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Contents

JCCA Vol 53 No 4 ISSN 00083194

223 President's Message
Dr. Eleanor White, DC, MSc

225 JCCA Editorial Board
Dr. Mathieu Piché, DC, PhD

226 Chiropractic Researchers
Dr. Katherine MacAdam, BScKin, MSc, DC

Commentary

227 Chiropractic research capacity in Canada in 2008 – Phase 3
Kent Stuber, BSc, DC, MSc, André Bussières, DC, FCCS(C), MSc, Allan Gotlib, BSc, DC

Editorial

231 Sports Chiropractic in Canada
Mohsen Kazemi, RN, DC, FCCSS(C) DACRB, FCCRS(C)

233 The ability of parents to accurately report concussion occurrence in their bantam-aged minor hockey league children
Craig J. Coghlin, BA, CSCS, DC, Bryan D. Myles, BSc, DC, Scott D. Howitt, BA, CK, CSCS, DC, FCCSS(C), FCCRS

251 Exercise related transient abdominal pain-a case report and review of the literature
Brad Muir, HBSc(Kin), DC, FCCSS(C)

261 Traumatic anterior shoulder dislocation: a case study of non-operative management in a mixed martial arts athlete
Kevin Sims, BHSc, DC, Andreo Spina, BKin, DC, FCCSS(C)

272 Nine year longitudinal retrospective study of Taekwondo injuries
Mohsen Kazemi, RN, DC, FCCSS(C), FCCRS(C)
Artur Chudolinski, HBSc, DC
Matt Turgeon, DC, Aaron Simon, DC, Eric Ho, DC, Lianne Coombe, DC

282 Conservative management of symptomatic Carpal Bossing in an elite hockey player: a case report
Peter Kissel, BA(Hon), DC

290 Golf-related stress fractures: a structured review of the literature
Alexander D. Lee, BSc, DC

300 Conservative management of posterior interosseous neuropathy in an elite baseball pitcher's return to play: a case report and review of the literature
Andrew Robb, BA, DC, Sandy Sajko, BPHE, MSc, DC, FCCSS(C)

311 Psoas Major: a case report and review of its anatomy, biomechanics, and clinical implications.
Sandy Sajko, BPHE, DC, MSc, Kent Stuber, BSc, DC, MSc

319 Anatomy and physical examination of the knee menisci: a narrative review of the orthopedic literature
Michael D. Chivers, BPhE, DC, FCCSS(C)
Scott D. Howitt, BA, CK, CSCS, DC, FCCSS(C), FCCRS(C)

334 Ganglion cyst on the posterior cruciate ligament: a case report
Jaclyn A. Durante, BSc, DC

Editorial Board

Forging Our Future

Dr. Eleanor White, DC, MSc

Dr. Eleanor White, DC, MSc
President
Canadian Chiropractic Association

It is an honour to be asked to make comments in this Journal regarding the chiropractic profession in Canada from the perspective of the CCA.

The association functions within an interesting and very typical Canadian duality. Although we represent the chiropractic profession on the national stage, health policy and delivery are primarily provincial in nature. Thus the CCA has two distinct member designations; Charter Members comprised of the provincial chiropractic associations, and individual registrants. This elastic web of national and provincial interests functions best, when viewed as interconnected and the CCA often has the privilege of exposure to differing perspectives, each addressing excellence in its own jurisdiction. The similarities far outweigh the differences and the opportunity to learn from one another cannot be ignored. Success hinges upon commonality in vision and commitment to excellence.

The national association is well suited to the art of fusion; working together to find the best answer or most appropriate policy as well as assuming a leadership role in pan-professional issues. A recent event in Canada is an excellent example of the coalescing of thought and action that I believe must take place.

In spring 2009, a Canadian Leadership Summit was held in Toronto, attended by all provincial advocacy and regulatory bodies, and all of our national chiropractic partners, comprising our educational and examining institutions, the specialty colleges, risk management, our research foundations and our national regulatory federation. The need to commit to a pan-Canadian professional approach to the advancement of chiropractic was addressed directly. The collective body unanimously agreed to adopt the identity statements developed by the WFC, positioning the chiropractic profession as "spinal health care experts" and to define this more colloquially as the 'Back Doc' in our business model. Secondly, the collective committed to formally pursuing an integrative role within mainstream health care.

This accomplishment cannot be trivialized. Many who have been involved in chiropractic leadership may view this as self-evident, but this stated commitment to a common direction is a big step for many within the profession. We, like other groups have a disparate membership, representing not only differing styles of practice, but adhering to diverse philosophies. Historically, the call has been made for "unity." Indeed, we have been united by

goals of service to the public and our patients, and by our standards of practice, our education, our legislation, and our licensing requirements. However, as a goal unto itself, the value of unity may have been a veiled call to tolerance. We have often been told that there is room under the tent for all of us and that we are united by our name, and the nature of our two historic realities – the adjustment and the subluxation. As education has evolved, and health human resource issues have become important, inter-professional competencies are overlapping. The game has changed. We must change too.

We are now called upon, as a profession, to evolve and perhaps more quickly than we even now admit. Unity has taken on a new context and importance. Tolerance has become a liability at times. We are called upon now to understand our profession intimately, honestly and publicly. We must be committed in our endeavour to understand the mechanisms underlying the benefits of chiropractic care. This documented understanding will also compel us to trim away behaviours and practices that are not beneficial to the patient, irrelevant to practice, and quite frankly less than helpful in the advancement of the role of chiropractic in mainstream healthcare. In this context, unity becomes an ethical commitment to the pursuit of excellence and to the brave exploration of our own claims. As we work within our own profession and with other professions and jurisdictions to truly determine the uniqueness of chiropractic care, we will gain the knowledge to stake our ground.

So, what will that ground be? Where does our commitment to advancing integration within mainstream healthcare take us? Imagine pre-screening for orthopedic procedures, hospital privileges, working in the ER, multidisciplinary clinics, inter-professional continuing and post graduate education. Seeds are taking root, but it will take a pan-professional commitment to have these scenarios become common practice.

We have evolved greatly in the last decade, and the CCA has played a major role in this evolution. We have seen the development of excellent Clinical Practice Guidelines, soon to be housed within the new Research Chair at McGill University; and have established Research Chairs and Professorships in universities across the country. We have advanced federal government relations greatly and anticipate many more successes. We stand resolute in the support of our administrative members and partners in the profession. But we must think, and we must think creatively and well into the future and see where we need to know ourselves better, commit to excellence, and no longer tolerate unprofessional behaviour if we are to advance this profession as an integral partner in the health of Canadian citizens.

I am fortunate to have been involved in provincial, national and international chiropractic associations and I am always struck by the strength of the commitment of the volunteers and the administrators in this work. I have also been lucky to have been able to take these lessons to other areas of service in the provincial and federal community and I am convinced that the capacity of chiropractors to improve the health care of Canadians has not been even entertained by most. I like the word "capacity." It is a word widely used in the health human resources world. The best way to describe the benefit of chiropractic care is that it increases the capacity of function of its recipients. Patients experience an increase in their daily functional capacity, whether with respect to their working or recreational life. Our functional capacity as a profession is greatly untapped and I believe that as we understand the mechanism of our clinical success better, we can communicate it more confidently and effectively. This will ultimately result in a better understanding and acceptance of our contributions to general healthcare, leading to a greater use of our professional capacity.

We must move ahead with clarity and in a timely fashion as the healthcare world is evolving quickly, and each and every one of us has a responsibility to contribute to the success of the chiropractic profession. Our resolve this year to commit to this path was needed, but our next steps must follow quickly, surely and boldly.

Dr. Mathieu Piché, DC, PhD

Dr. Mathieu Piché, DC, PhD

The JCCA is delighted to announce the appointment of Dr. Mathieu Piché, DC, PhD to the Editorial Board.

Dr. Piché graduated in 2002 from the chiropractic doctoral program at Université du Québec à Trois-Rivières (UQTR). He obtained a Masters degree in Cell Biology and Biophysics from UQTR in 2004 for his work on neural plasticity of sensory systems in rodents. In July 2009, he obtained his PhD in Neurological Sciences from Université de Montréal for his work on endogenous modulation of pain in humans. He is currently an Assistant Professor in the Department of Chiropractic at UQTR where he teaches neurophysiology. He is a co-researcher for the chiropractic research chair at UQTR and for CIHR funded projects on pain neurophysiology at Université de Montréal. His research interests include the physiology and pathology of endogenous pain modulation and the impact of spinal pain on autonomic regulation in humans and rodents.

In 2005, Dr. Piché was awarded a prestigious CIHR Fellowship from the Clinical Research Initiative in the Institute of Gender and Health. One of only six recipients, this 4 year award allowed Dr. Piché to undertake his research on the neurophysiological mechanisms of chronic pain with Dr. Pierre Rainville PhD and Dr. Mickael Bouin MD, PhD at Université de Montréal.

His current publications include:

1 Piche M, Cohen-Adad J, Nejad MK, Perlbarg V, Xie G, Beaudoin G, Benali H, Rainville P. Characterization of cardiac-related noise in fMRI of the cervical spinal cord. Magn Reson Imaging. 2009; 27:300–310.

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Profile – Dr. Katherine MacAdam, BScKin, MSc, DC

Dr. Katherine MacAdam, BScKin, MSc, DC

Dr. Kate MacAdam attained her Bachelor of Science in Kinesiology degree from the University of New Brunswick in 2000 and majored in Athletic Therapy. Her work with varsity athletes introduced her to sport chiropractic treatment for athletic injuries. Dr. MacAdam was accepted to the Canadian Memorial Chiropractic College and had the intention of pursuing graduate study in sports chiropractic sciences. Upon graduating from CMCC with clinic honours in 2005, Dr. MacAdam was accepted in the Chiropractic Sports Sciences (Canada) sports sciences residency program and concurrently pursued a graduate degree.

Dr. MacAdam completed a Master of Science Degree in Kinesiology from the School of Health and Human Performance Dalhousie University in May 2009, under the supervision of Dr. Carolyn Savoy. Her Master Thesis entitled, "Does guided imagery as an adjunct to chiropractic treatment affect pain and disability in physically active adults with chronic low back pain?" concluded that guided imagery may be beneficial as an adjunct to spinal manipulative therapy in the treatment of chronic low back pain. The guided imagery CDs which Dr. MacAdam developed for the use of the study are now available for distribution for patients with chronic low back pain. She has presented her research at peer-reviewed conferences and has submitted her research for publication.

Her first published research was in the Journal of Manipulative and Physiological Therapeutics in 2005. As an undergraduate student, she was a co-author with Dr. Howard Vernon in the study entitled, "Validation of a sham manipulative procedure for the cervical spine for use in clinical trials."

Dr. MacAdam is a professional member of the Canadian Sport Psychology Association and works as a mental performance consultant as well as a chiropractor. She specializes in utilizing guided imagery as an adjunct to chiropractic treatment for injury healing, pain management, stress and anxiety reduction and injury rehabilitation. Dr. MacAdam has been an instructor for Sport Psychology at the School of Health and Human Performance at Dalhousie University since 2006 and is a mentor for the Canadian Institutes of Health Research. The mentor program is for "researchers" or "scientists" to mentor students/youth in their respective fields.

Dr. MacAdam's future plans consist of continuing her education by pursuing an Interdisciplinary PhD from Dalhousie University.

Chiropractic research capacity in Canada in 2008 – Phase 3

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André Bussières, DC, FCCS(C), MSc*
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Dr. Kent Stuber, BSc, DC, MSc

Dr. André Bussières, DC, FCCS(C), MSc

Dr. Allan Gotlib, BSc, DC

Introduction

Previous work on the topic of chiropractic research capacity in Canada has shown that currently less than 1% of chiropractors across the country are conducting research on a full time basis.[1] That survey consisted of two parts, a first sent to the entire profession nationwide and a second sent only to those with post-graduate training or who indicated in the first part that they were currently conducting research.[1] The main findings from the first part of the survey indicated that there were 94 chiropractors who either have or are in the process of completing a master's degree, along with 30 who either possess or are completing a PhD.[1] There was a noticeable discrepancy in terms of researcher distribution across the country with the vast majority being found in Ontario and Quebec and the remaining provinces and territories suffering from a considerable lack of researchers.[1]

The second part of the survey found that there are 20 full time chiropractic researchers and 55 part time researchers.[1] Once again the vast majority of these researchers were found in Quebec and Ontario.[1] Among the full time researchers 8 were conducting clinical re-

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search, 9 were doing epidemiological research, 6 were neurophysiologists, and there were 4 biomechanists.[1] The full time researchers averaged 17.8 publications in peer-reviewed journals over the past 5 years, compared with 3.2 papers by the part time researchers in the same time span.[1] Ten of the full time researchers indicated receiving government funding for their work, 5 received institutional funding, 3 received private funding, and 4 received funding from the profession.[1] By comparison 4 of the part time researchers indicated receiving government funding for their work, 12 received institutional funding, 3 received private funding, and 3 received funding from the profession.[1]

Key message

- *Less than 1% of chiropractors in Canada are actively engaged in research*
- *Chiropractic researchers in Canada are substantially under-funded.*
- *Many chiropractic researchers and graduate students are solely self-funded.*
- *Finding new ways to secure funding for chiropractic researchers is imperative.*
- *There is an urgent need to continue to build chiropractic research capacity.*

Phase 3

In this final part of our survey, we focused on the "financial aspects" of funding health research, health researchers and researchers in training positions. We attempted to quantify in a general sense the amount of funding which supports chiropractic research and researchers in Canada. This information is important for many reasons. For example:

- It will allow us to better focus strategic planning over the next 5 year period to prioritize and partner strategically
- It will help us continue to build capacity and create new funding opportunities
- It will help us accelerate the application of knowledge
- It will identify gaps in capacity, research and funding
- It will help us increase the number of trainees and researchers

The purpose of this project is to quantify the funding levels and delineate the types of funding obtained by chiropractic researchers in Canada.

Methods

The sample population for the Phase III survey consisted of respondents to the Phase II survey. Known researchers and graduate students who did not reply to the earlier Phases were also included. These researchers were e-mailed a survey that asked for specific information regarding their funding. The survey was sent on five different occasions to encourage higher response rates. Professors were asked to name all federal and provincial funding agencies from which they had received financial support in 2008. Such federal agencies included the Canadian Institutes of Health Research (CIHR), Natural Sciences and Engineering Research Council (NSERC), Social Sciences and Humanities Research Council (SSHRC), Canada Foundation for Innovation (CFI), Canada Research Chairs (CRC), Health Canada or similar national/international funders such as the Cochrane Opportunities Fund. Provincial funding agencies included the Michael Smith Foundation for Health Research (MSFHR), Ministry of Health, Workplace Safety and Insurance Board (WSIB), University, Hospital, Saskatchewan Health Research Council, Nova Scotia Health Research Foundation (NSHRF) as examples. Other relevant funding agencies, whether national or international, included charities, corporations, regulatory boards, Canadian Chiropractic Research Foundation (CCRF), Foundation for Chiropractic Education and Research (FCER), Research Institutes, etc. We asked respondents to tell us the amount and in each case if this was:

1. Salary Award (name of award, amount), or
2. Operating Grant (grant program, amount, project title, PI, Co-PI, Co-investigators)
3. Contractual or tenured university or hospital salary as an employee (other than a competitive salary award).

Chiropractors in research training positions were asked to include grants, scholarships, bursaries, training assistant positions, and research assistant positions in 2008. This category included graduate students/candidates and post doctoral fellows.

The information was collected and displayed as aggre-

Table 1 *Engaged vs not engaged in research or research training in year 2008*

	Clinician DC	Professor (academic appointment)	Graduate student	Post-doctoral trainee
Not engaged in research or research training (have a Masters degree or PhD but not active in research in 2008)	28	4		
Engaged in research or research training in 2008 • received no funding • funded	 3 1	 3 13	 12* 23	 0 1

1. Year 2008 (N = 90) Non-responders = 2.
2. One third of chiropractors who have research training were not involved in research in 2008.
3. One third of graduate students (one PhD, 11 masters level) had no funding (self-funded).*

Table 2 *Total salary awards and operating grants in the year 2008*

	Clinician DC	Professor (academic appointment)	Graduate student	Post-doctoral trainee
Engaged in research or research training AND received funding Year 2008	1	13	23	1
Salary award	$5,000	$ 699,470[a]	$ 131,200[b] $ 357,000[c]	$60,000
Operating Grant PI Co-PI Co-Investigator		$3,409,526[d] $2,507,518 (23) $ 287,231 (3) $ 614,777 (10)	$1,346,805[e] $ 223,425 (1) $ 30,000 (1) $1,093,380 (6)	

Year 2008 (N=90) Non-responders = 2.
[a] University/College salaries not included – 7 of the 13 Professors received competitive salary awards from Federal/Provincial funding agencies. [b]13 master students. [c]10 PhD candidates. [d]Number of grants = 36 Range ($17,500–$504,330). [e]Number of grants = 8 Range ($30,000–$506,367). (#) bracketed number indicates number of grants.
As a PI or Co-PI combined – Total grants in 2008 above $300,000 threshold.
Researcher #1 1,078,983. Researcher #2 504,330. Researcher #3 401,111. Researcher #4 322,300 (75.6% of grants held by 4 researchers).

gate data only. All information was treated with strict confidentiality and all original materials were destroyed after relevant sanitized information was entered in the aggregate pool.

Results

The survey was sent to 90 participants, 88 of whom responded (97.8% response rate). Tables 1 and 2 depict the results of the survey. Of the 88 respondents who had formal research training, 32 (36%) indicated that they were not involved in research in 2008. Eighteen (20%) of the researchers, including 12 graduate students (1 PhD, 11 master's level) indicated that they had no funding and that their research was therefore self-funded. The 12 graduate students without funding account for approximately 1/3 (12 of 35) of chiropractors who are currently university graduate students. Thirty-eight (43%) of the respondents indicated that they had received some funding for their research.

Table 1 sets out the distributions of competitive awards received in 2008, while Table 2 sets out the actual amounts of the funding.

One clinician received $5000, while 7 of 13 academic professors received a total of $699,470 in competitive salary awards (range $21,000–$287,000). A total of 13 masters students received $131,200 while 10 PhD candi-

dates received $357,000 in competitive stipend awards. One post-doctoral fellow received $60,000 in a competitive stipend award.

With respect to competitive operating grants, 12 of 13 academic professors held a total of $3,409,526 in 36 separate grants (range $17,500–$504,330), while 3 graduate candidates held a total of $1.346,805 in 8 separate grants (range $30,000–$506,367).

We separated the operating grant component according to whether the respondent indicated they were the principal investigator (PI), co-principal investigator (Co-PI), or co-investigator. PI professors held 23 grants totalling $2,507,518 while PI graduate candidates held 1 grant totalling $223,425. Co-PI professors held 3 grants totalling $287,231 while Co-PI graduate candidates held one grant totalling $30,000. Co-Investigator professors held 10 grants totalling $614,777 while Co-Investigator graduate students held 6 grants totalling $1,093,380.

Four chiropractic researchers holding the highest total operating grants awarded to either a PI or Co-PI represented 75.6% of all PI and Co-PI awards granted.

Discussion

This preliminary survey has identified various gaps in capacity, research and funding which must be addressed in the coming years in order to advance the body of knowledge defining the profession. Too few chiropractic researchers and exceedingly low levels of research funding represent barriers to advancing the profession.

In 2008, reported chiropractic research activity in Canada was supported by approximately $4 million in competitive funding. It is noteworthy that in the same year, one of Canada's major federal health funding agencies (CIHR) alone reported expenditures of $974.1 million (fiscal 2007–2008).

On our preliminary data, only 4 chiropractic researchers in Canada held 75.6% of the competitive operating grants as a PI or Co-PI. This finding alone warrants a strategic major investment by the profession and its partners to strengthen chiropractic health research capacity. This investment is required in order to provide governments across Canada with the high quality chiropractic evidence necessary to integrate and facilitate health care delivery by chiropractors to Canadians.

One successful strategy realized to date has been the Consortium of Chiropractic Researchers which now has some 23 members who are professors at universities across Canada. Recently the Consortium held a successful Workshop funded by CIHR and the CCRF. The researchers presented their current innovative, multi-disciplinary state of the art research developments in the chiropractic discipline to further refine the Chiropractic Research Agenda and ensure its congruency with CIHR. Such collaborative relationships foster greater networking opportunities and new research streams and develop cross disciplinary linkages. These researchers collectively pool their output to improve the health of Canadians and secure our profession's place in both our health research system and health care system.

However, finding new ways to secure funding for chiropractic researchers is imperative. Canadians are disadvantaged by not enjoying the benefits of new chiropractic knowledge that addresses the economic burdens of health, injury, disease and disability that so many Canadians endure needlessly.

Conclusion

It is clear that focused strategies are required to continue to build capacity and create new funding opportunities structured to increase the number of chiropractic research trainees and researchers and that they be supported with appropriate levels of funding to facilitate their important work.

While still few in numbers, it appears that a core group of highly dedicated researchers with clinical training in chiropractic is ready to undertake collaborative research. Professorships and Research Chairs in major universities across the country are now being established, including the recently announced position at McGill University. With such opportunities, many more researchers and trainees are expected to consider a fulltime career in research. Future efforts of the Canadian Chiropractic Research Foundation (CCRF) will hopefully explore new funding models for chiropractic research, further refine the chiropractic agenda in Canada, foster interdisciplinary research and develop new chiropractic knowledge.

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Sports Chiropractic in Canada

Dr. Mohsen Kazemi, RN, DC, FCCSS(C), DACRB, FCCRS(C)*

Dr. Mohsen Kazemi, RN, DC, FCCSS(C), DACRB, FCCRS(C)

The December issue of the JCCA has been devoted to highlighting the significant advances being made by Sports Chiropractic in Canada.

In Canada, Sports Chiropractic is regulated by the College of Chiropractic Sports Sciences (Canada), CCSS(C). Chartered as a Specialty College of the chiropractic profession in 1984, the CCSS(C) is an educational and coordinating organization that acts as the Regulatory College for Chiropractic Sports Specialists.[1]

CCSS(C)[1] defines the Sports Chiropractor as, "Chiropractic Sports Specialists keep active Canadians at their best by treating neuromusculoskeletal dysfunction. Using chiropractic adjustments, soft tissue therapies, modalities and rehabilitative measures, sports chiropractors aim to restore and enhance the body's neuromusculoskeletal system for peak performance. Chiropractic Sports Specialists are experts in the diagnosis and treatment of sport related injuries. They are proficient in injury prevention and rehabilitation measures, and have a sound knowledge base of return-to-play criteria."

The Chiropractic Sports Sciences Residency Program consists of following education, placements and written requirements. The core academic curriculum includes: research methodologies, biostatistics and critical appraisal, sports injury management, sports nutrition, sports psychology, chiropractic sports jurisprudence, high performance evaluation and testing, and first responders. Placements (minimum of 1000 hours) include: fracture clinic, sport orthopedics, sports imaging facilities, sports teams/events, biomechanics/research/chiropractic clinic, and multidisciplinary sports injury clinic. Written requirements includes: 4 book reviews, 4 case reports, 1 literature review, and completing one major research project of scientifically admissible quality.

Upon completing the program and requirements the resident will be eligible to sit for the CCSS(C) Fellowship exam, which is written and practical in format. Successful candidates would receive the prestigious "Fellow of the College of Chiropractic Sports Sciences (Canada)" and be recognized by the designation FCCSS(C). This is one of

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only five chiropractic specialties in Canada recognized by the Canadian Federation of Chiropractic Regulatory and Educational Accrediting Boards (CFCREAB).[1]

Sports chiropractic has been constantly raising the professional platform in Canada. What started as a chiropractor providing care for individual athletes and local teams has grown to the point that most professional and National sports teams now have a chiropractor on their health care teams. Sports Fellows are now included in the Canadian Core Health Care Teams for major and minor games. Many Canadian Sports Chiropractic Fellows are now part of or the head of the Health Care teams for several sports. CCSS(C) is affiliated with the Canadian Olympic Committee (COC) and the Canadian Sports Centres. Members of the CCSS(C), along with sports Medical Doctors, sports Physiotherapists, Athletic Therapists and Sports Massage Therapists make up the Service Provider Expert Group which advises the COC on healthcare policy development and provider selection for various International Games.[1]

However, looking at the literature, there is a paucity of research in Sports Chiropractic effectiveness and treatment. This is one of the most hindering obstacles for Sports Chiropractic progress and inclusion in the health care of athletes. As such it is our responsibility to focus and encourage research in Sports Chiropractic. For this very reason and the fact that there is no Sports Chiropractic Journal to showcase the Sports Chiropractors' research, this issue of the JCCA is devoted to showcasing our Canadian Sports Fellows and residents' research. I hope that this Sport Issue of JCCA would encourage and inspire you to get involved in research in Sports Chiropractic.

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1 CCSS(C) (2008). http://www.ccssc.ca/index.phpresearch

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The ability of parents to accurately report concussion occurrence in their bantam-aged minor hockey league children

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Dr. Bryan D Myles, BSc, DC
Dr. Scott D. Howitt, BA, CK, CSCS, DC, FCCSS(C), FCCRS*

Objective: *The objective of this study was to assess the ability of hockey parents/guardians to recognize concussion symptoms in their 13–14 year old (Bantam-aged) children.*

Outcome Measures: *The outcome measures were the ability to recognize different signs and symptoms listed on the Sport Concussion Assessment Tool (SCAT) as well as 8 detractors consisting of signs and symptoms not associated with post concussive syndrome. Additional questions assessing the parents' knowledge of concussion management and recognition abilities were also posed.*

Participants: *Parents of Bantam-aged minor hockey league athletes volunteered for the study.*

Methods: *The study investigators distributed questionnaires during the warm up period or following their children's games to the study participants. Following questionnaire completion, participants were provided with an information package outlining the correct signs and symptoms of concussion.*

Results: *The mean number of correct responses to signs and symptoms of concussion was 21.25/25 for the mothers and 20.41/25 for the fathers. The mean number of detractors identified as not associated with concussion was 5.93/8 for the mothers and 4.85/8 for the fathers, indicating that mothers were more capable of recognizing the signs and symptoms than fathers. An analysis of variance including sporting experience in the model did not strengthen the relationship between parent gender and test outcome.*

Conclusion: *This investigation revealed that there is still a disconnect in regards to key components of recognizing a concussion, such as difficulty with sleep,*

Objectif : *L'objectif de cette étude était d'analyser la capacité des parents/tuteurs de hockey à reconnaître les symptômes de commotion chez leurs enfants de 13 et 14 ans (âges bantam).*

Critères d'évaluation : *Les critères d'évaluation étaient la capacité à reconnaître les divers signes et symptômes énumérés par l'outil d'évaluation des commotions dans le sport (SCAT), ainsi que huit facteurs défavorables composés de signes et symptômes qui ne sont pas associés au syndrome post-commotion. D'autres questions qui évaluent les capacités des parents par rapport à la connaissance du contrôle et de la reconnaissance des commotions ont également été posées.*

Participants : *Des parents d'athlètes de hockey mineur d'âge bantam qui se sont proposés pour participer à l'étude.*

Méthodes : *Les chercheurs de l'étude ont distribué des questionnaires aux participants à l'étude au cours de la période d'échauffement ou après les parties des enfants. Une fois les questionnaires remplis, on a fourni aux participants une trousse d'information indiquant les signes et symptômes exacts de commotion.*

Résultats : *Le nombre moyen de réponses correctes vis-à-vis des signes et symptômes de commotion était 21,25/25 pour les mères et 20,41/25 pour les pères. Le nombre moyen de facteurs défavorables déterminés comme n'étant pas associés à la commotion était 5,93/8 pour les mères et 4,85/8 pour les pères, ce qui indique que les mères étaient plus en mesure de reconnaître les signes et symptômes que les pères. Une analyse de la variance comprenant l'expérience dans les sports au sein*

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disorientation symptoms, and emotional irritability. Mothers have displayed an ability to better differentiate between true and false signs and symptoms of concussion as compared to fathers. Continued education and awareness of mild traumatic brain injury in athletes should address the misconceptions amongst parents in regards to the true signs and symptoms of a concussion.
(JCCA 2009; 53(4):233–250)

KEY WORDS: hockey, bantam, concussion, chiropractic

du modèle n'a pas renforcé la relation entre le sexe du parent et les résultats au test.

Conclusion : *Cette enquête a révélé qu'il existe toujours une coupure en ce qui a trait aux éléments clés permettant de reconnaître une commotion, comme la difficulté à dormir, des symptômes de désorientation et l'irritabilité émotionnelle. Les mères ont affiché une meilleure capacité à faire la différence entre les vrais et faux signes et symptômes de commotion que les pères. La sensibilisation continue des traumatismes cérébraux légers chez les athlètes doit aborder les idées fausses des parents en ce qui concerne les véritables signes et symptômes d'une commotion.*
(JACC 2009; 53(4):233–250)

MOTS CLÉS : hockey, bantam, commotion, chiropratique

Introduction

Concussion is a common type of head injury that can occur in most contact sports. In the "Summary and Agreement Statement of the First International Symposium on Concussion in Sport, Vienna 2001," concussion is defined as a "complex pathophysiological process affecting the brain, induced by traumatic biomechanical forces."[1]

It is thought that some coaches, trainers, and more importantly, parents or guardians don't realize exactly how a concussion occurs. There are several different constructs in which a concussion occurs and can include the following:

1. Concussion may be caused either by a direct blow to the head, face, neck or elsewhere on the body with an 'impulsive' force transmitted to the head.
2. Concussion typically results in the rapid onset of short-lived impairment of neurological function that resolves spontaneously.
3. Concussion may result in neuropathological changes but the acute clinical symptoms largely reflect a functional disturbance rather than structural injury.
4. Concussion results in a graded set of clinical syndromes that may or may not involve loss of consciousness. Resolution of the clinical and cognitive symptoms typically follows a sequential course.
5. Concussion is typically associated with grossly normal structural neuroimaging studies.[2]

Perhaps the most common misconception when it comes to recognizing a concussion is the issue surrounding loss of consciousness. Most concussions occur without a loss of consciousness.[1,2] In fact, loss of consciousness is just one of many possible signs and symptoms of a concussion.[1,2] Loss of consciousness may be suggested if the victim cannot recall events before or after the incident, however this is more consistent of a person experiencing signs of amnesia. Another misconception surrounding concussion is the idea that you must be hit in the head for a concussion to occur. A significant blow to either the neck, face, jaw, or elsewhere in the body can result in a concussion as long as the force is transmitted to the head.[1,2] Thus, a concussion can occur from what is normally viewed as a legal body check in the sport of hockey. An analogous model used to understand this phenomenon is that of a sponge in a bucket of water (brain within the skull). Under normal movements the sponge will move within the water freely, but with a jarring force to the bucket the sponge will make contact with the sides of the bucket.

There are many physical, cognitive, and emotional signs and symptoms associated with having sustained a concussion as listed below in Table 1.[1,2]

Table 1 *The Signs and Symptoms of Concussion*

Physical Symptoms	Cognitive Symptoms	Emotional Symptoms	Physical Signs
Headache	Confusion	Depression	Loss of consciousness
Dizziness	Amnesia	Irritability	Poor coordination
Nausea	Disorientation	Moodiness	Easily distracted
Feeling 'Unsteady'	Poor concentration		Poor concentration
Feeling 'Dinged'	Memory disturbance		Slow responses
Feeling 'Stunned'			Vomiting
Feeling 'Dazed"			'Glassy eyed'
Describe 'Bell rung'			Photophobia
'Seeing stars'			Aphasia
Visual Disturbances			Personality change
Tinnitus			Inappropriate behavior
Diplopia			Decreased physical ability

Not all of these indicators would be present in a concussion, but if one is present, a concussion should be considered as a possible diagnosis and appropriate referral needs to be advised.[2]

Early detection and documentation is critical in the management of the concussed athlete. The Vienna & Prague concussion conferences of 2001 and 2004 respectively confirm that the grading of concussions has been abandoned and the management of a concussion is to occur on a case by case basis.[1,2] These proceedings also led to the development of a Sport Concussion Assessment Tool (SCAT card), see Appendix A. The SCAT was developed to create a standardized tool to be used for patient education and physician assessment of a concussion. It was developed by combining a variety of existing tools, including: Sideline Evaluation for Concussion-Colorado Head Injury Foundation, Inc [Society, 1990 (revised May 1991)], Management of Concussion Sports Palm Card-American Academy of Neurology & Brain Injury Association,[3] Standardized Assessment of Concussions-SAC,[4] Sideline Concussion Check-UPMC/Thinksafe/Sports Medicine New Zealand Inc and the Brain Injury Association, McGill Abbreviated Concussion Evaluation (ACE) (unpublished), National Hockey League Physician Evaluation Form (unpublished), UK Jockey Club Assessment of Concussion[5] and Maddocks questions.[6]

The memory questions utilized in the SCAT were modified from the validated Maddocks questions in order to make them less football specific.[6] The Maddocks questions included: Which ground are we at? Which team are we playing today? Who is your opponent at present? Which half is it? How far into the quarter is it? Which side scored the last goal? Which team did we play last week? Did we win last week?[6]

The importance of recognizing even a minor concussion is reinforced through the literature which reports that once an athlete experiences a concussion, the likelihood that they experience a subsequent concussion is increased; and the symptoms experienced in the second concussion may be more severe and take an extended period of time to resolve.[7–9] The key to this concept is that once an initial concussion has been recognized, the involved athlete should not return to play until all symptoms have resolved or the risk of subsequent concussion is increased.[1,10–21]

A main issue in regards to the occurrence of concussions in minor hockey involves body checking. With the rising incidence of traumatic brain injury in hockey[22–24] some

authors fear that many Canadian youth are exposed to the lasting effects of concussions, some of which are not fully realized until the brain completes its maturation. Marchie et al. acknowledge that while many injuries can be caused by the body checking that occurs in hockey, concussion is a major concern due to the severity of the possible sequelae.[24] Of these sequelae, some of the more notable include headache, cognitive/memory and executive-function disturbances, and/or visual abnormalities.[25–30]

The widespread knowledge of the signs and symptoms of a concussion in contact sports such as hockey would further enhance the accuracy of recognizing this condition. This could be facilitated by knowledge transfer amongst coaching staffs, parents/guardians, and even the players themselves. The National Hockey League (NHL) and Ontario Hockey League (OHL) have surveillance systems in place for the epidemiological analysis and control of concussions during the game, but the incidence of concussion in youth hockey is poorly established. Reporting of concussions in minor hockey is largely based on the players themselves reporting an injury to the bench staff who in turn must recognize the injury and its severity.[31] This relies on the assumption that the bench staff, or even the hockey player, is knowledgeable in the important signs and symptoms of concussion. Unfortunately in many cases, this is where the line of communication may stop. When parents/guardians are unaware of what their child has experienced during the game, what is assumed to be a mild headache, or a moody child, could be considered normal.

Although in recent years there have been increases in the knowledge surrounding concussions through educational seminars hosted by associations such as the Greater Toronto Hockey League and Think First Canada, significant knowledge gaps still exist. Currently, studies are being done in diagnostic modalities, imaging, and concussion evaluation. Neuropsychological testing has been found to be an impressive way to assess severity and resolution, while shorter, computer based tests (such as CogSport or ImPACT) are becoming popular adjuncts.[2,28,32–46] Although there has been substantial research completed on concussions, this information is not consistently delivered to players' parents or guardians as it is filtered down through physicians, coaches, and hockey organizations. In a recent study by Valovich-McLeod et al., coaches were surveyed as to their understanding of sport-related concussion.[47] This study found that previous coaching experience was predictive of better symptom recognition, and that between 49.4% and 61.5% of coaches were able to answer correctly to four true/false questions on concussion. With this in mind, our study further aims to determine if parents/guardians are capable of identifying the signs and symptoms of a concussion in order to ensure the child receives the proper medical attention.

Methods

A questionnaire was developed in order to gather information from parents of Bantam aged (13–14 year old) athletes participating in the 7th Annual Penguins International Winter Classic hockey tournament (Appendix B). The tournament was hosted on January 19–21st, 2007, at the Chesswood and Westwood arenas in Toronto, Ontario, Canada.

Bantam-aged athletes were chosen because it has been found by Willer et al. that that there is a trend for an increasing occurrence of concussion as age increases.[48] Additionally the study by Emery[49] analyzed the risk of injury in different age groups of hockey players and found that 45% of all injuries occurred due to body checking. The relative risk of injury in Bantam aged hockey players was 3.72.[49] Thus, for the current study, Bantam athletes were chosen because players have been in a body contact age group for at least two seasons and thus would have an increased risk of having sustained a concussion compared to younger players. Additionally, more parents or guardians are typically in attendance for Bantam-aged players compared to the older midget aged players.

This questionnaire was screened for content/wording and approved for inclusion in this study by the Canadian Memorial Chiropractic College Institutional Review Board for Research prior to collecting any data. The bulk of the questions used in this study's questionnaire were taken from the Sport Concussion Assessment Tool (SCAT) (Appendix A).[2] The SCAT was evaluated for face and content validity on the basis of scientific literature[50] and clinical experience of the authors.

Our questionnaire (Appendix B) consisted of four demographic questions (a-d), and six additional questions specifically targeting the parent's knowledge of concussions, signs, and symptoms (numbered 1–6). The questionnaires were distributed to the parents or guardians by hand during the warm up prior to each game or directly

following the game. After filling out the questionnaire the parents were given the answer key to the questions posed (Appendix C).

The demographic questions were designed to indicate the level of bantam hockey the child was currently participating in, the child's current age, the parents' sporting experience, and their relationship to the child. Information regarding the level of Bantam hockey was collected in order to determine if higher levels of competition would have an effect on the parents' knowledge. Study investigators were also interested in the parents' sporting experience to determine if the level of experience of a parent would have a bearing on their knowledge of the signs and symptoms. The next six questions under the heading of 'Questionnaire' were designed to determine the parents' ability to recognize the signs/symptoms and guideline information that is currently being used to determine whether a child has received a blow to the head or body that may be diagnosed as a concussion. Questions 1–4 and 6 were of the Yes/No type while question 5 was unique. Question 5 consisted of twenty-five true or false questions asking the parents to identify signs and symptoms of concussion (see Appendix B, #5). The questionnaire instructed the parents to check true or false depending on whether they believed the items were correct signs or symptoms of a concussion. Of the twenty-five questions there were seventeen true answers and eight false answers.

Specifically the 8 false detractors, which were considered "red herrings" included: difficulty with urination, lowered pulse rate, difficulty with defecation, hearing voices, sinus congestion, feelings of euphoria, inability to swallow, and chest pain. These incorrect signs or symptoms were included in order to avoid the possibility of people guessing all the correct answers, and to make the questionnaire a more thought provoking process.

After the guardian of the child had completed the questionnaire, the questionnaire was collected and placed into an envelope in which it was stored until data analysis could begin.

Data Analysis

For each completed survey the number of correct responses was tabulated for the 25 items (true signs recognized and false signs denied) for a score out of 25 allowing for a normal distribution.

Data analysis used a two sample T-Test to analyze the correct responses to the signs and symptoms both associated and not associated with concussion (the 8 'red herrings'). An analysis of variance was utilized to compare the differences in responses to identifying the correct and incorrect signs and symptoms of concussion between the two groups of mothers or fathers with the additional factor of their own participation in sport.

Results

One hundred and seventeen questionnaires were completed and collected from the 120 handed out for a 97.5% response rate. Inclusion criteria for the acceptance of the questionnaire into this study were that all questions were answered and that the questionnaire was fully completed.

Three questionnaires were discarded from the study. Two questionnaires were discarded because they were not completely finished, and one was discarded because no demographic information was listed. Our final number of collected questionnaires used for analysis was 114 (N = 114).

The demographic information revealed that 57% of the athletes were competing at the single 'A' level, 42% were competing at the 'AA' level, and 1% were competing at the 'AAA' level (All star levels progressing from A to AAA). 44% of the athletes were 13-years of age and 56% were 14-years of age. Just under 10% (9.65%) of the guardians participated at a self-rated "high level" of sport, 43.86% of guardians participated at a self rated "medium" level of sport, 29.82% played at a self rated "low level" of sport and 16.67% did not participate in any sport. Of the guardians questioned, 52.63% were mothers of the bantam aged participants and 47.37% were the fathers of the participants. The questionnaire's demographic information revealed that no male or female legal guardians other than parents were questioned. The demographic information did not show any difference in the answers of the parents of varying sporting experience or with different levels of play for their children.

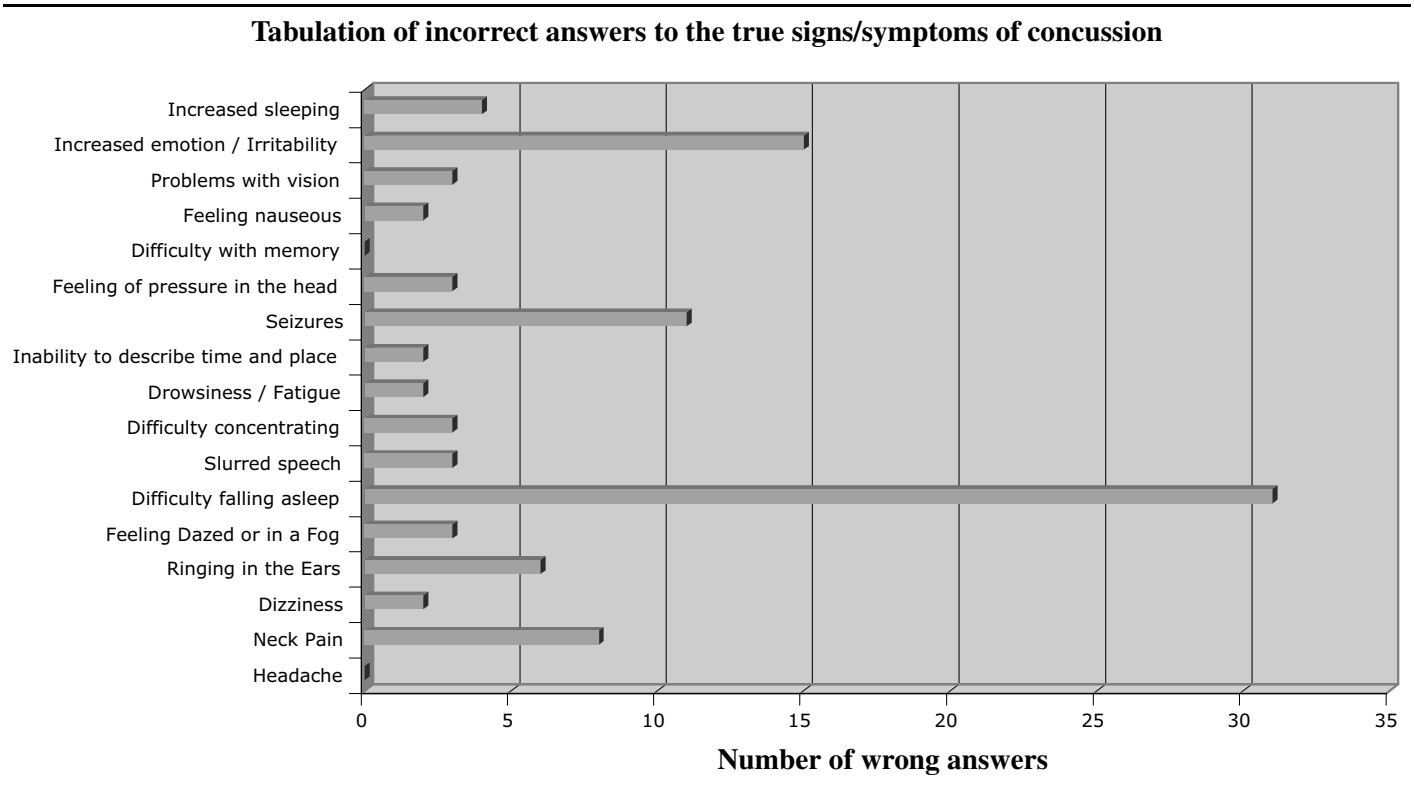
Questions 1–4 were generally answered correctly by the 114 respondents see table 2 below.

