effects are based on level 4 evidence. When considering these treatments, potential adverse events, healthcare costs, and likelihood of efficacy should be taken into account.

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