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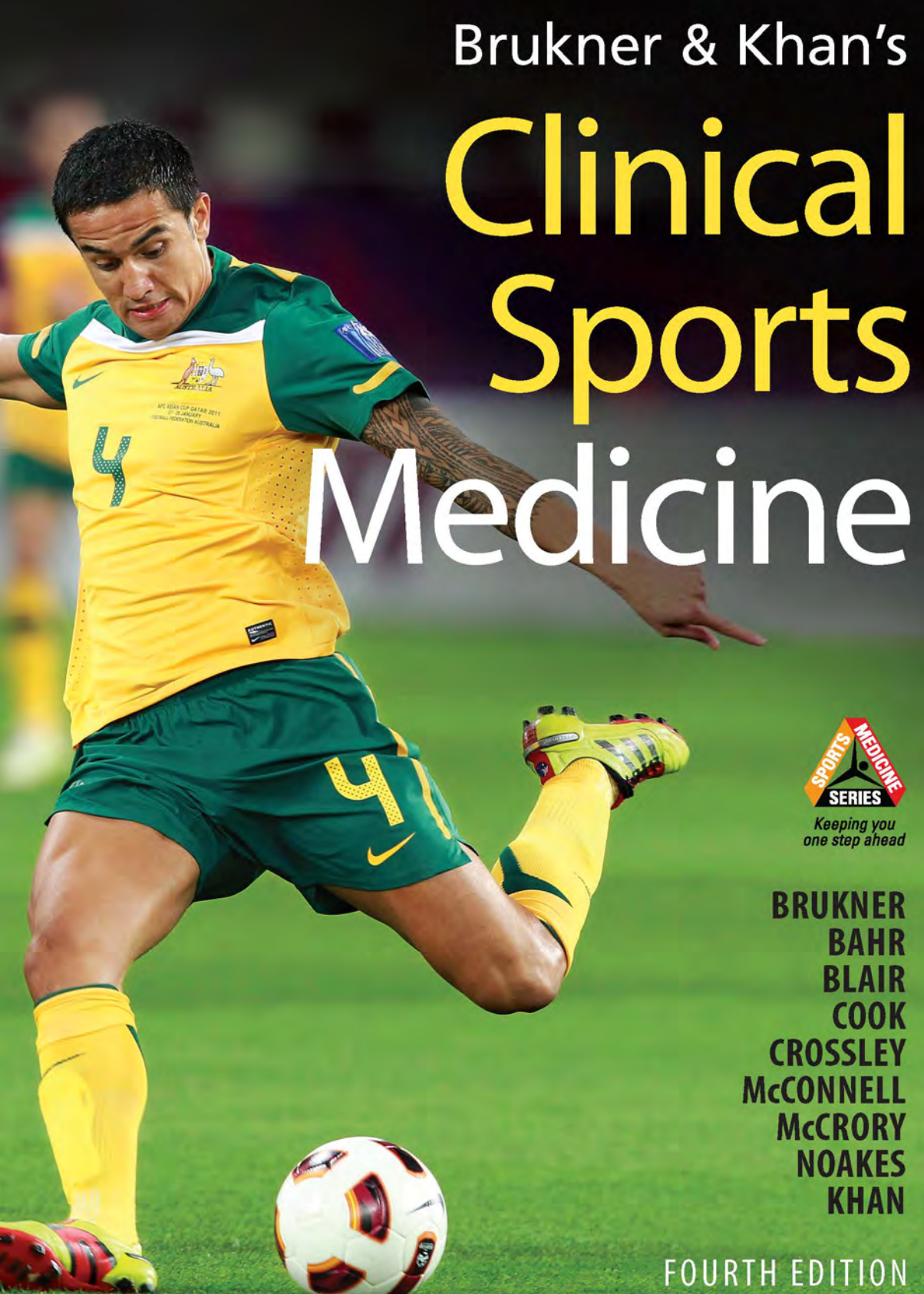

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We dedicate this fourth edition to the Clinical Sports Medicine community—to each clinician, educator, and policy maker committed to improving health through the power of physical activity.

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First published 1993
Second edition 2001
Revised second edition 2002
Third edition 2006
Revised third edition 2009

National Library of Australia Cataloguing-in-Publication Data
Author: Brukner, Peter.
Title: Brukner & Khan's Clinical Sports Medicine / Peter Brukner, Karim Khan.
Edition: 4th ed.
ISBN: 9780070998131 (hbk.)
Notes: Includes index.
Previous ed.: Clinical Sports Medicine, 2007.
Subjects: Sports medicine.
Sports injuries.
Other Authors/Contributors:
Khan, Karim.
Dewey Number: 617.1027

Published in Australia by
McGraw-Hill Australia Pty Ltd
Level 2, 82 Waterloo Road, North Ryde NSW 2113
Publisher: Fiona Richardson
Senior production editor: Yani Silvana
Production editor: Jess Ní Chuinn
Publishing and digital manager: Carolyn Crowther
Editorial coordinator: Fiona Collison
Copy editor: Jill Pope
Illustrator: Vicky Earle (anatomical figures)
Proofreader: Mary-Jo O'Rourke
Indexer: Russell Brooks
Cover design: Georgette Hall
Internal design: David Rosenmeyer
Typeset in 9/11.5 pt Scala by Midland Typesetters, Australia
Printed in China on 80gsm matt art by iBook Printing Ltd

Foreword to the first edition (1993)

Sport in Australia is ingrained in the national consciousness more widely, deeply, and indelibly than almost anywhere else in the world. When a prominent sportsperson sustains a sporting injury, either traumatically or from overuse, becomes excessively fatigued, or fails to live up to expectations, this assumes national importance. It is even more relevant nowadays with greater individual participation in sporting activities. The same type of problems occur for recreational athletes, middle-aged people wanting to become fit, or older people wishing to sustain a higher level of activity in their later years.

In *Clinical Sports Medicine* the authors take sport and exercise medicine out of the realm of the elite athlete and place it fairly and squarely where it belongs—as a subspecialty to serve everyone in the community who wishes to be active.

The book is organized in a manner that is sensible and usable. The chapters are arranged according to the anatomical region of the symptom rather than diagnostic categories. This results in a very usable text for the sports physician, general/family practitioner, physiotherapist, masseur, or athletic trainer whose practice contains many active individuals.

Practical aspects of sports medicine are well covered—care of the sporting team and concerns that a clinician might have when traveling with a team. In all, this is an eminently usable text which is timely in its production and will find an important place among clinicians involved in the care of active individuals.

John R Sutton MD, FRACP
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This foreword was written by the late Professor John Sutton before his untimely death in 1996; it is retained in this textbook out of profound respect for this champion of the integration of science, physical activity promotion, and multidisciplinary patient care.

Foreword to the fourth edition

Humans were not designed to sit at desks all day and in front of televisions all evening, and this physical inactivity is related to a host of health-related issues. Increasing physical activity is one very powerful way to mitigate many of the health issues we face today, and programs such as the Healthy People 2020 initiative and the Exercise is Medicine campaign encourage individuals to remain active throughout their lifetime.

As people become more involved in sport and exercise, sports medicine becomes increasingly important, and *Clinical Sports Medicine* has understandably become what we in the US refer to as the "PDR" (Physicians' Desk Reference) of sports medicine. For my UK colleagues the translation is "BNF" (British National Formulary). This text is extremely comprehensive, covering fundamental principles of biomechanics, diagnosis and treatment, regional musculoskeletal injuries, and medical problems. The text also addresses those practical issues of sports medicine that are often missing from other texts, such as dealing with athletic teams, covering endurance events, and working with the elite athlete.

The organization of the text makes it remarkably easy to use, including such features as color-coded book sections, flow diagrams to reinforce concepts, and tables that clearly organize information. Vicky Earle's anatomical drawings are truly among the best in the business. All these features put an astounding wealth of information at the reader's fingertips. This information has been assembled by a group of over 100 experienced and world-class physical therapists, physicians, and scientists. These co-authors provide up-to-date references when available, and clearly state when evidence is lacking.

This updated, fourth edition includes 200 new photos/graphics and 13 new chapters on current topics, including Integrating evidence into clinical practice, Principles of activity promotion, and Medical emergencies in sport. The editors continue to add to the clinically relevant topics with one of my favorites being what I call "How to manage the patient who has seen everyone and wants a cure from you!" (Chapter 41).

An innovative and exciting addition to this edition is the integration of the *Clinical Sports Medicine* masterclasses that allow you, through videos and podcasts on the *Clinical Sports Medicine* website, to learn directly from the experts. These masterclasses will be continually updated and they provide a remarkably dynamic component to the text.

It is exciting to watch *Clinical Sports Medicine* evolve substantially with each edition. The editors' focus of this text is to "help clinicians help patients" and they have clearly hit their mark. This book is an absolute must-have for any sports medicine professional.

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Brief contents

Part A Fundamental principles

Part B Regional problems

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Preface

"Helping clinicians help patients" has been the clear focus of *Clinical Sports Medicine* from its inception. This fourth edition (CSM4) builds unashamedly on its 20-year history. Twenty-year history? The more than 100 contributing authors average 15 years of practical experience each, so you are holding well over 1500 years of distilled clinical wisdom in your hand!

If you will permit us some level 5 evidence (expert opinion—see all-new Chapter 3), CSM4 provides clinicians in sports and exercise medicine and physiotherapy/physical therapy at least five major benefits:

- The wholehearted commitment from leading clinical faculty from all over the English-speaking world means that CSM4 provides the reader with an authoritative text—you can trust these authors.
- At 1270 pages and 67 chapters, CSM4 already carries 25% more pages than the best-selling third edition. Our ruthless editing to focus on clinical relevance means this edition contains 40% new material. CSM4 provides a comprehensive base for your clinical library. We provide some specific examples below.
- With more than 1000 color images (photos and graphics), the book paints a million words (1000 pictures each painting 1000 words!) over and above its 1270 pages! More than 200 of those images are new to this edition—customized for CSM4's learners—further extending the book's clarity and usability.
- Every copy of CSM4 comes with a code that gives you online access to more than four hours of assessment and treatment video and audio material. Called *Clinical Sports Medicine* masterclasses, this material is integrated with the text and will be free of charge to book owners for 12 months from registration at www.clinicalsportsmedicine.com. You have "the expert in the room."
- Reflecting the expanding evidence base for our field, we include an introduction to evidence-based practice (Chapter 3). All authors aimed to incorporate the best available level of evidence via text, tables, and current references. The online content of CSM4 will benefit from regular updates, adding further to the usefulness of this text for busy clinicians.

In short, CSM4 provides excellent value as an authoritative clinical foundation for physiotherapists, medical practitioners, osteopaths, massage therapists, podiatrists, sports/athletic trainers, sports therapists, fitness leaders, and nurses. It has also proven popular for students in sports physiotherapy, medicine, and human movement studies/kinesiology.

Editors and authors

As the task of editing a book of this magnitude was beyond the two of us, the CSM4 reader now benefits from the wisdom and productivity of seven sports and exercise medicine greats—Roald Bahr, Steven Blair, Jill Cook, Kay Crossley, Jenny McConnell, Paul McCrory, and Timothy Noakes.

The quality of our chapter authors, representing more than 14 countries, grows with each edition. Among our all-star cast, we are particularly grateful to Håkan Alfredson, Elizabeth Arendt, Carl Askling, Kim Bennell, John Drezner, Richard Frobell, Per Holmich, Mark Hutchinson, Gwen Jull, Pekka Kannus, Ben Kibler, Nicola Maffulli, Lorimer Moseley, George Murrell, Kevin Singer, and Willem van Mechelen.

New chapters

The new chapters in this edition are:

Chapter 1 Sports and exercise medicine: addressing the world's greatest public health problem
Chapter 3 Integrating evidence into clinical practice to make quality decisions
Chapter 4 Sports injuries: acute
Chapter 5 Sports injuries: overuse
Chapter 16 Principles of physical activity promotion for clinicians
Chapter 23 Wrist pain
Chapter 24 Hand and finger injuries
Chapter 28 Hip-related pain
Chapter 45 Military personnel
Chapter 47 Medical emergencies in the sporting context
Chapter 48 Sudden cardiac death in sport
Chapter 52 Renal symptoms during exercise
Chapter 54 Exercise to treat neurological diseases and improve mental health

A plethora of new, clinically relevant content

Here is just a sampler of new approaches to specific "hot topics" with a few of the contributing authors:

- The all-new Chapter 28 Hip-related pain clarifies the concept of **femoroacetabular impingement (FAI)**, its diagnosis and management
- The latest **concussion** guidelines based on the Zurich consensus meeting (with Paul McCrory)
- A fully revamped discussion of **neck pain** (with Gwen Jull)
- Further tips on management of **tendinopathies** (with Jill Cook, Hakan Alfredson, and Ben Kibler)
- Discussion of whether **ACL injuries** should be managed operatively or conservatively (with Richard Frobell and Liza Arendt)
- A revolution in **pain science** and its implications for clinical practice (with Lorimer Moseley)
- How to prevent **hamstring problems** from being a major burden—prevention and treatment strategies (with Carl Askling and Anthony Schache)
- A practical approach to **leg pain**, including compartment pressure testing (with Mark Hutchinson and a demonstration on the masterclasses website)
- How to manage **the patient who has seen everyone**—and now wants a miracle cure from you (with Jim Macintyre)
- Prevention of sudden **cardiac death** and a practical approach to **sports cardiology** (with Jon Drezner and Sanjay Sharma)
- **Exercise in the heat** as well as **prevention of hyponatremia** (with Tim Noakes)
- **Drugs**—based on latest WADA guidelines

We could have made this list much longer but instead we use a toll-free part of the *Clinical Sports Medicine* masterclasses website (www.clinicalsportsmedicine.com) to take you on a tour.

No single profession has all the answers required to treat the ill or injured sportsperson and to provide exercise advice as needed. CSM4 was created by a champion team of co-authors and critical reviewers tremendously committed to the vision of "Helping clinicians help patients." We are confident that whatever your training, *Clinical Sports Medicine* fourth edition will reinforce and refine existing knowledge and techniques, and introduce useful new approaches for your clinical practice as well as for your teaching of our wonderful vocation. Enjoy this first hybrid print and digital *Clinical Sports Medicine*.

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Australian Athletics team 1990–2000, World Championships Tokyo 1991, Gothenburg 1995, Seville 1999

Australian team, World Cup Athletics, Havana 1992

Australian Mens Hockey team 1995–96

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Drugs in Sport—What the GP Needs to Know 1996, 2000

The Encyclopedia of Exercise, Sport and Health 2004

Essential Sports Medicine 2005

Clinical Sports Anatomy 2010

Awards

Medal of the Order of Australia 2006

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Vicky is a highly experienced medical illustrator who has been involved in the design and production of a wide variety of surgical procedural and medical illustrations that have been used in journals, books, conferences, lectures, and legal presentations. Her keen interest in *Clinical Sports Medicine* stems not only from a great appreciation of the human body and its capabilities, but also from a decade of racing experience as a championship rower and paddler—and knowing first-hand the many injuries that accompany these activities.

Acknowledgments

No need to apologize, let me look at what needs to be done. Immediate email response from an extremely busy co-author when asked to contribute to this fourth edition.

This completely updated print and online resource is unashamedly founded on the previous three editions. To date, this text has satisfied more than 80 000 clinicians and provided core material for students who focus on the care of active people in Australia, New Zealand, Africa, Asia, Europe, and the Americas. Japanese readers have their own translation. The overwhelming support for this clinically based textbook means we are particularly indebted to our partners in all previous editions.

Specific thanks for the fourth edition go to chapter co-authors listed with their affiliations on pages xxxvi–xli. Expert co-authors provide the crucial innovation and timeliness that *Clinical Sports Medicine* users demand. We are both humbled and privileged to be sharing cover authorship with seven amazing colleagues and friends—Drs Cook, Crossley, McConnell, Bahr, Blair, McCrory, and Noakes (ladies first, of course). We would love to have listed more names on the cover but the designer overruled us on that one! A further 109 co-authors made this book happen. It takes a community to create *Clinical Sports Medicine*—and we are grateful for every single member of that hardworking international community.

Because this edition fully embraces digital media, we especially acknowledge those co-authors who contributed to this innovation. Particular thanks go to Dr Mark Hutchinson, and the team in Chicago, for providing critical and substantial content for the online masterclasses.

Vicky Earle has gained international recognition for her artwork; thank you for continuing to translate clinical innovation in ways that jump to life for users. The University of British Columbia (Department of Family Practice—Faculty of Medicine as well as Faculty of Education) provided essential support (KK), as did the Olympic Park Sports Medicine Centre, The University of Melbourne and Liverpool Football Club (PB). *Clinical Sports Medicine* benefits from the continuity, consistency, and integration honed over two decades, and from the expertise and freshness of cutting-edge international chapter authors. We seek out the world's best and we appreciate their responding to our calls! It has been a pleasure to work with every member of the *Clinical Sports Medicine* 4th edition team.

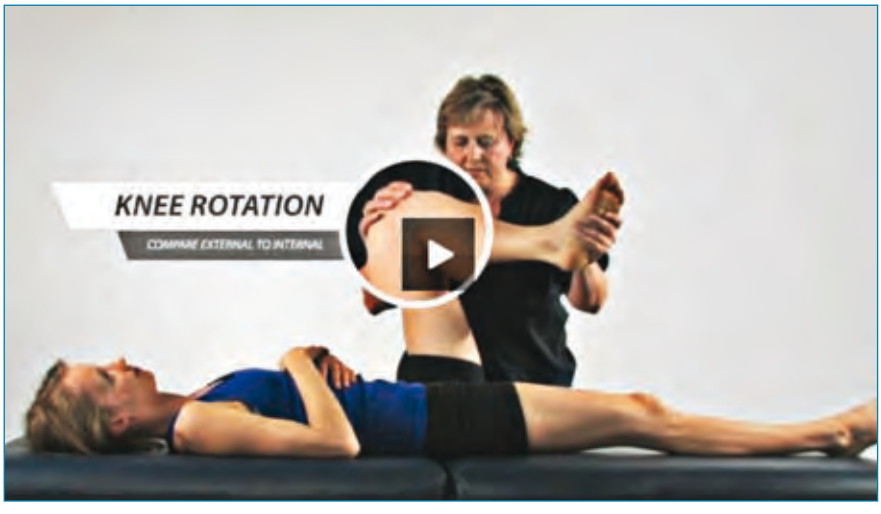
We give special thanks to our publishing team, who efficiently developed Brukner and Khan dreaming into the book you hold in your hands: publishing director Nicole Meehan, who has been a visionary leader; publishers Elizabeth Walton and Fiona Richardson; production editors Yani Silvana and Jess Ní Chuinn; and freelance editor Jill Pope. McGraw-Hill's support of all our crazy ideas has allowed us to generate a few good ones; thanks for your judgment and filtering! Within the authors' multi-faceted production team in three countries, Zuzana Machotka and Callista Haggis earn special thanks—for their skill, attention to detail, and good humor even under pressure. Finally, axiomatically, the most profound thanks we reserve for our long-suffering friends and families: Diana and Heather, we both know that words are not enough!