Question 5 tested the ability of the respondent to identify the signs and symptoms that are truly associated with a concussion and deny those that are not. Figure 1 reveals the number of incorrect responses in the attempt to identify the true signs and symptoms of concussion. The

Table 2 *Percentage of Correct Responses Questions 1–4*

Question	Number of correct answers (N=114)
1. Does a loss of consciousness determine whether a concussion has occurred?	87 (76.32%)
2. Can a player who has suffered a concussion return to play in the same day?	109 (95.61%
3. A concussion may be caused by a blow to the neck, jaw, or elsewhere in the body?	99 (86.84%)
4. Is it necessary for a player to be medically evaluated after having their bell rung?	101 (88.6%)

Figure 1 *The number of incorrect answers for the signs and symptoms not identified as being associated with concussion that are indeed related to concussion*



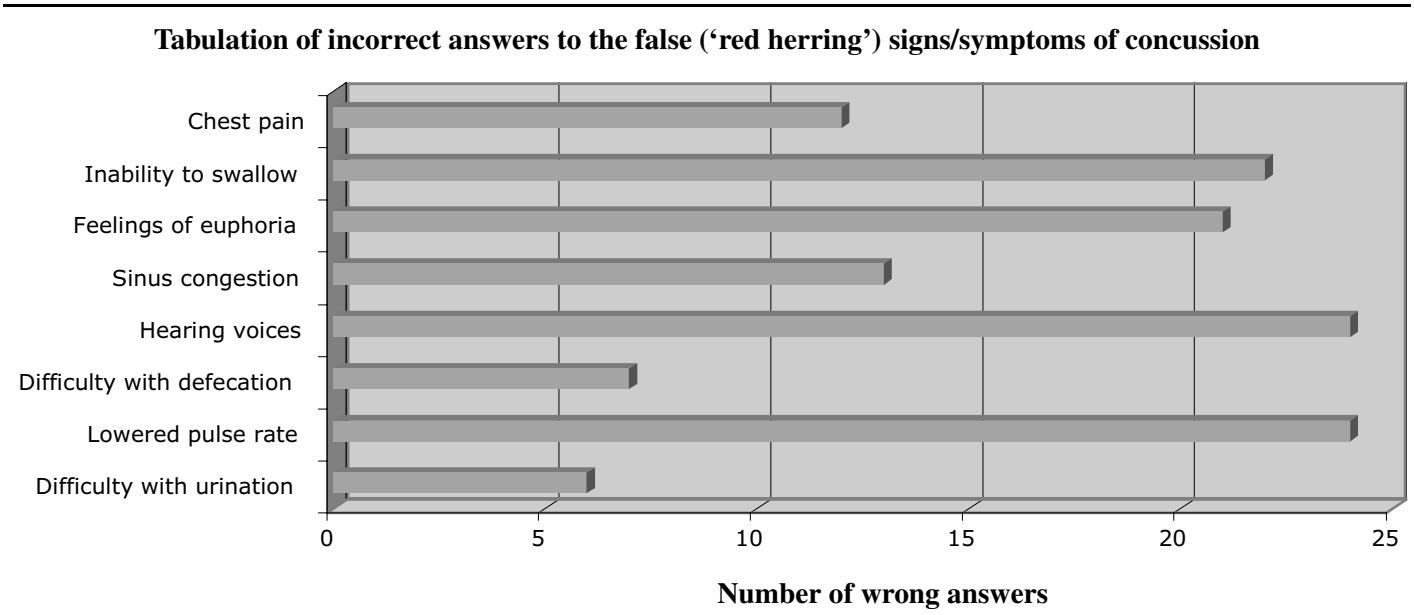
number of wrong answers in identifying the false signs and symptoms or 'red herring' detractors are displayed in the figure 2 below.

Considering the correct responses to questions 1–5, the 93 respondents or (81.58%) who answered "no" to question 6 *(Has your child ever suffered a concussion?)* has to be viewed with some degree of skepticism.

Statistical Analysis

A two-sample t test was done to compare the scores mothers and fathers received for the 25 questions. Of the 60 mothers the mean score was 21.25 (standard deviation 2.07, 95% confidence interval 20.71–21.79) while the 54 fathers mean score was 20.41 (standard deviation 2.54, 95% confidence interval 19.71–21.10). The two-sample

Figure 2 *The number of incorrect answers for sign and symptoms identified as being associated with concussion that are not related to concussion*



t test for the 8 false detractors revealed that the mothers mean score was 5.93/8 (standard deviation 2.15, 95% confidence interval 5.38–6.49) while fathers mean score was 4.85/8 (standard deviation 2.65, 95% confidence interval 4.13–5.57). Thus a difference does exist, with the mothers having a better ability to discriminate mild traumatic brain injuries, recognizing the signs or symptoms associated with concussion. An additional analysis of variance that included sporting experience in the model did not strengthen the relationship between parent gender and the test outcome (25 detractors: $F = 3.02$, $p = 0.0848$, 8 false detractors: $F = 5.04$, $p = 0.0268$)

Discussion

The results of this study indicate that in general the parents surveyed were successful in correctly identifying the signs and symptoms of a concussion in their child. Our analysis of data revealed that mothers were more knowledgeable than fathers in identifying the correct signs and symptoms of concussion. Interestingly, mothers were significantly more capable of recognizing the false signs and symptoms added as detractors. Unfortunately, there have been no other studies analyzing the ability of parents to recognize signs and symptoms, so it is difficult to comment on any contrasting or supporting results.

The 2001 Vienna and the 2004 Prague Statements[1,2] were instrumental in aiding the overall awareness of common signs and symptoms of a concussion and resulted in the creation of the Sport Concussion Assessment Tool (SCAT) (Appendix A). The SCAT makes it simpler and more convenient for coaching staffs and parents to become aware of the signs and symptoms of a concussion. Although this study shows that parents are generally aware of the signs and symptoms put forth in the guidelines, it sheds light on areas of confusion that may be overlooked in educational seminars or materials.

In comparison to the correct signs and symptoms, the incorrect signs and symptoms ('red herring' detractors) were not answered as well (review Figure 1 and 2). Both hearing voices and lowered pulse rate were incorrectly indicated as true signs/symptoms by 47.4% of the participants. Feelings of euphoria and inability to swallow were answered incorrectly by 43.9% of participants. We suggest that parents indicating these detractors as correct signs and symptoms could be overcautious in analyzing changes in their children's health or demeanor, or simply

that they had the perception that all of the detractors were indeed correct. In the current study, only one subject was found to have chosen all true responses. Although this could potentially lead to an increased reporting and subsequent demand to health care providers, we suggest that this is not likely to occur in reality and that more attention be paid to the commonly overlooked true signs and symptoms.

Of particular importance to recognizing a concussion, it should be noted that all participants answered both the headaches and difficulty with memory correctly as indeed signs/symptoms of a concussion.

The three signs and symptoms that were most commonly not recognized as being part of a concussion were: difficulty falling asleep (54.4% answered incorrectly), inability to describe time and place (28.9% answered incorrectly), and increased emotion/irritability (23.7% answered incorrectly).

The parents of Bantam-aged minor hockey league athletes in this study are reasonably knowledgeable in the common signs and symptoms of concussion. This is a similar result to the recent Valovich-McLeod study[47] that found that although coaches are knowledgeable, there are certain signs and symptoms that continue to be overlooked.

Difficulty falling asleep is reported thoroughly throughout the literature as being one of the most common sequelae of a mild traumatic brain injury, along with poor memory and fatigue.[51–53] However, the study by Chan et al.,[54] analyzed a group of participants who had experienced no head injury and found that sleep disturbance was indicated by 50.6% of their sample. They argued that many of the symptoms indicated on a typical post concussion checklist are commonly endorsed by a normal uninjured population. This was further supported by a similar study by Iverson et al.,[55] who argued that the typical symptoms noted in post concussive syndrome (PCS) are not unique to this syndrome. Yet in the Kaufman et al. study,[56] the researchers found that participants who had suffered a minor head injury showed lower sleep efficiency with more awake time and with more awakenings lasting more than 3 minutes. There has been recent research into the specificity of the commonly indicated signs and symptoms of PCS,[57–59] but this debate is beyond the scope of this paper.

Inability to describe place and time is a typical cognitive feature of a concussion. Often referred to in the literature as being disoriented, the inability to describe place and time can be considered highly indicative of a brain injury. The fact that the current study indicated this feature as being answered incorrectly 28.9% of the time may be concerning to health care providers. Several authors[1,2,11,50,59,60] note disorientation as being one of the best prospectively validated indicators that a concussion has occurred. The importance of asking the injured athlete questions regarding their orientation is further emphasized in several papers.[11,50,53,60–64]

Maddocks[53] further addresses the sensitivity of the typical orientation questions such as, "What year is it?" and "How old are you?" noting that these questions are typically sensitive in traumatic motor vehicle accidents, but may be less sensitive when examining a more subtle head injury such as those incurred during sport. In his study, Maddocks compared the correct answers by concussed and non concussed athletes. These questions ranged from recently acquired items (date, time, ground, quarter, last goal, etc) to more long term memory items (name, date of birth, age, year, etc). For those that were found to have sustained a concussion, the number of subjects answering the more recently acquired memory items correctly was significantly lower. In Young's study,[63] it was noted that many of these memory and orientation questions could be affected by the athletes' level of intelligence. Because of this, they studied the answers to common orientation and memory questions in non-injured athletes. The results of this study showed that some tests (serial sevens) were only answered correctly half the time, whereas a test such as the 'months of the year in reverse' (MOYR) was answered correctly by over 90% of participants. Thus, these researchers postulate that it may be wiser to use a test such as MOYR as a sideline evaluation tool.

Irritability is also described by Johnston et al.[50] as part of a wide variety of subjective findings that one may discover in a concussed athlete. These researchers also note that the time to resolution from these symptoms is quite variable and difficult to predict. As with many of the studies listed previously, irritability is commonly experienced by those suffering a concussion.[1,2,50,54,58–60,65–66] These mood disturbances typically become noticeable at later stages of post concussion, approximately 4–8 weeks later.[60] Often it is debated in the literature as to whether irritability is caused by neural damage incurred along

with the concussion, or whether it is due to the effects of concussion on slowing cognitive functioning and subsequently causing increased emotional distress in the patient.[67] Regardless of the true cause, the fact remains that irritability should be recognized as a symptom of sustaining a mild traumatic brain injury.

In interpreting the data presented in this study, it is important to be aware of the methodological limitations present. Despite adding detractors to the option list for question #5, there is still the possibility that parents simply chose 'true' for the entire section. The study investigators found that many parents were either unwilling to accept a questionnaire or rushed when it came to completing the questionnaire. In order to complete the questionnaire in less time, it is possible some parents may have simply chosen 'true' for all or most of the statements, without thoroughly reading them. This 'all true' scenario was documented only once within our sample, which implies that the questions were taken seriously and answered to the best of the respondents' abilities. Analyzing the way the selections were actually made in this one questionnaire (deliberate penmanship), further suggests that the subject considered all signs and symptoms to indeed be correct.

The SCAT card had been previously evaluated for face and content validity by Johnston, McCrory, et al.,[50] and thus for the present study, the SCAT signs and symptoms were incorporated into the questionnaire with additional false detractors to increase difficulty. Still, it must be considered that the questionnaire may not be measuring what it purports to, as the wording of the questions may not have been equally understood by all participants.

As this study was only directed to parents of children participating in the Bantam age group, the results can only be applied to this particular 13–14 years age group. It could be argued that parents of children in higher or more competitive age groups, or those with multiple children, may have more exposure to the topic of concussions and thus may have a more complete knowledge set. Conversely, one could also suggest that parents of players in a younger, pre-body checking age group may not yet be concerned with concussions in their children, and thus may be unaware of common signs and symptoms. Regardless of this, the study investigators would suggest an early start to the education process of parents with respect to concussion awareness in their children.

Conclusion

According to this study, it appears that the efforts of organizations such as ThinkFirst Canada, the Greater Toronto Hockey League, and individual primary health care providers within the community have largely been successful in increasing the awareness of parents to the signs and symptoms of concussions. However, a complete dissemination of concussion knowledge from researchers and health professionals to parents, still shows a disconnect. Key components of recognizing a concussion, such as difficulty with sleep, disorientation symptoms, and emotional irritability should be better known. The signs and symptoms of concussion require further knowledge transfer to all concerned parties. Continuing attempts to raise awareness for the proper and swift recognition of mild traumatic brain injury in young athletes is recommended. Further investigations towards the identification of concussion and the most effective ways to publicize this information would be prudent to enhance knowledge transfer of this condition.

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Appendix A—SCAT card (Sport Concussion Assessment Tool)

Name: _____ Date: _____

Sport/Team: _____ Mouth Guard? Y N

1) SIGNS

Was there loss of consciousness or unresponsiveness? Y N
Was there seizure or convulsive activity? Y N
Was there a balance problem/unsteadiness? Y N

2) MEMORY

Modified Maddocks questions (check those correct)

At what venue are we? ___ Which half is it? ___ Who scored last? ___ What team did we play last? ___
Did we win last game? ___

3) SYMPTOM SCORE

Total number of positive symptoms (from "**SYMPTOMS**" box on other side of the card) = _____

4) COGNITIVE ASSESSMENT

(Check those correct)

5 word recall (Examples)		Immediate	Delayed (after concentration tasks)
Word 1 _____	cat	___	___
Word 2 _____	pen	___	___
Word 3 _____	shoe	___	___
Word 4 _____	book	___	___
Word 5 _____	car	___	___

Months in reverse order (circle those incorrect) Jun-May-Apr-Mar-Feb-Jan-Dec-Nov-Oct-Sep-Aug-Jul

OR

Digits backwards (check those correct)

5–2–8	3–9–1	___
6–2–9–4	4–3–7–1	___
8–3–2–7–9	1–4–9–3–6	___
7–3–9–1-4–2	5–1–8–4-6–8	___

Ask delayed 5-word recall now

5) NEUROLOGICAL SCREENING

	Pass	**Fail**
Speech	___	___
Eye Motion and Pupils	___	___
Pronator Drift	___	___
Gait Assessment	___	___

Any neurologic screening abnormality necessitates formal neurologic or hospital assessment.

6) RETURN TO PLAY

ATHLETES SHOULD NOT BE RETURNED TO PLAY THE SAME DAY OF INJURY.

When returning athletes to play, they should follow a stepwise symptom-limited program, with stages of progression. For example:

1. rest until asymptomatic (physical and mental rest)
2. light aerobic exercise (e.g. stationary cycle)
3. sport-specific training
4. non-contact training drills (start light resistance training)
5. full contact training after medical clearance
6. return to competition (game play)

There should be approximately 24 hours (or longer) for each stage and the athlete should return to stage 1 if symptoms recur. Resistance training should only be added in the later stages.
Medical clearance should be given before return to play.

INSTRUCTIONS:
This card is for the use of medical doctors, physiotherapists or athletic therapists. In order to maximize the information gathered from the card, it is strongly suggested that all athletes participating in contact sports complete a baseline evaluation prior to the beginning of their competitive season. This card is a suggested guide only for sports concussion and is not meant to assess more severe forms of brain injury.

Signs:
Assess for each of these items and circle – Y (yes) or N (no)

Memory:
Select any 5 words (an example is given). Avoid choosing related words such as "dark" and "moon" which can be recalled by means of word association. Reach each word at a rate of one word per second. The athlete should not be informed of the delayed testing of memory (to be done after the reverse months and/or digits). Choose a different set of words each time you perform a follow-up exam with the same candidate.

SYMPTOMS: Headache, "pressure in the head," neck pain, balance problems or dizziness, nausea or vomiting, vision problems, hearing problems or ringing in the ears, "don't feel right," feeling "dinged" or "dazed," confusion, feeling slowed down, feeling like in a "fog," drowsiness, fatigue or low energy, emotional, irritable, difficulty concentrating or remembering

Concentration/Attention:
Ask the athlete to recite the months of the year in reverse order, starting with a random month. Do not start with December or January. Circle any months not recited in the correct sequence.

For digits backwards, if correct, go to the next string length. If incorrect, read trail 2. Stop after incorrect on both trials.

Neurologic Screening:
Trained medical personnel must administer this examination. These individuals might include medical doctors, physiotherapists or athletic therapists. Speech should be assessed for fluency and lack of slurring. Eye motion should reveal no diplopia in any of the 4 planes of movement (vertical, horizontal and both diagonal planes). The pronator drift is performed by asking the patient to hold both arms in front of them, palms up, with eyes closed. A positive test is pronating the forearm, dropping the arm, or drift away from midline. For gait assessment, ask the patient to walk away from you, turn and walk back.

Return to Play:
A structured, graded exertion protocol should be developed; individualized on the basis of sport, age and the concussion history of the athlete. Exercise or training should be commenced only after the athlete is clearly asymptomatic with physical and cognitive rest. A doctor should ideally make final decision for clearance to return to competition.

This tool represents a standardized method of evaluating people after concussion in sport. This tool has been produced as part of the Summary and Agreement Statement of the Second International Symposium on Concussion in Sport, Prague, 2004. For more information see the "Summary and Agreement Statement of the Second International Symposium on Concussion in Sport" in the:
Clinical Journal of Sports Medicine 2005; in press
British Journal of Sports Medicine 2005; **39**: 196–204
Neurosurgery 2005; in press
Physician and Sportsmedicine 2005; in press

Appendix B – Questionnaire

Demographic Information:

a) Please indicate the level of Bantam Hockey your child participates in:

1 A ❑
2 AA ❑
3 AAA ❑

b) Please indicate your child's current age:

1 12 years ❑
2 13 years ❑
3 14 years ❑

c) Have ***you***/do ***you*** participate in high level (ex. Pro or semi pro), medium level (ex. Competitive leagues), or low level (ex. Recreational) sports:

1 High level ❑
2 Medium level ❑
3 Low/Rec. level ❑
4 No sport participation ❑

d) What is your status of guardianship to the participating child?

1 Mother ❑
2 Father ❑
3 Male Legal Guardian ❑
4 Female Legal Guardian ❑

Questionnaire:

1) Does a loss of consciousness determine whether a concussion has occurred? (Please check one)
❑Yes **1** ❑No **2**

2) Can a player who has suffered a concussion return to play in the same day? (Please check one)
❑Yes **1** ❑No **2**

3) A concussion may be caused by a blow to the neck, jaw, or elsewhere in the body? (Please check one)
❑Yes **1** ❑No **2**

4) Is it necessary for a player to be medically evaluated after having their bell rung? (Please check one)
❑Yes **1** ❑No **2**

5) The following are signs and symptoms of concussion.
(Please circle True or False)

T	F	Headache
T	F	Neck pain
T	**F**	Difficulty with urination
T	F	Dizziness
T	**F**	Lowered pulse rate
T	F	Ringing in the ears
T	F	Feeling dazed or in a “fog”
T	**F**	Difficulty with defecation
T	F	Difficulty falling asleep
T	F	Slurred speech
T	F	Difficulty concentrating
T	F	Drowsiness/fatigue
T	**F**	Hearing voices
T	**F**	Sinus congestion
T	F	Inability to describe time and place
T	F	Seizures
T	**F**	Feelings of euphoria
T	**F**	Inability to swallow
T	**F**	Chest pain
T	F	Feeling of “pressure” in the head
T	F	Difficulty with memory
T	F	Feeling nauseous
T	F	Problems with vision
T	F	Increased emotion/irritability
T	F	Increased sleeping

6) Has your child ever suffered a concussion? (Please check one)
❑Yes **1** ❑No **2**

Appendix C—Information handed out to parents after survey.

Thank you for participating in our study! The purpose of this study was to assess the ability of a parent/guardian to recognize the **proper** signs and symptoms of a concussion in their child. You were asked to answer ***"yes/no"*** or ***"True/False"*** to a series of statements. Here is a list of the ***CORRECT*** answers:

Q#1 Does a loss of consciousness determine whether a concussion has occurred? (Please check one)
ANSWER: no, a concussion can occur without a loss of consciousness!!

Q#2 Can a player who has suffered a concussion return to play in the same day? (Please check one)
ANSWER: absolutely not

Q#3 A concussion may be caused by a blow to the neck, jaw, or elsewhere in the body?
ANSWER: yes, a concussion can occur from a blow to anywhere in the body if the force is subsequently transferred to the head

Q#4 Is it necessary for a player to be medically evaluated after having their bell rung?
ANSWER: YES

Q#5 This question asked you to circle "true/false" for a variety of signs and symptoms. These are the ***CORRECT*** signs and symptoms of a concussion:

Headache	Neck pain
Dizziness	Ringing in the ears
Feeling dazed or in a "fog"	Difficulty falling asleep
Slurred speech	Difficulty concentrating
Drowsiness/fatigue	Inability to describe time and place
Seizures	Feeling of "pressure" in the head
Difficulty with memory	Feeling nauseous
Problems with vision	Increased emotion/irritability
Increased sleeping	

The following signs and symptoms were ***INCORRECT:***

Difficulty with urination	Lowered pulse rate
Difficulty with defecation	Hearing voices
Sinus congestion	Feelings of euphoria
Inability to swallow	Chest pain

Additional Information on Concussions

A concussion is defined as a traumatically induced physiological disruption of brain function with a short period of altered or loss of consciousness. This cannot be seen on CT or X rays. It affects the way your child thinks and remembers things and may cause a variety of symptoms.

What do I do if my child suffers a concussion??
Your child should STOP playing immediately! They should not be left alone and should be taken for evaluation at soon as possible. If the child has lost consciousness, they should be taken to the hospital by ambulance. Do not move the child until paramedics arrive.

How long will it take for my child to recover?
Signs and symptoms often last for 7–10 days, but my last longer. In some cases, the child may take weeks or months to recover. If your child has had a previous concussion, it may take longer for them to recover.

How is a concussion treated?
The best treatment for concussion is rest. They should not exercise, go to school or do any activities that may make them worse. If your child goes back to activity too soon, their symptoms may get worse, or be present longer.

When can my child return to school?
Sometimes a child that suffers a concussion can find it difficult to concentrate and could potentially feel ill or get a headache if they are in school. Children should stay at home if their symptoms get worse while attending school. Once they feel better, they can try going back to school at first for half days and if they are okay with that, then they can go back full time.

When can my child return to sport?
It is very important that your child not go back to sports if he/she has any concussion symptoms or signs. Return to sport and activity must follow a step-wise approach:

1) No activity, complete rest. Once back to normal and cleared by a doctor, go to step 2.
2) Light exercise such as walking or stationary cycling, for 10–15 minutes.
3) Sport specific activity (ie. skating in hockey, running in soccer), for 20–30 minutes.
5) "On field" practice with body contact, *once cleared by a doctor.*
6) Game play.

Note: Each step must take a minimum of one day. If your child has any symptoms of a concussion (e.g. headache, feeling sick to his/her stomach) that come back either during activity, or later that day, your child should stop the activity immediately and rest for 24 hours. Your child should be seen by a doctor and cleared again before starting the step wise protocol again.

When should I take my child to the doctor?
Every child who gets a head injury should be seen by a doctor as soon as possible. You should take him/her back to the doctor IMMEDIATELY if, after being told your child has a concussion, he/she has worsening of symptoms such as:

1. being more confused
2. has a headache that is getting worse
3. vomits more than once
4. doesn't wake up
5. has any trouble walking
6. has a seizure
7. has strange behaviour

No child should go back to sport until they have been cleared to do so by a doctor.

Please take note of the true signs and symptoms of a concussion and seek proper evaluation if you suspect your child has potentially sustained a concussion. If you would like more information about the purposes or results of this study, please feel free to contact us:

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Exercise related transient abdominal pain: a case report and review of the literature

Dr. Brad Muir, HBSc(Kin), DC, FCCSS(C)*

Exercise-related transient abdominal pain (ETAP) is more commonly known to athletes as a runner's stitch. Many athletes also report shoulder tip pain (STP) associated with the ETAP. Although widely known, ETAP remains under analyzed and under reported in the medical literature. Often thought of as benign and self-limiting, ETAP has been shown to be very detrimental to the performance of many athletes from novice to elite. This case report of an elite triathlete with ETAP and subsequent review of literature, outlines the various theories about the etiology of ETAP, the epidemiology associated with it, some differentials to consider, and how chiropractic care may benefit those suffering from ETAP.
(JCCA 2009; 53(4):251–260)

KEY WORDS: exercise, abdominal pain, stitch, cramp, runner's stitch, runner's abdominal pain, subcostal pain

La douleur abdominale passagère liée à l'exercice (DAPE) est mieux connue par les athlètes sous le nom de point du coureur. De nombreux athlètes rapportent également une douleur à la pointe de l'épaule (DPE) associée à la DAPE. Quoiqu'elle soit bien connue, la DAPE est peu analysée et peu déclarée dans la littérature médicale. On a démontré que la DAPE, souvent considérée bénigne et autolimitative, peut être très nuisible au rendement de bon nombre d'athlètes des catégories novice à élite. Le rapport de cas d'un triathlète de haut niveau avec DAPE et la revue de la littérature ultérieure soulignent les diverses théories concernant l'étiologie de la DAPE, l'épidémiologie qui y est associée, certains différentiels à considérer et comment les soins chiropratiques peuvent profiter aux personnes qui souffrent de DAPE.
(JACC 2009; 53(4):251–260)

MOTS CLÉS : exercice, douleur abdominale, point, crampe, point du coureur, douleur abdominale du coureur, douleur sous-costale

Introduction

Exercise-related transient abdominal pain (ETAP) is more commonly known to athletes as a stitch, stitch in the side, side cramp, side ache and subcostal pain.[1,2] Many athletes also report shoulder tip pain (STP) associated with the ETAP.[1] Although widely known, ETAP remains under analyzed and under reported in the medical literature.[2]

Case report

History

A 20 year old triathlete presented to the clinic complaining of a 1.5 to 2 year history of right sided abdominal pain that only occurred with training. He has been a triathlete for 4 years and has always been aware of some cramping but never this severe. The onset was unclear

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but the athlete felt it might have been due to a new bike that wasn't set up for him properly and seemed to force him into a more kyphotic posture in the thoracic spine. The cramping was aggravated by his heart rate getting over 170 bpm which occurred frequently during "intensity training," the impact of running, oblique exercises during gym workouts and performing a side bridge exercise. He stated that the pain would start during the swim portion of the triathlon and worsened with the bike and run. The pain was always localized to the upper right quadrant of his abdomen in the area immediately inferior to the ribs. He related that the pain would progress to the right diaphragm on occasion but did not mention any related shoulder tip pain. During an episode, the pain would start as a "tightening" and progress to a "sharp pain." He also reported trouble breathing when the pain would get intense. He could push himself through the pain without the pain getting any worse but, on two occasions, was unable to complete races; one during the run phase in 2003 and one during the swim phase in 2004. The intensity of the pain would often reach a 9 on a 10 point scale (10 being the worst pain) with a varied time in onset during training.

Other associated symptoms included tight "hips" and cramping in his right posterior ribs with running. He has had an x-ray and an ultrasound of his abdomen which were read as normal. His previous treatment for this injury included acupuncture, ART to his psoas and iliacus, and a core stability program with little success.

The patient was a student and he did not report having the abdominal pain with any of his normal activities of daily living.

Physical examination

A physical exam revealed bilateral hip tightness particularly of the hip flexors, extensors and abductors. Muscle testing revealed inhibition in the psoas bilaterally (right > left), gluteus medius bilaterally (right > left), and the right external rotators of the hip. (Added note by author: in this case inhibition was used to describe non-painful, non-neurologic weakness of these muscles.) Motion palpation revealed restrictions in the thoracic spine and thoracolumbar junction. In particular, the T9 facet on the right and its corresponding costovertebral joint recreated a milder version of the pain of chief complaint in the abdomen. Tender points revealed by digital palpation were found in the psoas, rectus femoris, gluteus medius, spinal erectors, latissimus dorsi and iliacus bilaterally, and the right TFL, QL, internal oblique, and transverse abdominus. None of these tender points recreated the pain of the chief complaint.

Diagnosis and treatment

The patient was subsequently diagnosed with exercise-related transient abdominal pain (ETAP) with associated lower kinetic chain myofascial dysfunction as well as thoracic and thoracolumbar joint dysfunction. He was subsequently treated with ART®, spinal manipulative therapy and exercise to affected areas for four treatments over the course of a month. Exercise therapy included psoas, rectus femoris and QL stretches, core stability exercises on the stability ball, gait and balance training on a balance beam and running drills stressing proper gait mechanics (eg. walking A's).

The patient was asked to keep a log of activity, training intensity and ETAP intensity over the course of the month. His training intensity remained high while training 5–6 days per week. His ETAP intensity varied from a low of 2/10 to a high of 8/10 (on two training days) and was consistently below 5/10. The patient was extremely happy with the decrease in pain with treatment (9/10 to 5/10 on average with a low of 2/10) but was unable to continue treatment due to extensive travel in order to comply with the plan of management.

Discussion

Epidemiology

It has been reported that "GI symptoms such as nausea, vomiting, belching, bloating, heartburn, chest pain, GI cramps, side ache, and diarrhea are experienced by 20–50% of athletes."[3] They are receiving more and more attention by athletes, coaches and medical practitioners due to the detrimental affect these symptoms can have on performance.[3] In their study, Peters et al[3] found that runners experienced more lower GI symptoms than upper (71% versus 36%) while cyclists had both upper (67%) and lower (64%). This was confirmed by the triathletes in their study who experienced more lower (79%) than upper (54%) while running and both upper (52%) and lower (45%) while cycling.[3] Side ache was considered to be a part of the lower GI symptoms.

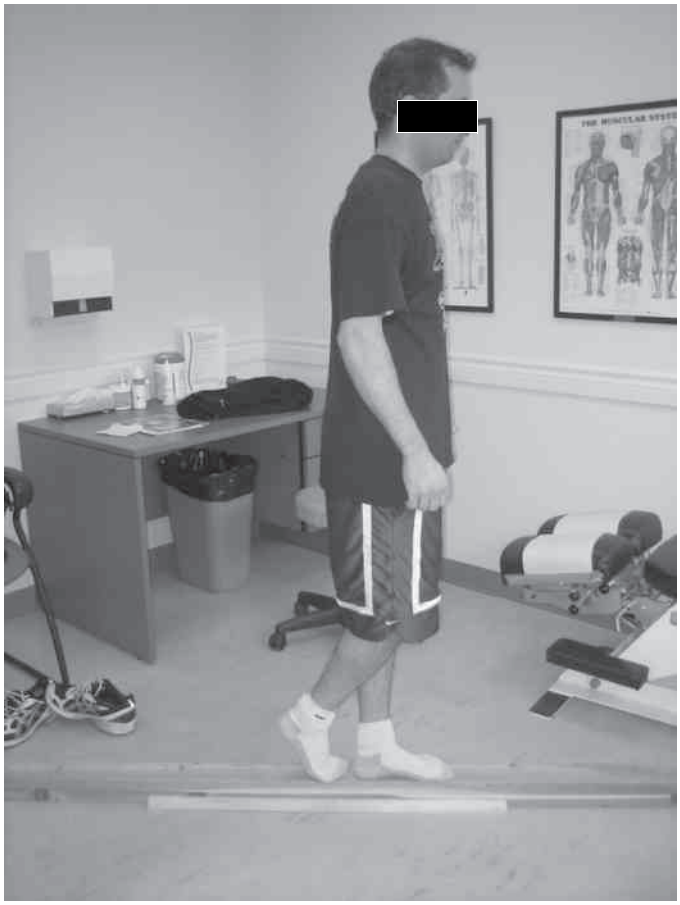

Figure 1 *Balance beam exercises*

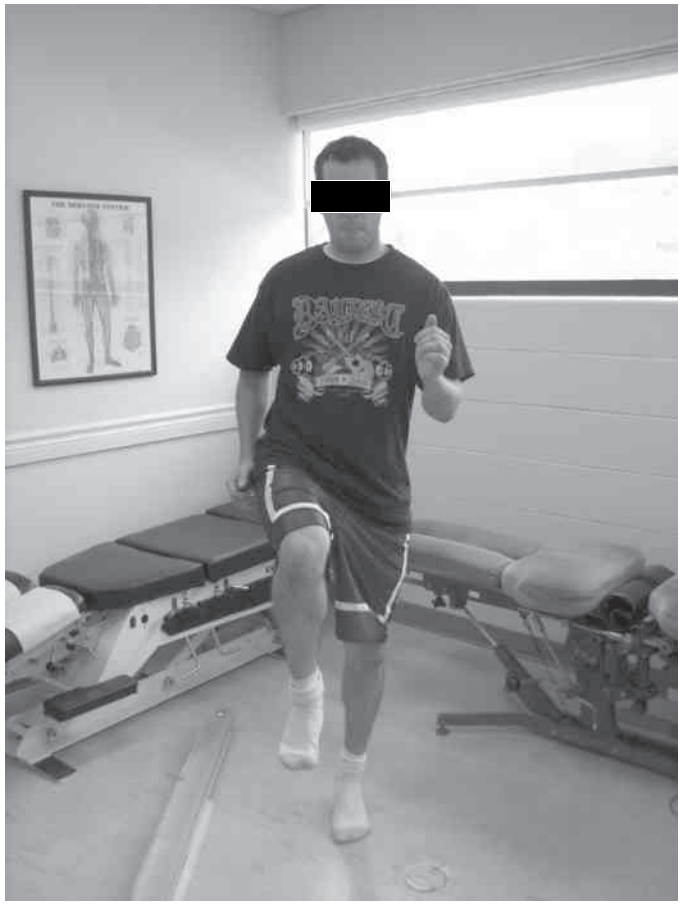

Figure 2 *Walking A's for gait mechanics*

Morton and Callister,[2] in a study of 965 athletes in 6 sports found that 61% of athletes had experienced ETAP over the period of 1 year. Broken down by sport, swimmers reported having had ETAP the most (75%), followed by runners (69%), horseback riding (62%), aerobics (52%), basketball (47%) and cycling (32%). In another study by Morton, Richards and Callister,[4] it was found that during a 14 km community walk/run 31% of the participants had experienced ETAP and 42% of those said it negatively affected their performance. ETAP does not seem to affect the athlete during every exercise session. Morton and Callister[2] found that 52% of the athletes reported it occurred less than 10% of the time while 82% said it occurred in no more than 20% of their exercise sessions.

Although often thought of as mild and benign, in those athletes that experienced ETAP, 84% of them reported that ETAP caused them to reduce their intensity level (72%) or stop the activity altogether (12%).[2] These results indicate that over half of the athletes polled felt that ETAP had a deleterious effect on their performance. Our athlete described several incidents where he was forced to slow down as a result of ETAP and in a couple of instances had to stop completely.

Shoulder tip pain (STP) has been found to be a component of ETAP for some athletes but not all.[2] In their survey, Morton and Callister[2] defined the STP as non-injury related that localized to the lateral 1/3 of the trapezius border extending to the acromion consistent with referred pain from the diaphragm. They found that 34% of the athletes had non-injury related shoulder pain but only

47% of those localized it to the aforementioned part of the trapezius. This constituted 14% of the total respondents. Again broken down by sport, runners experienced the most STP at 17%, followed by swimming (15%), aerobics (11%), basketball (10%) and cycling (4%) (horseback riding was not included).[2] In our case, the athlete did not report any STP associated with his ETAP.

ETAP and STP are also not mutually exclusive. Most often those with STP had ETAP (18%) but there were some athletes that only reported STP (8%).[2,5]

Location and character

It is a commonly held clinical belief that ETAP occurs mostly on the right side and is a benign cramping sensation relieved after a few seconds of rest but this is not entirely true for every athlete with ETAP.[2]

ETAP is well localized in 79% of the cases and in the same spot in 62%.[2] The most common area for ETAP is the right middle third of the abdomen (just adjacent to the umbilicus) in 58% of athletes followed by the left middle third (43%) and umbilical area (21%)(percentages were >100% due to people experiencing ETAP in more than 1 area).[2] ETAP, however was experienced to a lesser degree in all areas of the abdomen.[2] Our athlete consistently reported the location of his ETAP to be in the right upper quadrant of the abdomen.

Using Melzack and Torgersons 10 pain descriptors, Morton and Callister[2] found that the pain was described most often as sharp (35%), cramping (27%), stabbing (15%), aching (9%) and pulling (6%). In terms of severity, they found that ETAP was most severe at 4.7 +/– 0.1 on a 10 point numerical rating scale with stabbing being the most severe followed by sharp, aching, cramping, and pulling.[2]

Those athletes who reported their ETAP to be the most severe experienced ETAP more frequently, had more residual soreness and felt that the pain took longer to resolve.[2]

Factors affecting ETAP and STP

Very few studies have looked at the factors that affect ETAP and STP. It has been thought that ETAP is more pronounced in younger athletes and those athletes that are less fit.[5] In an attempt to elucidate these and other factors affecting ETAP, Morton and Callister,[5] in their survey of 965 athletes, asked for subjective data on height, weight, age, and training volume and frequency. Their findings are summarized below.[5]

- **Age:** Consistent with clinical observation, the prevalence, severity and frequency of ETAP and STP were both significantly decreased with increasing age. Although the severity of the pain on the 10 point numerical scale was found to significantly decrease with age, the descriptors used for the pain sensation were not significantly affected by age. The reason for the changes seen due to age were not easily explained using the current theories of the cause of ETAP and STP.
- **BMI:** The prevalence and frequency of ETAP and STP were not affected by BMI although those with a higher BMI had more localized ETAP and reported more severe STP but not ETAP.
- **Gender:** Few gender differences with respect to ETAP and STP were found. Men were more likely to report ETAP as "aching" compared to women.
- **Training:** Training more frequently significantly decreased the frequency of ETAP in those surveyed but had little effect on the severity and prevalence. These two measures also seemed unrelated to the number of years in the sport or training volume. Morton and Callister[5] concluded that, contrary to the popular belief that novice athletes who were less fit would experience more severe ETAP and STP, athletes of all levels were just as susceptible.

Provoking factors

Morton and Callister[2] have also surveyed athletes with respect to those factors that may provoke ETAP. Because of its transient nature, many athletes, in an attempt to explain their symptoms, link certain "things or conditions" with their ETAP. The following is a list of the subjective factors that the athletes reported as provoking their ETAP:

- eating (52%) and drinking (38%) – these could be broken down further into specific types of food and drink – high sugar content (37%), water (30%), fatty foods (16%), fruit and fruit juices (15%), and dairy products (10%);
- poor fitness level;
- high exercise intensity; competition;
- lack of warm-up;
- exercising in cold conditions.

Peters[6] attempted to determine if the osmolality of supplements administered to triathletes during bouts of running and cycling increased their gastrointestinal symptoms. Each athlete completed three trials with different supplementation separated by one week. The three supplements included a hypertonic (higher osmolarity) high energy drink of 40 mg of carbohydrate in 100 ml of water, a semi-solid supplement mixture of banana, white bread, marmalade, and water and an equal volume of a placebo fluid with flavouring similar to the high energy drink. Following the ingestion of the supplement prior to each bout of exercise, a hypotonic thirstquencher (4 mg of carbohydrate (orange juice) and 100 ml of water) was administered at fifteen minute intervals during each bout. The placebo group received a thirstquencher flavoured placebo in a similar time interval. With respect to ETAP (side ache in the study) and the osmolality of the supplement, there was a significant difference found between the 1st running bout and the 2nd cycling bout for the high energy and semi-solid supplements ($P < 0.01$) as well as the placebo supplement ($P < 0.05$). There was also a significant difference for the high energy supplement and side ache ($P < 0.01$) between the 1st cycling bout and the 1st running bout. This suggests that hypertonic fluids may be slightly more provoking factor in ETAP during running compared to cycling but all three supplements did provoke ETAP.[2,6] There was no significant difference between the three supplements and the occurrence of ETAP. Peters,[6] in his discussion, suggested that the supplements may have been "mildly hypertonic" during the exercise bouts due to the mixing of the hypotonic thirstquencher throughout each trial. This may have contributed to the insignificant findings.

Plunkett[7] also attempted to determine if osmolality played a role in the development of ETAP. He found during progressive bouts of exercise, hypertonic fluids caused a significantly greater amount of ETAP compared to no fluids and hypotonic fluids. The fluids used in order of increasing osmolarity were no fluids, water, Exceed (a commercial energy drink), Coca-cola, and Duphalac (a solution of the sugar lactulose). This suggests that hypertonic fluids may play a role in ETAP during sustained bouts of exercise.

More research is needed to determine if the osmolality of food or fluids has an influence on ETAP and/or its intensity.

Etiology – proposed theories

The exact etiology of ETAP has yet to be elucidated although there are numerous proposed theories.

Diaphragmatic ischemia

One of the traditional causative explanations of ETAP is diaphragmatic ischemia due to the shunting of blood from the respiratory muscles to the gut or to the muscles involved in movement.[2]

Origins of this theory are rooted in the referral of the diaphragm to tip of the shoulder, the subjective association of eating and drinking and increasing intensity (increased respiration) with ETAP, and the most common location of ETAP being the periumbilical/sub-diaphragmatic regions.[2,5,7]

This theory is unlikely because of the pain of ETAP being as low as the iliac and hypogastric regions and the low incidence level of STP (14%) compared to ETAP (61%).[5] A study done by Roussos and Macklem[8] refuted the shunting theory in part by showing that during decreased cardiac output, the respiratory muscles may actually deprive the rest of the body of blood. This makes sense from a survival standpoint – breathing is the most essential component of life. Plunkett[7] reported on another study that showed that following the ingestion of a large meal, there was no change in diaphragmatic movement under fluoroscopy with those runners experiencing ETAP. Although the diaphragm may be involved in ETAP, this suggests that diaphragmatic ischemia was not the cause.

Visceral ligament stress

Another early competing theory for the cause of ETAP and STP was stress on the visceral ligaments (gastrophrenic, lienophrenic and coronary ligaments) connecting the diaphragm to the abdominal organs.[2,7] Early anecdotal evidence suggested that ETAP may be caused by the vertical jolting in such sports as camel riding, horseback riding and driving vehicles "off road."[7] Morton and Callister[2], and Plunkett[7] report that Sinclair was the first to propose the theory of visceral ligament stress. Visceral ligament stress could account for STP referral from the diaphragm, the varied location of ETAP in the abdomen which Sinclair felt was due to referred pain from the ligaments themselves, and the subjective increase in ETAP following meals.[2,7] Plunkett[7] also report-

ed a case of a conscious patient who reported ETAP-like pain during abdominal surgery when there was traction on the mesentery.

Although the above observations lend credence to the visceral ligament theory, the fact that abdominal pain is usually along the midline and described as dull and not well localized is in contrast to the pain of ETAP.[2] ETAP is usually well localized, all over the abdomen and can be sharp or stabbing in character.[2]

Plunkett[7] attempted to test the first two theories of ETAP by having athletes digest fluid of varying absorbability and use various physical methods in an attempt to increase or decrease the amount of ETAP. Their results, although in a small sample size, showed little evidence for the diaphragmatic ischemia theory. More of their outcomes pointed in the direction of the visceral ligament stress theory although conflicting results still left some doubts as to this theory's applicability.

Muscle cramp

A considerable number of athletes (27%) describe ETAP as a cramping sensation[2] and in turn feel that a muscular cramp may be the root cause. Although the varied location of ETAP and good localization may be explained by the muscle cramp theory, its ability to explain the cause of STP is not. Muscle cramping is not highly regarded in the literature as a possible theory of ETAP and even its own cause is not well understood.[2]

Morton and Callister[9] recorded surface EMG in the area of pain during an episode of ETAP in 14 symptomatic individuals. There was no change in EMG activity immediately post exercise following the onset of ETAP compared to immediately after the pain had subsided. Both recordings were taken with the subject semi-reclined and with them holding their breath in order to reduce the activity of the abdominals and diaphragm respectively. They also recorded EMG during specific tests designed to activate the respiratory musculature which was detected at the site of ETAP yet was not recorded during an episode of ETAP. This suggests that respiratory muscle cramping was not responsible for the pain associated with ETAP.[9]

Morton and Callister[10] compared spirometry results before and after an episode of ETAP in 14 subjects that developed symptoms during a treadmill test. They found that there were no significant changes in spirometry values suggesting that the diaphragm is not directly related to the cause of ETAP. This further reinforces that diaphragmatic ischemia and respiratory muscle cramping are not involved in the etiology of ETAP.

Parietal peritoneum irritation

The irritation of the parietal peritoneum is another competing theory of the etiology of ETAP.[2] The parietal peritoneum consists of the layers of tissue that line the abdominal wall and the pelvic cavity. The visceral peritoneum is the layer of tissue lining the abdominal organs separating them from the parietal peritoneum by a potential space known as the peritoneal cavity. This cavity is filled with a serous fluid to alleviate friction between the two layers.[11]

In their study in 2000, Morton and Callister[2] felt that ETAP may be a form of exertional peritonitis secondary to friction between the parietal and visceral folds. Their argument is summarized below:

- the multi-level innervation of the viscera explaining the varied location of ETAP;
- the innervation of the subdiaphragmatic portion of the parietal peritoneum by the phrenic nerve could, potentially, cause STP;
- the highly localized and sharp pain with irritation of the abdominal part of the parietal peritoneum;
- the distension of the stomach with a large meal may increase the friction between the surfaces;
- the friction between the surfaces could be affected by the osmotic gradient in the viscera explaining the subjective changes in ETAP with hypertonic food and drink;
- the fact that the parietal peritoneum is sensitive to movement when irritated;
- the change in sensation in ETAP with increasing intensity of movement which would be consistent with an increase in the amount of friction.

These points led them to the conclusion that ETAP was linked to the irritation of the parietal peritoneum.[1,2]

Thoracic facet referral

Morton and Aune[12] reported on a case of ETAP in an elite 25 year old middle distance runner that experienced increasingly frequent bouts of increasingly severe ETAP

following an injury to the thoracic spine. The exact pain of ETAP for this patient was recreated with palpation of the thoracic facet that corresponded to the dermatomal level of innervation to the painful area. Following this discovery, they examined 17 other runners to further investigate this theory. They found that in 47% of the athletes, their exact sensation of ETAP was reproduced with palpation of the thoracic facets from T8 to T12 and in another 35% they reported pain referral toward the site of ETAP. They also reported that an increased thoracic kyphosis is provocative of ETAP.[12] This theory is consistent with the findings of ETAP with jolting activities such as horseback riding, all terrain driving/racing and camel racing. The dermatomal innervation of the abdomen by the various levels of the thoracic spine[11] may also explain ETAP's varied locations and its consistent localization. Thoracic facet referral does not explain STP however.

In our case, the athlete reported a similar sensation (just less intense) to his ETAP with palpation of his right mid-thoracic spine facet and costovertebral area. This suggests that his ETAP in part was associated with some thoracic spine dysfunction.

Psoas major and Quadratus lumborum

The medial arcuate ligament of the diaphragm forms a fascial arch covering the psoas major muscle and blending with its fascia.[11,13] The lateral arcuate ligament of the diaphragm has a similar attachment with the quadratus lumborum muscle.[11,13] These muscles play an important role in the segmental stabilization of the lumbar spine.[14,15] It is conceivable that tightness/dysfunction in these muscles could result in the subdiaphragmatic pain of ETAP and STP secondary to the attachment to the diaphragm. This could explain the prevalence of ETAP in those sports that cause vertical jolting (horse back riding, camel racing) and those that require repetitive hip flexion (running, cycling, swimming, aerobics). It also explains the increase in ETAP during intense intervals with these sports because of the increase in the static and dynamic activation of the psoas and quadratus lumborum muscles and the increase in diaphragm activation secondary to increased oxygen demand. It does not, however, explain the various locations of pain in the abdomen with ETAP.

Our athlete had considerable tightness, dysfunction and inhibition in his psoas and QL bilaterally (right greater than left) which may have contributed to his ETAP.

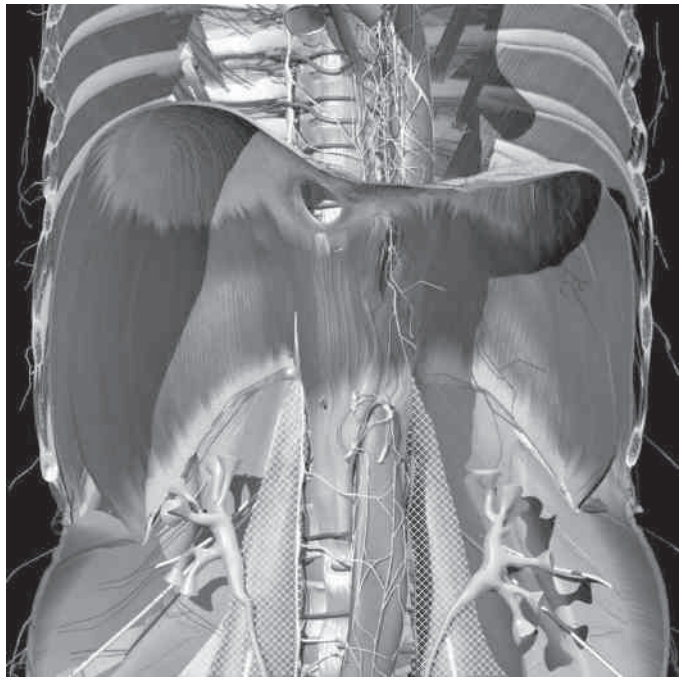

Figure 3 *Psoas major fascia continuous with medial arcuate ligament.* (courtesy and copyright Primal Pictures Ltd www.primalpictures.com)

Differential diagnoses

Eichner[16] outlines several differential diagnoses that need to be considered when an athlete presents with symptoms of ETAP. Also, because ETAP has a relatively consistent localization and character of pain, a change in location and intensity in an athlete with a previous diagnosis of ETAP must be re-examined and other differentials must be ruled out based on the location of the pain. Although there are many causes of abdominal pain, the following outlines a few differential diagnoses in which symptoms are increased with exercise including:[16]

- **upper left quadrant**: Splenic enlargement may cause upper left quadrant pain and is common in mononucleosis. Splenic infarction must be considered in athletes with sickle cell trait when training or competing at altitude.
- **flank pain**: An athlete with acute, severe flank pain especially with radiation into the groin may be experiencing renal colic due to kidney stones. If the flank

pain is acute and bilateral, acute renal failure must be considered as well as rarely diagnosed exercise-induced loin pain syndrome.
- **upper quadrant pain above the costal margin**: Especially in rowers, a stress fracture of the ribs or intercostal muscle strain must be entertained with stabbing pain above the costal margin.
- **upper medial quadrant**: With midline pain in the upper quadrant, abdominal wall muscle strain may mimic ETAP and must be ruled out.
- **lower quadrant**: If the pain presents in the lower quadrant pain, exercise associated intestinal ischemia could be the cause.

Eichner[16] reminds us of the old adage, depending on what part of the world you are from, when you hear hoof beats you think horses but it may also be zebras. The following include several zebras easily mistaken for classic ETAP:

Chronic constipation

Anderson[16,17] reported the case of a 14 year old female runner who reported ongoing, well localized, periumbilical pain that was worsening over the period of 4–5 months prior to examination. The pain was initially described as a dull ache that progressed to a sharp pain when running (rated as 6/10) but always resolved within a few minutes of stopping. On presentation, her pain had increased in frequency and severity (now rated as 9/10) and was forcing her to stop running. Her physical exam and laboratory studies were within normal limits. An x-ray showed a moderate amount of stool located diffusely throughout her colon but was within normal limits. On consultation with a gastroenterologist, he suggested the jarring from running may be provoking a clinically silent and relatively mild constipation. The patient was prescribed psyllium and within 10 days her pain with running had resolved. On follow-up 6 months later, she had continued to maintain her fibre intake and was still pain free with running.

Adhesion of ascending colon to anterior abdominal wall

Lauder and Moses[16,18] reported the case of a 28 year old elite, male triathlete with no previous history of abdominal pain who suffered an acute attack of sharp right-sided abdominal pain during the run portion of a triathalon. After the first episode, he continued to have a similar pain during training while running and while swimming to a lesser degree. His pain continued to be severe during other races and he subsequently underwent further evaluation that revealed adhesions between the ascending colon and the anterior abdominal wall. The adhesions were surgically lysed and the athlete was able to resume training and subsequent races in a pain free state.

Chronic cholecystitis

Dimeo et al[16,19] reported on a 29 year old elite, male, long distance runner that had had a 12 year complaint of severe, stitching type, upper right quadrant abdominal pain with strenuous, intense training and racing. The pain had worsened a few months prior to presentation and in the prior few weeks had progressed to a similar upper right quadrant pain following eating fatty meals. Laparoscopic abdominal surgery was performed and revealed congenital adhesions anteriorly between the gallbladder and liver to the abdominal wall and posteriorly between the gallbladder, transverse colon and liver. The adhesions were sectioned and the gallbladder was removed revealing a chronic cholecystitis on histological examination. No post-operative complications occurred and on two year follow-up, he had raced several times with no return of symptoms.

Median Arcuate Ligament Syndrome

Desmond and Roberts[20] reported on a case of ETAP in a 21 year old elite middle distance runner. He reported recurrent upper abdominal discomfort and diarrhea with increased intensity of training. He had had symptoms since he was 15 years old. He was found to have compression of the celiac artery by the median arcuate ligament. The median arcuate ligament is a tendinous band uniting the medial portion of the two crura of the diaphragm.[11] His symptoms of diarrhea and ETAP were completely relieved by surgery and he was able to return to competitive running.

Treatment

Thoracic mobilization/manipulation

In their case study, Morton and Aune[12] reported that mobilization of the affected thoracic facet along with mobi-

lizing exercises over a four week period reduced the pain of ETAP and allowed the patient to return to competitive running.

In a pilot study by Schiller[21], spinal manipulative therapy of the thoracic spine was found to be more effective than placebo treatment for mechanical thoracic spine pain. This suggests that SMT may be effective in the treatment of ETAP if thoracic spine symptoms are present. DeFranca and Levine[22] also found some success with manipulation of the thoracic spine for T4 syndrome.

Our patient was manipulated in the thoracic spine as part of his treatment regimen which may have contributed to his reduction in symptoms.

Soft tissue therapy and stretching

Our patient was treated with ART® of his psoas and quadratus lumborum specifically as well as other gluteal and hip musculature for a period of 1 month. Follow-up stretching of the QL and psoas were prescribed for the patient. Treatment and stretching of the patient's psoas and QL may have contributed to his reduction of symptoms.

Dietary modifications

A diet log and food/beverage intake schedule during training may reveal any consistent triggers of ETAP and lead to subsequent dietary alterations in an attempt to prevent the onset of ETAP. Hypertonic supplements (food or fluid) may produce more ETAP then hypotonic supplements although more research needs to be done in this area.[6,7] By monitoring intake during training, athletes with food or fluid triggers may be able to identify and avoid these triggers thus avoiding a decrease in performance due to ETAP. It is also suggested that non-symptomatic supplementation during training be maintained during race days.

Our athlete did not report any food or drink triggers.

Other

In their survey, Morton and Callister[2] asked how those athletes that suffered from ETAP treated it. Their treatments included bending over forward (18%), stretching the affected site (22%), deep breathing (40%), and pushing on the affected area (31%). Deep breathing and bending forward were reported as the most effective by those that that suffered ETAP more severely.[2] In the study by Plunkett,[7] three methods of reducing stitch were employed including: contraction of the abdominals; modified breathing; and tightened abdominal belt. They found that stitch was relieved rapidly and significantly with these maneuvers and its intensity was restored within 30–60 seconds of them being stopped.

Our athlete did not report any self-treatment methods.

Conclusions

ETAP is a very common condition known to most athletes. Although most often thought of as minor annoyance during training and competition, Morton and Callister[2] have shown that it can be severe enough to cause an athlete to stop completely. Its cause at this time is unknown but several theories have been put forward. This suggests that more time should be spent on elucidating its cause and subsequent treatment in an effort to reduce its impact on those athletes that suffer from ETAP.

Limitations and directions for future study

One of the limitations of this case study was the inability of the patient to continue treatment. It is feasible, although unlikely due to continued training, that symptom resolution was due to natural history or other training modifications. With continued treatment longer than 1 month, the ETAP intensity may have returned or resolved completely. An analysis of his swimming, running and cycling biomechanics may have been beneficial to elucidate training errors but was not undertaken.

Future study is needed to determine the underlying cause of ETAP, in particular whether it can be explained by one unified theory or if various subtypes of ETAP exist. Many injuries are the result of a combination of factors including training errors, poor biomechanics, muscular imbalance and poor nutrition. To find the individual cause in a multisport athlete can be like trying to find several needles in the proverbial haystack but finding them could help further direct treatment and prevention strategies.

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Traumatic anterior shoulder dislocation: a case study of nonoperative management in a mixed martial arts athlete

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Objective: *To present an evidence-informed approach to the nonoperative management of a first-time, traumatic anterior shoulder dislocation.*

Clinical Features: *A 30-year-old mixed martial arts athlete, with no prior shoulder injuries, presented one day following a first-time, traumatic anterior shoulder dislocation. An eight-week, individualized, intensive, nonoperative rehabilitation program was immediately begun upon presentation.*

Intervention and Outcome: *Management consisted of immobilization of the shoulder in external rotation and a progressive rehabilitation program aimed at restoring range of motion, strength of the dynamic stabilizers, and proprioception of the shoulder. Eight weeks post-dislocation the patient had regained full range of motion and strength compared to the unaffected limb and apprehension and relocation tests for instability were negative.*

Conclusion: *This case illustrates successful management of a first-time, traumatic, anterior shoulder dislocation using immobilization in external rotation combined with an intensive rehabilitation program.*
(JCCA 2009; 53(4):261–271)

KEY WORDS: shoulder, dislocation, rehabilitation

Objectif : *Présenter une approche fondée sur des faits vis-à-vis du traitement non chirurgical d'une première dislocation traumatique de la partie antérieure de l'épaule.*

Caractéristiques cliniques : *Un athlète d'arts martiaux mixtes de 30 ans, sans blessures antérieures à l'épaule, s'est présenté un jour à la suite d'une première dislocation traumatique de la partie antérieure de l'épaule. Un programme de réadaptation non chirurgical intensif et individualisé de huit semaines a commencé immédiatement après l'examen.*

Intervention et résultats : *Le traitement consistait en une immobilisation de l'épaule en rotation externe et un programme de réadaptation progressif ayant pour but de rétablir l'amplitude des mouvements, la force des stabilisateurs dynamiques et la proprioception de l'épaule. Huit semaines après la dislocation, le patient a retrouvé toute l'amplitude des mouvements et la force comparativement au membre non touché et les tests d'appréhension et de recentrage étaient négatifs.*

Conclusion : *Ce cas illustre le traitement réussi d'une première dislocation traumatique de la partie antérieure de l'épaule au moyen de l'immobilisation en rotation externe avec un programme de réadaptation intensif.*
(JACC 2009; 53(4):261–271)

MOTS CLÉS : épaule, dislocation, réadaptation

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Introduction

The shoulder is the most frequently dislocated joint in the body. Anterior dislocation accounts for 94–98% of shoulder dislocations.[1] The incidence of anterior shoulder dislocation has a bimodal distribution with peaks occurring in the second and sixth decade.[2] Initial traumatic dislocation is most commonly the result of a posterior directed force placed on an abducted and externally rotated shoulder. Less commonly, a dislocation may occur as a result of an anteriorly directed force placed directly on the posterior aspect of the humeral head.

Many complications to anterior shoulder dislocations are reported in the literature. A Bankart lesion, an avulsion of the capsulolabral complex from the glenoid rim, is reported to occur in 80–97% of anterior shoulder dislocations.[3] A Bankart lesion is often referred to as the "essential lesion" in a dislocation as it pulls the labrum away from the glenoid and disrupts the attachment of the inferior glenohumeral ligament (IGHL), greatly reducing anterior stability. A displaced labrum reduces the depth of the glenoid by half, and a lax IGHL has been shown to double glenohumeral translation.[1] Hill-Sachs lesions are impression fractures of the articular surface of the humeral head that occur as the dislocated humeral head is snapped back against the glenoid rim. They also occur quite frequently, in approximately 80% of anterior dislocations.[4] While these lesions are usually inconsequential, some authors suggest that a defect greater than 30% of the articular surface may contribute to instability.[5] Axillary nerve injury is a potentially serious consequence of dislocation. Damage to the nerve is cited to occur in 5–35% of anterior dislocations and is most likely due to the nerve's vulnerability as it travels over the subscapularis tendon and inferior to the glenohumeral capsule.[6] Avulsion of the IGHL from its insertion on the humerus, known as humeral avulsion of the glenohumeral ligament (HAGL), is a rare complication of dislocations. The actual incidence of HAGL lesions associated with first-time dislocations is unknown, but they are found in 7–9% of patients with recurrent instability.[7]

The most common complication of anterior shoulder dislocation is recurrence. Research implicates age as the single most important prognostic factor in the development of recurrent anterior dislocations.[8,9,10] A patient's age at the time of initial dislocation is inversely related to the development of recurrence. The literature suggests the recurrence rates of anterior dislocations are quite high in those under twenty ranging from 66–97%, while they are lower in those over forty ranging from 0–21%.[8,10] Since age is the major prognostic factor in the development of recurrent instability, it therefore has a significant role in clinical management. As the literature suggests the patient in this case was at a reduced risk for recurrent dislocation (based on age), an informed decision was made to manage this case nonoperatively.[9,10]

The nonoperative management of anterior shoulder dislocations, as reported in the literature, generally consists of immobilization of the shoulder in a sling and a progressive, individualized rehabilitation program including strength and proprioceptive training. This case discusses an unusual mechanism of injury for first-time, traumatic anterior shoulder dislocation as well as nonoperative management using an intensive, individualized program.

Case

A 30-year-old Mixed Martial Arts (MMA) athlete presented one day following a traumatic, anterior shoulder dislocation. Prior to injury, the athlete was participating in Jiu Jitsu training (a form of martial arts which focuses on ground fighting, or grappling, with emphasis on joint locks, chokes, and various forms of submissions) in which he was sparring with his training partner. The object of this exercise is to force the opponent to concede defeat (or "tap out") with the application of a submission hold. At the time of incident, the patient was in his opponent's "guard" position whereby the opponent was lying supine with his legs wrapped around the patient's waist. The opponent forcefully pulled the patient's left arm into a position of horizontal adduction causing dislocation. The patient described utilizing a self reduction technique to relocate the articulation. The patient had no prior shoulder injuries and this was his first episode of dislocation.

The patient indicated that post-reduction he had immediately begun a program of cryotherapy (10/10/10 protocol several times per day) and was currently taking Ibuprofen. He was instructed to continue this protocol for a period of five days. On examination the patient's left glenohumeral joint range of motion was globally restricted by approximately 20 degrees (external rotation was not attempted) and painful. Apprehension and relocation tests were positive. Radiographs of his left shoulder were

unremarkable. Following physical examination a treatment program was immediately initiated as follows:

Week 0–1:
The initial treatment program consisted of Interferential Current followed by Microcurrent application along the anterior shoulder in a direction parallel to the fibers of the anterior glenohumeral capsule. Interferential Current was set at 120Hz, continuously for pain relief. Microcurrent therapy included settings of 300Hz/300microamps for edema control and 0.3Hz/30microamps for tissue healing.[11] During microcurrent treatment, the patient's shoulder was held in slight (approximately 10 degrees) external rotation. Theoretically a position of slight external rotation was used to take advantage of the piezoelectric effect, which states the orientation of collagen deposition can be controlled using both mechanical and electrical force.[12,13] As well, external stretching or mechanical loading is believed to induce the expression of growth factors beneficial to tissue regeneration and repair.[14] During this period the patient's shoulder was immobilized in a sling in 10–15 degrees of external rotation and he was instructed to remove the sling only when in the shower and performing his prescribed exercises (see figures 1 and 2). In order to maintain this position during sleep, the patient was instructed to position himself on the unaffected side while the affected arm was secured to the torso using tensor bandages. With the elbow flexed to 90 degrees, the weight of the arm was supported on a pillow to maintain a slightly externally rotated position.

Initial rehabilitation focused on addressing scapular stability, maintaining range of motion to prevent stiffening, and maintaining strength in the affected limb using isometric exercises. The patient was instructed on scapular "setting" techniques and was provided with a program consisting of 'low row,' 'robbery' and 'lawnmower' exercises, as described previously.[15] The patient was also instructed to perform isometric exercises into shoulder flexion, extension, abduction, adduction, external rotation, and internal rotation while the shoulder was maintained in a neutral position against the torso. All contractions were to be held for as long as could be tolerated and were performed for three sets each, twice daily. The patient was encouraged to record contraction times in a log and to increase the duration of recorded times on each subsequent occasion when possible.

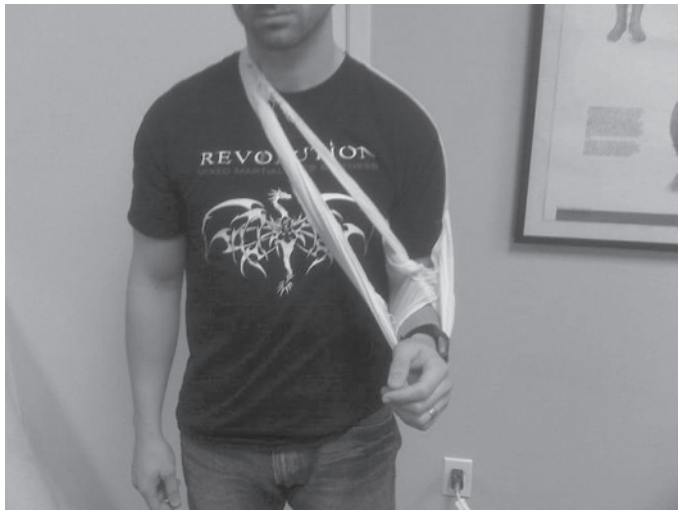

Figure 1 *Immobilization in external rotation with use of a sling. Front view.*

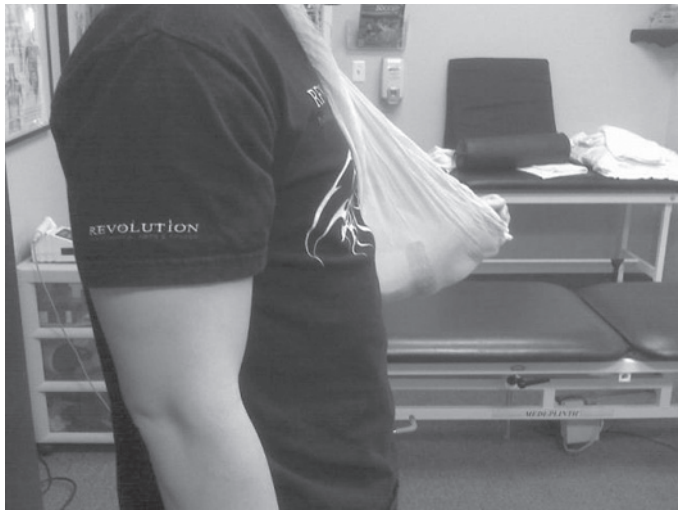

Figure 2 *Immobilization in external rotation with use of a sling. Side view.*

Week 1–2:
During the second week the patient continued his routine as indicated above. In addition, self assisted range of motion exercises (using the asymptomatic limb) were initiated as well as codman sets (several times daily).

Week 2–3:
During the third week, Active Release Techniques® soft

tissue treatment was applied to the anterior capsule of the glenohumeral joint as well as the rotator cuff musculature. The intent of this treatment was again to elicit the piezoelectric effect on the soft tissue structures and augment proper tissue healing.[12,13,14] Slow and controlled end-range mobilizations were also performed into external rotation (avoiding abduction) for the same purpose. Terminal range isometric exercises in several directions were performed by the patient while the treating practitioner provided resistance. The patient was asked to gradually build up tension as the motion was resisted with counter-pressure. The positions were to be held for as long as tolerated. At this time the patient was instructed to perform active range of motion exercises; however, the combined motions of abduction and external rotation were to be avoided.[16] Finally, 'wall alphabet' exercises were introduced with daily frequency for three sets to failure.

Week 3–5:
Treatment during this period continued as above with the addition of dynamic strengthening exercises. These exercises included a 61bs medicine ball chest pass on the rebounder, as well as pushups from a kneeling position. Each was performed every other day for three sets to failure. Progression to full pushups was possible during the 4th week. Proprioceptive training was also initiated at this time which included rhythmic stabilization drills (see figures 3 and 4), as well as upper body rocker board balancing.

Week 5–7:
As the patient continued to demonstrate strength gains, a gradual return to weight training was permitted. The patient performed several exercises as tolerated including bench press, reverse grip pull downs, dead-lifts, squats, lunges, and core strengthening (eg. bird-dog, side bridge, abdominal crunch). In addition the difficulty of proprioceptive training was progressed to upper body rocker board balancing with a Swiss ball placed under the pelvis.[17] At that time, isometric exercises were also being performed in an abducted, externally rotated position, as were slow and gradual passive mobilizations.

Week 7–8:
The patient was progressed to plyometric exercises to maximize strength gains and enhance proprioception.

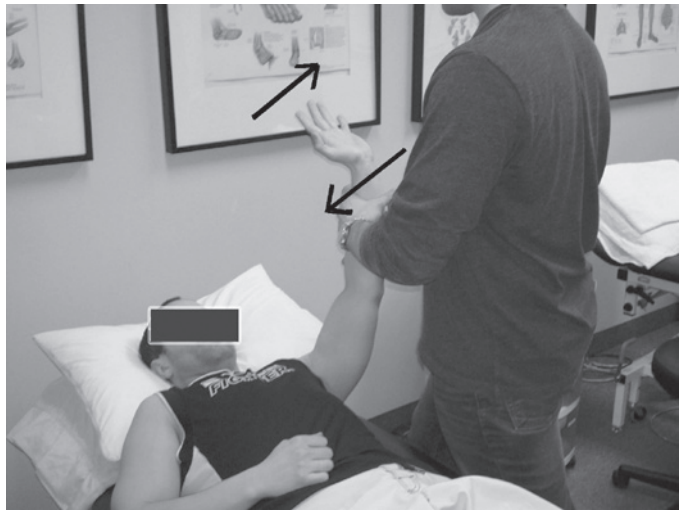

Figure 3 *Rhythmic stabilization drills. Patient attempts to resist clinician initiated perturbations. Medial to lateral.*

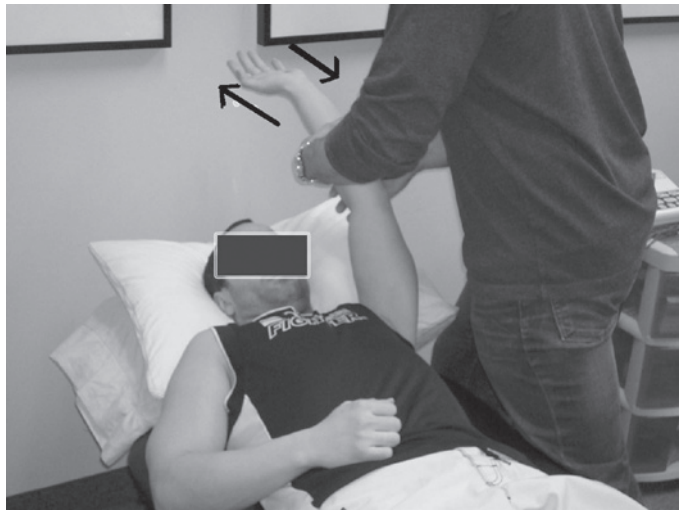

Figure 4 *Rhythmic stabilization drills. Patient attempts to resist clinician initiated perturbations. Superior to inferior.*

Plyometric training is purported to enhance proprioception through repetitive, maximal stimulation of mechanoreceptors as the shoulder is rotated to near end-range motion.[18] Plyometric exercises included 'stability push-ups' (see figures 5, 6 and 7), medicine ball throws on the rebounder with the arm in an externally rotated and ab-

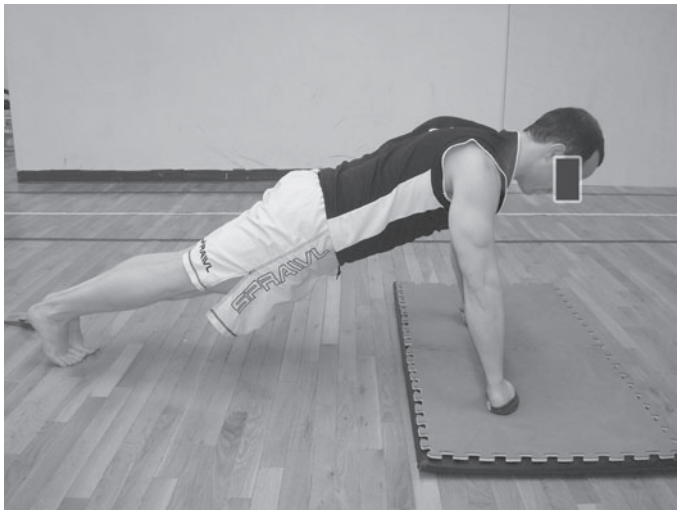

Figure 5 *'Stability pushups' Patient performs a plyometric pushup while simultaneously resisting horizontal abduction of the affected limb. Side view.*

Figure 7 *'Stability pushups' Patient performs a plyometric pushup while simultaneously resisting horizontal abduction of the affected limb. Mid exercise.*

Figure 6 *'Stability pushups' Patient performs a plyometric pushup while simultaneously resisting horizontal abduction of the affected limb. Front view.*

ducted position, and plyometric bench press on the Smith machine (see figures 8, 9 and 10).

Following this eight-week rehabilitation program, the patient was permitted to gradually return to pre-dislocation training. At this time he had regained full range of motion and strength compared to the unaffected limb and apprehension and relocation tests were no longer positive.

Discussion

When determining appropriate management of first-time, traumatic anterior shoulder dislocations, the clinician is faced with two options: operative or nonoperative management. Age at the time of initial dislocation is inversely related to the recurrence rate which has important implications for treatment recommendations. The literature suggests a primary surgical approach to first-time, traumatic anterior dislocations in younger individuals may be warranted.[19,20,21,22] When a comparison is made between operative and nonoperative management, there is a highly statistically significant decrease in frequency of recurrent dislocations for those managed surgically.[19] It is important to realize such conclusions are based on studies where the majority of subjects are young (less than 30), male, and tend to be highly active and/or involved in high risk or contact sports. Currently there is no evidence from randomized controlled trials (RCTs) to infer conclusions for patients that are female, older than 30, or inactive. It appears immediate operative stabilization in patients younger than thirty years may be a prudent approach for young individuals involved in competitive,

Figure 8 *Plyometric bench press on Smith machine. Start position.*

Figure 9 Plyo*metric bench press on Smith machine. Mid exercise – pushing bar into air.*

Figure 10 *Plyometric bench press on Smith machine. "Catching" the bar.*

contact, or high risk sports although current evidence is not overwhelming.

Unfortunately, the primary outcome measure used in most trials comparing operative and nonoperative management is recurrent dislocation. While functional outcome measures such as the Western Ontario Shoulder Stability Index (WOSI), range of motion, strength or return to sport are logically important measures of successful management, they are seldom used in clinical trials. Interestingly, in one of the few RCTs examining such measures, Kirkley et al (2005) were unable to demonstrate statistically significant difference on WOSI scores at five-year follow-up when comparing arthroscopic surgery and nonoperative management in subjects younger than thirty years.[21] Surgically treated individuals did not rate their shoulders significantly better than those conservatively managed.

Ultimately, the determining factor when contemplating whether to manage a case operatively or nonoperatively is the expectation that risks incurred with one type of management outweigh those incurred by the other. The risks associated with surgical repair for anterior dislocations include infection, nerve damage, decreased range of motion, and pain.[10] The major risks associated with managing a shoulder conservatively is recurrent instability and the additional glenohumeral damage sustained by subsequent dislocations.[23] While risks associated with nonoperative management appear minimal, given the high rate of recurrence, a patient may conclude that the risk of subsequent episodes of instability is unacceptable and elect for early stabilization through surgery.

For patients aged greater than thirty, nonoperative management is suggested to be the initial course of care.[23] A reasonable approach to caring for these patients includes a discussion of any relevant research, an evaluation of current sport or work participation, and elucidation of the

patient's feelings on management. When warranted, nonoperative management generally consists of immobilization of the shoulder in a sling and a progressive, individualized rehabilitation program including strength and proprioceptive training. Since the literature indicated our patient was at a reduced risk for a recurrent dislocation, the case was managed with an intensive, nonoperative approach using the best available evidence.[9,10]

Immobilization

Immobilization of the shoulder post-dislocation has been performed for thousands of years. Since the time of Hippocrates, conservative management of these injuries has generally involved immobilization in an internally rotated and adducted position for varying time periods. The pathoanatomical justification for immobilization in such a position is to allow for healing/scarring of the torn capsule and anterior soft tissue structures which should ultimately limit a patient's range of motion.[24] Scarred, shortened soft tissues theoretically hinders a patient's ability to move their shoulder into the position most often associated with an anterior dislocation: 90 degrees abduction and full external rotation (ER). The evidence base for this type of management is uncertain at best. The majority of large RCTs and literature synthesises on the topic suggest no benefit (no reduction in recurrence rate) to immobilization in a position of glenohumeral internal rotation (IR).[8,10,24,25,26]

Due to the high rate of Bankart lesions associated with anterior dislocations, authors have recently queried whether immobilization of the shoulder in ER, rather than IR, has the potential to reduce recurrence rates after initial dislocation.[3,27,28,29,30] The pathoanatomical justification for immobilization in ER is to tension the subscapularis and anterior soft tissue structures, thereby approximating the Bankart lesion to the glenoid rim. Better coaptation of the capsulolabral tear to the glenoid rim would augment the resistance imparted by IGHL and restore the chock block effect of the labrum, greatly enhancing static stability anteriorly.[31] There is preliminary evidence indicating a reduction in recurrent dislocations when immobilized in a position of ER.[3,28]

Miller et al. (2005) measured contact pressures between the labrum and glenoid as the shoulders of ten cadavers were rotated in an arc from 60 degrees IR to 45 degrees ER. No detectable contact force was measured in internally rotated shoulders, however, the contact force increased significantly as the shoulder moved through neutral to ER and reached maximum contact at 45 degrees ER. Itoi et al. (2001) and Seybold el al. (2008) used Magnetic Resonance Imaging to determine the position of the torn capsulolabral complex in vivo in individuals with acute anterior shoulder dislocations. Axial slices of recently dislocated shoulders revealed shoulder IR resulted in a displaced labrum from the glenoid while ER (at least 10 degrees) approximated the torn labrum to the glenoid.[27,30] Seybold et al. (2008) suggested the benefit to immobilization in ER was in part due to the effect the position had on edema in the joint capsule. In a position of IR, edema remained anterior, distending the shoulder capsule. However, in a position of ER the anterior soft tissue structures became taught, shifting edema posterior in the capsule, thereby permitting coaptation of the Bankart lesion to the glenoid.[30]

A recent randomized trial by Itoi et al. demonstrated immobilization in ER rather than IR may translate into improved clinical results. At two year follow-up, intention-to-treat analysis demonstrated a relative risk reduction of 38.2% for those immobilized in ER compared with the risk associated with the conventional method of immobilization in IR.[3] The study's results suggest that if seven patients are managed with immobilization in ER rather than IR, one recurrent dislocation should be prevented. While the results appear encouraging, this study is not without its limitations. The authors do report a conflict of interest, return to sport numbers were quite low, and clinically important outcome measures such as strength testing or range of motion measurements were not included.

Managing acute, traumatic anterior shoulder dislocations with conventional immobilization in IR is not supported by the available evidence. Due to preliminary evidence suggesting immobilization in ER (at least 10 degrees), as opposed to IR, may be an effective option for reducing recurrent dislocation rates a clinical decision was made to immobilize the patient in this case in this position. The notion does however require further evaluation before adopting immobilization in ER as the standard of care.

Rehabilitation

The shoulder is an inherently unstable joint sacrificing stability for mobility. Stability of the shoulder is provided by its static and dynamic stabilizers. The glenoid fos-

sa, labrum, and joint capsule/glenohumeral ligaments are the static stabilizers of the shoulder. Muscles that attach in and around the glenohumeral joint and the scapulothoracic joint are considered the dynamic shoulder stabilizers. These muscles, which include the rotator cuff, deltoid, long head of the biceps and scapulothoracic muscles, provide stability by enhancing joint compression, resisting humeral translation, and maintaining optimal contact between the glenoid and humeral head.[32]

The principle objective of rehabilitation is to return the patient to a pain-free condition with a high level of functionality as quickly and safely as possible. Rehabilitation of the shoulder after dislocation aims at enhancing the dynamic muscular restraints of shoulder stability. The evidence base pertaining to rehabilitative exercises in the management of anterior shoulder dislocations is quite poor. A recent Cochrane review was unable to locate RCTs for any aspects of conservative management of anterior shoulder dislocations including rehabilitation.[33] The literature discussing rehabilitation protocols is primarily expert opinion, therefore, there is a lack of solid evidence from which conclusions can be drawn.[34]

The shoulder girdle is a complicated system formed by multiple articulations with an intricate network of muscles, tendons, ligaments and other connective tissues.[35] It is important clinicians be able to apply a knowledge of shoulder anatomy and kinematics during rehabilitation to protect structures that have potentially been injured post dislocation. Safe and effective rehabilitation of the shoulder therefore requires an understanding of the anatomical and biomechanical characteristics of the shoulder complex. For example, a common recommendation immediately after an anterior dislocation is to avoid the combined ranges of motion of external rotation and abduction.[16,36] The purpose of this recommendation is to evade positions with the potential of driving the humeral head anteriorly placing strain on injured tissues and potentially delaying healing. The patient in this case was prohibited from combining these two ranges of motion for the first five weeks of the rehabilitation program.

Obtaining a stable scapular base is another important concept to consider when rehabilitating the shoulder. The scapula is intimately associated with the position of the humeral head during movement and helps maintain balance between the glenohumeral joint and scapulothoracic joint.[37] Exercises focused on the scapular pivoters (primarily the serratus anterior and trapezius) can be started early in the rehabilitation program and were begun immediately upon presentation in this case. Manual resistance of scapular motion, rowing, or push-up pluses are all reasonable exercises to improve scapular stabilization. It is also important to perform exercises in the scapular plane during rehabilitation. This ensures optimal length-tension relationships for the function of the rotator cuff and deltoid musculature and avoids straining soft tissues recently damaged during the dislocation.[35,38] Movement in the scapular plane is also associated with a high degree of congruence between the glenoid and the humeral head, which enhances stability and protects the shoulder.[16]

Restoring normal function of the rotator cuff musculature is also an important goal in the rehabilitation of anterior shoulder dislocations. Re-establishing muscular strength and endurance of these muscles results in reduced strain on the static stabilizers and enhances joint function and stability. There is evidence to suggest altered electromyographic activity of the supraspinatus muscle in individuals with anterior instability when compared to healthy controls.[39] Altered function of the supraspinatus results in superior migration of the humerus, which has been shown to increase susceptibility to dislocation.[40] The infraspinatus and teres minor control external rotation of the humerus and reduce anterioinferior capsuloligamentous strain.[41] The subscapularis muscle should be a focus in any rehabilitation program as it has been shown to be a main muscular stabilizer in the abducted and externally rotated position (the position in which most dislocations occur).[40] Rehabilitation of the rotator cuff in this case initially began as isometric exercises through manual resistance provided by the practitioner then progressed to dynamic strengthening exercises and eventually plyometric exercise.

A rehabilitation program post-dislocation should be individually tailored to meet a patient's needs. While the evidence discussing rehabilitation is limited, expert opinion suggests a program should promote safe attainment of range of motion, while strengthening the dynamic stabilizers of the shoulder girdle.[16,34]

Proprioception

Proprioception is an afferent neural input from the periphery which conveys information regarding joint movement, joint position, and force applied to or present within a

joint.[42,43] Essentially, during shoulder movements mechanoreceptors within surrounding tissues (joint capsule, ligaments, and muscles) are stimulated through length and tension changes in the tissues. Proprioceptive information from these mechanoreceptors is integrated with other afferent information (vision, vestibular input, etc.) in the motor cortex resulting in either muscle activation or inhibition. The integrated information enables coordinated motor patterns and enhances reflex activity and joint stiffness, all of which influence shoulder joint stability.[44] Deficits in proprioceptive capabilities have been demonstrated in individuals who have previously dislocated their shoulders.[42,43]

Why do proprioceptive deficits develop after an anterior shoulder dislocation? Tissue lengthening associated with shoulder dislocation is most likely the principal cause. Decreased proprioceptive capabilities in patients with known history of shoulder dislocation have been shown to improve significantly after surgical repair to restore capsular tension when compared to healthy controls.[45,46] Tibone et al (1996) demonstrated no significant differences between normal subjects and subjects with clinical anterior instability using somatosensory cortical evoked potentials.[47] Given that joint mechanoreceptors were stimulated with electrical rather than mechanical stimulation and subjects demonstrated differing mechanical capsuloligamentous properties, the results imply proprioceptive input pathways remain intact in those with instability. Simply put, mechanoreceptors are not damaged or non-functioning rather they are not being adequately stimulated due to tissue lengthening post-trauma. Interestingly, Tibone et al (1996) also found the latency response time for mechanoreceptors within the shoulders of this population to be approximately 3 msec.[47] A short latency time may indicate that proprioceptive exercise is suitable for protecting the shoulder from subsequent injury as reflexive shoulder muscle contractions could be fast enough to adequately protect the shoulder. Tissue damage that occurs with an anterior shoulder dislocation not only affects mechanical restraint by stretching and tearing capsuloligamentous tissues, it also alters proprioceptive input into the central nervous system, potentially predisposing individuals to future episodes of instability.

Given the important role that proprioception plays in shoulder stability and function, it is important for clinicians to become aware of methods currently available to restore these mechanisms altered by injury. Perturbations, closed kinetic chain, and plyometric exercises are a few of the techniques described in the literature.

Naughton et al. (2004) was able to improve proprioception in fifteen subjects with previous anterior dislocations using an upper-body wobbleboard training regimen. Subjects with a Swiss ball placed under their pelvis stabilized themselves with active movements at their shoulders while balancing on a wobbleboard. After one month of training (ten minutes a day, five to six days a week), subjects not only significantly improved discrimination of shoulder movements when compared to healthy controls but also perceived their shoulders to be stronger and more stable.[17] During week 3–5 in this case the patient began proprioceptive exercises including rhythmic stabilization drills and upper body rocker board balancing.