Guided tour of your book

The principal text in its field, this fourth edition of *Clinical Sports Medicine* continues to provide readers with quality, up-to-date content. The engaging material has been contributed by leading experts from around the world. Look out for these key features, which are designed to enhance your learning.

Integrated learning resources

New to this edition is the *Clinical Sports Medicine* website containing masterclasses with video and audio content.



The authors have worked with specialists to film key clinical procedures, including video clips demonstrating physical examinations, key rehabilitation exercise programs, and joint injections. Much of this video content has been commissioned for this edition.

www.clinicalsportsmedicine.com.

Wherever this icon appears in the book, go to the website to view a video or listen to a podcast. Access is via the pincode card located in the front of the book.

For easy reference, a summary of the online content (where relevant) is given at the end of each chapter.

CLINICAL SPORTS MEDICINE MASTERCLASSES

www.clinicalsportsmedicine.com

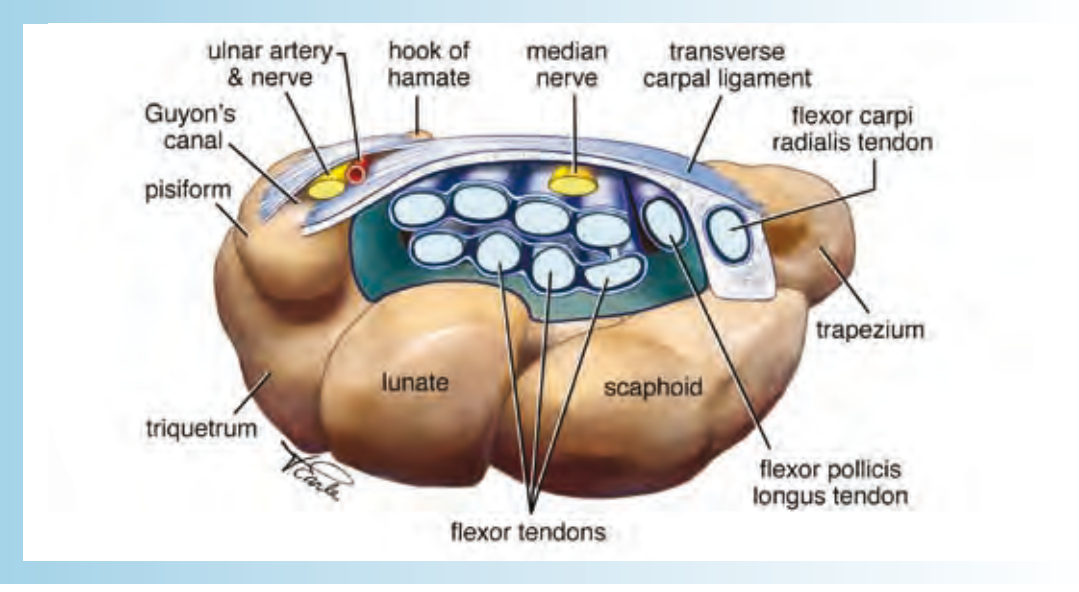
- Listen to the interview with chapter authors.
- See demonstration of biomechanical assessment.
- See a demonstration of the original low-Dye technique augmented with reverse sixes and calcaneal slings anchored to the lower leg.

RECOMMENDED WEBSITES

Barton CJ, Bonanno D, Menz HB. Development and evaluation of a tool for the assessment of footwear characteristics: www.ncbi.nlm.nih.gov/pmc/articles/PMC2678108/?tool=pubmed

First-class content

As with previous editions the emphasis is on treatment and rehabilitation. The chapters in Part B, which address regional problems, are heavily illustrated with clinical photos, relevant imaging, and anatomical illustrations.



The list of **world-renowned contributors** has grown even longer in this edition and brings a truly global perspective to the book.

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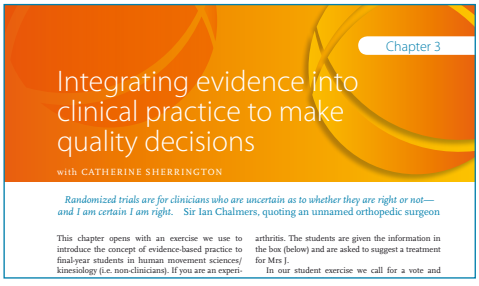
Practice pearls are a valuable feature that provide clinical tips and important information to keep in the forefront of your mind.

In the large majority of hamstring strains, the injured muscle is biceps femoris (reported as 76–87%).[5] Semimembranosus injury is uncommon; semitendinosus injury is rare.



Chapter 3

Integrating evidence into clinical practice to make quality decisions

with CATHERINE SHERRINGTON

Randomized trials are for clinicians who are uncertain as to whether they are right or not—and I am certain I am right. Sir Ian Chalmers, quoting an unnamed orthopedic surgeon

This chapter opens with an exercise we use to introduce the concept of evidence-based practice to final-year students in human movement sciences/kinesiology (i.e. non-clinicians). If you are an experi-

arthritis. The students are given the information in the box (below) and are asked to suggest a treatment for Mrs J.

In our student exercise we call for a vote and

The importance of **evidence-based practice** is emphasized, with a new chapter on this topic. In addition, there is a comprehensive list of references at the end of every chapter.

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Part A

Fundamental principles

Chapter 1

Sports and exercise medicine: addressing the world's greatest public health problem

with JENNIFER DAVIS and STEVEN BLAIR

Exercise in the prevention of coronary heart disease: today's best buy in public health.
Jeremy Morris, 1994

The three previous editions of *Clinical Sports Medicine* focused on how to practice sports and exercise medicine. This chapter takes us back one step to "why?" Why practice sports and exercise medicine?

The burden of physical inactivity and sedentary behavior

Where to start? Surf the web, read any magazine, look around you as you walk down the street. The problem of physical inactivity is not subtle, and this chapter aims to provide a launching pad for the sports clinician—a key agent in the war against physical inactivity.

The one trillion dollar argument (US alone!)

The year 2000 seems like antiquity, but even then physical inactivity cost the US $1 trillion annually.[1] This information gained enormous exposure and the fundamental data and methods that underpin those calculations still apply. Methods to analyze economic burden of disease have been refined,[2] updated, and expanded[3] to include many additional costs. Thus, the costs of physical inactivity can only have increased in the past decade (Table 1.1). Note that a week of physical *inactivity* is estimated to incur the same health costs as a week of smoking.[4]

Physical fitness—more health benefits than smoking cessation or weight loss

Having identified that physical inactivity is a problem, we can look for a solution. How can the problem of physical inactivity be addressed? Physical activity! It is known that physical fitness provides more health benefits than smoking cessation or losing weight.[5] Numerous systematic reviews expound the many health benefits of physical activity, but systemic reviews are complex, predictable, and unemotional—a perfect combination for boring the general public and policy makers alike.

However, brief slogan-like ("sticky") messages (see also Chapter 16 for more on "sticky messages") are useful in helping convince people that physical activity is a remarkable medical therapy. Some include:

- For health, daily walking (30 minutes) is eight times as powerful as losing weight.
- Physical activity provides twice the health benefits as giving up smoking.[5,6]
- Low fitness kills more Americans than does 'smokadiabesity'—smoking, diabetes, and obesity *combined*.[7]

These sticky messages reflect data from Steven Blair's epidemiological study at the Cooper Institute in Texas (Fig. 1.1).[5] Note that "attributable fraction" refers to the proportion of deaths in the *population* that are due to the specific risk factor. It differs from "individual level" risk profiling.

The molecular mechanisms that explain the health benefits of physical activity

Experimental and mechanistic data shows how physical activity promotes health at the cellular and subcellular level. Some examples of exercise-induced health benefits at the molecular level that many patients find interesting and that can help motivate some are shown in the box opposite.

Table 1.1 Conditions precipitated by physical inactivity and resulting health care costs in the US

Unhealthy condition	Annual cost of condition in US$
Hypertriglyceridemia Hypercholesterolemia Hyperglycemia Insulin resistance Increased thrombosis Increased resting blood pressure Increased risk of myocardial ischemia Increased incidence of lethal ventricular arrhythmias Decreased cardiac stroke volume and maximal cardiac output	286.5 billion
Obesity	238 billion
Type 2 diabetes	98 billion
Breast and colon cancer	107 billion for all cancers
Osteoporosis	6 billion
Sarcopenia	300 billion for all disabilities
Back pain	28 billion
Gallstone disease	5 billion
Decreased psychological wellbeing	(cost not known)
Total	**1000 billion = 1 trillion**

ADAPTED FROM BOOTH ET AL.[1]

This was published in 2000 so is likely to be an *underestimate* today.

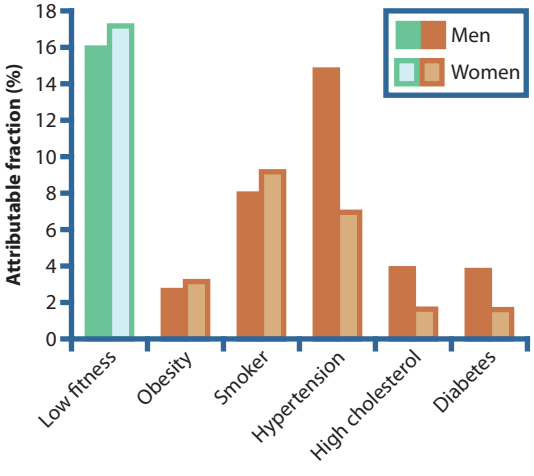

Figure 1.1 Attributable fractions (%) for all-cause deaths in 40 842 (3333 deaths) men and 12 943 (491 deaths) women in the Aerobics Center Longitudinal Study. The attributable fractions are adjusted for age and each other item in the figure

BLAIR[5]

Examples of exercise-induced health benefits at the molecular level

Preventing type 2 diabetes

Running on a treadmill stimulates key enzymes for energy sensing/signaling, including an important one called AMP kinase (AMPK). This protein helps remove fatty acids during muscular contraction and limits fatty acid biosynthesis. Exercise also enhances muscle membrane glucose transport capacity by recruiting a critical transport protein, GLUT-4, to the sarcolemma and T tubules where the protein can be active. Increasing the expression of GLUT-4 in skeletal muscle can be considered a crucial way of "mopping" glucose out of the bloodstream and into muscle and, hence, reducing the demand for insulin.[8]

Brain function

1. Both resistance training and endurance (aerobic) training can improve brain function. Convincingly,

continued

continued

the improvement in brain function can be shown using functional magnetic resonance imaging (fMRI) (Fig.1.2).

2. Animal studies show that improvements in brain function arise through improved blood flow and via particular hormones, including insulin-like growth factor 1 (IGF-1) and "brain derived neurotrophic factors" (BDNF).

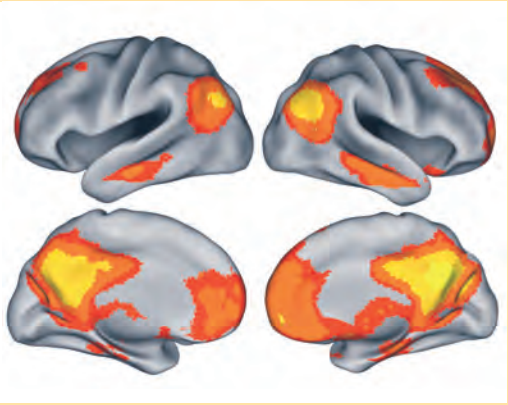

Figure 1.2 Sophisticated contemporary brain imaging, including functional MRI and "connectivity" mapping (illustrated), demonstrates that brain function improves with exercise training

Putting it all together—the economic imperative

"Exercise—the best buy in public health" concluded Jeremy Morris. A recent success story? Not at all! That was the title of a 1994 paper![9] The evidence has piled up since then: personal, regional, and national economic benefits accrue to those who are physically active.[6, 10] But how can we encourage adoption of this most powerful behavior—physical activity as medicine!

Practical challenges

Physical activity was not a societal burden when survival depended on it. Because we have engineered physical activity out of contemporary society, sedentary behavior is an easier choice. Similarly, poverty in various forms can make it very difficult for an individual to be active. Although clinicians are an important part of the team that promotes physical activity, society will need to make a concerted effort at various levels. This multilevel approach has been codified as the socioecological model of behavior change (Fig. 1.3).[11]

Consider the difference in likelihood of physical activity for Roald in Norway and a nameless inhabitant of a mythical urban wasteland. Roald's government provides tax benefits for healthy behavior and he lives close to a large forest with attractive walking paths. He can ride to work safely on a dedicated bike lane. His community promotes free public cross-country skiing by grooming and lighting the paths. His friends consider skating to be a great social activity. All levels of school-age children do at least 30 minutes of exercise each day as part of the school curriculum. All five elements of the socioecological model are working toward Roald and his family having an active lifestyle.

The darkest hour is just before the dawn

This chapter is written in a spirit of optimism. The human race has faced major challenges previously in public health and this problem—physical inactivity—is remediable. The remainder of this book is dedicated to keeping people active by preventing and treating musculoskeletal conditions in those who want to be active and by prescribing therapeutic exercise for those who have medical conditions.[12]

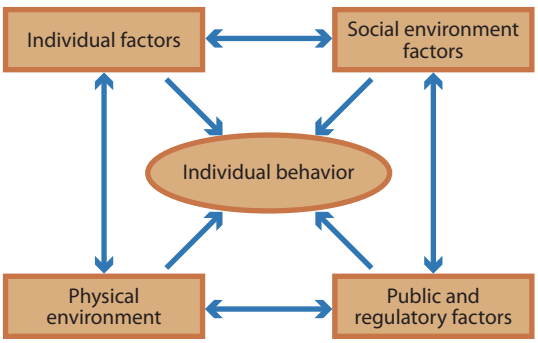

Figure 1.3 Socioecological model of physical activity

A

RECOMMENDED WEBSITES

British Journal of Sports Medicine: www.bjsm.bmj.com
Exercise is Medicine: www.exerciseismedicine.org
International Society of Physical Activity and Health: www.ispah.org

RECOMMENDED READING

Blair SN. Physical inactivity: the biggest public health problem of the 21st century. *Br J Sports Med* 2009;43(1):1–2.

Booth FW, Chakravarthy MV, Gordon SE, Spangenburg EE. Waging war on physical inactivity: using modern molecular ammunition against an ancient enemy. *J Appl Physiol* 2002;93(1):3–30.

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Chapter 3

Integrating evidence into clinical practice to make quality decisions

with CATHERINE SHERRINGTON

Randomized trials are for clinicians who are uncertain as to whether they are right or not—and I am certain I am right. Sir Ian Chalmers, quoting an unnamed orthopedic surgeon

This chapter opens with an exercise we use to introduce the concept of evidence-based practice to final-year students in human movement sciences/kinesiology (i.e. non-clinicians). If you are an experienced clinician or an expert on evidence-based practice you may want to skip over this chapter!

The "case" for the students to consider involves Mrs J, a 55-year-old woman. Students are told she presents with persistent knee pain due to osteoarthritis. The students are given the information in the box (below) and are asked to suggest a treatment for Mrs J.

In our student exercise we call for a vote and every year the first ballot results in option 1 (surgery) receiving about 80% of the votes! We then lead an open-class discussion and emphasize that the quality of the data should carry more weight than the clinical training of the person providing the advice. Students

Which evidence carries most weight?

You are asked to advise Mrs J, a 55-year-old woman with knee osteoarthritis, as to whether or not knee arthroscopy is a good idea. You have your own personal opinion, and you obtain the following four pieces of further information. Which of the four options carries the most weight with you? Would you advise that surgery is a good idea?

1. Dr X, an expert knee surgeon, advises in favor of surgery because "I have done hundreds of these operations and *obtained good or excellent results in over 90% of them*." The surgeon offers you and your friend the phone numbers of patients who can provide testimonials. You call a few of these patients and they all vouch for surgery.
2. A published study of cases done by another surgeon, Dr Y, shows that 75% of patients who have had this type of surgery reported improvements. Overall *75% of patients had an "excellent or good" outcome*. Patients were recruited and interviewed two years after the surgery.
3. A published study examined patients who had presented with knee pain to a specialist in osteoarthritis two years earlier. One group of patients had undergone arthroscopic surgery, the other had not. *Patients who had undergone surgery reported playing more golf and tennis than those who had not undergone arthroscopic surgery*. The paper concluded that surgery was associated with superior outcomes compared to conservative management.
4. A physiotherapy student obtained ethics approval to attend doctors' offices and recruit patients with knee osteoarthritis. The surgeon decided to allocate patients randomly to either "surgery" or "no surgery." Two years later, the student interviewed the patients again and found that *both groups of patients* (those who had had surgery and those who had not) *had similar levels of pain and function*. Both groups had pain scores of around 50 out of 100 where 100 is severe pain.

review the options and many begin to see the limitations of options 1, 2, and 3. The evidence in option 4 is designed to mimic an important randomized trial that addressed this question.[1] (We deliberately avoid the word "randomized" as students are sensitized to this being important, even before they really understand study design.)

The aim of this introduction to the course is for students to link quality of evidence and decision making. This sounds axiomatic, but our experience over many years reinforces that at first students fail to distinguish "evidence" from "eminence." Students find this practical exercise much more meaningful than a soporific lecture on "research methods." Students are then primed to engage with the literature with a view to making "quality decisions" together with patients.

Life before evidence-based practice

Clinicians trained after the year 2000 might be surprised that the term "evidence-based medicine" first appeared in 1991.[2] Professor Paul McCrory describes that dark period before as a time of "eminence-based practice" but he jests. Nevertheless, a certain amount of clinical training relied on wisdom passing down from mentor to mentor. When clinical trials were few, the opinionated veteran was king. This is understandable in an emerging field.

Sackett and the McMaster contribution

Dr David Sackett and colleagues from McMaster University[3–5] described a pedagogical approach to evidence-based practice (Fig. 3.1). This type of health care reflects "the conscientious, explicit and judicious use of current best evidence in making decisions about the care of individual patients. Evidence-based practice integrates individual clinical expertise with the best available clinical evidence from systematic research."[5] Since the mid 1990s, evidence-based practice has been facilitated by the Cochrane Collaboration (www.cochrane.org), which conducts and publishes high-quality systematic reviews of randomized trials of effects of interventions to address a wide range of health problems.