Swanik et al. (2002) was able to demonstrate significant improvement in proprioception in subjects trained with plyometric exercise. The internal rotators of twelve female division I swimmers were trained with elastic tubing (progressing to the Pitchback System) two days a week for six consecutive weeks. Subjects in the plyometric group improved significantly in five of six proprioceptive tests when compared to controls.[18] It was surmised that plyometric training enhanced proprioception through repetitive, maximal stimulation of mechanoreceptors as the shoulder was rotated to near end-range of motion during the exercises. Plyometric drills were included near the end of the rehabilitation of this case. Medicine ball throws while on the rebounder and plyometric bench press on the Smith machine were instituted in week 7–8 to maximize strength of the shoulder stabilizers and enhance proprioception.

Overall, investigation into the literature correlates proprioceptive deficits and anterior shoulder instability. Anterior dislocation likely results in mechanical laxity that ultimately alters motor control of the dynamic stabilizers, predisposing individuals to further shoulder injury. Proprioception can be improved through perturbations, closed kinetic chain and plyometric exercises in individuals exhibiting deficits. Unfortunately, no prospective studies currently exist from which conclusions can be drawn as to the effects such training has on recurrent dislocation rates. Ultimately, the literature remains unclear whether improving proprioception at the shoulder joint improves overall prognosis.

Conclusion

Anterior dislocation is a common condition encountered by health care practitioners. Age at the time of initial dislocation is inversely related to the recurrence rate and has important implications for treatment recommendations. Except in the younger athlete, where a primary surgical approach to first-time acute anterior dislocations may be warranted, conservative management should be the initial course of treatment.[9,10,21,23] Conservative management of anterior shoulder dislocations should consist of immobilization of the shoulder and a rehabilitation program including strength and proprioceptive training. Preliminary evidence suggests immobilization in ER may be an effective option for reducing recurrent dislocation rates.[3,27,28,29,30] Individualized, progressive rehabilitation programs aimed at restoring normal range of motion and strength of the dynamic stabilizers of the shoulder are recommended in the literature.[16,34] Research suggests proprioception can be improved through perturbations, closed kinetic chain, and plyometric exercises in individuals exhibiting deficits.[17,18]

To date, despite the high incidence of anterior shoulder dislocations the evidence base regarding conservative management of these injuries remains quite poor. This case demonstrates potential management of a first-time anterior shoulder dislocation using immobilization in ER, combined with an intensive rehabilitation program. Prior to implementing the type of nonoperative management discussed in this case as treatment protocol, further investigation in larger, controlled trials is required.

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Nine year longitudinal retrospective study of Taekwondo injuries

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This retrospective longitudinal study aims to describe reported Taekwondo injuries and to examine associations between competitor experience level, age and gender, and the type, location, and mechanism of injury sustained. Additionally, we examined whether recent rule changes concerning increased point value of head shots in adult Taekwondo competition had affected injury incidence.

This study was a summation of 9 years of data of competition injury reports, which included 904 injury reports spanning 58 individual competitions. The data was collected on standardized injury reports at time of injury during competition. Care was provided to the athletes, but the type of care provided was not included in the study. Participants included athletes injured during competition who sought care by the health care team, and for whom an injury report was filled out. The data analysis was performed at the Canadian Memorial Chiropractic College.

The three most common locations of presenting injury were the head (19%), foot (16%), and thigh (9%). The most common mechanism of presenting injury was found to be a defensive kick (44%), followed by an offensive kick (35%). The most commonly diagnosed injuries were contusions (36%), sprains (19%), and strains (15%). Coloured belts had a higher incidence of contusions, while black belts sustained more joint irritation injuries. Black belts were more likely to suffer multiple injuries. Colored belts suffered more injuries while receiving a kick, while black belts had a larger influence of past

Cette étude rétrospective longitudinale a pour but de décrire les blessures déclarées de taekwondo et d'examiner les associations entre le niveau d'expérience des compétiteurs, leur âge et leur sexe, ainsi que le type, l'endroit et le mécanisme de la blessure subie. De plus, nous avons examiné si des changements récents aux règlements pour augmenter la valeur en points des coups à la tête lors d'une compétition de taekwondo entre adultes ont eu des répercussions sur la fréquence des blessures.

Cette étude était un résumé de 9 ans de données provenant de rapports sur les blessures subies lors de compétitions, qui comprenaient 904 rapports de blessures pour 58 compétitions individuelles. Les données ont été recueillies lors de rapports de blessures normalisés tout de suite après la blessure lors de la compétition. Des soins ont été prodigués aux athlètes, mais le type de soins fourni n'a pas été inclus dans l'étude. Les participants comprenaient des athlètes blessés lors d'une compétition qui ont reçu des soins de la part de l'équipe soignante et pour qui on a rempli un rapport de blessure. L'analyse des données a été réalisée au Canadian Memorial Chiropractic College.

Les trois endroits les plus communs de blessure étaient la tête (19 %), le pied (16 %) et la cuisse (9 %). Le mécanisme le plus commun ayant provoqué la blessure était un coup de pied défensif (44 %), suivi par un coup de pied offensif (35 %). Les blessures les plus souvent diagnostiquées étaient les contusions (36 %), les entorses (19 %) et les foulures (15 %). Les athlètes avec ceinture

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history of injury. We found no significant difference in location or type of injury when comparing pre versus post rule change. The most common locations of injury are head, foot, and thigh respectively, and are areas for concern when considering preventative measures. Colour belt competitors are more likely to sustain contusions, which the authors believe is due to more aggressive tactics and lack of control. Those more likely to be injured tend to be younger than 18 years. Recent rule changes have no significant effect on head injuries.
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KEY WORDS: Taekwondo, injury, athlete, competition

de couleur présentaient une plus grande fréquence de contusions, alors que les athlètes avec ceinture noire présentaient plus d'irritation des articulations. Les athlètes avec ceinture noire étaient plus enclins à subir des blessures multiples. Les athlètes avec ceinture colorée souffraient de plus de blessures en recevant un coup de pied, alors que les athlètes avec ceinture noire avaient une plus grande influence de leurs antécédents de blessures. Nous n'avons pas découvert de différence importante vis-à-vis de l'endroit ou du type de blessure lors de comparaisons entre avant le changement aux règlements et après. Les endroits les plus communs pour les blessures sont la tête, le pied et la cuisse respectivement et sont à considérer lors de l'établissement de mesures préventives. Les compétiteurs avec ceinture colorée ont de plus fortes chances de subir des contusions; les auteurs croient que cela s'explique par des tactiques plus agressives et un manque de contrôle. Les personnes le plus à risque d'être blessées sont celles de moins de 18 ans. Les récents changements aux règlements n'ont pas d'effets importants sur les blessures à la tête.
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MOTS CLÉS : taekwondo, blessure, athlète, compétition

Introduction

Taekwondo is a Korean martial art practiced in over 184 countries.[1] The sport gained full medal status at the 2000 Olympics in Sydney, Australia,[1] and draws participants of all ages due to its perceived fitness benefits and popularity. A beginner starts at a rank of 'white belt' and when he or she has achieved sufficient proficiency of Taekwondo skills, they move through several higher ranks, respectively the colored belts (In increasing rank: Yellow, Orange, Green, Purple, Blue, Brown, Red), and black belts (with increasing rank based on Dan level, from 1st to 9th). To qualify for provincial tournament the athlete has to hold a first degree black belt from the WTF and has to weigh in the day before the tournament within the weight category they had applied to. Local tournaments have no such stipulations, though athletes are segregated by gender, age, and weight. Points in competition are awarded for full contact kicks to the body and head. A match consists of three rounds of two minutes each, and requires cardiovascular and muscular endurance. Due to the physical demands imposed on the athlete and the forces involved in full contact sparring, injury risk must be addressed.

It is plausible to reason that more training should correlate with lower injury incidence, and this is supported by research indicating a significant inverse relationship between children's Taekwondo rank and their aggression.[2] Martial arts athletes practice between two to four times per week and all must demonstrate a level of proficiency to advance in rank.[3] Thus, improvement of technique application in a competition setting should decrease the athlete's risk for injury during a match. However, as skill level increases, so do physical demands during combat, as well as force generated. More skilled athletes are likely to use dangerous techniques or execute fundamental ones with greater strength and speed. It is

therefore also possible that injury incidence may be higher in elite athletes. Interestingly, a recent study of karate injuries found that injury incidence increased with competitor rank, and with number of years practicing.[4]

Differences in injury rates between young and adult Taekwondo athletes have been studied in the past, and Beis et al 2001 found that young competitors, male or female, were more likely to sustain injury during competition than their adult counterparts.[5] It is likely that young Taekwondo athletes require extra safety precautions, for example, greater referee vigilance or extra protective equipment.

Previous studies have shown gender differences in Taekwondo with respect to type and mechanism of injury and injury rates. A recent study found greater rates of injury in male black belt competitors, and reported concussion injuries in male competitors only.[6] Previous studies have also noted increased force generation and aggression in male competitors.[7]

Although recent studies have found that the most common Taekwondo injury is that of the lower limb,[6] much attention is given to head injury, most likely owing to the severity of such trauma. This is interesting, as the frequency of head blows and concussions is reported to be high in Taekwondo.[8] Kicks are allowed to the head region, excluding the posterior aspect. The incidence of cerebral concussions in Taekwondo was reported by Zemper and Pieter in 1994 to be more than 2 times greater than in college football games, based on number of exposures.[9] In 2003, rule changes introduced an increase in point value of successful head contacts in adult competition to 2-points, compared to a standard 1-point value for kicks to the torso, plus an additional point for an eight-count knockdown.[1] This likely adds incentive to score more points via head strikes and thereby increases the risk of head injury.

The most common diagnoses reported in the literature for Taekwondo athletes are contusions and lacerations, followed by sprains and strains, knee lesions, broken limbs, and broken noses.[10,11] A recent study ranked the top 5 injuries as: sprain, joint dysfunction, contusion, laceration and strain, with concussion a close 6th.[6] The most common mechanism of injury in Taekwondo is receiving a kick, followed by delivering a kick.[10,11]

Past research has focused on injury incidence,[5,6,9,12,13] and the physical characteristics and training habits that predispose athletes to injury.[2,14,15,16] This study attempts to discern the injury incidence in Canadian provincial level Taekwondo competition and to describe the effect of recent rule changes and athlete experience on the types, locations, and mechanisms of injury.

Method

The sample population consisted of male and female Taekwondo athletes of all experience and age levels who competed in Canadian provincial and local interclub tournaments over the last 9 years, suffered an injury and sought care from the health care team at time of competition. For the purposes of this study, an athlete was considered injured if any of the following conditions applied: 1) any circumstance that forced the Taekwondo athlete to leave the competition; 2) any circumstance for which the referee or athlete had to stop competition; 3) any circumstance for which the athlete requested medical attention.[17]

Injuries were recorded on an injury form[6] describing the athlete as well as the nature, site, severity, and mechanism of injury. The injury reports were used for medical record-keeping purposes, and were later used as part of this study. The data was not initially collected for research purposes. Study data included 904 injury reports spanning 58 competitions. The data set consisted of 664 competitors, some suffering single and some suffering multiple injuries.

The study data was gathered retrospectively via injury reports over a 9 year period. The injury forms[6] were used to provide care for the athletes, and documentation of injuries was mandatory as per Ontario law. Oral or written informed consent for treatment was obtained. With an underage athlete, consent for treatment was obtained through the parents or guardian. The first author (the health care provider for these athletes) was the only person who had possession of the injury forms, keeping athlete identities confidential. Canadian Memorial Chiropractic College (CMCC) was the location of the study. The study passed an REB approval at CMCC (May 14, 07, certificate# = 0705X03).

The software program "R-project" (University of Auckland) was used to analyze the gathered data. Descriptive statistics were used to describe the sample in terms of demographic factors: population number, number of males and females, age range and mean age, and experience level expressed as belt rank. Means were

used to describe the outcome measures listed above (number of reported injuries, injury incidence, body part injured, injury type, and injury mechanism). The experience levels were compared by injury incidence, type, and mechanism using contingency tables and the Chi-square test for independence with Yate's continuity correction. The difference between the number of injuries and experience level was examined using a Chi-square test with Yate's continuity correction. The difference between pre and post rule change injury incidence was correlated using a Chi-square test with Yate's continuity correction. The authors used 0.05 as the standard for statistical significance.

Only the primary author had access to injury forms and identities. Photocopies were produced of injury reports with identities deleted, replaced with a unique number for data entry.

Results

The data included 532 single injury reports; 363 male, 142 female, and 27 without recorded gender. The data also included 367 repeat injury reports from 132 individual competitors; 84 male, 44 female, and 4 without recorded gender. The average number of injuries for athletes with repeat injuries was 2.78.

The age range for injured male competitors was 6 to 58 years old (mean = 18.9 years), with a median age of 16. The age range for females was 4 to 47 years old (mean = 17.1 years), with a median of 15. 16 reports lacked age information and were excluded, leaving 888 injuries. 510 injuries were competitors under age 18 and 378 injuries were competitors over age 18. In both age categories, males sustained the majority of injuries. In the under 18 category, there were 333 males and 177 females injured. In the over 18 category, there were 272 males and 106 females injured.

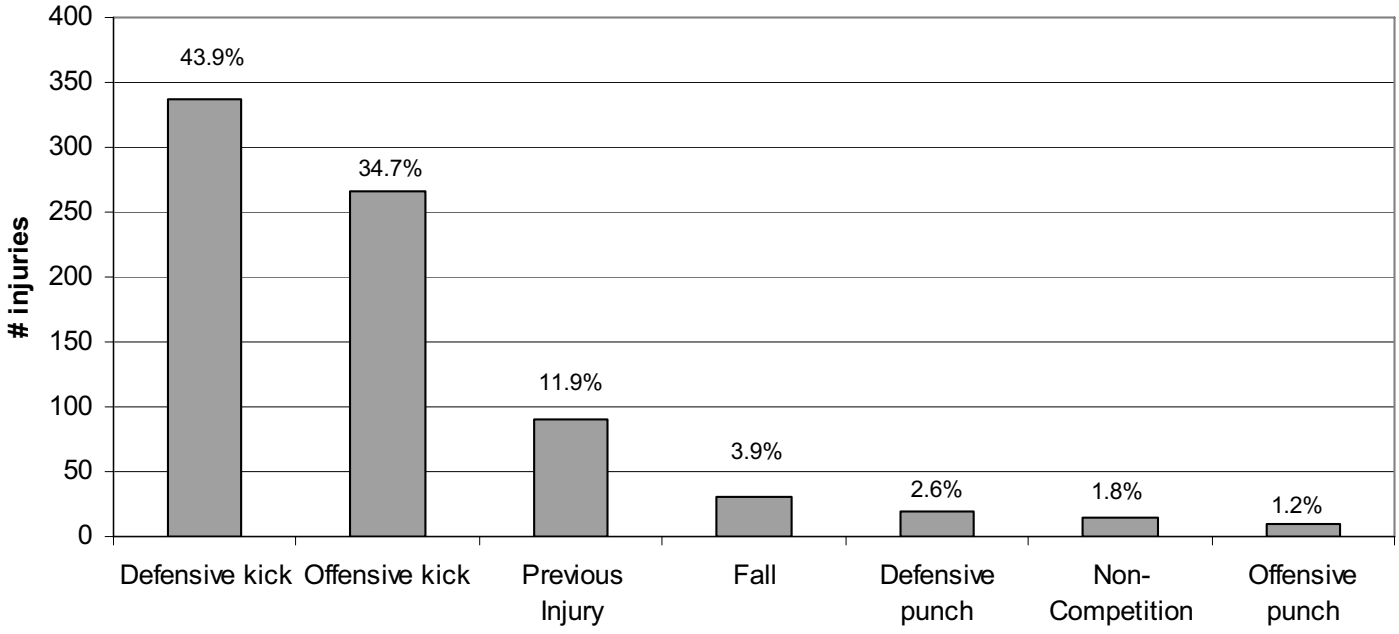
Of the 664 competitors, there were 447 males, 186 females, and 31 injury reports that lacked gender information and were excluded. Of the injured competitors, males sustained an equal proportion of total injuries as females, as well as head injuries (90) than females (33), and injuries received due to a kick, with 231 males and 97 females sustaining kick related injuries. Females were as likely to sustain injury from a defensive kick (n = 57) as from an offensive kick (n = 40), while males were almost twice as likely to sustain injury while receiving a kick (n = 147) than when delivering one (n = 84). The top three diagnoses for males were, in order: contusion (n = 125), sprain (n = 61), and strain (n = 45). Similar findings were discovered for females (contusions = 46, sprain = 23, strain = 22). An analysis on the effects of gender on location of injury yielded non-significant results ($X^2 = 3.97$, df = 3, p = 0.14). A similar finding was observed for gender and diagnosis of injury ($X^2 = 8.52$, df = 9, p = 0.48), as well as mechanism ($X^2 = 8.33$, df = 6, p = 0.21), and number of injuries sustained ($X^2 = 2.23$, df = 1, p = 0.14).

The mechanism of injury was not filled in for 137 of the injury forms and thus the analysis of mechanism of injury included 767 injuries (84.85% of total reports). The most common mechanism of injury was the defensive kick (337, 43.94%), followed by the offensive kick (266, 34.68%), and fall (30, 3.91%). However, 91 injuries (or 11.86%) may have been attributed to a previous injury. Full data with regard to injury mechanism is summarized in Figure 1. The defensive kick and punch categories included injuries sustained in the process of receiving a kick or a punch respectively. The offensive kick and punch categories included injuries sustained during the delivery of a kick or a punch respectively. A fall included injuries sustained during a fall, as long as injury was not sustained prior to the fall itself. For example, a competitor receiving a kick to the head, who then fell and sustained an ankle injury, would only have the ankle injury counted as a fall related injury. A non-significant trend of competitors aged over 35 sustaining an injury while receiving a punch was observed.

The location of injury was not reported in 13 of the injury reports, and thus the analysis of location of injury was based on 891 injuries (98.56% of total reports). The most common injured body region was the head (169 injuries, 18.86%), followed by foot (144 injuries, 16.16%), thigh (84 injuries, 9.43%), knee (75 injuries, 8.42%), ankle (71 injuries, 7.97%), back (68 injuries, 7.63%), and leg (55 injuries, 6.17%). Due to their proximity and functional relationship, foot and ankle injuries were pooled into a single category (215 injuries, 24.13%), making the foot and ankle the most likely area of injury. The categories were then pooled into functional categories of Upper Limb, Lower Limb, Head and Neck, and Torso. Full data is summarized in Table 2 and Table 3.

The injury diagnosis was missing in 24 of the reports and thus the analysis of injury diagnosis is based on 880

Figure 1 *Mechanism of injury*



injuries (97.35% of total reports). The most common diagnosed injury was contusion (319, 36.25%), followed by sprain (166, 18.86%), and strain (129, 14.66%). Table 4 summarizes the occurrence data.

For the purpose of correlation of experience and injury mechanism, diagnosis, and location, number of injuries, as well as pre and post rule change injury location, repeat injury reports were not used in the calculations, as the independence of a repeat injury could not be established. We could not be sure if a repeat injury was the result of a competitor's level of experience or simply due to the presence of recent injury itself. Thus the data set was pared down to a set of 659 injury reports, including the first injury suffered by a unique competitor. Black belt Dan level competitors were pooled into a single Black Belt category, as the Dan categories themselves were small. Of the 659 reports, 160 lacked identification of competitor experience level and were excluded from calculations pertaining to experience, leaving a pool of 499 injury reports (55.20% of total reports).

The location of injury was independent of the competitor's level of experience ($X^2 = 3.6615$, df = 3, p = 0.30).

Of the 499 injury reports included in the analysis, 63 did not include a mechanism of injury and were excluded for the purpose of experience versus mechanism calculations, and thus the following were performed on 436 injury reports (48.23% of total reports). The mechanism of injury was dependent of the competitor's experience level ($X^2 = 16.5053$, df = 6, p = 0.01128). A 2-sample test for equality of proportions with continuity correction was performed and it was observed that the proportion of injuries suffered during a defensive kick to all injuries was significantly higher for colored belt athletes (0.54) versus black belt athletes (0.40) ($X^2 = 6.5511$, df = 1, p = 0.01). The proportion of a history of injury versus all other types of injury sustained was significantly higher for black belt athletes (0.12) versus colored belt athletes (0.04) ($X^2 = 7.0923$, df = 1, p = 0.008). Black belt athletes (0.38) showed a trend towards injuries suffered during an offensive kick as compared to colored belts (0.30) ($X^2 = 1.9428$, df = 1, p = 0.1634). Table 5 offers a summary of the mechanism of injury versus experience level data.

Injury diagnosis was dependent of the competitor's experience level ($X^2 = 34.7444$, df = 9, $p < 0.001$). Again, a

Table 2 *Location of injury*

Location	Frequency	Percent
Head	169	18.86
Foot	144	16.16
Thigh	84	9.43
Knee	75	8.42
Ankle	71	7.97
Back	68	7.63
Leg	55	6.17
Neck	39	4.38
Toes	39	4.38
Hand	26	2.92
Trunk	19	2.13
Hip	19	2.13
Finger	17	2.02
Wrist	17	1.91
Shoulder	14	1.57
Forearm	13	1.46
Arm	11	1.23
Elbow	11	1.23
Total	891	100

Table 3 *Percentage of injury by body region*

Body Region	Frequency	Percent
Lower Limb	487	54.7
Head and Neck	208	23.3
Upper Limb	109	12.2
Torso	87	9.8

2-sample test for equality of proportions with continuity correction was performed and it was observed that the proportion of contusions to all injuries was significantly higher for colored belt competitors (0.48) versus black belt competitors (0.29) ($X^2 = 16.2138$, df = 1, $p < 0.001$). It was also observed that the proportion of joint dysfunctions/irritations to all injuries was significantly higher in black belt competitors (0.11) versus colored belt competitors (0.01) ($X^2 = 12.3451$, df = 1, $p < 0.001$). Table 6 summarizes the injury diagnosis versus experience level data.

Table 4 *Percentage of injuries by diagnosis*

Diagnosis	Frequency	Percent
Contusion	319	36.25
Sprain	166	18.86
Strain	129	14.66
Fracture	69	7.84
Joint Dysfunction	64	7.27
Concussion	52	5.91
Other	41	4.66
Laceration	18	2.05
Dislocation	11	1.25
Epistaxis	11	1.25

The presence of single versus multiple injuries was dependent on the experience level of the competitor, as black belt competitors sustained significantly more multiple injuries ($X^2 = 22.3792$, df = 1, $p < 0.001$). Table 7 summarizes the number of injuries versus Experience level data.

For the purpose of examining the effect of recent point scoring rule changes, a pool of 659 injury reports were used for the examination of rule change influences on injury location. Location of injury was found to be independent of whether injury occurred prior to the 2003 rule change, or after it ($X^2 = 0.3299$, df = 3, $p = 0.9543$). A subsequent proportional analysis was performed and no significance was determined.

Discussion

The present study is the first to examine injury incidence across a longitudinal time span of competition events, as well as to examine the effect of experience level on injury type, location and mechanism. The analyzed data spans from 1997 to 2006 and includes over 50 individual competitions. Such quantity of data should offer a clearer picture of injury incidence in Taekwondo competition.

Table 5 *Mechanism of injury versus Level of Experience*

Mechanism of injury	Color belt	Percent(%)	Black belt	Percent(%)
Defensive kick (statistically significant, $X^2 = 6.5511$, df = 1, p = 0.01)	73	53.68	120	40.00
Offensive kick	42	30.88	115	38.33
Defensive punch	8	5.88	7	2.33
Offensive punch	2	1.47	4	1.33
Fall	5	3.68	13	4.33
non competition injury	1	0.74	4	1.33
previous injury history (statistically significant, $X^2 = 7.0923$, df = 1, p = 0.008)	5	3.68	37	12.33
total	136	100	300	100

Table 6 *Diagnosed injuries versus Level of Experience*

Diagnosis of injury	Color belt	Percent(%)	Black belt	Percent(%)
Concussion (statistically significant, $X^2 = 16.2138$, df = 1, p < 0.001)	7	4.55	31	8.99
laceration	5	3.25	3	0.87
contusion	74	48.05	100	28.99
sprain	27	17.53	71	20.58
strain	20	12.99	55	15.94
fracture	9	5.84	26	7.54
dislocation	0	0.00	4	1.16
joint irritation /dysfunction (statistically significant, $X^2 = 12.3451$, df = 1 p < 0.001)	2	1.30	38	11.01
nose bleed	2	1.30	6	1.74
other	8	5.19	11	3.19
total	154	100.00	345	100.00

Table 7 *Number of injuries versus Level of Experience (statistically significant, $X^2 = 22.3792$, df = 1, p < 0.001)*

Number of injuries	Color belt	Percent(%)	Black belt	Percent(%)
single	140	90.91	245	71.01
multiple	14	9.09	100	28.99
total	154	100	345	100

The majority of injuries occurred in competitors under age 18, comparable to a previous study[5] which found that younger competitors, male or female, were more likely to sustain injury during competition than their adult counterparts. No statistical significance was found with respect to age and diagnosis, mechanism, and location of injury. There was, however, a trend that competitors aged 35 and older were more likely to sustain an injury while receiving a punch, possibly due to a reduced confidence in ability to perform a kick.

The data showed an equal proportion of reported injuries in males as females, though it must be restated that the sample only represents athletes who presented to the medical team for care, and not a complete sample of all competitors involved in each tournament. This differs from previous study[6] which reports higher injury rates in male black belt competitors. The study[6] also found concussion injuries in male competitors only. In our study, the proportion of males with head injuries did not differ from that of the females. The demographics of Taekwondo appear to consist of three times more men than women,[6,9,18] and this was reflected in our data set. In our data set, gender does not appear to affect the risk of sustaining injury, nor does it affect the diagnosis, mechanism, or location of injury. A recent meta-analysis by Lystad et al reported similar findings.[19]

The three most common locations of injury were the head, foot, and thigh. However, when the hip, thigh, knee, leg, ankle, foot and toe categories were summed into a lower limb category, these injuries exceeded head injuries for the black belt category only. Past research[6,8,9,19] cites the most common injury locations as the lower limb followed by the head. This is not surprising due to the use of the lower limb as the primary striking weapon, and the head's role as the primary target. In agreement with past study [11] the most common mechanisms of injury in this study were defensive kicks, and offensive kicks respectively. According to the literature,[10,11] contusions and lacerations occur significantly more frequently than other types of injuries. In this study, however, the most commonly diagnosed injuries were contusions, followed by sprains, then strains. Lacerations were not found to be common. As the literature states, the rate of non-serious injury is much higher than that of serious or life threatening injuries.[10,11,19] Thus, though the rates of injury have been reported to be high in Taekwondo, owing to its competitive nature, current rules and regulations adequately protect the athlete from serious injury.

The recent rule change increased the value of head shots in black belt competition, and was expected to cause an increase in head injuries. Contrary to this, no significant difference was found with regard to injury location or type when comparing injuries *pre-* versus *post-*rule change. It appears the 2003 rule change does not put competitors at increased risk of potentially serious head injury.

Prior to this study, the relationship between experience level and injury was not thoroughly examined. One could surmise that a novice is more prone to injury than a master due to the skill gap between the two. The novice athlete may compensate by increasing their aggression. Indeed, Skelton et al[2] found an inverse relationship between aggression and Taekwondo rank. Conversely, as skill level of a competitor increases, so does the physical load imposed on their body during the delivery and reception of blows. It is also plausible that an experienced fighter has been exposed to more dangerous techniques, and is comfortable using them during competition. Higher competitor rank has been positively correlated with higher injury incidence in karate competition.[4] Based on this, one could just as easily presume that a more skilled competitor is more likely to sustain an injury than would a novice. Our data revealed that the black belt competitors were more likely to sustain multiple injuries than their colored belt counterparts. Whereas, the colored belts were more likely to sustain single injuries compared to the black belts in our study. Lystad et al reported that level of play did not influence injury incidence.[19]

With respect to experience and location of injury, lower limb and head injuries were the most common in all groups, with no significant differences between experience groups. This is to be expected, as the head is a vulnerable location and black belt competitors seeking competition victory would choose it as a target. However, this was a surprising finding among colored belt competitors since no contact or only light contact were allowed for colored belt competition. The colored belts' lack of experience and control may have been contributing to this higher than expected rate of head injury.

When experience and injury mechanism were examined it was noted that colored belt competitors appear to suffer injury most likely while receiving a kick, possibly

due to poor defensive techniques. This may serve as a reminder to novice athletes to focus more on their defensive capabilities than their striking. Black belt competitors appeared to have a significant influence on injury due to a past history of injury to the same region. It is to be expected that injuries occur over an individual's Taekwondo career, and may influence not only match outcome but injury prevalence. Experienced competitors would be wise to learn from past experience and make sure not to overly stress areas of previous injury. Black belt competitors also trended to suffer injury while delivering a kick. Future studies should address where the elite level athlete is at greatest risk for injury which would affect their performance. In addition, further studies are recommended investigating effective injury prevention strategies. As Taekwondo emphasizes the use of the lower limb in combat, it is not surprising that all groups chose the kick as their primary defensive and offensive weapon.

When experience level and injury type were examined, all experience levels suffered similar injuries, with the top three injuries being contusion, sprain, and strain. However, it was found that colored belt competitors had significantly more contusions than black belt competitors. We speculate that this may be due to less experienced competitors attempting more aggressive, less controlled strikes, causing injury to both themselves and their opponents. The findings appear to support the hypothesis that less experienced competitors may attempt to compensate with more aggressive tactics. This also supports the WTF recommendation which requires that elite level tournament competitors should hold a minimum rank of black belt.[1] This recommendation appears to offer a measure of inherent injury prevention. Future studies should make an effort to examine if the difference noted in this study exists between each colored belt level. An additional variable to be considered is the amount of time an individual has spent in full time training, as various schools, and individuals have differing rates of rank progression. Black belt competitors appear to have suffered more joint dysfunction/irritation due to competition. Haldeman[20] defines joint dysfunction quoting Drum (1973) as, "Joint mechanics showing area disturbances of function without structural change; subtle joint dysfunctions affecting quality and range of joint motion. They are diagnosed with the aid of motion palpation, as well as stress and motion radiography investigation" [p. 623]. Greenman[21] states: "Joint dysfunction is characterized by findings of misalignment, relative fixation, loss of normal range-of-motion and end-play, tenderness, and tissue texture abnormality" [p. 13–14]¸ Kazemi and Pieter[6] also reported joint dysfunction to be the second most common injury sustained by male athletes (13.7/1,000 A-E).

The current data collection system underestimates repetitive stress injuries and their effects. Sub-categorization of the previous injury category would help to flush out repetitive injuries. Record keeping for regional, provincial and national level competition must be improved so that detailed statistics of injury incidence may be tracked over a long term basis. A viable option is to adopt a standardised classification system to be utilized by all martial arts medical teams, allowing for cross- and inter-sport comparisons. The Orchard Sports Injury Classification System would in part serve as a starting point,[22] but would not serve as a complete system for martial arts. Other researchers have also noticed a need for a standardized classification system for injury reporting in Taekwondo.[19,22] We believe martial art injury documentation should at least include information about the mechanism of injury. A limitation of this study was the need to remove a large number of injury reports due to missing data. Thus, some calculations were more reliable than others, as they reflected a larger percentage of the full sample of reports. This is partly a result of some competitors not providing all the necessary information and health team members not completely filling out the report. This was compounded by the presence of injured competitors who felt their injury was not severe and thus decided to terminate their visit partway through the examination. These records were not destroyed as part of record keeping protocol. A standardized protocol for injury report completion should be developed to prevent future missing data. The refinement of the standardized injury report form published by Kazemi and Pieter,[6] and used by the health teams, would also serve to benefit not only the researcher, but the injured athletes as well. New injury reports should also include such information as whether the athlete completed the match, if a stoppage was called, and in what round, as well as an injury severity ranking. Such measures will serve to further research in Taekwondo injury and to develop strategies to avoid injury and reduce time loss.

Conclusion

The results of this study show that the three most common locations of reported injury in this study were the head, foot, and thigh, respectively; these remain as areas of concern for injury prevention. Recent rule changes do not appear to increase reported head injury incidence, and thus cause no increased risk of significant injury to competitors in this cohort. Colour belt competitors are more likely to sustain injuries, particularly in the form of contusions, possibly due to more aggressive tactics and lack of control. Younger athletes (under 18) sustained more injuries than older athletes, presumably for the same reason. The vast majority of injuries were suffered by all competitors due to either receiving a kick or delivering one. This is expected in a sport focused on the use of the lower limb as a primary weapon. The data in this study spanned competitors of all ages and experience levels and thus we believe it represents the average Taekwondo participant and reflects the true risk of participation in the sport. Future studies of such injury statistics should help elucidate the athletes and body regions at highest risk of injury, and steps may be taken to prevent such injury in these individuals.

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Conservative management of symptomatic Carpal Bossing in an elite hockey player: a case report

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Objective: *To present the characteristics and create awareness of symptomatic carpal bossing and discuss potential etiologies and the role of conservative management through the presentation of an athlete with traumatic onset of symptomatic carpal bossing.*

Clinical features: *This case report outlines the presentation and conservative management of an elite eighteen year old hockey player with symptomatic carpal bossing after a traumatic on ice collision. Carpal bossing is a bony, dorsal prominence in the quadrangular joint of the wrist that is inconsistently symptomatic.*

Intervention and outcome: *A conservative treatment plan consisting of education, reassurance, avoidance of aggravation, and soft tissue therapy allowed return to play in two weeks without restrictions or need for surgical consultation.*

Conclusion: *With inconsistent recurrence rates and surgical complications, the role of conservative management for symptomatic carpal bossing deserves further exploration. The conservative practitioner should be aware of the signs and symptoms of symptomatic carpal bossing to institute suitable treatment.*
(JCCA 2009; 53(4):282–289)

KEY WORDS: boss, carpal, athlete, hockey, trauma

Objectif : *Présenter les caractéristiques et faire de la sensibilisation concernant le carpe bossu symptomatique et discuter des étiologies potentielles et du rôle du traitement conservateur par la présentation d'un athlète avec une apparition traumatique du carpe bossu symptomatique.*

Caractéristiques cliniques : *Ce rapport de cas indique la présentation et le traitement conservateur d'un joueur de hockey d'élite de 18 ans présentant un carpe bossu symptomatique à la suite d'une collision traumatique sur la glace. Le carpe bossu est une protubérance osseuse dorsale de l'articulation quadrangulaire du poignet qui est symptomatique de façon non constante.*

Intervention et résultats : *Un plan de traitement conservateur de sensibilisation, de rassurance, d'évitement de l'aggravation et de traitement des tissus mous a permis un retour au jeu après deux semaines sans restrictions ou besoin de consultation chirurgicale.*

Conclusion : *Des taux de récidive non constants et des complications chirurgicales font en sorte que le rôle du traitement conservateur pour le carpe bossu symptomatique mérite qu'on s'y attarde davantage. Le praticien conservateur doit savoir reconnaître les signes et symptômes du carpe bossu symptomatique afin de prodiguer le traitement approprié.*
(JACC 2009; 53(4):282–289)

MOTS CLÉS : bossu, carpe, athlète, hockey, traumatisme

Introduction

The carpal boss is a bony and inconsistently symptomatic prominence appearing between the base of the second and third metacarpals, the trapezoid, and capitate on the dorsal wrist (figure 1).[1] This joint, termed the quadrangular joint of the wrist, is the only area in which carpal

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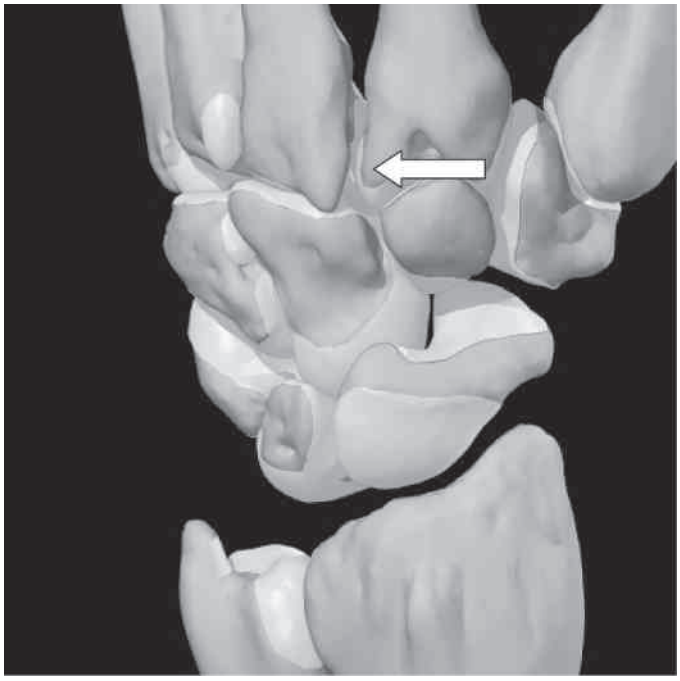

Figure 1 *The dorsal wrist with the white arrow illustrating the quadrangular joint.* (Image courtesy of and copyright to Primal Images ltd)

bossing appears.[2] Carpal boss etiology is a subject of contention with speculative reports on trauma, degeneration, instability, an accessory ossicle and most recently partial or complete osseous coalition appearing in the literature.[2–6] The undefined etiology makes the incidence difficult to ascertain. The pathophysiology of carpal bossing has never been elucidated with only speculative reports on why this bony prominence occurs.

Literature on the treatment of the symptomatic carpal boss is surgical.[1,4,7–13] Conservative management is attempted prior to surgical care, however the descriptions of outcome measures, course of conservative therapy and treatment regimen are poor. The indications and contraindications for surgery are similarly inadequate. Further confounding the topic are reports in prospective and retrospective longitudinal studies stating an inconsistent recurrence rate between 0 and 50% for simple resection, and a lack of sufficient follow up for recent advances in arthrodesis.[1,4,7,8,11–14] This questions the appropriateness of surgical management while leaving the conservative practitioner without definitive management criteria or prognosis. In an athlete, surgery results in missed playing time and a rehabilitation protocol that can take weeks to months.

This report will highlight a case of symptomatic carpal bossing in an elite eighteen year old hockey player and examine the relevant diagnosis, anatomy, etiology, pathophysiology, and the role of conservative management for symptomatic carpal bossing.

Case presentation

An eighteen year old male provincial level hockey player was acutely injured in game, and presented to the team chiropractor on the bench. The player, a defenceman, was hit into the boards by an opposing player with the dorsal aspect of his right wrist first contacting the boards while flexed. The subsequent addition of his mass and that of the opposing player further compressed the wrist between the boards and the players own body.

The player, right hand dominant, was otherwise healthy based upon a preseason assessment with no previous injury to the wrist. Upon equipment removal edema was noted on the dorsal aspect of the right hand and wrist. The primary complaint was poorly localized to the dorsal aspect of the right wrist over the second and third carpometacarpal joints, and the length of the second and third metacarpals. The player rated the intensity of pain as 8 out of 10. Active range of motion of the right wrist revealed limitations in flexion by fifty percent compared to the left due to moderate pain on the dorsal aspect of the right wrist in the area of the primary complaint. Mild pain was produced in the same area at end range of extension, ulnar and radial deviation. Active supination and pronation were full and pain free. Passive range of motion of the right wrist produced identical findings as active range of motion testing. Resisted extension of the right wrist produced moderate pain in the area of the primary complaint and was graded four out of five compared to the left wrist. Resisted flexion, ulnar deviation, radial deviation, supination and pronation were graded five out of five with mild pain. Grip strength was graded as four out of five when compared to the left side. Resisted metacarpophalangeal joint extension produced mild pain in the area of primary complaint. All other active, passive and resisted range of motion testing of the digits was within normal limits.

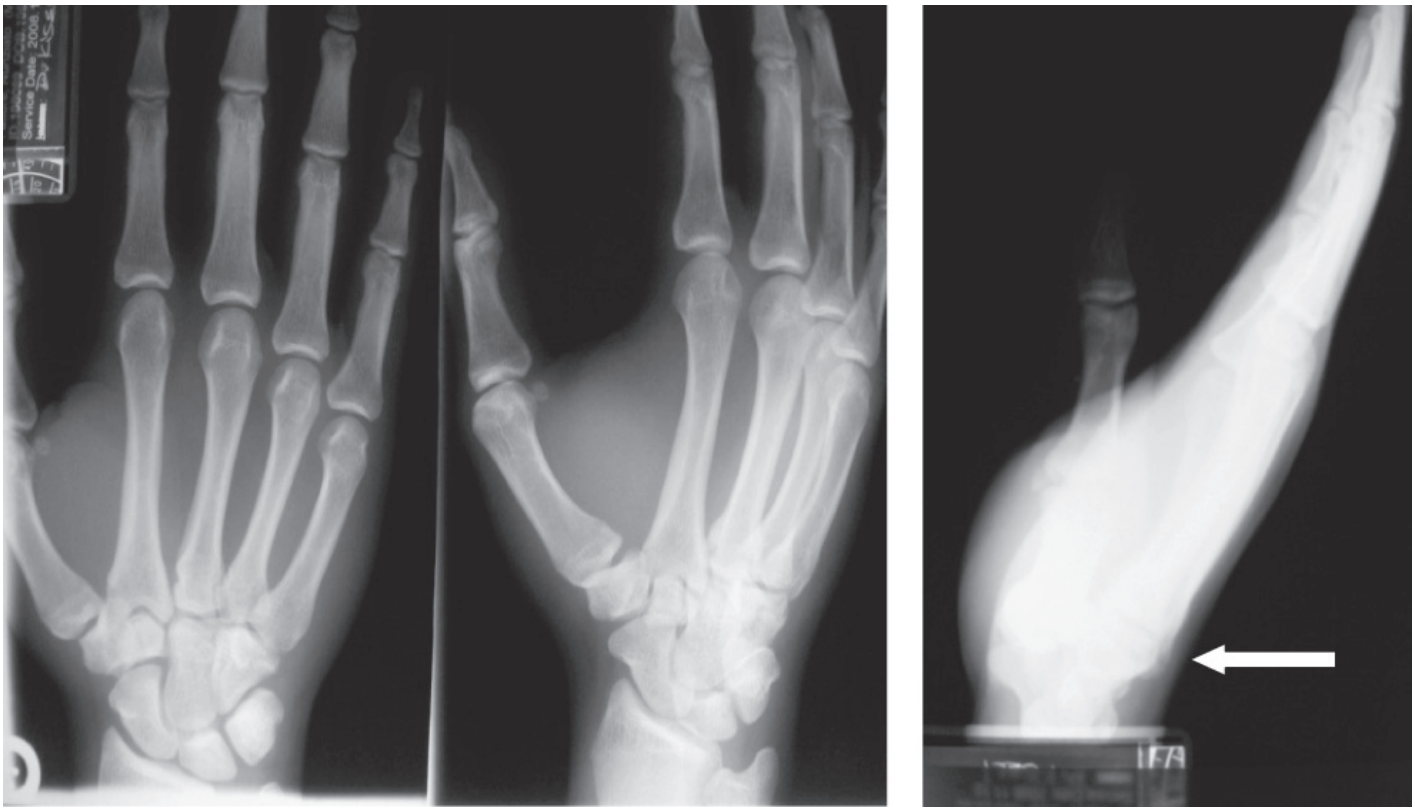

Figure 2 *x-ray imaging series with white arrow showing a dorsal projection on the lateral view at the area of the quadrangular joint diagnosed as an os styloideum.*

A neurological evaluation including sensory, motor and deep tendon reflexes of the upper limb was performed and deemed within normal limits.

Palpation revealed tenderness over the length of the right second and third metacarpals, the capitate and trapezoid. Anterior/posterior shear testing of the second and third metacarpals caused moderate poorly localized pain. Axial compression of the second and third carpometacarpal joints reproduced the patient's dorsal wrist pain. A 128Hz tuning fork was felt as painful at the base of the second and third metacarpals, the trapezoid and capitate compared to vibration in the same areas on the left wrist.

The player was given ice and was not cleared to return to play. Suspecting fracture, the player was instructed to attend a facility for radiographic imaging. The following day the player had not gone for imaging and presented with the same physical findings but only mild swelling allowing palpation of a bony projection in the area of the quadrangular joint. The patient had radiographic imaging of the right wrist seen in figure 2.

The patient was diagnosed by a chiropractic radiologist with an acute os styloideum of the right wrist and was held out of competition for two days. A treatment plan consisting of reassurance and education on symptomatic carpal bossing to reduce fears of a more ominous wrist injury and Active Release Technique (ART®) soft tissue therapy of the extensor carpi radialis longus and brevis, extensor digitorum and extensor retinaculum was instituted twice weekly for two weeks. The player returned to practice without contact for the first two practices post injury. One week from the injury the player returned to full play with intensity rated at 0 out of 10. The player was only symptomatic with full active or passive wrist flexion combined with direct pressure over the bony prominence. The player continued play for the final two months of the hockey season without re-aggravation.

Discussion

Athletes sustain a variety of muskuloskeletal injuries. Hand and wrist injuries account for 3–9% of all athletic injuries.[15–16] A ten year longitudinal study of injuries sustained at the Olympic Training Centre in the United States revealed 8.7% of 8311 injuries involved the wrist and hand with 64% of the diagnoses representing sprains and contusions.[15] In collision sports, the incidence of hand and wrist injuries accounts for 15% of all injuries.[15]

The incidence of the symptomatic carpal boss is not well defined. There is a paucity of reports of symptomatic carpal bossing in athletes where a literature search produced one case report in a swimmer.[3] A dorsal ossicle in the quadrangular joint was first described by Saltzmann in 1725.[18] In 1931 Fiolle described a case of a bony wrist exostosis producing a functional disturbance providing the name carpe bossu.[19] Bassoe and Bassoe were the first to describe the incidence of bossing based on an x-ray study of 450 hands in 1955 suggesting the carpal boss as an accessory ossicle present in 1–4% of the population.[20] Because the etiology has been challenged, so too has the accuracy of the reported incidence. In a cadaveric anatomic study of the second through fifth carpometacarpal joints, Nakamura found carpal coalitions only in the area of the second and third carpometacarpal joints in 14/80 or 18% of dissected wrists.[6] Alemohammed found similar results, uncovering a partial osseous coalition in all cadavers with clinical signs of a bony prominence representing 19% of their sample.[2] These two studies suggest the true incidence of carpal bossing to be much greater than previously thought, however because the studies were cadaveric it is not possible to conclude the incidence of symptomatic carpal bossing.

The clinical presentation of symptomatic carpal bossing is variable. Data from surgical publications describing patient characteristics taken as continuous data suggests a mean age of 32.25 with a range of 11–75.[1,4,7,8,11,12] Presentation is most often in the dominant arm with varied reports on male to female distribution.[1,4,7,8,11,12] A review suggests patients may present secondary to direct trauma, advanced age and recurrent strain.[20] The literature demonstrates antecedent trauma in just 23–27% of cases, however a traumatic incident was present in this case.[1,4,7,8] Advancing age does not seem likely as the aforementioned mean age in the literature is 32 years. Repetitive strain, specifically forced wrist extension, is thought to aggravate bossing symptoms due to a tenosynovitis of the extensor carpi radialis longus and brevis at their insertions on the dorsal aspect of the base of the second and third metacarpals. There is variable presentation of ganglions seen in 0–30% of subjects.[1,4,7,8] The pain presentation is also variable, but improves with rest.[20]

Diagnosis of carpal bossing is made on clinical and imaging findings after ruling out various differential diagnoses. Ganglion cysts are the most common cause of dorsal wrist protuberances.[21] Differential diagnosis between a ganglion and carpal bossing is based on location and palpation. Between 60 and 70% of all ganglions occur over the scapholunate ligament.[21] Carpal bossing is always over the quadrangular joint. Carpal bossing has a hard consistency, while ganglions are mucin filled creating a cystic consistency that can be transilluminated.[20] Dorsal ganglia have been suggested to spontaneously resolve in 58% of cases.[22] Because the carpal boss is a bony prominence, there are no reports of spontaneous remission. Intraosseous ganglions can cause a similar pain presentation as the carpal boss.[23] Additional differential diagnoses include benign bony lesions such as aneurysmal bone cysts, unicameral bone cysts, enchondromas, osteochondromas and osteoid osteomas as well as malignant tumors and locally invasive tumors such as osteosarcomas, and giant cell tumors.[20]

Physical diagnosis begins with direct observation and palpation for a dorsal bony prominence accentuated with wrist flexion, using the opposite wrist for contrast. The clinician must keep in mind that when present 11–21% of bossing is seen bilateral.[2,6] Swelling and a dorsal ache are observed inconsistently.[20] Fusi describe the malalignment test where the examiner distracts the second and third metacarpal with concomitant supination and pronation while the metacarpal-phalangeal joints are held in flexion.[8] The authors claim this distorts the anatomic relationship of the quadrangular joint. When explored, the second and third carpometacarpal joints have one degree of freedom (flexion and extension), while the fourth and fifth have three.[24] Studies indicate only 1–3 degrees of flexion occur through the quadrangular joint, while 15 and 30 occur through the fourth and fifth carpometacarpal joints respectively indicating this area has very little motion.[24] This calls into question if distorting the anatomy at this joint can occur at all via the malalignment test. Lorea found the malalignment test positive in all 32 patients with a surgically con-

firmed carpal boss but only when utilizing the third digit.[11] Clarke described the stress test with palmar directed pressure on the second and third metacarpalphalangeal joints while stabilizing the metacarpals from the ventral side meant to aggravate degenerative arthritis. However degeneration has not been confirmed in carpal bossing. Anatomic studies only found degeneration in the metacarpal articulations 3 to 5, with none in the quadrangular joint.[6] Neither the malalignment or stress tests were utilized in this case example.

Though an acute os styloideum appeared on the routine wrist imaging series seen in Figure A, radiographic imaging of the carpal boss is difficult due to superimposition of structures on a standard wrist series.[18] The carpe bossu view was originally described as a lateral view of the wrist with the hand flexed and supinated thirty to forty degrees.[18] This projection mimicks the 30–40 degree dorso-radial projection of the boss.[18] The normal axis of the metacarpocapitate articular surface is oblique by twenty to thirty degrees. By adding twenty to thirty degrees of ulnar deviation the articulation of the boss if present will be exposed.[4] Hultgren found that four of sixteen patients with a surgically confirmed boss had preoperative radiographs that were inconclusive.[7] A negative radiograph cannot rule out carpal bossing. When bossing is present on radiographic imaging, there is no sclerosis or reduction of joint space typically noted indicating that a degenerative process may not be the etiology of the carpal boss.[1] Apple noted increased local uptake in the quadrangular joint utilizing bone scintigraphy in a case report on carpal bossing.[24] In a larger sample, Clarke found a positive bone scintigraphy in 12 out of 18 carpal bosses confirmed surgically.[12] Magnetic Resonance Imaging has been shown to pick up the bony anomaly however the high cost and long wait times call into question if this imaging modality is practical.[26]

Carpal boss etiology is undefined. Hypotheses of trauma, minor stress, instability, ganglion, accessory ossicle, ligamentous microrupture, chronic periositis, ununited fracture, degeneration, partial bony coalition and combinations of the above inundate the reader with theories based on scant evidence.[1–20] Commonly proposed etiological explanations for carpal bossing involve an os styloideum (accessory ossicle) causing aberrant biomechanics and a resultant degeneration, and a recently suggested etiology of a partial incomplete bony coalition.

An accessory ossicle, or os styloideum, in the quadrangular joint was identified in 1894 by Thompson. When present, this ossicle is suggested to be completely isolated in 2% of cases, but more commonly fused to the proximal third metacarpal (94%), the capitate (3.5%) or trapezoid (0.5%).[20] The relationship to the carpal boss was not drawn until 1955 by Bassoe and Bassoe who suggested that the os styloideum represented the symptomatic carpal boss.[17] This abnormal joint configuration could predispose an individual to the development of a highly localized degenerative arthritis making this area more susceptible to the effects of repetitive trauma. Normal ossification of the capitate occurs in infancy, and the trapezoid by 7 to 8 years.[27] The metacarpals have ossification centres in the distal epiphysis for metacarpals 2 through 5, however a second epiphyseal center in the proximal second and fifth metacarpal has been observed in up to 6% of the population.[28] The second metacarpal represents 33% of the pseudoepiphyses, however this fuses with the diaphyses by age fifteen to twenty.[28] These explanations for the etiology of the accessory ossicle are deficient and require further exploration. The presence of an os styloideum is rare, however was diagnosed on plain film radiograph in this case.

The major axes of stress in the normal wrist are the scapholunate and quadrangular joints.[29,30] The middle longitudinal arch is said to act as a fulcrum for the opposition of the first ray with the fourth and fifth rays.[30] Because the third metacarpocapitate joint receives great stress, degenerative joint disease may result from an inability of this abnormal joint configuration to withstand ordinary daily stresses. The evidence for degeneration is murky. Anatomically, dissection studies only show degeneration in the fourth and fifth carpometacarpal joints.[2,6] The age range of carpal boss does not coincide with degenerative changes revealing bossing in individuals as young as eleven years old, and a mean age of 32.[1,4,7,8,11,12] The radiographic appearance of carpal bossing shows no sclerosis of the joint margin or narrowing of the cartilage space.[1] The second and third carpometacarpal joints are extremely stable with one degree of freedom questioning if this stable joint is prone to degeneration at all.[24] In a study of 3156 residents of Tecumseh Michigan, there was no evidence of degeneration via x-ray in any carpometacarpal joints other than the first ray.[31]

The first suggestion of a partial osseous coalition oc-

curred in a 1995 surgical study which identified a bony anomaly in the quadrangular joint with an occurrence of 63%, most frequently a separate ossicle that was fused in part or completely to the metacarpals. Frequently the fused ossicle bridged both the respective metacarpal and carpal bones.[8] This possible etiology was further explored by Nakamura and again by Alemohammad.[2,6] Both studies found partial osseous coalition only in the area of the quadrangular joint, present in all cadavers with clinical signs of a carpal bossing.[2,6] The partial coalition was always on the dorsal aspect of the wrist. When present none of the five normal dorsal ligaments that cross the dorsal quadrangular joint were present.[6] It was hypothesized that carpal bossing is actually a congenital partial carpal coalition. A symptomatic carpal boss may result from a fracture or breakdown of the coalition similar to those seen in tarsal coalitions.[2] Though limitations in these studies as they pertain to this case exist; the in vitro nature of the study, the lack of strong objective criteria to establish clinically the dorsal protuberance and a sample not representative of a symptomatic bossing population, these studies do provide evidence to support a partial coalition as an etiological explanation. Coalitions of carpal bones are a failure of demarcation of joint spaces during embryogenesis caused by incomplete cavitation of a common embryologic carpal precursor during the fourth to eighth week intrauterine.[32–34] Coalitions can be cartilaginous or bony.[33] A partial coalition or synchondrosis occurs when there is some degree of joint space formation, often resembling a pseudoarthrosis.[32] Partial coalitions are more likely to cause symptoms than complete coalitions due to stress loading activities, while complete coalitions are symptomatic only after fracture.[32] The absence of adequate inta-articular cartilage between the incomplete coalition and normal carpus is thought to cause intolerance of forceful activity and is seen on magnetic resonance imaging as bone marrow edema.[34] The most commonly cited carpal coalition is the lunotriquetral coalition.[33] Minaar found thirty six wrists with carpal coalitions, classifying them in four types (table 1). Type 1 (Proximal pseudarthrosis) and Type 2 (Proximal osseous bridge with distal notch) are reported as potentially symptomatic, while Type 3 (complete fusion) and Type 4 (fusion with other carpal anomalies) are suggested to be asymptomatic.[33] The incidence of carpal coalition has been cited as 0.1–0.7% of the population with a familial tendency.[32–34] No studies on carpal bossing have explored the role of genetics. Carpal coalition has been cited as bilateral in up to 61.5%, while the anatomic carpal boss studies cite a bilateral occurrence of 11–21%.[2,6] The etiological explanation of a partial or complete congenital osseous coalition is the most promising explanation available. It is possible that the os styloideum may represent a partial or complete osseous coalition seen on radiographic imaging.

Table 1 *Minnaar Classification of lunotriquetral coalitions*

Type 1	Proximal Pseudarthrosis
Type 2	Proximal osseous bridge with distal notch
Type 3	Complete Fusion
Type 4	Fusion with Other Carpal Anomalies

No studies with adequate size on the efficacy of conservative management of carpal bossing exist. The treatment literature available is surgical and offers scant accounts of failed conservative therapy as an indication for surgery, however the conservative care attempted is variable and poorly defined.[1,4,7,8,11,12] Clarke utilized corticosteroid injections in a sample of 13 surgical candidates with no relief in 11 though the authors noted difficulty with the injection due to the distorted anatomy present.[12] Curtiss suggested immobilizing the hand and wrist for three to four weeks after initial diagnosis.[35] In a recent study Lorea included a conservative treatment regimen of splinting at night and during heavy manual activities and corticosteroid injections. This was responsive in 16 patients, with only 7 of the remaining 16 opting for surgery.[11] A twenty year review by Fusi suggested four to six weeks of rest, minimally restrictive reinforced elastic wrist brace, and the use of oral non-steroidal anti-inflammatory agents with no report on success.[8] The literature suggests that surgery is reserved only for those whose symptoms are chronic and interfere with either occupational or recreational activities, and not just for cosmetic deformities or only occasional discomfort (table 2).[1,8] The only conservative study with follow up was performed on 34 patients with 43 lesions treated with an explanation of bossing and advice on avoidance of provocation. Only 11 patients were followed from 1 to 7 years. All reported the

Table 2 *Artz and Posch Indications and Contraindications for Surgery*

Surigcal indications	1) Persistent pain in thelocation of the carpometacarpal boss with motion and with repeated stress to the wrist sufficient to interfere with the patient's occupation 2) Repeated bumping of the lesion in a patient whose occupation required that they work with their hands in close quarters, producing persistent pain in this region 3) Snapping of the digital extensor tendons over the carpometacarpal boss causing sufficient pain and tenosynovitis to require excision of the lesion
Surgical contra-indications	1) Only occasional discomfort or pain with unusual use of the wrist were not ordinarily considered an indication for surgery 2) Cosmetic deformity without other symptoms

boss remained stable in size, with disappearance of pain seen in 3.[1] The athlete presented in this case report was treated with a conservative approach based upon the recommendation of conservative management prior to surgical consultation. A treatment regimen of soft tissue therapy, education and avoidance of provocation produced superb results as qualified by the athlete's speedy return to play and decrease on numerical rating scale.

Surgical management of symptomatic carpal bossing has evolved greatly since it was first described by Carter in 1941 who suggested prompt recurrence advocating against surgery.[36] The surgical technique of wedge excision to the level of normal cartilage and cancellous bone followed by reconstitution of the dorsal capsular structures predominates.[4] The recurrence rate has a reported range of 0 to 50% in the literature, with great variation based on the definition of successful outcome.[1,4,7,8,11,12] Citteur explored the cause of recurrence suggesting the excision of the dorsal ligamentous structures causing continued instability in the joint.[37] However when a partial osseous coalition is present, there is complete absence of these ligaments questioning the validity of this study.[6] Vermeulen suggested the depth of wedge excision caused resultant instability showing a 55% excision resulting in instability, yet a clinical wedge excision does not exceed 33%.[38] Lorea explored the use of resection utilizing radial bone grafting and staple arthrodesis resulting in no complications, however the sample was only seven and the follow up only seventeen months.[11] Clarke looked at excision compared to fusion surgery, finding a fifty percent good and fifty percent poor outcome in each group where the definition of successful outcome was complete resolution of symptoms and patient satisfaction.[12] Because surgical complications and recurrence abound, an extended conservative management trial is advisable saving surgery for the most symptomatic patients with functional and occupational disturbances.

Conclusion

The role of conservative management for the symptomatic carpal boss deserves further exploration. Currently, there is an undefined best surgical procedure and unsatisfactory surgical outcome. In an athlete a surgical procedure would require four to six weeks without play in addition to the consult and procedure, significantly impacting their season. With a conservative approach of reassurance, education, a graded return to play and therapy, the player in this case was able to continue play one week after the original injury. It must be noted that there are limitations of basing a conclusion on one subject. Further research contrasting conservative and surgical care is required to compare the outcomes of these management styles.

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Golf-related stress fractures: a structured review of the literature

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Background: *Stress fractures are troublesome injuries. Sites of occurrence are activity-related and specific anatomical sites are endemic to certain sports. Little is known about stress fracture patterns in golf.*

Objective: *A structured review of the literature was conducted to identify the occurrence and injury sites of golf-related stress fractures.*

Methods: *A literature search of MEDLINE, CINAHL, and SPORTDiscus was conducted using a combination of controlled vocabulary and truncated text words to capture all articles relevant to golf-related stress fractures. Articles meeting inclusion criteria were descriptively analyzed.*

Results: *The search resulted in 164 articles, of which 13 met the inclusion, and reported 44 cases of golf-related stress fractures. Seven anatomical injury sites were identified with rib stress fracture being the most commonly reported. Stress fractures occurred on the golfer's lead-side in 80% of cases.*

Conclusion*: Golf-related stress fractures are infrequent injuries. The ribs were the most common stress fracture site, and a predilection for lead-side involvement was reported.*
(JCCA 2009; 53(4):290–299)

KEY WORDS: golf; stress fracture; golf injury; golf injuries; bony stress injury; athletic injuries

Contexte *: Les fractures de stress sont des blessures pénibles. Les sites d'occurrence sont fondés sur les activités et les sites anatomiques particuliers sont endémiques à certains sports. On en sait peu sur les fractures de stress au golf.*

Objectif : *Un examen structuré de la littérature a été réalisé dans le but de déterminer la fréquence et les sites de blessures des fractures de stress associées au golf.*

Méthodes : *Un recherche dans la littérature de MEDLINE, CINAHL et SPORTDiscus a été effectuée à l'aide d'une combinaison de vocabulaire contrôlé et de mots de textes tronqués pour obtenir tous les articles pertinents aux fractures de stress associées au golf. Les articles qui correspondaient aux critères d'inclusion étaient analysés d'un point de vue descriptif.*

Résultats : *La recherche a permis de trouver 164 articles, desquels 13 correspondaient à l'inclusion, et a rapporté 44 cas de fractures de stress associées au golf. Sept sites de blessures anatomiques ont été déterminés, la fracture de stress aux côtes étant celle qui a le plus souvent été rapportée. Les fractures de stress se produisaient du côté dominant des golfeurs dans 80 % des cas.*

Conclusion : *Les fractures de stress associées au golf sont des blessures qui ne sont pas fréquentes. Les côtes constituaient le site le plus commun de fractures de stress et une prédilection pour le côté dominant a été rapportée.*
(JACC 2009; 53(4):290–299)

MOTS CLÉS : golf; fracture de stress; blessure de golf; blessures de golf; fracture de stress osseuse; blessures de sport

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Introduction

With the remarkable ability of golfers, such as Tiger Woods and Vijay Singh, who captivate audiences worldwide, it is not surprising golf is becoming increasingly popular. In the United States, there are over 26 million golfers, and approximately 6 million of these golfers play more than 25 rounds a year.[1] The sport has typically been regarded as low impact with most participants seemingly unaware of the potential for golf-related injuries.[2] Epidemiological studies[3,4] have shown, golf injuries are not uncommon. A recent study reported an annual aggregate injury rate of 15.8 injuries per 100 players.[3] In a study of 703 golfers, greater than 80% of injuries were attributed to overuse, and injury prevalence increased if one played four or more rounds of golf a week or hit over 200 balls a week during practice.[4]

The prevalence of golf injuries may be attributed to the repetitive asymmetrical loads imparted to the body by the golf swing. This has been purported to create distinct side-specific injury patterns in golfers.[2,5] Such injuries have been described as lead-side or trail-side, depending upon the side of the body involved when performing the golf swing. For example, in a right-handed golfer the lead-side refers to the left side of the body and the trail-side to the right. In support of this notion are the side-specific injury patterns that have been identified in male and female golfers.[2] For instance, in professional male golfers, the low back, lead wrist, and lead shoulder have been reported as the most common sites of injury; whilst in females the lead wrist, lead hand, and low back were most common.[2]

As previously noted, greater than 80% of golf injuries have been attributed to overuse.[4] In sports, an overuse injury of particular concern is the development of a stress fracture. A stress fracture is a partial fracture to bone that occurs as a result of repetitive sub-maximal loading in the absence of acute trauma.[6] Overtime, if not removed, this repetitive loading will exceed the adaptive capacity of bone; eventually resulting in a stress fracture.[7] Two types of stress fractures have been described; an *insufficiency fracture* (resulting from normal stress applied to abnormal bone) and a *fatigue fracture* (normal bone subjected to repetitive stresses over time leading to mechanical failure).[8] Fatigue stress fractures are often associated with an activity that is 1) new or different for the person, 2) strenuous, and 3) repeated with a frequency that ultimately produces signs and symptoms.[7,8] Sites of occurrence of stress fracture are activity-related and certain sites are endemic to certain sports.[7–10] It is important to identify stress fractures promptly, as continued activity without treatment may result in a complete or catastrophic fracture.

While specific stress fractures have been identified in many sports[7–10], little is known about the anatomical injury sites of stress fractures in golf. A structured review of the literature of golf-related stress fractures can help identify stress fracture sites and their occurrence. Currently, such a review does not exist. The purpose of this article is to review the literature on the occurrence and injury patterns of golf-related stress fractures.

Methods

A literature search was conducted during April 2009 using MEDLINE (1950–2009), CINAHL Plus with Full Text (1937–2009), and SPORTDiscus with Full Text (1985–2008) through EBSCO Publishing. Search terms included controlled vocabulary from these databases (MeSH terms: "Fractures, Stress," "Fractures, Bone," "Fractures, Closed," "Cumulative Trauma Disorder," and "Golf"; CINAHL headings: "Fractures, Stress," "Fractures, Bone," "Fractures, Closed," "Cumulative Trauma Disorder," and "Golf") and truncated text words ("stress fracture*," "fracture*," "cumulative trauma disorder*," "golf*," and "golf injur*"). All terms from the controlled vocabularies were exploded and searched as major concepts when available.

Controlled vocabulary search terms "Fractures, Stress," "Fractures, Bone," "Fractures, Closed," "Cumulative Trauma Disorder"; and text words "stress fracture*," "fracture*," "cumulative trauma disorder*" were combined with the Boolean operator "OR" to capture all relevant articles pertaining to a closed fracture. Similarly, the controlled vocabulary search term "Golf," and text words "golf*," and "golf injur*" were combined with the Boolean operator "OR" to obtain maximum exposure for relevant articles pertaining to golf. The results of the two searches were combined with the Boolean operator "AND" to identify articles that would be utilized for screening for inclusion. In addition, scanning of reference lists was performed from the retrieved studies to identify any articles that may have been missed from the literature search.

Relevant articles were screened using abstracts and citations. Articles were included if they met the following criteria: the primary focus was the reporting of bony stress injuries or stress fractures; increased participation in golf was ascribed as the primary causative factor leading to injury; the article was published in a peer-reviewed journal; was at minimum a case report experimental design; and was written in English.

Results

A total of 164 articles were retrieved from the literature search. Upon applying inclusion criteria, 144 articles did not report a golf-related stress fracture as its primary focus; one article could not be retrieved due to the inability to contact the journal or primary author; one article was a small opinion piece; and six articles that met the criteria were excluded as they were not written in English. Thus, 13 articles were accepted for the review.

Of the 13 articles accepted, five were retrospective case series and eight were case reports. These studies identified seven different sites of golf-related stress fractures: rib,[11–16] hook of the hamate,[17] tibial diaphysis,[18,19] proximal phalanx,[20] sternum,[21] ulnar diaphysis,[22] and acromion[23]. Of the sites reported, rib stress fracture was the most frequently reported in three case series[11,13,15] and three case reports,[12,14,16] summating to 30 reported cases (see Table 1). The hook of the hamate stress fracture was described in one case series,[17] identifying seven cases (see Table 2); a tibial stress fracture was reported in one case report[18] and one case series,[19] accounting for three cases (see Table 3); and the other stress fracture sites each were reported in one case report,[20–23] respectively (see Table 4). A total of 44 cases of golf-related stress fractures were included in this review. Nearly 80% of the stress fractures occurred on the golfer's lead-side (35 cases); and side-specificity of injury was not reported in 1 case of stress fracture of the sternum.

Discussion

The majority of golf injuries reported in the literature are relatively benign and are classified as muscular strains and sprains.[1–3,24,25] Although, the true incidence of golf-related stress fractures is unknown, the results of this review identified 13 articles in the English literature that reported a total of 44 cases of golf-related stress fractures. An epidemiological study by Fradkin et al[25] found fractures accounted for 3.8% of self-reported golf injuries. While the authors did not sub-classify the types of fractures identified, it is likely a subset of the fractures reported included stress fractures. Interpreting the results of Fradkin et al[25] in conjunction with the results of the present review, it seems the occurrence of stress fractures in golf is not common. Reflective of the rarity of these injuries, the articles included in this review consisted of case series and case report experimental designs.

While golf-related stress fractures were seemingly rare, they seemed to have a predilection for certain anatomical sites. The most common stress fracture site was the ribs followed by the hook of the hamate. The ribs and hook of the hamate accounted for 68% and 16% of reported golf-related stress fractures, respectively. The less common sites included the tibial diaphysis, proximal phalanx, sternum, ulnar diaphysis, and acromion. Stress fractures were predominantly reported to have occurred on the golfer's lead-side (35 cases) as opposed to the trail-side (8 cases). Considering there seemed to be an anatomical preference of stress fracture sites in golfers, a detailed review of each site is presented.

Stress Fracture of the Ribs: Rib stress fracture was the most common golf-related stress fracture reported in this review (see Table 1). Golfers who presented with stress fracture of the ribs were almost exclusively novice golfers who participated in high-volume, high-frequency practice/training regimens. The site of occurrence was most often the lead-side posterolateral ribs with ribs four to six most frequently involved. Symptoms were typically reported as having presented with a gradual onset of vague posterolateral thorax pain that often prevented the golfer from further play. The diagnosis was often delayed as they were treated for a muscle strain or thoracic spine joint dysfunction with either a lack of therapeutic benefit or worsening of symptoms. Radiographic evidence of fracture was often unremarkable, and bone scintigraphy was frequently needed to confirm the diagnosis. Healing was usually uneventful with relative rest, and golfers were often able to return to play with no sequelae.

Rib stress fracture was reported to occur on the lead-side in 22 cases and on the trial-side in eight cases. When rib stress fracture was reported to occur on the golfer's lead-side, the proposed mechanisms of injury ascribed by the authors included: the repetitive striking of the ground

Table 1 *Articles reporting rib stress fractures in golf*

Author	Cases	Location	Subject Characteristics	Proposed Mechanism	Treatment	Outcome
Rasad[11]	3	• Case 1: Lead-side posterolateral ribs 4 & 6 • Case 2: Lead-side posterolateral ribs 4 & 5 • Case 3: Lead-side posterolateral ribs 6 & 7	All were beginning golfers in their first year of participation	Increased training intensity coupled with improper technique	Relative rest	Not Reported
Lord & Carson[12]	1	• Lead-side posterolateral ribs 6, 7 & 8	Beginning golfer in first year of participation	Increased training intensity & lead-side serratus anterior fatigue	Relative rest for 6 weeks followed by serratus anterior rehabilitation for 4 weeks	Returned to golf in 10 weeks and remained asymptomatic
Orava et al[13]	5	• Case 1: Trail-side, rib 7 at mid-axillary line • Case 2: Trail-side, ribs 6 & 7 at mid-axillary line • Case 3: Trail-side, anteromedial 7th rib • Case 4: Lead-side, 3rd rib at mid-axillary line • Case 5: Trail-side, posterolateral 6th rib	All 5 were beginning golfers in their first year of participation	Increased training intensity coupled with improper technique	Relative rest	All patients returned to golf and remained asymptomatic Timeframe for return to golf was case-specific with an average return to golf of 5.8 weeks.
Read[14]	1	• Trail-side, 5th Anterolateral rib	Amateur golfer for 13 years	Faulty technique: failure to rotate the thorax through the golf swing	Not Reported	Not Reported
Lord et al[15]	19	• 16 lead-side rib injuries • 3 trail-side rib injuries • Ribs 4–6 most commonly injured • All ribs were injured on the posterolateral aspect	18 beginners, 1 professional	Sudden increase in golfing activity & serratus anterior fatigue	Relative rest followed by serratus anterior and general conditioning rehabilitation	Not Reported
Goyal et al[16]	1	• Lead-side, posterior 4th & 5th ribs	Golf experience not described	High golfing frequency	Not Reported	Not Reported

Table 2 *Article reporting hook of the hamate stress fractures in golf*

Author	Cases	Location	Subject Characteristics	Proposed Mechanism	Treatment	Outcome
Aldridge et al[17]	7	All cases involved injury to the lead-side hook of the hamate	Competitive PGA tour golfers	Cumulative loading from the golf club handle and anatomical structures inserting on to the hook of the hamate	Surgical excision of the fractured fragment in all cases	Return to golf in 3 months; however, 4 patients returned to golf earlier with no deleterious effects

Table 3 *Articles reporting tibial stress fractures in golf*

Author	Cases	Location	Subject Characteristics	Proposed Mechanism	Treatment	Outcome
Gillies et al[18]	1	Lead-side tibial diaphysis	Professional Golfer	Repetitive chronic torsional force on a fixed tibia led to prodromal "shin" pain. A powerful "drive" caused a complete fracture	Immobilization in a long leg cast	Not Reported
Gregori[19]	2	Lead-side tibial diaphysis in both cases	2 Professional Golfers	Repetitive torsional force on a fixed tibia led to prodromal "shin splints" A powerful "drive" caused a complete fracture	Case 1: Immobilization in a long-leg cast Case 2: Intramedullary nailing and long-leg cast immobilization	Case 1: Return to golf after 9 months Case 2: Return to golf after 10 months

Table 4 *Articles reporting other types of stress fractures in golf*

Author	Cases	Location	Subject Characteristics	Proposed Mechanism	Treatment	Outcome
Gramp et al[20]	1	Lead-hand second proximal phalanx	Novice Golfer	Altered biomechanics and overuse	Relative rest for 2 months	Resolution of imaging findings with a 2 month follow-up MRI
Barbaix[21]	1	Sternum	Semi-professional golfer	Not reported	Exploratory surgery to rule out other pathology resulted in 9 weeks of rest	Not reported
Koskinen et al[22]	1	Lead-side ulnar diaphysis	Amateur golfer	Externally rotated grip and increased training intensity	Relative rest and correction of grip	Return to golf 19 weeks after onset of symptoms; however, occasional pain was still experienced
Hall et al[23]	1	Lead-side base of the acromion at the spine of the scapula	Amateur golfer	Powerful muscular contraction of the posterior fibres of the deltoid	Sling immobilization, analgesia, and physiotherapy	Rapid functional recovery; however, time frame was not reported

with the club[11] and fatigue of the lead-side serratus anterior.[12,15,16] The majority of reported cases made mention of the role of serratus anterior fatigue as a possible injury mechanism.[12,13,15,16] This supposition was based on electromyography data of the golf swing that reported constant activity of the lead-side serratus anterior; whereas, the serratus anterior of the trail-side exhibited a discrete on-off firing that coincided with scapular protraction and retraction respectively.[26,27] This muscle activation pattern is thought to render the serratus anterior of the lead-side more susceptible to fatigue.[12,15] With high-volume and high-frequency training, fatigue may cause decreased activation of the serratus anterior. A decrease in activation of this muscle has been shown to shift the concentration of compressive and tensile loads towards the posterolateral aspect of the ribs.[28] The frequent descriptions of posterolateral rib stress fracture reported in the golf literature is suggestive of this injury mechanism.[11,12,13,15]

In contradiction to the more prevalent lead-side injury, when rib stress fracture occurred on the golfer's trail-side, authors of these articles attributed this discrepancy in injury location to different injury mechanisms. One author[14] reported a failure of the golfer to rotate the body properly through the golf swing and another author[13] postulated a possible thoracic cage impingement mechanism of the trial-side ribs. These injury mechanisms were

thought to contribute to increased stress localization to the ribs of the golfer's trail-side, subsequently causing rib stress fractures at this site.

Considering stress fracture of the ribs was the most common golf-related stress fracture reported; clinicians who treat golfers should be cognizant of the potential for this injury. Rib stress fracture should be included in the differential diagnosis when a golfer presents with vague thoracic and/or thorax pain.

Stress Fracture of the Hook of the Hamate: Hook of the hamate fractures are rare; however, they are endemic in sports involved in gripping a club, racquet, or bat.[17] Hook of the hamate fractures in golfers have long been reported in the literature since the first description by Milch in 1934.[29] The most cited mechanism of injury was the golfer striking an object other than the ball, causing the end of the club to abut the hook of the hamate of the lead wrist. Recently, some authors have questioned the validity of this acute traumatic mechanism and have proposed a mechanism of repetitive bony overload causing a stress-induced injury.[17,30,31]

Championing this contention, Aldridge et al[17] reported seven cases of highly competitive golfers with fractures of the hook of the hamate of their lead hand with no temporal relationship between onset of pain and a traumatic event (see Table 2). All patients were amateur or professional golfers on the Professional Golfer's Association (PGA) tour. As competitive golfers the subjects were required to perform over 200 golf swings a day. All subjects reported a gradual onset of vague hand pain over the hypothenar region. Upon examination, all subjects reported palmar tenderness to palpation over the hypothenar region and exacerbation of symptoms during their golf swing. They reported no antecedent trauma.

Fractures of the hook of the hamate were confirmed in all seven golfers by either radiography, CT, or MRI. All fractures were complete, but minimally displaced. Each patient underwent excision of the fracture fragment and returned to pre-existing level of play within 3–6 months. The authors theorized the golfers sustained a stress fracture to the hook of the hamate as a result of repetitive low-grade impaction of the end of the golf club on the hypothenar region, coupled with persistent tensile loading of the musculo-ligamentous attachments of the hook of the hamate.

It remains plausible there may be a subpopulation of golfers with hook of the hamate fractures that resulted from a stress-induced injury over-time. Repetitive loading of the hamate from the end of a golf club may weaken the bone to such a level that a seemingly low load such as striking a golf ball off the tee or hitting a ball "fat" results in a fracture of the hook of the hamate.[17] If this hypothesis were true, it would call into question the mechanism of injury of many of the reports of hook of the hamate fractures discussed in the literature. This could potentially affect the results of this present review, as 12 studies were excluded because their primary focus were hook of the hamate injuries described as a fracture rather than as a stress fracture. As recommended by Aldridge et al[17], further study is needed to ascertain if golfers experience prodromal symptoms and/or imaging findings that are consistent with a stress reaction of bone prior to hook of the hamate fractures.

Stress Fracture of the Tibial Diaphysis: Tibial stress fractures are often transverse fractures that involve the diaphysis.[10] Spiral and oblique tibial stress fractures are rare.[19] Three cases of tibial stress fracture (2 spiral and 1 oblique) have been reported in professional golfers, and all three cases reported similar injury characteristics (See Table 3).[18,19] In all cases, the lead tibia was affected, and the patients reported prodromal symptoms of "shin splints" localized to the affected tibia. Acute fracture incidents occurred during the generation of a powerful swing off the tee. In all three descriptions, spectators reported hearing a loud audible "crack" as the golfers completed their swings.

Spiral and oblique tibial fractures are often caused by torsional forces on the tibia.[32,33] During a golf swing, the lead leg is secured to the ground by spiked shoes. This locks the lead leg in place, and subsequently, the lead tibia must resist a large external torsional load. The torsional load applied to the lead tibia during the golf swing may explain why the reported fractures were spiral and oblique fractures rather than the more common transverse tibial fractures found in runners.[18,19]

Professional golfers frequently participate in golfing activities. Aside from other physical training, they may strike an average of 800 balls/week.[2] Increased training intensity, a secured lead foot to the ground, and a large repetitive torsional force applied to the tibia may explain

the generation of prodromal "shin splint" symptoms. Albeit, these symptoms are not common in golfers, their presence should warrant prompt investigation and management to prevent major injury.

Stress Reaction of the Hand: Similar to the assumption that bony stress injury can occur at the hook of the hamate from repetitive abutment from the golf club[17], Grampp et al[20] presented a case of overuse edema in the second proximal phalanx of the hand of a 29 year old female golfer (see Table 4). The golfer presented with mild pain and swelling localized to the medial second proximal phalanx of her lead hand. The symptoms commenced following one week of daily training sessions of golf. Radiographs demonstrated moderate soft tissue swelling adjacent to the base of the second proximal phalanx and no bony pathology. A MRI examination utilizing Short Tau Inversion Recovery (STIR) sequences identified diffuse increased signal intensity in the proximal and distal portion of the second proximal phalanx accompanied by focal increased signal intensity of the distal second metacarpal. These findings were reported to be consistent with bone marrow edema. The authors proposed the alteration in signal intensity represented a threshold between a bone bruise and the physiologic stress response of bone corresponding to bone remodeling. The authors attributed the cause of her symptoms to overuse and altered biomechanics; however, details of the precise biomechanical cause were not reported.

Stress Fracture of the Sternum: Sternal stress fractures are rare. The only previous sternal stress fracture reported in sport occurred in a wrestler.[34] In 1996, Barbaix[21] reported a case of a stress fracture of the sternum in a semi-professional golfer (see Table 4). The patient reported having purchased new clubs one month prior to participation in a tournament. In the weeks prior to the tournament, the golfer increased his practice volume and intensity to familiarize himself with his new clubs. He slowly developed pain and swelling at the 3rd and 4th ribs at the sternum. On the day of competition the pain was so severe he withdrew from the tournament. An ultrasound test that consisted of the application of 1 MHz continuous ultrasound at an intensity of 1 W/cm^2 provoked pain within seconds, and abated when it was reduced to 0.5 W/cm^2. Bone scintigraphy revealed a hot spot in the sternum consistent with the diagnosis of a stress fracture. The patient sought a second opinion from an orthopedic surgeon who performed exploratory surgery, and in the absence of pathology the diagnosis of stress fracture of the sternum was confirmed. The authors did not provide a discussion to explain the mechanism that lead to the focal increased stress localized to the sternum. It was unclear whether the stress fracture occurred in the midline, lead-side, or trail-side of the sternum.

Stress Fracture of the Ulnar Diaphysis: Stress fractures of the ulnar diaphysis are rare, and have been reported in tennis, volleyball, softball, and body builders.[9] The most common site is at the junction between the middle and distal third of the ulna.[9,22] Koskinen et al[22] reported a case of a 44 year old recreational female golfer who presented with lead-side dorsal wrist pain that worsened over time (see Table 4). The patient denied any incident of trauma. Upon examination, her wrist was slightly swollen and palpation of the ulnar side of the distal forearm was tender. Radiographs revealed a spiculated periosteal reaction on the radial side of the distal ulnar diaphysis with no signs of bone destruction. After 10 weeks, a palpable, non-tender, 2 cm long protuberance was located on the distal third of the ulna. A MRI revealed cortical thickening and an area of low signal intensity on T1-weighted images consistent with fracture healing. Mild edema was also noted at the fracture site with marked edema of the interosseous membrane. A radiograph one week following the MRI investigation revealed a lucent line with periosteal callus formation consistent with a stress fracture. The patient was treated with relative rest and technique modification. Her golf instructor recognized the affected lead hand was held in excessive external rotation while gripping the golf club.

Stress fractures of the ulnar diaphysis are believed to be a result of overuse of the flexors of the wrist. In the above case, due to the golfer's excessive external rotation of her lead wrist, it is possible that her lead forearm underwent excessive supination at the end of the golf swing which caused repetitive torsion of the interosseous membrane in conjunction with overuse of the forearm flexors.

Stress Fracture of the Acromion: Fractures of the scapula are quite rare, and occur most often in concurrence with a considerable traumatic force.[35] Hall and Calvert[23]

presented an unusual case of a stress fracture of the acromion in a 42 year old female who had recently begun to golf (see Table 4). The patient presented with a 15 day history of left shoulder pain that started immediately after striking a ball off the tee. Her pain prevented her from subsequently participating in golf-related activities. Upon examination, mild swelling and localized tenderness was noted over the spine of the left scapula. Radiographs revealed an undisplaced linear fracture at the base of the acromion that extended into the spine of the scapula. Hematology and bone densitometry were normal. The patient was treated with sling immobilization, analgesics, and physical therapy. Her fracture fully healed and she returned to normal activities. The authors suspected the mechanism of injury was due to the contraction of the posterior fibres of the deltoid as the club swung forward to strike the ball.

Limitations and Future Directions

A major limitation of this study was the articles included in this review consisted of case series and case report literature; representing level IV evidence.[36] Caution should be used when interpreting results from such a review, as results drawn from level IV evidence may over or underestimate the truth and may be non-generalizable due to inherent biases in study methodology.[37] Given the rarity of these injuries it is not surprising there is an absence of prospective studies investigating the topic.

A problem encountered when analyzing the retrieved articles was the lack of uniform reporting of clinical cases. Intervention details and patient outcomes were inconsistently reported. These limitations made it difficult to generate conclusions about the mechanisms leading to injury and the effect of the interventions prescribed. The majority of reports made attempts to rule out co-morbid conditions affecting bone quality; however, it was difficult to definitively establish if the fractures were fatigue or insufficiency fractures. It cannot be disregarded that, in some cases, the development of stress fracture may have been the result of a co-morbid condition or mere coincidence.

The present review did not include articles written in languages other than English. During the application of inclusion criteria, six relevant articles were excluded because they were not written in the English language. Upon review of the abstracts, the six articles were case reports reporting stress fracture of the ribs in golfers. Considering the present review included a total of 30 cases of rib stress fractures, it is unlikely the inclusion of an additional six cases would substantially alter the interpretation of the review.

Given the infrequent occurrence of these injuries, case-control research designs may have utility in future research to identify predictive variables leading to stress fractures in golf. Results from these studies could be utilized to develop intervention strategies to prevent these injuries. Stress fractures represent an overuse injury generated from repetitive sub-maximal loading.[6,7] Consequently, research endeavors that evaluate the effects of golf swing mechanics on the physiological load of the participant may also contribute to the development of effective intervention programs to help golfers safely enjoy their sport.

Conclusions

Although infrequent, golf-related stress fractures do occur. While definitive conclusions cannot be made from level IV evidence, the results of the review do highlight certain similarities relating to injury patterns. Golf-related stress fractures appeared to be site-specific with a preponderance on the golfer's lead-side. The majority of stress fractures were reported in beginner and novice golfers who had recently increased their practice/training volume or intensity. The most common site of stress fracture was the golfer's lead-side ribs. Stress fractures in novice golfers were frequently reported to be found in the ribs; whereas, in professional golfers they occurred at the hook of the hamate and tibial diaphysis.