Applying Sackett's approach to the case of Mrs J (boxed item p. 11), we note that many patients with that clinical presentation have been encouraged to have immediate arthroscopy, based on "expert opinion." They have not been provided with the full range of options that have been evaluated in research. Armed with the information that is freely available through

'Evidence-based practice' is the integration of *best research evidence* with *clinical expertise* and *patient values*—*Dave Sackett*

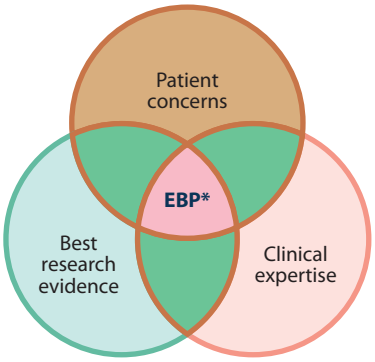

Figure 3.1 Schematic illustration of how clinical skills, evidence from research, and patient desire should overlap to provide the "quality decision" for the patient
* evidence-based practice

PubMed, the "best evidence" is that a well-conducted randomized controlled trial (RCT), systematic review, or meta-analysis (Fig. 3.2) suggests that arthroscopy is no better than placebo. The pieces of evidence provided in options 1 to 3 in the boxed item on page 11 represent a much lower level of evidence—data with much greater potential for bias and, hence, potentially flawed conclusions. However, "evidence" is not synonymous with randomized trials alone. If there is a question about clinical prognosis, or patient experiences, the best evidence comes from other study designs.[6] (See also Recommended reading.)

Different study designs provide different quality evidence (Fig. 3.2). The levels in this figure map

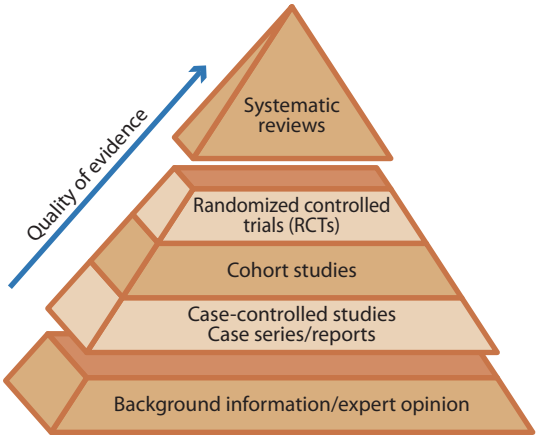

Figure 3.2 Hierarchy of study designs

closely, but not perfectly, to the Oxford "levels of evidence" shown in Table 3.1. We use the Oxford levels of evidence in this book.

Table 3.1 Levels of evidence

Level of evidence	Study design
Level 1	Systematic review of homogenous RCTs, individual RCT with narrow confidence interval
Level 2	Individual cohort study or low-quality RCT
Level 3	Individual case-control studies, non-consecutive cohort study
Level 4	Case series
Level 5	Expert opinion

This seems obvious—so what is the problem?

Evidence-based practice has intrinsic appeal; however, execution is the challenge. There are not enough individual RCTs, let alone systematic reviews or meta-analyses, to provide a body of evidence for every clinical encounter. For example, your patient might be an elite athlete who earns over US $150 000 per week; however, unfortunately, the relevant RCT was conducted in recreational athletes whose only reward was pleasure.

Also, clinical trials only provide data on "average effects" of interventions; your clinical experience means you can adjust those average effects to estimate what might happen in an individual patient. For example, a highly motivated individual might be expected to do better than average with an exercise intervention—where compliance is important.

In your office, you need to marry three things—the patient's wishes, the clinical evaluation you performed to make a diagnosis, and the evidence (Fig. 3.1). These elements were all part of Sackett's original definition of evidence-based practice.[7] Unfortunately, some "radical" advocates of pseudo-evidence-based practice forget the importance of the patient's wishes and your clinical evaluation, and they focus purely on the evidence component. If given license, those folk (usually non-practicing) disempower clinicians who work with real people; these radicals devalue clinicians' previous experience and patient wishes. If you are a clinician, don't be disempowered. Embrace evidence-based practice as additional value for your patients.

Evidence-based practice helps you distinguish evidence from propaganda (advertisement), probability from certainty, data from assertions, rational belief from superstitions, and, ultimately, science from folklore.[8]

By incorporating new evidence, your skills are continually updated—you are not stuck in a time warp where you practice today as you did in your year of graduation!

In summary, the health professions combine the art of caring for people with the best that science has to offer. The healing part can be likened to the community "shaman"—or healer. The patient's perspective and wishes are critical to reaching a "quality decision." The days of paternalism should be behind us. And remember that the plural of "anecdote" is not "data"![9]

In summary, the purpose of this chapter is to provide a perspective on evidence-based practice and to encourage interested readers to follow up with their own searches on the topic. Use the Recommended reading below. Evidence-based practice and clinical reasoning form key parts of the curriculum for students in all health disciplines; this chapter is not meant to provide a comprehensive text for that! Enjoy your evidence-based practice classes and remember that the key is to integrate clinical acumen with the evidence to meet the patient's needs (Fig. 3.1)! That way you'll make quality clinical decisions!

RECOMMENDED WEBSITES

Centre for Evidence-Based Medicine: www.cebm.net
The Cochrane Collaboration: www.cochrane.org

RECOMMENDED READING

Herbert R, Jamtvedt G, Mead J et al. *Practical evidence-based physiotherapy*. Edinburgh: Elsevier, 2005.

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Chapter 28

Hip-related pain

with JOANNE KEMP, KAY CROSSLEY, ANTHONY SCHACHE, and MIKE PRITCHARD

Bo says he felt his hip come out of the socket, so he popped it back in, but that's just impossible, no one's that strong. Bo Jackson's trainer after the American football and baseball All-Star dislocated his hip when tackled during the 1990 NFL playoffs

Until recently, the hip joint was not thought to be a significant cause of problems in the athletic population, although hip disorders have long been recognized in the pediatric population (Perthes disease, slipped femoral epiphysis) and older people (osteoarthritis). It was not until the advent of, firstly, MRI, and then hip arthroscopy, that it was realized that the incidence of hip labral and acetabular rim pathology was high, and that anatomical variants such as femoroacetabular impingement (FAI) were a common underlying cause of groin pain.

Hip pain is a common cause of activity restriction in sportspeople. Hip and groin pain is the third most common injury reported in the Australian Football League (AFL),[1] accounting for between 5 and 15% of all football-related injuries;[2] it is also prevalent in many other sports, including tennis, football of all codes, and hockey.

The likelihood of a sportsperson sustaining an injury to the hip joint can be increased by the demands of the sport, in particular, sports that require repetitive hip flexion, adduction, and rotation.[2–4] Hip joint injury can also be caused by the inherent individual anatomical variations within the joint, such as FAI or developmental dysplasia of the hip (DDH).[5–11]

The range of motion of the hip is critical in determining the likelihood of intra-articular damage during sporting activity. The demands of range of motion vary between all sporting activities and the levels of activity. As range of motion decreases, the risk of impingement-related damage increases, especially with contact sports.

Intra-articular hip pathologies contribute to both a reduced ability to participate in sporting or physical activities as well as pain and also reduced function during activities of daily living. There is also considerable evidence that hip pathologies are strong contributors to hip, groin, and pelvic pain in young adults.[12, 13]

Burnett et al.[14] demonstrated that 92% of patients with an arthroscopically confirmed labral tear complained of moderate to severe groin pain. Philippon et al.[10] described labral tears and FAI in 100% of professional National Hockey League (NHL) ice hockey players presenting for hip arthroscopy or the treatment of longstanding hip and groin pain. Injury to the ligamentum teres of the hip has been cited as the third most common cause of hip and groin pain in the sportsperson.[15–17]

In this chapter, we:

- review the functional anatomy of the hip
- provide a clinical approach to assessment of what is often a longstanding problem
- detail the pathologies and management of the many important conditions that are now recognized to cause pain around the hip region.

This chapter should be read in conjunction with the chapter on groin pain (Chapter 29).

Functional anatomy and biomechanics

The hip has three functions:

- It allows mobility of the lower limb.
- It transmits loads between the upper body, trunk, and lower limb.
- It also provides a stable base in weight-bearing activities.

The anatomical structure of the hip allows it to perform these functions.

The hip joint is supported by a number of dynamic and passive supports—these include its bony morphology, passive restraints such as capsule and ligaments, and a complex system of interplaying muscle groups. The biomechanics of the hip joint are generally under-reported in the literature and so are poorly understood. An appreciation of the functional anatomy of the hip and the role of the various structures surrounding the hip will assist in this understanding (Fig. 28.1).

Morphology

The hip joint (femoroacetabular joint) is a tri-planar synovial joint, formed by the head of femur inferiorly and the acetabulum superiorly. The acetabulum sits within the bony pelvis and is normally anteverted (forward-facing) by approximately 23°[18] (Fig. 28.2a overleaf). The acetabulum also faces inferiorly and laterally.

The head and neck of the femur are also anteverted—this refers to the most superior aspect of the femoral head and the femoral neck (Fig. 28.2b overleaf). This angle is normally between 10° and 15° in adults. The head of femur also faces superiorly and medially. A reduction in either femoral or acetabular anteversion is considered to increase the risk of hip pathology.

The relationship between the head and neck of the femur, called the head–neck offset, is also very important when discussing the hip joint. This refers to the difference between the greatest diameter of the spherical femoral head and the diameter of the neck measured around the femoral neck axis in any plane (Fig. 28.2c overleaf) and is normally approximately 20 mm in people without hip pain.[8] A reduced head–neck offset[19] (also referred to as a cam lesion) is considered to increase the risk of hip pathology and will be discussed in detail below.

The morphological structure of the hip joint allows the hip to achieve its three planes of movement, being flexion and extension, adduction and abduction, and external and internal rotation.

Acetabular labrum

The acetabulum forms the socket of the hip joint, and is lined with articular cartilage. The acetabular labrum (Fig. 28.3 on page 513) is a ring of fibrocartilage and dense connective tissue which is attached

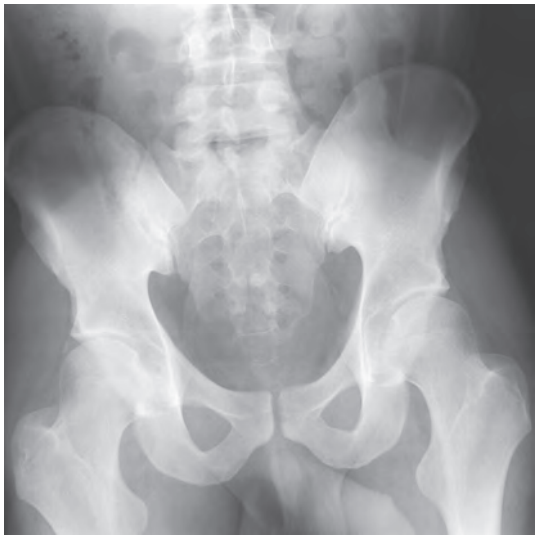
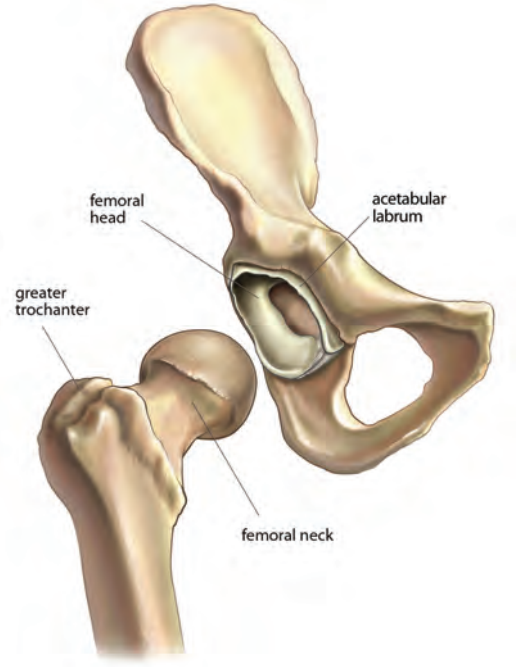


Figure 28.1 Anatomy of the hip and groin area
(a) Plain X-ray of the pelvis



(b) The hip joint

to the bony acetabular rim. The acetabular labrum is thinnest in its anterior aspect.

The blood supply of the labrum enters though the adjacent joint capsule. Only the outer one-third of the labrum is vascularized.[20] Nocioceptive free nerve endings are distributed throughout the acetabular labrum, suggesting a pain-producing capacity.[21, 22]

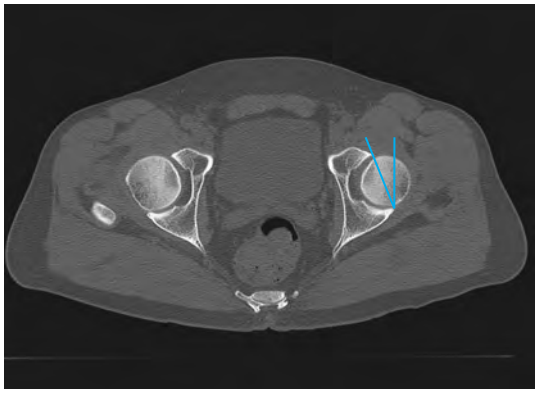
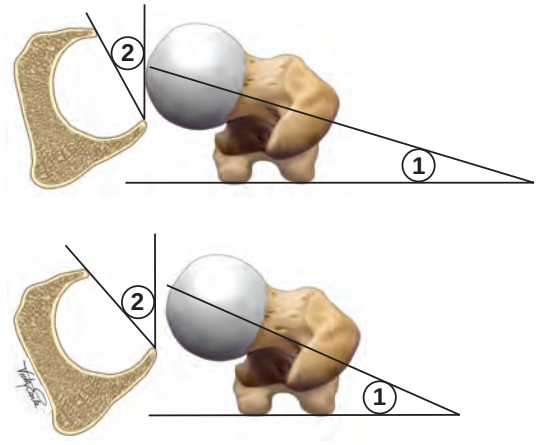
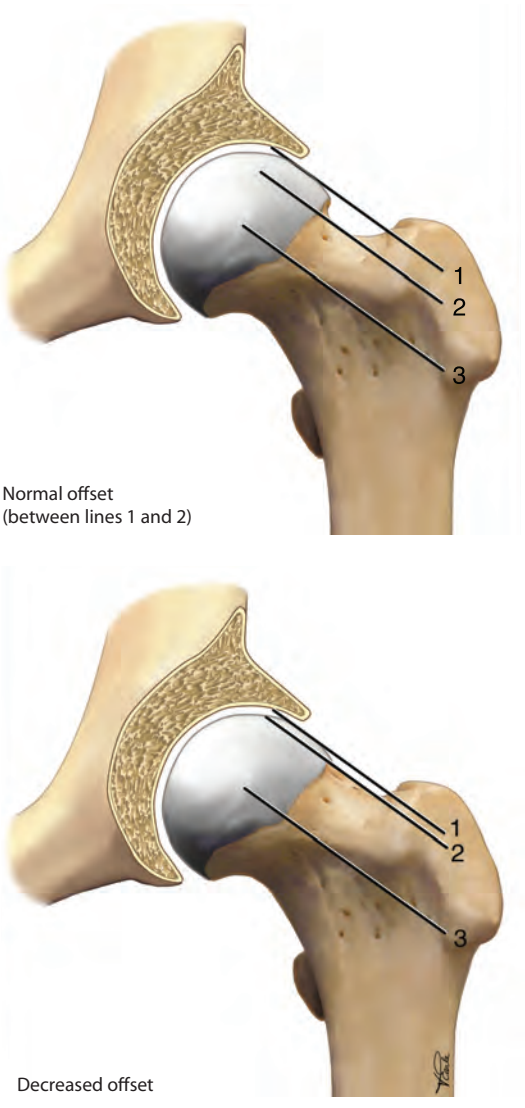


Figure 28.2 **(a)** CT showing acetabular anteversion[18]



(b) Transverse views of a normal hip (upper figure) and a dysplastic hip (lower figure)[19]
(1) Angle of torsion—rotation of the femoral neck relative to the shaft (transcondylar axis), normally 10–15° of anteversion. Dysplastic hips usually have increased angle
(2) Acetabular anteversion angle—the anterior direction of the acetabulum, is normally 20–40° of anteversion. Dysplastic hips usually have increased angle but it may be decreased



(c) Femoral head–neck offset[19]
The head–neck offset is the distance between the most superior aspect of the femoral head (line 1) and the femoral neck (line 2). Line 3 is first drawn through the long axis of the neck, then parallel lines are drawn representing the superior aspect of the femoral head (line 1) and the femoral neck (line 2). The offset is the distance between lines 1 and 2. A decreased offset (lower figure) leads to reduced clearance and subsequent impingement

The acetabular labrum has several functions. These are primarily to deepen the acetabulum, to distribute the contact stress of the acetabulum over a wider area (increasing contact area by 28%)[23] and assisting in synovial fluid containment and distribution.[22–24]

Ligaments of the hip

The transverse acetabular ligament (Fig. 28.3) traverses the acetabular notch, connecting the anterior and posterior edges of the labrum. The deepest layer of labral tissue blend into this ligament. The transverse acetabular ligament is under greatest load in weight-bearing, widening the acetabular notch and placing the transverse acetabular ligament under a tensile load.[24]

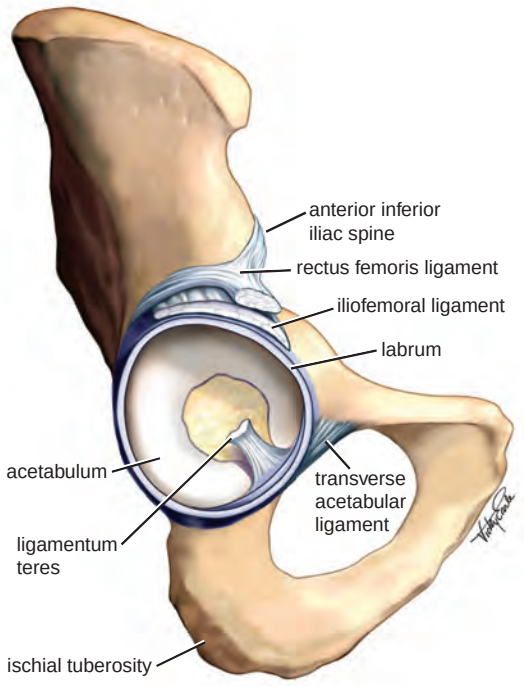


Figure 28.3 Transverse acetabular ligament, acetabular labrum, and ligamentum teres (resected)

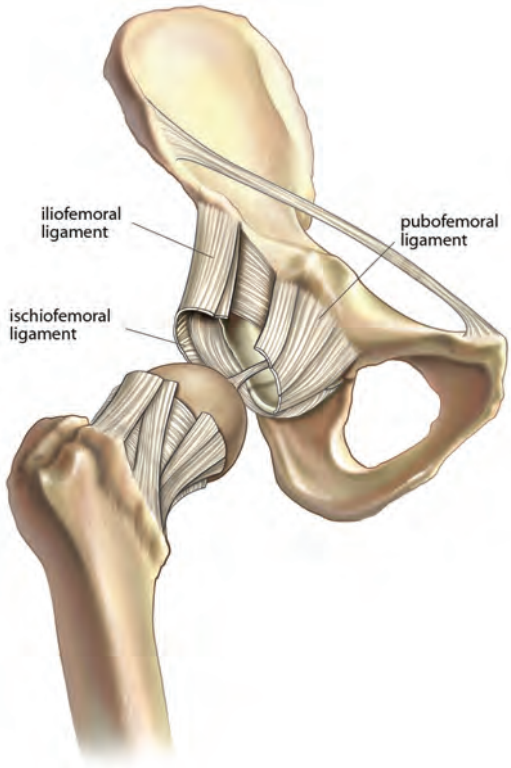


Figure 28.4 Capsular ligaments of the hip

The ligamentum teres (Fig. 28.3) is an intra-articular ligament, arising from the fovea of the head of the femur, becoming triangular in shape, with an anterior and posterior branch, which insert into the anterior and posterior aspect of the transverse acetabular ligament respectively.[25] It is covered by the synovium within the hip.[15]

The ligamentum teres is also rich in free nerve endings, which are mechanoreceptors.[26] The ligamentum teres was originally thought to be a histological vestige which becomes redundant early in childhood; however, it is now assumed that the ligamentum teres plays an important proprioceptive role, especially in weight-bearing activities.[15]

The iliofemoral ligament (Y ligament of Bigelow) reinforces the anterior capsule and originates from the anterior iliac spine, fanning into an inverted Y shape to insert into the intertrochanteric line (Fig. 28.4). It is taut in hyperextension and also provides stability in relaxed standing.