The articles included in this review represented the current state of the literature. With the increasing popularity of golf worldwide it is important clinicians are aware of both the common and rare injuries that can occur as a result of golf participation. The intent of this review is to increase awareness of these injuries to better assist the clinician when evaluating the golfer who presents with an atypical injury presentation.

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Conservative management of posterior interosseous neuropathy in an elite baseball pitcher's return to play: a case report and review of the literature

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This report documents retrospectively a case of Posterior Interosseous Neuropathy (PIN) occurring in an elite baseball pitcher experiencing a deep ache in the radial aspect of the forearm and altered sensation in the dorsum of the hand on the throwing arm during his pitching motion. The initial clinical goal was to control for inflammation to the nerve and muscle with active rest, microcurrent therapy, low-level laser therapy, and cessation of throwing. Minimizing mechanosensitivity at the common extensor region of the right elbow and PIN, was achieved by employing the use of myofascial release and augmented soft tissue mobilization techniques. Neurodynamic mobilization technique was also administered to improve neural function. Implementation of a sport specific protocol for the purposes of maintaining throwing mechanics and overall conditioning was utilized. Successful resolution of symptomatology and return to pre-injury status was achieved in 5 weeks. A review of literature and an evidence-based discussion for the differential diagnoses, clinical examination, diagnosis, management and rehabilitation of PIN is presented.
(JCCA 2009; 53(4):300–310)

KEY WORDS: radial nerve, posterior interosseous nerve, radial neuropathy, baseball, arm injury.

Ce rapport documente rétrospectivement un cas de neuropathie interosseuse postérieure (NIP) s'étant produit chez un lanceur élite au baseball qui avait une forte douleur dans la face radiale de l'avant-bras et une sensation modifiée de la face dorsale de la main du bras qui lance lors du mouvement du lancer. Le premier objectif clinique était de contrôler l'inflammation du nerf et du muscle avec un repos actif, un traitement par micro-courant, une thérapie au laser à faible niveau et la cessation des lancers. On a réussi à minimiser la mécano-sensibilité à la région commune de l'extenseur du coude droit et à la NIP par un soulagement des douleurs myofaciales et des techniques d'augmentation de la mobilisation des tissus mous. La technique de mobilisation neurodynamique a également été administrée pour améliorer la fonction neurale. On a mis en place un protocole propre aux sports aux fins de maintien des mécaniques de lancer et de conditionnement général. La résolution réussie de la symptomatologie et le retour à l'état qui prévalait avant la blessure ont été réalisés en cinq semaines. Une revue de la littérature et une discussion fondée sur des preuves pour les diagnostics différentiels, l'examen clinique, le diagnostic, le traitement et la réadaptation pour la NIP sont présentées.
(JACC 2009; 53(4):300–310)

MOTS CLÉS : nerf radial, nerf interosseux postérieur, neuropathie radiale, baseball, blessure au bras

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Introduction

Posterior Interosseous Neuropathy (PIN) is a peripheral nerve injury commonly characterized by a sensation of a deep ache in the posterior forearm which can be accompanied by weakness of the forearm extensors and brachioradialis, and/or sensory alterations, or a combination of both. In the upper extremity PIN occurs with less frequency than median and ulnar mononeuropathies.[1] Among athletes, the incidence of PIN is unknown. Mechanical soft tissue injuries can present in a self-limiting nature and with similar symptomatology (pain and weakness) as a peripheral neuropathy which can make differentiating each condition a challenge.[2]

This report highlights a case of PIN in an elite baseball pitcher associated with throwing. Many case reports of peripheral neuropathies unresponsive to conservative care typically involve surgical management.[3,4] Other neuropathies of a repetitive nature provide recommendations of rest, nonsteroidal anti-inflammatory medications, physiological modalities, and rehabilitation regimes in the early stages of management.

Case Report

A 21 year-old male, right-handed pitcher, drafted to play professional baseball presented with an insidious onset of pain, fatigue, and altered sensation of the right posterior forearm of 1-week duration. This pain was experienced while pitching, more specifically during follow-through**,** both at maximal and submaximal efforts. The onset of pain and fatigue commenced with the initiation of pitching and progressively worsened with throwing to a maximum of 35 pitches which resulted in the discontinuation to pitch. The pain was rated as a 6/10 on the visual analogue pain scale and was described as a deep ache in the radial, posterior compartment of the forearm. The pain subsided upon cessation of throwing. The patient reported an altered sensation over the dorsum of the thumb and index finger, and acknowledged that the wrist and finger extension felt "weaker and more fatigueable" since the onset of the pain. The patient reported no previous injury of this nature to the throwing arm. The patient denied any history of trauma, surgery, or previous injury to the right upper extremity and/or neck.

Upon visual inspection, no swelling, redness, ecchymosis, deformity, spontaneous muscle activity (i.e. fibrillations), or muscle wasting of the forearm when compared to the non-throwing arm was observed. Active and passive ranges of motion of elbow extension elicited difficulty attaining a bony end-feel at end range of extension due to tightness in the antecubital fossa. Elbow flexion for all range types was within normal limits and a muscular end feel obtained. All active and passive ranges of motion of the wrist and hand were also within normal limits, while supination was limited at the end range actively due to the patient verbalizing discomfort in the proximal radial forearm. Passive forearm pronation combined with wrist flexion elicited patient discomfort in the proximal radial forearm. Resisted wrist extension with the elbow extended reproduced the patient's chief complaint**.**

Direct palpation of the supinator was markedly tender with a "snapping band" effect over the Arcade of Froshe. Proximal to the elbow joint, on the lateral aspect of the distal humerus, direct palpation of the brachioradialis and brachialis elicited tenderness. Palpation over the radial nerve branches that transverse the elbow joint (see figure 1) proved to be non-pain provoking. Joint play of the radiocapitellar and ulnotrochlear joints proved to be unremarkable and non-pain provoking. Direct palpation over the common extensor origin was unprovocative.

Orthopaedic testing revealed Cozen's Test (resisted wrist extension from a flexed elbow and pronated and extended wrist position) reproduced the patient's chief complaint. Repeat muscle testing, using a 15-pound dumbbell resulted in progressive fatigueability of the wrist extensors. A Modified Cozen's Test (modification with the elbow fully extended) also reproduced the chief complaint producing greater fatigueability and discomfort in the radial forearm. Pain and fatigueability subsided within minutes of stopping the test. Mill's Maneuver (passive extension of the elbow from a flexed, pronated forearm and flexed wrist/finger position) elicited tightness in the forearm and proximal to the elbow joint. Active cervical ranges of motion and Maximal Cervical Compression test (cervical spine lateral flexion, extension, and vertical compression) were unprovocative. Shoulder examination was unremarkable for any capsular pathology, rotator cuff pathology, acromioclavicular joint pathology, or glenohumeral labrum pathology. Elbow examination was unremarkable for Valgus-Overload Syndrome, Postero-lateral instability, and ligamentous insufficiencies. Thoracic outlet syndrome testing was deemed to be unremarkable for reproduction of symptomatology.

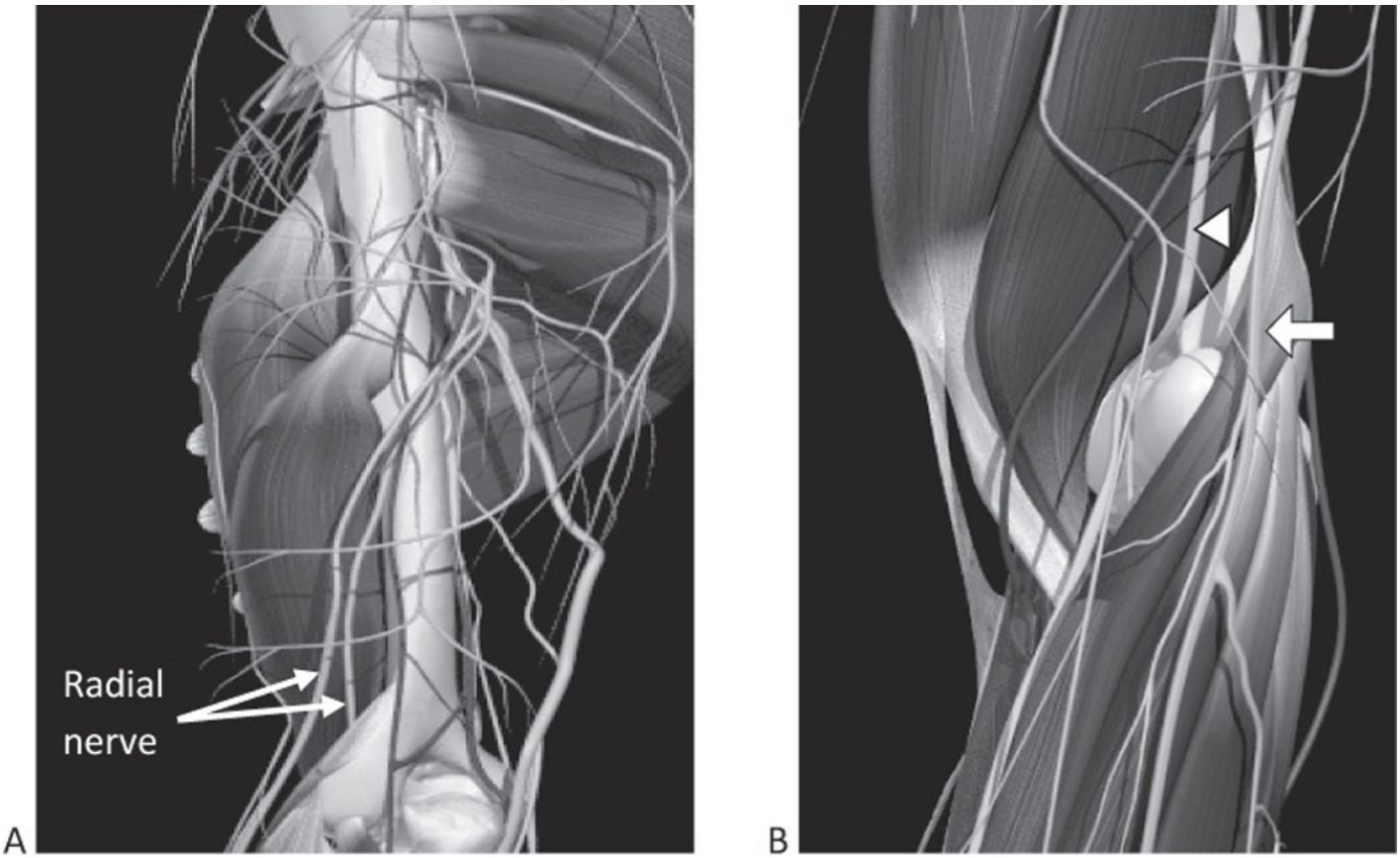

Figure 1 *Graphic depiction of the Radial Nerve at the distal humeral and elbow joint. (A) Radial Nerve branches coursing along the posterior compartment of the upper arm superficial to the brachioradialis. (B) Radial Nerve trunk and its branches (posterior interosseous (arrow head) and superficial radial (small arrow) nerves) coursing distally through the elbow and forearm region.* (3D anatomy images copyright Primal Pictures Ltd.)

The Upper Limb Tension test for the radial nerve trunk was used to assess the radial nerve as described in previous studies[5,6] (see figure 2). The protocol involves shoulder girdle depression with the elbow flexed to 90 degrees; this is followed by forearm pronation, elbow extension, wrist and finger flexion, shoulder abduction and cervical spine lateral flexion to the opposite side (see figure 1). Reproduction of the patient's chief complaint and altered sensation over the dorsum of the radial 3 digits which included the thumb was achieved. Pin sensation (sharp and dull) was symmetrical and intact over the dorsum of the thumb and index finger, the antero-lateral border of the forearm, and the postero-lateral border of the forearm. Position and vibratory senses were present, deep tendon reflexes were 2+ and symmetric in the upper extremity.

The clinical impression was Posterior Interosseous Neuropathy, as a first degree injury or neuropraxia, with an associated myofasciopathy of the supinator, brachioradialis, and brachialis which was consistent with the history and clinical findings.

The patient was treated six times over two weeks with soft tissue therapy (Active Release Techniques), augmented soft tissue mobilization technique (Graston Techniques) as presented in figure 3, and low level laser therapy to the affected muscles. Neural gliding was employed (figure 4) for the radial nerve. Mobilizations with

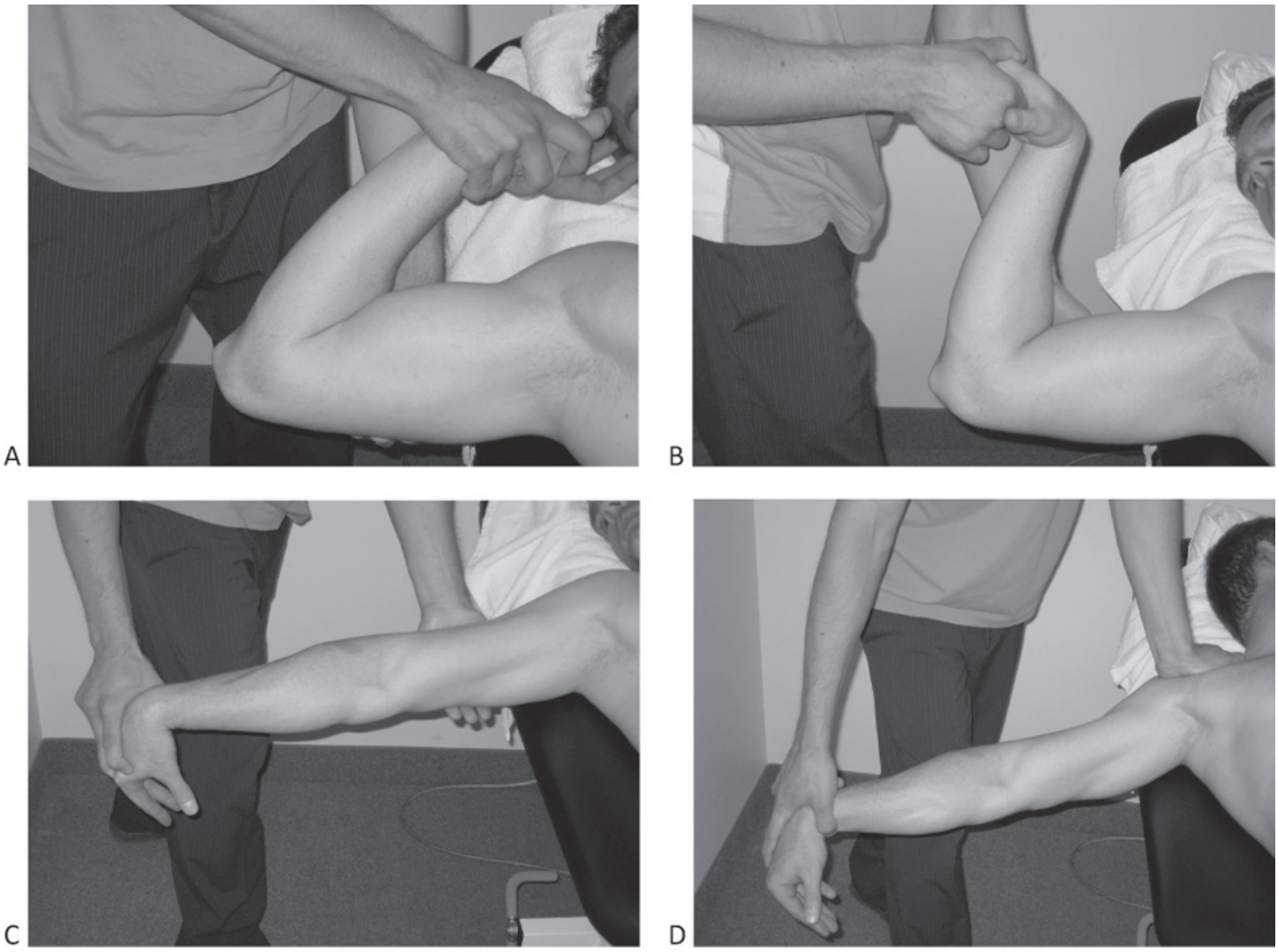

Figure 2 *Upper Limb Tension Test: Radial Nerve Protocol. (A) Starting Position: shoulder is in 90 degrees of abduction, elbow in full flexion, the forearm is fully pronated, the wrist and fingers placed in full extension and ipsilateral lateral flexion of the cervical spine. (B) Initiation of motion is first conducted by placing the fingers and wrist in to full flexion while maintaining the elbow, forearm, shoulder, and cervical spine positions. (C) Elbow extension and forearm pronation is started while still maintaining the shoulder in 90 degrees of abduction. (D) The shoulder is depressed and the cervical spine is laterally flexed to the contra-lateral side.*

movement were administered to the radiocapitellar and ulnotrochlear joints. Rehabilitative exercises for the right elbow and forearm were performed simultaneously with microcurrent therapy applied to the radial tunnel and distal course of the radial nerve. A sport specific rehabilitation program was performed to maintain conditioning of the lower extremity, core, and throwing shoulder. Instructions were to avoid pitching during the treatment period, except for light throwing to focus on appropriate motor control and technique.

Having a clear prognosis and clearance to participate in a baseball try-out camp is dependent upon having a healthy arm to pitch.

Upon the third visit the patient was referred to his

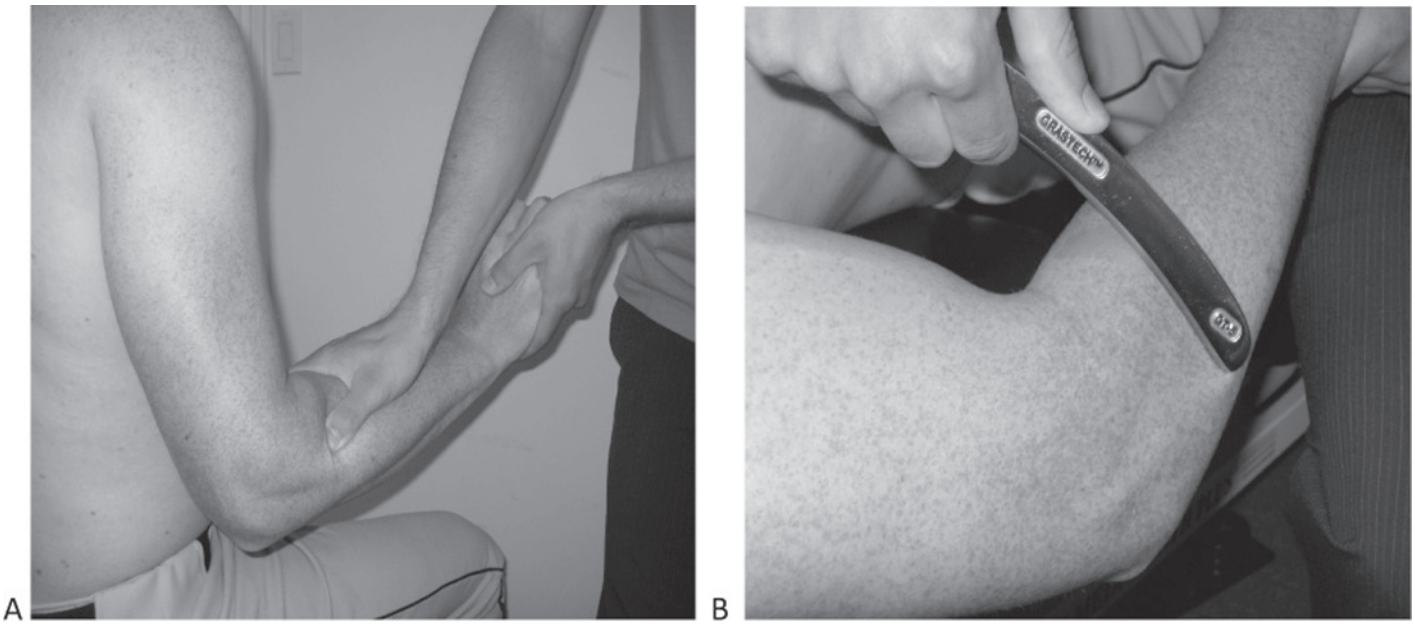

Figure 3 *Myofascial Release of the Elbow employing (A) Active Release Technique and (B) Graston Technique*.

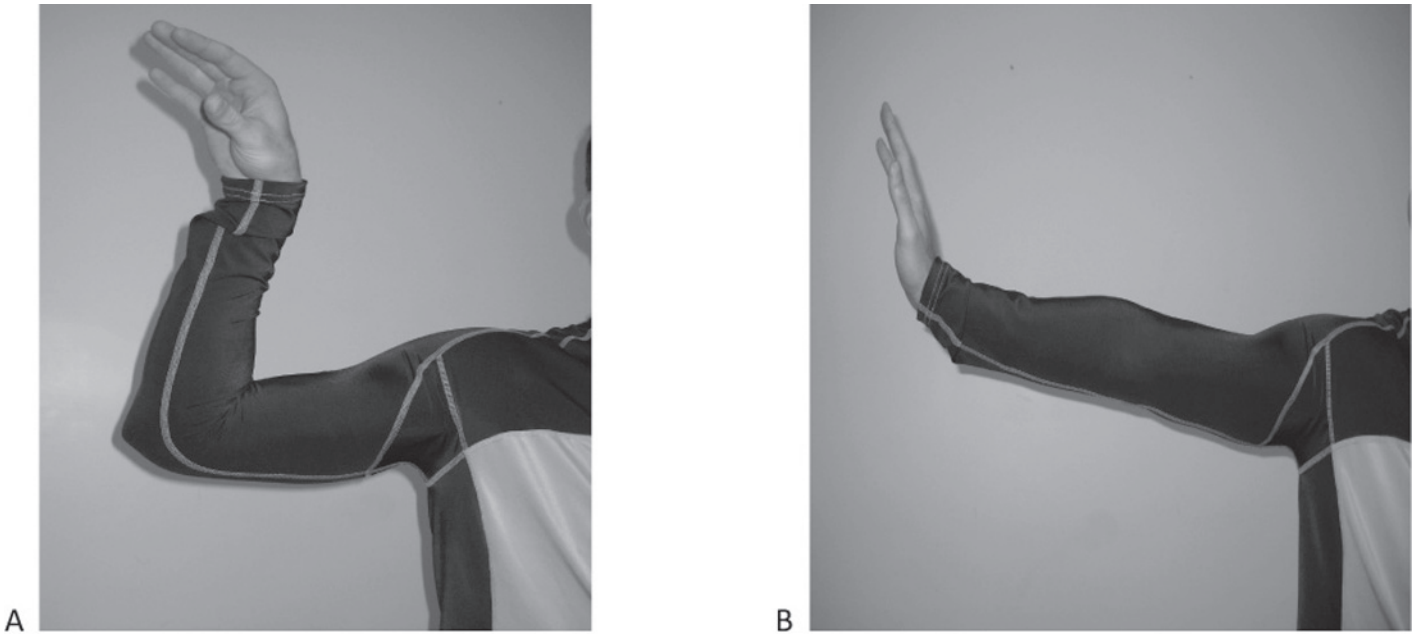

Figure 4 *Neuromobilization Technique for the radial nerve. (A) Start with the wrist and fingers in flexion, elbow in flexion, shoulder at 90 degrees of abduction. This is intended to permit maximal radial nerve excursion at the wrist and less at the elbow joint. (B) Commence by placing the wrist and fingers into extension and the elbow into extension. This is intended to permit maximal excursion of the radial nerve at the elbow joint and less at the wrist joint*.

medical doctor for a nerve conduction study to confirm the severity and ascertain prognosis of the injury. The nerve conduction analysis measuring the amplitudes of the motor action potentials was graded as within normal limits. It should be noted that patient's right radial nerve conduction velocity value was graded as within lower end-range of the normal limits measuring 53 m/s but this result was still above what is typically viewed upon as pathological (a decrease of equal or greater than 20%). This nerve conduction velocity study revealed a slight decrease in values from the norm for both the sensory and motor conduction velocities of the radial nerve at the elbow of the throwing arm when compared to the normative population ranges (64 m/s).[7] The remaining nerve conduction velocity study was normal and no advanced diagnostic imaging was taken.

The patient attended a scout camp in the third week, for 10 days, with minimal reproduction of symptoms in the posterior forearm and had his pitches measured over 85 mile per hour on average. Upon return from the camp, the patient was treated four additional times over a 14 day period with the same treatment plan as described above. At the end of the last treatment (38 days post first visit), the patient experienced a pain free throwing motion. The patient no longer reported any stiffness in the elbow, wrist extensor strength was powerful and absent of fatigueability during dumbbell testing, and his pitches were regularly measured at speeds over 90 mph which was a velocity previously achieved prior to injury.

Discussion

Peripheral neuropathies have been described in case reports and reviews among baseball pitchers involving the suprascapular nerve,[2] axillary nerve,[2] ulnar nerve,[2] musculocutaneous nerve,[8] and the lateral femoral cutaneous nerve.[9] In baseball pitchers, nerve injury has been cited in the literature accounting for approximately 2% of all diagnosed shoulder injuries.[2] There is a paucity of literature describing the occurrence of radial neuropathy in baseball and athletes alike. As a result, the incidence of radial neuropathy in athletes is unknown. The majority of the literature assessing the management of peripheral neuropathies consists predominantly of surgical nerve decompression.[8,9] This case report is a first to document a variant radial neuropathy in a baseball pitcher with conservative management.

The prevalence of nerve injuries are found to be rare among throwing athletes and can be a challenge to diagnose as a result of overlapping presentations for muscle pain and weakness. Consequently, delayed diagnosis can compromise prognosis with progressive neurological sequalae and deterioration of pitching performance. Even with resolution, determining the plan of management in the absence of definitive identification of tissue damage is limited.[2] Understanding how pitching exposes the nerve to insult will assist with tissue identification and direct treatment initiatives.

The proposed mechanism for injury in this case is the mechanics during the late stages of the throwing motion, the follow-through. The follow-through is defined from maximal shoulder internal rotation to a balanced fielding position after the ball has been released.[10,11] This is the phase of throwing that exhibits high levels of eccentric loading at the forearm along and along with repeated supination-pronation as a potential effect that places stress to the soft tissues of the throwing arm.[12]

The muscles involved with the deceleration phase are the rotator cuff muscles, triceps brachia, brachialis, supinator, and forearm extensor muscles. These muscles undergo eccentric loading that can theoretically compress or apply excessive tensile loading along the course of the radial nerve. This theory has not been substantiated in the literature, however observing the similarity of the position of the follow-through and the upper limb tension test for the radial nerve (see figure 5) suggests a plausible mechanism for assault to the radial nerve. This requires further investigation to demonstrate a relationship between radial nerve injury and the follow-through in pitching.

The pathophysiology of nerve injuries can be instigated by mechanical events involving repetitive movements as suggested with throwing or through direct blunt trauma.[13,14] The effect, either tensile or compressive, on the nerve stimulates the nervi nervorum, causing disruption of the microvasculature, and the deformation of the connective tissue. Irritation potentiates a transient inflammatory response (i.e. macrophagic activity) that can induce chemosensitive and immunosensitive reactions with prolonged exposure producing a noxious response to be perceived as pain.[13] Furthermore, the product of swelling is a consequence of compromised microcirculation at the endoneurial level and may lead to hypoxia, endoneurial edema, and restricted axonoplasmic flow.[14] Endoneurial edema

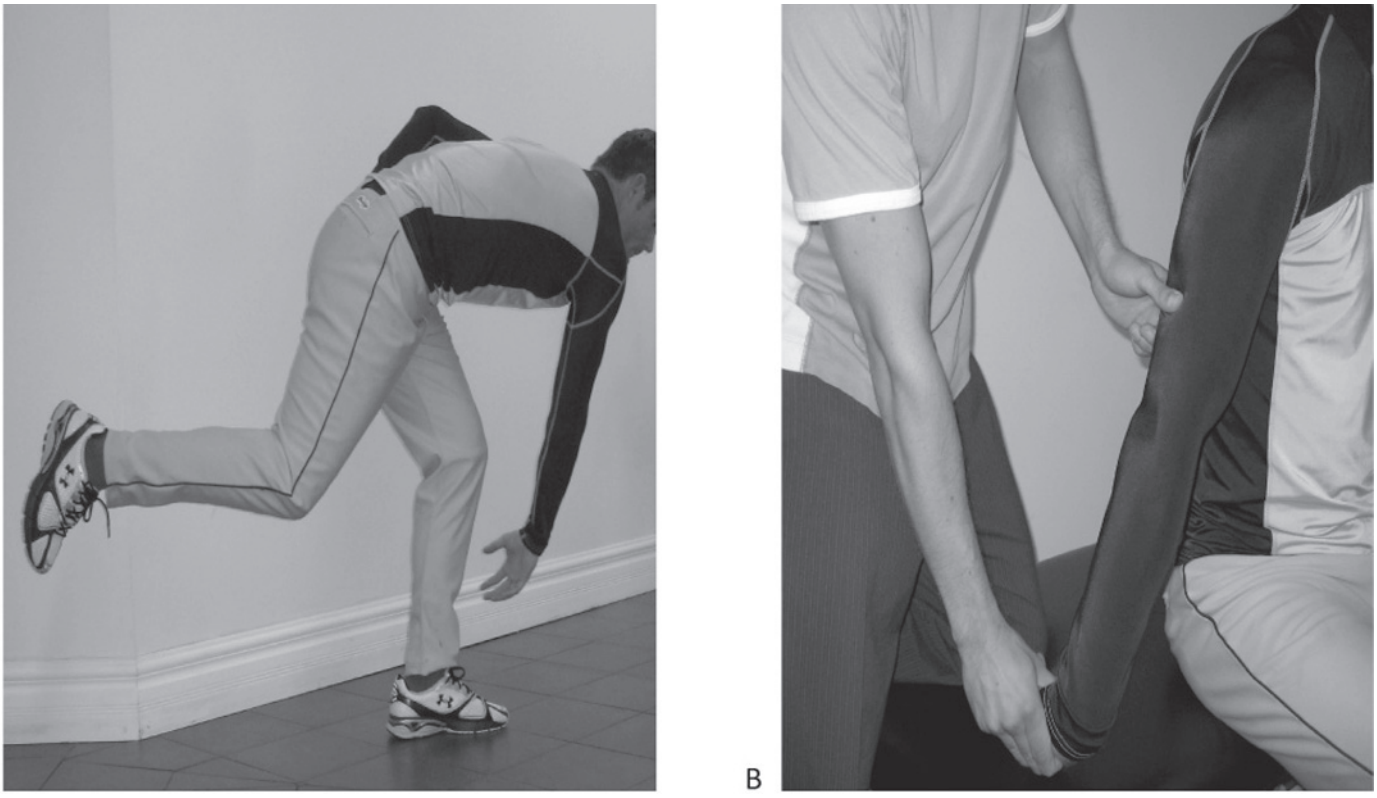

Figure 5 *Comparison of the Follow-through (A) and the Upper Limb Tension Test for the Radial Nerve (B). The extension that is observed in the follow-through is similar to the extension necessary to test the radial nerve as presented in the Upper Limb Tension Test.*

accumulation is compounded by the lack of lymph tissue present at this level of the nerve to facilitate flow of fluids.[15] Continued mechanical exposure, in this case throwing, potentiates increased nerve mechanosensitivity, propagating inflammatory responses inflicting chemosensitivity to the nerve and the surrounding musculature.[14,15] The manifestation can result in dysesthesia, hyperesthesia, hypoesthesia, paresis, and/or paresthesia. Clinical presentations include dysfunction, fatigueability, and disability. Muscular tightness also occurs as a result of excessive fibrosis, neural budding, and reflexive muscle tone to protect the injured nerve from further damage. Management involves eliminating any mechanical stimuli that are prevalent to reduce irritation and promote proper healing.

Differential diagnostic considerations of pathology afflicting the radial forearm were ruled out on the basis of the history, physical examination, and nerve conduction study. An exertional posterior forearm compartment syndrome was ruled out in the absence of pressure, disproportionate pain, and paresthesia. Cervical discogenic and/or radiculopathy were ruled out with a normal cervical and neurological screen. A possible referral from the rotator cuff was ruled out on the basis of normal palpatory findings, resisted muscle testing and specific orthopaedic testing. A suspicion of a stress reaction or fracture to the radial head and neck was not considered as local pain was not identified. Suspicion of a local space occupying lesion (i.e. ganglion cyst, lipoma, intraneurial neuroma) should always be suspected with neuropathies but a normal nerve conduction study and relatively quick resolution of symptomatology assisted with ruling this out. Consideration of a possible entrapment or compression of the nerve should be suspected when examining this patient, however only diagnostic imaging (magnetic resonance imaging or diag-

nostic ultrasound) or surgical exploration is definitive.[16,17] The clinical examination does suggest the consideration of a concomitant grade I muscle strain of the supinator, brachioradialis, and extensor carpi radialis longus (ECRL) as a result of the patient reporting pain upon palpation and reduced ranges of motion.

A neuropathy is typically defined as a nerve injury that exhibits neuro-pathophysiological adaptations, lacks a definitive pathophysiological cause (entrapment/compression, neuroma, ganglion cyst, lipoma, etc.) as confirmed on advanced diagnostic imaging (MRI, ultrasound), aggravated with movement and/or unequivocal electrophysiological testing.[15] In addition, the presentation of neurologically related symptomatology can be categorized into Sunderland's or Seddon's Classification of a nerve injury (Table 1). Nerve "Entrapment or Compression" can be definitively identified during surgery when the nerve is dissected and observed to demonstrate the constricted region that is associated with the neurological presentation.[16,17] However, there are protocols manual therapists perform that attempt to identify and localize the area of suspected "entrapment" or irritation (i.e. Upper Limb Tension Tests).[5,6]

The current understanding for conservative management of peripheral neuropathy emphasizes reducing mechanosensitivity to the peripheral nervous system and restoring neurodynamic function.[31] A fundamental understanding of the pathophysiology and the magnitude of involvement between nonneural tissue (muscle, fascia) and neural tissue are necessary.[13,15]

Treatment to restore non-neural tissue function can consist of a variety of soft tissue techniques consisting of myofascial release and augmented soft tissue mobilization. Myofascial release (Active Release Techniques ©) has been successfully employed for various soft injuries: lateral epicondylosis,[18] dorsal interosseous strain,[19] hamstring tightness,[20] and trigger thumb.[21] Augmented soft tissue mobilization (Graston Technique ©) has been used in the treatment of thoracic spine dysfunction,[22] trigger thumb,[18] costochondritis,[23] and subacute lumbar compartment syndrome.[24] These two techniques are theorized to breakdown adhesive and fibrotic deposits at the level of the tissues, restore physiological range of motion, and provide biofeedback through manual application or instrument assisted applications thereby reducing noxious stimulation. The theoretical mechanism of action suggests that stimulation at the site of the injury increases the concentration levels of satellite cells (mesenchymal cells with highly differentiating capabilities) that differentiate into myoblasts to facilitate the production of healthy connective tissue.[25] The implementation of myofascial therapy has demonstrated resolution of symptomatology within 4 weeks.[18]

Physical modalities can be employed to control the inflammatory response and promote healing of the nerve through the utilization of microcurrent therapy and low-level laser therapy directed over the nerve trunk between the BR and Brachialis and radial tunnel. Microcurrent is an electrotherapeutic modality inducing a subsensory electric charge to the skin surface producing no sensation during treatment. Microcurrent has been shown to significantly reduce pain thresholds over a 5 day period.[26]

Low level laser therapy (LLLT) involves applying wavelengths of energy, between 630 and 1064 nm, at varying doses to penetrate specific depths.[27] The conditions known to be treated with LLLT include tendinopathy,[28] arthropathies,[29] and myofasciopathies.[30] Laser has been shown to increase collagen synthesis and reduce pain thresholds in as little as 10 and 21 days respectively since the start of treatment.[27–30]

Neuromobilization was implemented using the nerve gliding technique designed to facilitate the enhancement and restoration of nerve mobility through the numerous nerve beds (see figure 3). Nerve gliding is strategic joint movements designed to elongate the nerve bed at one joint while simultaneously reducing the length of another nerve bed at an adjacent joint. Nerve gliding exhibits greater excursion of the nerve through the nerve bed than nerve tensioning.[31] Nerve gliding is theorized to apply less tension to the affected nerve because of the segmental partitioning of the nerve beds to minimize the ectopic axonal discharge (i.e. fibrillations, dysesthetic pain) and to permit extravasation of intraneural edema and reduce symptoms.[31] Caution is warranted with excessive nerve tensioning as this can impede microcirculation and exacerbate neural symptomatology.[32]

A common rehabilitative strategy prescribed to overhead athletes is the "Ballistic Six "exercises as suggested by Pezzullo and colleagues.[33] These exercises were used as rehabilitative and preventative strategies. The concept emphasized that throwing should involved both eccentric and concentric movements that mimicked the various

Table 1 *Classification of Nerve Injuries*

Seddon's	Sunderland's	Dysfunction	Pathology	Mechanism	Prognosis
Neuropraxia	Type 1	Focal conduction block – motor, proprioception most common; Sensory, sympathetic loss	Myelin injury of large fibers Axons intact No Wallerian degeneration	Electrolyte imbalance; Disruption of myelin; Ischemia due to compression or traction	Transient to hours or days (anoxic or ionic imbalances); Mechanical compression or stretch take weeks to months
Axontomesis	Type 2	Loss nerve conduction at and below injury site	Disrupt axon with Wallerian degeneration; connective tissue intact	Compression	Recovery of function proportionate to distance from end of nerve
	Type 3	Loss of nerve conduction at and below injury site	Axon continuity and endoneurial architecture lost. Peri/epineurial structures intact	Compression	Scarring due to endoneurial deformation, edema, and hemorrhaging; poor prognosis; potential surgical intervention
	Type 4	Loss nerve conduction at and below injury site	Near complete deformation of nerve architecture, except for perineurium	Compression	Intraneural scarring and axonal misdirection; Poor prognosis requiring surgical intervention
Neurotomesis	Type 5	Loss of nerve conduction at and below injury site	Complete deformation of nerve architecture	Traction, laceration, compression	Recovery necessitates surgical resection, with incomplete acquisition of function.

Adopted from Zachazewski, Magee, and McQuillen. Chapter 20 Peripheral Nerve Injuries, page 658.[36]

phases of the pitching motion. The sport specific movement patterning and conditioning is also suggested to prevent injury and enhance ball velocity. This upper extremity plyometric regime has been demonstrated to increase concentric internal rotation and eccentric external rotation and pitching velocity (~2 mph) in 8 weeks. This regime was performed for 3 sets of 10–20 repetitions, 2 days per week. The exercises included: latex tubing external rotation, latex tubing 90/90 external rotation, overhead soccer throw using a 6-lb medicine ball, 90/90 external rotation side-throw using a 2-lb medicine ball, deceleration baseball throw using a 2-lb medicine ball,

and baseball throw using a 2-pound medicine ball. This protocol has been implemented in a similar manner with collegiate players.[34] Modification of this program was implemented to avoid the throwing based exercises to minimize irritation to the throwing arm until full range of motion and strength testing was pain free. This program was implemented to maintain sport specific movement patterns and conditioning of the upper extremity.

The limitations of this case report are those inherent with the type of study design.[35] This study lacked objectivity by not using a dynamometer to assess muscular strength. The subjective nature by which the muscle strength was assessed was not incorrect to clinically identify muscle weakness when compared to the unaffected forearm; notwithstanding it does not accurately validate and reliably assess fatigueability pre and post conservative management. The absence of diagnostic imaging to visualize the nerve injury site did not permit definitive correlation of symptomatology with pathophysiological changes to the nerve. This limitation results in the use of the categorization of the injury as a neuropathy to generalize the clinical entity even though the presentation is consistent with a neuropraxia/grade I nerve injury. Furthermore, because of multi-modal approach it is difficult to ascertain the contributions of each therapy mode would have on the early recovery. Finally, the natural history of the injury cannot be ruled out as an explanation for the resolution of symptomatology in a short time frame. Finally, this is the first report of conservative management of a variant radial neuropathy and important to understand that generalizing the results of this report are individualized to this particular patient.

This case report demonstrated the successful application of soft tissue therapy, electrophysiological interventions, neuromobilization techniques, and rehabilitative regimes. The goal was to restore an elite pitcher to pre-injury status afflicted with a neuropraxia of the PIN due to a repetitive use mechanism associated with baseball pitching. To provide assistance to clinical decision-making practices with neuropathy presentations the results of this case are for hypothesis generation in advanced research methodology trials involving the non-surgical management of neuropathies.

Conclusion

Peripheral nerve injuries in pitchers are rare. Diagnosing peripheral nerve injuries that present with only muscle weakness and pain can be challenging as mechanical muscle injuries can present concomitantly. This case of Posterior interosseous Neuropathy was a diagnosis of exclusion, upon ruling out various pathologies that afflicting the forearm. Uncertainty of the true pathophysiology (i.e. compression or entrapment causing nerve injury) was evident yet imaging was unnecessary as the resolution of symptomatology was swift with conservative management. The application of a multimodal conservative approach to managing a peripheral neuropathy can be a potential option for treatment but further research investigations should be implemented as this may be a viable alternative to surgery.

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Psoas Major: a case report and review of its anatomy, biomechanics, and clinical implications

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A 25-year-old male professional hockey player with right sided hip pain was diagnosed with myofascopathy of the right psoas major and rectus femoris. The patient maintained a conservative treatment regimen and was prescribed a four week active strengthening program. The program progressed from resisted concentric exercise to eccentric abduction/adduction exercises along with balance training, core stabilizing and endurance exercises in the first two weeks. In the final two weeks the program progressed to include sport specific exercises. At three weeks the patient was able to participate in non-contact practice and was clear for full contact at five weeks. The anatomy, biomechanics, and function of the psoas major muscle are discussed as is its influence on lumbar spine stability. Evidence-based evaluation and management strategies for psoas dysfunction are presented.
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KEY WORDS: Psoas, case report, exercise, stability, biomechanics

Un joueur de hockey professionnel de 25 ans avec une douleur à la hanche droite a été diagnostiqué avec un syndrome myofascial au muscle grand psoas droit et au muscle droit antérieur. Le patient a maintenu un régime de traitement conservateur et on lui a prescrit un programme de renforcement actif de quatre semaines. Le programme a progressé d'un exercice concentrique contracté à des exercices d'abduction et d'adduction excentriques avec un entraînement lié à l'équilibre, ainsi que des exercices de stabilisation et d'endurance lors des deux premières semaines. Au cours des deux dernières semaines, le programme a progressé afin d'inclure des exercices propres au sport. À trois semaines, le patient était en mesure de participer à une pratique sans contact et a été autorisé au contact sans restriction après cinq semaines. L'anatomie, la biomécanique et la fonction du muscle grand psoas sont discutés, tous comme son influence sur la stabilité de la colonne lombaire. L'évaluation fondée sur les preuves et les stratégies de traitement du dysfonctionnement du psoas sont présentées.
(JACC 2009; 53(4):311–318)

MOTS CLÉS : psoas, rapport de cas, exercice, stabilité, biomécanique

Introduction

Some researchers and anatomists still refer to the hip flexor muscle complex as one unit or as the iliopsoas.[1,2] The psoas muscle differs from the iliacus in that it has a different architecture, innervation and more importantly, a different function. The psoas muscle is comprised of both the

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psoas major and minor, but as the psoas minor is often absent in individuals,[3] this paper will focus on the psoas major. A better understanding of the role of the psoas muscle and its impact on lumbopelvic stability may improve the clinical management of individuals suffering from lower back pain. The objective of this paper is to provide a brief case report followed by an evaluation of the literature on the psoas major muscle, specifically its anatomy, biomechanics and function, along with management strategies for psoas major dysfunction. It is hoped that this will enhance the clinician's understanding of this condition and its diagnosis.

Case Presentation

A 25-year-old male professional hockey player presented with right sided hip pain after 5 days of pre-season training camp. The pain was characterized as sharp, had progressively worsened since its insidious onset three days prior, and was aggravated by weight bearing on the right leg and striding out when skating. Rest, ice, athletic taping to minimize hip extension and abduction, and non-steroidal anti-inflammatories decreased the patient's pain somewhat, but striding out when skating continued to exacerbate the condition. The athlete did not recall any particular action or incident that caused the pain but did have history of multiple "groin pulls" throughout his career. No burning, numbness or tingling was present in the hip, groin, thigh or genitals. The patient denied any associated signs and symptoms, previous surgery to the area, or family history of arthritic diseases.

Inspection of the region revealed no ecchymosis or divot deformity. Active, passive and resisted ranges of motion of the lumbar spine and knee were full and pain free bilaterally, while right passive hip extension and resisted hip flexion were limited due to pain. The right psoas major was tender to palpation as was the proximal aspect of the rectus femoris. The strength of the psoas major and rectus femoris were graded as 4/5 using the Grading Motor Strength Scale, while all other hip and groin muscles were graded as 5/5 with manual testing. Muscle testing for rectus femoris was performed with the patient in lateral recumbent position with the hip extended and the knee flexed; whereas the testing for psoas major was performed with the patient supine with the hip flexed approximately 30°, abducted 10° and externally rotated.[4]

The patient was diagnosed with myofasciopathy of the right psoas major and rectus femoris. Myofasciopathy can be distinguished from a contusion or muscle strain in that the involved trauma is due to excessive tensile force that overstrains the myofibers and deep fascial layers surrounding the muscles and typically occurs near the myotendinous junction. The patient continued with the ice, taping and non-steroidal anti-inflammatories and was prescribed a four week active strengthening program. The program progressed from resisted concentric to eccentric abduction/adduction exercises, balance training, core stabilizing and endurance exercises in the first two weeks as denoted in Table 1. In the remaining two weeks of the rehabilitation program, the patient continued the previously prescribed exercises, while hockey specific exercises were cautiously introduced (initially under supervision) including sumo squats, side lunges and use of a skating slide board. The patient was able to participate in non-contact practices after three weeks and was cleared for full contact at five weeks.

Discussion

Psoas Major Anatomy

The psoas major is the largest muscle in cross section at the lower levels of the lumbar spine.[5] It has fibrous attachments to the anterior aspect of all lumbar transverse processes and to the anteromedial aspect of all the lumbar discs and adjoining bodies with the exception of the L5/S1 disc.[6] For their relative positions on the spine, the attachments on the transverse processes are named the posterior attachments and those on the disc and bodies are called the anterior attachments. These attachments constitute the individual fascicles. The fascicles of the psoas major are approximately similar in length throughout the lumbar spine and have a unipennate fiber orientation. Muscle fiber length within the anterior fascicles ranges from 3 to 8 cm and 3 to 5 cm in the posterior fascicles.[7] The fascicles are oriented inferolaterally and come together as a common tendon which descends over the pelvic brim and shares a common insertion with the iliacus muscle on the lesser trochanter of the femur.

The fascial relations of the psoas major to the surrounding tissues warrant special attention as these links influence the biomechanics of these interlaced structures. The medial arcuate ligament is a continuation of the su-

Table 1 *Active strengthening program*

Exercise	Timeframe (weeks)	Comment
Isometric exercises	0–2	
Concentric exercises	1–4	
Eccentric abduction/adduction exercises	2–4	
Balance training	0–4	
Core stabilizing and endurance exercises	0–4	
Sumo squats	3–4	Sport specific exercise, introduced under supervision at first
Side lunges	3–4	Sport specific exercise, introduced under supervision at first
Skating slide board	3–4	Sport specific exercise, introduced under supervision at first

perior psoas fascia that continues superiorly to the diaphragm. The right and left crus constitute the spinal attachment of the diaphragm. They attach to the anterolateral component of the upper three lumbar vertebral bodies. The crus and their fascia overlap the psoas major and appear to be continuous with this muscle until they come more anterior and blend with the anterior longitudinal ligament.[8] As the psoas descends, its inferomedial fascia becomes thick at its inferior portion and is continuous with the pelvic floor fascia.[9] This forms a link with the conjoint tendon, transverse abdominus, and the internal oblique.[10] As the psoas major courses over the pelvic brim, the fascia of the posterior fascicles attach firmly to the pelvic brim.

Psoas Major Biomechanics and Function

The function of the psoas major is another area of controversy and uncertainty in the literature. It is well established that the psoas functions as a primary flexor of the hip joint[6,10–14] but it is the other actions that are not well understood. There are several hypotheses that have been put forward that are worthy of consideration.

The electromyographic work of Basmajian[11] was the first to investigate the role of the iliopsoas. He concluded that the psoas major could not be separated from the iliacus with regards to their collective action of a hip joint flexor. Keagy et al.[15] performed electromyographic studies on the psoas major in five patients with wire electrodes placed directly into the muscle. Recordings made during various activities indicated that psoas played a significant role in advancing the limb while walking and in controlling deviation of the trunk when sitting. The action of the psoas in rotation, abduction, and adduction of the hip was slight and variable.

Nachemson[16,17] showed that the psoas major was active during upright standing, forward bending, and lifting. These observations prompted the inference that the psoas major may function as a lumbar spine stabilizer. Others have since proposed and found evidence for various roles that the psoas major may play with respect to lumbar spine stability and movement. These roles include psoas major being a flexor of the lumbar spine on the pelvis,[18] a lateral flexor of the lumbar spine,[19] a stabilizer of the lumbar spine,[10,13,14,20] stabilizer of the hip,[3,6,21] power source for bipedal walking and running,[22] and controller of the lumbar lordosis when supporting difficult lumbar loads.[23]

Yoshio et al.[24] used cadavers to analyze the psoas major in its dynamic phase (as a flexor of the hip joint) as well as in its static phase (involving fixation of the hip joint to maintain a sitting or standing position against gravity). Their results suggest that the psoas major works phasically: (1) as an erector of the lumbar vertebral column, as well as a stabilizer of the femoral head onto the acetabulum (from 0°–15°); (2) exerting decreased stabilizing action, in contrast to maintaining the erector action

(from 15°–45°); and (3) as an effective flexor of the lower extremity at the hip joint (from 45°–60°). They further concluded that the function of the psoas major as a hip stabilizer is overshadowed by its action of stabilizing/erecting the lumbar vertebral column.

Over the last decade, new insights into muscle function and the role of muscles in providing dynamic stability have emerged. Some muscles may have stabilization of the lumbosacral spine as their primary role, while others appear to have multiple roles and these multiple roles may be dependent upon spinal position and the loads being transmitted to the spine (i.e. low load vs. high load).[25–27]

Recent research on lumbar musculature and how it relates to individuals suffering from lower back pain has progressed through the use of advanced imaging techniques. Dangeria and Naesh[28] conducted a clinical prospective cohort study examining the cross-sectional area of the psoas major in healthy volunteers and subjects with unilateral sciatica caused by a disc herniation. These authors demonstrated that in most patients with a lumbar disc herniation there was a significant reduction in the cross-sectional area of the psoas major on the affected side only and most prominently at the level of the disc herniation. They suggested that a correlation exists between the reduction in the cross-sectional area of the psoas major (Spearmann's rho = 0.8; P = 0.05) and the duration of continuous sciatica of the affected side but that no correlation exists between the amount of disc herniation and reduction in psoas major cross-sectional area. Similarly, Danneels et al.[29] examined the trunk muscles (paraspinal, psoas and multifidus) in chronic low back pain patients and healthy control subjects employing computerized tomography at three different lumbar levels. These authors found no significant differences in the cross-sectional area of the psoas major or paraspinals but they did find significant differences existed in the cross-sectional area of the multifidus at the L4 spinal level. Barker et al.[30] investigated the cross-sectional of the psoas major in the presence of unilateral low back pain through the utilization of magnetic resonance imaging (MRI). These authors found that there were statistically significant differences in cross-sectional area of the psoas major between sides (median reduction was 12.3%) at the levels of L1-L5 and that there was a positive correlation between a decreased cross-sectional area of the psoas major and the duration of symptoms. In another MRI study, Hides et al.[31] assessed the effects of prolonged bed rest on the truck muscles. This study showed that the cross-sectional area of certain muscles decreased or were unaffected by bed rest as one would imagine but surprisingly found that the psoas major and rectus abdominis actually increased in cross-sectional area. The authors attributed this increase or hypertrophy to increases in muscle tone and to the possibility that the subjects maintained a flexed truck position during bed rest, resulting in a psoas muscle shortening.

More recently Dickx et al.[32] used muscle functional magnetic resonance imaging (mfMRI) to evaluate changes in lumbar muscle activity with induced muscle pain. This study was one of the first to examine patients with acute low back pain and how it affects activity of the trunk musculature. mfMRI was obtained under three different conditions: a resting MRI was obtained after the subjects laid supine for 30 minutes; an MRI was obtained after trunk extension at 40% of one-repetition maximum without pain; and an MRI was obtained after the subjects were injected with hypertonic salt into the right longissimus muscle to induce pain and then subjects were required to again perform the back extension exercise while experiencing low back muscle pain. There were no significant changes in the psoas major muscle recruitment between resting and exercise leading the authors to conclude that the psoas major was not significantly recruited during trunk extension exercises. During the trunk extension exercises with pain induced, the authors reported that there was a statistically significant reduction in the psoas major activity bilaterally and at multiple levels whereas previous studies found it to be ipsilateral and on the symptomatic side.[28,30]

Psoas Major and Lumbar Spine Stability

A common model of lumbar stability shows the musculature surrounding the spinal vertebrae forming a cylinder. The top of the cylinder is the diaphragm, the bottom is the pelvic floor, and the wall is formed by segmentally attaching abdominal and posterior spinal musculature, specifically the transversus abdominus and the segmental fibers of lumbar multifidus.[33] There is growing evidence that demonstrates how these muscles coordinate their activity to stabilize the spine. For example, transversus abdominis has been shown to co-contract with: the diaphragm;[34] the pelvic floor;[35] and the deep fibres of lumbar multifidus.[36] According to this model, the psoas major is ideally located

to assist in a stabilizing role. Psoas major has intimate anatomical attachments to the diaphragm and the pelvic floor. This unique anatomical location allows the psoas major to act as a link between these structures and may help in maintaining the stability of the lumbar cylinder mechanism. This can be thought of conceptually as a supporting rod in the middle of the cylinder. Early biomechanical literature suggested that the psoas major might aid in the stabilization of the lumbar spine through its large potential to generate compressive forces, which would result in increased spinal stiffness.[30]

McGill[37] conceptualizes lumbar spine stability as a fishing rod placed upright and vertical with tensioned guy wires attached at different levels along its length and those guy wires being attached to the ground in a circular pattern. Here the rod represents the lumbar vertebrae and the guy wires are the various muscles attaching to the lumbar spine. Reducing the tension on one of the muscles (wires) will allow the spinal segment (rod) to buckle and allow spinal injury to occur. Juker et al.[12] showed that the psoas major counteracts the action of iliacus during hip flexion. They believe that the iliacus would torque the pelvis into anterior pelvic tilt and that the psoas major works against these forces, adding to the stiffness within the pelvis and the lumbar spine. An activated and stiffened psoas major will contribute some shear stiffness to the lumbar motion segment.[38,39]

Psoas Major Clinical Presentation and Management

Myofascial pain from or mysofasciopathy of the psoas major muscle will often present as anterior hip and/or lower back pain. Referral areas include the anterior thigh.[40] The psoas major muscle can be considered as a pain source in athletes, office workers or anyone who spends much of their day sitting. Psoas major myofascial pain is thought to be prevalent in certain sports including soccer, dance, and hockey (as in the case presented above).[40] Myofascial psoas major pain is different from that of psoas tendinitis, psoas bursitis or coxa saltans and these are among the strongest differential diagnoses, along with tears of the hip labrum. Table 2 provides a list of possible differential diagnoses for psoas major myofascial pain. It should be noted that the snapping or popping of coxa saltans produces pain at the anterior aspect of the groin, and patients can often reproduce the snap or pop themselves.

In the physical examination, postural analysis may indicate an increased lumbar lordosis and posterior pelvic tilt. Gait analysis may reveal a shortened stride on the affected side and conducting a functional squat test may cause pain or indicate hip flexor weakness.[40,41] Strength testing of psoas major can be conducted in numerous ways (generally supine) as long as the patient resists the examiner's attempt to extend the hip.[4,42–44] Assessment of active and passive hip ranges of motion is important, particularly active flexion and extension and passive extension (generally performed with the patient prone).[45] Palpation can be conducted with the patient either supine or side-lying[44,45] but should likely involve the examiner flexing the hip to 30° and palpating the psoas major muscle medial to the anterior superior iliac spine and deeper into the abdomen. When the examiner feels they are palpating the psoas major muscle, having the patient flex their hip against resistance should allow the examiner to feel the psoas major contract. The examiner must consider patient comfort when palpating the psoas major as it may be extremely tender, ticklish, or more invasive than the patient's comfort level allows. Still, reproduction of the patient's pain on palpation of the psoas major muscle belly with tightness and tenderness are strong indicators of psoas major myofasciopathy.[40] The psoas major mus-

Table 2 *Differential diagnoses for psoas major myofascial pain*

Psoas bursitis	Osteoarthritis
Psoas tendinitis	Labral tear
Psoas strain (major and/or minor)	Intra-articular bodies
Psoas abscess	Joint infection
Coxa saltans	Inflammatory arthritides/ Gout
Iliacus	Femoral stress fracture
Iliotibial band	Avascular necrosis of femoral head
Rectus femoris	Femoral bone tumour
Adductor muscles	Hernia
Lumbar spine or sacroiliac joint referral	Obturator nerve entrapment

cle is intimately linked with the iliacus, psoas minor (if present), adductor group, and quadriceps muscles (rectus femoris in particular as it also aids with hip flexion), thus evaluation of these muscles for strength, flexibility, and palpating for tonicity and tenderness is necessary to aid management decisions.

It is important to evaluate lumbar ranges of motion when assessing patients with suspected psoas major myofasciopathy, particularly as active and passive extension may be limited by a tight psoas major.[45] The flexibility of the psoas major muscle can be further assessed with orthopaedic testing using the Thomas test, Yeoman's test or Gaenslen's test.[40,43,44] The Thomas test is traditionally thought to help differentiate tight hip flexors (including primarily psoas major) from tight quadriceps femoris muscles.[43,44] Both Yeoman's test[44] and Gaenslen's tests[43,44] are generally acknowledged as sacroiliac joint provocation maneuvers, however these tests do involve passive hip extension and observation of hip extension restriction and pain during these maneuvers could implicate psoas major, particularly if pain is elicited anteriorly.[45]

A final examination procedure of interest for ruling out coxa saltans is the Snapping Hip Test.[40,41] This involves the examiner attempting to reproduce the snapping of the hip with the patient supine with a flexed and abducted hip that is brought into extension and adduction by moving the hip into neutral position while palpating and listening for a snap or click.[40,46] A modification of this maneuver involves adding external hip rotation to the initially flexed and abducted position and in returning the patient to neutral position adding in internal rotation to the adduction and extension required.[40]

The conservative treatment of a psoas major myofasciopathy has not been previously reported in the literature. Like many other myofascopathies, most of the literature surrounding them is anecdotal at best. Initial treatment should focus on immobilizing the injured muscle for approximately three to five days to prevent further retraction of the strained muscle while attempting to reduce pain by using various modalities.

After the acute treatment phase, the clinician may add soft tissue mobilization and light resistance training (isometric muscle contraction/activation exercises) and progress towards weight-bearing exercises to facilitate more rapid and intensive capillary in-growth to the damaged area, as well as improved myofiber regeneration. It is important to maintain the ranges of motion of the lumbosacral spine and hip joints and prevent arthrogenic muscle inhibition through proprioceptive exercises and joint manipulation/mobilizations, as these joints may have been coincidentally injured. During the rehabilitation program, a gradual progression of exercises should be implemented beginning with isometric training followed by concentric training. Once these exercises are tolerated the patient should begin eccentric dynamic training.

The decision to initiate sport-specific training should be based upon the following criteria: whether the patient can perform basic movements that utilize the injured muscle without pain; whether the patient has similar strength levels between the injured muscle and its contralateral counterpart; and whether the patient is able to stretch the injured muscle to approximately the same length as the contralateral muscle. This phase of rehabilitation should be supervised and a gradual progression of sport-specific activities should be employed.

Conclusion

The critical elements of correctly diagnosing a psoas major myofasciopathy include the absence of bruising and significant swelling, with the presence of restricted ranges of motion and muscular pain on palpation and resisted specific muscle testing. Clinicians should be aware of the anatomy and biomechanical influence that this muscle has on lumbar spine biomechanics and stability when assessing and treating patients. It is thought that a stable spine along with increased muscle endurance is protective and therefore may help to reduce the incidence of low back pain. It is difficult to draw conclusions from a case report but based on the literature it is the authors' contention that psoas major myofasciopathy should be considered among the differential diagnoses for low back pain. Increased knowledge of this condition should aid clinicians in selecting the most appropriate methods for its treatment and rehabilitation.

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Anatomy and physical examination of the knee menisci: a narrative review of the orthopedic literature

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Objective: *The objective of this study was to review the physical examination tests available to a practitioner in order to arrive at a clinical diagnosis or suspicion of a meniscal lesion.*

Background: *The menisci transmit weight bearing forces and increase stability of the knee. The menisci also facilitate nutrition, provide lubrication and shock absorption for the articular cartilage and promote knee proprioception. The combinations of torsional and axial loading appear to be the cause of most meniscal injuries. Diagnosis of acute knee injuries has long been a topic for discussion throughout the orthopedic literature. Many clinical tests and diagnostic studies have been developed to increase the clinician's ability to accurately diagnose these types of disorders of the knee.*

Conclusion: *The accuracy of all diagnostic tests is thought to be dependant upon the skill of the examiner, and the severity and location of the injury. The multitude of tests described to assess meniscal lesions suggests that none are consistently reliable. However, recent research has focused on a composite score to accurately predict meniscus lesions. The combination of a comprehensive history, multiple physical tests and diagnostic imaging for confirmation is typical for a clinical meniscal lesion diagnosis while the gold standard remains the arthroscopic procedure itself.*
(JCCA 2009; 53(4):319–333)

KEY WORDS: knee, meniscus, tests, orthopedic

Objectif : *L'objectif de cette étude était de passer en revue les examens physiques que peut faire passer un praticien pour en arriver à un diagnostic clinique ou un soupçon de lésion méniscale.*

Contexte : *Le ménisque transmet les forces de charge et accroît la stabilité du genou. Le ménisque facilite également la nutrition et fournit de la lubrification et une absorption des chocs pour le cartilage articulaire, en plus de promouvoir la proprioception du genou. Les combinaisons de charge de torsion et axiale semblent être la cause de la plupart des blessures méniscales. Le diagnostic d'une blessure aiguë au genou est depuis longtemps un sujet de discussion dans la littérature orthopédique. Bon nombre de tests cliniques et d'études diagnostiques ont été créés pour accroître la capacité d'un clinicien à diagnostiquer avec exactitude ces types de troubles du genou.*

Conclusion : *On croit que l'exactitude des tests diagnostiques dépend de l'aptitude de l'examinateur, ainsi que de la gravité et l'endroit de la blessure. La multitude de tests décrits pour évaluer les lésions méniscales suggère qu'il n'en existe pas qui soit fiable de façon constante. Cependant, la recherche récente a mis l'accent sur un résultat combiné pour prédire avec exactitude les lésions méniscales. La combinaison d'antécédents détaillés, de nombreux tests physiques et d'imagerie diagnostique aux fins de confirmation est typique pour le diagnostic clinique d'une lésion méniscale, bien que la norme d'excellence reste la technique arthroscopique.*
(JACC 2009; 53(4):319–333)

MOTS CLÉS: genou, ménisque, tests, orthopédique

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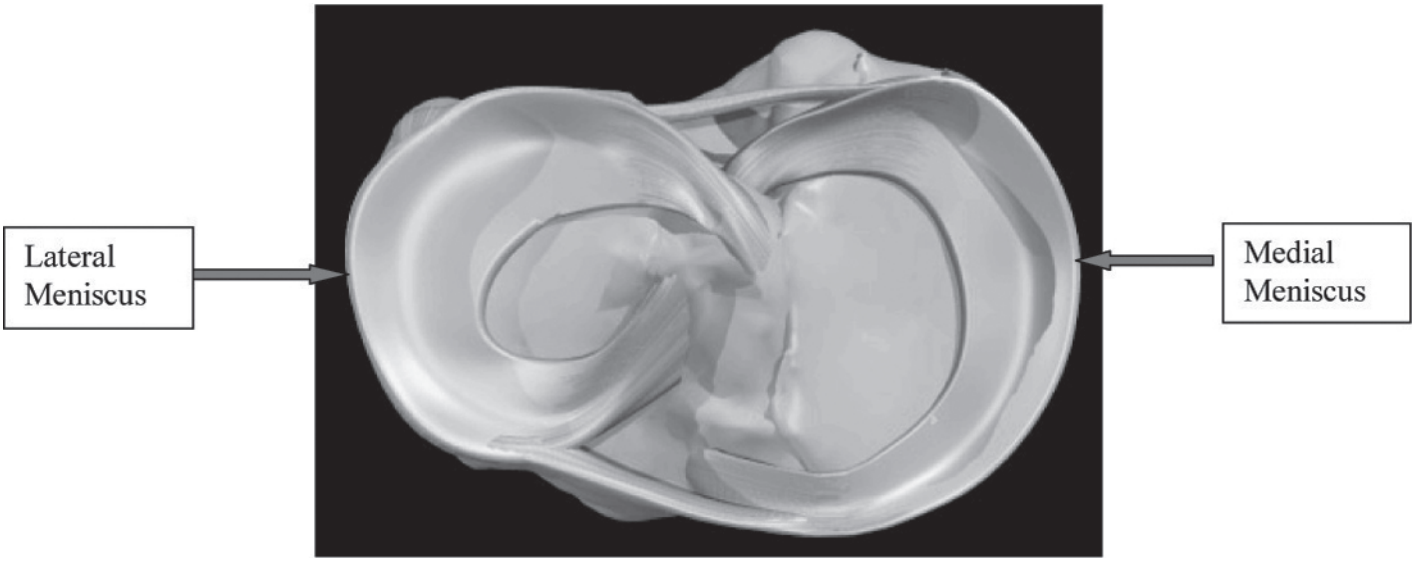
Figure 1 *The superior tibial plateau with the menisci* (courtesy Primal Pictures Ltd. www.primalpictures.com)



Anatomy

The tibio-femoral, or knee joint is the largest articulation in the body. It is a double condyloid joint with 20° of freedom of motion.[1] The condyles of the femur rest in an incongruent manner on the shallow convex surface of the tibia, thus the knee joint relies on other structures to provide both static and dynamic stability.[1] This stability is accomplished by the various soft tissue structures: the anterior and posterior cruciate ligaments, the medial and lateral collateral ligaments, the menisci, the capsule and the muscles crossing the joint.[1–4]

Historically, the medial and lateral cartilages (menisci) of the knee were thought to be vestigial structures that had no real significance with regard to dynamic knee function.[5] However, orthopedic literature clearly shows that the medial and lateral menisci serve very important roles in knee stability, function and dynamic loading and are thus important in maintaining proper joint health.[1–9]

The medial and lateral menisci although similar in function have subtle differences in their shape and thus their biomechanical role in the knee. The knee joint menisci are a pair of wedge shaped (in cross-section), semilunar fibrocartilage structures that are attached to the tibial plateau along the periphery via the coronary ligaments and to the femur via the ligaments of Humphrey and Wrisberg.[10] In addition, the menisci are attached to the patella via the patellomeniscal ligaments which are thickenings of the anterior joint capsule.[3,11] When viewed from above the medial meniscus appears 'C' shaped while the lateral meniscus appears more 'O' shaped[3] (see the shape in Figure 1) The surface of each meniscus is concave superiorly, providing a congruous surface to the femoral condyles and is flat inferiorly to accompany the relatively flat tibial plateau.[3,10] (see the contact depicted in Figure 2)

The medial meniscus is much less mobile during joint motion than the lateral meniscus owing in large part to its firm attachment to the knee joint capsule and medial collateral ligament (MCL).[3] On the lateral side the meniscus is less firmly attached to the joint capsule and has no attachment to the lateral collateral ligament. In fact, the posterior horn of the lateral meniscus is separated entirely from the posterolateral aspect of the joint capsule by the tendon of the popliteus muscle as it descends from the lateral epicondyle of the femur.[3]

The menisci are made of type 1 collagen.[5] Gray (1999) has shown that the menisci are collagenous structures, however the different regions of each meniscus have different cell types. The predominant cell in the inner third are chondrocytic in nature and termed fibrochondrocytes.[7,12] In the middle third the primary cell is fibrocytic

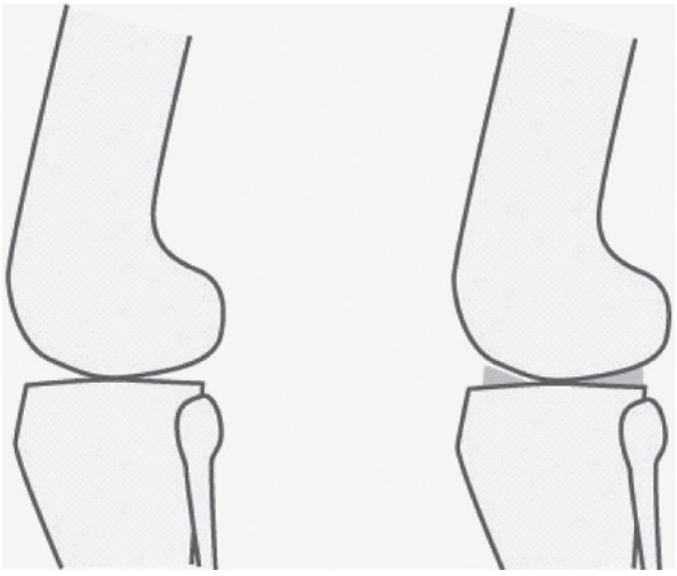


Figure 2 *Contact area between the convex articulating surface of the femur and the flat tibial plateau is increased by the menisci*

and in the outer third of the meniscus the cells are fibroblastic (active fibrocytes).[7] The different cell type in the outer periphery of the meniscus as compared to the central portion of the meniscus helps explain some of the healing properties of the different regions.[13] Bullough et al (1970) demonstrated that the majority of the collagen was arranged in a circumferential orientation which provides "hoop stress" and allows the menisci to elongate as the femur places axial and torsional loads upon the knee joint. Interspersed among the longitudinal fibers are radially oriented fibers that act as "tie rods" to provide additional structural rigidity.[14]

The menisci are supplied mainly by the superior and inferior medial and lateral geniculate arteries which form a peripheral plexus within the synovial and capsular tissues of the knee.[6,13] Nourishment to the outer third of the menisci is provided by small branching capillaries from this genicular plexus that penetrate the knee capsule.[6] Anorczky and Warren (1983) have shown that only the peripheral 10–30% of the meniscus or what has been termed the "red zone" receives this blood supply. In the middle third or the "red/white zone" the blood supply is very limited and in the "white zone" or inner third of the

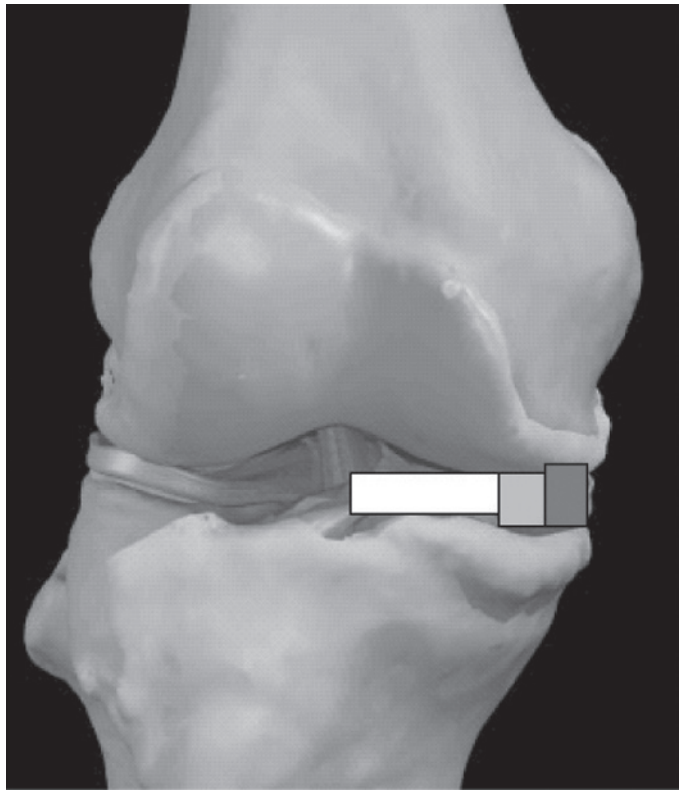


Figure 3 *Showing the red zone (dark grey box), red/white zone (light grey box) and white zone (white box), which represents the blood supply to the meniscus* (courtesy Primal Pictures Ltd. www.primalpictures.com)

meniscus there is no active blood supply with a reliance on diffusion and circulation of the synovial fluid for nourishment.[13] (see the zones of the meniscus in Figure 3) The tenuous blood supply of the inner regions of the menisci is one of, if not the main detrimental factor in the poor healing properties of the meniscus.[2–7,9,13–16]

Biomechanical studies demonstrate that in extension at least fifty percent of the axial forces are transmitted through the meniscus.[1–3,7,12] Conversely in flexion the meniscus transmits eighty-five to ninety percent of axial forces.[1,3,7] In addition to transmitting weight bearing forces and increasing stability, the menisci facilitate nutrition, provide lubrication and shock absorption for the articular cartilage and promote knee proprioception.[1–9]

The combination of torsional and axial loading appear to be the cause of most meniscal injuries.[2,3,7,17] The anatomical arrangement of the knee's soft tissue structures

ensures that during flexion with the tibia internally rotated, the posterior horn of medial meniscus is pulled in towards the center of the joint.[7] This movement can produce a traction injury of the medial meniscus tearing it from its peripheral attachment along the medial joint capsule of the knee.[7,18]

Meniscal tears are classified by their complexity, plane of rupture, direction, location and overall shape.[3] Specifically they may be classified as vertical longitudinal, oblique, horizontal cleavage, radial, and complex.[3] The "bucket handle" tear is the most common type of vertical longitudinal tear and generally amenable to repair.[3] Anatomy and body mechanics dictate that in isolated injuries to the menisci, the medial meniscus is injured more often than the lateral, and more specifically, the posterior horn more-so than the anterior horn.[2,17–19] Seventy-five to ninety percent of all tears sustained during sports such as football, baseball, basketball, and soccer involve the medial meniscus.[3]

Diagnosis of acute knee injuries has long been a topic for discussion throughout the orthopedic literature. Many clinical tests and diagnostic studies have been developed to increase the clinician's ability to accurately diagnose disorders of the knee. Torn menisci or ligamentous structures within the knee cause significant pain and disability and thus, require expeditious management.[9,17–21] Prompt accurate diagnosis of a meniscus injury is necessary considering the diverse and potentially degenerative consequences of this injury for patients, especially those athletically inclined.[17,19]

In athletes, isolated meniscus tears typically result from a single traumatic event.[17,19] Effusion is usually minimal and develops over a twenty-four to forty-eight hour period after the inciting injury.[2,3,6–9,17–21] The occurrence of subsequent swelling secondary to haemarthrosis is approximately twenty percent in a peripheral (vascular) meniscus tear.[8,17,19] The acute meniscus tear is often associated with an immediate limited range of motion especially in flexion.[20,22,24]

The presence of chronic recurrent pain and swelling after exercise is also indicative of a meniscal tear irritating the joint.[3,8,21] Other common symptoms of a meniscus tear include locking, catching, buckling and joint line pain along either the medial or lateral joint line corresponding to the site of injury.[3,6,8,9,17–21] A history of degenerative joint disease or anterior cruciate ligament injury both in the acute setting or with a chronic ACL deficiency, also predisposes one to a meniscus tear.[3]

Important historical questions should include the location of pain, duration of pain, a change in activities, trauma, an acute noise or an audible "pop," swelling surrounding the knee joint, a feeling of give way weakness or buckling, locking or catching of the tibiofemoral articulation, and possibly associated hip, back or thigh pain.[3,8,9,19,21] Concomitant or differential diagnosis' to be considered include degenerative joint disease of the knee, osteochondral defect, cruciate/collateral ligament injury, patellar dislocation, plica, capsular lesion, muscle strain. Of course the practitioner must also rule out the possibility of a vascular injury which carries the risk of more serious sequelae.

A specific history and physical examination should give the clinician clues toward the diagnosis of a lesion of either meniscus. Although the location of pain will be similar it should also be noted that degenerative meniscal tears may present spontaneously without trauma or apparent mechanism. Clinically, the orthopedic literature abounds with several descriptions of physical examination procedures and diagnostic tests to specifically assess the integrity of the menisci, such as joint line tenderness (JLT),[24] Apley compression test,[23] Anderson medial-lateral grind test,[25] McMurray's test,[26] Bounce home maneuver[24] and newer tests such as the Axially loaded pivot shift test,[10] KKU knee compression rotation test[27], Ege's test[22] and Thessaly test.[28] Therefore, we reviewed the medical literature to summarize the available evidence about the diagnostic accuracy of the diagnostic procedures for assessing meniscal lesions of the knee. We conducted a literature search of MEDLINE (1966–2007) to identify articles written in English. The Medical Subject Headings (MeSH) terms "knee," "knee injury" and "meniscus" were combined with the terms "physical examination," "diagnosis," "clinical examination," "sensitivity," "specificity," "accuracy," and "screening." In addition the cited references of relevant publications were examined to indentify articles not found on the initial searches. Articles comparing physical examination procedures to the findings of magnetic resonance imaging (MRI) were excluded.

The Tests

Palpation of the medial and lateral joint lines is the most basic procedure of the meniscal assessment.[24] Flexing the

Figure 4 *Apley's Test*

Figure 5 *Anderson Grind Test*

knee and adding tibial rotation either internally or externally allows for easier palpation of the periphery of the medial and lateral meniscus respectively.[24] Muellner et al (1997) have stated that the most important finding in patients with a meniscal tear is localized tenderness along the joint line. It is estimated that sixty to eight percent of patients with meniscal lesions will have a joint line that is painful upon palpation.[24] Palpation of the joint line is induced through digital pressure by the practitioner into the "red zone" of the meniscus. (refer to the "red zone" depicted in Figure 3)

The Apley compression test was originally described by Apley in 1947. Since then the test has been known as "Apley's test." In his article he described the procedure of the test as follows: For this examination the patient lies on his face. To start the examination, the surgeon grasps one foot in each hand, externally rotates as far as possible, and then flexes both knees together to their limit. When this limit is reached, he changes his grasp, rotates the feet inward and extends the knees together again. The surgeon then applies his left knee to the back of the patient's thigh. It is important to observe that in this position his weight fixes 1 of the levers absolutely. The foot is grasped in both hands, the knee is bent to a right angle and the powerful external rotation is applied. This test determines whether simple rotation produces pain. Next, without changing the position of the hands, the patient's leg is strongly pulled upward, while the surgeon's weight prevents the femur from rising off the table. In this position of distraction the external rotation is repeated. Two things can be determined 1) whether or not the maneuver produces pain and 2) still more important, whether the pain is greater than in rotation alone without the distraction. If the pain is greater, the distraction test is positive and a rotation sprain may be diagnosed. Then the surgeon leans well over the patient and with his whole body weight compresses the tibia downward onto the table. Again he rotates powerfully, and if addition of compression had produced an increase in pain, this grinding test is positive and meniscal damage is diagnosed.[23] (refer to the compression and rotation as shown in Figure 4)

The Anderson medial-lateral grind test was originally described by Anderson and published by Anderson and Lipscomb in 1986.[25] The test is performed with the patient supine. The examiner cradles the test leg between the trunk and the arm while the index finger and thumb of the opposite hand are placed over the anterior joint line. A valgus stress is applied to the knee as it is passively flexed to 45°; then a varus stress is applied to the knee as it is passively extended, producing a circular motion to the knee. The motion is repeated, increasing the varus and valgus stresses with each rotation of the knee.[25] (refer to the flexion and valgus stress shown in Figure 5)

The McMurray or "McMurray's" test is arguably the

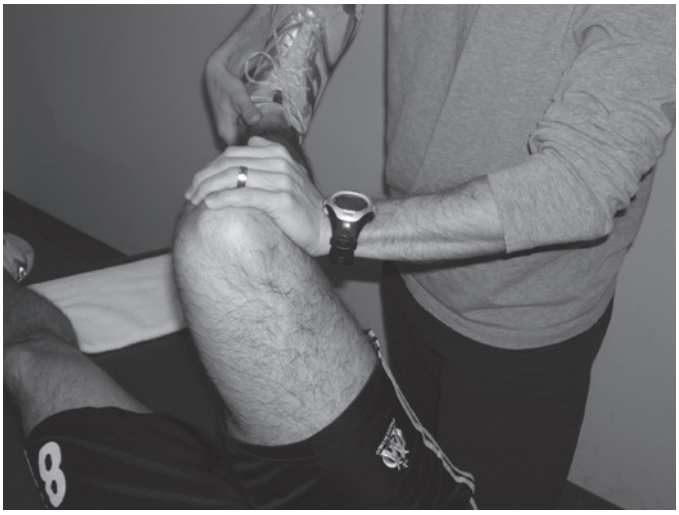


Figure 6 *McMurray's Test*

Figure 7 *Bounce Home Test*

most commonly used test to diagnose tears of the meniscus.[24] The test was first described in 1940 by McMurray.[26] He described the test as follows: In carrying out the manipulation with the patient lying flat, the knee is first fully flexed until the heel approaches the buttock; the foot is then held by grasping the heel and using the forearm as a lever. The knee being now steadied by the surgeon's other hand, the leg is rotated on the thigh with the knee still in full flexion. During this movement the posterior section of the cartilage is rotated with the head of the tibia, and if the whole cartilage, or any fragment of the posterior section is loose, this movement produces an appreciable snap in the joint. By external rotation of the leg the internal cartilage is tested, and by internal rotation any abnormality of the posterior part of the external cartilage can be appreciated. By altering the position of flexion of the joint the whole of the posterior segment of the cartilages can be examined from the middle to their posterior attachment. Probably the simplest routine is to bring the leg from its position of acute flexion to a right angle, whilst the foot is retained first in full internal, and then in full external rotation. When the click occurs with a normal but lax cartilage, the patient experiences no pain or discomfort, but when produced by a torn cartilage which has already given trouble, the patient is able to state that the sensation is the same as he experienced when the knee gave way previously.[26] (refer to the valgus stress and rotation shown in Figure 6)

The Bounce home test evaluates for a possible limitation in the ability of the knee to fully extend.[24] A lack of full knee extension may signify a torn meniscus fragment, an other intra-articular pathology such as a loose body or joint effusion, or a displaced bucket handle tear that requires immediate orthopedic consultation.[24] The test is performed with the patient lying supine and the examiner holding the heel of the foot in his hand. After passively flexing the knee, the knee is then allowed to passively extend. The knee should fully extend and "bounce home" with a sharp endfeel.[29] If full extension of the joint is not complete or has a rubbery end feel, there is probably a torn meniscus or some other blockage present.[29] There have been two modifications of the bounce home test described in the literature. Oni described a modification in which the knee is forcibly extended in a quick jerk.[30] He so named this modification the knee jerk test.[30] With this test pain will occur in the area of tissue injury. In addition, Shybut and McGinty described a modification of simply forced hyperextension of the knee. A blocking of forced extension indicates a positive test and could be the result of a tear of the meniscus.[31] (refer to the hyperextension shown in Figure 7)

Recently four newer diagnostic tests have been published in the sports medicine literature.[10,22,27,28] The Axially loaded pivot shift test as described by Kurosaka et al (1999), is a modification of the pivot shift maneuver as

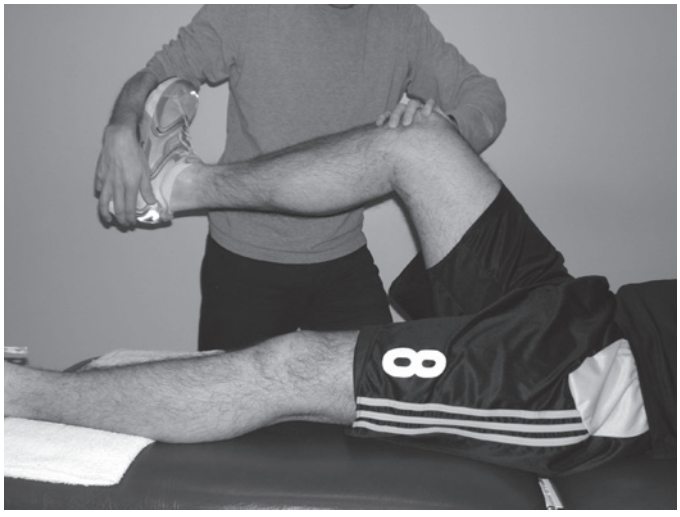


Figure 8 *Axially Loaded Pivot Shift Test*

Figure 9 *Knee Compression Rotation Test*

described by MacIntosh, which is used for the diagnosis of ACL ruptures.[10] For this test the patient lies supine with the knee in full extension. The examiner applies a valgus force to the proximal tibia in full internal rotation. Next, an axial load is transmitted through the tibia and the knee is brought into 30–45° of flexion. The maneuver is repeated whilst returning the knee to full extension again with axial compression.[10] The test is regarded as positive when the patient feels specific pain along the joint line or a click is appreciated by the examiner.[10] (refer to the valgus stress with an internally rotating leg shown in Figure 8)

The knee compression rotation test was developed by Sarachai et al (2007). To carry out this test the patient lies supine on the examining table. The knee is passively flexed by grasping the patient's ankle, with the other hand positioned over the joint line. Knee compression is created by pushing the tibia onto the femur, then the tibia is rotated on the femur both internally and externally. This maneuver is repeated at 120°, 90°, 60°, 30°, and 0°.[26] A positive test is indicated when the patient experiences joint line pain, or the examiner appreciates a click or grinding along either the medial or lateral joint line.[27] (refer to the compression and rotation shown in Figure 9)

Ege's test as described by Akseki et al (2005), is performed with the patient standing with the knees in extension and the feet held 40 cm apart. To detect a medial meniscus tear, the patient squats with both lower legs in maximal external rotation and stands up slowly.[22] For lateral meniscal tears both lower extremities are held in maximum internal rotation while the patient squats and returns to standing.[22] The test is positive when pain and/or a click is felt by the patient or is audible to the examiner.[22] (refer to the squat with rotation as shown in Figures 10 a,b)

The Thessaly test is also a weight bearing test. It was originally described by Karachalios et al (2005). It attempts to reproduce dynamic load transmission in the knee joint. The examiner supports the patient by holding his outstretched hands. The patient then rotates his knee and body both internally and externally three times keeping the knee in slight flexion at 5°.[28] The same testing procedure is then repeated with the patient maintaining the knee in more flexion at 20°.[28] A positive test results in joint line discomfort or locking or catching.[28] (refer to the slightly flexed knee with rotation as shown in Figures 11 a,b)

The diagnostic accuracy of these tests has often been questioned, and a review of the available sports medicine and orthopedic literature reveals conflicting results in regards to their purported usefulness.[24,32,33]

With newer surgical advancements, arthroscopy has become the gold standard for the accurate diagnosis of internal derangements of the knee including cruciate ligament tears, tears of the menisci, cartilage defects and

Figure 10a *Ege's Test (Internal Rotation)*

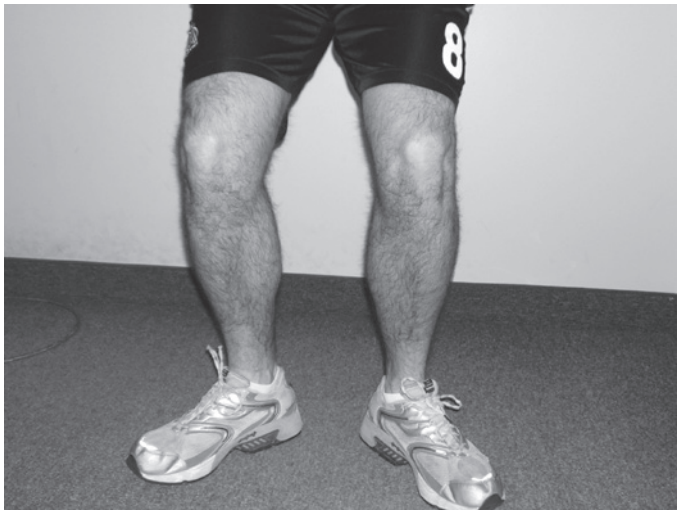


Figure 10b *Ege's Test (External Rotation)*

Figure 11a *Thessaly Test (Internal Rotation)*

Figure 11b *Thessaly Test (External Rotation)*

other less common causes of significant intra-articular knee pain.[33] It is commonplace in the literature investigating diagnostic methods in the physical examination of the knee to compare the findings of the physical examination to the findings at arthroscopy. Thus, in the proceeding discussion all of the reported sensitivities, specificities, and predictive values are in comparison to the findings of arthroscopy which as the gold standard is deemed to have a 100% sensitivity and specificity.