The pubofemoral ligament arises from the anterior surface of the pubic ramus and inserts into the intertrochanteric fossa (Fig. 28.4). It is taut in abduction and extension, and also reinforces the anterior capsule.

The ischiofemoral ligament arises from the posterior surface of the acetabular rim and labrum, and extends into the femoral neck just proximal to the greater trochanter (Fig. 28.4). Its fibers run in a spiral pattern and are also taut in hyperextension.

The iliofemoral, pubofemoral, and ischiofemoral ligaments act to restrain hyperextension, which is of particular relevance in relaxed standing.

Chondral surfaces

Both articular surfaces of the hip are lined with articular cartilage. These chondral surfaces rely upon adequate function of the synovium and movement of synovial fluid within the joint to provide nutrition, because articular cartilage is avascular. As both the acetabular labrum and ligamentum teres have been reported to attach to the synovium, they may also play a role in the nutrition and normal function of articular cartilage within the hip joint.

Joint stability and normal muscle function

The bony morphology, acetabular labrum, ligamentum teres, other ligaments, and capsule of the hip

joint all provide passive stability to the hip joint. Dynamic stability is provided by a complex interplay between various muscles surrounding the hip joint. The concept of deep hip stabilizers, the "hip rotator cuff," has been present for some years,[27] but has grown in popularity in recent years. In particular, the primary hip stabilizers are thought to provide a posterior, medial, and inferior force on the femur to control the position of the head of femur within the acetabulum. Ultimately, the dynamic control provided by the deep hip stabilizers has potential to minimize stress on vulnerable structures, such as the anterosuperior acetabular labrum, and the anterosuperior acetabular rim (Fig. 28.5).

Recent reports have described the roles of hip muscles, with respect to muscle morphology, primary action of joint movement, and lines of action in relation to the axes of joint movement[27–29] (Fig. 28.6). Some muscles have greater capacity to generate torque over larger ranges of motion (prime movers), while other muscles are better placed to act as dynamic hip joint stabilizers.

Detailed descriptions of muscle morphology have increased the understanding of the potential roles of individual muscles to act as dynamic hip stabilizers.[29] Muscles with a larger physiological cross-sectional area (PCSA) relative to muscle fiber length (MFL) (i.e. PCSA [cm²] : MFL [cm] >1.0) generate large forces over small length changes and, hence, are considered to be joint stabilizers[29] (Table 28.1). In contrast, those muscles with smaller PCSA relative to muscle fiber length (i.e. PCSA [cm²] : MFL [cm] <1.0) are considered to be "prime movers" of a joint. Generally the hip muscles tend to act as either joint stabilizers or prime movers. However, there are two muscles with both large PCSA and large MFL (gluteus maximus and adductor brevis), which suggests that these muscles are required to act as both stabilizers and prime movers.

The six short hip external rotators (SHER) (obturator internus and externus, superior and inferior gemellus, quadratus femoris and piriformis) have the capacity to provide hip joint compression and, hence, dynamic stability during most weight-bearing and non-weight-bearing activities.[28, 30] The gluteus medius is the dominant hip abductor, and is the primary lateral stabilizer of the hip during one-leg stance activities.[29]

For the patient with hip pain and/or pathology, the clinician should also consider the lines of actions for each of the deep hip stabilizers.[28] For example, although all of the SHER have capacity to provide

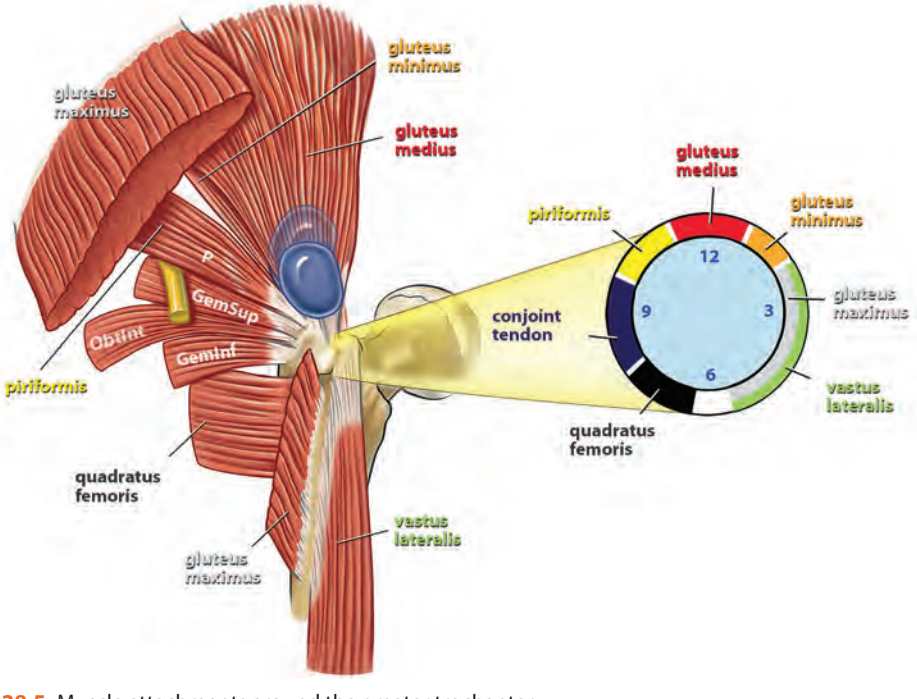


Figure 28.5 Muscle attachments around the greater trochanter

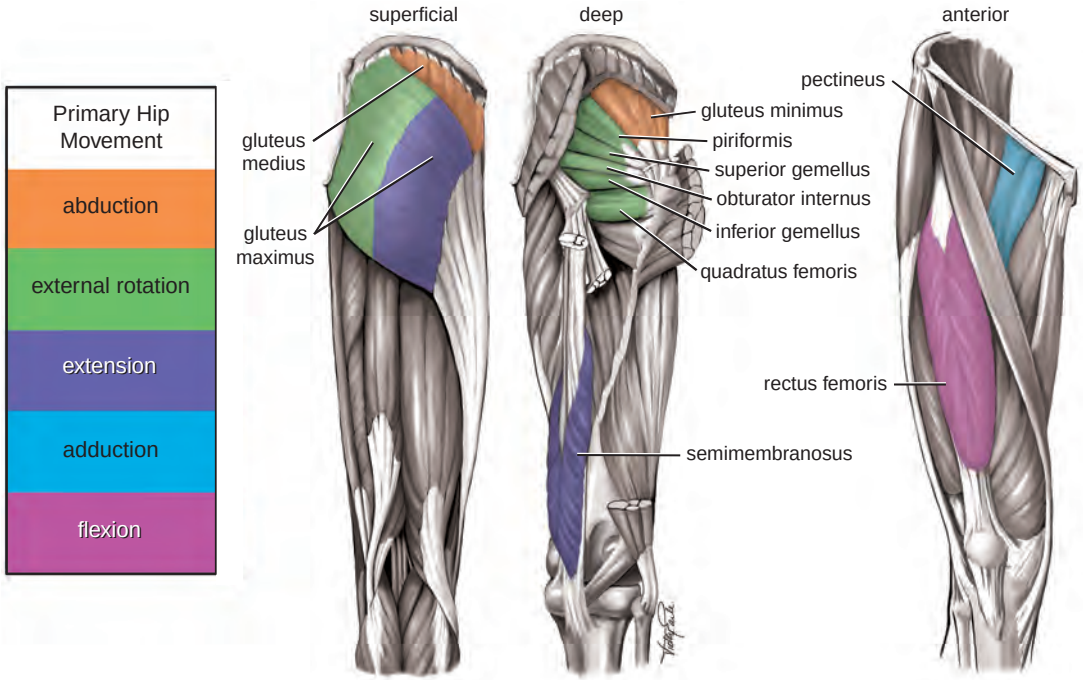


Figure 28.6 Muscles around the hip showing their primary direction of movement

Table 28.1 Hip muscles with primary function as hip stabilizer—primary and secondary actions[(a)]

Stabilizers (PCSA [cm^2] : MFL [cm] >1.0)	Primary action	Secondary action
Gluteus maximus	Extension External rotation	Adduction
Gluteus medius	Abduction	Extension External rotation (posterior fibers) Internal rotation (anterior fibers)
Gluteus minimus	Abduction	Flexion External rotation (posterior fibers) Internal rotation (anterior fibers)
Piriformis	External rotation	Abduction
Quadratus femoris	External rotation	Adduction
Obturator internus	External rotation	
Inferior gemellus	External rotation	
Superior gemellus	External rotation	
Obturator externus		External rotation Adduction
Pectineus	Adduction	Internal rotation
Semimembranosus	Extension	
Rectus femoris	Flexion	Abduction

[(a)] Primary action and line of action in relation to axis of movement of hip joint in the anatomical position[28, 29]

dynamic hip stability in the anatomical position, the quadratus femoris also has a line of action that is inferomedial. Therefore it has a greater capacity to resist superior translation of the hip. Similarly, the gluteus maximus and four SHERs (piriformis, gemellus inferior and superior, obturator internus) have a line of action that is posteromedial, and may be able to resist anterior force of the hip.[31]

In contrast, although the gluteus medius is an important lateral stabilizer of the hip, its line of action is both medial and superior[28] making it the greatest contributor to both medial and superior hip contact force during walking.[31] Furthermore, the anterior fibers of gluteus medius and minimus become hip internal rotators when the hip is flexed.[32, 33] The relevance of these factors to the rehabilitation of the patient with hip pain and pathology is described below.

Clinical perspective: making sense of a complex problem

Pain related to the hip joint is commonly seen in athletic populations. Of sportspeople with longstanding adductor-related groin pain, 94% have radiological signs of FAI.[34] Of sportspeople with hip and groin pain, 22% have labral tears and 55% of people with mechanical symptoms of the hip have confirmed labral pathology.[19]

However, despite this prevalence, hip-related pain and associated pathologies have not been well managed in the athletic population until recently. Weir et al.[34] reported a mean duration of hip and groin pain in sportspeople of 22 weeks, with the maximum duration 250 weeks (5 years), while many other studies report symptom duration of greater than 2 years.[19] Byrd and Jones[16] reported an average of 7 months from initial assessment, and multiple other diagnoses being made, before a definitive diagnosis of hip pathology was made.

Hip pain also commonly coexists with other groin-related pathology, such as adductor symptoms, iliopsoas symptoms and pubic symptoms. This makes definitive diagnosis and provision of appropriate management programs difficult and often multifactorial[35] (see also Chapter 29).

Causes of hip injuries and pain are shown in Table 28.2. Hints for differentiating hip pain from lateral thigh pain are shown in the box below.

Femoroacetabular impingement

Femoroacetabular impingement (FAI) describes a morphological variant seen in approximately 20% of the general population—it is not in itself pathology.[12] There are three types of FAI described.

Hints for differentiating hip joint and lateral thigh pain

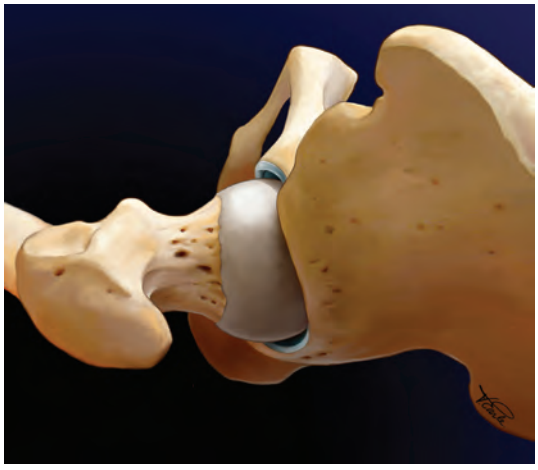
- Groin or anterior pain plus loss of range of movement, clicks, or catching generally means joint pathology.
- Lateral thigh pain plus full range of movement (once muscle spasm is taken into account) generally means lateral soft tissue structures.
- Both groin and lateral pain plus some joint restruction/signs generally means both.

Table 28.2 Causes of hip injuries/pain

Common	Less common	Not to be missed
Anterior pain		
Synovitis	Os acetabulare	Synovial chondromatosis
Labral tear	Ligament teres tear	Avascular necrosis of head of femur
Chondropathy	Stress fracture	Slipped capital/upper femoral epiphysis (SUFE)
Osteoarthritis	Hip joint instability (hypermobility or developmental dysplasia of the hip	Perthes
	Traction apophysitis (AIIS—rectus femoris; ASIS—sartorius; lesser trochanter—iliopsoas)	Tumor
Lateral pain		
Greater trochanter pain syndrome	Referred pain from lumbar spine	Fracture of neck of femur
Gluteus medius tears and tendinopathy		Nerve root compression
Trochanteric bursitis		Tumor

The first type is impingement due to a cam lesion (Fig. 28.7b), sometimes referred to as a Ganz lesion. This describes the reduction in femoral head–neck offset described previously, which results in additional bone being seen at the head–neck junction. This additional bone is also referred to as the "bump." Cam lesions are most commonly seen on the anterior, superior, or anterosuperior aspect of the femoral neck, and are seen in 78% of people with FAI.[36]

The second type of FAI seen is referred to as "pincer impingement" (Fig. 28.7c). This refers to bony change seen in the acetabulum and is seen in 42% of people with FAI.[36] This can either manifest as a deep acetabulum, which is most commonly seen anteriorly,[12, 37] or as a retroverted acetabulum, which leads to an apparent deeper anterior acetabular wall.



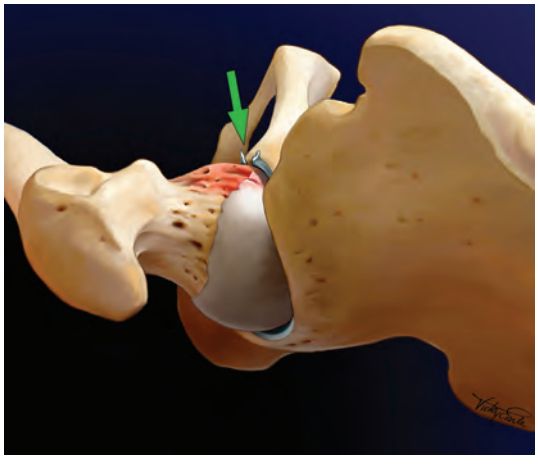
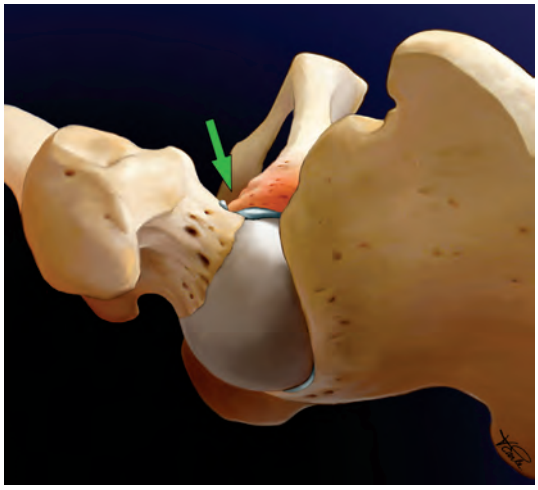


Figure 28.7 Hip joints with and without femoroacetabular impingement (FAI)
(a) Hip without FAI
(b) Cam lesion
Additional bone arises as a "bump" from the femoral surface



(c) Pincer lesion—bone spur extends from the acetabular surface

The third type of FAI seen is the mixed presentation where both cam and pincer lesions are seen; this is seen in 88% of people with FAI.[36]

It is unclear whether the development of cam deformity is due to overactivity at the epiphyseal plate between the femoral head and neck during periods of rapid growth in adolescents caused by repetitive torsional forces at the hip; or whether it is due to genetic tendencies. There is now some evidence that FAI has a familial pattern, with siblings being three times more likely to have FAI than controls.[38]

PRACTICE PEARL

FAI in itself is not a pathology, it is simply a variation of bony structure within the normal range seen.[38–40] Of the 20% of the population with FAI, only 23% of these people complain of hip pain.[36]

FAI, however, can result in pathology causing pain when the hip joint is placed into a position of impingement in a repetitive fashion during sporting activities, or in a single major traumatic incident such as a motor vehicle accident, or a large fall when the hip is in the position of impingement. The incidence of radiological signs of FAI in sportspeople with longstanding adductor-related groin pain has recently been reported as being 94%.[34]

The most common position of impingement is flexion with internal rotation and sometimes adduction.[2, 10, 39, 40] With most cam and pincer lesions located on the anterior or superior aspect of the hip,

these positions are most likely to bring these surfaces together and cause impingement.

Several authors have postulated an association between FAI and an increased likelihood of developing other intra-articular hip pathologies—these primarily being labral tears, chondropathy, and ultimately osteoarthritis of the hip.[5, 6, 8, 11, 38]

PRACTICE PEARL

As there is no conclusive evidence at this stage that indicates that all sportspeople with morphological features of FAI will develop hip pathology, prophylactic surgery to correct deformities in athletes who do not have signs of hip pathology is not recommended.

The early identification of FAI in sportspeople with hip and groin pain is essential. Unfortunately, there is no gold standard in clinical diagnosis of FAI. Clinical signs that are often reported to indicate the presence of FAI include reduced range of hip internal rotation, particularly when the hip is flexed, and a positive FADIR (flexion, adduction, internal rotation) test.

Positive FADIR testing is common in FAI-related damage and radiological examination is required. Plain radiographs can be useful and, generally, a correctly centered plain AP view of the pelvis, along with extended lateral femoral neck X-rays taken at 45° and 90°—the "Dunn views"—will indicate the presence of the morphological features of FAI when read by an experienced radiologist (Fig. 28.8).

Sportspeople who present with FAI and have hip or groin pain should be encouraged to avoid the position of impingement as much as possible. This position of impingement is usually flexion, internal rotation and adduction, or any combination of these (Fig. 28.9). This may involve activity modification

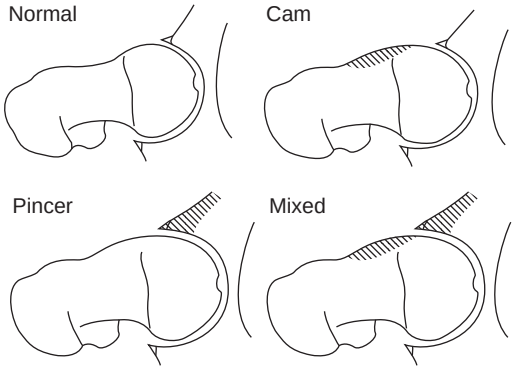


Figure 28.8 Radiological appearances of the types of FAI

Figure 28.9 Hip impingement during football

on a day-to-day basis, as well as during athletic pursuits. For example, in footballers, this may involve playing in a different position which requires less time changing direction and getting down low to the ball. It may also involve reducing the time spent on the field. Maximizing dynamic neuromotor control around the hip also assists in achieving this goal.

Factors that may contribute to the development of hip-related pain

Certain factors may contribute to the development of hip-related pain. These factors all alter the loads on the hip joint, thus placing structures within and around the hip joint under duress, which may eventuate in pain. These contributing factors can be classed as either extrinsic or intrinsic factors.

Extrinsic factors

Extrinsic factors include the type of sports played, particularly those involving repeated combined hip flexion, abduction and adduction, and loaded rotational or twisting movements. Extrinsic factors may

also include the volume of sport and activity undertaken, footwear worn, and type of surface played upon. Of these extrinsic factors, the type of sport and volume of load undertaken are probably the most important when evaluating the sportsperson with hip-related pain.

Repeated hip flexion, abduction and adduction, and rotation and twisting are reported throughout the literature as influencing the likelihood of the development of hip pathology. The clinician must examine these loads in detail and modify them accordingly for sportspeople who experience hip-related pain.

Intrinsic factors

Intrinsic factors can also influence the development of hip pain and pathology. These factors may also alter loads within the joint, predisposing the hip to injury. Intrinsic factors are considered as either "local" or "remote," and both must be considered for comprehensive assessment of the sportsperson with hip-related pain. Identifying these factors via thorough assessment is essential if the clinician is to successful modify the loads within the joint to protect potentially vulnerable structures.

Local factors

The following local factors may contribute to the development of hip-related pain. These are shown Table 28.3 overleaf.

Reduced hip flexion

Reduced hip flexion may indicate the presence of FAI. In sportspeople with hip pain, this must be assessed to ensure the sportsperson has adequate range of hip flexion to meet the demands of the sport, particularly sports that involve repetitive end-range flexion (e.g. football, gymnastics).

Hip flexion can be assessed reliably in supine position, with the contralateral thigh stabilized with a seatbelt (Fig. 28.10a on page 521), using either a goniometer or inclinometer. Any pain experienced at the end range of flexion must be noted.

Reduced hip internal rotation

Reduced range of hip internal rotation may also indicate the presence of morphological changes such as FAI, slipped upper femoral epiphysis (SUFE), Perthes, or dysplasia that may predispose the sportsperson to hip pathology. Many sports demand certain ranges of hip internal rotation, and these ranges must be established if a sportsperson is to participate in the sport without the risk of hip pain. This should be assessed in both a neutral range of hip flexion, and at 90° of hip flexion.

This range can be assessed reliably with either an inclinometer or goniometer, with the patient prone for hip neutral, and sitting for 90° of flexion, with the contralateral thigh stabilized (Fig. 28.10b on page 521).

Reduced hip extension

Reduced hip extension may predispose to hip pain, as it is possible that loads are placed on the anterior margins of the joint as the sportsperson attempts to gain more range during the end stage of stance in running and gait. The anterior margins of the joint are considered to be highly vulnerable to injury, and must be protected from overload. Hip extension range of motion can be measured reliably in supine position at the end of the plinth, with an inclinometer (Fig. 28.10c on page 521).

Increased femoral adduction/internal rotation during functional tasks

Increased femoral adduction and/or internal rotation during functional tasks may place the hip in a position of impingement, thus increasing loads on vulnerable joint margins such as the acetabular labrum and acetabular chondral rim. This is especially important for the patient with lost range of motion.

Increased femoral adduction motion should be assessed in both static and dynamic activities, such as a single-leg squat, walking, and running. Videotaping the sportsperson performing functional tasks may assist the clinician in identifying increased adduction/internal rotation (Fig. 28.10d on page 521).

Remote factors

The following remote factors may contribute to the development of hip-related pain.

Proximal factors

Increased pelvic tilt and/or lumbar hyperextension may increase the load on the anterior margins of the hip, due to the more distal placement of the anterior acetabular rim. This increased load may be a source of increased hip pain and eventually anterior hip pathology. The clinical assessment of pelvic symmetry and lumbar spine is outlined in Chapter 26.

Inadequate control of the lumbopelvic segments may result in a number of asymmetries, which alter the loads on the hip joint. In particular, lateral pelvic

Table 28.3 Local factors that can contribute to the development of hip-related pain

Factor	Possible mechanisms—structural	Possible mechanisms—functional	Confirmatory assessments
Reduced hip flexion range of motion	Cam lesion Pincer impingement Dysplasia		Plain X-ray AP pelvis Dunn view 45°/90° Positive FADIR
Reduced hip internal rotation range of motion	Acetabular retroversion Reduced femoral head–neck offset (cam lesion) Pincer impingement Femoral retroversion Osteoarthritis changes (osteophytes)		Plain X-ray AP pelvis Dunn view 45°/90°
		Reduced strength hip internal rotators Tight gluteals and piriformis Muscle spasm	Hand-held dynamometry Muscle length tests
Reduced hip extension range of motion	Acetabular anteversion Dysplasia		Plain AP X-ray
		Tightness hip flexors, quadriceps Reduced hip extensor strength Posterior pelvic tilt	Muscle length tests Hand-held dynamometry, and manual muscle tests
Increased femoral adduction/internal rotation motions	Developmental dysplasia of the hip Acetabular or femoral anteversion		Plain X-ray AP pelvis Dunne view
		Reduced hip abductor strength Reduced hip extensor strength Reduced hip external rotator strength Reduced neuromotor control/proprioception	Hand-held dynamometry, and manual muscle tests Single-leg balance challenge, and force platform

tilt may increase load on both the lateral and medial structures of the hip joint, due to the increased adductor and internal rotation moment seen on the stance leg.

Control of the hip and lumbopelvic control can be assessed using the single-leg squat (Chapter 8), other single-leg activities, and gait- or sports-specific activities. In some cases, the sportsperson should also be videotaped while running, particularly when fatigued, as altered control may become more pronounced. The demands on the lumbopelvic region for the individual's sport must be considered, as this may predispose certain athletic groups to fatigue and subsequently altered load on the hip joint.

Distal factors

Increased subtalar pronation may lead to an increase in tibial internal rotation. This may lead to an overload on the iliotibial band and the lateral structures of the hip. Increased iliotibial band tension leads to increased compression over the greater trochanter, and the development of gluteus medius and minimus

Figure 28.10 Assessment of local factors
(a) Measurement of hip flexion range of motion (using a simple inclinometer)

(b) Measurement of hip internal rotation range of motion in sitting

(c) Hip extension range of motion

(d) Increased femoral adduction and internal rotation moment

tendinopathy, and trochanteric bursitis. Increased tibial internal rotation may also create increased internal rotation of the femur, thus heightening load on the hip, particularly in sportspeople with an increased risk of impingement. The clinical assessment of the subtalar joint is outlined in Chapter 40.

Adequate range of ankle dorsiflexion during the stance phase of gait is essential in order to minimize excessive loads further up the kinetic chain. If this movement is limited, the gait pattern may be altered to achieve onward forward propulsion of the sportsperson. One adaptation commonly seen is an increase in hip adduction and rotation at the middle of the stance phase of gait. This may increase load on the hip joint in similar ways to those outlined above. The clinical assessment of ankle dorsiflexion range of motion is outlined in Chapter 38.

Clinical assessment

History

It is important to obtain a full history from the patient prior to undertaking a physical examination or obtaining any investigations. This history should include:

- age, general health, past medical history (including presence or absence of childhood conditions such as

"clicky hips," slipped upper femoral epiphysis (SUFE) or infantile dysplasia) and medications
- weight and height (BMI)—BMI >25 can increase severity of symptoms of osteoarthritis and tendinopathy, as well as increase joint loads
- exact mechanism of injury (if known), including onset as sudden or insidious
- time since onset of symptoms
- pattern of symptoms since onset (worsening, improving or not changing)
- family history—there may be a genetic predisposition to FAI and osteoarthritis of the hip[38]
- presence of mechanical symptoms such as locking, clicking, or giving way—suspect labral or possibly ligamentum teres pathology
- location of pain—hip pathology may present as groin, lower back, lateral hip, buttock, or thigh pain,[41] and secondary sources of pain such as muscle spasm may be present, complicating the assessment
- nature of pain (intensity, severity, constancy, time of day, latency)—will provide clues as to presence of inflammation, synovitis, bursitis, or tendinopathy in addition to intra-articular pathology
- neurological signs and low back pain—the lumbar spine can refer pain to the hip and should be eliminated as a potential primary source of pain
- aggravating factors—be specific regarding position of hip and potential for impingement during these activities, how long it takes for these activities to provoke pain, and latent pain
- current level of activity (frequency and intensity of lower limb loading)—tendon-related pain may need to be assessed over a period of three days
- factors easing pain—be specific regarding positions of ease as well as time required for pain to ease
- current sporting history—including level of sport (community, state, national, and position played within the team); certain positions will place the hip under more load, such as midfield in AFL football
- previous sporting history—certain sports played may increase the likelihood of a hip injury (e.g. dancing, gymnastics, martial arts, tennis, hockey)
- desired level of future sporting activity—this is important to establish in order to determine level of intervention as well as future risk of injury
- activities of daily living—including occupation, length of time spent sitting, amount of hip flexion and rotation and degree of manual labor within occupation, family situation including the presence of young children
- any past treatment, including investigations, conservative treatment, or surgical intervention.

Examination

Examination of the hip is in the *Clinical Sports Medicine* masterclasses at www.clinicalsportsmedicine.com.

Examination involves:

1. Observation
   - (a) standing
     - (i) general lower limb alignment
     - (ii) femoral alignment
     - (iii) pelvic symmetry
     - (iv) muscle tone and symmetry
   - (b) walking
     - (i) pain
     - (ii) limp
     - (iii) lateral pelvic stability—Trendelenburg sign
   - (c) supine
     - (i) leg length
2. Active movements
   - (a) hip flexion/extension
   - (b) hip abduction/adduction
   - (c) hip internal/external rotation at both neutral and 90° flexion
   - (d) bent knee fall-out
3. Passive movements
   - (a) adductor muscle stretch (Fig. 28.11a)
   - (b) anterior impingement test (hip quadrant)—flexion, adduction, internal rotation (Fig. 28.11b)
   - (c) internal rotation at 90° flexion
   - (d) flexion, abduction, and external rotation (FABER or Patrick's test) (Fig. 28.11c)
   - (e) quadriceps muscle stretch
   - (f) psoas muscle stretch/impingement (Thomas position) (Fig. 28.11d overleaf)
4. Tests of muscle function
   - (a) adductor squeeze test (Fig. 28.11e overleaf)
   - (b) hand-held dynamometry of hip muscle strength bilaterally
     - (i) flexion/extension
     - (ii) adduction/abduction
     - (iii) internal/external rotation
   - (c) Real-time ultrasound assessment of deep hip stabilizers and deep core
   - (d) de-rotation test[42] (Fig. 28.11f overleaf)
5. Palpation
   - (a) adductor muscles/tendons/entheses
   - (b) pelvis including pubis symphysis, ischial tuberosities, proximal hamstring attachment
   - (c) iliopsoas in muscle belly and at anterior hip joint (Fig. 28.11g overleaf)

(d) superficial hip abductors including tensor fascia lata, gluteus medius, superior gluteus maximus
(e) greater trochanter and tendons of gluteus medius and minimus

6. Functional movements
(a) single-leg squat (Fig. 28.11h on page 525)
(b) hopping (to reproduce pain)
(c) forward hop
(d) step up and down on the affected leg (observe stability, pain level, and pain location) (Fig. 28.11i on page 525)
(e) side step up and down on the affected leg
(f) hip hitch (in neutral, internal, and external rotation), keeping the knee extended (Fig. 28.11j on page 525)
(g) kicking (if appropriate)
(h) balance and proprioception

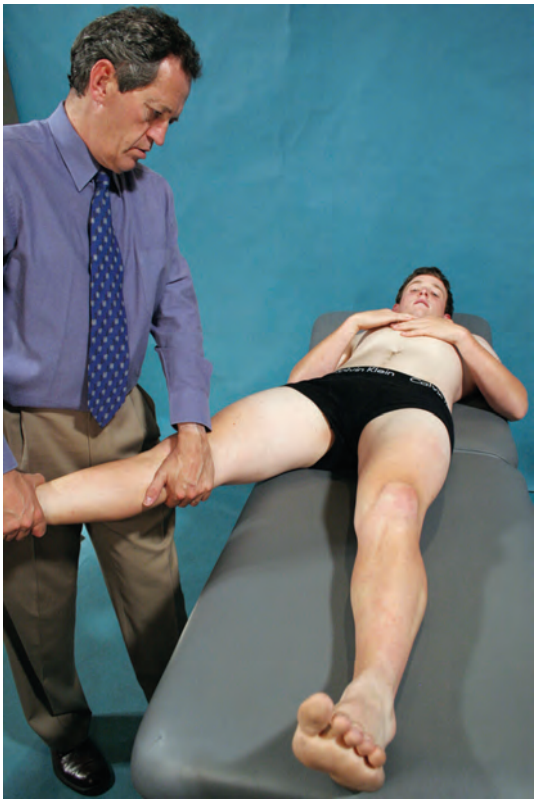
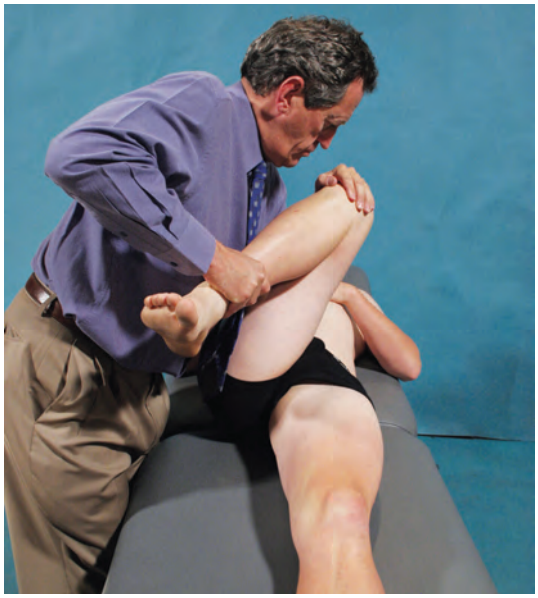
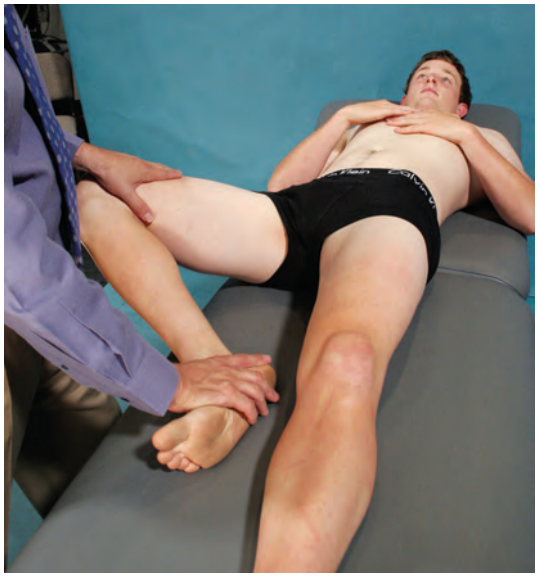


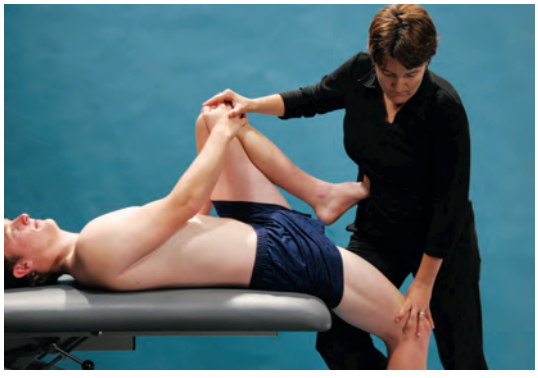
Figure 28.11 Examination of the patient with hip-related pain
(a) Passive movement—adductor muscle stretch