The Value of the Tests

McMurray's Test

Originally described in 1940, McMurray's test has been the subject of much debate and is still considered the primary test for the examination of the knee menisci.[9] Anderson and Lipscomb investigated 100 meniscus tears and found that McMurray's test was positive in 58% thus establishing its diagnostic accuracy. Fowler and Lubliner (1989) conducted a prospective study of 161 consecutive patients to evaluate the predictive value of five common clinical tests for meniscus tear. All patients were suspected of having a meniscal tear with or without concomitant ligament pathology.[34] A positive McMurray had a high correlation with meniscal tear with a specificity of 95%.[34] Evans et al.(1993) evaluated 104 consecutive patients for the accuracy of the McMurray test for the diagnosis of meniscal tear and also looked at the inter-examiner reliability in performing the test. Patients with suspected anterior or posterior cruciate ligament tears were excluded.[35] The authors found that for inter-examiner reliability only a "thud" on the medial joint line with associated tibial external rotation was significantly associated with a medial meniscus tear.[35] The sensitivity of the medial thud was 16% and the specificity was 98% with a positive predictive value of 83%.[35] The sensation of pain on the medial joint line was not correlated with a tear of the medial meniscus.[35] Interestingly, Evans et al (1993) found that for the diagnosis of lateral meniscus tears McMurray's test was less than accurate. They found that lateral joint line pain with internal rotation as originally described by McMurray[26] had a poor correlation with lateral meniscus tears, with a positive predictive value of only 29%.[35] Sensitivity and specificity calculations for McMurray's test in diagnosing lateral meniscus tears were not performed. Corea et al. (1994) also compared the McMurray test to surgical findings, using 93 patients with a suspected meniscus injury. He found a sensitivity of 59%, a specificity of 93%, and a positive prediction value of 83%.[36] On the basis of low sensitivity they considered the McMurray test to be of limited value in clinical diagnosis.[36] Kurosaka et al (1999) evaluated a battery of tests in their cohort of 160 knees, including McMurray's test. They found that none of the tests used had a diagnostic accuracy over 75%, and that for all the tests used the sensitivity was lower than the specificity indicating that only a small percentage of patients with meniscus lesions exhibited a positive sign on physical examination.[10] For McMurray's test the authors found an overall sensitivity of 37%, a specificity of 77% and a diagnostic accuracy of 45%.[10] Akseki et al (2005) compared the results of their new diagnostic test, Ege's test to the results of McMurray's test and JLT. They found that all three tests were well correlated to the arthroscopic findings.[22] Specifically, McMurray's test had a kappa score of 0.321 indicating a fair agreement between a positive test and surgical findings.[22] The authors stratified their results into medial and lateral tears as diagnosed by each specific test.[22] For McMurray's test they established a sensitivity of 67%, a specificity of 69%, a diagnostic accuracy of 69%, a positive predictive value of 80% and a negative predictive value of 53% for lesions of the medial meniscus.[22] They also ascertained a sensitivity of 53%, a specificity of 88%, a diagnostic accuracy of 82%, a positive predictive value of 59% and a negative predictive value of 88% for tears of the lateral meniscus.[22] Karachalios et al (2005) also stratified their calculations into tears of either the medial or lateral meniscus. For McMurray's test they found a sensitivity of 48%, a specificity of 94% and a diagnostic accuracy of 78% for injuries of the medial meniscus.[28] For lesions of the lateral meniscus the authors found a sensitivity of 65%, a specificity of 86% and a diagnostic accuracy of 84%.[28] The authors also examined McMurray's test in patients with a combined ACL injury and a meniscus injury. With combined injuries McMurray's test had a sensitivity of 45%, a specificity of 76% and a diagnostic accuracy of 74%.[28] Recently, Surachai et al (2007) examined McMurray's test and found an overall sensitivity of 70%, a specificity of 82%, a diagnostic accuracy of 73%, a positive predictive value of 92% and a negative predictive value of 48%.[27] In their investigation Surachai et al (2007) found higher percentages for McMurray's test when used in the diagnosis of medial menis-

cus tears as compared to lateral lesions.[27] In their study of the composite knee examination, which included a battery of tests, Lowery et al (2006) found a sensitivity of 20%, a specificity of 96% and a positive predictive value of 84% for McMurray's test. In a meta-analysis Scholten et al (2001) reviewed the available medical literature concerning various tests of the knee examination including the presence of effusion, joint line tenderness, McMurray's test and Apley's compression test. They identified eleven studies of substantial quality that investigated McMurray's test.[37] Scholten et al pooled the results of the individual studies deemed high quality and found an overall sensitivity of 48% and a specificity of 86%.[37] The authors did find that McMurray's test was the only physical examination procedure that had a favorable estimated positive predictive value.[37] The negative predictive value of all the tests reviewed was poor.[37] In another published review, Solomen et al (2001) found that the overall sensitivity of McMurray's test was 53% and the specificity was 59%. More recently in a rigorous systematic review, Ryzewicz et al (2007) identified five research studies evaluating McMurray's test. The authors noted that of the articles reviewed there was a large difference in the reported overall diagnostic accuracy of the test. They speculate that this is due to the varying criteria for clinically diagnosing a meniscus tear using McMurray's test.[38] For example, some articles used a palpable or audible thud as a positive test, whereas others used solely the recreation of pain to constitute a positive result. As such, this detection bias plays a role in the differing results between studies. A lower threshold for a positive test will obviously detect more tears, thus improving the sensitivity however this comes at the expense of specificity.[38]

Joint Line Tenderness

In the most recent investigation evaluating joint line tenderness (JLT), Wadley et al (2007) attempted to determine the positive predictive value of posterior joint line tenderness in diagnosing posterior horn meniscus tears. Seventy-one patients scheduled for arthroscopy of their knees took part in the study. Each one of three examiners blinded to the history or previous examination of the patients assessed each knee for joint line tenderness. Their results indicate a positive predictive value of 60% with 33 of 55 patients with JLT having a posterior meniscus tear.[39] The overall sensitivity was 84.6% but the specificity was only 31.2%.[39] In 75.8% of the patients, all three examiners found the same point of maximal tenderness. This corresponded to a kappa score of 0.48 suggesting good inter-examiner reliability.[39] Rose et al (2006) examined 129 knees with a definite history of knee trauma and associated knee pain. They found that tenderness of the joint line had a higher diagnostic accuracy (93%), sensitivity (95%) and specificity (93%) for lateral meniscus tears as compared to tears of the medial meniscus.[32] Similarly, Eren (2003) examined 104 knees with a history of trauma and found that tenderness of the joint line had a higher diagnostic accuracy (96%), sensitivity (89%) and specificity (97%) for the diagnosis of lateral meniscus lesions. In accordance with the results of Rose et al (2006), the rates obtained for tears of the medial meniscus were much lower.[40] In both studies, patients with positive findings for cruciate ligament or collateral ligament injuries were excluded from the study. These three investigations are unique in that they are the only studies performed to date evaluating joint line tenderness as the sole physical examination procedure for diagnosing tears of the meniscus. Five other studies[10,22,25,28,34] evaluated joint line tenderness in combination with other commonly used physical examination procedures. Anderson and Lipscomb (1986) examined the medial-lateral grind test, McMurray's test and joint line tenderness in a series of 100 patients with suspected meniscus tears. The authors found that joint line tenderness was 77% accurate for the diagnosis.[25] The authors did not report the sensitivity, specificity or positive predictive value. Fowler and Lubliner (1989) examined joint line tenderness tests in a cohort of 165 knees. Of those, 125 had joint line tenderness producing a sensitivity of 85% and a specificity of 29%.[34] Akseki et al (2005) compared joint line tenderness to Ege's test, one of the newer diagnostic procedures. In examining 150 knees they found a higher sensitivity for the diagnosis of medial tears (88% vs. 67%), a higher specificity for the diagnosis of lateral tears (80% vs. 44%) and a higher diagnostic accuracy for lateral tears (77% vs. 71%).[22] In examining patients with suspected meniscal pathology with or without an associated ACL rupture and a history of precipitating trauma, Kurosaka et al (1999), calculated a sensitivity of 55% a specificity of 67% and a diagnostic accuracy of 57% for joint line tenderness. Of their cohort 68% had chronic ACL deficiency and acute injuries were excluded in this study.[10] Shelbourne et al (1995) evaluated the correlation of joint line

tenderness with meniscus tear in patients with acute ACL injuries. One hundred and seventy three patients were evaluated for joint line tenderness, and then the meniscus was inspected during subsequent ACL reconstruction.[33] They found that in a knee with an ACL tear, joint line tenderness was neither sensitive nor specific for the presence of an associated meniscal tear.[33] Karachalios et al (2005) investigated five physical examination tests and compared the results to magnetic resonance imaging (MRI). For joint line tenderness, they found a sensitivity of 71% and specificity of 87% and an overall diagnostic accuracy of 81% for the medial meniscus.[28] For the lateral meniscus they found a sensitivity of 78%, a specificity of 90% and a diagnostic accuracy of 89%.[28] With a combined ACL and meniscus lesion they found JLT to be 65% sensitive, 80% specific and had a diagnostic accuracy of 80%.[28] The authors found significant heterogeneity of the sensitivity and specificity of all tests except for Apley's test.[28] Lowery et al (2006) documented a sensitivity of 65% and a specificity of 65% for diagnosing meniscus tears using JLT. They also found a positive predictive value of 55%.[15] Scholten et al (2001) reviewed nine studies that examined the results of joint line tenderness. The overall sensitivity and specificity for joint line tenderness generated from the pooled analysis of all the available literature was 77% and 41% respectively.[37] In addition, the pooled positive predictive value for using joint line tenderness in diagnosing a meniscus lesion was not substantially increased indicating poor additional diagnostic value.[37] Solomon et al (2001) in their review found an overall mean sensitivity of 79% and a specificity of 15%. They calculated that the logical regression (LR) for a positive test was 0.9 (95% CI, 0.8–1.0) and for a negative test 1.1 (95% CI, 1.0–1.3).[18] Essentially the regression means the probability of an anticipated outcome, with a negative test being more probable. More recently, Ryzewicz et al (2007) performed a systematic review on the diagnosis of meniscus tears. In it they identified four studies of acceptable methodological quality investigating joint line tenderness. Their conclusions are very similar to those of Scholten et al (2001), in that joint line tenderness alone is not sufficient for the diagnosis of tears of the meniscus.[38]

Bounce Home Test (Forced Hyperextension)

There are currently no research studies investigating the sensitivity, specificity or diagnostic accuracy of the bounce home test as originally described. Fowler and Lubliner (1989) found that pain with forced hyperextension generated a sensitivity of 44% and a specificity of 86% in their cohort of patients. Kurosaka et al (1999) also included the forced hyperextension test as described by Shybut and McGinty[31] into their investigation. They found a sensitivity of 47%, a specificity of 67% and a diagnostic accuracy of 51% for the maneuver.[10] Lowery et al (2006) found that forced knee hyperextensions generated a sensitivity of 36%, a specificity of 86% and a positive predictive value of 73%. The knee jerk test as described by Oni[27] has not been investigated in the literature.

Apley's Test

Fowler and Lubliner (1989) in their thorough investigation of the clinical examination of the knee meniscus examined the utility of Apley's test. They reported a sensitivity of 16% and a specificity of 80%.[34] More recently Kurosaka et al (1999) reported a sensitivity of 13% and a specificity of 90%. In addition, they reported a diagnostic accuracy of 28%.[10] Karachalios et al (2005) also reported on the clinical examination results of Apley's test with lesions of the medial meniscus determined to have a sensitivity of 41%, a specificity of 93% and a diagnostic accuracy of 75%. Furthermore, for lesions of the lateral meniscus they found a sensitivity of 41%, a specificity of 86% and a diagnostic accuracy of 82%.[28] For combined lesions of the ACL and either the medial or lateral meniscus Karachalios et al (2005) attained a sensitivity of 20%, a specificity of 84% and a overall diagnostic accuracy of 59% for Apley's test. It should be noted that the authors used MRI as the gold standard.

(Anderson's) Media-Lateral Grind Test

Anderson and Lipscomb (1986) reported on and investigated a manipulative test for the diagnosis of meniscus tears. They compared the results of the medial-lateral grind test to those of McMurray's test in one hundred patients with a suspected meniscus tear. All patients with a suspected ligament tear of the cruciates or collaterals were excluded.[25] There were a total of 100 tears of the meniscus occurring in 93 patients.[25] The authors report that the medial-lateral grind test was positive in 68% of the knees examined, thus reporting a diagnostic accuracy of 68%.[25] The authors did not report sensitivity and specificity.

Thessaly Test

Karachalios et al (2005) examined the sensitivity, specificity and diagnostic accuracy of this new dynamic weight bearing test. In this investigation subjects were examined clinically with tests of JLT, McMurray's, Apley's and the newly described Thessaly test at 5° and 20° of knee flexion.[28] Two hundred and thirteen patients with a suspected meniscus injury on the basis of the clinical history and mechanism of injury (Group A) were investigated and compared to a group of 197 volunteers with no injury to the meniscus (Group B).[28] All patients underwent an MRI of the knee and those in Group A had further therapeutic arthroscopy.[28] The results indicate that for injury to the medial meniscus the Thessaly test at 5° knee flexion is 66% sensitive, 96% specific, and 86% accurate.[28] For the lateral meniscus it is 81% sensitive, 91% specific, and 90% accurate.[28] The Thessaly test at 20° of knee flexion is 89% sensitive, 97% specific, and 94% accurate for medial lesions and 92% sensitive, 96% specific and 96% accurate for lateral lesions.[28] For combined lesions of the ACL and either of the menisci the Thessaly test at 5° of knee flexion was 65% sensitive, 80% specific and had a diagnostic accuracy of 80%.[28] The test performed at 20° of knee flexion performed at 80% sensitivity, 91% specificity and had a diagnostic accuracy of 90% for these combined ligament and meniscus injuries.[28] It is evident that with an associated ACL tear the test becomes less accurate. Overall this new dynamic test performed better than all other tests evaluated in this study. As a result, the authors recommend the Thessaly test as the preferred first line test for the diagnosis of meniscus tears.[28]

Ege's Test

Akseki et al (2005) compared the value of this new weight bearing diagnostic test with the two other most commonly used tests: JLT and McMurray's test. Of 150 knees in 150 patients, 89 reported a specific history of trauma.[22] All patients had a radiographic examination and underwent arthroscopic surgery.[22] There were no statistically significant differences between the three tests in detecting a meniscus tear, however there were evident differences in terms of the sensitivity, specificity, and diagnostic accuracy. Ege's test had a sensitivity of 67%, a specificity of 81% and a diagnostic accuracy of 71% for tears of the medial meniscus.[22] For the lateral meniscus Ege's test had a sensitivity of 64%, a specificity of 90% and a diagnostic accuracy of 84%.[22] Also, Ege's test had the highest overall positive predictive value for medial meniscus tears (86%).[22] Overall, the accuracy of Ege's test was equal to that of JLT and was superior to McMurray's test. It was also the most specific test for both medial and lateral meniscus tears. The authors concluded that by using Ege's test they were able to diagnose meniscal lesions as accurately as JLT but with an increased specificity and thus, Ege's test should be incorporated into routine knee examination.[22]

KKU Knee Compression Rotation Test

Surachai et al (2007) compared the sensitivity, specificity, and diagnostic accuracy of this new test with McMurray's test. Sixty-eight patients with suspected meniscus injury and who were scheduled for arthroscopy were examined using the KKU knee compression rotation test and McMurray's test.[27] KKU knee compression rotation test was positive in 46 of 51 torn menisci, while McMurray's was positive in 39 of 51 torn menisci.[27] This generated an 86% sensitivity, 88% specificity and 87% diagnostic accuracy for the KKU knee compression rotation test, which was far superior to the results of McMurray's test.[27] In 35 cases of meniscus lesions both tests were positive. This had a bearing on the sensitivity, increasing it to 90% when both tests were positive, however the specificity and diagnostic accuracy were not significantly affected by the addition of a positive McMurray test.[27]

Axially Loaded Pivot Shift Test

Kurosaka et al (1999) assessed the significance of the Axially loaded pivot shift test in the diagnosis of chronic meniscus lesions. One hundred and sixty knees were examined using this test as well as McMurray's test, Apley's test, JLT and pain on forced extension.[10] All patients had knee symptoms for at least 8 weeks and were examined for isolated tears of the meniscus or with an associated ACL rupture.[10] The results indicate that 68% percent of the knees examined had a chronic ACL deficiency and that there were 144 meniscus tears evident at arthroscopy.[10] On the clinical examination the Axially loaded pivot shift test had the highest sensitivity (71%) compared to all other tests.[10] The test also showed high specificity (83%) and had a significantly higher diagnostic accuracy than the other tests used.[10] Incidentally only 13 knees exhibited a click with this test, with the majority of patients examined

experiencing pain with a positive result. This was more apparent in those with chronic ACL deficiency.[10]

Composite Score

Often the diagnosis of a meniscus tear is a difficult one to make even for the experienced clinician. This is because a variety of signs and symptoms of lesions of the meniscus have been proposed in the literature. Much like the examination of specific diagnoses in other joints of the body no one test has been shown to be pathognomonic for the diagnosis of a meniscus tear.[15] The clinician relies upon an accurate history/mechanism and a variety of physical examination procedures to arrive at a diagnosis of a lesion of the meniscus. It is from the results of these tests that the clinician then formulates a treatment plan. Recently, the literature has begun to explore the accuracy of a set of tests performed at the same time in the generation of a specific diagnosis of a meniscus tear. Referred to as a composite score, it attempts to assess the efficacy of a variety of procedures in detecting pathology. Scholten et al (2001) in their review conclude that the physical examination procedures they analysed (effusion, JLT, McMurray's test) in isolation do not seem to be very helpful in guiding clinical decision making. They state that combining the results of the various tests might improve the diagnostic accuracy of detecting a meniscus lesion.[37] Oberlander et al (1993) investigated the accuracy of the clinical examination for intra-articular knee disorders, including tears of the meniscus. Of 296 knees included in the study the correct diagnosis was made 56% of the time.[41] Included, were other intra-capsular knee pathology besides solely meniscus lesions. When only one knee pathology was present the diagnostic accuracy was 72%.[41] As the number of pathologies increased the diagnostic accuracy of the knee examination decreased. Specifically, the authors documented a sensitivity of 87%, a specificity of 93% and a diagnostic accuracy of 90% for tears of the medial meniscus.[41] For the lateral meniscus the sensitivity was 81%, the specificity was 93% and the diagnostic accuracy was 95%.[41] O' Shea et al (1996) examined 156 knees for various pathology. Using a standardized composite knee exam the authors recognized the correct primary diagnosis in 83% of the knees.[29] Of those given a secondary diagnosis (57 knees) the diagnosis was correct at a rate of 54%.[29] Sixty-eight knees were diagnosed with medial meniscus tears, 60 of which were correctly diagnosed on physical examination.[29] This generated a sensitivity of 88%, a specificity of 77% and a diagnostic accuracy of 82%.[29] Of 49 lateral meniscus tears, only 25 were correctly diagnosed pre-operatively.[29] This produced a sensitivity of 51%, a specificity of 90% and a diagnostic accuracy of 78%.[29] Muellner et al (1997) examined a cohort of 93 competitive athletes. Group 1 included 57 patients who also underwent arthroscopic surgery. Group 2 consisted of 36 patients who underwent further MRI. Of those patients in Group 1 the diagnosis of a meniscal lesion was confirmed in 51 of 57 patients.[19] Of the 44 medial meniscus lesions all were diagnosed preoperatively.[19] Seventeen medial menisci were normal at arthroscopy of which 13 were correctly diagnosed.[19] The sensitivity was 100%, the specificity was 76%, the diagnostic accuracy was 93%, the positive predictive value was 91% and the negative predictive value was 100%.[19] Of the 13 torn lateral menisci, 12 were correctly identified on examination.[19] Forty four lateral menisci were normal, 43 of which were determined on physical examination.[19] The sensitivity calculated was 92%, the specificity was 98%, the diagnostic accuracy was 96%, the positive predictive value was 92% and the negative predictive value was 98%.[19] Solomon et al (2001) reviewed nine meniscal studies of which five reported the accuracy of the composite examination (averaged the results of several meniscal tests). The mean sensitivity of the composite examination was 77% and the specificity was 91%.[37] Recently, Lowery et al (2006) investigated the composite examination for meniscus pathology in a cohort of 635 knees with either acute or chronic injury.[15] The examination consisted of McMurray's test, forced hyperextension, JLT and pain with forced flexion, in addition to a history of mechanical symptoms.[15] Two hundred and nine knees then underwent arthroscopic evaluation, while the remainder did not.[15] The results indicate that with a history of mechanical symptoms and a positive result on four tests examining the meniscus the sensitivity is 11%, the specificity is 99% and the positive predictive value is 92%.[15] Overall, as the number of findings indicative of meniscus pathology increased so did the PPV of finding a lesion of the meniscus. Interestingly the authors found that when concurrent ACL pathology was present, the positive predictive value of the composite examination decreased to 67%.[15] Ryzewicz et al (2007) indicated that a composite examination for meniscal injuries performs much better than any individual

Table 1 *Summary of Ranges for Sensitivity and Specificity of the Tests*

Test	Sensitivity Range	Specificity Range
McMurray's	16–70%	59–98%
Joint Line Tenderness	55–95%	15–97%
Bounce Home (forced hyperextension)	36–47%	67–86%
Apley's	13–41%	80–93%
Thessaly	65–92%	80–97%
Ege's	64–67%	81–90%
KKU (compression rotation) (1 study)	86%	88%
Axial Loaded Pivot Shift	71%	83%
Composite Score	11–100%	77–99%

test used in isolation. This suggests that a synthesis of examination findings used in conjunction with an historical account of the knee injury (mechanism) must be refined for an accurate diagnosis of a meniscus lesion.[38]

Discussion

The sports medicine and orthopedic literature is full of various articles describing various signs and symptoms as well as diagnostic procedures in an attempt to ensure an accurate diagnosis of a meniscus lesion. Historically, from a physical examination perspective, this has included joint line tenderness, McMurray's test and Apley's compression test. Recently, there have been several newly developed physical examination procedures to further address this diagnostic challenge. Our goal was to collect and summarize the available evidence concerning the diagnostic accuracy of both the older and newer diagnostic tests for assessing injuries of the knee meniscus. On the basis of the current review, it is evident that there is a lack of consistency amongst the tests and that the more recent tests have not yet undergone rigorous scientific investigation. The accuracy of these tests seems to be relatively poor suggesting, if used in isolation, there is little evidence to support the notion that an accurate diagnosis of a meniscus lesion can be made through a physical examination, with the current orthopedic tests. Arthroscopy is truly the only gold standard. The recent research has focused on a composite score to accurately predict meniscus lesions and the research in this area is promising as the results more accurately predict the clinical situation of not relying solely on one test. Further, the composite examination can improve the clinician's ability to diagnose meniscus tears more accurately. This improved diagnostic ability allows the physician to better counsel the patient regarding the possible management options. Unfortunately at this time it has not been well established as to which tests should be included in the composite examination. From our review it seems logical that any meniscus evaluation should include both McMurray's test as well as joint line tenderness. The Thessaly test, has shown some promise as a good screening procedure, however future research should attempt to further define its diagnostic accuracy.

Conclusion

Future research in the area of diagnosing meniscus tears is warranted. Currently the need for utilizing more advanced diagnostic methods or specialty referral can really only be based on the severity of the patient's complaints and a high index of suspicion by the practitioner. A composite score in which a practitioner uses a battery of tests to suggest the diagnosis is recommended. Further investigations into the statistical value of all the commonly used tests, which combinations of tests should be used and the importance of combining the physical examination with aspects of history taking is needed before any further evidence based recommendations can be made.

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Ganglion cyst on the posterior cruciate ligament: a case report

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Objective: *To present the diagnostic and clinical features of a ganglion cyst located on the posterior cruciate ligament and create awareness amongst clinicians of this uncommon diagnosis.*

Clinical Features: *A 24-year old woman complaining of intermittent left knee pain brought on by an increase in mileage during her training for a half-marathon. A diagnosis of mild chondromalacia patella and a ganglion cyst on the posterior cruciate ligament was made via diagnostic imaging.*

Intervention and outcome: *Patient was followed up with imaging. The patient chose to withdraw a surgical consult due to patient preference. No conservative treatment was provided.*

Conclusion: *Although chondromalacia patella is the more probable, a secondary diagnostic consideration in this patient could be a ganglion cyst. A ganglion cyst on the posterior cruciate ligament is an uncommon diagnosis and the clinical manifestations are variable and non-specific. It is important to be aware of its clinical features and to obtain appropriate methods of imaging to generate the diagnosis promptly.*
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KEY WORDS: intra-articular cysts, knee, ganglia, posterior ganglion cyst

Objectif : *Présenter les caractéristiques cliniques et diagnostiques d'un kyste ganglionnaire situé dans le ligament croisé postérieur et sensibiliser les cliniciens à propos de ce diagnostic rare.*

Caractéristiques cliniques : *Une femme de 24 ans se plaignant de douleur intermittente au genou gauche provoquée par une hausse du kilométrage lors de son entraînement pour un demi-marathon. Un diagnostic de légère chondromalacie de la rotule et un kyste ganglionnaire sur le ligament croisé postérieur a été posé au moyen de l'imagerie diagnostique.*

Intervention et résultats : *On a suivi la patiente avec l'imagerie. La patiente a choisi d'obtenir l'avis d'un chirurgien à la suite d'une orientation. Aucun traitement conservateur n'a été prodigué.*

Conclusion : *Bien que la chondromalacie de la rotule soit la plus probable, un kyste ganglionnaire pourrait être une autre considération de diagnostic avec cette patiente. Un kyste ganglionnaire sur le ligament croisé postérieur constitue un diagnostic rare et les manifestations cliniques sont variables et non spécifiques. Il est important d'en connaître les caractéristiques cliniques et d'avoir les méthodes d'imagerie appropriées pour parvenir rapidement au diagnostic.*
(JACC 2009; 53(4):334–338)

MOTS CLÉS : kystes intraarticulaires, genou, ganglion, kyste ganglionnaire postérieur

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Introduction

Intra-articular ganglion cysts of the knee are uncommon. During an autopsy in 1924, Caan first reported a ganglion cyst on the anterior cruciate ligament (ACL).[1] Intra-articular cysts are predominantly incidental findings on MRI and arthroscopy with the reported prevalence of 0.2% to 1.3% and 0.6% respectively.[2,3] Ganglion cysts are found to be more often associated with the ACL rather than the posterior cruciate ligament (PCL). Krudwig et al reported 85 intra-articular ganglions of the knee in 8,000 arthroscopically examined knees over a 15 year period.[4] Of that amount, 49 ganglions were associated with the ACL and 16 associated with the PCL, while the remaining were associated with the menisci, infrapatellar fat pad, and medial plica.[4] In this case report a ganglion cyst on the PCL is presented and its etiology, clinical presentation and means of diagnosis are discussed.

Case

A 24-year-old female training for a half marathon presented with left knee pain. She reported no history of trauma. The pain began as mileage increased in the training program. She reported that she could hardly walk after a 16 km run. Running 5 km and less was well tolerated. Pain lasted for 2–3 days at a time and mainly occurred with running and lunges. She described pinpoint sharp pain with light touch located on the most superomedial aspect of the patella. There was pain with kneeling, and at full extension. She also reported an intra-articular transient clicking sensation while moving the knee from flexion to extension. The patient began pain free activities of swimming and cycling to maintain fitness until competing in the half marathon, which she did not run pain free.

Physical examination did not reveal any swelling or bruising. Upon palpation of the superomedial corner of the patella (which measured a diameter of 1cm by 1 cm) the patient experienced extreme tenderness. Pain occurred during active range of motion from 90 degrees of flexion to full extension. Joint line tenderness, McMurray's, Lachman's, anterior drawer, posterior drawer, slocums, Clarke's test, patellar compression test, and patellar lateral apprehension sign were all found to be negative.

Plain film radiographs of the knee were performed and displayed mild joint effusion with blurring of the posterior border of the Hoffa's fat pad and no bone abnormality or loose body (figure 1 and 2). Diagnostic ultrasound examination was performed on both knees and found to be a normal study. A multisequence MRI examination of the left knee was performed revealing mild chondromalacia patella with early delamination of the medial facet, mild infrapatellar fat pad inflammation, and a very small Baker's cyst with no effusion. Lastly, the MRI revealed a multiloculated ganglion cyst adjacent to the PCL posteriorly; extending medially measuring 2 cm (figure 3). The entire process to determine the diagnosis was 3 months.

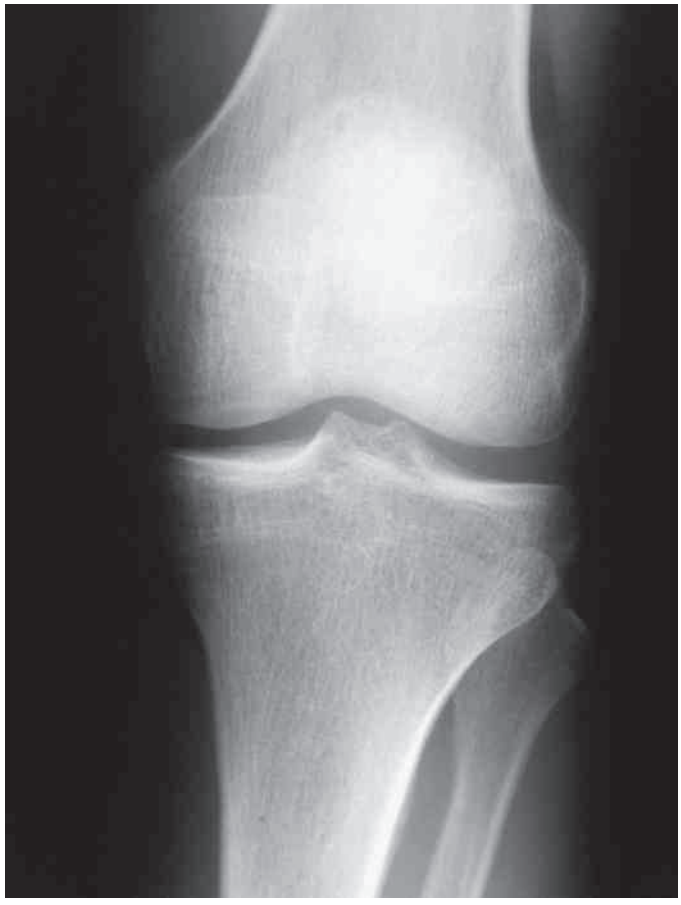

Figure 1 *Plain film Radiograph AP left knee shows no abnormalities*

The patient did not have a surgical consult, but rather modified activity as stated above with swimming and cycling prior to the half marathon. The patient preferred to avoid surgery due to potential complications that can arise.

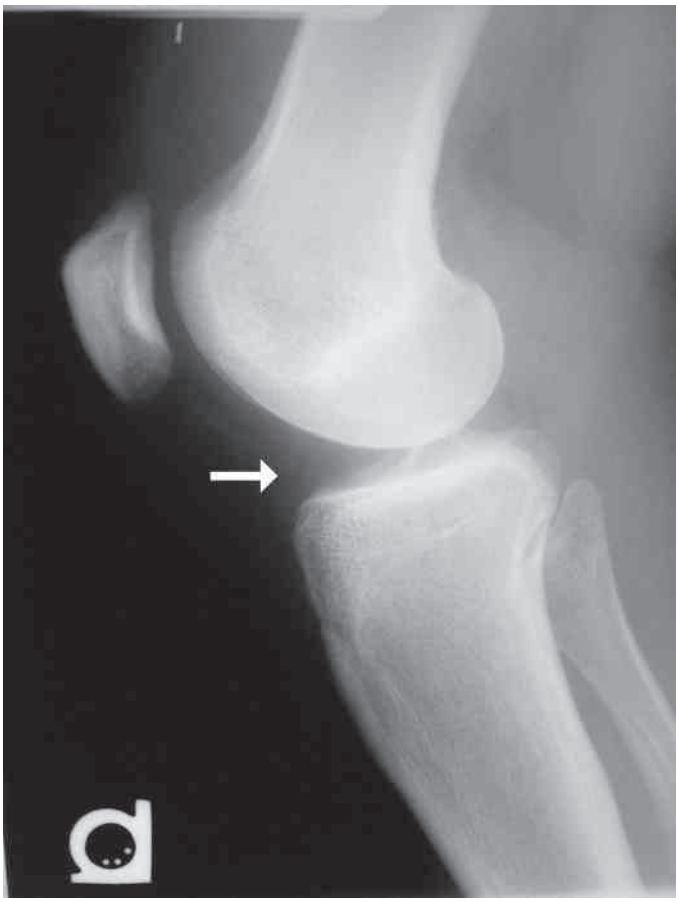

Figure 2 *Plain film Radiograph Lateral left knee shows mild effusion blurring Hoffa's Fat pad*

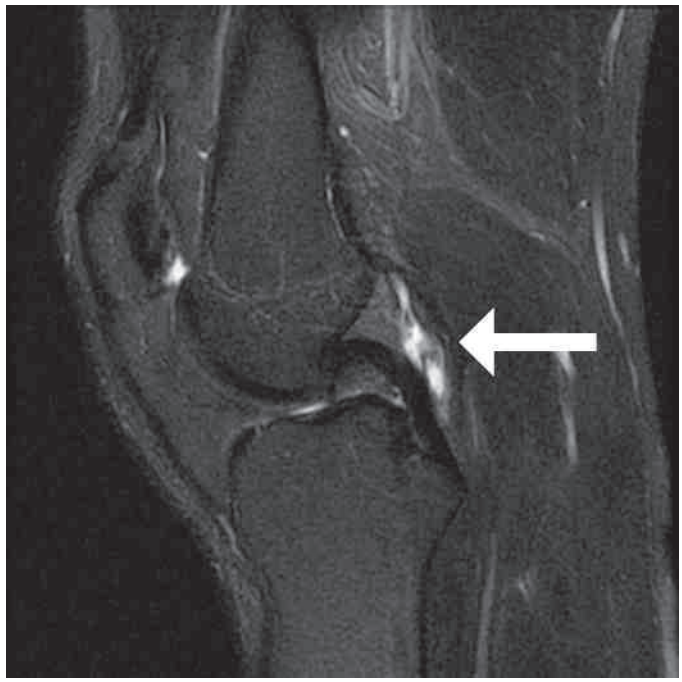

Figure 3 *MRI Sagittal T2 FES S shows Cystic mass posterior to the PCL*

Discussion

Ganglions can arise as cystic lesions that contain a glassy, clear and jelly-like fluid and can occur within muscle, menisci and tendons.[5,6] Ganglia are usually located in areas under continuous mechanical stress.[7] Peri-articular cysts of the knee occur frequently, with Baker's cyst being the most common followed by antefemoral and synovial cysts.[8] However, intra-articular ganglion cysts of the knee are uncommon and mainly arise from the alar folds of the cruciate ligaments.[9] The reported incidence on MRI is 0.2% to 1.3% and 0.6% in arthroscopic studies.[2,3] Although it can occur at any age, the peak incidence of intra-articular ganglion is approximately 20–40 years of age and occurs most commonly in males.[7,10] PCL ganglia are less common than ganglia associated with the ACL. Clinical presentation of PCL ganglia often mimics internal derangement of the knee.[5] They are difficult to diagnose due to the lack of specific clinical signs and symptoms making diagnosis a slow process.

The aetiology of ganglion cysts is unknown. The most widely held physiological explanation attributes cyst formation to mucoid degeneration of collagen and connective tissues.[3,5] A more recent theory postulates that a cyst is formed due to trauma or tissue irritation.[3,5] Additional theories explaining the possible etiology of ganglion cysts include: herniation of the synovium, displacement of synovial tissue during embryogenesis and proliferating pluripotential mesenchymal cells.[3] The suspected aetiology of ganglion cysts of the PCL may be due to mechanical stress experienced on the posterior aspect of the cruciate ligament during knee motion, however this remains a theory.[11]

Ganglia associated with the PCL are usually well defined lobulated and multiloculated along the surface of the ligament.[11] The ganglion is smooth-walled, translu-

cent and white containing clear, highly viscous mucin that consists of hyaluronic acid, albumin, globulin and glucosamine.[11] The site of origin of the main cystic component is posterior to the PCL in 87.5% of cases and anterior to the PCL in 12.5% of cases.[9]

The clinical manifestations of ganglion cysts associated with the PCL are variable and non-specific.[11] The most consistent symptom is knee pain, which is usually intermittent.[3,10,11] The patient may also experience a sensation of fullness during knee motion, limitation in knee range of motion, and flexion or extension block.[10,11,12] In some rare occasions there may be swelling, effusion, joint line tenderness, pain with McMurray's, and a clicking sensation.[11] Orthopaedic examination of the knee will reveal a stable knee.[6] Functional activities, such as running, stair climbing or squatting may exacerbate the symptoms, which were the main findings reported by the patient in this case.[11] Chondromalacia patella most commonly affects the lateral facet of the patella,[13] but was shown to affect the medial facet in the case presented. The most common clinical criteria used to diagnose chondromalacia patella are anterior knee pain with crepitus when flexing and extending the knee, buckling, locking, stiffness and pain with prolonged sitting (Movie Sign), swelling and tenderness.[14] The following orthopaedic tests are found to be the most consistently positive in eliciting the symptoms of chondromalacia patella, patellofemoral compression test and Clarke's test.[14,15] Often times there is aberrant biomechanics of the patellofemoral joint noted as a predisposing factor to developing chondromalacia patella.[13] Symptomatic features of chondromalacia patella and ganglion cysts are somewhat similar leading to a delayed or missed diagnosis. The MRI findings in this case revealed mild chondromalacia patella, and is the most probable explanation of the patient's pain. However, a ganglion cyst should also be considered as a source of pain in this case along with chondromalacia patella. It is suggested that symptoms arising from a ganglion cyst may be associated with the location, size and dimension of the ganglion, which may change over time.[5] The clinical features of a ganglion cyst on the PCL may mimic internal derangement of the knee and should be differentiated from meniscal tears, meniscal cyst, synovial proliferation disorders, synovial chondromatosis, synovial hemangioma and synovial sarcoma.[12]

PCL ganglion cysts are typically diagnosed incidentally while ruling out serious pathology with diagnostic ultrasound and MRI. Plain film radiography is used to rule out intra-articular loose bodies.[6] If this is negative an MRI evaluation should take place to determine if pathology exists.[6] MRI is the modality of choice as it is the most sensitive, specific, accurate and non-invasive method for depicting cystic masses including size and location.[4] MRI evaluation is also used to exclude neoplastic lesions and to detect additional intra-articular pathologies.[4] MRI is a superior tool used to identify anatomic and morphologic relationships of synovial tissue to surrounding structures, such as bone, vessels, and other soft tissues.[6] Ganglia demonstrate fluid characteristic with intermediate signal intensity on T1 and increased signal intensity on T2 spin-echo or gradient-recalled-echo.[16] Signal from a cyst is usually homogenous but also may be inhomogeneous reflecting a degree of fibrous/myxoid change.[16] CT scan and diagnostic ultrasound may also be used to evaluate the knee. Intra-articular cysts appear as well-defined lesion of water density on CT evaluation and as hypoechoic cystic focus on diagnostic ultrasound.[12,16] Special imaging is very useful to determine the diagnosis of a ganglion cyst associated with the PCL promptly.

The main approaches for treatment of PCL ganglion cysts are surgical procedures. The most common and preferred approach to treatment is arthroscopic resection.[1,2,4,7,9] This is due to the fact that it allows a search for associated injuries, enables complete excision, has a decreased recurrence rate and usually results in rapid recovery.[3,4,9] However, arthroscopic resection is expensive, requires hospitalization, and can lead to potential complications, such as ligamentous and popliteal artery injury and infection.[9] CT and ultrasound-guided needle aspiration have also been utilized to treat cystic lesions.[3,7] Ultrasound guided aspiration is quick, readily available, cost effective, and does not emit radiation as compared to CT guided aspiration.[7] However, there are instances in which recurrence of cysts is possible using CT or ultrasound guided aspiration techniques.[4]

Conclusion

Ganglion cysts associated with the PCL are uncommon but can lead to considerable pain. Patients may experience a sensation of fullness during knee motion, limitation in knee range of motion, and occasional swelling, effusion and joint line tenderness. Although orthopaedic

evaluation of the knee will reveal a stable knee, functional activities, such as running, stair climbing or squatting may exacerbate the symptoms. Cystic lesions are mainly diagnosed incidentally using diagnostic ultrasound or MRI. However, MRI is the modality of choice. Painful PCL ganglion cysts can be treated using CT or ultrasound-guided needle aspiration. However, arthroscopic resection is the preferred technique due to better outcomes achieved. It is important to be aware of the clinical manifestations of PCL ganglia to help obtain appropriate methods of imaging, which will generate the diagnosis promptly and guide practitioners in educating their patients regarding treatment options.

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