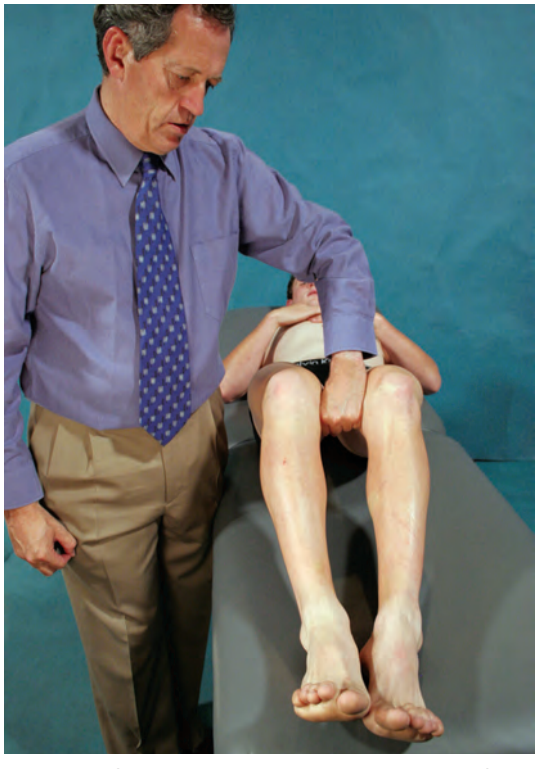
(b) Passive movement—anterior impingement (hip quadrant: flexion, adduction, and internal rotation—FADIR). This is a combined movement that is performed if hip range of motion is normal in single planes



(c) Passive movement—flexion, abduction, and external rotation (FABER or Patrick's test). Range of motion, apart from extreme stiffness/laxity, is not that relevant. Some caution needs to be exercised, as it is possible to sublux an unstable hip in this position. Pain felt in the groin is very non-specific. Pain in the buttock is more likely to be due to sacroiliac joint problems. However, pain felt over the greater trochanter suggests hip joint pathology



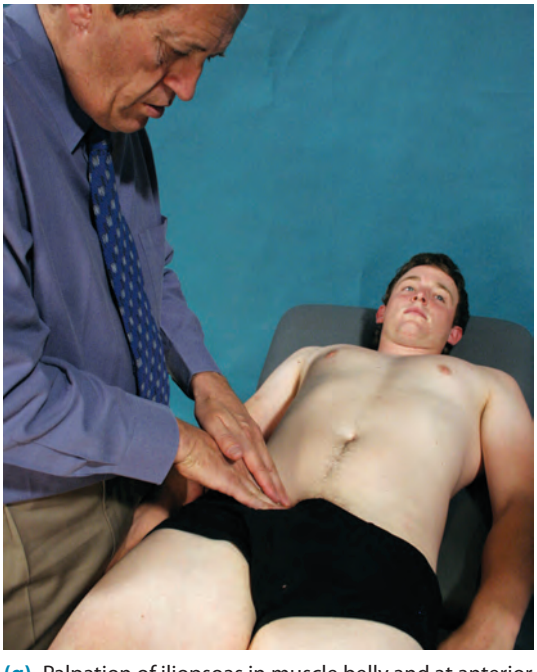
(d) Passive movement—psoas stretch (Thomas position). Pain in the hip being stretched suggests psoas abnormality. Pain in the hip being compressed can be significant for anterior impingement of the hip joint



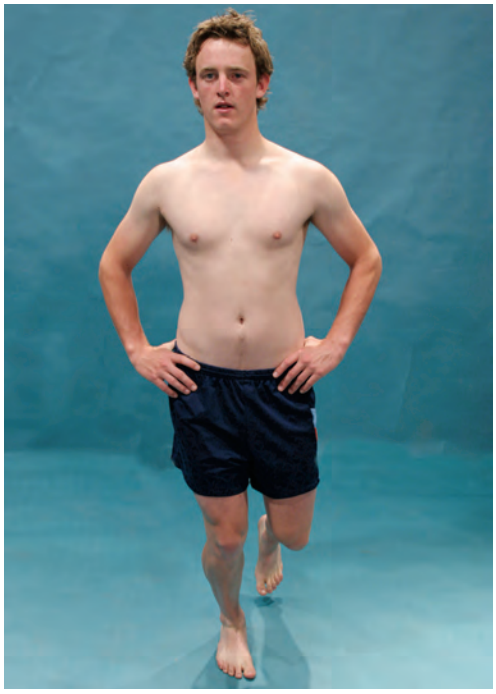
(e) Resisted movement—squeeze test. Examiner places fist between knees as shown. Patient then adducts bilaterally against the fist

We emphasize that each individual clinician needs to develop his or her own technique for examining this joint. Between the masterclasses online and this outline, you see a range of options you can incorporate into your practice.

(f) De-rotation test
In 90° flexion, the hip is taken into external rotation, and the patient asked to return the leg to the axis of the table against resistance. The test result is positive when the usual pain is reproduced



(g) Palpation of iliopsoas in muscle belly and at anterior hip joint



(h) Functional movements—single-leg squat. Note pain, degree of femoral adduction and internal rotation, and lateral pelvic stability—Trendelenburg sign

(i) Functional movements—step up and down (observe stability, pain)

(j) Hip hitch (in neutral, internal and external rotation), keeping the knee extended

Investigations

X-ray, MRI, and ultrasonography are the mainstay of imaging for diagnosis of hip pathology. Plain X-rays are often overlooked by clinicians, but usually should be the first investigation ordered as they can provide valuable information. FAI can often be seen on an AP view of the pelvis and Dunn views of the hip (Fig. 28.8). Similarly these X-rays can also guide the clinician to the presence of osteoarthritis, and abnormalities in morphology such as acetabular dysplasia, acetabular retroversion or anteversion, the presence of os acetabulare, and not-to-be-missed pathologies such as a slipped upper femoral epiphysis, Perthes disease, tumors, fractured neck of femur, and avascular necrosis. Unfortunately a plain radiograph does not provide information about soft tissue injuries such as labral, chondral, or ligamentum teres pathology.

CT scans are generally not utilized as a diagnostic tool for hip pathology, as most diagnostic information is usually obtained by clinical examination, a plain radiograph, and MRI. However, surgeons will often obtain specific CT scans preoperatively to

assist them in planning surgery for the treatment of FAI. The POD (Pritchard–O'Donnell) "position of discomfort" protocol CT gives surgeons specific three-dimensional images (Fig. 28.12) providing information regarding impingement sites, femoral head–neck morphology and version, acetabular overhang and version, and joint space loss anterosuperiorly. Surgeons may obtain specific CT scans preoperatively to assist them in planning surgery for the treatment of FAI.

MRI is commonly used in the diagnosis of soft tissue injuries of the hip. Pathologies such as labral tears, ligamentum teres tears, tendon and bursae pathology and, occasionally, chondral defects may be seen on an MRI scan. Unfortunately the sensitivity and specificity of MRI to these injuries is not high, and false-negative results are often noted.

Due to the depth of the joint, diagnostic ultrasound is not especially useful for the diagnosis of intra-articular hip pathology; however, it can be useful in determining the presence of bursae of the greater trochanter or iliopsoas tendon, and tendinopathy of these regions. Real-time ultrasound can be used to assess the function of the deep lumbar and hip stabilizing muscles.

Diagnostic injections of local anesthetic are used frequently in the hip to determine the presence of intra-articular pathology of the hip. These are generally performed under imaging guidance. A reduction in symptoms following an injection generally confirms the presence of intra-articular pathology, although a negative response does not necessarily indicate that no pathology is present, and further investigation and management may still be warranted.

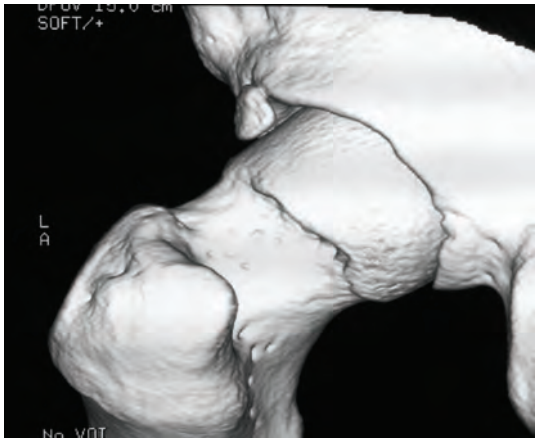


Figure 28.12 The POD (Pritchard–O'Donnell) "position of discomfort" protocol CT gives surgeons specific 3-dimensional images providing information regarding impingement sites, femoral head–neck morphology and version, acetabular overhang and version, and joint space loss anterosuperiorly

Labral tears

Tears of the acetabular labrum (Fig. 28.13) are seen commonly in the athletic population, with 22% of sportspeople with groin pain having labral tears, and 55% of patients with mechanical symptoms and hip pain having labral tears.[19, 22, 43–45] The etiology of labral tears is well described in the literature.

The presence of both FAI[5, 8, 10, 37, 46] and developmental dysplasia of the hip (DDH)[47, 48] has been repeatedly shown to increase the risk of a labral tear. This is thought to be due to impingement of the labrum in the presence of FAI and increased shear forces on the outer joint margins including the labrum, in the presence of DDH.

The prevalence of labral tears in the US and Europe is greatest anteriorly.[19, 22, 43, 49] Various causes for the high number of anterior labral tears have

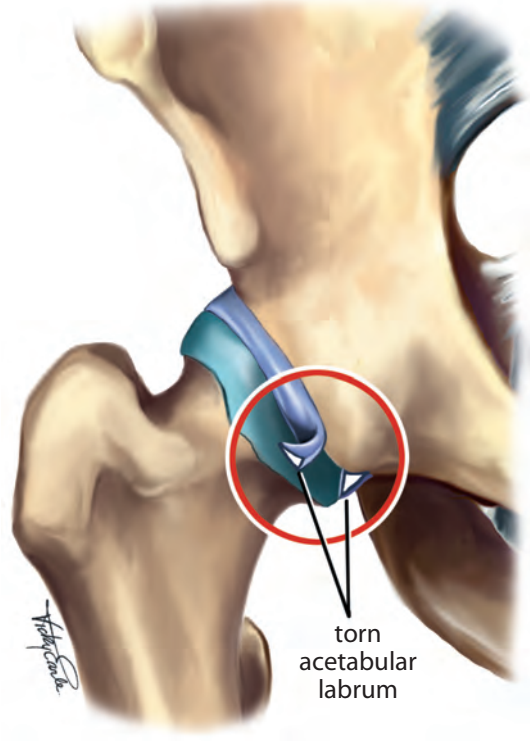
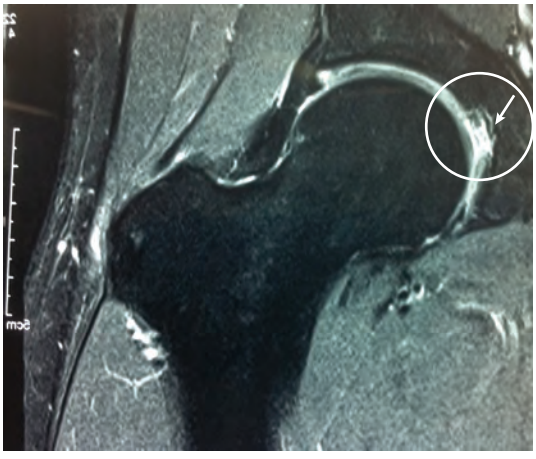


Figure 28.13 Labral tear
(a) Pathology



(b) MR arthrogram (MRA) with gadolinium contrast leaking through a labral tear (circle)

been postulated, including reduced thickness of the labrum anteriorly, the prevalence of FAI lesions seen anteriorly resulting in anterior impingement, and common functional activities, especially those with repetitive twisting and pivoting of the hip.[10]

The reduced bony support seen anteriorly in the hip due to the anteverted position of the acetabulum, which results in higher shear forces on anterior soft tissue structures, is also a likely cause of labral pathology. It has been shown that in the last 20–30% of the stance phase of gait, and in more than 5° of hip extension, increased forces are placed on anterior soft tissue structures by the head of femur.[19, 50]

Tears of the acetabular labrum are usually classified as type I or type II tears.[22, 51] Type I is described as a detachment of the labrum from the articular hyaline cartilage at the acetabular rim. Type II is described as cleavage tears within the substance of the labrum. The location of these tears relative to the vascularization of the labrum must be considered when establishing the potential for healing of the tear, and the most appropriate type of intervention.

The identification of labral tears in patients remains difficult. The patient often complains of mechanical symptoms such as locking, clicking, catching, and giving way. The location of pain is usually reported to be within the anterior hip or in the anterior groin region, although some patients report pain in the posterior buttock.

Clinical examination is also difficult, as most clinical tests have poor sensitivity and specificity regarding the type and location of pathology, although the FADIR (Fig. 28.11b) and FABER (Fig. 28.11c) are often described as being appropriate for the diagnosis of labral pathology.

Radiological investigations remain unreliable, with MRA the only investigation having a reasonable degree of sensitivity and specificity[19] (Fig. 28.13b).

Labral tears are often suspected, but not confirmed until patients present for hip arthroscopy, which remains the gold standard for diagnosis of labral pathology.[19, 22]

Sportspeople with labral pathology may respond to conservative management, and this should usually be trialed prior to undergoing surgery, except possibly in those patients with large bumps who are engaging in activity that demands more range of motion than the sportsperson safely achieves before impingement. Management should be directed to unloading the damaged labrum, which is almost always anterior and/or superior.

Repetitive hip flexion, adduction or abduction, and rotation at the end of range should be avoided through activity modification. Improving hip joint neuromotor control via activation of the deep stabilizing muscles, initially in an unloaded and then a progressively loaded manner, appears to assist in the unloading of the labrum.

Gait retraining may also be undertaken to minimize excessive hip extension at the end of stance phase of gait, as increased hip extension has been demonstrated to increase the loads on anterior hip joint structures.[52] Neuromotor control of the hip should be maximized and any remote factors influencing the mechanics of the hip should be addressed.

Ligamentum teres tears

Tears of the ligamentum teres (Fig. 28.14 overleaf) are seen frequently in sportspeople undergoing hip arthroscopy, and are being reported more frequently in the literature. Studies have found up to 70% of sportspeople undergoing hip arthroscopy for FAI and labral tears also have tears of the ligamentum teres.[10] Tears of the ligamentum teres are classified as:

- type I—a partial tear
- type II—a complete rupture
- type III—a degenerate ligament.[53]

The mechanism of injury for ligamentum teres most commonly involves forced flexion and adduction, and often internal or external rotation.[53] Twisting motions and hyperabduction injuries have also been reported to cause a tear to this ligament.

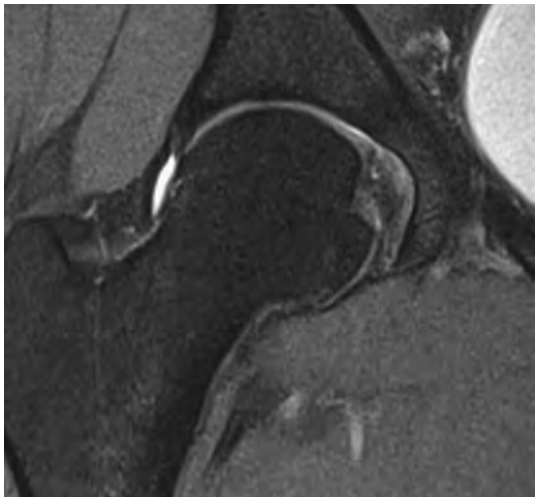


Figure 28.14 Ligament teres tear

With the likelihood of the ligamentum teres playing a large proprioceptive and stabilization role of the hip becoming increasing recognized, the prompt diagnosis and management of these injuries in the sportsperson is essential. Likewise, any surgical procedure that sacrifices the ligamentum teres through open dislocation should be carefully considered.

The symptoms of a ligamentum teres tear appear to be similar to other intra-articular hip pathologies, with most patients complaining of deep groin and/or medial/anterior thigh pain, catching, reduction in range of motion, and night pain.[53, 54]

Clinically these patients will also often present with significant increased tone of the adductor muscle group, and an injured ligamentum teres when touched with a radiofrequency probe intraoperatively can generate an adduction moment force powerful enough to reduce the hip while in traction.[55] These patients also present with reduced strength of the hip external rotators and extensors.

There are presently no established radiological or clinical tests that reliably identify tears of the ligamentum teres, and hip arthroscopy is the gold standard in diagnosis of these tears. However, Pritchard et al. have recently developed a clinical test that may have clinical utility.[56]

The test aims to place the femoral head and neck into a position that avoids bony and soft tissue impingement, while placing traction on the ligamentum teres by moving the hip through a full range of internal and external rotation.

The test is performed with the patient's hip flexed to full flexion without tilting of the pelvis (Fig. 28.15).

Figure 28.15 Position from which to internally and externally rotate the hip in the ligamentum teres test

The hip is then extended by 30 degrees. From this position (full flexion minus 30 degrees), the hip is moved into full abduction and then adducted 30 degrees (full abduction minus 30 degrees). The hip is then internally and externally rotated through full range. The presence of pain is considered a positive test.

Often ligamentum teres pathology is not confirmed until a sportsperson presents for surgery, but it should be suspected in episodes involving the mechanism of injury mentioned above. The principles of management of ligamentum teres pathology are similar to those for labral pathology, with a particular emphasis on regaining neuromotor control, excellent proprioception, and avoiding positions that place the ligament under most stress through activity modification.

The sportsperson with a ligamentum teres injury often presents with extremely overactive long adductors, which can be a source of considerable additional discomfort. This should also be managed with appropriate myofascial techniques, trigger point dry needling, and *gentle* stretching.

Synovitis

Synovitis (Fig. 28.16) is often seen in sportspeople with other intra-articular hip pathology—whether FAI, labral tears, ligamentum teres tears or chondropathy. One surgical study found synovitis coexisting in 70% of sportspeople with hip joint pathology.[2] It is rarely seen as a primary entity. Synovitis can cause considerable pain in the hip joint, with night pain and pain at rest being common presentations.

Synovitis is a concern to the clinician because of the pain and the associated changes in muscle

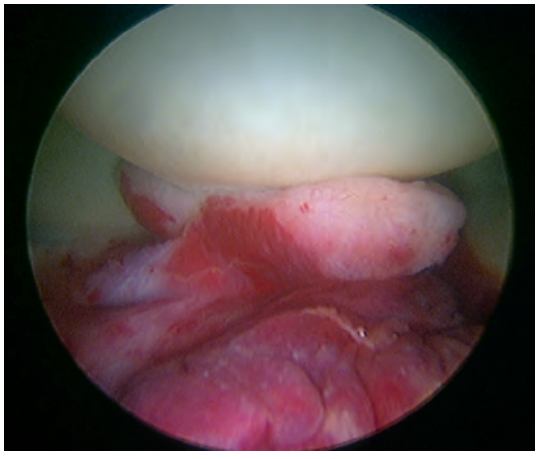


Figure 28.16 Synovitis

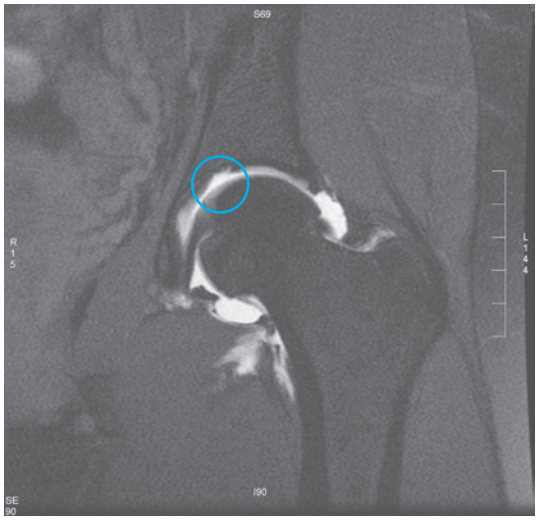
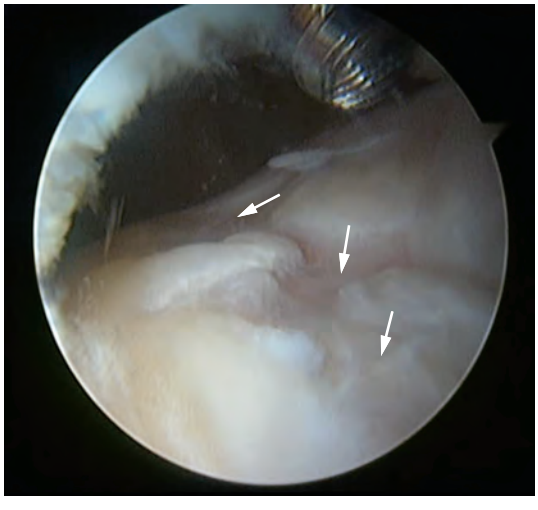


Figure 28.17 **(a)** Chondral lesion (acetabular side)



(b) Arthroscopic view of severe chondral damage secondary to FAI

activation that are seen around the hip in the presence of pain. In addition, the implications of synovial dysfunction on cytokine production, nutrition, and hydration of articular cartilage, which may already show signs of chondropathy, are significant for the long-term health of the hip joint.

Management should be aimed to addressing the other coexisting pathology, restoring normal neuromotor control around the hip, modifying loads, and also anti-inflammatory treatment such as oral non-steroidal anti-inflammatory drugs (NSAIDs) or intra-articular injection.

Chondropathy

Changes to the chondral surfaces of the hip are often seen in conjunction with other hip pathologies (Fig. 28.17). It is well reported that the presence of FAI,[5, 6, 11, 39] decreased acetabular anteversion,[7] labral pathology,[19, 48] and developmental dysplasia of the hip (DDH)[6, 7, 48] will lead to an increased risk of chondropathy and ultimately osteoarthritis of the hip. In patients with significant labral pathology, chondral loss is often up to 70% of the full thickness—or Outerbridge grade III or IV.[57]

A study examining hip pathology in AFL footballers found that full-thickness chondral loss of >30% of the acetabular depth anteriorly was found in 52% of players, who also all had labral tears and FAI.[2] It is also proposed that the presence of longstanding synovitis may also affect the nutrition of chondral surfaces, possibly exacerbating chondral damage.

The majority of chondral lesions are seen on the anterior or superior aspect of the acetabular rim, at the chondrolabral junction.[2, 57] This is not surprising considering that this is also the location for the majority of cam and pincer lesions, and the majority of labral tears.

Developmental dysplasia of the hip (DDH) also involves a reduction in the bony coverage of the femoral head by the acetabulum; thus the anteriorly directed forces of the femoral head will be concentrated on a smaller surface area on the anterior aspect of the joint.

The clinical diagnosis of chondropathy may be confirmed with plain radiographs, although early chondral changes will not visible. MRI may identify earlier chondral lesions, although the extent of

chondropathy is often only evident on hip arthroscopy.[2, 57]

Chondropathy is difficult to manage and may be difficult to confirm in the early stages without arthroscopic confirmation. If suspected, the management again is similar to that of labral pathology, as the majority of chondral lesions of the hip occur in the anterior aspect of the acetabular rim at the chondrolabral junction. As such, this region should be unloaded in the same fashion as labral pathology, with an emphasis on regaining normal neuromotor control of the hip. Recent evidence has shown atrophy in inferior gluteus maximus and hypertrophy in gluteus medius in osteoarthritis, with atrophy also occurring in gluteus medius in severe osteoarthritis.[58, 59]

Attempts to minimize synovitis should also be made, as the synovium and synovial fluid play an important role in articular cartilage nutrition. Obesity and lack of exercise aggravate the symptoms of osteoarthritis. The outcomes of hip arthroscopy for individuals with significant chondral loss are generally worse than for those with no chondral loss.[10, 17, 60, 61] Where chondral surface damage is found, conservative measures should be attempted first, and in some cases the sportsperson should be counseled to modify the amount of weight-bearing activities they undertake.

Rehabilitation of the injured hip

Rehabilitation of the injured hip requires careful consideration of the interplay between pain and loading (including progression of exercises and activities). Importantly, due to its role in all activities of daily living, including simple activities such as sit-to-stand, standing, and walking, it is hard to "rest" the hip. It is vital that the patient and the clinician have a good understanding around monitoring joint loads and the loading response.

The general principles of management of hip pathology are straightforward and consistently reported in the small amount of literature available concerning rehabilitation of the hip.[10, 19, 62–65] Unfortunately there is no evidence available other than clinical commentaries that evaluate the effectiveness of particular principles of rehabilitation of the hip.

The three most commonly reported principles of rehabilitation are shown in the box.

This section discusses each of these general principles, and then applies them to commonly seen conditions of the hip. We then outline the application of these principles of management to patients post–hip arthroscopy surgery.

Unloading and protecting damaged or potentially vulnerable structures

The most effective way to unload and protect specific structures of the hip varies slightly for different pathology, based on the understanding of the functional anatomy and biomechanics of the hip. When addressing the loads on structures outlined below, the principles of management of neuromotor control and remote factors should also be applied. Managing the load of the hip can be particularly difficult as the sportsperson has to walk about simply for activities of daily living. Thus it is vital that their ability to walk, stand, and perform everyday activities such as getting in and out of a chair is managed in such a way that these activities do not aggravate the underlying pathology.

Restoration of normal dynamic and neuromotor control

Restoration of dynamic and neuromotor control around the hip follows the same principles as other joints.

Phase 1: Deep hip stabilizer retraining

The short hip external rotator (SHER) muscles are those with the greatest capacity to provide dynamic stabilization of the hip (see above). Retraining of these deep hip stabilizers may be undertaken in the early stages of rehabilitation. As it does with other pain conditions,[66, 67] clinical observation indicates that pain appears to inhibit effective activation of the SHER muscles. Therefore, pain must be well controlled.

The initial step involves educating the patient in the role of the SHER muscles to provide dynamic hip stability, and the location and actions of these muscles. The second step involves facilitating independent

Three key principles of rehabilitation

1. Unload and protect damaged or potentially vulnerable structures within and around the joint.
2. Restore normal dynamic and neuromotor control around the hip joint.
3. Address other remote factors that may be altering the function of the kinetic chain.

contraction of these muscles. This is often best commenced in 4-point kneeling (Fig. 28.18a), where the patient is taught to activate the SHER muscles and then perform an isometric external rotation contraction against minimal resistance. The aim is to produce a low-level tonic hold of these muscles. In this position (90° hip flexion), the contribution from the larger external rotator (gluteus maximus) is reduced (see the section about joint structure and muscle function earlier in this chapter), thus enabling more specificity of activation for the SHER muscles.

Both the patient and the clinician must be confident that the deep hip stabilizers are activated and a real-time ultrasound machine may assist with providing feedback. Progression of the retraining includes providing different levels of resistance, number of repetitions, and speed of movements. Other progressions include increasing the amount of hip flexion, and decreasing the support (i.e. lifting one hand) to increase the balance demands and challenge to lumbopelvic stability.

Further progressions include activation of the deep hip stabilizers (Fig. 28.18b) in a variety of degrees of hip range of motion and in various functional positions as the activity of the sportsperson demands, and can be assessed using a real-time ultrasound in these varying positions. For example, a sportsperson who performs regularly in positions of hip flexion such as a deep squat should ultimately perform muscle activation in this position.

Figure 28.18 Deep hip stabilizer strengthening exercises

(a) Activation of the SHER muscles in 4-point kneel with theraband and resistance. The degree of difficulty can be progressed by decreasing or increasing the level of resistance, changing the speed of activation and increasing the number of repetitions. The challenge to the core can also be increased by lifting one hand off the floor in this position, and the degree of hip flexion or extension, and abduction or adduction can be altered based on the needs of the sportsperson

(b) Progression of activation of deep hip stabilizers into a closed chain position, ensuring adequate deep hip external rotators, gluteus maximus, lateral pelvic, and core stability

Phase 2: Gluteus maximus retraining

Gluteus maximus plays an important role in generating extension and external rotation torque, and has the potential to provide hip stabilization by resisting anterior hip force.[28, 31] Facilitation of independent gluteus maximus contraction may be best commenced prone (Figs 28.19a, b overleaf), where the patient is taught to perform an isometric external rotation contraction against minimal resistance (low-level tonic hold of these muscles). As with the SHER muscles, feedback may assist in ensuring that the muscle is activated. Since the gluteus maximus is more superficial, feedback may be provided by palpation, surface EMG biofeedback, or real-time ultrasound machine.

The activation of the gluteus maximus should be undertaken in a variety of degrees of hip range of motion determined by the functional demands the athlete's activity requires, and can be assessed using a real-time ultrasound in these varying positions. For example, hip abduction and external rotation, or hip adduction and internal rotation for a sportsperson who performs cutting maneuvers, or in hip flexion for a sportsperson who is required to perform in a deep squatting position. It should be then progressed from open chain to closed chain and then functional positions.

Figure 28.19 Gluteus maximus retraining exercises—examples of activation of gluteus maximus, in combination with SHER muscles in prone. Real-time ultrasound assessment can also be undertaken in this position

(a) Prone—knee extension

(b) Knee flexion

Phase 3: Generalized strengthening exercises

Generalized hip strengthening exercises should only be commenced when the patient and clinician are confident that the key stabilizing muscles can be activated and the activation maintained. During this phase, the aim is to restore muscle function (strength, endurance) and proprioception. This phase remains low-impact (Fig. 28.20a). Exercises should initially be undertaken with specific activation of the deep stabilizers prior to commencing the exercise. This ensures that the sportsperson has adequate control of the hip prior to placing it under load, which will assist in protecting vulnerable or damaged structures within the hip.

Generalized hip strengthening exercises should be undertaken, based on clinical assessment. For example, hip abductors (predominantly gluteus medius) should be targeted for those with reduced hip abduction strength.

Strengthening exercises need to be targeted to the needs of the individual, progressed according to patient responses, and targeted to the sporting/physical requirements. For example, a sportsperson who regularly jumps and lands (such as a netballer or gymnast) should incorporate these actions into their rehabilitation program (Fig. 28.20b).

Exercises are frequently commenced in prone (to ensure specificity and isolation of muscle activations) or in 4-point kneeling and then progressed into functional/weight-bearing positions, bilaterally and then unilaterally (Figs 28.20c–e).

Phase 4: Functional and sports-specific activity

Once good neuromotor control of the deep hip stabilizers and global hip muscles has been regained, functional and sport-specific activities should be assessed, and then undertaken, both to retrain these

Figure 28.20 Generalized hip strengthening exercises

(a) An example of low-impact functional retraining of deep hip stabilizers, ensuring adequate activation of these muscle groups in a challenging situation without excessive impact or load through vulnerable hip structures

(b) A jumping and landing task ensuring adequate activation of deep hip, lateral pelvic, and core stabilizers at take-off and landing

movement patterns but also ensure the sportsperson can cope with these activities without failing.

Any retraining of functional activities should focus on pre-activation of the deep hip stabilizers, adequate control of the lumbar spine and pelvis during the activity, and correct alignment of the femur during weight-bearing tasks (Fig. 28.21 overleaf).

Retraining of hip stabilizers should be performed in the positions that place the hip at greatest risk of overload, such as direction change and pivoting, deep squatting, and kicking. They should also be undertaken in a repeated fashion, again to ensure the sportsperson does not fail in a controlled environment.

Criteria for returning to sport

The decision regarding a patient's readiness to return to sport is made using clinical judgment of the individual's functional capacity. In the absence of robust scientific evidence, the following criteria are suggested:

- performance on the one-leg hop test (or other single-leg functional tests) at least 90% of the uninjured side (if unilateral symptoms)

(c) and **(d)** Two examples of later-stage functional activities with concurrent SHER muscle activation, core activation, and functional balance and proprioceptive challenges

(e) Examples of later-stage functional exercises incorporating deep hip and core stability with proprioceptive and neuromotor control retraining

- performance on strength tests at least 90% of the uninjured side (if unilateral symptoms)
- performance on strength test indicates balance of muscle strength (e.g. external rotation strength similar to internal rotation strength; flexion similar to extension, abduction similar to adduction).

Address other remote factors that may be altering the function of the kinetic chain

As outlined previously, a number of remote factors (e.g. lumbopelvic control) are likely to influence the rehabilitation of hip pain and pathology. Therefore, all potential contributing factors should be addressed and treated appropriately.

Surgical management of the injured hip

Hip arthoscopy is the gold standard for the diagnosis of early chondral, labral, or ligamentum teres pathology, and has indications ranging from diagnostic purposes through to removal of loose bodies, labral tears, chondral lesions, FAI, version abnormalities, and dysplasia.

Hip arthroscopy has evolved substantially over the last 10 years, with improved technique and dedicated instrument design. It is now commonly performed to manage intra-articular hip pathologies, including labral tears.[68] Hip arthroscopy has revolutionized hip surgery, since this minimally invasive procedure is associated with considerably less morbidity than open procedures.

Open surgical indications include femoral and acetabular osteotomies for dysplasia and for treatment of unusual FAI morphology. Internationally, the number of hip arthroscopy procedures now performed is growing rapidly, with 30 000 procedures performed in 2008, with this number expected to increase by 15% annually.[69]

The basic principles of arthroscopic hip surgery are to treat damaged tissues to allow the healing process to be maximized. This often involves debridement of the irreparable tissue, and stimulation of a

Figure 28.21 Retraining of functional activities—single-leg hop for distance

healing response. The joint is assessed for mechanical optimization to assist with healing and help prevent further damage through mechanical insult. Range of motion is critical in this assessment.

Clinically, patients presenting for hip arthroscopy surgery tend to be grouped into two categories:

- those diagnosed with soft tissue pathology resulting from bony morphological variations, requiring reshaping on one or both sides of the joint to increase the available range of motion prior to impingement
- those not requiring bony intervention but presenting with soft tissue injuries requiring intervention.

The first group includes patients with FAI which may be cam, pincer, or mixed impingement. This group have coexisting labral pathology, ligamentum teres pathology, or chondral lesions.

Incidental findings of FAI-related morphology without typical associated pain need no intervention, merely education and observation.

The second group includes those with soft tissue pathologies, but without morphological change requiring surgical intervention. Soft tissue pathologies include labral pathology, ligamentum teres pathology, chondral lesions, synovial pathology, loose bodies, crystalline hip arthropathy, infection, and any combination of these. Patients with these lesions may have co-existing issues such as dysplasia or hypermobility which predispose them to such injuries, but do not require surgical intervention. This group also includes patients with essentially normal morphology but who undergo a massive single episode of excessive range (usually rotation) which causes trauma to the associated soft tissues.

Hip arthroscopy is a demanding procedure with a steep learning curve requiring advanced training. Debridement of labrum, ligamentum teres, and chondral surfaces require less recovery time and less rehabilitation than more complex procedures (e.g. labral refixation and associated rim excision, and femoral head–neck reshaping for cam deformity).

The body of evidence examining outcomes following hip arthroscopy is growing rapidly, although most studies are case series (level IV) evidence. The majority of the literature focuses on outcomes following surgery for FAI, labral pathology, chondropathy, or combined pathology.

Three systematic reviews have examined outcomes following hip arthroscopy.[60, 61, 70] They each concluded that short-term outcomes are generally promising for hip arthroscopy treatment of FAI and labral pathology, although outcomes are generally poorer if significant chondropathy is observed; further long-term studies are required.

Ten-year outcome studies examining outcomes in hip arthroscopy have reached similar conclusions to the systematic reviews, mostly finding good outcomes unless significant chondral loss is present, with a number of those with significant chondral loss eventually progressing to joint replacement surgery.[17, 71, 72] These studies looked at all pathologies, and are somewhat limited by the enormous change in surgical technique that has occurred in the last decade.

Two specific studies looked at outcomes of hip arthroscopy in elite sportspeople.[2, 10] The populations included were AFL footballers and NHL ice hockey players, both of which have a high rate of hip and groin pain. Both studies examined FAI and labral pathology and found good short-term outcomes, patient satisfaction, and return-to-sport levels, although each study was limited by the short follow up of two years. Further longer term follow-up in sportspeople needs to be undertaken in order to conclusively understand the outcomes of these procedures in sportspeople.

Rehabilitation following hip arthroscopy

Rehabilitation following hip arthroscopy has been described in the literature in a number of clinical commentaries, and essentially follows the same conservative principles of management outlined above.[62, 64, 73] The individual pathology treated during hip arthroscopy must be considered when designing a postoperative rehabilitation program to ensure the hip is adequately unloaded and protected while healing. This generally involves a period of partial-weight-bearing as tolerated on crutches until a pain-free normal gait pattern is achieved.

Generally osteochondroplasties performed for the correction of FAI must be protected for at least six weeks, as should microfracture surgery performed for chondral defects.

Labral debridement and repairs should be protected for 4–6 weeks, ensuring the sportsperson avoids potential positions of impingement through activity modification and normalization of neuromuscular control around the hip.

Injuries to the ligamentum teres should be protected for six weeks by avoiding end-range positions that place the ligament under stress, and ensuring excellent neuromotor and proprioceptive control around the hip.

During this initial protective phase, the sportsperson should commence active rehabilitation of the deep hip stabilizers, initially in an isolated fashion, and then progressing into functional activity in a safe manner. During this time the therapist should also address any overactivity of the secondary stabilizers such as the long adductors, the proximal gluteals, tensor fascia lata, and the hip flexors.

Once this protective phase is complete, the sportsperson should undertake a dynamic rehabilitation program ensuring full strength of all muscle groups around the hip, normal function of the whole kinetic chain, and sport-specific activity. A full assessment of the muscle strength and function around the hip using real-time ultrasound and hand-held dynamometry at this time can also assist in providing targeted exercise programs to address any ongoing residual deficits in strength or muscle activation. Generally most sportspeople return to full sport between three and five months postoperatively following hip arthroscopy, although this varies depending on the level and type of sport played, as well as the specific pathology and surgery performed.

Os acetabulare

An os acetabulare (or os acetabuli) is defined as "a separated fragment of bone at the rim of the hip socket." They have traditionally been regarded as an unfused secondary ossification centre. The orientation of the cartilaginous growth plate is more *parallel* to the joint surface (Figs. 28.22 a, b).

In sportspeople, an os acetabulare is seen in conjunction with FAI and is thought to be a fatigue fracture (Figs 28.22c, d).

The separation line is *perpendicular* to the joint surface. Similar fatigue fractures had previously been described in severely dysplastic hips.

In a study of 495 patients treated surgically for FAI,[74] a large osseous fragment at the anterolateral rim was found in 18 hips. All patients presented radiographically with a femoral head showing an aspherical extension producing a "cam" impingement. Sixteen hips had a retroverted acetabulum, indicating anterior overcover. Preoperative MRIs confirmed a fragment composed of labrum, articular cartilage, and bone. The gap between the stable acetabulum and the rim fragment had a vertical orientation. All patients had been exposed to a physically demanding profession or contact sport, and in 15 hips no memorable traumatic episode was present. The mechanism

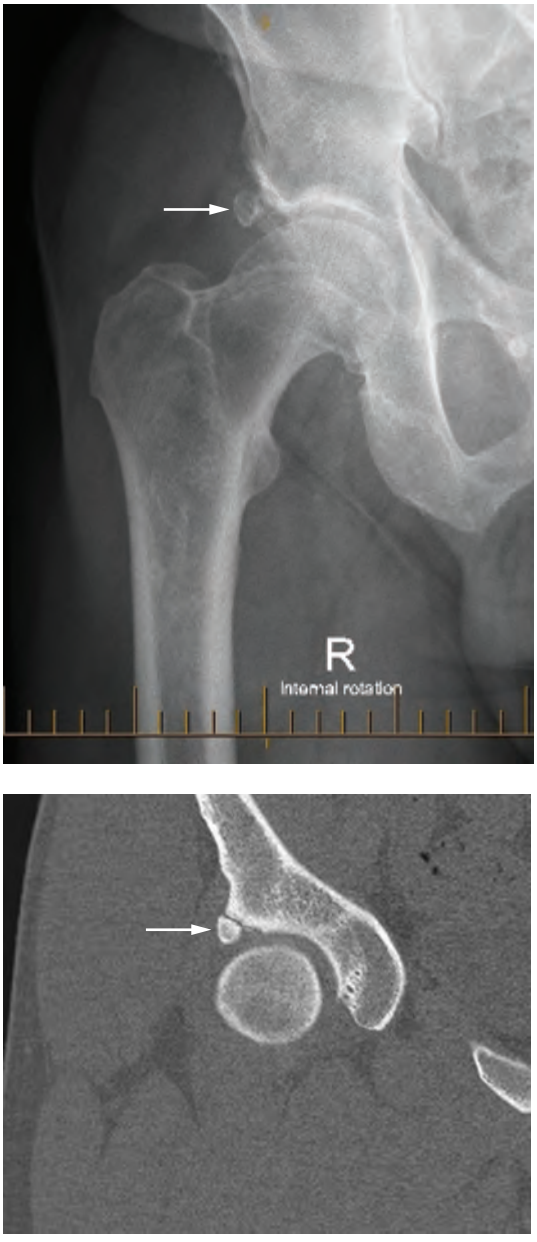
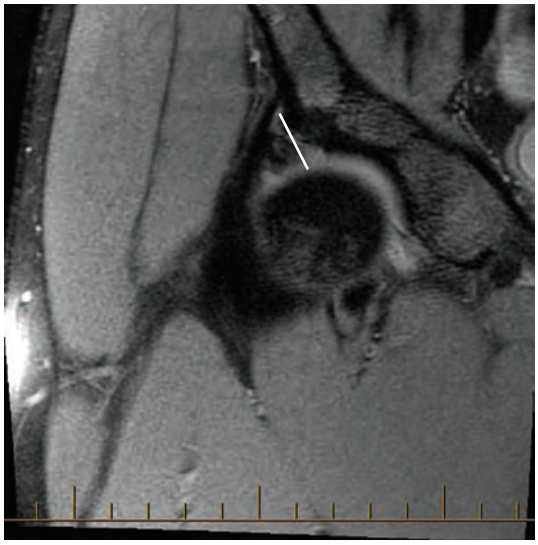
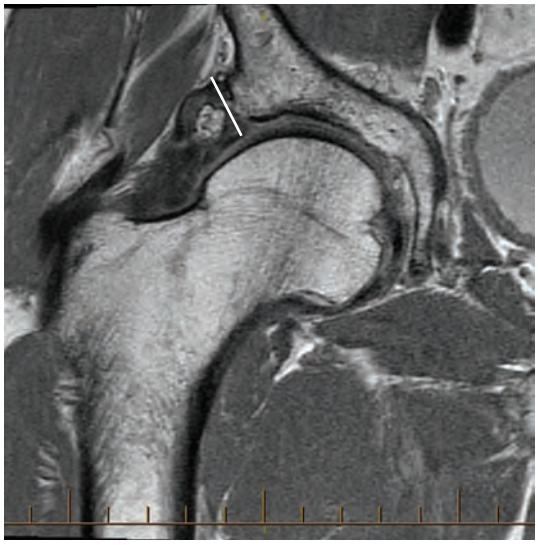
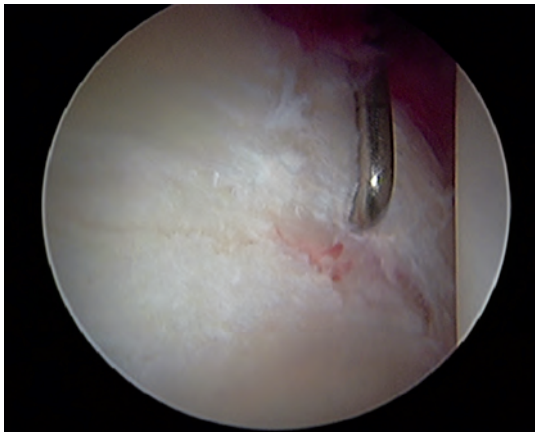


Figure 28.22 Os acetabulare (arrows)
(a) X-ray **(b)** CT scan of true os acetabulare showing orientation of the cartilaginous growth plate is more *parallel* to the joint surface

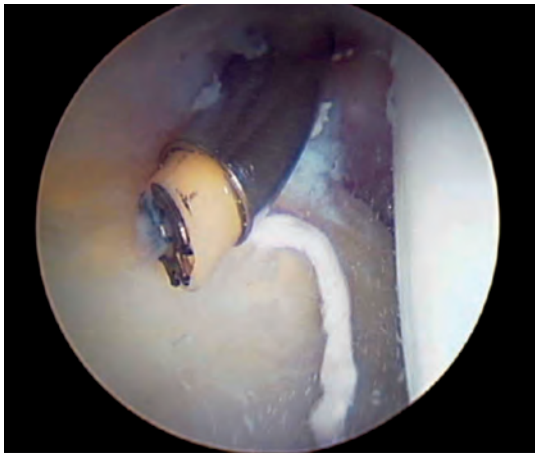




(c) and **(d)** are MRIs of os acetabulare due to fatigue fracture. Note the separation line *perpendicular* to joint surface



(e) The labrum and articular cartilage can be damaged at the site of the os acetabulare and can be unstable to probe



(f) Toothpaste-like calcific debris may be removed from the labrum

leading to this acetabular rim fragment is thought to be fatiguing due to femoro-acetabular impingement. The aspheric portion of the head is jammed into the acetabulum and with time causes a stress fracture of the area of overcoverage of the anterior acetabulum.

The treatment of symptomatic os acetabulare is commonly achieved during hip arthroscopy. During acetabular rim assessment, the labrum and articular cartilage is probed. It is common for the labrum and articular cartilage to be damaged at the site of the os acetabulare (Fig. 28.22e) and can be unstable to probe. During labral takedown and rim excision, the os acetabulare is removed, and the labrum refixed to the rim of the acetabulum. If the fragment of bone is very large, removal may result in insufficient bony coverage. In these cases, refixing of the fragment is preferred.

Occasionally, toothpaste-like calcific debris (Fig. 28.22f) similar to calcific tendinopathy of the shoulder is removed from the labrum, and is thought to represent a response to labral injury, which can mimic os acetabulare on plain X-ray.

Lateral hip pain

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Lateral hip pain is a common presentation particularly among distance runners and women over the age of 40. Traditionally lateral hip pain was thought to be due to trochanteric bursitis. However, it appears that tendinopathy of the gluteus medius and/or minimus and bursa pathology probably co-exist. The term "greater trochanter pain syndrome" (GTPS) is now used to describe this condition.[75, 76]

Greater trochanter pain syndrome (GTPS)

The anatomy of the greater trochanter and its associated tendons and bursae is shown in Figure 28.23. There are two bursae around the greater trochanter. The gluteus medius bursa lies beneath the tendon of the gluteus medius and medial to the greater trochanter. The trochanteric bursa is lateral to the greater trochanter.

Gluteus medius tendinopathy presents with tenderness to palpation of the gluteus medius muscle, and can be triggered by sudden falls, prolonged weight-bearing on one extremity for long periods, activity overuse, or sporting injuries. Most commonly, this situation is observed in middle-aged women who have commenced unaccustomed exercise (e.g. vigorous walking or joining a gymnasium).

Patients report pain over the greater trochanter which may extend into the lateral thigh, and even the lateral leg. The pain tends to be episodic but worsening over time. Frequently, pain lying on the affected side at night is the most distressing symptom, although pain with, or following, weight-bearing activities is likely to be identified. As with other tendon problems, the cumulative load over three days needs to be drawn from the patient in order to identify the aggravating factors.

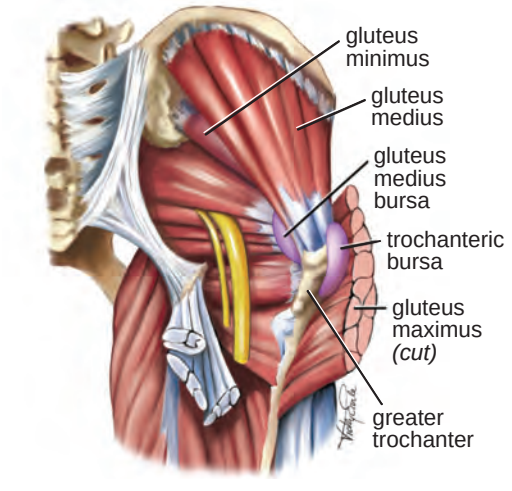


Figure 28.23 The anatomy of the greater trochanter and its associated tendons and bursae

Palpation of the greater trochanter produces the "jump sign"—the person nearly leaps off the bed. Range of movement tests for flexion, adduction, abduction, and the rotations in 0° and 90° flexion are normal or slightly increased, although muscle spasm may affect these. The FABER test is frequently positive, while Ober's test (Chapter 34) may or may not be positive.

Resisted external rotation and abduction muscles tests are reported to aid with the diagnosis; however there is very limited evidence to support this. The de-rotation test (Fig. 28.11f) may assist.

The step up and down test (Fig. 28.11i) may help differentiate between tendinopathy, tears, and hip osteoarthritis. Those with more severe GTPS report higher levels of pain with stepping up forwards onto the step, and down sideways off the step. Those with less severe presentation have pain with the hip hitch with external and/or internal rotation. A report of groin pain with these activities is likely if the person has hip joint pathology (e.g. chondropathy, osteoarthritis) in addition to lateral hip pain.

A Trendelenburg gait, and weakness may be present. Differentiating between pain inhibition and true weakness is important. These symptoms specifically affect runners, possibly due to the tilting of the pelvis with running.

Diagnostic ultrasound can be performed to determine if fluid is present in the bursa or thickening exists about the bursa, and to look for echogenic changes that are consistent with tendinopathy and tears.[77, 78] Magnetic resonance imaging (MRI) demonstrates tendinopathy and tears of the gluteus medius[79, 80] (Fig. 28.24).

Treatment

The principles of treating GTPS are similar to the treatment of other tendinopathies:

- Control pain by minimizing the compression on the greater trochanter and managing the load on the tendons.
- Strengthen the gluteal muscles.[81]
- Treat the comorbidities.[82]

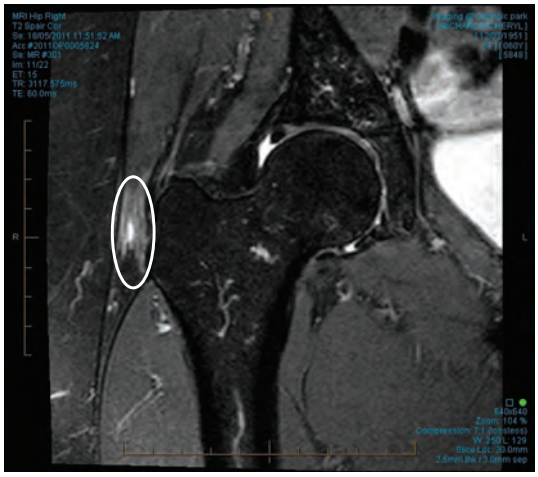
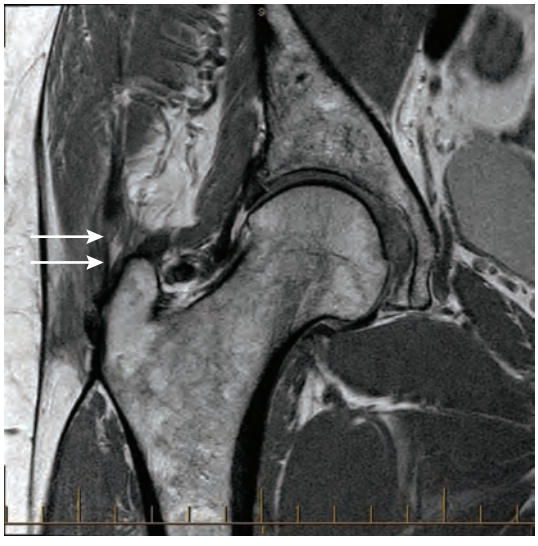


Figure 28.24 MRI appearances of gluteus medius abnormalities
(a) Tendinopathy



(b) Tendon tear

Managing pain

In the acute phase, treatment of GTPS consists of relative rest, ice, iliotibial band soft tissue work to improve compliance, taping, gaining good gluteal muscle control, and NSAIDs and paracetamol. Patients should be checked for hip abduction and rotation control in activities of daily living (e.g. getting out of a chair, climbing stairs, and standing). As the patient improves, sport-specific activities such as running, jumping, and hopping should be analyzed. Runners should avoid banked tracks or roads with excessive camber when resuming their running program.

Shock wave therapy has been shown to be effective in the treatment of GTPS.[81, 83]

Recalcitrant cases may respond to a local corticosteroid injection. A peritendinous ultrasound-guided corticosteroid injection has been shown to be an effective treatment of gluteus medius tendinopathy; 72% of the patients showed a clinically significant improvement in pain level, which was defined as a reduction in the VAS pain score of ≥30%.[84] However, another study demonstrated that less than 50% of subjects had a positive outcome three months after the injection.[81]

It is essential that a corticosteroid injection is only regarded as one part of the treatment—as a means to reduce pain and enable the patient to commence a muscle strengthening and postural control program, which is the key to the treatment process.

Strengthening the gluteal (and other lower leg/trunk) muscles

The following exercises are designed to enhance the control and strength of the gluteal muscles in people with GTPS, and can be taught in addition to lumbopelvic control work (Chapter 14). Importantly, in people with GTPS, positions of hip adduction may be associated with increased pain and hence exercises such as "clams" into hip adduction may be best avoided.

In prone, with the leg slightly abducted, knee flexed 90°, the patient is instructed to medially and laterally rotate their hip within pain limits. Gaining excellent control of this movement through range—including lateral rotation—provides both concentric and eccentric activity of gluteus medius and minimus muscles in an unloaded situation. This exercise should be repeated in varying degrees of hip flexion (prone over pillows or a bolster), as this targets the anterior fibers of gluteus minimus and medius and the middle fibers of gluteus medius. Progression of this exercise is to add ankle weights (using response to load as a guide).

Hip extension and abduction in prone over pillows (hip flexion) and/or bilateral bridging (knees and feet apart to reduce hip adduction) is also useful (Fig. 28.18 on page 531).

Hip abduction strengthening should be avoided in the initial stages of GTPS because it provokes symptoms. Hip abduction should only be commenced when the patient has good control of the deep hip stabilizers, and it should commence in positions of

hip abduction initially. As the acute stage resolves, hip abductor strengthening is important and may be achieved in the aquatic environment.

As the patient gains control and strength of gluteal muscles, the clinician should progress the difficulty of exercises. Options include single-leg standing with contralateral hand on a bench or backing against a wall (Fig. 28.25a) to gain static pelvic control, progessing to no assistance (Fig. 28.25b), and more challenging balance exercises (Fig. 28.25c). Rubber-band type (resistance) exercises need to be introduced very carefully, and the response monitored carefully. Pilates-style sliding boards have also been used (Fig. 28.25d).

Treat the comorbidities

Hip-related comorbidities (e.g. osteoarthritis, labral tears) frequently coexist with GTPS, and they should be addressed as outlined above.[82] Furthermore, referred pain from the spine should be assessed and treated as appropriate.

Gluteus medius tendon tears

Gluteus medius and minimus tendon tears are common in older patients. This can be confirmed on MRI[85] (Fig. 28.24b). In patients where the tear remains symptomatic despite conservative management, various surgical options are currently available. Up to 60% obtain relief from an arthroscopic bursectomy, although some patients will go on to a repair of the tendon.[86, 87]

Examination of the hip is in the *Clinical Sports Medicine* masterclasses at www.clinicalsportsmedicine.com.

(a)

(c)

(b)

(d)

Figure 28.25 Examples of exercises that provide increasingly greater challenges to improve strength and control of gluteal muscles

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